Distribution of myofibroblasts in stage II and IV pressure ulcers

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Distribution of myofibroblasts in stage II and IV pressure ulcers

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ABSTRACT

Aim : In this study, we examined the distribution of myofibroblasts (MF) in stage II and IV pressure ulcers, using histological and immunohistological methods, and also discuss the relationship between the distribution of their depths and wound contraction. Method : Eight pressure ulcers obtained from cadavers were investigated. According to the NPUAP classification, three subjects were Stage II, and five subjects were Stage IV with undermining. Paraffin sections of pressure ulcers were made, stained and examined by light microscopy. Anti α-smooth muscle actin antibody was also used to specifically identify MF. The specimens of stage IV ulcers were cut in the same direction to observe the wound roof, transition area and wound bed in one section. All subjects gave their informed consent to have their skin tissue examined after their death. Results : Partial-thickness wound -Stage II : No granulation tissue was present. No MF were observed. Full-thickness wound-Stage IV with undermining : Numerous MF were distributed in the connective tissue of the wound roof and at the transition area, and along the granulation tissue in the wound bed. As a result, the distribution of MF in pressure ulcers with undermining showed a belt and J-shaped form from the wound roof to bed. Summary : No MF in partial-thickness wounds were observed. These findings suggest that partial-thickness wounds heal not by wound contraction but instead by epithelialization. The distribution of MF in a J-shaped form seems to allow for the effective closure of the undermining, because the contraction of a belt of MF in the roof and wound bed pulls the transition part from the roof to the wound bed toward the center of the ulcer.

KEY WORDS

myofibroblast, pressure ulcer, undermining, α-smooth muscle actin, contraction

Introduction

The healing process of skin wounds entails the distinct but temporally overlapping processes of inflammation, re-epithelialization, granulation tissue formation, and tissue remodeling[1]. Partial-thickness wounds which lack an entire epithelium, but possess a partial dermis without containing any adnexal structures tend to rapidly heal by re-epithelialization with either minimal or no scaring. On the other hand, full-thickness wounds, which are deeper than the dermis and also include adnexal structures, do not heal completely by re-epithelialization and thus they need the formation of granulation tissue and wound contraction associated with a reduction in the wound area. Since such wound contraction causes about a 40% decrease in the full thickness wound area[2], while also shorting the time required for re-epithleialization, wound contraction is therefore considered to play a critical role in the healing of such wounds.

Myofibroblasts (MF) in granulation tissue are known to be related with wound contraction[3-5]. Due to mechanical stress and inflammation, fibroblasts

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differentiate into proto-myofibroblasts and then transforming growth factor $\beta 1$ (TGF-$\beta 1$), which is produced by inflammatory cells, promotes the modulation of proto-myofibroblasts into differentiated MF. Finally, MF begins to disappear after epithelialization. Although MF looks similar to fibroblasts under light microscopy, some special structures of MF can be observed by transmission electron microscopy; such as bundles of microfilaments with dense bodies in between them, gap junctions between MF, supermature focal adhesions in vitro[6] and a fibronexus in vivo[7] which are all specialized contacts with the extracellular matrix, and moreover, stress fibers[3] and α-smooth muscle actin (α-SMA)[8] appear in MF. Although α-SMA is present in vascular smooth muscle cells, the presence of α-SMA represents the most reliable marker of the myofibroblastic phenotype[9]. The contractive activity of MF has been shown both in vivo[10] and in vitro[11] and the orientation of MF varies in the different layers of granulation tissue; including alterative, exudative, exudativo-productive, and cicatrizing layers from surface to deep[12]. In the exudative layer, the long axis of MF is perpendicular to the surface of wound, whereas in the exudativo-productive layer and cicatrizing layer, the long axis is parallel to the surface. This indicates that the changes in the orientation of MF to serves transmit contractile force in order to effect wound closure.

Pressure ulcers are a type of wound that frequently occur in elderly 65 years of age and older[13, 14]. The treatment of such wounds is a serious issue regarding the medical care of the elderly. Pressure ulcers are areas of local tissue injury, which develop where soft tissues are compressed between bony prominences and any external surface for a long time[15]. Moreover, some factors that contribute to the development of pressure ulcers are shear force, friction, moisture, and poor nutrition. Pressure ulcers are usually clarified into four stages according to the NPUAP classification[16], from stages I－IV. Stage I pressure ulcers appear as a defined area of persistent redness, stage II pressure ulcers demonstrate partial-thickness skin loss involving the epidermis or dermis, or both, stage III pressure ulcers show full-thickness skin loss involving the damage or necrosis of subcutaneous tissue, and stage IV pressure ulcers demonstrate full-thickness skin loss with extensive destruction, tissue necrosis, or damage to the muscle bone or supporting structures. Moreover, stage III or IV pressure ulcers usually possess a cave or undermining of the wound edges which is created by the excavation of the subcutaneous or muscle tissue.

The length of time that pressure ulcers require to heal depends on the stage of the pressure ulcers; approximately 7-16.7 days for stage I, approximately 19.8-28.1 days for stage II, approximately 101 days for stage III or IV without undermining, and approximately 236.5 days for stage III or IV with undermining[17－19]. Konya et al[19]. reported that 46.7% of Stage III and IV pressure ulcers had wound contraction and at the time that wound closure granulation tissue increased from the roof of undermining and wound contraction occurred. Although there have been some reports in which pressure ulcers were histologically studied[20, 21], there have been few studies describing on MF in pressure ulcers, and Mori et al[22]. clarified that α-SMA expressing MF were observed in the wound bed, granulation tissue and the subsequent fibrous layers of stage III and IV pressure ulcers and also reported MF to be involved in the contraction and healing of pressure ulcers. However, the subjects studied by Mori et al[22]. mostly showed a wound bed and wound roof in stage III and IV pressure ulcers with undermining. As a result, it is unclear whether the distribution of MF continues from the wound roof to wound bed or not. The aim of this study was therefore to examine the distribution of MF in stage II and IV pressure ulcers. We have tried to clarify the distribution of MF from the wound roof to the wound bed while also noting that no MF were present in stage II pressure ulcers.

Materials and Methods

1. Materials

Tissue specimens of pressure ulcers were obtained from eight cadavers undergoing autopsy and anatomical practice from 2000 to 2004. Eight tissue specimens of pressure ulcers were used; three tissues specimens of stage II, and five tissue specimens of stage IV with undermining. The specimens of stage IV comprised only a small part of ulcers including the undermining; the wound roof, transitional area and

wound bed, and measured 2.5 cm in maximum length and 1.5 cm in maximum width. One of the three stage II pressure ulcers was observed in the trochanter region, while the others were found in the sacral regions. Unfortunately, we could not obtain any tissue specimens of stage III pressure ulcers. As a result, we must evaluate stage III pressure ulcers in a future study.

2. Methods

The obtained tissue specimens were fixed with 10% formalin for 12 hours, then paraffin sections, 5 μm in thickness, were made using the normal method. The specimens of stage IV pressure ulcers with undermining were cut in the same direction as shown in Figure 2a for the observations of the wound roof, transition area and wound bed in one section. Then they were stained with Hematoxylin-Eosin (H-E). Moreover, for the immunohistological staining of MF, 5 μm paraffin sections, from which the paraffin was removed, were then washed with 0.01 M phosphate buffer saline (pH 7.4)(PBS), blocked with internal peroxidase using 0.03% H_2O_2, washed with PBS, stained with anti-smooth muscle actin/HRP (DACO Japan) for 60 min at room temperature, washed with the same PBS, stained with DAB, washed with PBS, and then were nuclear-stained with hematoxylin. For the control, N-universal negative control-mouse (DACO Japan) was used. We observed the specimens under light microscopy.

All subjects and their families gave their informed consent to have their bodies and skin tissue specimens examined after their death.

Results

1. Partial-thickness Stage II wound

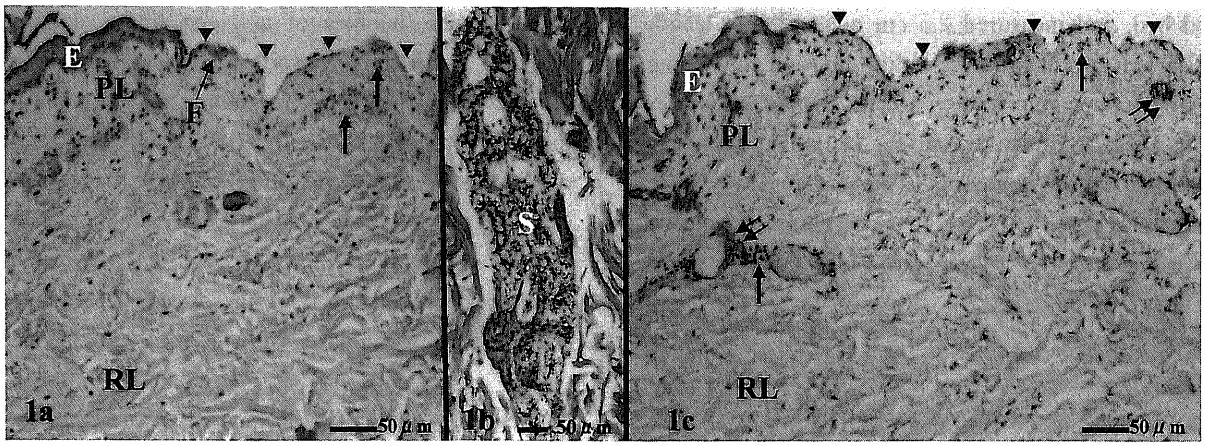
The epidermis was removed from the dermis and the superficial dermis, from the papillary layer to a part of the reticular layer, was covered with or without fibrinoid tissue (Fig. 1a). The superficial infiltration of lymphocytes and macrophages was observed in all samples but dermis bleeding and edema were found in 2 out of 3, and no normal bundles of collagen fibers had formed due to the injury to collagen fibers, and moreover, no elastic fibers were seen in comparison to the normal dermis. In the deep dermis sweat glands, bundles of collagen fibers, and elastic fibers (Fig. 1b) were observed in the same manner as for the normal dermis. Therefore, no granulation tissue was observed.

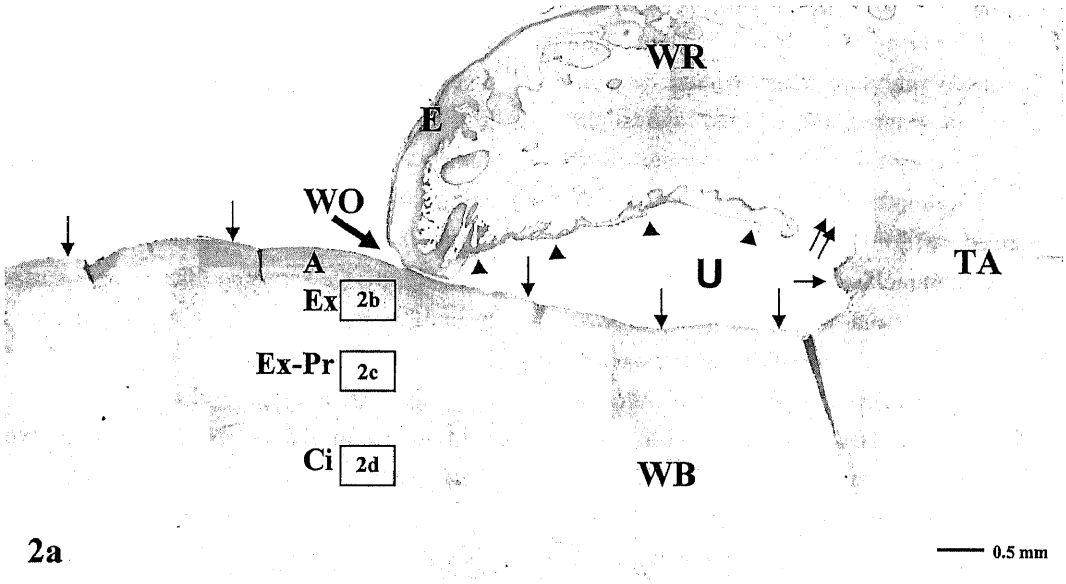
The only cells expressing α-SMA were vascular and sweat gland smooth muscle cells (Fig. 1c). No α-SMA-expressing fibroblasts or MF were found even in the wound bed of the superficial dermis, although many fibroblasts were present.

2. Stage IV pressure ulcers with undermining

The epidermis stopped at the wound orifice in three subjects and was elongated over almost the entire inner surface of the wound roof in another two subjects (Fig. 2a). The wound orifice of the former was not covered with fibrinoid tissue and this might indicate that the shear force and friction may cause the fibrinoid tissue near the wound orifice to strip. Thick fibrinoid tissue covered the inner surface of the wound roof, the transition area, and wound bed. In the dermis of the wound roof a lot of inflammatory cells as lymphocytes and macrophages were seen, while a lot of blood vessels were observed between the collagen bundles including fibroblasts. Underneath the fibrinoid tissue granulation tissue was seen which consisted of a great number of lymphocytes, macrophages and plasma cells, and a lot of thin blood vessels containing many red blood cells of which some were perpendicular to the surface, and collagen fibers (Fig. 2b). Such granulation tissue corresponds to the exudative layer[12]. And underneath, granulation tissue corresponding to an exudativo-productive layer was present, which included a smaller number of inflammatory cells and blood vessels but a larger number of collagen fibers containing more fibroblasts than the exudative layer of granulation tissue (Fig. 2c). Next, under the exudative-productive layer of granulation tissue, a cicatrizing layer of granulation tissue, that is, fibrous tissue or scar tissue was observed consisting of a large number of collagen fibers and fewer number of fibroblasts and blood vessels than the exudative-productive layer (Fig. 2d).

In all subjects (Figs. 3a, 3b, 3c, and 3d) α-SMA were positive on the vascular smooth muscles. Although the quantity of MF differed among our obtained samples, some MF were always observed in





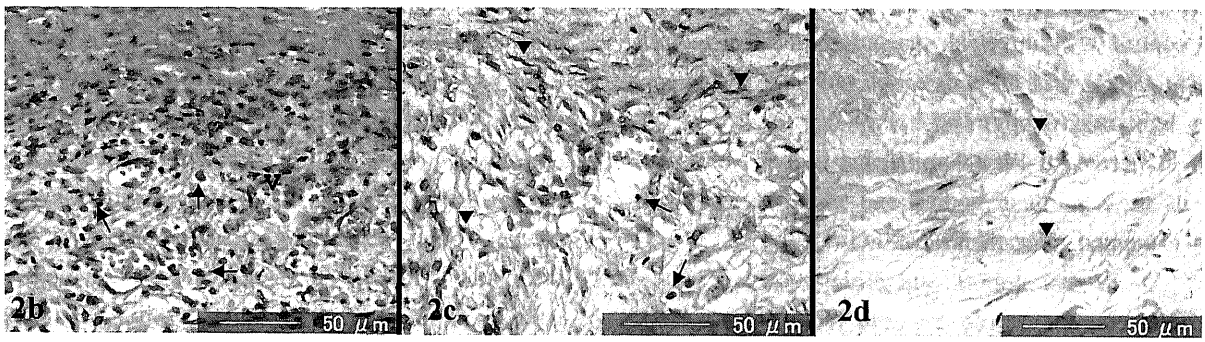


Fig. 1. A partial thickness lesion classified as a stage II pressure ulcer: (1a) and (1b) Features of H-E staining. (1c) Feature of α-SMA staining. Note the lack of an epidermis (E) and a superficial dermis area (arrow heads) which is covered with or without fibrinoid tissue (F). The arrows indicate inflammatory cells. A sweat gland (S in (1b)) is present in the dermis. No structures were stained with anti α-SMA antibody except for blood vessels (double arrows in (1c)). PL : the papillary layer of the dermis, RL : the reticular layer of dermis.

Fig. 2. A full thickness lesion classified as a stage IV pressure ulcer with undermining : Features of H-E staining. (2a) The epidermis (E) extends from the wound orifice (WO) to the inner surface of the wound roof (WR) (arrow heads) and stops on the way. A part of the connective tissue of the transition area (TA) is exposed to the undermining (U)(double arrows). The surface of the ulcer is almost completely covered with thick fibrinod tissue or the alterative layer (A) (arrows). Granulation tissue consists of the exudative (Ex), exudativo-productive (Ex-Pr), and cicatrizing (Ci) layers of granulation tissue in the wound bed (WB). In Ex (2b) a lot of inflammatory cells (arrows) and blood vessels (v) are observed among the few collagen fibers. In Ex-Pr (2c) a few inflammatory cells (arrows) are present among increased collagen fibers and fibroblasts (arrow heads). In Ci (2d) only a few inflammatory cells are found but a lot of collagen fibers and fibroblasts (arrow heads) are observed.

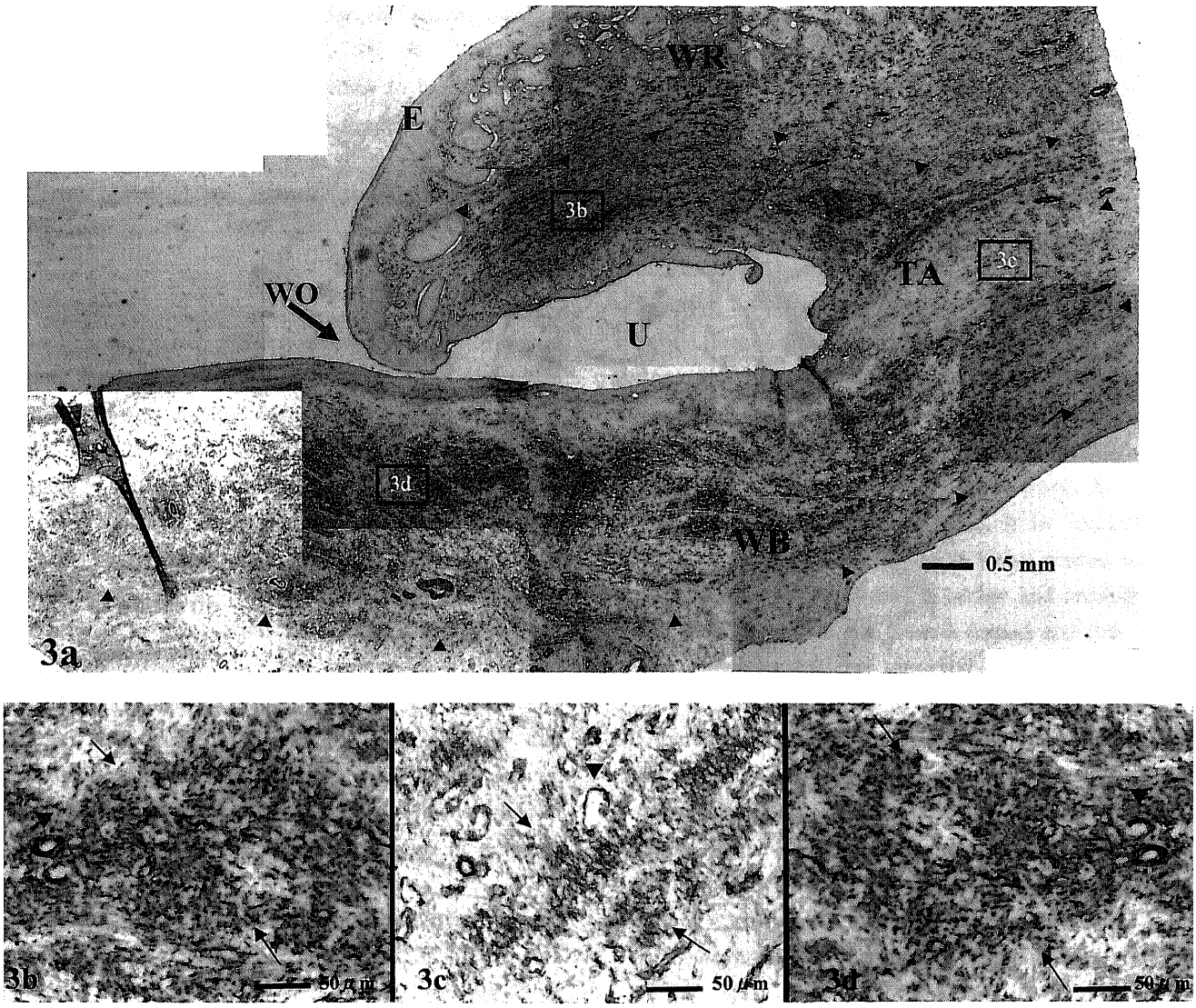


Fig. 3. A full thickness lesion classified as a stage IV pressure ulcer with undermining: Features of α-SMA staining. (3a) This feature closely corresponds to the feature of Fig. 2a. MF and blood vessels with α SMA are darkly stained. Although there are fewer MF in the transition area (TA) than in the wound roo (WR) and the wound bed (WB), it clear that MF are present in TA (Fig. 3c). Two MF bundles of WR an WB in TA become thinner until the edge of the photograph and the MF are as small in number as th MF in TA. It is thus not considered unsuitable to connect the distributions of MF in WR, TA, and WE with gap junctions between MF and fibronexuses between MF and collagen fibers[7]. As a result, the shap of the distribution of MF from WR, TA to WB develops into a J-shaped form (arrow heads). Figs. 3b, 3c and 3d are enlarged photographs corresponding to b, c, and d in Fig. 3a. In Figs. 3b, 3c, and 3d the arrov heads indicate blood vessels stained with α-SMA and arrows indicate a cluster of MF. WO: wound ori fice, U: undermining.

the wound roof, the transition area and the wound bed of all stage IV pressure ulcers with undermining.

MF were present in the dermis of the wound roof shaped like a strip parallel to the surface and this distribution of MF was found to be the same as the distribution of MF by observing a few non-serial sections and thus MF most likely concentrically surround the wound orifice and wound roof. In the transition area, MF sometimes gathered to form a lump. In the wound bed a cluster of MF were distribute parallel to the surface like a broad band. MF wer almost situated in the exudativo-productive layer o granulation tissue where abundant fibroblasts i Hematoxylin-Eosin stained sections, and a few M were observed in the exudative and cicatrizing layer Like MF in the wound roof, MF in the wound be are concentrically distributed. The cytoplasmic proc esses of MF protruded to all directions and the lon

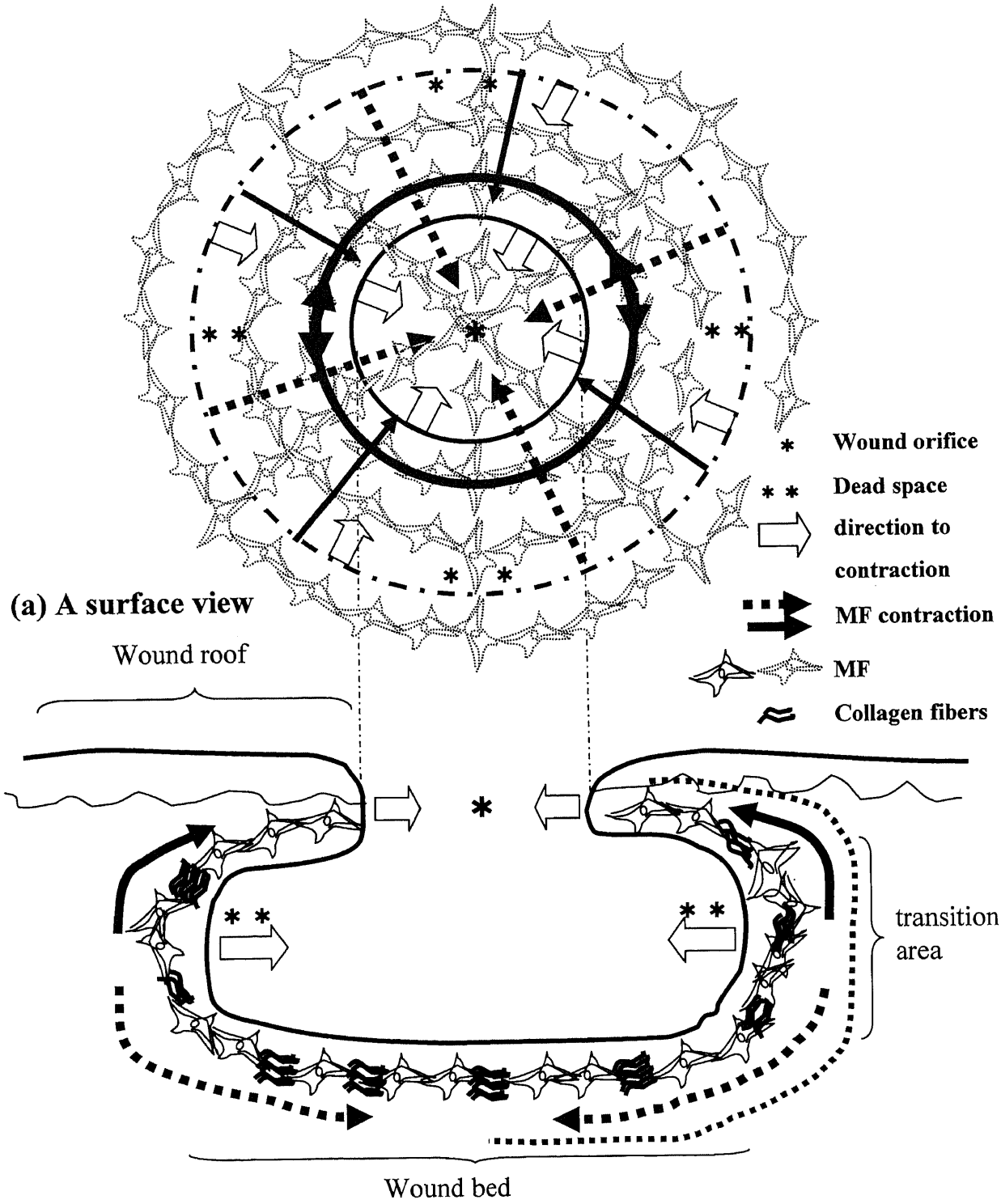


Fig. 4. Drawing of the distribution and orientation of MF in a pressure ulcer with undermining. (a) A surface view. (b) A sectional view. MF are concentrically and radially distributed in the wound roof and wound bed on a surface view (a). MF are distributed from the wound roof, the transition area to the wound bed in a J-shaped form on a half of a sectional view (dotted line in (b)). MF connected to each other and collagen fibers (collagen fibers are not drawn in a surface view). The arrows indicate the orientation of the contraction of MF toward the center of the ulcer. The open arrows indicate the contractive orientation of the wound orifice (*) and the dead space (**) in the area of undermining at the transition area in the time of MF contraction. Next, the area of undermining and wound space gradually grows closer together.

axis of MF were more parallel to the wound surface than perpendicular to it (Figs. 3b, 3c, 3d). As a result, the distribution of MF seems to be a J-shaped form from the wound roof and the transition area to the wound bed in the sectional plane and be concentric and radial in the wound roof and bed from a front view (Fig. 4).

Discussion

In this study MF were not observed in the wound bed of stage II pressure ulcers. Since these ulcers are partial-thickness wounds which occur in the papillary layer of dermis deep, while the adnexal structures remain in the deep dermis under the wound bed and so no granulation tissue forms, these ulcers probably heal by re-epithelialization alone and without wound contraction[1]. It may thus be natural that MF, which are present in granulation tissue and are involved in wound contraction[4,5], are not observed. On the other hand, if the wound depth of stage II pressure ulcers reaches the deep dermis, it may be possible that granulation tissue will form and MF will also appear. However, since we could not obtain such materials at this time, we plan to elucidate this point in a future study.

However, since the shear force and friction as well as pressure all play roles in the formation of pressure ulcers[15], the stage II pressure ulcers investigated in this study received both shear force and friction, thus resulting in mechanical stretching being applied to the collagen fibers and fibroblasts. On the other hand, under mechanical stress, fibroblasts tend to differentiate into proto-myofibroblasts and then TGF-β1 secreted by inflammatory cells influences the transition from proto-myofibroblasts to myofibroblasts[4,5]. Therefore, since stage II pressure ulcers tend to receive mechanical stress, even though they only reach the superficial layer of dermis, it is possible that MF can be formed. However, in this study no MF were observed in any stage II pressure ulcers. The disappearance of myofibroblasts after epithelialization is completed has been well estabalished[4], and so some interaction between epidermis and connective tissue may have influence on the differentiation of fibroblasts into myofibroblasts. This also remains a question to be clarified in future studies.

MF in the wound bed of stage IV pressure ulcers are distributed parallel to the surface of the ulcer and in granulation tissue and fibrous tissue thus indicating the distribution of MF to be useful for the contraction of the wound bed[22]. This also supports our finding that a thick band of MF is present in the exudativo-productive layer of granulation tissue and parallel to the surface of the wound bed. To our knowledge, however, there are no previous reports on the distribution and presence of MF in the wound roof of stage IV pressure ulcers with undermining. On the other hand, Tanaka et al[23]. who made full-thickness wounds in mice and examined the distribution of MF during wound healing reported that when the wound area decreased to about one third of its original size, a bundle of MF in granulation tissue tended to connect the wound ridges like a bridge and thereafter the MF disappeared as the wound healed and left a scar line. However, MF are observed in hypertrophic scars after burn wound[5], human knee arthrofibrosis tissue[24], Dupuytren's contracture[25], and pulmonary fibrosis[26]. In addition, they are also scattered in scar tissue in order to contract the involved tissue and produce collagen fibers. MF therefore plays a critical role in decreasing the area of the wound, thereby speeding up wound closure, or forming a pathological scar.

In the present study no hypertrophic scars were observed, but a band and a J-shaped distribution of MF in stage IV pressure ulcers with undermining were seen in a sectional view. As a result, if the ulcer could heal, the amount of granulation tissue would likely increase and at the same time the wound area of the orifice and wound bed decreases due to the contraction of MF, as Konya et al. reported[19]. Moreover, it is noteworthy that we obtained subjects with stage IV pressure ulcers with undermining who demonstrated the wound roof and wound bed to be connected by a transition area, and the distribution of the MF in the wound roof and wound bed could be observed in the same section of a pressure ulcer with undermining, and thus we could examine the relationship between the distribution of MF and the contraction of pressure ulcers with undermining. Therefore based on the concentric and radial distribution of MF in the wound roof and wound bed, and the distribution of MF in a J-shaped form from the wound roof

the transition area to the wound bed, and moreover, MF connect to each other with gap junctions and an extra cellular matrix via fibronexuses[7], the following wound contraction mechanism is thus suggested to contribute to the closure of pressure ulcers with undermining (Fig. 4). MF concentrically surrounding the wound orifice contract to reduce the area of the wound orifice. The contraction of a radial distribution of MF pulls the wound bed toward the center of the bed. MF, which are radially distributed in the wound roof and bed, contract and thereby pull the transition area toward the center of the pressure ulcer. The contraction of MF distributed in a U-shaped form in the transition area thus gradually squeezes the dead space of undermining. Such a contraction of MF decreases the area of pressure ulcers and closes the undermining of pressure ulcers as the granulation tissue proliferates.

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ステージⅡ，Ⅳの褥瘡における筋線維芽細胞の分布

北川　敦子，中谷　壽男，真田　弘美，稲垣美智子

要　　旨

【目的】本研究の目的は，ステージⅡとⅣの褥瘡における筋線維芽細胞の分布を明らかにし，創収縮との関係を検討することである。

【方法】対象は，StageⅡ３部位，ポケットのあるStageⅣ５部位（NPUAP分類）の計８部位であった。褥瘡部位は１部位が大転子部位，ほかは仙骨部位であった。褥瘡組織は，剖検および献体から採取し，ステージ別の筋線維芽細胞（MF）の分布を検討した。組織学的手法は定法に従い，HE染色，α-SMA免疫染色を施し，光学顕微鏡にて観察した。倫理的配慮は，本人および家族より同意を得て行った。

【結果】StageⅡ：肉芽組織の形成およびMFは認められなかった。ポケットを有するStageⅣ：すべての対象においてMFは，ポケットの被蓋部，移行部の結合組織中，創底部の肉芽組織中に分布していた。そのため褥瘡の断面で見ると，帯状のMFの集団は創被蓋部，移行部，創底部とＪ字型に配列していた。

【結論】StageⅡの褥瘡ではMFは出現していなかった。これは浅い褥瘡の治癒は創収縮ではなく，表皮化でおこることを示唆している。ポケットを有するStageⅣの褥瘡では，MFはポケット被蓋部，移行部，創底にかけてＪ字型に存在することから，MFが収縮すると，移行部を創の中心に引くようにして，効率良くポケットを収縮させて褥瘡を閉鎖させることが示唆された。