INTERNATIONAL JOURNAL OF KELOID RESEARCH

PROCEEDINGS OF THE 5TH INTERNATIONAL

KELOID SYMPOSIUM

JUNE 6-8, 2025 | *SHANGHAI, CHINA*

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KELOID RESEARCH FOUNDATION

www.keloidresearchfoundation.org

Dear Friends and Colleagues:

On behalf of the Keloid Research Foundation, and in collaboration with Drs. Xiaoli Wu and Wei Liu of the Department of Plastic and Reconstructive Surgery, Shanghai Ninth People's Hospital, Shanghai Jiao Tong University School of Medicine, we are delighted to extend a warm welcome and cordially invite you to join us at the 5th International Keloid Symposium to be held in Shanghai, China, on June 6th, 7th, and 8th, 2025.

This symposium marks the return of the event to China after the successful 2019 meeting in Beijing. With a population of over 1.4 billion, China treats a significant number of keloid cases annually using a variety of therapeutic techniques, including some novel and unique methods that are widely practiced in the country. As a nation with a rich history, Traditional Chinese Medicine has also played an important role in keloid treatment, often complementing Western medical approaches such as intralesional injections, cryotherapy, surgery, and radiotherapy to enhance therapeutic outcomes. This unique integration of Traditional Chinese Medicine and Western Medicine, which is also likely practiced in other Asian countries, will be presented and discussed at the symposium.

The aim of this symposium is to bring together experts from around the world to share their latest achievements in keloid research and therapy, including basic scientists, dermatologists, general and plastic surgeons, ENT and maxillofacial surgeons, oncologists, laser specialists, and rehabilitation physicians.

This three−day meeting will feature intensive, interactive exchanges of ideas, the latest technologies, and broad experience in the management of keloid disorder, complemented by excellent social activities and exceptional food and dining experiences.

We look forward to welcoming you to Shanghai for this unique event in June 2025. It promises to be an exceptional gathering where leading experts from around the globe will come together to exchange knowledge, explore cutting−edge advancements, and foster collaborations in the field of keloid research and therapy. We are confident that this symposium will be a memorable and enriching experience for all attendees, both professionally and culturally.

Yours sincerely,

The Organizing Committee
5th International Keloid Symposium

Xiaoli Wu, MD, PhD
ShanghaiJiaoTong University
Shanghai, China

Wei Liu, MD, PhD
Shanghai Second Medical University
Shanghai, China

Michael H. Tirgan, MD
Keloid Research Foundation
New York, USA

Jonathan Tsao, MD
Keloid Research Foundation
Toronto, Canada

VENUE

The 5th International Keloid Symposium will be held in the:
Auditorium of Shanghai Ninth People´s Hospital
Address: 8/F, Building 1, No. 639, Zhizaoju Road, Shanghai, China.

The Shanghai Ninth People´s Hospital Affiliated to Shanghai Jiao Tong University School of Medicine is a grade−A tertiary comprehensive hospital that specializes in plastic surgery, oral medicine, orthopedics and other conditions.

LAMONT R. JONES, MD, MBA

THE 2025 RECIPIENT OF THE JOUNI UITTO, MD, PhD
INTERNATIONAL LECTURESHIP

Lamont R. Jones, MD, MBA, is a graduate of Xavier University of Louisiana and completed medical school and otolaryngology, head and neck surgery, training at the University of Michigan. He also completed a facial plastic and craniofacial reconstructive surgery fellowship at SUNY UpState Medical University and an MBA at Michigan State University Broad School of Business. Dr. Jones is Associate Chief Medical Officer of the Henry Ford Medical Group and member of the Department of Otolaryngology at Henry Ford Health. He is a NIH funded, international expert on the treatment and pathogenesis of keloids. Dr. Jones research focuses on keloid biomarker discovery to better understand keloid biology and to identify therapeutic targets. His other clinical interests include craniofacial reconstruction, facial trauma, and facial cosmetics.

DAY 1

PROGRAM
THE 5TH INTERNATIONAL
KELOID SYMPOSIUM

JUNE 6, 2025
FRIDAY

	TIME	SPEAKER	TITLE
	08:00–08:30	*Registration and Morning Coffee*	
	08:30–09:00		**Welcome to Shanghai, & Opening of the 5th International Keloid Symposium**
Session 1	09:00–10:00	**Open Forum: Understanding the True Nature of Keloid Disorder: Toward a Rational and Effective Therapeutic Strategy**	
		Chair: Wei Liu, MD, PhD	**Co–Chair:** Michael H Tirgan, MD
	09:00–10:00		Panel Discussion and Q/A
	10:00–10:30	*Coffee Break*	
Session 2	10:30–12:20	**What's New in Keloid Pathophysiology and Treatment: A 2025 Update**	
		Chair: Xiaoli Wu, MD, PhD	**Co–Chair:** Frank Niessen MD, PhD
	10:30–10:50	*Frank Niessen MD, PhD*	Keloid Research and Therapy: A Worldwide Update on Scientific and Clinical Progress
	10:50–11:10	*Wei Liu, MD, PhD*	An Integrative Approach to Unravel Keloid Pathogenesis and Optimize Therapeutic Outcomes
	11:10–11:30	*Xiaoli Wu, MD, PhD*	Innovative Advances in Keloid Therapy: Breakthrough Techniques from China
	11:30–11:50	*Michael H Tirgan, MD*	Changing the Paradigm in Keloid Treatment: An Oncologist's Insights
	11:50–12:20		Panel Discussion and Q/A
	12:20–13:30	*Lunch Break*	
Special Session	12:30–13:30	*Keloid Research Foundation (19 floor conference room)*	*Keloid Nomenclature Working Group: Aiming to Harmonize Definitions and to Adopt Standard Nomenclature This Session is Open to all speakers*

	TIME	SPEAKER	TITLE
Session 3	**13:30–15:30**	**Evidence–Based Non–Surgical Strategies for Keloid Treatment**	
		Chair: Akash Gunjan, PhD	**Co–Chair:** Bingrong Zhou MD, PhD
	13:30–13:45	*Akash Gunjan, PhD*	Leveraging Machine Learning Using Clinical Data to Predict Response to Intralesional Steroids in Keloid Patients
	13:45–14:00	*Xiaoli Wu, MD, PhD*	LCR Technique for Keloid Treatment
	14:00–14:15	*Tony Chan, MBChB, FCSHK*	Ten Years' Clinical Experience with Intralesional Cryosurgery and Adjunctive Therapies for Keloid Management in Hong Kong
	14:15–14:30	*Bingrong Zhou, MD, PhD*	Novel Needle–Type Electrocoagulation and Combination Pharmacotherapy: Basic and Clinical Studies on Efficacy and Safety in Treating Keloids
	14:30–14:45	*Joanne Smith*	Innovative Compression Technology for Ear Keloids: Reducing Recurrence Rates Effectively
	14:45–15:00	*Ka–Wing Wong, MS, OT*	Innovative Application of Scanning, Printing and Pressure Sensors Technologies to Improve Patient Experience in Auricular Keloids Management
	15:00–15:30		Panel Discussion and Q/A
	15:30–15:45	*Chair: Weil Liu, MD, PhD* *Speaker: Bingrong Zhou, MD, PhD*	CO2 Laser For the Treatment of Facial Atrophic Acne Scars
	15:45–16:00	***Coffee Break***	
Session 4	**16:00–18:15**	**Rethinking Keloid Surgery: Toward Predictable and Long–Lasting Outcomes**	
		Chair: Youbin Wang, MD,PhD	**Co–Chair:** Lamont Jones, MD, MBA
	16:00–16:15	*Youbin Wang, MD,PhD*	Facial Keloid Treatment with Keloid Subepidermal Vascular Network Fap (KSVNFs): A Long–Term Follow–up Study
	16:15–16:30	*Jinglong Cai, MD, PhD*	Treatment Method and Efficacy for Pediatric Keloids
	16:30–16:45	*Ioannis Goutos, MD*	Surgical Management of Large Volume Keloids
	16:45–17:00	*Renliang He, MD, PhD*	Trephination Technique in Treating Keloids
	17:00–17:15	*Mingming Pan*	Retrospective Review of a Single Tertiary Care Medical Center's Surgical Keloid Population Over a 30–Year Period
	17:15–17:30	*Chunmei Wang, MD, PhD*	From Planning to Practice: Key Surgical Techniques in Personalized Keloid Management
	17:30–17:45	*Bianyou Sun*	Sponsor: Treatment and Management Over 10 Thousands Keloid Patients in Beijing Bakang Hospital
	17:45–18:15		Panel Discussion and Q/A
Reception	**18:30–19:30**	**Registered attendee**	
Dinner	**19:30–21:30**	**Registered attendee**	

DAY 2

PROGRAM
THE 5TH INTERNATIONAL
KELOID SYMPOSIUM

JUNE 7, 2025
SATURDAY

	TIME	SPEAKER	TITLE
	08:00–08:30	***Registration and Morning Coffee***	
Session 1	08:30–10:30	**Molecular and Cellular Insights into Keloid Formation**	
		Chair: Yixin Zhang, MD, PhD	**Co-Chair:** Nanze Yu, MD, PhD
	08:30–08:45	*Yixin Zhang, MD,PhD*	Molecular and Immune-Mechanism of Keloid Development
	08:45–09:00	*Mark Fear, PhD*	Understanding the Mechanisms of Keloid Recurrence and Exploring Novel Therapeutic Options
	09:00–09:15	*Zelian Qin, MD, PhD*	The lnhibition of ENO1 Expression Targets the Glycolytic and Lactylation to Regulate Keloid Fibroblast Function
	09:15–09:30	*Chenyu Huang, MD, PhD*	Asporin Inhibits Collagen Matrix-Mediated Intercellular Mechano-Communications Between Fibroblasts During Keloid Progression
	09:30–09:45	*Nanze Yu, MD, PhD*	Understanding and Clinical Practice of Vascular Abnormalities in Keloid Disease
	09:45–10:00	*Chengcheng Deng, PhD*	BMP2-Induced Adam12+ Fibroblasts Dictate Skin Scarring and Fibrosis.
	10:00–10:30		Panel Discussion and Q/A
	10:30–10:50	***Coffee Break***	
Session 2	10:50–12:40	**Evidence-Based Radiotherapy in Management of Keloids**	
		Chair: John Glees, MD FRCR DMRT	**Co-Chair:** Jonathan Tsao, MD
	10:50–11:05	*Jonathan Tsao, MD*	Adjuvant Radiotherapy for the Treatment of Keloids
	11:05–11:20	*Henry Weatherburn, PhD*	A Data-Based Approach to a Keloid Post-Excisional Radiotherapy Treatment (PERT) Pathway
	11:20–11:35	*John Glees, MD, FRCR, DMRT*	Case Studies of Patient Outcomes of Re-Treatment of Recurrent Keloid Tumours by Fractionated Superficial X-Ray Radiotherapy
	11:35–11:50	*Zhongling Qiu*	Isotope Therapy to Prevent Post-Surgical Keloid Recurrence
	11:50–12:05	*Hui Yao, MD*	Electronic Beam Irradiation for Keloid Post-Surgical Treatment
	12:05–12:20	*Jinhu Xu, MD*	Sponsor: Superficial X-ray for Keloid Treatment
	12:20–12:40		Panel Discussion and Q/A
	12:40–13:30	***Lunch Break***	
Special Session	12:40–13:30	*Keloid Research Foundation, 19 floor conference room*	Harmonizing Clinical Endpoints: Response Criteria Working Group for Keloid Trials (open to all speakers)

	TIME	SPEAKER	TITLE
Session 3	**13:30–15:30**	**Surgical Treatment of Keloids in Conjunction with Radiation Therapy**	
		Chair: Xiaodong Chen, MD, PhD	**Co–Chair:** Henry Weatherburn, PhD
	13:30–13:45	*Jinglong Cai, MD, PhD*	Can Keloids Be Cured? Evidence–Based Criteria for Patient Selection and Treatment
	13:45–14:00	*Henry Weatherburn, PhD*	A 15 Year Single Centre Audit of the Outcome of Fractionated Superficial X–Ray Radiotherapy Treatment of Recurrent Keloid Tumours
	14:00–14:15	*Xiaodong Chen, MD, PhD*	Nucleoresection Combined with Punch Extraction and Radiation Therapy: a Keloid Surgical Technique Worth Promoting
	14:15–14:30	*Wei Liu, MD, PhD*	Use of Punch Biopsy Procedure to Treat Wide–Spread Keloids
	14:30–14:45	*Xiaomei Han, MD*	Full–Process Management of Auricular Keloids: The Role of the Innovative ABC Surgical Technique in a Multidisciplinary Treatment Framework
	14:45–15:00	*Dongyun Yang, MD*	Distal Reduction Sutures and Keloid Surgical Treatment
	15:00–15:30		Panel Discussion and Q/A
	15:30–15:45	*Chair: Haiguang Zhao* *Speaker: Wei Liu, MD, PhD*	Sponsor: Application of Wound Tension Reduction Device in the Treatment of Keloids
	15:45–16:00	*Chair: Haiguang Zhao* *Speaker: Wenbo Wang, MD, PhD*	Sponsor: Application of Hyaluronic Acid and Its Functionalization Technology in the Field of Scars
	16:00–16:20	***Coffee Break***	
Session 4	**16:20–18:10**	**Advances in Non–Surgical Management of Keloid Patients**	
		Chair: Cecilia Li, PhD	**Co–Chair:** Patricia Yap, MD
	16:20–16:35	*Cecilia Li, PhD*	Novel Concept and Clinical Practice of Keloid Pressure Therapy
	16:35–16:50	*Eva Chintia Yessica Manalu, BMedSc*	Bridging a Therapeutic Gap: Hydrocortisone Occlusion Dressing after Keloid Excision in Indonesia's National Referral Hospital – A Case Series
	16:50–17:05	*Patricia Yap, MD*	Keloid Tumors and Telemedicine – a Patient's Journey
	17:05–17:20	*Yuting Huang, RN*	Coping Tendencies Play a Partial Mediating Role between Social Support and Anxiety/Depression among Chinese Keloid Patients
	17:20–17:35	*Haiguang Zhao*	Combined therapy of drug injection and laser for keloid
	17:35–17:50	*Lina Ndjock Mbolong, MD*	Understanding the Emotional Impact of Living with Keloids: A Clinical Perspective on Body Image and Psychological Burden
	17:50–18:10		Panel Discussion and Q/A
Reception	18:20–19:00	*Registered attendee*	
Dinner	19:00–21:30	*Registered attendee*	

DAY 3

PROGRAM
THE 5TH INTERNATIONAL
KELOID SYMPOSIUM

JUNE 8, 2025
SUNDAY

	TIME	SPEAKER	TITLE
	08:00–08:30	***Registration and Morning Coffee***	
Session 1	08:30–10:00	**Complexities in Keloid Disorder: Clinical Realities Across Age Groups**	
		Chair: Juliette Loute, MSc	**Co-Chair:** Dan Deng, MD, PhD
	08:30–08:45	*Michael H. Tirgan, MD*	Pediatric Keloids: Case Review, Clinical Patterns, and Treatment Recommendations
	08:45–08:55	*Dan Deng, MD, PhD*	Pediatric Keloid Experience and Treatment at Shanghai Children's Medical Center
	08:55–09:05	*Wei Liu, MD, PhD*	Therapeutic Principle for Pediatric Keloids
	09:05–09:20	*Xiqiao Wang, MD*	Microbiome Dysbiosis Dominated by Rhodococcus Occurs in Keloids
	09:20–09:35	*Juliette Louter, MSc*	How do I Deal with the Keloid Disease: a Qualitative Study Exploring Coping Strategies in a Multicultural Adult Patient Population
	09:35–10:00		Panel Discussion and Q/A
	10:00–10:30	**The Annual Jouni Uitto, MD, PhD International Visiting Professorship and Lecture in Molecular Dermatology Clinical Biomarkers**	
		Chair: Michael H. Tirgan, MD	**Co-Chair:** Wei Liu, MD, PhD
	10:00–10:20	*Lamont Jones, MD, MBA*	The Future of Keloid Disease´s Diagnosis and Treatment
	10:20–10:30		Panel Discussion and Q/A
	10:30–10:45	*Chair: Wei Liu, MD, PhD* *Speaker: Xiaoli Wu, MD, PhD*	Sponsor: The Application of Silicone Products in Scar Treatment
	10:45–11:05	***Coffee Break***	

	TIME	SPEAKER	TITLE
Session 2	**11:05–12:30**	**Abstract Presentation – Clinical Science**	
		Chair: Akash Gunjan, PhD	**Co-Chair:** Chunhui Xie, MD
	11:05–11:15	*Chunhui Xie, MD*	Application of Core Excision in the Treatment of Keloids
	11:15–11:25	*Yan Hao, MD*	Clinical Observation of Subepidermal Vascular Network Flaps in Keloid Patients
	11:25–11:35	*Bing Li, MD, PhD*	Surgery Approaches and Experiences for Keloid Treatment and the Adjunctive Therapy
	11:35–11:45	*Akash Gunjan, PhD*	Rationale Based Repurposing of FDA-Approved Agents for Keloid Management: An in Vitro Study
	11:45–11:55	*Wenbo Wang, MD, PhD*	An Innovative Single-Stage Approach of High-Tension Keloid Excision and Reconstruction Using Acellular Dermal Matrix and Epidermal Skin Grafting
	11:55–12:05	*Zongan Chen, MD, PhD*	Keloids and inflammation: The Critical Role of IL-33 in Epidermal Changes
	12:05–12:30		Panel Discussion and Q/A
	12:30–13:30	***Lunch Break***	
	12:30–13:30	*Michael H.Tirgan, MD*	Bridging Progress: What We Learned in Shanghai and What Lies Ahead – Strategic Planning for Amsterdam 2026(Speakers)
Session 3	**13:30–15:00**	**Abstract Presentation – Basic Science**	
		Chair: Frank Niessen MD, PhD	**Co-Chair:** Lamont Jones, MD, MBA
	13:30–13:40	*Lian Zhang, MD*	Integrated Multi-Omics Unveils the Epigenetic Landscape in the Pathogenesis of Keloid
	13:40–13:50	*Tianhao Li, PhD*	TWIST1 Promote TGF-β Receptor I in Keloid Fibroblasts via Regulating the Stability of MEF2A
	13:50–14:00	*Tianhao Li, PhD*	Integrated Analysis of TWIST1 in Keloid Pathogenesis: Single-Cell Transcriptomics Reveals Fibroblast Heterogeneity and a Novel MEF2A-TBR1 Regulatory Axis
	14:00–14:10	*Mengjie Shan, MD, PhD*	Multi-Omics Analyses Reveal Bacteria and Catalase Associated with Keloid Disease
	14:10–14:20	*Yixin Sun, MD, PhD*	Single-Cell RNA Sequencing Revealed Key Factors of EMT to Promote Fibroblast Activation and Immune Infiltration in Keloid
	14:20–14:30	*Junxian Wen, PhD*	Endothelial Dysfunction in Keloid Formation and Therapeutic Insights
	14:30–15:00		Panel Discussion and Q/A
	15:00–15:30	***Coffee Break***	

	TIME	SPEAKER	TITLE
Session 4	**15:30–17:10**	**Basic and Clinical Research**	
		Chair: Michael H. Tirgan, MD	**Co–Chair:** Wei Liu, MD, PhD
	15:30–15:40	*Michael H. Tirgan, MD*	Contact Cryotherapy in Treatment of Keloid Lesions, Indications and Case Studies
	15:40–15:50	*Tan Yingrou, PhD*	Dual siRNAs Nanoplex Targeting IL–4RA and SPARC Enhance Collagen Reduction in IL–4 Activated Skin Fibroblasts
	15:50–16:00	*Qiannan Li, MD*	Tissue Chondrification and Ossification in Keloids with Primary Report of Five Cases
	16:00–16:10	*Michael H. Tirgan, MD*	Cartesian Model of Clinical Behavior of Keloid Disorder
	16:10–16:20	*Xinwen Kuang, MD, PhD*	Multi–Dimensional Effects of Hydrogen–Rich Materials on Wound Healing and Alleviating Skin Fibrosis
	16:20–16:30	*Shuo Li, MD*	Establishment of a Deep Learning–Based Automated Segmentation and Blood Perfusion Prediction System for Keloids
	16:30–16:40	*Zhijin Li, PhD*	Unraveling the Vascular Tapestry: Endothelial Dysfunction in Keloid Formation and Therapeutic Insights
	16:40–17:10		Panel Discussion and Q/A
	17:10–17:30	*Wei Liu, MD, PhD* *Xiaoli Wu, MD, PhD* *Michael H. Tirgan, MD* *Jonathan Tsao, MD*	***Closing Remarks***

SHANGHAI,CHINA

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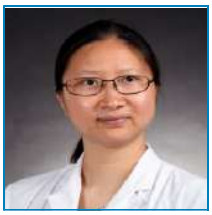

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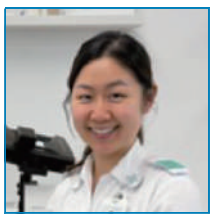

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Contents

Venue /

Lamont R jones MD, MBA– The 2025 Recipient of the Jouni Uitto, MD, PhD International Lectureship /

Program /

Speakers /

DAY 1: FRIDAY JUNE 6, 2025

Session 1 *09:00–10:00*

Open Forum: Understanding the True Nature of Keloid Disorder: Toward a Rational and Effective Therapeutic Strategy / 000

Session 2 *10:30–12:20*

What´s New in Keloid Pathophysiology and Treatment: A 2025 Update

- *An Integrative Approach to Unravel Keloid Pathogenesis and Optimize Therapeutic Outcomes / 000*
- *Innovative Advances in Keloid Therapy: Breakthrough Techniques from China / 000*
- *Changing the Paradigm in Keloid Treatment: An Oncologist´s Insights / 000*

Session 3 *13:30–15:30*

Evidence–Based Non–Surgical Strategies for Keloid Treatment / 000

- *Leveraging Machine Learning Using Clinical Data to Predict Response to Intralesional Steroids in Keloid Patients / 000*
- *LCR Technique for Keloid Treatment / 000*

- *Ten Years' Clinical Experience with Intralesional Cryosurgery and Adjunctive Therapies for Keloid Management in Hong Kong. / 000*
- *Novel Needle-Type Electrocoagulation and Combination Pharmacotherapy: Basic and Clinical Studies on Efficacy and Safety in Treating Keloids / 000*
- *Innovative Applicati on of Scanning, Printing and Pressure Sensors Technologies to Improve Patient Experience in Auricular Keloids Management / 000*

Session 4 *15:45–18:15*

Rethinking Keloid Surgery: Toward Predictable and Long–Lasting Outcomes / 000

- *Facial Keloid Treatment with Keloid Subepidermal Vascular Network Fap (KSVNFs): A Long-Term Follow-up Study / 000*
- *Treatment method and efficacy for pediatric keloids / 000*
- *Surgical Management of Large Volume Keloids / 000*
- *Trephination Technique in Treating Keloids / 000*
- *From Planning to Practice: Key Surgical Techniques in Personalized Keloid Management / 000*
- *Treatment and management over 10 thousands keloid patients in Beijing Bakang Hospital / 000*

DAY 2: SATURDA JUNE 7, 2025

Session 1 *08:30–10:30*

Molecular and Cellular Insights into Keloid Formation / 000

- *Molecular and Immune-Mechanism of Keloid Development / 000*
- *Understanding the Mechanisms of Keloid Recurrence and Exploring Novel Therapeutic Option / 000*
- *The lnhibition of ENO1 Expression Targets the Glycolytic and Lactylation to Regulate Keloid Fibroblast Function / 000*
- *Asporin Inhibits Collagen Matrix-Mediated Intercellular Mechano-Communications Between Fibroblasts during Keloid Progression / 000*

- *Understanding and Clinical Practice of Vascular Abnormalities in Keloid Disease / 000*
- *BMP2-Induced Adam12+ Fibroblasts Dictate Skin Scarring and Fibrosis / 000*

Session 2 *10:50–12:40*

Evidence-Based Radiotherapy in Management of Keloids / 000

- *Adjuvant Radiotherapy for the Treatment of Keloids / 000*
- *A Data-Based Approach to a Keloid Post-Excisional Radiotherapy Treatment (PERT) Pathway / 000*
- *Case Studies of Patient Outcomes of Re-Treatment of Recurrent Keloid Tumours by Fractionated Superficial X-Ray Radiotherapy / 000*
- *Electronic beam irradiation for keloid post-surgical treatment / 000*
- *Superficial X-ray for keloid treatment / 000*

Session 3 *13:30–15:30*

Surgical Treatment of Keloids in Conjunction with Radiation Therapy / 000

- *Can Keloids Be Cured? Evidence-Based Criteria for Patient Selection and Treatment / 000*
- *A 15 Year Single Centre Audit of the Outcome of Fractionated Superficial X-Ray Radiotherapy Treatment of Recurrent Keloid Tumours / 000*
- *Nucleoresection Combined with Punch Extraction and Radiation Therapy: a Keloid Surgical Technique Worth Promoting / 000*
- *Use of Punch Biopsy Procedure to Treat Wide-Spread Keloids / 000*
- *Full-Process Management of Auricular Keloids: The Role of the Innovative ABC Surgical Technique in a Multidisciplinary Treatment Framework / 000*
- *Distal reduction sutures and keloid surgical treatment / 000*
- *Application of Hyaluronic Acid and Its Functionalization Technology in the Field of Scars / 000*

Session 4 *16:20–18:10*

Advances in Non–Surgical Management of Keloid Patients / 000

- *Bridging a Therapeutic Gap: Hydrocortisone Occlusion Dressing after Keloid Excision in Indonesia's National Referral Hospital – A Case Series / 000*
- *Keloid Tumors and Telemedicine – a Patient's Journey / 000*
- *Coping Tendencies Play a Partial Mediating Role between Social Support and Anxiety/Depression among Chinese Keloid Patients / 000*
- *Combined therapy of drug injection and laser for keloid / 000*
- *Understanding the Emotional Impact of Living with Keloids: A Clinical Perspective on Body Image and Psychological Burden / 000*

DAY 3: SUNDAY JUNE 8, 2025

Session 1 *08:30–10:00*

Complexities in Keloid Disorder: Clinical Realities AcrossAge Groups / 000

- *Pediatric Keloids: Case Review, Clinical Patterns, and Treatment Recommendations / 000*
- *Pediatric Keloid Experience and Treatment at Shanghai Children's Medical Center / 000*
- *Therapeutic principle for pediatric keloids / 000*
- *Microbiome Dysbiosis Dominated by Rhodococcus Occurs in Keloids / 000*
- *How do I Deal with the Keloid Disease: a Qualitative Study Exploring Coping Strategies in a Multicultural Adult Patient Population. / 000*
- *Special Lecture:The Future of Keloid Disease's Diagnosis and Treatment/ 000*

Session 2 *11:05–12:30*

Abstract Presentation – Clinical Science / 000

- *Application of Core Excision in the Treatment of Keloids / 000*
- *Clinical Observation of Subepidermal Vascular Network Flaps in Keloid Patients / 000*
- *Surgery Approaches and Experiences for Keloid Treatment and the Adjunctive Therapy / 000*
- *Rationale Based Repurposing of FDA−Approved Agents for Keloid Management: An in Vitro Study / 000*
- *An Innovative Single−Stage Approach of High−Tension Keloid Excision and Reconstruction Using Acellular Dermal Matrix and Epidermal Skin Grafting / 000*
- *Keloids and inflammation: the critical role of IL−33 in epidermal changes / 000*

Session 3 *13:30–15:00*

Abstract Presentation – Basic Science /

- *Integrated Multi−Omics Unveils the Epigenetic Landscape in the Pathogenesis of Keloid / 000*
- *TWIST1 Promote TGF−β Receptor I in Keloid Fibroblasts via Regulating the Stability of MEF2A / 000*
- *Integrated Analysis of TWIST1 in Keloid Pathogenesis: Single−Cell Transcriptomics Reveals Fibroblast Heterogeneity and a Novel MEF2A−TBR1 Regulatory Axis / 000*
- *Multi−Omics Analyses Reveal Bacteria and Catalase Associated with Keloid Disease / 000*
- *Single−Cell RNA Sequencing Revealed Key Factors of EMT to Promote Fibroblast Activation and Immune Infiltration in Keloid / 000*
- *Endothelial Dysfunction in Keloid Formation and Therapeutic Insights / 000*

Session 4 *15:30–17:10*

Basic and Clinical Research / 000

- *Contact Cryotherapy in Treatment of Keloid Lesions, Indications and Case Studies / 000*
- *Dual siRNAs Nanoplex Targeting IL−4RA and SPARC Enhance Collagen Reduction in IL−4 Activated Skin Fibroblasts / 000*
- *Chondrification and Ossification in Keloids with Primary Report of Five Cases / 000*
- *Cartesian Model of Clinical Behavior of Keloid Disorder / 000*
- *Multi−Dimensional Effects of Hydrogen−Rich Materials on Wound Healing and Alleviating Skin Fibrosis / 000*
- *Establishment of a Deep Learning−Based Automated Segmentation and Blood Perfusion Prediction System for Keloids / 000*
- *Unraveling the Vascular Tapestry: Endothelial Dysfunction in Keloid Formation and Therapeutic Insights / 000*

DAY 1: FRIDAY JUNE 6, 2025

SESSION 1 *09:00 – 10:00*

OPEN FORUM: UNDERSTANDING THE TRUE NATURE OF KELOID DISORDER: TOWARD A RATIONAL AND EFFECTIVE THERAPEUTIC STRATEGY

DAY 1: FRIDAY JUNE 6, 2025

SESSION 2 *10:30 – 12:20*

WHAT´S NEW IN KELOID PATHOPHYSIOLOGY AND TREATMENT: A 2025 UPDATE

Integrative Approach to Dissect Keloid Mechanism and Optimize the Therapeutic Effect

Wei Liu, MD, PhD

Department of Plastic and Reconstructive Surgery, Shanghai Ninth People's Hospital, Shanghai Jiao Tong University School of Medicine

Keloid is a challenging disease boasting a high recurrence rate after various therapies. This situation has remained largely unchanged over the past decades. The most likely reason is that keloid is commonly regarded as a pure skin lesion, and all efforts in mechanistic and therapeutic investigations are solely targeted at keloid tissue/cells. In contrast, traditional Chinese medicine views all diseases as the results of an imbalanced internal environment of individuals or an imbalance between individuals and their external environments. An optimal therapy should involve restoring the balance along with targeted treatment.

With this in mind, we hypothesize that an imbalanced microenvironment is the key factor interacting with vulnerable skin cells to initiate keloid development and lead to its recurrence. To prove this, we have conducted a series of investigations on potential contributing factors to keloid constitution, including the effects of lifestyle, food types and nutrition, biorhythm disturbances, gut microbes and metabolic abnormalities, inflammatory status and hormonal fluctuations, psychological stress, and climate influence. Preliminary results reveal that these environmental factors do contribute to keloid constitution, making individuals prone to keloid development and recurrence. Based on this, an integrated approach has been employed to adjust and restore the normal balance of the internal and external environments of afflicted patients, such as through food and lifestyle adjustments, stress relief, the use of anti −inflammatory drugs, and Chinese herbal medicine. Interestingly, with the restoration of environmental balance, traditional local therapies, such as surgery, radiotherapy, and drug injection, achieve better outcomes in terms of reducing recurrence and enhancing the cure rate. This talk will provide an overview of the concept, recent advancements, experimental supportive evidence, and clinical outcomes.

Advancements of Keloid Therapy Techniques

Xiaoli Wu

Department of Plastic and Reconstructive Surgery Shanghai Ninth people's hospital, Shanghai Jiao Tong University School of Medicine. China.

Keloid is a challenging disease. Traditional therapies, such as surgery, radiotherapy, drug injections, and cryotherapy, have been applied to keloid treatment for many years. However, recent advancements in the pathophysiology of keloids and technological innovations have led to the development of new treatment modalities that can improve therapeutic outcomes and reduce recurrence rates.

Emerging techniques include surgical excision, punch surgery, innovative suturing methods, CO2 fractional laser therapy, microplasma, intense pulsed light (IPL), diode portable laser (EUFOTON), novel silicon pressure therapy, combined laser and radiotherapy, and multi-modality approaches. The broad application of these innovative methods in treating a large number of Chinese patients has resulted in significant progress in keloid treatment in China, greatly benefiting those affected by this condition.

This presentation will provide an overview of these cutting-edge techniques and their clinical applications.

Changing the Paradigm in Keloid Treatment: An Oncologist's Insights

Michael H Tirgan MD

Keloid Research Foundation

Background:

Keloid Disorder (KD), while benign, shares several biological characteristics with malignancies, including uncontrolled proliferation, local tissue invasion, and a high recurrence rate after treatment. Historically managed as a dermatologic or surgical condition, keloid lesions have consistently resisted conventional therapies, especially surgical excision. Drawing from oncologic principles, this presentation explores the genetic landscape of KD, contrasts it with cancer biology, and presents a new framework for understanding and treating this disorder.

Methods:

We conducted a comparative review of published genomic data on keloid tissues and matched normal tissues, alongside a literature analysis of cancer genetics. Parameters included clonality, somatic mutation burden, and gene expression profiles. Additionally, clinical outcomes from a cohort of over 3,000 keloid patients treated by the author were analyzed, with particular focus on treatment modality and recurrence rates.

Results:

KD exhibit stable but abnormal gene expression patterns in all cells within the lesion and often in adjacent, clinically unaffected skin. Unlike cancer—which typically involves clonal expansion driven by acquired somatic mutations—keloids lack this genetic heterogeneity. Instead, the genetic predisposition in keloids is systemic or constitutional, present uniformly across cells. This fundamental difference explains the ineffectiveness and high recurrence rates observed with surgical excision, which fails to address the root cause of the disease.

Conclusion:

KD, though non−malignant, behaves biologically as a tumor and should be treated accordingly. Its systemic genetic nature makes surgical intervention ineffective and often counterproductive. By applying principles from oncology—specifically cell−targeted destruction using intralesional chemotherapy and cryotherapy—we can achieve more durable responses and shift the treatment paradigm. This approach encourages a reclassification of KD from a dermatologic nuisance to a serious fibroproliferative disease requiring biologically rational, tumor−directed therapy.

DAY 1: FRIDAY JUNE 6, 2025

SESSION 3 *13:30–15:30*

EVIDENCE–BASED NON–SURGICAL STRATEGIES FOR KELOID TREATMENT

Predicting Response to Intralesional Steroids in Keloid Patients Using Machine Learning Models Trainedon Clinical Survey Data

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BACKGROUND:

Intralesional triamcinolone acetonide (ILTA) is a widely used first-line corticosteroid therapy for keloid disorder, yet many patients show limited or no response, while a significant number see a worsening of their keloids. Given the variability in steroid treatment outcomes, there is a pressing need for tools that can predict patients' steroid responsiveness prior to initiating therapy. *We hypothesized that machine learning (ML) models trained on keloid patient-reported data could be leveraged to provide accurate, pre-treatment predictions for their response to steroid therapy, enabling more personalized and effective treatments for keloid patients.*

METHODS:

An IRB-approved online survey-based dataset of self-reported responses from 1,940 keloid patients was used to obtain data for 940 patients (290 male, 650 female) who had provided sufficient information on their steroid treatment for our analysis. This data was used to develop and validate several ML models, including Neural Networks (NN) and Random Forest (RF). Features included demographics, lesion morphology and location, growth history, number of ILTA injections, and triggering factors. After preprocessing and handling class imbalance using SMOTE-ENN (Over-sampling using SMOTE and cleaning using ENN), models were trained using 5-fold cross-validation. Feature importance was evaluated using SHAP (SHapley Additive exPlanations) and permutation importance.

RESULTS:

NN and RF models demonstrated high accuracy (~95%)in predicting patient response to ILTA.SHAP analysis identified patient gender and keloid lesion morphology as the most influential features. Younger female patients with flat or nodular lesions were more likely

to respond favorably to treatment, while persistent lesion growth over time was associated with resistance. Notably, the models maintained strong predictive performance even when growth history or ILTA injection data were excluded,highlighting their applicability in treatment–naive patients or those lacking detailed medical records.

CONCLUSION:

ML models, particularly NN and RF, can accurately predict steroid response in keloid patients based on basic clinical data. These models offer a promising tool for identifying patients unlikely to benefit from ILTA and may guide clinicians in personalizing treatment plans. Additionally, feature analyses suggest gender–and morphology–specific patterns of steroid responsiveness, providing insight into the biological variability of keloid treatment outcomes. This approach represents an important step toward precision medicine in keloid management.

Disclosure Declaration:

The authors have no conflicts of interest to declare.

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Laser Combined with Radiotherapy for Keloid Treatment: A Novel and Efficient Comprehensive Therapy with a Lower Recurrence Rate

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BACKGROUND:

Keloid is a kind of benign skin fibroproliferative disease with a high recurrence risk. At present, comprehensive therapy is commonly used in treatment, but the high recurrence rate is still an urgent problem to solve. Moreover, the existing comprehensive treatment methods also have various problems, such as high cost, complex process, multiple side effects and so on.

METHODS:

99 patients with keloids who received ablative fractional carbon dioxide laser combined with radiotherapy (LCR) from February 2018 to November 2019 were studied and analyzed retrospectively. The LCR treatment was composed of one session of ablative fractional CO_2 laser and two sessions of electron beam irradiation. The patient received ablative fractional CO_2 laser first with an energy range of 360−1008mj. Radiotherapy was performed with 6MeV electron beam secondly. The first pass was completed within 24 hours after laser therapy, and the second pass was completed on the 7^{th} day after that. 9Gy energy was used for radiotherapy. The efficacy was evaluated by Patient and Observer Scar Assessment Scale (POSAS) before treatment and 6, 12 and 18 months after treatment. At the same time, the recurrence, side effects and satisfaction with the results were evaluated by online questionnaire.

RESULTS:

The total POSAS scores at 18 months follow-up was significantly lower than that before treatment (31.3 ± 11.0 vs 61.5 ± 13.7, P < 0.0001). During the follow-up period, 12.1% of the patients had recurrence, of which 11.1% had partial recurrence and 1.0% had complete recurrence. The overall satisfaction rate was 97.0%. No serious adverse reactions were observed during follow-up.

Table1. Patient and Observer Scar Assessment Scale before and 18 months after the therapy

	Before Treatment	18 Months after Treatment	P-value
Total Score	61.5±13.7	31.3±11.0	<0.0001
Patient Score	38.6±10.8	19.5±9.8	<0.0001
Pain	3.4±2.6	1.6±1.2	<0.0001
Pruritus	4.6±2.7	2.2±1.6	<0.0001
Color	8.2±2.2	4.6±2.7	<0.0001
Stiffness	7.5±2.4	3.9±2.5	<0.0001
Thickness	7.7±2.6	3.7±2.6	<0.0001
Irregularity	7. 3±2.5	3.6±2.3	<0.0001
Observer Score	**23.0±6.0**	**11.7±3.0**	**<0.0001**
Vascularity	3.6±1.6	1.8±0.7	<0.0001
Pigmentation	6.2±1.8	2.5±0.7	<0.0001
Thickness	4.1±1.7	2.2±0.8	<0.0001
Tension	4.5±1.7	2.5±0.8	<0.0001
Elasticity	4.5±1.4	2.5±0.7	<0.0001

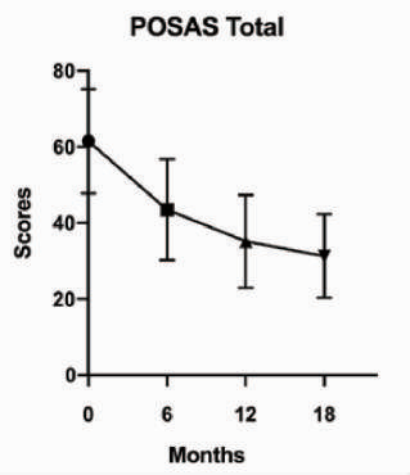

Fig 1. Patient POSAS total score changes before (T0),6 months (6M), 12 months (12M), and 18 months (18M) after the treatment. The figure indicates a significant difference between follow–up moments ($p<0.0001$).

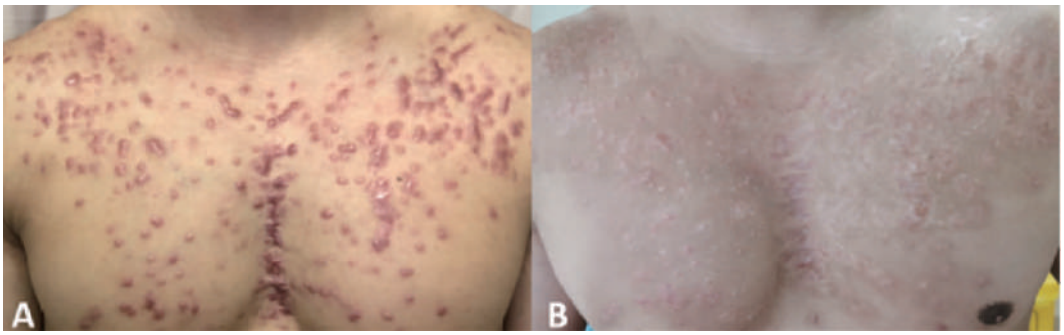

Fig 2. A 30–year–old male (case 57) with multiple keloids developed from acnes on the anterior chest for 1 year (A); 18 months after LCR therapy (B).

CONCLUSION:

LCR is a new comprehensive treatment for keloid which has not been reported by other scholars yet. The research results show that it has the benefit of good curative effect, low recurrence rate and high safety. This paper provides a theoretical basis for widely developing this method clinically.

This abstract reports on original research. There search is not a clinical trial and was not funded by any government agency, pharmaceutical or biotechnology companies or other foundations.

Ten Years' Clinical Experience with Intralesional Cryosurgery and Adjunctive Therapies for Keloid Management in Hong Kong

Tony Tung-Fei Chan

Genesis Minimally Invasive Surgery Centre, Hong Kong

BACKGROUND:

Surgical excision followed by radiotherapy remains the standard for keloid treatment but is often limited by accessibility and recurrence. Intralesional cryosurgery offers a minimally invasive alternative by directly targeting keloid tissue. Over the past decade, I have applied intralesional cryosurgery combined with adjunctive triamcinolone/5FU injections and laser/light therapy in clinical practice to optimise outcomes and reduce recurrence.

METHODS:

This presentation summarises my clinical experience treating patients with keloids using intralesional cryosurgery. The procedure involves inserting a cryoprobe directly into the keloid base to induce controlled freezing and necrosis. Adjunctive triamcinolone/5FU injections are administered post-procedure to reduce recurrence, followed by laser/ light therapy sessions to improve scar texture and pigmentation. Treatment outcomes are illustrated through representative case examples and longitudinal clinical observations.

RESULTS:

Over ten years, patients treated with this combined approach demonstrated consistent reduction in keloid volume and symptomatic relief, with improved scar pliability and pigmentation. Recurrence rates appeared lower compared to historical outcomes with excision alone. The minimally invasive nature of intralesional cryosurgery resulted in favourable tolerability and patient satisfaction.

CONCLUSION:

Intralesional cryosurgery combined with triamcinolone/5FU injection and laser/light therapy represents a practical, effective treatment strategy for keloids, especially in settings where expertise of surgery and radiotherapy are limited. Sharing this decade-long clinical experience aims to provide valuable insights and encourage wider adoption of this less invasive approach. Further prospective studies are warranted to validate and refine treatment protocols.

Novel Needle-type Electrocoagulation and Combination Pharmacotherapy: Basic and Clinical Studies on Efficacy and Safety in Treating Keloids

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Background and aim:
Keloids cannot be effectively treated using monotherapy regimens. This study aimed to evaluate the efficacy and safety of ablation (a novel needle-assisted electrocoagulation technique) combined with pharmacotherapy (corticosteroid and 5-fluorouracil [5-FU] injections) in removing keloids and to investigate the underlying biological mechanisms.

Methods:
The effects of energy consumption and duration of needle-assisted electrocoagulation on the ablation zone were tested in porcine liver tissue, which simulates human skin. The regulatory effects of ablation combined with pharmacotherapy on collagen deposition, cell proliferation, and angiogenesis were analyzed in a keloid-bearing nude mouse model in vivo. In a clinical trial involving six patients with keloids, the Vancouver Scar Scale (VSS) and Patient and Observer Scar Assessment Scale (POSAS) scores were graded before treatment and 1 month after one cycle of ablation combined with corticosteroid and 5-FU therapy.

Results:
Higher energy consumption and longer duration of electrocoagulation resulted in a larger ablation zone and higher surface temperature. Ablation combined with pharmacotherapy significantly reduced keloid volume in nude mice, upregulated MMP-1 and MMP-3, downregulated COL I and COL III, and inhibited angiogenesis and proliferation. This combination also significantly reduced the VSS and POSAS scores in patients with keloids after treatment without any obvious adverse events.

Conclusion:

Our findings show that electroablation combined with pharmacotherapy effectively reduces keloid volume by inhibiting collagen deposition, angiogenesis, and cell proliferation. Thus, this novel combination may serve as a safe therapeutic approach for keloid removal.

Keywords:

collagen; electrolysis; keloid; laser ablation.

Innovative Application of Scanning, Printing and Pressure Sensors Technologies to Improve Patient Experience in Auricular Keloids Management

Ka-Wing Wong, Ching-Sheung Chan, Wai-Kin Ching

Department of Occupational Therapy, North District Hospital, Hospital Authority, HKSAR

BACKGROUND

Auricular keloids pose significant clinical challenges due to their high recurrence rates, cosmetic impact, and potential discomfort for patients. Compression or pressure therapy by splinting as adjunct treatment has been reported to be effective. An innovative application of 3D scanning and printing technology as well as pressure sensors to increase the precision of splint fitting, thereby shortening the therapist–patient contact time and reducing the frequency of clinic visits. The aim of this study is to explore the feasibility of technology applications to improve the efficiency and comfort level for patients in auricular keloid management.

METHODS

A case study methodology was used. Following the occupational therapist´s keloid assessment during the first therapy session, the auricular keloids were scanned using a portable 3D scanning technology. An individual ear model was made by 3D printing for testing and fitting of the compression splint without the presence of the patient. Pressure sensors were applied to measure the optimal pressure provided by the compression adjustable splint. On the second session, the therapist might use the ear model to empower the patient and teach them how to fit the splint. Minor adjustments would be made if necessary to increase the level of comfort of the compression splint. Patients' conditions were followed up by mixed tele–mode and physical–mode follow–ups according to the patient's level of competency in self–management and the needs of the compression splint renewal. Users' satisfaction survey, adherence to compression splints, keloid conditions, and logic model framework analysis would be used to evaluate the inputs, outputs, and outcomes of this innovation.

RESULTS

Three patients with auricular keloids with sizes ranging from 1 to 2 cm^2 completed the 6-month therapy. Keloid volume, measured via comparative 3D imaging, was reduced by an average of 34%. Vancouver Scar Scale scores improved from a mean of 8.7 at baseline to 6.7 at 6 months, reflecting positive changes in vascularity, pliability, and height. The self-administering survey reported an average daily splint-wearing time of 17 hours, demonstrating strong adherence to therapy. All 3 patients reported the therapy arrangement, including the duration of assessment, scanning, fitting, and frequency of follow-ups, was highly satisfactory.

CONCLUSION

The integrative application of low-cost, market-available technologies in scanning, ear model printing, and pressure sensors to auricular keloid management is promising to improve the efficacy and adherence to the compression therapy by reducing the patient visiting time and frequency without compromising the quality of the services.

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Disclosure Declaration

All authors declare that they have no relevant or material financial interests that relate to the research described in this paper.

DAY 1: FRIDAY JUNE 6, 2025

SESSION 4 *15:45–18:15*

RETHINKING KELOID SURGERY: TOWARD PREDICTABLE AND LONG–LASTING OUTCOMES

Facial Keloid Treatment with Keloid Subepidermal Vascular Network Flap (KSVNFs): A Long-term Follow-up Study

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Objective

To investigate the early and long−term efficacy of a modified "keloid core excision method" preserving the keloid subepidermal vascular network flap (KSVNFs) combined with postoperative electron beam radiotherapy in patients with multiple facial keloids.

Methods

A retrospective analysis included 94 patients (846 lesions) treated between January 2017 and January 2020. The surgical procedure involved a modified keloid core excision technique preserving KSVNFs. Single− or double−pedicle KSVNFs flaps with an aspect ratio ≈1 were designed according to lesion size. Keloid core tissue was removed through layered dissection while preserving the integrity of the subcutaneous vascular network. Wounds were closed using vertical mattress sutures combined with interrupted sutures. Adjuvant radiotherapy (6 MeV electron beam, 9 Gy/fraction) was delivered in two fractions (24 hours postoperatively and postoperative day 7). Early outcomes (postoperative day 9) focused on flap necrosis rate, while long−term follow−up (until March 2025) evaluated recurrence rates and patient satisfaction (assessed via a Likert 5−point scale, with 5 indicating optimal satisfaction).

Results

Early evaluation (postoperative day 9) revealed a flap necrosis rate of 2.5% (21/846), with 3 cases (3.2%) of delayed wound healing and superficial infection, all resolved by local debridement. Long−term follow−up (median 90.6 months, range 60−96 months) demonstrated an 84% cure rate (79/94). Complications included localized hyperpigmentation in 6 patients (6.4%) and mild atrophic scarring in 4 patients (4.3%). Patient satisfaction assessment showed 41 cases (43.6%) "very satisfied," 44 cases (46.8%) " satisfied," and 9 cases (9.6%) "dissatisfied," yielding an overall satisfaction rate of 90.4% with a mean score of 4.4±0.6 points.

Conclusion

The KSVNFs preserving technique combined with electron beam radiotherapy demonstrates superior tissue preservation (flap necrosis rate: 2.5%), durable efficacy (16% recurrence vs. literature–reported 25 – 40% for conventional methods), and high patient satisfaction (90.4%). This combined protocol represents an optimal treatment strategy for multiple facial keloids, offering significant clinical value in aesthetic restoration.

Treatment Methods and Efficacy for Pediatric Keloids: A Clinical Study

Jinglong Cai, Chief Physician

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Objective

To investigate the treatment methods and efficacy for pediatric keloids.

Methods

A total of 100 pediatric keloid patients (63 males, 37 females; aged 1–12 years) treated between June 2018 and December 2023 were enrolled. Keloids were caused by surgery, burns, scalds, or trauma, located on the chest, abdomen, shoulder/back, jaw/neck, or joints, with a disease duration of 1–5 years. The "Cai Jinglong Keloid Treatment Protocol" was applied, tailoring therapies based on keloid characteristics. For thin keloids (<3mm thickness), intralesional drug injections combined with superficial radiotherapy (SRT-100) or punch reduction, CO_2 fractional laser, and adjuvant therapies (oral/topical medications, physical rehabilitation) were used. For thick keloids (≥3mm), surgical excision (single or staged) or partial debulking with layered tension-reducing sutures was performed, followed by SRT-100 within 24 hours and adjuvant therapies. Patients were informed of recurrence risks and advised to undergo 3-month follow-ups. Follow-up duration ranged from 6 months to 6 years (mean: 3 years). Outcomes were evaluated via photographic comparisons, Vancouver Scar Scale (VSS), Sawada Clinical Grading (for color, texture, thickness, hardness), and Visual Analog Scale (VAS) for itch/pain.

Results

- Short-term (6 months): 70% of lesions showed no signs of recurrence (redness, induration, elevation, or symptoms). Scores decreased significantly: VSS (10±2.5 vs. 4.6±0.7), Sawada (10±2.6 vs. 3.8±0.8), VAS (9±1.3 vs. 3.2±0.6), achieving a 70% control rate.

- Mid-term (12 months): 80% of lesions exhibited no recurrence. Scores further improved: VSS (2.3±0.5), Sawada (1.8±0.3), VAS (1.7±0.4), yielding an 80% cure rate.

– Long–term (24 months): All lesions remained recurrence–free, with VSS, Sawada, and VAS scores reduced to 0, indicating stable scar resolution.

Conclusion

The "Cai Jinglong Keloid Treatment Protocol," integrating dynamic and systematic approaches, effectively achieves long–term control and resolution of pediatric keloids.

Keywords:

keloids; treatment protocol; pediatrics; surgery; radiotherapy; drug injection

Surgical Management of Large Volume Keloids

Ioannis Goutos,MD

Queen Mary University of London

BACKGROUND

Keloid scars represent a complexpathological entity with multiple approaches described in the literature for clinical management. Therapeutic considerations become even more challenging in patients with large volume lesions.

METHODS

Retrospective review of a single surgeon experience treating large volume keloidal lesions in a variety of anatomical areas. Management modalities included local flaps as well as tissue expansion in combination with radiotherapy.

RESULTS

Favourable outcomes were attained both in terms of recurrence as well as aesthetic outcome in all patients.

CONCLUSION

Local flaps and tissue expanders represent valuable surgical modalities for the management of large volume keloidal lesions.

Trephination Tecknique in Treating Keloids

He Renliang

Keloid is a benign skin tumor caused by excessive proliferation of fibrous tissue and an imbalance in extracellular matrix (ECM) metabolism. It has a tendency to grow outward and is prone to recurrence, often accompanied by functional or aesthetic complications such as pain and itching. In addition to patient heterogeneity, trauma, inflammation, and tension are important factors contributing to keloid formation. Currently, comprehensive surgical-based treatments remain the primary approach. Reducing wound tension is critical to preventing abnormal scar hyperplasia. Therefore, preoperative design parallel to Langer's lines (anatomic tension lines) or the use of S-shaped incisions, combined with tissue expanders, can reduce mechanical tension. Common intraoperative techniques include V-Y plasty, Z-plasty, W-plasty, local flap transfer, intradermal sutures, and specialized tension-reducing suturing methods. Postoperative strategies involve skin tensioners, wound tapes, adhesives, mechanical closure devices, non-invasive adjustable tension systems, and pressure therapy. However, enlarged surgical wounds and prolonged operative time increase surgical complexity, risks, and healthcare costs.

Keloid Trephination Technology involves using a skin punch to extract keloid tissue in situ via volume reduction, drainage, and tension balancing. This technique is characterized by its in-situ precision, minimally invasive nature, and suitability for clinical adoption and development.

Retrospective Study on the Effectiveness of Punch Drilling Combined with Superficial X-ray Radiotherapy and Intralesional Drug Injection for Keloid Treatment

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Background:

Keloids, pathological scars with complex etiology including genetic predisposition and trauma, remain a therapeutic challenge due to high recurrence rates. This study intends to conduct a retrospective study on the patients who received punch drilling therapy for keloid in our hospital to provide a new method and objective basis for the treatment of keloid.

Methods:

A retrospective analysis of 42 keloidpatients treated at Dermatology Hospital of Southern Medical University was conducted. Outcomes were assessed using Vancouver Scar Scale (VSS) and Visual Analogue Scale (VAS) scores preoperatively and scar image data were collected, alongside complication rates and patient satisfaction surveys.

Results:

Significant reductions were observed in both scar scores: Median VAS decreased from 3.5 (range: 2.0−8.0) to 1.5 (1.0−4.0), and VSS from 7.0 (3.0−12.0) to 5.0 (1.0−8.0) ($P < 0.001$). Postoperative complications were minimal, with 95% of patients reporting marked improvement in scar appearance and satisfaction.

Conclusion:

The combination of punch drilling, adjuvant radiotherapy, and intralesional steroids injection demonstrates high efficacy in keloid treatment, characterized by low invasiveness, rapid recovery, and reduced recurrence. This protocol provides a promising strategy for refractory keloid management.

Keywords:

Keloid, Punch Drilling, Superficial Radiation Therapy, Intralesional Drug Injection;

Treatment and Management Over 10 Thousands Keloid Patients in Beijing Bakang Hospital

Bianyou Sun

Although keloids can be comprehensively treated through personalized plans developed by multidisciplinary collaboration, they still have a high recurrence rate. Based on years of clinical experience and case data from Beijing Bakang Hospital, it is concluded that the combination of treatment and close follow−up management is crucial to effectively reduce the recurrence rate of keloids, and the coordination of the two is of vital importance.

DAY 2: SATURDAY JUNE 7, 2025

SESSION 1 *08:30–10:30*

MOLECULAR AND CELLULAR INSIGHTS INTO KELOID FORMATION

Molecular and Immune-Mechanism of Keloid Development

Yixin Zhang, MD, PhD

Keloids are problematic scars characterized by excessive fibroblast proliferation and collagen deposition that extend beyond the original wound and often recur after treatment. To identify clinically actionable biomarkers and therapeutic targets, we conducted immune profiling on both blood and lesional tissue from a hospital-based cohort (104 keloid patients, 512 healthy controls, 100 patients with other scar or inflammatory skin conditions). Flow cytometry and single-cell RNA sequencing revealed a significant depletion of cytotoxic $CD8^+$ T lymphocytes (CTLs) in peripheral blood and within keloid margins. In the scar tissue, residual CTLs displayed high expression of the inhibitory NKG2A/CD94 receptor complex, which corresponded with elevated serum levels of soluble HLA-E (sHLA-E). Quantitative assays demonstrated that sHLA-E distinguished keloid patients with 83.7% sensitivity and 92.2% specificity, showing minimal cross-reactivity in hypertrophic scars or unrelated dermatologic diseases.

We then assessed response to combined intralesional triamcinolone and 5-fluorouracil therapy in a treatment subgroup. Patients with favorable outcomes exhibited pronounced reductions in sHLA-E post-treatment, whereas incomplete suppression of sHLA-E was linked to higher recurrence rates on follow-up. These results implicate the NKG2A/CD94 - sHLA-E axis in immune evasion within keloid pathology and position sHLA-E as both a diagnostic marker for keloid risk and a prognostic indicator of treatment efficacy. Incorporating sHLA-E monitoring into clinical practice could enable personalized strategies to prevent keloid formation and recurrence.

Understanding the Mechanisms of Keloid Recurrence and Exploring Novel Therapeutic Options

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BACKGROUND:

Despite a plethora of treatment options for keloid, including surgery, intralesional corticosteroid injections, cryotherapy, radiotherapy and chemotherapy, continued disease progression and/or recurrence remain a clinical challenge. This is in part due to incomplete understanding of keloid pathophysiology. We are investigating the cellular changes that occur between primary and recurrent keloids, as well as investigating the potential for targeting keloid extracellular matrix (ECM) to disrupt fibroblast activity as a therapeutic approach for keloids.

METHODS:

To understand the changes that occur in recurrent keloids, we have compared fibroblasts isolated from a primary and recurrent keloid from the same individual using flow cytometry, immunohistochemistry and a range of functional assays including migration, collagen production and matrix deposition. We also recently completed a phase 1c clinical trial investigating the effects of a Lysyl Oxidase inhibitor (PXS−6302) in normotrophic scars on the ECM. Based on the findings we are now commencing a clinical trial to explore the impact of PXS−6302 in keloids to examine the effects of targeting ECM stability on keloid pathophysiology.

RESULTS:

In comparison to the primary keloid, fibroblasts from the recurrent keloid tissue showed a significant decrease in COLI expression at both the RNA and protein level. Surprisingly fibroblasts from recurrent keloid appear to have lower proliferation and migration than those from the primary keloid, with changes in subtypes of fibroblast, including an increase in the percentage of CD140a positive fibroblasts in recurrent keloids as well as

changes in expression levels of other surface proteins known to be important in fibroblast activity. In the clinical trial of PXS−6302 in normotrophic scars, hydroxyproline levels were significantly reduced after 3 months, with optical coherence tomography (OCT) imaging showing changes in both vessel and collagen density in treated scars.

CONCLUSION:

Fibroblasts from primary and recurrent keloids appear to have significant phenotypic changes that may be a response to previous treatment and/or changes in the cell populations as the disease progresses. This may impact on treatment options most suited to primary and recurrent keloids. The effects of PXS−6302 in normotrophic scars suggests Lysyl Oxidase inhibition can effectively stimulate matrix remodelling. This approach may provide a strategy to reduce fibroblast activity in keloids.

The lnhibition of ENO1 Expression Targets the Glycolytic and Lactylation to Regulate Keloid Fibroblast Function

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Objectives

The pathogenesis of keloids has been associated with metabolic reprogramming from oxidative phosphorylation to aerobic glycolysis (Warburg effect). Lactate is a metabolite of the Warburg effect that can induce lactylation and stimulate chromatin gene transcription. Hypoxic microenvironments may promote the development of keloids by enhancing the Warburg effect. However, whether excessive lactate production causes lactylation in keloid fibroblasts (KFb) and whether lactylation, proliferation, and collagen deposition can be reduced by inhibiting glycolysis in KFb are still unknown.To identify the lactylation in keloid fibroblasts (KFb), and examine the potential of inhibiting glycolysis in the treatment of keloid.Distinct from tumor therapy, which completely kills tumor cells, this study aimed to improve cellular function and reduce collagen synthesis in KFb by inhibiting glycolysis under normoxic and hypoxic (simulating a hypoxic microenvironment in vivo) conditions.

Methods

Gene expression and protein levels were evaluated with immunohistochemical staining, real−time quantitative polymerase chain reaction (qRT - PCR), and Western blotting for normal skin tissue and keloid tissue, as well as cultured primary fibroblasts. ENO1 in KFb was knockdown by lentivirus infection, and qRT−PCR, Western blotting, incucyte live−cell tracking system, Seahorse XF96, and transwell assays were used to detect the gene expression, protein level, cell viability, metabolism, migration and invasion of KFb with ENO1 knockdown and 2DG treatment.

Results

We found that the expression of ENO1 in keloid tissue and KFb was significantly higher than that in normal skin tissue ($P < 0.05$). The mRNA and protein expressions of ENO1 in KFb were significantly higher than those in NFb under normoxia and hypoxia condition, respectively ($P < 0.05$), and hypoxia would stimulate the expression of ENO1 in KFb and NFb ($P<0.05$). The mRNA and protein expressions of Glut1, LDHA, and COL1 in KFb were significantly decreased after inhibiting the expression of ENO1

compared with the negative control group under normoxia and hypoxia conditions, respectively ($P < 0.05$). Glucose consumption and lactic acid production, extracellular acidification rate, non−glycolytic acid, glycolysis, glycolytic capacity, and glycolytic reserve were significantly decreased ($P < 0.05$); the mitochondrial basal respiration, maximal respiration, and OCR/ECAR values were significantly higher than the control group ($P< 0.05$). Cell proliferation, migration, and invasion were also significantly inhibited ($P < 0.05$). Furthermore, after inhibiting the expression of ENO1, the phosphorylation of PI3K/ AKT pathway in keloid fibroblasts was significantly decreased ($P > 0.05$).

Conclusions

Our study confirmed for the first time that lactylation occurred in KFb, and inhibiting the overexpression of the glycolytic enzyme ENO1 in KFb inhibited glycolysis and ameliorated aerobic respiration, cell viability, migration, invasion, collagen synthesis, lactate production, and lactylation under normoxic and hypoxic conditions. Our results provide new insight into the pathogenesis keloids and reveal novel treatment strategies.

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Asporin Inhibits Collagen Matrix-Mediated Intercellular Mechanocommunications Between Fibroblasts During Keloid Progression

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Background:
Keloids are fibrotic lesions that grow unceasingly and invasively and are driven by local mechanical stimuli. Unlike other fibrotic diseases and normal wound healing, keloids exhibit little transformation of dermal fibroblasts into α–SMA$^+$myofibroblasts.

Methods and Results:
Differential gene expression in the keloid–leading edge relative to surrounding healthy skin showed that asporin is the most strongly expressed gene in keloids and its gene–ontology terms relate strongly to ECM metabolism/organization. Experiments with human dermal cells (HDFs) showed that asporin overexpression/treatment abrogated the HDF ability to adopt a perpendicular orientation when subjected to stretching tension. It also induced calcification of the surrounding 3D collagen matrix. Asporin overexpression/treatment also prevented the HDFs from remodeling the surrounding 3D collagen matrix, leading to a disorganized network of thick, wavy collagen fibers that resembled keloid collagen architecture. This in turn impaired the ability of the HDFs to contract the collagen matrix. Asporin treatment also made the fibroblasts impervious to the fibrous collagen contraction of α–SMA$^+$myofibroblasts, which normally activates fibroblasts. Thus, by calcifying collagen, asporin prevents fibroblasts from linearly rearranging the surrounding collagen; this reduces both their mechanosensitivity and mechanosignaling to each other through the collagen network. This blocks fibroblast activation and differentiation into the mature myofibroblasts that efficiently remodel the extracellular matrix. Consequently, the fibroblasts remain immature, highly proliferative, and continue laying down abundant extracellular matrix, causing keloid growth and invasion. Notably, dermal injection of asporin–overexpressing HDFs into murine wounds recapitulated keloid collagen histopathological characteristics.

Conclusion:

This study shows that ASPN–mediated inhibition of mechanocommunications between fibroblasts promotes keloid growth. It also suggests that ASPN is a potential diagnostic biomarker and therapeutic target for keloids.

Understanding and Clinical Practice of Vascular Abnormalities in Keloid Disease

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Background:

Most of the widely used methods for the assessment of keloid treatment are subjective grading scales based on the opinion of an individual clinician or patient.Meanwhile, there is a growing need for reliable objective methods to evaluate keloid treatment, such as blood perfusion.

Methods:

This retrospective analysis was conducted on 99 patients with 176 keloid lesions treated at our hospital. Patients underwent both VSS assessments and LSCI measurements pre− and post −treatment. LSCI −derived perfusion values included absolute perfusion units for keloids and the normalized perfusion ratio, calculated against adjacent normal skin.

Results:

There was significant correlations with total VSS scores ($\rho=0.308$, $p<0.001$) and key subcomponents, including vascularity ($\rho=0.424$, $p<0.001$), pigmentation ($\rho=0.366$, $p<0.001$), pliability ($\rho=0.187$, $p=0.014$), and height ($\rho=0.163$, $p=0.032$). Linear regression analysis revealed a strong positive correlation between changes in absolute perfusion units and VSS (ΔVSS) ($R^2=0.539$, $p<0.01$). Clinical cases further illustrated that absolute perfusion units effectively reflects vascular abnormalities and scar severity.

Conclusion:

LSCI −derived absolute perfusion units provide a reliable, objective, and quantifiable supplement to the VSS for evaluating keloid severity and treatment outcomes. Its ability to detect subtle vascular changes highlights its potential to improve precision in keloid management and clinical decision−making.

BMP2-induced Adam12+Fibroblasts Dictate Skin Scarring and Fibrosis

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BACKGROUND

We and others found that Adam12+ fibroblasts were increased and essential for fibrosis in multiple fibrotic diseases. However, the key signaling that regulates the origin of Adam12+ fibroblasts, the cellular progeny of Adam12+ fibroblasts and the mechanism that Adam12+ fibroblasts promote fibrosis in fibrotic diseases remain elusive.

METHODS

We use lineage tracing, cell ablation and conditional knockout technologies to explore these questions in skin wounds.

RESULTS

We found that Adam12+fibroblasts were necessary for skin scarring and fibrosis and they promoted fibrosis by secreting periostin. Lineage tracing and single cell RNAseq results suggested that most of myofibroblasts, the important cells for scarring, were progeny of Adam12+fibroblasts. We next identified BMP2 as the essential upstream signal for the generation of Adam12+ fibroblasts and showed that Adam12+fibroblasts mainly originated from normal fibroblasts after skin injury. Conditional knockout of the BMP2 receptor in fibroblasts decreased the number of Adam12+fibroblasts, the expression of periostin and the degree of scarring and fibrosis after skin injury. In clinical samples, we found that BMP2, periostin and Adam12+fibroblasts were increased significantly in hypertrophic scar and keloid compared to normal scars, and enhancing BMP2 signaling aggravated skin scarring and fibrosis, implying that abnormally highly expressed BMP2 may lead to skin fibrotic diseases. Treatment of hypertrophic scar mouse model by BMP2 inhibitor decreased the degree of scarring and fibrosis.

CONCLUSION

These findings will help to understand skin abnormal scarring pathogenesis in depth and provide new targets for the therapy of fibrotic diseases.

DAY 2: SATURDAY JUNE 7, 2025

SESSION 2 *10:50–12:40*

EVIDENCE–BASED RADIOTHERAPY IN MANAGEMENT OF KELOIDS

Adjuvant Radiotherapy for the Treatment of Keloids

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Abstract:
Radiotherapy has been an effective primary and adjuvant therapy for many benign fibroproliferative disorders including keloids. The risk of recurrence after local excision of keloids is high. The literature supporting the important role of postoperative adjuvant radiotherapy for keloids in decreasing to less than 25% the risk of recurrence is reviewed. External beam radiation has been the principal modality but more recently evidence is described indicating the superiority of brachytherapy over other modalities.

Topic Code:
External Radiotherapy, Brachytherapy, Keloids

A Data-Based Approach to a Keloid Post-Excisional Radiotherapy Treatment (PERT) Pathway

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Background

A 2017 meta-analysis of 62 studies (27 superficial X-ray; 18 electron beam; and 17 brachytherapy) of keloid post-excisional radiotherapy treatment (PERT)concluded that, regardless of type of radiation employed, a recurrence rate of approximately 20% was achievable and that PERT was superior to radiation monotherapy (ref. 1). However,a subsequent Report of the 3rd International Keloid Symposium (IKS) in Beijing in 2019 identified that "the biggest obstacle to the development of optimal treatment for patients with keloids is the lack of data-driven treatment pathways" (ref. 2).

In 2022,Guidelines from the German Society for Radiation Oncology (DEGRO)approved radiotherapy after surgical intervention as a viable option for keloid treatmentbut only at Evidence Level 4 (Case Series), due to a lack of Evidence Level 1 Randomised Controlled Trial (RCT) data. DEGRO also advised that keloid radiotherapy as a monotherapy be only employed exceptionally (ref. 3). However, by 2025, only 4 registered keloid treatment research studies (3RCTs & 1 Cohort Study) were ongoing, none evaluating PERT (ref. 4).

To overcome this apparent lack of progress, this study aims to consider a novel approach to the analysis of PERT Audit outcome data presented at the 2nd, 3rd & 4th IKS (refs.5, 6& 7), with a view to re-formulating it as Evidence Level 2 Cohort Studies.In turn,this evidence will facilitate optimisation of aradiotherapy treatment dose regimen for PERT, as well as the development of a related optimal treatment pathway including a cost-effectiveness perspective. The applicability of this study approach to other keloid treatment modalities, e.g. laser therapy, cryotherapy, etc. will also be briefly considered.

Method

Historically, untreated keloid tumours are characterised by infiltration into surrounding normal tissue, progression over time and only rare instances of regression. As a result, virtually all untreated keloids will either remain stable or continue to grow (ref. 8).

Given this predictable behaviour, it can be argued that within the population of untreated keloids, a suitable comparator cohort can be identified to match any patient cohort in a

keloid Audit study. This matching can be based on factors such as keloid location, stage, patient age, gender, and ethnicity. Consequently, long−term outcome Audits of treated keloid patients can be considered Cohort Studies, qualifying their outcome data for Evidence Level 2 classification (ref. 9).

Applying this approach to control groups, the three previously referenced KFS Audits can be regarded as Cohort Studies. Data from these Audits will be analysed to assess treatment outcomes, then cross −referenced with radiobiological data to determine the optimal treatment regimen. Following this, the treatment equipment used will be evaluated for cost−effectiveness, ultimately informing the proposal of an optimal treatment pathway.

Results

Treatment outcome data inrefs. 5, 6 & 7(now seen as Cohort Studies):

Centre Location	Treatment Unit; kV or MeV	Treatment Dose (Gy); No. of fractions(#)	No. of Keloids or Patients	Recurrence Rate; Mean Follow-Up	Adverse Effects
Beijing, China (ref. 6)	Linear Accelerator (Linac); 6 or 7 MeV	18Gy/2#; #1w/in 24h-48h;& #2 1wk later	834 keloids	9.6%; 3.3 years	9.8%
Nairobi, Kenya (ref. 7)	SXRT Unit: e.g. 100kV	12Gy/1# w/in 24 h	523 patients	10.0%; 5 years	None "long term"
London, UK (ref. 8)	SXRT Unit: e.g.100kV	10Gy/1# w/in 24h	(80†; 102) 182 keloids	16.0%†& 14.7%; 5 years	Minimal side effects

No radiation induced cancers observed in any of the studies; †study published in 2003

Optimal patient outcomes were observed for a dose of 10Gy to 12 Gy of X −rays delivered as a single fraction within 24 hours of keloid excision. Treatment by electrons in 2 fractions, each of 9Gy, did not improve the recurrence rate but increased adverse effects significantly.

An alternative approach is to apply the LQ radiobiological model to calculate the radiobiologically effective dose (BED) and produce a BED comparison of single fraction treatment doses with their reported clinical implications. Using the BED formula:BED = nxd[1+d/(α/β)], for keloids α/β=2 to 3 and, using 2.5 (ref. 10):

Single Fraction Dose	$BED_{2.5}$(Gy)	Clinical Implications
8 Gy / 1#	33.6 Gy	Effective, lower toxicity but potential increasing recurrence risk
10 Gy / 1#	50.0 Gy	Higher effectiveness but fibrosis risk
12 Gy / 1#	69.6 Gy	Significantly higher BED, higher risk of fibrosis & poorer cosmesis

On these bases, an optimal treatment pathway would include an optimsed dose protocol of 10 to 12 Gy/1#, treated within 24 hours of keloid excision @ 100kV (with a higher kV for thick keloids and a lower kV for thin keloids).

A cost−effectiveness analysis of treatment pathways employing an SXRT unit or a linac for benign disease treatment, has shown that SXRT units are around 10X cheaper to purchase and 10X cheaper to maintain. The construction cost of a lead lined SXRT treatment room is also significantly less than that of a concrete bunker for a linac. Finally, physicist (dosimetry & QA) and radiographer (treatment) staffing costs are lower for an SXRT unit than a linac (ref. 11).

Conclusions

As nearly 100% of untreated keloids stabilise or progress, well structured long−term treatment outcome Audits can be viewed as Cohort Studies qualifying their outcome data for Evidence Level 2 classification

Keloid treatment outcome data and radiobiological calculations both suggest that a single 10−12Gy treatment fraction within 24 hours of surgery is the optimum dose for PERT for optimisation of recurrence and adverse effect rates. This also suggests that when 2 x 9Gy treatment fractions are employed, the second fraction may be unnecessary and only serve to increase the probability of adverse effects.

A quoted cost−effectiveness analysis showed that using an SXRT unit,as part of an optimal pathway for effective keloid management,was cost−effective compared with using a linac,

Ongoing data collection, involving routine auditing and collation of a range of parameters, preferably in a standardised format,is crucial for further refining data−driven radiotherapy treatment pathways for keloids,

Adopting a similar outcome data recording framework for treatment modalities other than radiotherapy, e.g. laser therapy, cryotherapy, etc., would facilitate comparison of outcomes and development of data−driven optimisation of treatment for each modality. In turn, inter−modality comparison of optimised treatment modalities could then be undertaken.

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Case Studies of Patient Outcomes of Re-Treatment of Recurrent Keloid Tumours by Fractionated Superficial X-Ray Radiotherapy

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Background

A 15–year audit (from 2010 to 2025) of the outcome of treatment of recurrent keloid tumoursby fractionated superficial X–ray radiotherapy (SXRT) was undertaken at Cancer Centre London. The audit result was that, of the 23 keloid recurrences which had developed in the cohort of 158 keloids originally treated,all were found to have been successfully re–treated: the mean period of follow–up was 6–years 8–months (ref.1).

A case series of individual patient outcomes has been selected from the 23 keloid recurrences to illustrate the process and success of re–treatment for a range of: body sites (e.g.earlobe, sternum, etc.);treatment doses;kVs; patient ethnicities; and gender.

Consideration was also given to establishing whether identifiable factors for keloid recurrence were present at initial treatment by surgery and SXRT with a view to establishing whether patients at greater risk of recurrence could be identified and, in turn, monitored by long term follow–up,so facilitating re–treatment at an early stage.

Methods

The radiation dose delivered post–surgery during the initial keloid tumour treatment was 10Gy of SXRT in 1 fraction (#), within 24 hours of keloid surgery.In terms of Biologically Effective Dose (BED), using $\alpha/\beta=3$ for skin, 43.3 Gy_3 was delivered (ref. 2).

When a patient experienced a keloid recurrence, following review,the protocol employed was for a course of SXRT treatment only:4# x 4Gy delivered at 3 monthly intervals over the course of 1 year (i.e. 16Gy in total). However, this treatment was discontinued after 1# (4Gy), 2# (8Gy) or 3# (12Gy), if,following delivery of this level of dose,a successful response were present prior to the next #.

Radiobiologically, for 1#, the BED is 9.3Gy3 and, assuming moderate tissue repair over each 3 month period, 12.6Gy3 for 2#, 13.7Gy3 for 3# and 14.1Gy3 for a full 4# course of treatment over a 12 month period (ref. 3).

Results

14 patients with a total of 23 keloids were successfully treated for keloid recurrence. Some examples of the treatments delivered are summarised in the table below by keloid site, number of keloids, ethnicity, etc.

Examples of Keloid Recurrence Patients Re–Treated with SXRT

Year of Treatment	Patient Gender (M/F)	Patient Ethnicity White(W);Black(B); Asian (A);Mixed Ethnicity(ME)	Number of Keloid Re-Treatment Sites	Location of Keloid(s)	SXRT kV
2011	M	B	2	Sternum; Shoulder	100kV; 60kV
2014	F	A	1	Lt Earlobe	160kV
2017	F	A	1	Lt Nasal Ala	60kV
2019	F	W	1	Sternum	60kV
2021	M	A	1	Lt Cheek	100kV

Notes:

Mean Follow–Up Period = 6years 8months (Minimum:1month; Maximum:13years 4months)
No cancers recorded

Treatment response was assessed prior to each scheduled quarterly treatment, as radiation response is delayed.If a successful response were observed,SXRTwas then discontinued.A successful response was defined as:elimination of the hallmarks of keloid recurrence, i.e. keloid re –growth or red/shiny skin; as well as symptom relief (from itching); and, importantly, patient satisfaction with the cosmetic and functional results. While a degree of thickening remained in treated areas for a minority of patients, they found this to be acceptable.

A review of the case studies was unable to definitively establish any consistent common factors predicting keloid recurrence due to its' limited sample size and the statistical power remaining low. However it was observed that a propensity to recurrence may be associated with: the initial thickness of the keloid; and/or inadequate initial treatment

margins. It was also observed that, when surgical excision then delivery of SXRT were performed for the initial treatment, recurrence may sometimes have been linked to incomplete removal of sutures, or failure of dissolvable sutures. Further data collection is necessary to strengthen these findings.

Conclusions

While limited in scope, this study demonstrates that SXRT, when fractionated at quarterly intervals, is a viable treatment for keloid recurrence with minimal side effects.

No definitive common factors for recurrence were conclusively identified, though initial keloid thickness, initial treatment margins and suture issues may contribute. A larger pooled dataset could help identify high –risk patients for closer monitoring and early intervention.

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Electron Beam Radiation Therapy for Postoperative Keloids

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Radiation therapy, as one of the effective adjuvant measures to suppress keloids, works through the direct and indirect effects of ionizing radiation to inhibit the excessive proliferation of fibroblasts, with the primary goal of preventing recurrence after keloid treatment. A medical linear accelerator generating 6 MeV electron beams is typically used for keloid treatment, offering an effective penetration depth of 1.5 – 2 cm, with precise control over radiation dose and depth. Clinically, postoperative radiotherapy for keloids requires mastery of electron beam techniques, including conformal lead shielding, electron beam compensation and field junction techniques, to enhance dose uniformity in the irradiated area while maximally protecting critical organs and normal tissues surrounding the surgical site.

To achieve effective keloid cure and reduce recurrence, electron beam radiotherapy is generally recommended within 24 – 48 hours postoperatively. For keloids in different anatomical locations, the total radiation dose, fractionation (single dose), and dose–time regimen should be determined based on the BED (biologically effective dose) calculation formula.

Dosimetric Characteristics of Superficial X-Ray Therapy（SRT） and Precautions in its Clinical Application

Xu Jinhu

Introduction:

Keloids are challenging fibroproliferative disorders with high recurrence rates after surgical excision. Superficial X－ray radiotherapy (SRT) has emerged as an effective adjuvant therapy to prevent recurrence. This presentation outlines the key aspects of SRT, including its scientific basis, dosimetry, clinical workflow, Clinical casesand safety protocols.

Key Content:

1. Schematic Diagram of Skin Structure

Visual overview of skin layers, highlighting the depth of keloid formation and SRT's targeted penetration.

2. Dosimetry Characteristics of Superficial X－rays

Energy range (e.g., 50 - 100 kv), depth－dose distribution, and optimal dosing (typically 12 - 20 Gy in fractionated regimens).

3. Treatment Process of SXRT

Step－by－step workflow: post－excision timing, field delineation, shielding techniques, and fractionation schedules.

4. Precautions for Clinical Application

Radiation safety measures (e.g., thyroid/eye shielding), contraindications (pregnancy, pediatric cases), and managing acute side effects (erythema, hyperpigmentation).

5. Clinical Cases

Demonstrative cases showing efficacy in reducing recurrence, with pre－/post－treatment images and long－term follow－up.

Conclusion:

Superficial radiotherapy is the optimal modality for preventing keloid recurrence post－surgery. Success depends on clinicians' understanding of dosimetry principles, tailored treatment planning, stringent radiation protection, and patient－centered safety protocols. SRT offers a balance of efficacy and tolerability when applied judiciously.

DAY 2: SATURDAY JUNE 7, 2025

SESSION 3 *13:30–15:30*

SURGICAL TREATMENT OF KELOIDS IN CONJUNCTION WITH RADIATION THERAPY

Cure Criteria and Treatment Model for Keloids

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Abstract:
Objective: To explore the cure criteria and treatment model for keloids.

Methods:
1. A clinical classification of keloids was proposed to guide treatment selection. Patients were categorized into four types based on lesion location and quantity: single-site solitary, multi-site solitary, single-site multiple, and multi-site multiple. Solitary lesions were further subclassified by area and thickness: small-thin (<5mm thickness, treatable via excision, cryotherapy, laser, radiotherapy, or drug injection), small-thick (>5mm thickness, requiring surgical excision), large-thin (<5mm thickness, needing flap transfer, tissue expansion, or skin grafting), and large-thick (>5mm thickness, requiring complex reconstruction). Multiple lesions were classified as isolated (treatable sequentially) or diffuse (requiring prioritized management of symptomatic or ulcerated lesions alongside systemic therapy).

2. Treatment strategies emphasized efficacy-risk balance, early intervention, and combination therapies (surgery, photoelectric techniques, radiotherapy, intralesional injections). The "Cai Jinglong Keloid Management Model" was implemented, focusing on systematic, comprehensive, and dynamic approaches.

3. The Cai Jinglong Model emphasized:
Systemic therapy: Addressing internal imbalances and infections via oral/topical medications.
Combination therapy: Integrating surgical and non-surgical methods.
Dynamic therapy: Long-term management with regular follow-ups and adaptive interventions.

Treatment stages included:
Wound healing promotion;
Early post-healing scar modulation (laser, radiotherapy, botulinum toxin);
Mature scar management (tailored surgical/non-surgical approaches);
Patient education on recurrence risks and mandatory 3-month follow-ups.

4. Three-phase efficacy criteria were established:
Short-term (6 months): 80% cure rate (no erythema, induration, or pruritus);
Mid-term (12 months): 90% cure rate;
Long-term (24 months): 100% cure rate (clinical endpoint).

5. Cure was defined as lesion flattening/softening with ⩾2-year symptom-free stability.

6. From January 2010 to June 2024, 2,246 patients were treated and evaluated via Vancouver Scar Scale (VSS), Sawada clinical grading, and VAS scores during 3-month follow-ups (mean 3 years).

Results:
Short-term: 80% cure rate; VSS 2.6±0.5, Sawada 1.8±0.6, VAS 1.2±0.4.
Mid-term: 90% cure rate; VSS 1.3±0.3, Sawada 0.9±0.2, VAS 0.7±0.3.
Long-term: 100% cure rate; all scores normalized to 0, achieving stable clinical endpoints.

Conclusion:
The Cai Jinglong Model achieves complete keloid remission through systematic, dynamic, and multidisciplinary strategies.

Keywords:
Keloid; Treatment model; Efficacy criteria; Cure standard

A 15 Year Single Centre Audit of the Outcome of Fractionated Superficial X-Ray Radiotherapy Treatment of Recurrent Keloid Tumours

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Background:

For patients who have experienced a keloid recurrence at a site previously treated by surgery and radiotherapy, the efficacy of a further course of radiotherapy for treatment of this recurrence has not been previously reported as a distinct study outcome.This study reports such results.

The results of two series of long−term audits of recurrence rates of keloid tumours, following surgical excision of the keloid and administration of a single (10Gy) dose of superficial X−ray radiotherapy (SXRT) within 24 hours, have previously been reported in 2003(ref.1) & 2022 (ref.2).The study period for the second series of patient treatments has now been extended to January 2025 and, during this entire second series, keloid recurrences were re−treated with fractionated SXRT. The outcome of this re−treatment is now reported.

Methods:

Outcomes of patient treatment of keloid tumours between 2010 and 2020, by surgical excision then SXRT within 24 hours,have previously been reported (refs.2& 3).This recurrence study has now been extended to January 2025 and recurrence results over a 15 year period derived (see Table 1). For this treatment regime of 10Gy in one fraction (1#), the Biological Effective Dose(BED), using $\alpha/\beta=3$ for skin,is43.3 Gy_3

Following a patient reporting a keloid recurrence, re−treatment was undertaken with fractionated SXRT only and usingthe following protocol: 4Gy administeredin 4#at 3 monthly intervals to a maximum dose 16Gy over 12 months. Treatment was discontinued after 4Gy, 8Gy or 12Gy if, at review,a successful outcome had been achieved at this dose. Here the BED3 is 9.3Gy_3 for 1#,and, assuming moderate tissue repair, 12.6Gy_3 for 2#, 13.7Gy_3 for 3# and 14.1Gy_3 if the full 4# course of treatment were to be delivered over a 12 month period.

Recurrences were usually small and the results of employing this radiotherapy re−treatment regime for a patient recurrence are summarised below (see Table 2).

Results:

Table 1 – Keloid Recurrence Rate Post Surgery and SXRT (2010–2025)

1. Centre	2. Dose (Gy)	3. SXRT Treatment	4. Patients: Number& Gender	5. Number of Keloids	6. Outcome at 4–6Week Post Treatment Review	7. % Relapse Rate After 1 year	8. % Relapse Rate After 5 years
Cancer Centre London 2010 – 2025 (January)	10Gy in 1 fraction	60kV, 100kV, or 160kV X-ray beam	87 patients 66% (F) 34% (M)	158 keloids	100% (158 keloids) Free from recurrence	6.8% (for 148* keloids)	14.7% (for102† Keloids)

* 10 keloids treated within previous year; †56 keloids treated within previous 5 years
For entire patient cohort (158 keloids): 23 (14.6%)relapsed during the 15 year period

Notes:

1.Minimal skin reaction – reddening /itching and/or pigmentation change for 6 –12 months

2. No cancers recorded

3. Time Period before Relapse: 3.5 months to 10 years

4. Mean Follow–Up Period:7years 6months (Minimum: 1 month; Maximum: 15 years)

Table 2 – Outcome of Keloid Recurrence Re–Treatments by Fractionated SXRT (2010–2025)

1. Centre	2. Dose (Gy)	3. SXRT Treatment	4. Patients: Number& Gender	5. Number of Keloids	6. Outcome at Quarterly Treatment Review Before Next Planned Fraction	7. % Relapse* Rate After 1 year	8. %Relapse* Rate After 5 years
Cancer Centre London	4Gy at 3 monthly intervals: Maximum dose 16Gy (i.e.4Gy/4#) over 12 months	60kV, 100kV, or 160kV X-ray beam	14 patients 50% (F) 50% (M)	23 keloids	Treatment discontinuedafter 2# or 3#in ~ 50% of patients: further treatment not required	None but for16Gy (4#/1yr)patient treatments: response ongoing& outcome yet tbc	None requiring further re-treatment: patients satisfied with outcome†

* Hallmarks of recurrence – keloid regrowth;itching; red, shiny skin: not present
+ Patients satisfied with treatment outcome, though minority had residual skin thickening

Notes:

1. Minimal skin reaction – reddening /itching and/or pigmentation change for 6 –12 months
2. No cancers recorded
3. Mean Follow–Up Period:6years 8months (Minimum:1month;Maximum:13years 4months)

An attempt was made to compare the results of this radiotherapy treatment regime with re–treatment results reported when either different doses and/or different fractionation or different radiotherapy modalities (i.e. a linear accelerator electron beam or brachytherapy) were employed. However,no reports of the success rates for the effectiveness of such re–treatment as a distinct study outcome were found to be available.

Conclusions:

Surgical excision of keloid tumours followed by superficial radiotherapy given within 24 hours in a single dose of 10Gy has proven to be highly effective with minimal side effects and a low 5−year recurrence rate of 14.8%.

In the event of a recurrence, the radiotherapy treatment can be safely repeated in smaller (4Gy) fractions at three monthly intervals, up to a maximum of 16Gy over a year if necessary.When keloid tumours were re −treated, an audit of the outcome of this treatment regime found no recurrences at 5 years though,in a minority of patients, there was some skin thickening.

While the patient numbers in this audit are small and its statistics have low power, they demonstrate that successful retreatment of keloids tumours with radiotherapy alone is possible, with minimalside effects.

It can therefore be proposed that,when considering re−treatment of keloid recurrences by radiotherapy, other centres consider following this treatment regime, then audit outcome data to facilitate comparison and collation of results. A similar approach could also be employed more generally to permit a comparison of outcomes of treatment when other treatment methods, e.g. steroid tape, cryotherapy, laser, etc., are used.

References:

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Nucleoresection Combined with Punch Extraction: A Keloid Surgical Technique Worth Promoting

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ACKGROUND

The treatment of large –area keloids remains challenging. Surgical intervention can effectively reduce keloid volume, creating favorable conditions for postoperative superficial radiotherapy. However, selecting the optimal surgical approach requires careful consideration by clinicians. Traditional direct excision with suturing or flap techniques, while reducing keloid volume, increases tension in the affected skin area –a key factor contributing to keloid formation and recurrence. Additionally, direct excision and suturing are not feasible for extensive keloids.

METHODS

Keloid nucleoresection addresses this by removing the core tissue while preserving the overlying skin, achieving "zero volume reduction without altering surface area," thereby avoiding postoperative skin tension. The combined punch extraction technique further optimizes outcomes by homogenizing skin flap thickness, draining subflap hematomas, and enhancing the efficacy of nucleoresection.

RESULTS

From November 2021 to October 2024, 369 keloid patients underwent keloid core excision combined with punch extraction, with routine postoperative superficial electron beam irradiation. Thefollow–up period ranged from 6 to 40 months. None of the patients experienced extensive or severe recurrence postoperatively. Partial localized minor recurrences tended to become flattened and softened after intralesional drug injections.The VSS and VASscores significantly decreased. ($p<0.05$)

CONCLUSION

The combination of keloid nucleoresection and punch extraction offers an effective treatment for large–area keloids and represents a surgical approach worthy of widespread promoting. Postoperative superficial radiotherapy can effectively reduce recurrence rates.

Use of Biopsy Punch Procedure to Treat Wide-spread Keloids

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Background:
Multiple spreading keloids over whole body represents a difficult challenge to physicians because surgical operation is not possible for wound coverage and drug injection is also not feasible considering sever side effect resulting from large area treatment.

Methods:
Biopsy punch was originally used for harvesting pathological tissue sample and the punch hole could be spontaneously healed via tissue regeneration. Because of this unique advantage, it has been commonly used for keloid treatment in China.

Results:
From mechanistic point of view, this procedure can significantly debulk the pathological tissue volume and thus significantly reducing symptoms of itching and sting via reducing paracrine production of pathological factors. In addition, it also efficiently disrupts the vascular network of keloid tissue, and thus help to control keloid development of recurrence. The punch diameter and the distance between the hole are the key parameters for achieving proper therapeutic results. In general, the thicker of the tissue, the bigger of punch diameter should be applied, and distance between holes similar to the hole diameter should be applied to control the proper hole density. To achieve rapid debulking, a denser punch procedure should be applied. In addition, post−surgical radiotherapy and laser therapy can be combined to effectively control the recurrence.

Conclusion:
This talk will present the application of this procedure for spreading keloids in the area of chest, back, extremity and mandibular regions, which demonstrates a feasible way of spreading keloid treatment.

Comprehensive Management of Large Auricular Keloids: The Role of the Innovative ABC Surgical Technique in a Multimodal, Multidisciplinary Approach

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BACKGROUND:

Auricular Keloids, particularly those resulting from ear piercings, present significant cosmetic challenges for patients. Conventional treatment methods for larger keloids often fail to prevent significant changes in ear contours and are associated with a high recurrence rate. Achieving both low recurrence and optimal aesthetic restoration remains a major challenge in clinical practice. This study introduces a structured, multidisciplinary team (MDT) - guided management protocol integrating the innovative ABC surgical technique with preoperative assessment, precise surgical intervention, and multimodal postoperative management. The ABC surgical technique was specifically designed to balance complete lesion removal with maximal ear contour preservation, making it a key component in optimizing outcomes. By leveraging a collaborative approach among surgeons (dermatologists or plastic surgeons), radiation oncologists, and the nursing team, this model aims to improve treatment efficacy, minimize recurrence, and enhance aesthetic results.

METHODS:

A total of 17 patients with 23 auricular keloid lesions were treated using an MDT-guided three-phase management strategy:

1. Preoperative Assessment & Planning (Multidisciplinary Collaboration)

Team Roles: A collaborative effort among Surgeons (dermatologists or plastic surgeons), radiation oncologists, and the nursing team ensured an individualized treatment approach. Three-Dimensional Evaluation: Comprehensive assessment of contour preservation, angle adjustment, and lesion minimization to guide precise surgical planning.Psychological support & patient education: Enhancing treatment adherence and preparing patients for postoperative care.

2. Surgical Procedure (ABC Surgical Technique, Led by the Surgical Team)

Arcuate incision design: Optimized flap planning to ensure adequate defect coverage and prevent deformities.Blind dissection for keloid lesion flap: Minimizing vascular disruption while preserving blood supply.Centrifugal keloid core serial shave excision: A gradual excision method balancing complete lesion removal with optimal contour restoration.

3. Postoperative Comprehensive Management (Collaborative Medical & Patient Engagement)

1) Medical Interventions

Postoperative radiotherapy as the primary modality, administered by radiation oncologists, with optimized dosage and irradiation strategies to prevent recurrence.

Adjunctive compression therapy, ensuring uniform pressure application to minimize the risk of lesion regrowth.

2) Patient–Guided Care (Nursing Team & Patient Collaboration)

Wound care education led by nursing professionals to prevent infections and promote healing.

Training on the proper use of compression devices, ensuring continuous and effective pressure distribution for optimal postoperative outcomes.

3) Long–Term Follow–Up (Ongoing MDT Monitoring & Adjustments)

Regular follow–up (2 months – 2 years) conducted by surgeons and the nursing team, monitoring lesion recovery and adjusting management strategies as needed.

Aesthetic evaluation of ear contour restoration, ensuring high patient satisfaction and refining postoperative rehabilitation approaches.

RESULTS:

Follow–up ranged from 2 months to 2 years (mean: 9 months).

Low recurrence rate: Only 1 recurrence (4.3%), significantly lower than conventional methods.

Aesthetic improvement: In non –recurrent cases, ear contours were well restored, appearing natural.

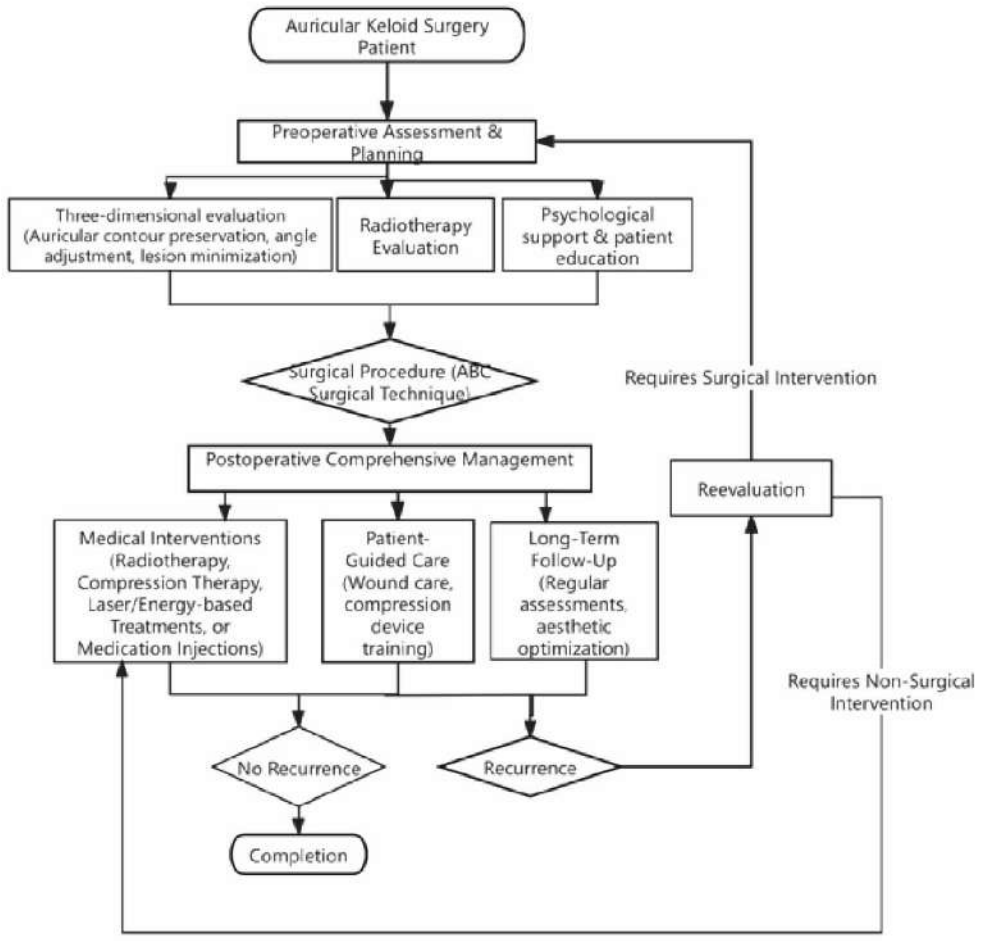
High patient satisfaction: Most patients reported excellent cosmetic outcomes.

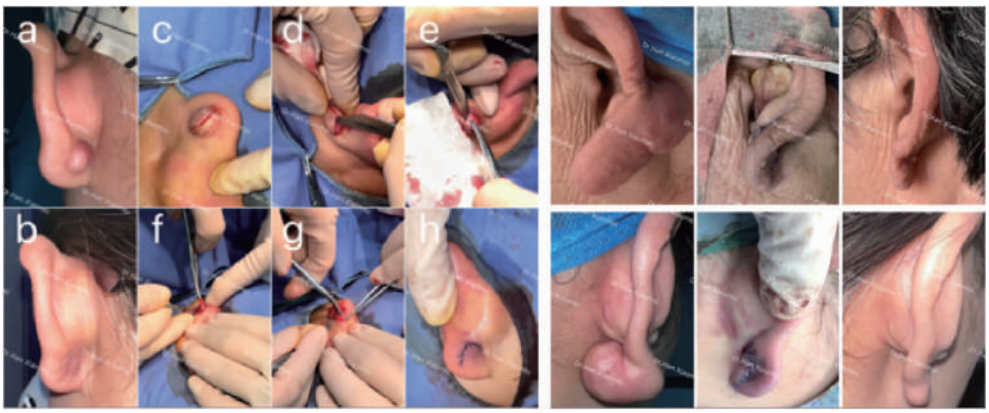
The ABC surgical technique, in combination with postoperative radiotherapy and compression therapy, significantly enhanced treatment effectiveness, demonstrating a synergistic effect in reducing recurrence while preserving ear aesthetics. The meticulous excision approach in the ABC technique further contributed to improved contour restoration and reduced postoperative complications.

CONCLUSION:

This study presents an MDT–based structured treatment protocol integrating preoperative assessment, the ABC surgical technique, and multimodal postoperative care to provide a systematic and highly effective approach for auricular keloid disorder management. The ABC surgical technique plays a pivotal role in this model, offering a refined surgical method that enhances precision, optimizes contour restoration, and reduces recurrence risk. By leveraging multidisciplinary team collaboration among surgical, radiation, and rehabilitation specialists, this model ensures low recurrence rates, superior aesthetic outcomes, and high patient satisfaction, offering a promising framework for future clinical application.

Comprehensive Workflow Management of Auricular Keloid Surgery Patient





Excision of Keloid without Radiotherapy

Yang Dongyun, Dongshang Med.

Objective:
Although radiotherapy can effectively control the recurrence of keloids, there are always some situations where radiotherapy is not suitable, such as in minors and glandular areas. Therefore, it is necessary to explore the feasibility of not performing radiotherapy after keloid resection.

Methods:
After complete resection of the keloid, the subcutaneous tissue is fully mobilized, followed by adequate distant tension reduction. Non-invasive procedures are strictly followed during the operation. No radiotherapy is administered postoperatively, and the recovery of the scar is followed up.

Results:
Postoperative scars not only had a better appearance but also showed no uncontrolled recurrence. Although mild hyperplasia occurred in some cases, it was effectively controlled through treatments such as medication and compression.

Conclusion:
Our long-term and extensive clinical practice has confirmed that, on the premise of adequate distant tension reduction and non-invasive operations, recurrence can be effectively prevented without radiotherapy postoperatively for many keloids and most hypertrophic scars. Therefore, radiotherapy should generally be avoided for scars in minors and glandular areas.

Application of Hyaluronic Acid and Its Functionalization Technology in the Field of Scars

Wenbo Wang, MD, PhD

Hyaluronic acid (HA) is one of the main components of the extracellular matrix (ECM) and plays a crucial role in wound healing through its binding to cell surface receptors, exerting important biological functions related to wound healing, such as promoting cell proliferation, adhesion, regulating inflammation, and alleviating pain.The functionalization technology of hyaluronic acid, achieved by chemically modifying the active groups, not only retains the original functions of hyaluronic acid but also imparts new material and biological properties suitable for clinical applications. This technology has been widely used in the biomedicine field, including targeted drug delivery, drug carriers, hydrogels, tissue engineering scaffolds, and cell matrices.Numerous clinical studies have proven that functionalized hyaluronic acid plays a vital role in promoting wound healing, preventing scar formation, and scar repair.

DAY 2: SATURDAY JUNE 7, 2025

SESSION 4 *16:20–18:10*

ADVANCES IN NON-SURGICAL MANAGEMENT OF KELOID PATIENTS

Bridging a Therapeutic Gap: Hydrocortisone Occlusion Dressing after Keloid Excision in Indonesia's National Referral Hospital: A Case Series

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[2] Division of Plastic Reconstructive and Aesthetic Surgery, Department of Surgery, Universitas Indonesia Hospital, Faculty of Medicine Universitas Indonesia, Depok, Indonesia

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BACKGROUND:

Commercial steroid tape is widely regarded as the benchmark adjuvant after keloid excision, yet it cannot be sourced in Indonesia. We therefore explored whether a simple hydrocortisone 1% cream sealed beneath a transparent polyurethane dressing could fill that therapeutic gap.

METHODS:

Twelve consecutive patients underwent excision followed, within 24 hour, by radiation. Starting on postoperative day 3, patients was applied hydrocortisone 1% covered with occlusion dressing daily, for 12 weeks. Outcomes are measured using Vancouver Scar Scale (VSS) and Patient/Observer Scar Assessment Scale (POSAS) were recorded every four weeks. Adherence and adverse events were logged at each visit.

RESULTS:

All twelve patients demonstrated significant improvements in scar height, redness, and softness as measured by VSS and POSAS. Patients reported high satisfaction with the ease and comfort of the treatment. Importantly, no significant adverse effects or skin irritation were observed, which are commonly associated with steroid tape.

CONCLUSION:

This preliminary series indicates that an inexpensive hydrocortisone–occlusion regimen can approximate the published efficacy of steroid tape while avoiding its supply limitations in Indonesia. A randomised controlled trial with a larger cohort is planned to validate these findings.

Keywords:

keloid; hydrocortisone; occlusive dressing; steroid tape substitute; Indonesia

Keloid scar and Telemedicine - apatient's

Yap Patricia

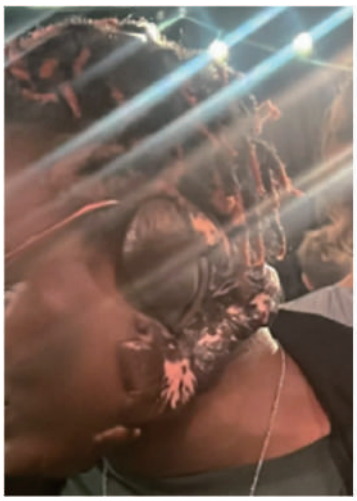
Background:

Telemedicine is an evolving method of telecommunication designed to provide healthcare at a distance.

Given the visual nature of keloid scars, telemedicine is an ideal method to treat patients who live far away from medical facilities. Telemedicine also provides a cost effective method of treatment for persons who live in remote and rural settings, who would otherwise not have access to medical care.

Methodology:

A young Nigerian woman (O.I) embarked on her journey to the 4th International Keloid Symposium in France to seek a solution to her keloid scars problem since the age of Two (2). O.I . asked DR Pat Yap to help her to treat her symptoms of pain and itchiness which have prevented her from getting a full night sleep. Despite using many of the latest treatments, all her existing keloid scars continued to grow with new keloid scars forming; either spontaneously or as a result of minor trauma. Dr. Yap informed her that all the existing treatment can be used adjunct to Keloflat corticosteroid cream.



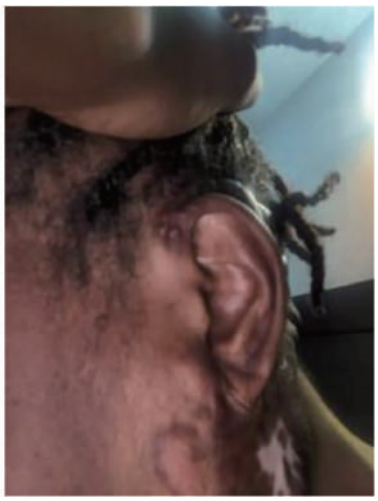


Fig1. O.I. was provided with Keloflat corticosteroid cream, she was asked to apply the cream with or without occlusion, after bathing twice a day. In the span of a week O. I. experienced mitigation of her symptoms; pain and itching, she is now able to sleep through the night. After O.I. used Keloflat corticosteroid cream for eight (8) months on her left ear she was then able to have it surgically removed by her plastic surgeon due to the softening of the keloid and ease of handling during surgery. After two weeks when the sutures were removed, she was able to use Keloflat corticosteroid cream to prevent keloid scars from forming.

In conclusion, telemedicine and the use of Keloflat corticosteroid cream provides a solution oriented approach to the management of keloid scars. It provides an opportunity for remote consultation, initiation of treatment and appropriate continuation of care, as needed. The Keloflat corticosteroid telemedicine approach can work in isolation but also serves as a helpful complement to other traditional modalities for keloid scar therapy – corticosteroid injections, surgery, laser therapy, cryotherapy and radiation

The combination of this approach and conventional treatments has the potential to improve patient outcomes in patients who would not otherwise have access.

Coping Tendencies Play Partial Mediating Role Between Social Support and Anxiety/Depression Among Chinese Keloid Patients

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BACKGROUND:

In addition to producing pain and itching, keloids can cause psychological symptoms, including anxiety and depression. We aimed to investigate the prevalence and underlying factors of anxiety/depression in Chinese keloid patients and to explore the mediating role between social support and anxiety/depression.

METHODS:

A total of 202 self−reported questionnaires were collected from keloid patients, including the General Information Questionnaire, Vancouver Scar Assessment Scale (VSS), Patient and Observer Scar Assessment Scale (POSAS), Dermatological Problems Quality of Life Inventory (DLQI), Social Support Rating Scale (SSRS), Simple Coping Styles Scale (SCSQ), Self−Efficacy Scale (GSES) and the Heart Attitude Assessment (HADS).

RESULTS:

The mean scores for anxiety and depression were 9.82±2.20 and 7.96±2.70, respectively. Annual income (OR=0.258), GSES score (OR=0.2955), pain symptoms (OR=1.281) and coping styles (OR:3.321) were significantly associated with HADS anxiety in keloid patients ($P<0.05$), whereas use of support (OR=0.607) was significantly associated with HADS depression in keloid patients ($p<0.05$). The area under the curve (AUC) for the combined anxiety ROC for annual income, coping styles, pain symptoms and GSES scores was 0.835. PROCESS analysis concluded that coping tendencies partially mediated

the relationship between social support and depression, with the mediating effect accounting for 38.58% of the total effect.

CONCLUSION:

Coping tendency in keloid patients played a partial mediating role between social support and depression. Future studies should further explore how training in coping tendencies can enhance the effectiveness of social support to more effectively prevent and reduce depressive symptoms.

Treatment of Keloids with Long-pulsed 532/1064nm Laser and Intralesional Injection

Haiguang Zhao, MD, PhD

Plastic Surgery Department, The Third Qi Lu Hospital affiliated to Shandong University School of Medicine

Background:
Keloid scars are highly prevalent in the general population and irritating both physically and mentally.There are various treatment methods available.

Objective:
Considering the diversity of existing therapies, long−pulsed 532/1064nm laser was rarely used for the treatment of keloids. The combination of this laser and intralesional injection was undertaken.

Case Presentation:
A 60−year−old woman had a large area of scar tissue on her chest. Due to the obvious red color of some scars, we used long −pulsed 532/1064nm laser treatment before injection therapy, while the remaining scar were treated with simple injection therapy. The interval between each treatment is one month. After 5 treatments, the scar symptoms, color, and height in the combined treatment area were significantly better than those at the injection site alone.

Conclusion:
Our case suggest therapy using a combination of long −pulsed 532/1064nm laser combined with intralesional injection seems safe and increases the recovery level.

Keywords:
long−pulsed 532/1064nm laser; Intralesional; Keloid; hypertrophic.

Understanding the Emotional Impact of Living with Keloids: A Clinical Perspective on Body Image and Psychological Burden

Lina Ndjock Mbolong, MD

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Introduction:

Keloids have been receiving more attention recently, along with growth in the body of skin−of−color research and yet, a major understudied feature of keloids is their impact on patients' quality of life. Keloids are more than just excess skin growth. They can bring out a wide variety of emotions and challenges. This brief presentation has as a goal to shed light on the emotional toll of living with keloid diseases, providing understanding to those living with theses lesions.

Methods:

Two studies summarized from literature:

A clinical observational study, conducted involving 61 patients with keloids located in socially exposed areas (face, neck, upper chest, arms). Each participant underwent a comprehensive evaluation including clinical examination, psychological assessment through structured anamnesis, and standardized psychometric tools: the Body Dysmorphic Symptoms Scale (BDSS)and the Rosenberg Self−Esteem Scale.

A screening of 340 results to identify 17 studies that directly assessed the impact of keloids on the quality of life through 7 biopsychosocial domains: pain and itching, mobility, visibility, gender and age, psychosocial, stigma and cosmetic.

Results:

Among the 61 participants, over 40% exhibited significant disturbances in body image (BDSS score > 6), with notable symptoms of self−consciousness, anxiety, and social withdrawal. The negative impact was particularly pronounced in individuals with larger or multiple lesions in highly visible locations. There was a clear correlation between the extent of keloid visibilityand reduced self−esteem, highlighting the need for psychological support as part of comprehensive keloid management.

Conclusion:

Keloid disorder extends beyond physical disfigurement; it deeply affects mental health and quality of life, especially in patients with visible lesions. Body image issues and reduced self−esteem are common, yet under−recognized, aspects of the disease. These findings underscore the urgent need to integrate psychosocial assessment and support into standard keloid care.

Treatment strategies should not only aim to reduce lesion burden but also address the emotional and psychological needs of patients living with this challenging condition.

DAY 3: SUNDAY JUNE 8, 2025

SESSION 1 *08:30–10:30*

COMPLEXITIES IN KELOID DISORDER: CLINICAL REALITIES ACROSSAGE GROUPS

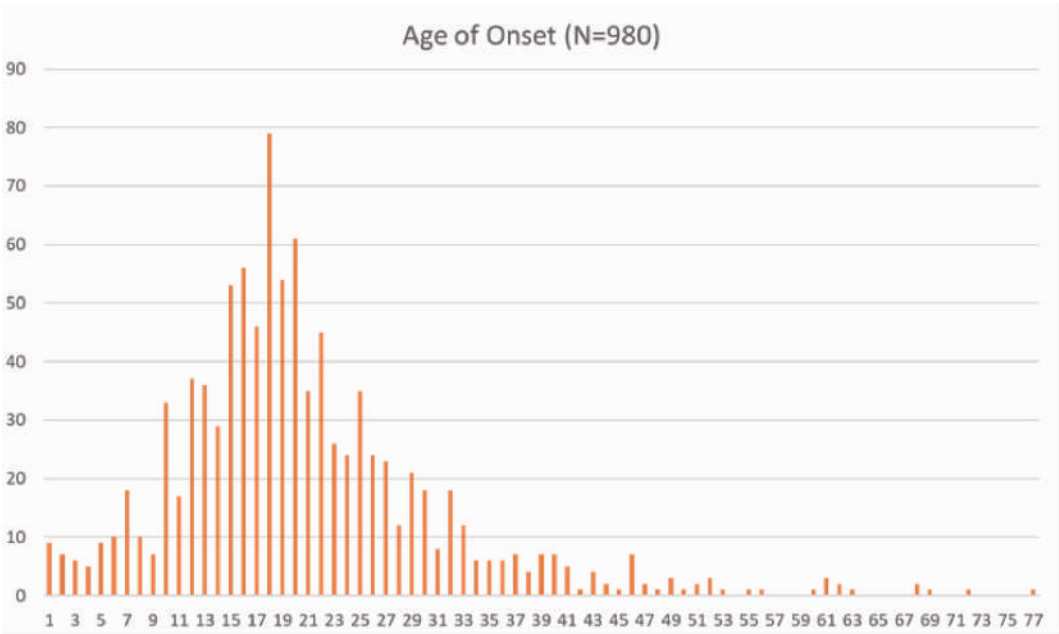
Challenges in the Treatment of Pediatric Keloids

Michael H Tirgan MD

Keloid Research Foundation

Background:

Approximately 63% of patients develop their very first keloid before they turn 18, making Keloid Disorder a pediatric chronic skin ailment. As shown in the graph below, children under the age of 10 are not spared and require proper treatment.



Methods:

Keloid removal surgery should be avoided in children at all costs, as it can result in detrimental outcomes that will be very difficult, if not impossible, to treat at a later date. Contact cryotherapy and intralesional chemotherapy have emerged as promising yet underutilized alternatives in the management of pediatric keloids.

Results:

Earlobes are common location for development of keloids among African American female children, as many families tend to pierce their children at a very young age. Other types of skin injuries can also lead to the formation of keloid in children who are genetically prone.

Conclusion:

This presentation will focus on review of clinical presentation of pediatric keloids as well as non–surgical management approaches and review of several case studies.

Combined Treatment of Post Surgery Keloids in the Pediatric Population, Cases Report

Dan Deng, Jinye Guan, Ruicheng Tian, Junwen Ge, Jingyi Yang, Chao Zhang, Meiyun Li, Zhuo Chen

Background:
Keloid scars present in the pediatric population a difficult treatment challenge due to lack of safety evaluation of chemotherapy and radiotherapy.Surgical treatment combined laser or steroid injection supposed to be options. We hypothesize that timing of steroid injection and pressure therapy may improve outcomes in combination with lesion excision.

chemotherapy, or post−surgery radiotherapy

Methods:
Five patients with 8 keloids occured after surgery were treated using steroid injection and pressure therapy, in combination with lesion excision. Strict follow−up was enforced, with continueous pressure clothes and repeat injections as needed at any sign of abnormal scar formation postoperatively. Dermascopy or 3D camera system were used to evaluate.

Results:
Of 8 lesions, 5 (62.5%) were treated successfully with no sign of lesion recurrence at 6 months of follow−up. Three lesions of long time follow−up and presented 12 months postoperatively with recurrence. These lesion were subsequently retreated.

Conclusions:
Steroids injection, pressure therapy and laser therapy in combination with excision is a well−tolerated and effective treatment of keloids in the pediatric population. Safety is the most important issue for evaluation. Contineous pressure and adherence to a strict follow−up regimen is crucial to success. Digital methods are promised to be widely applied for accurate observation.

The Therapeutic Principle for Pediatric Keloids

Wei Liu, MD, PhD

Department of Plastic and Reconstructive Surgery, Shanghai Ninth People's Hospital, Shanghai Jiao Tong University School of Medicine

Pediatric keloid represents a major challenge to physicians simply because chemotherapy and radiotherapy, which is the most valuable and commonly used therapies, are the anti-indication for pediatric patients. In my practice, the general principles for pediatric keloid include:

1. Conservative therapy is the first line choice by using pressure therapy, steroid cream or steroid tape or local drug injection.
2. As long as the keloid can be under control without aggressive invasion and enlargement, radical treatmentsuch as surgical removal plus irradiation will not be employed until the age around 16 years old.
3. When the keloid presents an aggressive enlargement and invasion manner, surgical debulking procedure such as nucleoresection procedure or partialresection is employed to remove large part of the keloid in order to improve the pathological niche environment by reducing local production of growth factors and inflammatory cytokines, and thus to stop the aggressive progression of the keloid. Skin graft and flap will never be applied in order to prevent new keloid formation. When needed, repeating procedures can be applied until the aggressive progression is hold.
4. Post-surgical irradiation wound not be applied to pediatric patients, rather, pressure therapy and topical drug application were usually employed to assist the recurrence control.

This principle has been used in my treatment for pediatric keloids, which works well. This talk will present some cases to share my experience.

Microbiome Dysbiosis Dominated by Rhodococcus Occurs in Keloids

Jiarong Yu[1], Zhigang Mao[2], Zongan Chen[2], Lei Yi[1], Xiaoli Wu[2], Xiqiao Wang[1]

Background:
Keloid is a pathological scar that grows like a tumor.Previous studies revealed that chronic inflammation in keloid contributes to its formation, however, the mechanism is poorly understood.

Objective:
Currently, numerous inflammatory diseases are correlated with microbiome dysbiosis, therefore, this study aims to investigate the presence of microbiome dysbiosis in keloids and its potential correlation with keloid formation.

Methods:
A total of 35 keloid and 36 normal skin (NS) samples were collected, and the keloid severity was evaluated using Vancouver Scar Scale (VSS)score. The microbiota in the tissues was assessed by 16S rRNA sequencing, followed by an investigation into the correlation between microbiota and clinic indices. In addition, 10 keloid and 10 NS samples were dissociated into single cells, and flow cytometry was used to analyze the proportion of T cells in keloids and NS tissues.

Results:
The richness of the bacteria community in keloid was significantly reduced than that in NS. Additionally, the microbiota composition in keloid was different from that in NS. At the phylum level, *Firmicutes* was significantly higher in keloid than in NS, while *Rhodococcus* was the dominant species in keloid at the genus level. *Acinetobacter*, was also found to be positively correlated with keloid formation. Furthermore, *Rhodococcus* demonstrated a higher predictive value for keloid severity than *Acinetobacter*. Interestingly, the bacterial composition varies during keloid progression. Compared to keloids<10 years, the proportion of genus *Rhodococcus* reduced significantly in keloids ⩾10 years, while the proportion of genus *Cutibacterium* significantly increased compared to keloids<10 years.The proportion of CD4 + and CD8 +T cells in keloid scar tissues was also significantly decreased compared with NS.

Conclusion:

Microbiome dysbiosis occurring in keloids was dominated by *Rhodococcus*, which may be correlated with the reductionof CD4+ and CD8+T cells. Targeting microbiome dysbiosis may be a prospective approach future keloid management.

Keywords:

Microbiome dysbiosis; Keloid; Inflammation; Vancouver Scar score; *Rhodococcus*

How do I Deal with the Keloid Disease: A Qualitative Study Exploring Coping Strategies in a Multicultural Adult Patient Population

Louter J.M.I.M, MSc[1], Niessen F.B, MD PHD[1], Lapid O, MD PHD[1,2]
Mullender M.G, Prof Dr[1], van de Grift T.C Dr PHD[1,3]

[1] Department of Plastic, Reconstructive and Hand Surgery, Amsterdam UMC location Vrije Universiteit Amsterdam, Amsterdam, The Netherlands.
[2] Pediatric Surgical Centre, Emma Children's Hospital, Amsterdam UMC location University of Amsterdam, Amsterdam, The Netherlands.
[3] Department of Psychiatry and Medical Psychology, Zaans Medical Center, Zaandam, the Netherlands.

Background
Abnormal, excessive keloids come with high recurrence rates and persisting complaints as pain and itch after semi-successful treatment. This affects patients' quality of life. The role of coping with chronic disease or disability remains unknown in this population. Coping strategies can contribute to (mental) health and quality of life, by supporting positive adaption to a chronic disease and coping with ever-changing challenges in life.

Aim The aim of this study is to identify and explore coping for a multicultural adult patient population who suffer from keloids.

Methods
This Ethical approved qualitative study explored patient experiences and subsequent coping strategies using semi-structured interviews. A purposive sampling (n=13) was performed, including patients with different demographic and clinical backgrounds. Interviews topics included reactions and feelings among the early stage of having keloids, living with keloids and other life-changing events, social-support, information, and support among the care for the keloid. Data were audio-recorded and transcribed. The data were analyzed using thematic analysis with inductive and deductive coding methods.

Results
The age from the participants ranged from 17 to 67 years, 7 were female and 5 had skin type 4 (moderate brown). The number of the keloids varied between 1 and 30. Four stressor groups were identified as physical, pre-treatment, treatment related and psychosocial stressors.Deductively identified coping strategies found were: problem-focused, emotion-focused, social-support, religious/spiritual, and avoidant coping.

Problem–focused coping was described as focusing on other aspects of their life which they think they could control, and obsessively controlling while leaving the uncontrollable aspects as keloid evolution and ineffective treatments behind, resulting in overachieving. Early–life events identified the urge for self–reliance, social context was identified to fit into the society and cultural background gave insights into not Western cultures experiencing the feeling of pressure to thrive in the modern society, survival and competing in the modern society and protection which formed their coping strategies.

Conclusion

In this study was found that patients suffering from keloid disorder use coping strategies as obsessively controlling other aspects of their lives, hiding the keloid, seeking help during exacerbations, and relying on social support. Addressing these coping strategies, professionals can enhance patients' adaption to living with a chronic disease.

DAY 3: SUNDAY JUNE 8, 2025

special lecture *10:00–10:30*

THE ANNUAL JOUNI UITTO, MD, PHD INTERNATIONAL VISITING PROFESSORSHIP AND LECTURE IN MOLECULAR DERMATOLOGY CLINICAL BIOMARKERS

The Future of Keloid Disease Diagnosis and Therapy

Lamont R Jones, MD, MBA

Keloid disease (KD) is characterized by painful and itchy benign fibroproliferative tumors of the skin which result from injury in genetically predisposed individuals. KD has high recurrence rates and there has been little progress in treating KD, mainly due to an incomplete understanding of its pathogenesis. The development of KD specific diagnostic and prognostic biomarkers has the potential to have significant clinical impact by identifying clinical subgroups and novel therapeutic targets.This presentation will describe how innovative spatial technology can be used to identify prognostic and diagnostic KD biomarkers in formalin fixed paraffin embedded tissue and their future use as novel therapeutic targets.

DAY 3: SUNDAY JUNE 8, 2025

SESSION 2 *11:05–12:30*

ABSTRACT PRESENTATION – CLINICAL SCIENCE

Application of Core Excision in the Treatment of Keloid

Chunhui Xie[1], Jiaao Yu[1], Xinxin Gao[1], Xiaoli Wu[2]

[1] *Burns Department, The First Hospital of Jilin University*

[2] *Department of Plastic Surgery, Shanghai Ninth People's Hospital, Shanghai Jiao Tong University School of Medicine*

Background:

In the treatment of keloid, surgery combined with radiation therapy is a very effective treatment, and the recurrence rate is relatively low. However, in some small areas with multiple keloids, direct surgical resection is difficult. If the wound tension is too large to be sutured directly after the resection of the pathological tissue, skin grafting or flap transfer is needed to close the wound and increase new wounds, and the local tension after suturing is too large, which may lead to new scars in the operative area. Therefore, we suggest that small keloid lesions, especially those with sloping edges, can be performed coreexcision.

Methods:

Retrospective analysis was performed on the patients who underwent core excision in our hospital from January 2021 to December 2021, and they were divided into high tension group (chest, shoulder, mandible, limb group) and low tension group (ear group) according to scar sites. For each scar, a separate core resection was performed, an incision was designed along the long axis of the scar, 1mm scar epidermal and dermal tissue were retained on both sides, the core collagen of the scar was removed, and the scar flap was sutured and fixed, and 12Gy electron-beam radiation therapy was performed once within 24 hours after surgery. After 1 year of follow-up, silicone gel and silicone sheet were routinely used for treatment. If scar hyperplasia occurred, local steroid injection was given, once every one month; if scar hyperplasia continued more than 6 months after surgery, carbon dioxide fraction laser treatment was given, and once electron-beam radiotherapy was given within 24 hours after treatment with a radiation dose of 12Gy. The patient and observer scar assessment scale (POSAS) was applied before and one year after treatment (recorded as after treatment) to evaluate the changes of keloid before and after treatment, and the efficacy was determined according to the efficacy criteria such as Liu Wenge.

Results:

A total of 52 patients were included, 331 cases of keloid, 298 cases were cured, 29 cases were effective, 4 cases were ineffective, the total effective rate was 98.79%. In 259 cases of high tension group, 231 cases were cured, 24 cases were effective, 4 cases were

ineffective, the total effective rate was 98.46%; In the hypotonic group of 72 cases, 67 cases were cured, 5 cases were effective, 0 cases were ineffective, and the total effective rate was 100%. The total POSAS score after treatment was significantly lower than that before treatment ($P<0.05$), and there was no significant difference between the high tension group and the low tension group.

Conclusion:

Core excision combined with postoperative electron-beam radiation therapy is a safe and effective treatment for keloid, especially for ear part.

Key words:

Keloid, Core excision, Radio therapy, Operation.

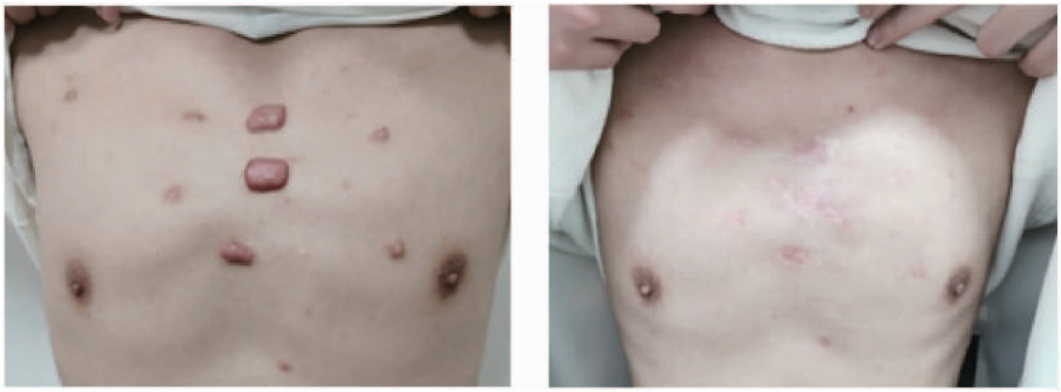


Fig 1. Left: Before treatment; Right: 12 months after treatment

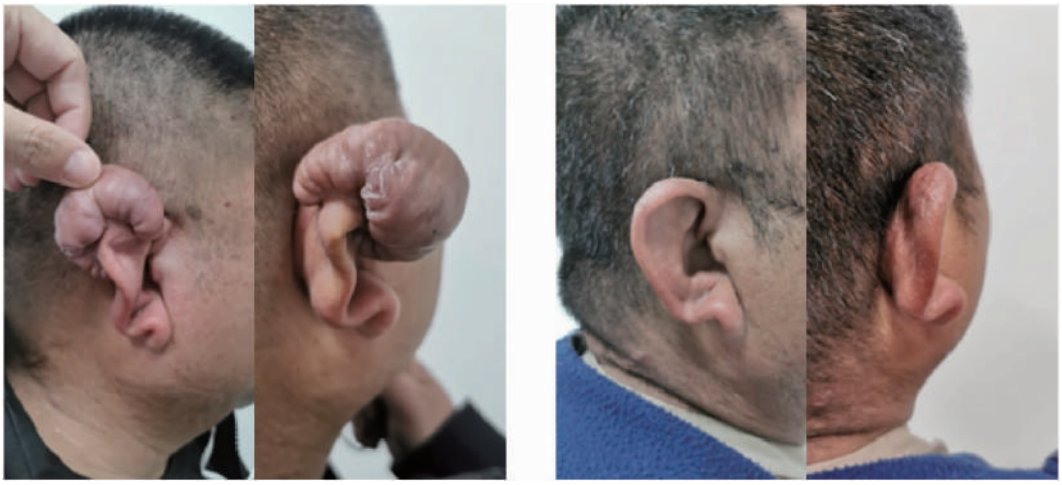


Fig 2. Left: Before treatment; Right: 13 months after treatment

Clinical Observation of Subepidermal Vascular Network Flaps in Keloid Patients

Hao Yan (First Author), Shan Mengjie, Liu Hao, Song Kexin, Chen Qiao, Meng Tian; Feng Cheng, Wang Zhi, Qi Zheng, Xia Yijun, Wang Youbin(Corresponding Author)

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BACKGROUND:

There are many different keloid treatment modalities. One surgical technique is to keep the ''shell'' of the keloid to over the defect. We named this "shell" keloid subepidermal vascular network flap (KSVNF), and we outlined the characteristics of this flap by observing 35 flaps in keloid patients.

METHODS:

A total of 35 KSVNFs were designed in 15 patients during 2020−2021. All patients underwent the operation and adjuvant radiotherapy as well as hyperbaric oxygen therapy. All flap lengths and widths were recorded, and the blood perfusion of the flaps was measured on the first day postoperation and the day of stitch removal. Flap survival and the quality of flaps were evaluated on the day of stitch removal. All harvested data were analyzed using the R (version 4.0.1) package.

RESULTS:

The mean blood perfusion on the first day postoperation (pod1) and the day of stitch removal was 120.4013 and 168.6900, respectively ($p = 0.02249$); 2 flaps had partial necrosis (5.714%). Receiver operating characteristic (ROC) curve analysis showed that when the length/width ratio was less than 1.05, the quality of the flap was good (AUC = 0.724), which suggests that the effective safe length/width ratio was 1.05.

CONCLUSION:

KSVNF is an applicable method for covering the remaining wound after keloid mass removal with sufficient blood perfusion and adequate skin quality. We recommend that the length/width ratio of the flap design not exceed 1.

Surgical Approaches and Experiences for Keloid Treatment and the Adjunctive Therapy

Bing Li and Tao Zhao

Xijing Hospital of the Air Force Medical University

BACKGROUND

Current treatment options for keloids include surgical excision and local corticosteroid injections. However, the high rate of recurrence remains a significant challenge. Current guidelines and expert consensus suggest that treatment strategies should be tailored based on the size of the lesion. Keloids with a diameter of less than 2 cm are typically managed with corticosteroid injections alone, while those larger than 2 cm are often treated with surgical excision followed by adjuvant radiotherapy. Despite these approaches, managing patients with multiple or scattered keloid lesions remains particularly difficult.

METHODS

We present several cases in which the choice of surgical method was guided by the anatomic location and the morphological characteristics of the lesions.

RESULTS

For larger keloids on the ear, core excision is the preferred surgical approach, as it helps preserve the natural shape of the auricle. Large keloids on the lower jaw or trunk often originate from acne or folliculitis. In such cases, the choice of surgical technique is guided by the JSW Scar Scale (JSS), the lesion's growth pattern, and its surface characteristics.

When the JSS elevation score is 3 and the lesional surface is rough, the optimal approach is complete excision, followed by low–tension primary closure, skin grafting, or the use of local flaps.

If the surface is smooth and the lesion remains within the wound boundary, core excision is the first choice. However, if the surface is smooth but the lesion extends beyond the wound boundary, excision followed by skin grafting using tissue harvested from the keloid itself is recommended.

In patients with multiple, partially confluent keloids, the micro–punch technique is employed when the JSS elevation score is less than 3, whereas core excision is indicated when the elevation score is 3.

Electron beam irradiation is administered within 24 hours post–surgery. Compression therapy, intralesional injections, and 595–nm pulsed dye laser treatments can be selected as adjuvant therapies to reduce recurrence and optimize outcomes.

CONCLUSION

The choice of surgical method can be guided by the anatomic location and the morphological features of the keloid lesions. Electron beam irradiation, compression therapy, intralesional injections, and 595–nm pulsed dye laser treatment can be selected as adjuvant therapies, based on the characteristics and distribution of the keloids.

Rationale Based Repurposing of FDA-Approvedagents for Keloid Management: Anin Vitro Study

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BACKGROUND

Effective management of keloid disorder remains elusive, as it often resists monotherapy and demonstrates a high rate of recurrence. Several FDA–approved drugs developed for treating both fibrotic and unrelated conditions have the potential to target key pathways involved in keloid pathogenesis, and some of these have been used for treating keloids with limited efficacy. However, the effects of combinations of these drugs have not been studied systematically. Given the multifactorial nature of keloid formation, we hypothesized that rationally designed drug combinations could produce additive/synergistic anti–fibrotic effects on keloids when administered locally at lower doses, thereby reducing side effects and the development of resistance to treatment.

METHODS

Primary keloid fibroblasts cultured from surgically removed keloids were expanded and used at low passage. All procedures involving human–derived tissues were approved by the Florida State University Institutional Review Board (IRB STUDY00001981, Approval Date: 05/08/2021) following informed consent from all donors. Cells were seeded in triplicate and either left untreated, or treated for seven days with single, double, triple, and quadruple combinations of the following FDA–approved drugs:

Triamcinolone Acetonide (TA), Nintedanib (Nin), Pirfenidone (Pir), Sulindac (Sul), Minoxidil (Mino), Vorinostat (Vor), Verteporfin (Vtf), and Verapamil (Vera) — administered with or without low–dose (1 Gy) ionizing radiation (IR). Relative cell viability was measured by counting cells on a Coulter Counter. Synergistic interactions among drug combinations were assessed using Bliss Independence analysis. Levels of type I collagen and alpha –Smooth Muscle Actin (α–SMA) were measured by indirect immunofluorescence and Western blotting.

RESULTS

At the concentrations employed, single–agent treatments yielded modest reductions in keloid fibroblast viability (~84.5%). In contrast, triple and quadruple combinations significantly decreased viability to 42.6% and 26.8%, respectively. Radiation increased the impact of most drugs. A dramatic reduction in type I collagen levels was observed with triple–drug combinations, particularly after 7 days of treatment, indicating a time–dependent anti–fibrotic effect.

CONCLUSION

This study demonstrates that rationale–based combinations of FDA–approved drugs can synergistically suppress keloid fibroblast viability, proliferation, and fibrotic markers *in vitro*. By targeting multiple fibrotic pathways at once, these drug combinations make it harder for keloid fibroblasts to develop resistance, a potential problem with single–drug treatments. Additionally, due to their additive/synergistic effects when used in combinations, lower doses of individual drugs are required, which would reduce the potential for adverse effects. These findings highlight the therapeutic promise of low–dose, multi–agent, locally administered therapies as a more effective approach for managing keloid disorder. *Combinations of these FDA approved agents can now be tested by clinicians for safety and efficacy in keloid patients following intralesional or topical administration, with or without superficial radiation.*

Disclosure Declaration

The authors have no conflicts of interest todeclare.

Funding

This research was not supported by any external funding.

An Innovative Single-stage Approach of High-tension Keloid Excision and Reconstruction Using Acellular Dermal Matrix and Epidermal Skin Grafting

Wenbo Wang, MD, PhD

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Abstract

The treatment of keloids, particularly in high-tension areas, is challenging due to their extension beyond the original wound boundaries and high recurrence rates, thereby rendering traditional treatments ineffective. In this study,we investigated the effectiveness of a novel single-stage treatment approach that combines acellular dermal matrix (ADM) with keloid-specific epidermal skin grafting.To further prevent recurrence after neo-skin formation, the treatment was followed by fractionated laser and radiation therapy (LCR). Seven patients with high-tension keloids, including one with keloids at two locations, were treated and followed-up for an average of 15.9 months. The patients showed significant improvements in wound healing and skin appearance, with a marked reduction in the Patient and Observer Scar Assessment Scale (scores from 91.1±5.6 to 23.8±6.1 [$p < 0.001$]). This approach effectively minimizes tension, reduces the likelihood of keloid recurrence, and serves as a viable alternative to conventional methods that often involve challenges related to donor-site acquisition. No recurrence was observed during the follow-up period, indicating a promising innovationin the management of extensive keloids and offering improved healing and aesthetic outcomes, particularly in high-tension areas.

Keywords:

Keloids, acellular dermal matrix, keloid epidermal skin grafting, single-stage treatment, high tension

Keloids and Inflammation: The Crucial Role of IL-33 in Epidermal Changes

ZongAn Chen, YaTing Yang, XiuXia Wang, LingLing Xia, WenBo Wang, XiaoLi Wu, Zhen Gao

From the Department of Plastic and Reconstructive Surgery, Shanghai Ninth People's Hospital, Shanghai Jiao Tong University School of Medicine, Shanghai 200025, China.

BACKGROUND:

Keloids are benign fibroproliferative disorders characterized by excessive collagen deposition and inflammation that extend beyond the original wound boundaries.

METHODS:

In this study, through clinical sample and high-throughput analysis, we investigate the role of interleukin-33 (IL-33) in the pathogenesis of keloids, highlighting its expression in keratinocytes and its interactions with infiltrating lymphocytes.

RESULTS:

Our results demonstrate that IL-33 levels are significantly elevated in the epidermis of keloid tissues, where it functions as an alarmin, promoting a chronic inflammatory response. We further reveal a feedback loop between IL-33 and interferon-gamma (IFN-γ), whereby IL-33 induces IFN-γ production in lymphocytes, which in turn stimulates keratinocytes to produce more IL-33. This loop contributes to impaired keratinocyte differentiation and skin barrier dysfunction, exacerbating the inflammatory environment.

CONCLUSION:

By elucidating the role of the IL-33/ST2 axis in keloid formation, this research provides valuable insights into potential therapeutic targets for managing this challenging condition.

DAY 3: SUNDAY JUNE 8, 2025

SESSION 3 *13:30–15:00*

ABSTRACT PRESENTATION – BASIC SCIENCE

Integrated Multi-omics Unveils the Epigentic Landscape in the Pathogenesis of Keloid

Lian Zhang

Keloids are benign skin tumors, but their molecular mechanisms of pathogenesis remain unclear. Through multi−omics analyses, we identified that keloid pathogenesis involves transcriptional dysregulation driven by DNA methylation reprogramming. Compared to normal scars, keloid tissues exhibit a global DNA hypermethylation pattern, which is similarly observed in keloid fibroblasts. RNA−seq analysis revealed significant upregulation of bone and cartilage−related signaling pathways, while hormone−related pathways were markedly downregulated in keloid samples. Furthermore, inhibition of aberrant DNA hypermethylation using a DNA methyltransferase inhibitor suppressed the growth of keloid fibroblasts. These findings indicate that DNA methylation plays a crucial role in keloid pathogenesis, and targeting the regulation of bone, cartilage, and hormone−related signaling pathways may provide novel therapeutic strategies for keloid treatment.

TWIST1 Promote TGF-β Receptor I in Keloid Fibroblasts Via Regulating the Stability of MEF2A

Tianhao Li[1], Mingzi Zhang[1], Yunzhu Li[1], Yixin Sun[1], Jiuzuo Huang[1], Ang Zeng[1], Nanze Yu[1*], Xiao Long[1*]

[1] Department of Plastic and Cosmetic Surgery, Peking Union Medical College Hospital, Chinese Academy of Medical Sciences & Peking Union Medical College, Beijing, China.

BACKGROUND:

The bHLH (basic helix−loop−helix) transcription factor TWIST1 (Twist−related protein 1) controls cell proliferation and differentiation in tissue development and disease processes. Recently, TWIST1 has been associated with keloid and TGF−β signaling pathway, yet the role of TWIST1 in keloid derived fibroblasts (KFBs) remains unclear.

METHODS:

In this study, TWIST1 down and up regulation in KFBs, mass spectrometry, chromatin immunoprecipitation, and immunoprecipitation were applied to explore the function of TWIST1 in KFBs and tissue.

RESULTS:

In keloid tissue, TWIST1 expression was markedly increased. In vitro, TWIST1 inhibition prevented keloid derived fibroblasts proliferation, invasion and activation. Mechanistically, the observed effects were mediated, by TWIST1−dependent degradation of MEF2A (Myocyte Enhancer Factor 2A). TBR1 (TGF β Receptor 1) was identified as a novel downstream target of MEF2A, which directly binds to its promoter. Overexpression of TWIST1 promoted the recruitment of MEF2A to the TBR1 promoter and restored TBR1 functional expression.

CONCLUSION:

Our findings identify a key role for TWIST1 in the pathogenesis of keloid and in keloid derived fibroblasts that is partially mediated via increased MEF2A dependent TBR1 expression. Inhibition of TWIST1 in KFBs may constitute a new therapeutic strategy for the treatment of keloid.

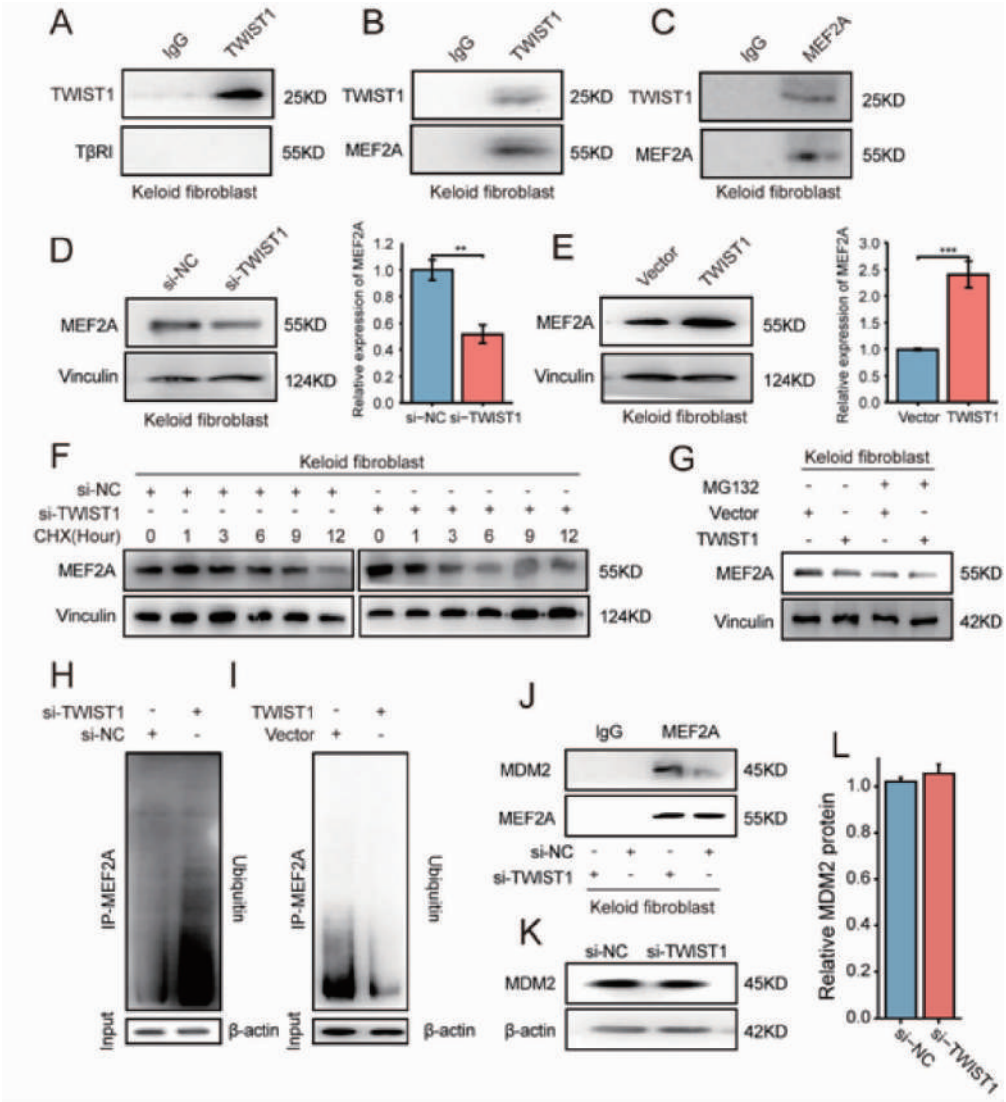

Figure 1:TWIST1 interacts with MEF2A and suppress ubiquitination and degradation of MEF2A. A, B. Cell lysates of keloid – derived fibroblasts were immunoprecipitated with anti –TWIST1 antibody, followed by immunoblotting with anti –TBRI or anti – MEF2A antibody, respectively; C. Cell lysates of keloid – derived fibroblasts were immunoprecipitated with anti–MEF2A antibody, followed by immunoblotting with anti– TWIST1 and anti –MEF2A antibody, Immunoglobulin G was used as a negative control; D. The protein expression of MEF2A in small interfering negative control and small interfering TWIST1 groups was detected by Western blot with densitometry analysis of Western blot results; E. The protein expression of MEF2A in vector and TWIST1 group was detected by Western blot with densitometry analysis of Western blot results; F. Western blot detected the alteration of MEF2A in small interfering

negative control and small interfering TWIST1 group with treatment of 10 μg/ml CHX for the indicated times; G. Keloid –derived fibroblasts transfected by Vector or TWIST1 virus were treated with 10 nmol/ml of MG132, and protein concentrations of MEF2A were determined; H, I. Lysates from keloid –derived fibroblasts modulated with down or up regulated TWIST1 were incubated with anti – MEF2A antibody. Immunoprecipitants were immunoblotted with an anti –Ub (ubiquitin) antibody; J. Coimmunoprecipitation of MDM2 and MEF2A in keloid –derived fibroblasts transfected with si –NC or si –TWIST1; K, L. Protein concentrations of MDM2 in keloid–derived fibroblasts transfected with si–NC or si–TWIST1 (*P<0.05, **P<0.01, ***P<0.001).

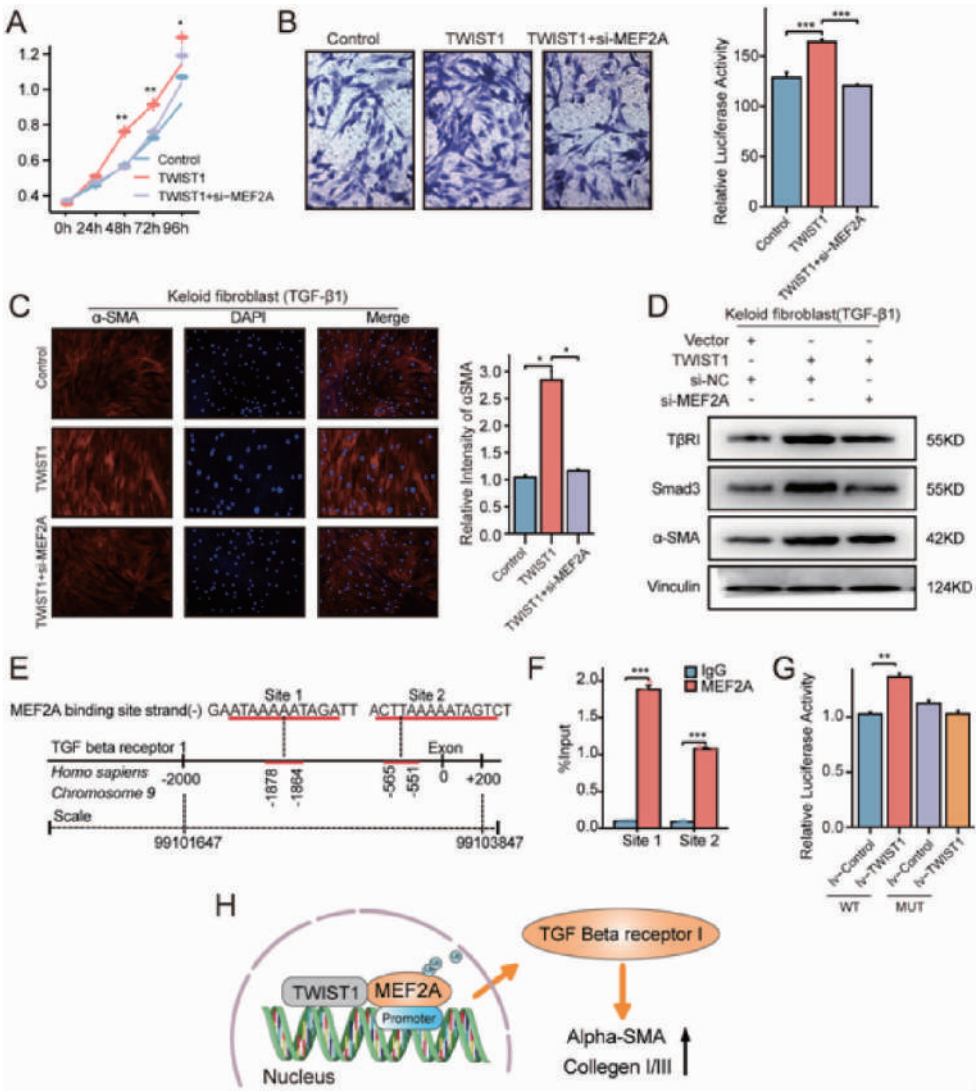

Figure 2: TWIST1 promoted fibrosis ability via MEF2A and TGF–β receptor I was a direct target of MEF2A in keloid derived fibroblasts. A. Keloid–derived fibroblasts transfected by Control or TWIST1 or TWIST1 with si–MEF2A were measured proliferation ability using growth curve; B. Cell invasion determined by transwell in pretreated keloid–derived fibroblasts; C. keloid–derived fibroblasts transfected by Control or TWIST1 or TWIST1 with si–MEF2A pretreated 5ng/ml TGF–β1 were measured by α–SMA using immunofluorescence assay; D. Protein levels of TBRI, Smad3 and α–SMA were detected by Western blotting; E. Prediction of MEF2A–binding sites in the TBR1 promoter region using JASPAR software; F.ChIP–qPCR assay showed the binding capability of MEF2A with different predicted binding sites; G. Dual–luciferase reporter assay indicated the promoter activity of MEF2A encoding wild–type (WT) or mutant MEF2A–binding site (MUT);H. The schematic representation of TWIST1 promoting expression of TGF–β receptor I by regulating the stability of MEF2A (*P<0.05, **P<0.01, ***P<0.001).

Integrated Analysis of TWIST1 in Keloid Pathogenesis: Single-Cell Transcriptomics Reveals Fibroblast Heterogeneity and a Novel MEF2A-TBR1 Regulatory Axis

Tianhao Li, Mingzi Zhang, Yunzhu Li, Yixin Sun, Jiuzuo Huang, Ang Zeng, Nanze Yu, Xiao Long

Department of Plastic and Aesthetic Surgery, Peking Union Medical College Hospital

BACKGROUND:

Keloid scarring is caused by a fibroproliferative disorder due to abnormal activation of genes, the underlying mechanism of which is still unclear. The basic helix−loop−helix transcription factor Twist−related protein 1 (TWIST1) controls cell proliferation and differentiation in tissue development and disease processes. In this study, we aimed to clarify the essential role of TWIST1 in the pathogenesis of keloids.

METHODS:

1.Single−Cell RNA Sequencing: Analyzed 28,064 cells from keloid and adjacent normal tissues to map cellular heterogeneity (n=4 patients).

2.Functional Assays:In vitro: CCK−8, Transwell, immunofluorescence, and Western blotting in KFBs treated with TWIST1 inhibitor harmine.Molecular Interactions: Co−immunoprecipitation (Co−IP), ubiquitination assays, chromatin immunoprecipitation (ChIP−qPCR), and dual−luciferase reporter assays to validate TWIST1−MEF2A−TBR1 interactions.

3.Pathway Analysis: Gene set enrichment (GSEA) for TGF−β, Eph−ephrin, and tumor−related pathways.

RESULTS:

1.Cellular Heterogeneity:

scRNA−seq identified expanded fibroblast (cluster c9) and VEC subpopulations (clusters c4, c5, c18) in keloids, linked to TGF−β and Eph−ephrin pathway activation.TWIST1 was significantly upregulated in KFBs and VECs ($p<0.01$).

2.TWIST1 Functional Roles:

Fibrosis: TWIST1 promoted collagen synthesis (COL1A1, COL3A1) and myofibroblast activation via TGF−β/Smad3. Harmine (TWIST1 inhibitor) suppressed TWIST1, reducing ECM deposition ($p<0.05$).

Angiogenesis: TWIST1 enhanced Eph−ephrin signaling (EFNB2−EPHA4) in VECs, driving pathological vascularization.

3.Mechanistic Insights:

TWIST1 stabilized MEF2A by inhibiting MDM2–mediated ubiquitination, prolonging its half –life.MEF2A directly bound the TBR1 promoter, enhancing TGF –β receptor expression (ChIP–qPCR fold enrichment=2.5, $p<0.001$).TWIST1 overexpression rescued TBR1 expression in KFBs, while MEF2A knockdown reversed this effect ($p<0.01$).

CONCLUSION:

Our research highlights a significant function of TWIST1 in the development of keloid and its related fibroblasts, partially facilitated by elevated MEF2A –dependent TBR1 expression. Blocking the expression of TWIST1 in KFBs could potentially pave a novel therapeutic avenue for keloid treatment.

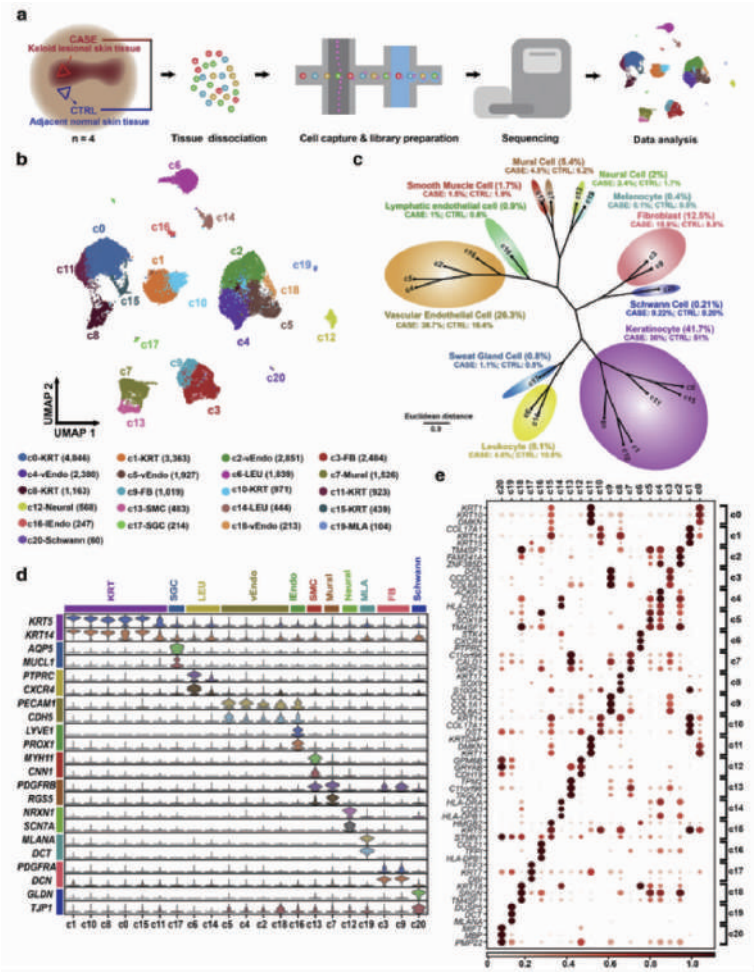

Fig.1Presents a comprehensive analysis of the cellular diversity and heterogeneity within keloid skin tissue through scRNA

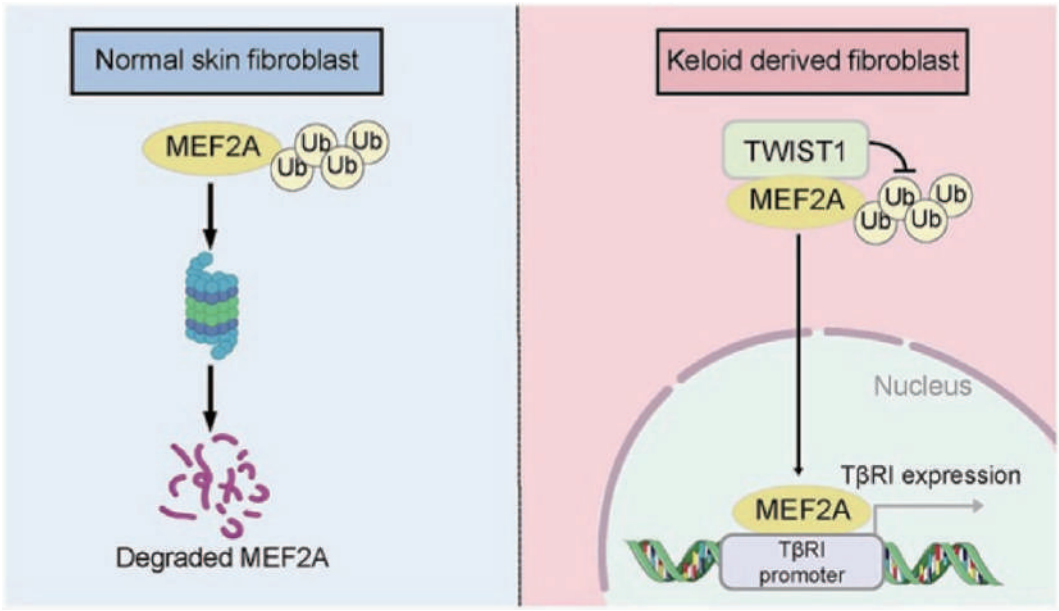

Fig.2 Schematic representation of TWIST1 promoting expression of TGF–β receptor 1 by regulating the stability of MEF2A

Multi-omics Analyses Reveal Bacteria and Catalase Associate with Keloid Disease

Mengjie Shan, PhD, Meng Xiao, Jiyu Xu, Wei Sun, Zerui Wang, Wenbin Du, Xiaoyu Liu, Yongsheng Huang, and Youbin Wang

Peking union medical college hospital

BACKGROUND:
The pathology of keloid and especially the roles of bacteria on it were not well understood.

METHODS:
In this study, multi-omics analyses including microbiome, metaproteomics, metabolomic and single-cell transcriptome were used to explore the roles of bacteria on keloid disease.

RESULTS:
We found that the types of bacteria are significantly different between keloid and healthy skin. The 16S rRNA sequencing and metaproteomics showed that more catalase (CAT) negative bacteria, Clostridium and Roseburia existed in keloid compared with the adjacent healthy skin. In addition, protein mass spectrometry shows that CAT is one of the differentially expressed proteins (DEPs). Overexpression of CAT inhibited the proliferation, migration and invasion of keloid fibroblasts, and these characteristics were opposite when CAT was knocked down. Furthermore, the cell-derived xenograft (CDX) model showed that Clostridium butyricum promote the growth of patient's keloid fibroblasts in vivo, while CAT positive bacteria Bacillus subtilis inhibited it. Single-cell RNA sequencing verified that oxidative stress was up-regulated and CAT was down-regulated in mesenchymal-like fibroblasts of keloid.

CONCLUSION:
In conclusion, our findings suggest that bacteria and CAT contribute to keloid disease.

Single-cell RNA Sequencing Revealed Key Factors of EMT to Promote Fibroblasts Activation and Immune Infiltration in Keloids

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Conflict of interest:

The authors have no financial or other conflicts of interest to disclose.

BACKGROUND:

Keloid is a fibrotic disorder of soft tissues secondary to abnormal wound healing. Previous studies have predominantly focused on fibroblasts, yet research on epidermal keratinocytes (KCs) remains limited. Existing evidence indicates that KCs in keloid differ from normal KCs in morphology and transcriptional profiles, with keloid KCs exhibiting EMT-like characteristics. However, the molecular drivers of EMT in keloid KCs and how EMT-primed KCs contribute to keloid pathogenesis and progression remain poorly understood.

METHODS:

We collected lesional tissues from 10 keloid patients and 5 normal skin samples for singlecell RNA sequencing and downstream bioinformatics analysis. In vitro molecular experiments (quantitative polymerase chain reaction [qPCR],western blotting [WB], enzyme-linked immunosorbent assay [ELISA]) and cellular assays (scratch assay, CCK8 proliferation assay, colony formation assay, immunocytochemistry) were performed. We established a bleomycin-induced skin fibrosis mice model and a keloid xenograft nude mice model. After administering the FOSL1 inhibitor SR11302 for two weeks, fibrosis-related indicators were assessed to evaluate the therapeutic efficacy of the drug.

RESULTS:

Single-cell sequencing revealed distinct keratinocyte compositions between keloid and normal skin. The proportions of basal-mig and spinous-mig keratinocyte subpopulations were significantly increased in keloid. Pathway enrichment analysis demonstrated that genes in these subpopulations were enriched in wound healing, epithelial-mesenchymal transition (EMT), and related pathways. Notably, basal-mig exhibited a pronounced EMT tendency. SCENIC analysis suggested that the transcription factor FOSL1 plays a critical role in the differentiation of basal-mig and spinous-mig subpopulations. In cell models, FOSL1 overexpression significantly enhanced the proliferation and migration of normal keratinocytes. ChIP-seq further revealed that FOSL1 binds to MED1 to form a super-enhancer complex, regulating downstream gene transcription. In keratinocytes, FOSL1 overexpression markedly increased the transcription and secretion of Matrix Metalloproteinase 3 (MMP3). Co-culture of FOSL1-overexpressing keratinocytes with normal and keloid fibroblasts enhanced fibroblast proliferation, migration, and inflammatory factors secretion, whereas these effects were significantly attenuated by MMP3 antibody treatment. In skin fibrosis mice models, FOSL1 inhibitor SR11302 can significantly alleviate fibrotic pathology.

CONCLUSION:

Keratinocytes overexpressing FOSL1 can upregulate the secretion of MMP3 and subsequently facilitate the activation of fibroblasts and immune cells infiltration, thereby promoting the initiation and progression of keloid.

Insights from Comparing Blood Perfusion Metrics and Vancouver Scar Scale in Keloid Assessment

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Background:

Accurate keloid assessment is crucial for effective treatment planning. The Vancouver Scar Scale (VSS) is widely used but relies on subjective observation, leading to variability in scoring. Laser Speckle Contrast Imaging (LSCI) has emerged as a promising technique for objective assessment of keloid vascularity by quantifying blood perfusion. This study investigates the correlation between LSCI-derived perfusion metrics and VSS scores to evaluate their clinical utility in keloid assessment and treatment monitoring.

Methods:

A retrospective study was conducted on 99 patients with 176 keloids. Blood perfusion ratios ($PU_{keloid/control}$) derived from LSCI were compared with pre- and post-treatment VSS scores. Correlation analysis and linear regression were applied to determine the relationship between perfusion metrics and VSS changes. Clinical cases were analyzed to validate the effectiveness of $PU_{keloid/control}$ in guiding treatment.

Results:

$PU_{keloid/control}$ demonstrated a significant correlation with total VSS scores (Spearman's ρ = 0.308, $p<0.001$) and its subcomponents, particularly vascularity (Spearman's ρ = 0.424, $p<0.001$). The strongest associations were observed between $PU_{keloid/control}$ and vascularity and pigmentation scores, emphasizing the role of perfusion in keloid severity. Linear regression analysis showed a strong association between changes in $PU_{keloid/control}$ and improvements in VSS scores ($R^2=0.539$, $p<0.01$), indicating that perfusion changes can reliably reflect therapeutic outcomes.

Further subgroup analysis revealed that $PU_{keloid/control}$ was superior to absolute PU_{keloid} values in evaluating both keloid lesions and post-surgical linear wounds. Patients who demonstrated a greater reduction in $PU_{keloid/control}$ post-treatment also exhibited more

substantial improvements in their VSS scores. Additionally, clinical cases demonstrated that high−perfusion areas identified via LSCI often corresponded with regions of persistent inflammation or poor therapeutic response, highlighting the potential role of targeted intervention strategies.

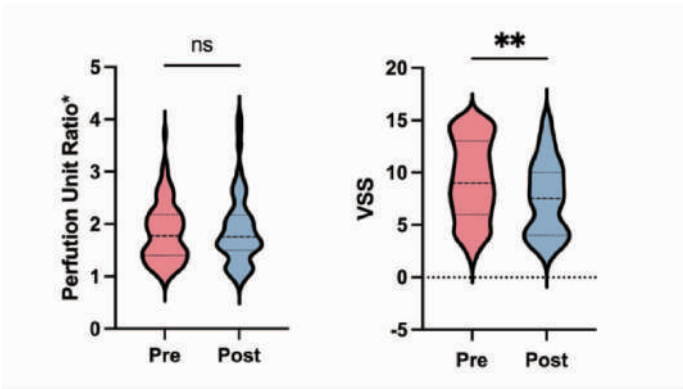

Figure 1. Comparison of pre– and post–treatment VSS scores and PUkeloid/control ratio.

Violin plots demonstrating significant improvements in VSS scores post−treatment, while no significant changes were observed in the PUkeloid/control ratio. *: The ratio of blood perfusion in the keloid region and adjacent normal skin.

Among the 44 patients (56 keloids) who underwent post−treatment follow−up, the most common therapeutic approaches included pulsed dye laser (PDL) combined with betamethasone and 5−fluorouracil (35.17%), and surgery with radiotherapy (19.64%). While overall VSS scores significantly improved after treatment (median: 9 [6 - 13] to 7.5 [4 - 10], $p = 0.005$), no significant change was observed in absolute PUkeloid/control values at the group level ($p = 0.942$). However, a strong correlation was noted between changes in perfusion ratio (ΔR) and changes in VSS scores (ΔVSS), suggesting that individualized perfusion changes may better reflect treatment outcomes.

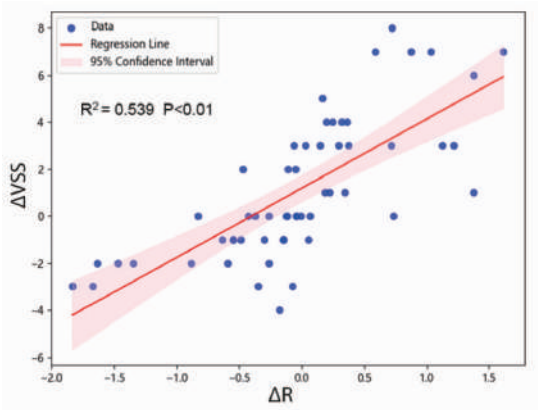

Figure 2. Correlation between ΔPUkeloid/control and ΔVSS

A scatterplot showing the linear regression analysis of the correlation between changes in PUkeloid/control ratio and VSS scores (ΔR and ΔVSS), with an R^2 value of 0.539. ΔVSS: Change in vancouver scar scale, ΔR: Change in perfusion ratio (PUkeloid/control).

Clinical cases further supported these findings. One patient exhibited a marked decrease in PU ratio following combination therapy, which corresponded with improved scar pliability and pigmentation. Conversely, another patient with persistently high PUkeloid/control despite treatment demonstrated poor clinical response, reinforcing the importance of vascular assessment in treatment planning.

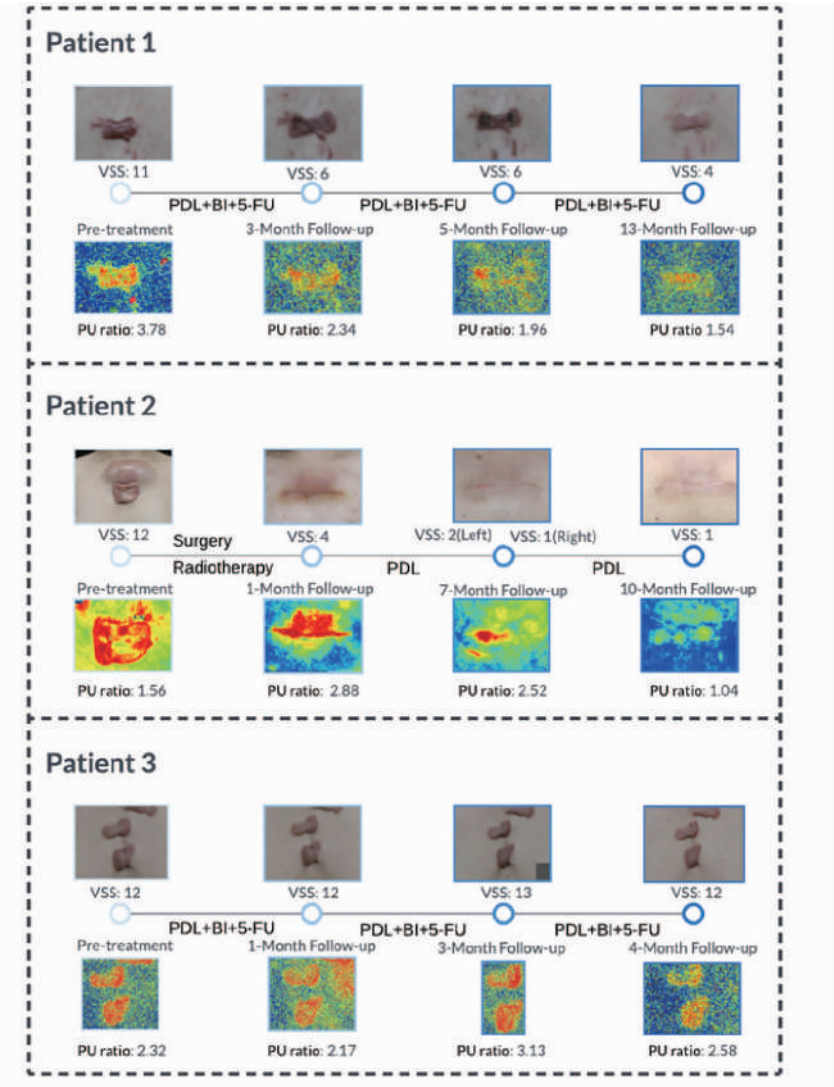

Figure 3. Clinical cases illustrating the utility of PUkeloid/control

Photographs and LSCI perfusion images from three patients, demonstrating the effectiveness of PUkeloid/control in monitoring keloid treatment outcomes. VSS: Vancouver Scar Scale, PU ratio: blood perfusion ratio (PUkeloid/control), PDL: Pulsed Dye Laser, BI: Betamethasone injection; 5−FU: 5−Fluorouracil.

Conclusion:

LSCI−derived PUkeloid/control provides an objective, non−invasive method for assessing keloid vascularity. This metric complements traditional tools like VSS, enhancing scar evaluation and treatment planning. Further studies are needed to optimize its clinical applications.

Identification of Original Research: YES
Identification of Clinical Trials: No
Funding Source: The work was supported by National High Level Hospital Clinical Research Funding (Grant nos. 2022 - PUMCH - B - 042, 2022 - PUMCH - B - 041, 2022 - PUMCH - A - 210 and 2022−PUMCH−C−025)
IRB/Ethics Committee Approval: The study was approved by the Ethics Review Committee of Peking Union Medical College Hospital (I−24PJ0121)
Disclosure Declaration: None

DAY 3: SUNDAY JUNE 8, 2025

SESSION 4 *15:30–17:10*

BASIC AND CLINICAL RESEARCH

Innovative Treatment Modalities for Keloids: Contact Cryotherapy

Michael H Tirgan MD

Keloid Research Foundation

Background:

Keloids represent a therapeutic challenge due to their high recurrence rates and resistance to conventional treatment. Surgical excision, often considered a first-line treatment, is associated with recurrence rates exceeding 50% even when combined with adjuvant therapies. Similarly, intralesional corticosteroid injections, another widely used modality, provide variable results, with recurrence rates ranging from 9% to 50% and frequent complications, including hypopigmentation, atrophy, and pain. These outcomes underscore the need for innovative and more effective treatments.

Methods:

Contact cryotherapy has emerged as promising yet underutilized alternatives in the management of keloids. Recent clinical studies have shown high response rates in appropriately selected patients, with significantly lower recurrence rates compared to traditional therapies, underscoring the potential of these treatments to redefine keloid management.

Results:

Contact cryotherapy results in cold-induced tissue destruction. Unlike traditional spray cryotherapy, contact cryotherapy achieves uniform tissue freezing while sparing surrounding healthy skin, leading to improved clinical outcomes. Cryotherapy not only reduces keloid volume effectively but also enhances symptom relief and prevents recurrence when used as a monotherapy or in combination with other modalities.

CONCLUSION:

This presentation will review the evidence supporting the efficacy and safety of these innovative treatment modalities, highlighting their potential to transform the current paradigm of keloid management. By leveraging the unique mechanisms and the precision of contact cryotherapy, clinicians can achieve superior outcomes, reduce recurrence rates, and improve the quality of life for patients with keloids.

Dual siRNAs Nanoplex Targeting IL-4RA and SPARC Enhance Collagen Reduction in IL-4 Activated Skin Fibroblasts

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BACKGROUND:

Currently, there is a need for more effective preventive methods or treatment for hypertrophic scars or keloids. To target important proteins involved in the T helper 2 inflammatory microenvironment and ECM production during scar formation, we designed dual siRNAs targeting interleukin-4 Receptor α (IL-4Rα) and secreted protein acidic and cysteine rich (SPARC) as a novel preventive solution and treatment to reduce pathological scars. In this study, the effect of dual siRNAs (siIL-4Rα and siSPARC) nanoplex in reducing collagen expression for interleukin-4 stimulated human dermal fibroblasts (HDFs) was investigated.

METHODS:

As unmodified siRNA is easily degraded by endogenous enzymes, we encapsulated the siRNAs using positively charged gelatin-tyramine (Gtn-Tyr) to form a nanoplex to achieve protection and targeted delivery to dermal fibroblast, studied gene knockdown, decrease in collagen production, and compared internalization of the siRNA-nanoplex by dermal fibroblasts with keratinocytes. Simultaneously, we tested the effect of siRNA nanoplex on cell cytotoxicity and proliferation.

Separately, three individuals with hypertrophic scars and keloid volunteered to test the dual siRNA-nanoplex delivered via microneedles for treatment and their scars were assessed at different timepoints.

RESULTS:

The results demonstrated that siRNA-nanoplex preferentially enhances fibroblast uptake in comparison to naked siRNA, and when compared with keratinocytes. Majority of the siRNA-nanoplex is also not present within lysosomal associated membrane protein-1

(LAMP −1) −positive late endosomes for degradation even after 24h of treatment. Simultaneously, the dual siRNAs nanoplex of siIL −4Rα and siSPARC could inhibit collagen production of HDFs in the presence of IL −4, and dual siRNAs nanoplex treatment does not cause any cytotoxic effect on cells or inhibit cell proliferation.

Volunteers that tested the microneedle delivered dual siRNA−nanoplex had reduction of scar volume together with reduction of pain or itch, with no adverse effects reported.

CONCLUSION:

Overall, targeting both IL−4Rα and SPARC using the siRNA nanoplex demonstrates a promising solution for preventing and treating hypertrophic scars or keloids.

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CATEGORY:

Original Research

FUNDING SOURCE:

Government agency

Tissue Chondrification and Ossification in Keloids with Primary Report of Five Cases

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Dermatology Department of Shuguang Hospital affiliated to Shanghai Traditional Chinese Medicine University
Plastic Surgery Department, Shanghai Ninth People's Hospital, Shanghai Jiao Tong University School of Medicine

Introduction:
Keloid is commonly regarded as a benign skin tumour. Some keloids clinically exhibit hard tissue texture similar to that of cartilage or bone. We hypothesized that the keloid pathological niche environment is likely to induce keloid MSCs towards chondrogenic and osteogenic differentiation and leads to cartilage or bone-like tissue formation.

Method:
The differences in tissue ossification, histology, mechanical properties, abnormal extracellular matrices and chondrogenic/ osteogenic gene expression among sclerous keloids (SKs), regular keloids (RKs) and normal skins(NKs) were carefully examined.

Results:
The sporadic ossified islets existed in SK group whereas no ossified/chondrified islet was found in other groups by micro-CT reconstruction. H&E, Masson trichrome and safranin O staining revealed lacuna-like structures in SKs, which were featured as bone/cartilage histology. Immunohistochemical staining showed over production of osteoprotegerin, type I and III collagen in SK group but similar production level of aggrecan among three groups. The biomechanical analysis demonstrated the weakest compliance of SK tissues. In addition, SK fibroblasts exhibited a relatively slower proliferation rate but higher expression levels of osteogenic and chondrogenic genes among all three groups. These cell populations also showed the strongest potential for lineage transformation.

Conclusion:
we first reported the presence of ossified and chondrified matrices in some extremely hard keloids in the present study.

Cartesian Model of Clinical Behavior of Keloid Disorder, Implementation and Update of the Keloid Staging System and Call for Establishment of International Keloid Registry

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Conflict of Interest:None
Funding: None
IRB Assessment: Determined by central IRB to be exempt and not requiring IRB approval.
Keywords: Keloid, Keloid Biology, Keloid Registry, Keloid Growth

ABSTRACT

Background: Keloid disorder (KD) has highly variable clinical behavior. KD ranges from patients who in their lifetime develop one or a few keloid lesions to those who within a few years develop very extensive disease involving various parts of their skin. To the author's knowledge, this very important aspect of the disorder has never been studied and thus never incorporated into an analysis of clinical or laboratory data from keloid patients.

Objective:

To assess clinical behavior and rate of progression of keloid disorder through an analysis of data from two large cohorts of keloid patients.

Material and Methods:

This is a retrospective analysis of two datasets. The first dataset was obtained from 971 consecutive patients seen by the author in his keloid specialty practice. Medical records and photographs of the lesions were analyzed.

The second dataset was obtained from an ongoing online keloid survey that the author launched in November 2011.Survey participants were asked to provide answers to numerous questions about their keloids, including an assessment of the growth rate of their keloids over time. The underlying studies for both datasets were approved by the Institutional Review Board (IRB). Descriptive statistics are provided. Patterns of clinical progression of KD are plotted in Cartesian tables.

Results:

A review of the clinical patterns of presentation and KD's duration allows for plotting the patients' results into Cartesian tables and therefore clustering patients with similar patterns of disease progression.Seventy−eight patients (8.02%) were found to have stage I KD beyond 15 years since the onset of their illness. On the other hand, 52 patients (5.35%) developed stage II disease,15 patients (1.54%) developed stage III disease and four patients (0.41%) developed stage IV disease within the first three years of the onset of their illness.

Approximately, one third of the survey patients reported having stable disease and no progression of their KD at one−, two−, five− and ten−year timepoints. About 10% of patients reported a 50% increase, and approximately 6% reported doubling of the size of their keloids at one−, two−, five− and ten−year timepoints.

Conclusions and Relevance:

Proper comparison of clinical and/or laboratory outcome data is meaningful only when such a comparison is made among patients with similar biology and clinical behavior. The author recommends that keloid researchers incorporatethe variable patterns of clinical behavior of KD in planning clinical or laboratory experiments, as well as in analysis of their data. Call is made for all keloid researchers to collaborate in establishing an international Keloid Registry.

A Study on the Multidimensional Effects of Hydrogen-Rich Materials in Wound Healing and the Alleviation of Skin Fibrosis

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Background:

Wound healing disorders and skin fibrotic lesions (e.g., keloids) are complex clinical challenges often leading to functional impairments. Hydrogen-rich materials, known for their antioxidant, anti-inflammatory, and metabolic regulatory properties, have emerged as promising therapeutic agents. This study evaluates the effects and molecular mechanisms of hydrogen-rich saline (HRS) and hydrogen-rich water (HRW) in wound repair and scar management.

Methods:

Animal Model: A full-thickness skin defect model was created in rabbits, divided into five groups: control, saline, HRS, VSD+saline, and VSD+HRS. Wound healing was assessed via closure rate and histopathological evaluation.

Cell Experiment: Human keratinocyte HaCaT cells were used in vitro to evaluate cell viability.

Metabolomics Analysis: Wound tissue samples underwent untargeted metabolomics analysis to identify differential metabolites and oxidative stress markers.

Clinical Trial: A double-blind randomized controlled trial involved 21 keloid patients, randomized into HRW (11 patients) and control groups (10 patients). Cytokine expression levels and keloid appearance were assessed using Western blotting, qPCR, ELISA, and the Vancouver Scar Scale (VSS).

Ethical Approval: The animal study protocol was approved by the Ethics Committee of Peking Union Medical College Hospital (Approval No.: XHDW-2023-063), and the clinical study passed ethical review (Review No.: ZS-3373). All participants provided informed consent.

Results:

Animal Experiment: The VSD+HRS group showed significantly higher wound healing rates and shorter closure time ($p < 0.05$). HE staining revealed superior epidermal

regeneration and granulation tissue formation.
Cell Viability: The VSD+HRS group exhibited significantly higher HaCaT cell viability ($p<0.05$).
Metabolomics Analysis: Forty-five differential metabolites were identified, with biotin metabolism pathways emerging as potential targets.
Oxidative Stress: VSD+HRS treatment reduced local oxidative stress in wound tissues.
Clinical Trial: HRW significantly reduced postoperative pain and pruritus frequency ($p<0.05$) and improved VSS and pigmentation scores ($p<0.05$).
Molecular Mechanisms: HRWT downregulated TRPV1 and HIF-1α expression ($p<0.05$) and upregulated anti-inflammatory IL-10 ($p=0.003$), while pro-inflammatory factors IL-6, TGF-β, and VEGF decreased ($p=0.030$, $p=0.002$, and $p=0.063$).

Conclusion:

Hydrogen-rich materials show therapeutic potential in wound healing and keloid management. HRS combined with VSD accelerates wound closure, while HRW therapy alleviates pruritus and promotes inflammation resolution. These findings provide scientific support for the application of hydrogen-rich materials in wound management and skin fibrosis treatment, though further optimization and mechanistic validation are needed.

Establishment of a Deep Learning-Based Automated Segmentation and Blood Perfusion Prediction System for Keloids**

Shuo Li, Mengdi Zhang, Binghan Wang

Department of Plastic Surgery, Peking Union Medical College Hospital, Chinese Academy of Medical Sciences and Peking Union Medical College, Beijing, 100730, People's Republic of China

BACKGROUND:

Keloids are pathological scars commonly arising after skin trauma, mediated by various pathological mechanisms. They exhibit dynamic changes, high internal heterogeneity, and biological characteristics similar to malignant tumors, with high recurrence rates and prolonged treatment cycles. Blood perfusion maps based on Laser Speckle Contrast Imaging (LSCI) can effectively assess the blood perfusion and growth status of keloids. However, conventional LSCI-based blood flow measurement processes are time-consuming and labor-intensive. Therefore, this study aims to develop a deep learning-based automated segmentation and blood perfusion prediction system for keloids, enabling remote, efficient, and objective evaluation of their blood perfusion and growth status.

METHODS:

This study retrospectively and prospectively included 3,000 patients, collecting macroscopic photographs of keloids and LSCI-measured blood perfusion maps. Using deep learning, macroscopic photographs were utilized as input to construct an automated keloid segmentation model and an image-based blood perfusion prediction model. During model development, robustness was validated by altering imaging conditions (e.g., shooting angles) or adding noise to images (e.g., brightness, contrast, and blur adjustments). In the prospective cohort, the constructed models were used to evaluate blood perfusion values before and after keloid treatment, and the results were compared with measured values to verify the accuracy of the models in real-world follow-up scenarios.

RESULTS:

In the retrospective study, the automated segmentation model achieved a correlation of 0.96 with measured values in identifying keloid area size. The image-based blood perfusion prediction model demonstrated a correlation of 0.75 between predicted and measured values. The blood perfusion prediction model exhibited superior robustness against noise interference compared to traditional models. In the prospective cohort study,

the blood perfusion prediction model achieved correlations of 0.64 and 0.76 with measured values before and after treatment, respectively.

CONCLUSIONS:

By integrating multi-stage deep learning models, this study established an automated segmentation and blood perfusion prediction system for keloids based on macroscopic photographs. The system demonstrated high segmentation accuracy and blood perfusion prediction performance, enabling objective, remote, and precise assessment of keloid growth status and severity. It facilitates early identification of poor prognosis cases, personalized follow-up monitoring, and guidance for secondary intervention timing. This approach significantly enhances clinical diagnosis and treatment outcomes, providing robust support for refined and personalized management of keloid patients.

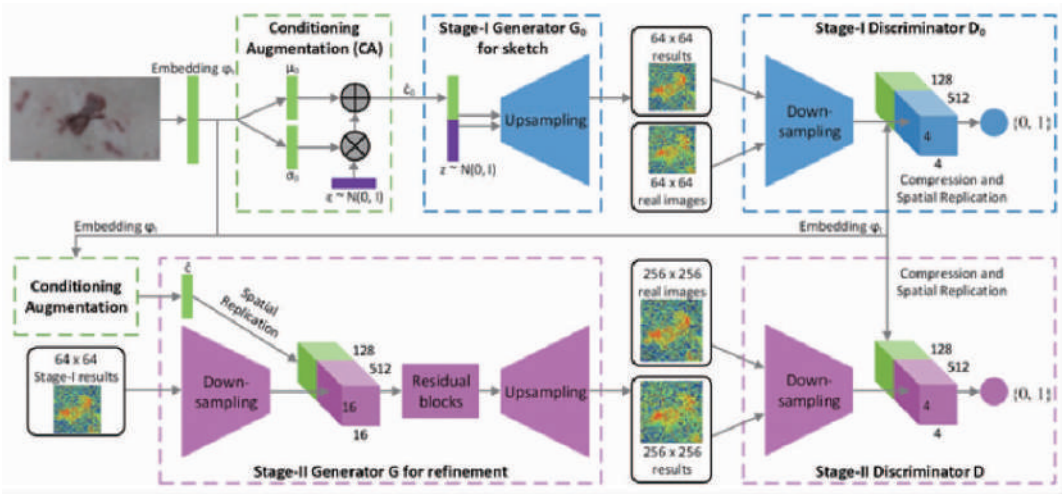

Figure: Generative Adversarial Network (GAN) Architecture Diagram

Unraveling the Vascular Tapestry: Endothelial Dysfunction in Keloid Formation and Therapeutic Insights

Zhijin Li[1], Junxian Wen[1,2], Yingrou Tan[2,3], Hong Liang Tey[2,4], Nanze Yu[1,5], Xiao Long[1], Xiaojun Wang[1]

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BACKGROUND:

For keloids, although fibroblast hyperproliferation and excessive extracellular matrix deposition have been extensively studied, less attention has been paid to the role of vascular dysregulation and endothelial dysfunction (ED) in keloid pathogenesis. Emerging evidence highlights abnormal angiogenesis, vascular irregularities, and endothelial injury as critical drivers of fibrosis in keloids.

METHODS:

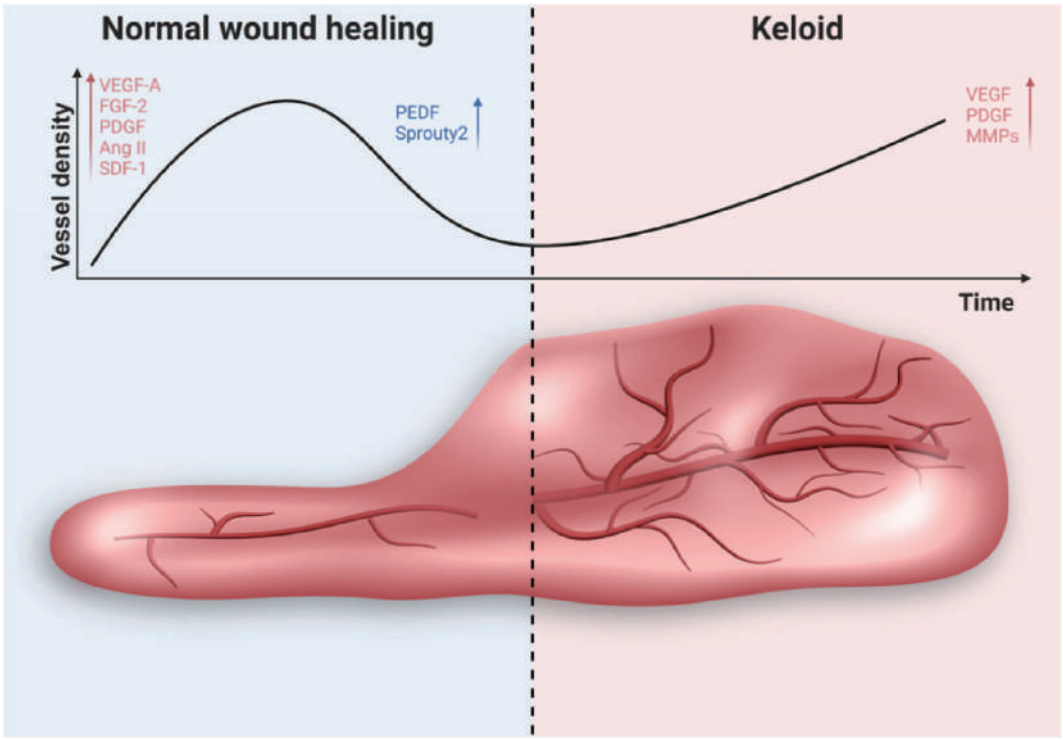
Four databases, including Medline, Embase, Web of Science, and the Cochrane Library, were searched from their inception dates to November 2024. The search was restricted to English −language publications to maintain consistency and focus. The search strategy included terms such as "Keloid", "Blood Vessels", "Microcirculation", and "Endothelium" to identify studies exploring vascular and endothelial mechanisms in keloid pathogenesis and treatment. Additionally, references cited in the selected articles were screened for further studies of interest to ensure comprehensive coverage.

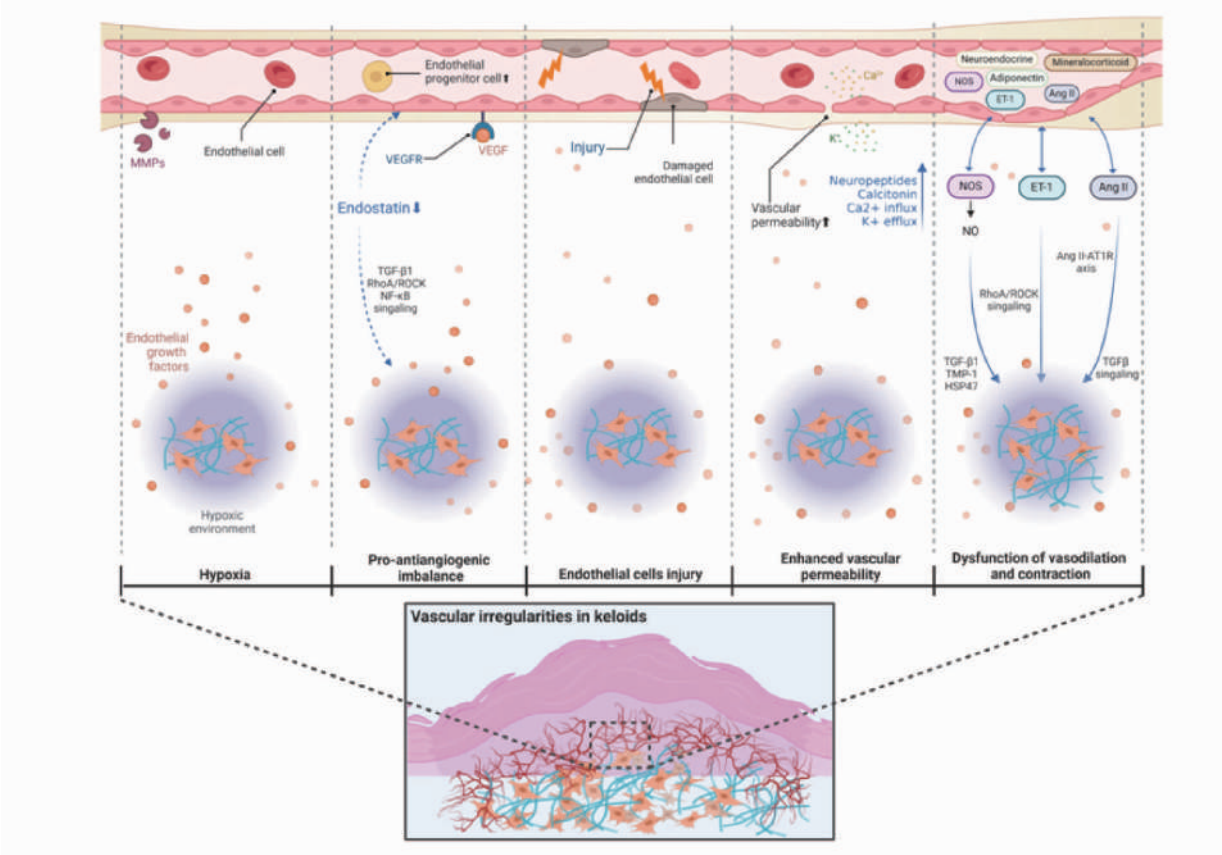
RESULTS:

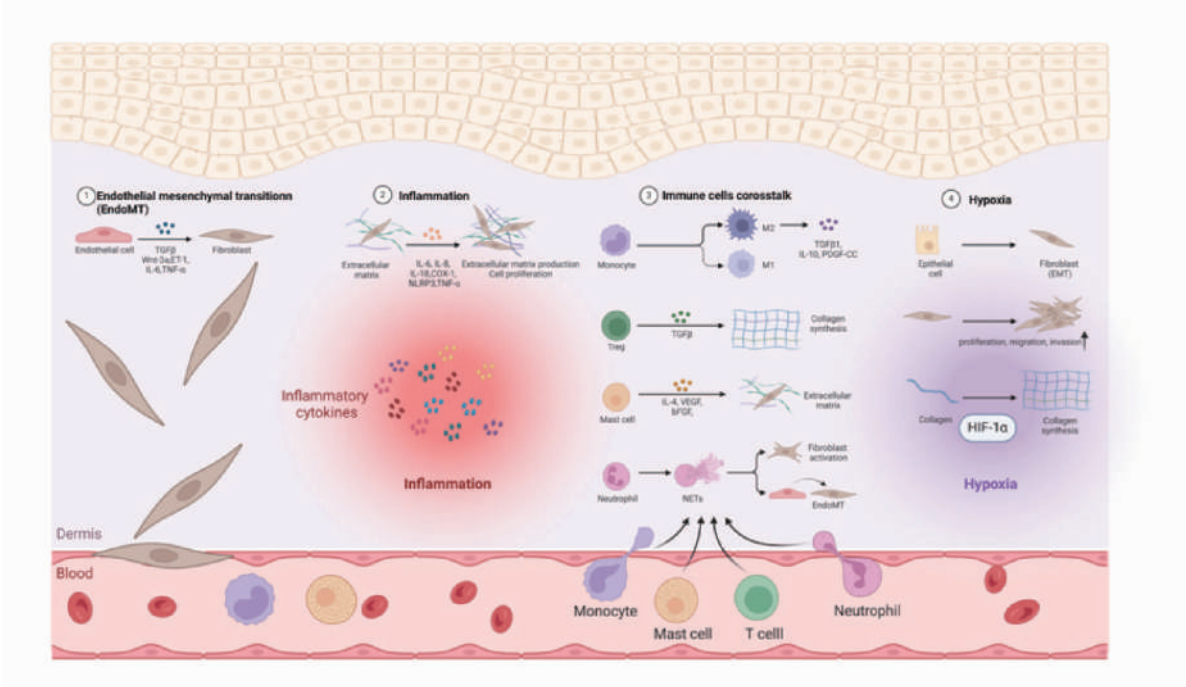
This review explores the direct and indirect mechanisms of ED in keloid progression, including endothelial−to−mesenchymal transition (EndoMT), inflammation, immune cell crosstalk, and hypoxia. Additionally, various treatment strategies targeting angiogenesis and ED, such as drugs, radiotherapy, hyperbaric oxygen therapy, compression, and laser treatments, are comprehensively reviewed. This review explores keloids through the lens of vasculature and endothelium, emphasizing the critical roles of vascular dysregulation and endothelial dysfunction. It aims to provide insights into the mechanisms of keloid formation and serve as a reference for developing future therapeutic strategies.

CONCLUSION:

Keloids, which are characterized by vascular irregularities and increased blood vessel density, exhibit imbalances in proangiogenic and antiangiogenic factors. These deviations in vascular architecture and the increase in blood vessel density contribute to a hypoxic microenvironment the mediates angiogenesis and collagen synthesis.Endothelial dysfunction in keloids involves a complex interplay between imbalanced pro −/ antiangiogenic factors and endothelial dynamics. ED directly impacts endothelial −to − mesenchymal transition and indirectly supports inflammation and immune cell crosstalk, thereby promoting fibrosis in keloids. Therapeutic interventions targeting ED include anti −VEGF drugs, radiotherapy, hyperbaric oxygen therapy, compression therapy, and laser treatments. In the future, continued exploration of the specific mechanisms underlying vascular endothelial dysfunction and the targeted development of effective keloid treatment methods will be crucial.







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