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Epithelial-mesenchymal transition in keloid tissue

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Fibroblasts at the wound site are recognized as the primary drivers of scar formation. They differentiate into myofibroblasts, the key mediators of fibrosis, which are responsible for collagen deposition and wound contraction. Repair processes cease when epithelialization is completed in normal wounds, whereas in keloid wounds, they may continue and result in excessive accumulation of unorganized extracellular matrix, forming problematic scars.

In addition to resident mesenchymal cells, fibroblasts and myofibroblasts are thought to be derived from multiple sources, including epithelial-mesenchymal transition (EMT) [1]. During this process, epithelial cells experience intercellular and intracellular changes, including dissociation of junctional complexes, loss of apical-basolateral polarity, and repression of epithelial markers. As a

result, epithelial cells lose many of their properties and take on characteristics of mesenchymal cells.

To investigate this process, an immunofluorescence assay using both epithelial and mesenchymal markers was performed using keloid tissues obtained from the anterior chest wall of a 70-year-old male patient (Fig. 1). After fixation, cells were incubated with primary antibodies against E-cadherin and vimentin (Abcam, Cambridge, UK), and subsequently labelled with fluorescently-tagged secondary antibodies. Counterstaining with 4',6-diamidino-2-phenylindole (DAPI) (VECTOR Laboratories, Burlingame, CA, USA) was also performed.

Cells were visualized using an LSM 700 Carl Zeiss confocal microscope (Carl Zeiss MicroImaging, Thornwood, NY, USA). E-cadherin and vimentin expression were simultaneously observed at the dermo-epidermal junction in keloid tissue, indicating the occurrence of EMT (Fig. 2).

Although further investigations with additional keloid specimens are needed, we hope that our study yields some insight into the mechanisms of keloid formation.

Notes**Conflict of interest**

No potential conflict of interest relevant to this article was reported.

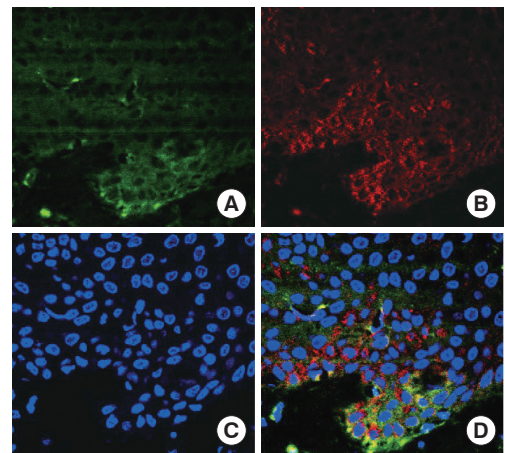


Fig. 2. Epithelial-mesenchymal transition (EMT) characteristics in keloid tissue. (A) Expression of the epithelial marker E-cadherin (green) and (B) the mesenchymal marker vimentin (red) was observed via immunofluorescence. (C) DAPI counterstain of nuclei (blue). (D) Co-expression of E-cadherin and vimentin at the dermo-epidermal junction was observed, indicating the occurrence of the EMT process (A-D, magnification $\times 630$).



Fig. 1. Patient with a keloid. A 70-year-old male with a keloid on the post-sternotomy area suffered from itchiness, pain, and skin tightening. The keloid was surgically excised intralesionally.

Ethical approval

The study was approved by the Institutional Review Board of Yonsei University Medical Center (IRB No. 4-2017-0259) and performed in accordance with the principles of the Declaration of Helsinki. Written informed consent was obtained.

Patient consent

The patient provided written informed consent for the publication and the use of his image.

Reference

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Versatility of the reverse sural fasciocutaneous flap for the reconstruction of lower leg defects caused by chronic osteomyelitis

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Recently, many studies have shown no difference in efficacy between musculocutaneous and fasciocutaneous flaps in the treatment of osteomyelitis [1]. The aim of this study was to examine the efficacy of the reverse sural fasciocutaneous flap for the reconstruction of chronic osteomyelitis defects on the distal lower leg. Between March 2013 and March 2018, five adult patients aged 38 to 85 years who underwent reconstruction with a reverse sural fasciocutaneous flap were included in this study (Table 1). These patients were diagnosed with chronic osteomyelitis at the Department of Orthopedic Surgery of Myongji Hospital and were referred to the Department of Plastic and Reconstructive Surgery for reconstruction of the soft tissue defects. Delayed distally-based fasciocutaneous reverse sural flaps were used in a 2-step procedure [2]. The patients were followed in our outpatient clinic and their healing status was quantitatively compared with previous findings by 3-phase bone scans, which all patients agreed to have performed for postoperative follow-up. Four of the five patients recovered progressively from osteomyelitis without complications, such as necrosis of the distal aspect of the flap or marginal dehiscence. These patients showed clinical resolution at the time of the last

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Table 1. Demographics and clinical characteristics of the patients

Patient no.	Age (yr)/sex	Diagnosis	Risk factors	Delay period (day)	Size of the flap (cm ²)	Complications	Duration of follow-up (mo)
1	68/male	Chronic osteomyelitis on the lateral malleolus caused by abscess formation	None	11	9 × 3	None	6
2	38/male	Chronic osteomyelitis on the calcaneus caused by surgical site infection	Smoker	10	14.5 × 4	None	7
3	62/male	Chronic osteomyelitis on the first metatarsal bone caused by diabetic foot	Diabetes mellitus, smoker	14	5 × 4	None	18
4	85/male	Chronic osteomyelitis on the tibia caused by an open tibiofibular fracture	Old cerebral infarction, peripheral arterial occlusive disease	15	12.5 × 5	None	6
5	65/male	Chronic osteomyelitis on the calcaneus caused by diabetic foot	Diabetes mellitus	14	15 × 6.5	Bone necrosis	12