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# Compressed Fixation Combined with Vacuum-Assisted Closure for Treating Acute Injury of the Heel Fat Pad

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Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
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**Background:** Treating acute injury of the heel fat pad is different from treating common soft tissue damage. Due to the paucity of literature on the topic, we described our initial experience treating acute injury of the heel fat pad to determine the ideal treatment method.

**Material/Methods:** A total of 53 patients with acute injury of the heel fat pad admitted to our hospital were selected for the study and were randomly divided into 2 groups: the compressed fixation combined with vacuum-assisted closure group and the only reimplanted and sewn group. Twenty-seven of the heel fat pads were compressed and fixed using a flat, hard piece of plastic and hollow screws; then, they were covered with a vacuum-assisted closure device. The other 27 were only sewn without tension. The clinical results were evaluated according to the American Orthopedic Foot and Ankle Society hindfoot score and the British Medical Research Council function evaluation criteria

**Results:** In the compressed fixation combined with vacuum-assisted closure group, flaps of 12 feet with retrograde avulsion injury survived successfully. Partial flap necrosis occurred in 8 feet. Seven feet underwent repair using the neurocutaneous vascular resupinated island flap. Results were excellent or good for 74% of patients according to the AOFS. However, in the only reimplanted and sewn group, results were excellent or good for 44% of patients according to the AOFS.

**Conclusions:** Compressed fixation with vacuum-assisted closure is effective for treating acute injury of the heel fat pad, with high success rates and good utility.

**MeSH Keywords:** External Fixators • Heel • Negative-Pressure Wound Therapy • Replantation

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## Background

The functions of the heel fat pad of the foot include weight-bearing, prevention of sliding, and shock absorption [1,2]. Failure to treat the damaged heel fat pad can seriously affect a patient's quality of life and ability to work. Treating acute injury of the heel fat pad is different from treating common soft tissue damage, and it presents a challenge because of the unique structure of its soft tissue [3]. Recent clinical reports have focused on reimplanting and sewing the heel fat pad during the first stage, but we found that the heel fat pad had a high necrosis rate using this technique.

Therefore, we decided to change the traditional treatment and explore a novel method of compressing fixation combined with vacuum-assisted closure. After regular scrubbing, a flat, aseptic, hard piece of plastic was placed to fully cover the heel. Then, 2–3 hollow nails were used to fix the plastic piece with even compression. Finally, the site was covered using a vacuum-assisted closure device. Due to the paucity of literature on this topic, we described our initial experience treating acute injury of the heel fat pad and retrospectively evaluated the clinical outcomes to determine the ideal treatment method for this injury.

## Material and Methods

We reviewed the medical records of 62 patients with acute injury of the heel fat pad hospitalized in our department of the Third People's Hospital of Jinan from January 2009 to January 2015. The inclusion criteria were: age 14–60 years, normal coagulation function, not combined with other soft tissue or bony injuries, no diabetes, no smoking during the perioperative period, and no mental disorders. Nine patients were excluded by these criteria and 53 patients were retrospectively evaluated.

The patients with acute injury of the heel fat pad were randomly divided into 2 groups: the compressed fixation combined with vacuum-assisted closure group and the only reimplanted and sewn group. The allocation to groups was entirely determined by the random numbers table. This was done by an individual who was required to make arrangement as a third party related to the study.

The mean age of 53 patients (44 males and 9 females) was 36 years (range, 30–46 years). Twenty-seven feet had a retrograde avulsion injury, 13 had a roll-off injury, 9 had a falling injury, and 5 had a crush-related injury. The acute processing time was within 6–8 h after injury, and for 2 patients it was 12 h after injury.

Of the 26 patients in the compressed fixation combined with vacuum-assisted closure group, 21 were males and 5 were females. Their mean age was 36.1 years (range, 31–46 years). Of the 27 feet, 14 had a retrograde avulsion injury, 6 had a roll-off injury, 5 had a falling injury, and 2 had a crush-related injury.

Of the 27 patients in the only reimplanted and sewn group, 23 were males and 4 were females. Their mean age was 36.4 years (range, 29–45 years). Of the 27 feet, 13 had a retrograde avulsion injury, 7 had a roll-off injury, 4 had a falling injury, and 3 had a crush-related injury (Table 1).

This study was approved by the Ethics Committee of the Third People's Hospital of Jinan (200806). Each patient provided

**Table 1.** Preoperative patient characteristics.

| Characteristic | Fixation and vacuum-assisted group (n=27 feet in 26 patients) | Only reimplanted and sewn group (n=27 feet in 27 patients) | p Value |
|---|---|---|---|
| Age (y) | 36.1±9.90 | 36.4±8.60 | .907 |
| Sex | | | |
| Male | 21 (80.77) | 23 (85.19) | .669 |
| Female | 5 (19.23) | 4 (14.81) | |
| BMI (kg/m$^2$) | 23.36±2.25 | 22.17±2.76 | .092 |
| Classification | | | |
| Retrograde avulsion injury | 14 (51.85) | 13 (48.15) | |
| Roll-off injury | 6 (22.22) | 7 (25.93) | |
| Falling injury | 5 (18.52) | 4 (14.81) | |
| Crush-related injury | 2 (7.41) | 3 (11.11) | |
| Interval from injury to surgery (h) | 3.20±3.17 | 3.50±3.05 | .725 |

Data presented as n (%) or mean ± standard deviation. Data presented as number of feet (%).





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written informed consent for participation. Four surgeons participated in these operations.

All 53 patients underwent combined spinal-epidural anesthesia. We used the horizontal or prone position to fully expose, observe, and treat the injury site. After regular scrubbing, large amounts of hydrogen peroxide and saline were flushed repeatedly, and a large amount of iodine liquid was used to immerse the wound for 10 min. Then, we used saline to wash the wound. Contaminants were thoroughly removed during minimally invasive surgery, and the obviously necrotic skin and fat tissue were excised. The skin connected to the surrounding tissue was retained. The fat in the area of the contusion was not deeply cleared because we managed to retain the shape and size of the fat according to color. The deep fascia was meticulously debrided as much as possible, and local pressure hemostasis was performed at the bleeding site to prevent damage to the remaining blood supply and to squeeze the hemorrhage and fluid between the plantar fascia and heel fat pad. Then, we reimplanted the residual fat pad according to the anatomical structure after repair. It was sewn back into position without tension [4]. In the compressed fixation combined with vacuum-assisted closure group, a flat, aseptic, hard piece of plastic was placed in the heel and clipped to fully cover the heel. Then, 2–3 hollow nails were used to fix the plastic with even compression at the bottom of the heel fat pad. According to the exterior color of the fat pad, the tightness of the hollow nail was adjusted. Finally, the site was covered with a vacuum-assisted closure device to adequately drain the hemorrhage and effusion. All patients received intravenous antibiotics for 48 h. Intravenous microcirculation-improving drugs and discutient drugs were administered for 7 days. If necrotic tissue boundaries were not obvious after 7 days, then the vacuum-assisted closure device was changed.

### Statistical analysis

Descriptive statistical analyses were performed using the SPSS statistical package, version 13.0 (SPSS Inc., Chicago, IL, USA) for Windows. Data are presented as mean ± standard deviation (SD). A P value of <.05 was considered to indicate statistical significance

## Results

Color, tension, blood vessel filling, ecchymosis, and swelling of the flap were observed daily to determine flap survival. Necrosis was confirmed if the flaps were black and dry and if there was no bleeding 14 days after surgery. In the compressed fixation combined with vacuum-assisted closure group, of 14 feet with retrograde avulsion, the flaps of 12 survived the initial repair period (Figure 1A–1D). Appearance and function were excellent. Partial necrosis of the flap occurred in 8 feet, including 2 with a retrograde avulsion injury, 4 with a roll-off injury, 1 with a falling injury, and 1 with a crush-related injury (Figures 2A–2D, 3A–3D). After further debridement, the vacuum-assisted closure device was changed. A full-thickness skin graft was used when the wound was full of fresh granulation tissue. Extensive necrosis occurred in 7 feet, including 2 with a roll-off injury, 4 with a falling injury, and 1 with a crush-related injury. These 7 feet underwent final repair with a neurocutaneous vascular resupinated island flap. However, in the only reimplanted and sewn group, 8 flaps of 13 feet with retrograde avulsion survived the initial repair period. Partial necrosis of the flap occurred in 5 feet, and in these feet a full-thickness skin graft was finally used. Extensive necrosis occurred in 14 feet that underwent final repair with a neurocutaneous vascular resupinated island flap, including 7 with a roll-off injury, 4 with a falling injury, and 3 with a crush-related injury.

The injured limbs were elevated to reduce swelling after the operation, without a plaster cast. Partial weight-bearing (supplemented with crutches) was encouraged at 3 weeks after the wound healed well, and full weight bearing should be done gradually when the crutches are no longer used.

All patients were followed up for an average of 18 months (range, 6–36 months). At the final follow-up, patients with successful healing of retrograde avulsion injury reported partial absence of plantar sensation, limited pain or no pain, no ulcer, and no sliding. Patients with partial necrosis had a local ulcer on the bottom of the grafted skin soon after walking. Over time, the grafted skin thickened and formed a callus, and no ulcer occurred. Patients with extensive necrosis had almost complete absence of plantar sensation. In the compressed fixation combined with vacuum-assisted closure group, excellent or good results were achieved by 74% according to the American Orthopedic Foot and Ankle Society hindfoot score [5], 15 feet achieved s4, and 78% had good function and quality of life according to the British Medical Research Council function evaluation criteria [6]. However, in the only reimplanted and sewn group, results were excellent or good for 44% according to the AOFAS. Only 8 feet achieved s4 and 48% had good function and quality of life according to the British Medical Research Council function evaluation criteria. The duration of hospitalization was shorter for the compressed fixation combined with vacuum-assisted closure group than for the only reimplanted and sewn group, and the difference was statistically significant ($p<0.5$, Table 2)

## Discussion

We found that compressed fixation with vacuum-assisted closure is effective for treating acute injury of the heel fat





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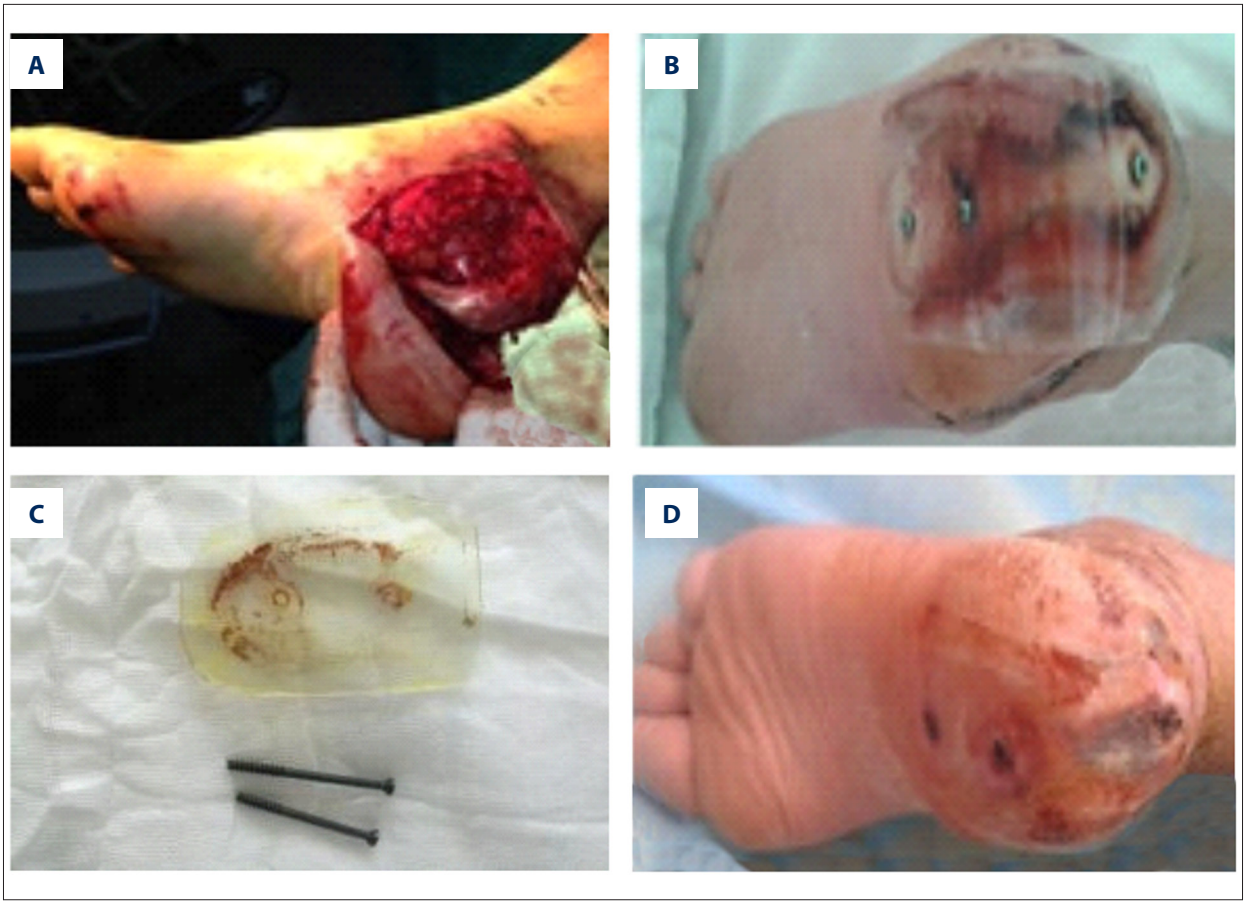


**Figure 1.** (**A**) Retrograde avulsion injury. (**B**) Reimplantation and compressed fixation. (**C**) Hard plastic and hollow nails. (**D**) Complete flap survival.

pad, with high success rates and good utility during the first stage. The results were excellent or good for 74% of patients according to the American Orthopedic Foot and Ankle Society hindfoot score (AOFS) in the compressed fixation combined with vacuum-assisted closure group. However, in the reimplanted and sewn group, results were excellent or good for only 44% of patients according to the AOFS. There was statistically significant difference ($p<.05$) in the duration of hospitalization between the 2 groups.

According to the traditional theory, injury of the heel fat pad poses significant challenges because of its unique anatomical structure and blood supply [3]. Jeng and Wei [4] reported that of 28 patients, 16 had complete or partial necrosis (57%) and underwent secondary surgery with free muscle flaps.

The heel fat pad is between the heel derma and plantar fascia, which is a dense band of fibrous tissue that originates from the medial calcaneal tuberosity. The structure of the heel pad is complex, with collagenous elements connecting the plantar fascia to the heel derma and collagen-reinforced chambers filled with fatty tissue [7,8] restricting its displacement when subjected to compressive loading and maintaining the integrity of the heel pad [9]. This means that the assembly of fat-filled collagen reinforcement can be considered an independent hydrostatic structure that supports, buffers, absorbs shock, and prevents sliding [10]. However, there is a loose fiber layer between the heel fat pad and the plantar fascia.

The blood supply of the heel fat pad mainly comes from the medial plantar artery, lateral plantar artery, and lateral calcaneal artery. The lateral plantar artery is the most important source of blood supply. These artery vessels emit branches that contribute to a vascular network. The smaller arterial branches from the vascular network distribute blood flow under the surrounding area of the heel fat pad. Although collateral circulation of the heel fat pad is abundant, the fat tissue itself in collagen-reinforced chambers has poor blood supply and regenerative ability. Therefore, the fat tissue easily develops liquefactive necrosis after injury.

The mechanism of injury is an important factor affecting treatment outcomes. The external forces of injury are initially concentrated on the back of the heel when a retrograde avulsion





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**Figure 2.** (**A**) Falling injury. (**B**) Reimplantation and fixation. (**C**) Vacuum-assisted closure device. (**D**) Partial necrosis. (**E**) Full-thickness skin graft.

injury occurs. The loose fiber layer between the heel fat pad and the plantar fascia is a result of stress breakthrough. This causes the heel fat pad along with the superficial fascia and skin to separate from the plantar fascia. This mechanism of injury was confirmed in 27 feet with a retrograde avulsion injury. As the shear stress decreases, the surrounding vascular network and chambers filled with fat are less severely affected. Furthermore, the injured heel fat pad and skin benefit from the abundant surrounding blood supply and collateral circulation compensations. Therefore, we can observe fresh blood exuding from the surface of the injured fat pad during debridement. Additionally, the pedicle of the retrograde avulsion flap can still provide a certain amount of blood supply [11]. These are important factors leading to high survival rates (74%) after the first stage of retrograde avulsion injury repair. However, with roll-off, falling, and crush-related injuries, the surrounding vascular network and fat tissue are extensively damaged. Complete or partial necrosis and secondary surgery are inevitable.

Based on the physiological and pathological characteristics of the heel fat pad, debridement should be performed with minimally invasive and noninvasive methods as much as possible to improve the flap survival rate. To rebuild the blood circulation, the use of only local pressing hemostasis at the





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**Figure 3.** (**A**) Roll-off injury. (**B**) Extensive necrosis. (**C**) Exposure of the calcaneus. (**D**) Flap repair.

**Table 2.** Postoperative patient data.

| Characteristic | Fixation and vacuum-assisted group (n=27 feet in 26 patients) | Only reimplanted and sewn group (n=27 feet in 27 patients) | p Value |
|---|---|---|---|
| Flap survival status | | | .046 |
| Survived the initial repair period | 12 (44.44) | 8 (29.63) | |
| Partial necrosis | 8 (29.63) | 5 (18.52) | |
| Extensive necrosis | 7 (25.93) | 14 (51.85) | |
| Hospitalization (days) | 18.2±2.17 | 21.3±4.36 | .002 |
| The AOFAS ankle-hind foot scale outcome | | | .046 |
| Excellent | 14 (51.85) | 8 (33.33) | |
| Good | 6 (22.22) | 5 (14.82) | |
| Poor | 7 (25.93) | 14 (51.85) | |
| The Maryland hindfoot score outcome | | | .024 |
| Excellent | 15 (55.56) | 8 (29.63) | |
| Good | 6 (22.22) | 5 (18.52) | |
| Poor | 6 (22.22) | 14 (51.85) | |

Data presented as n (%) or mean ± standard deviation. Data presented as number of feet (%).





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bleeding site (but not clamping, ligation, or electrocoagulation) is necessary to prevent damage to the remaining blood supply. Avoiding excessive clearing of the injured fat tissue is also important as a buffering function. This is different from treating a general avulsion injury of the skin. Management to retain the skin of the heel involves complete removal of necrosis after 7–10 days.

Adani et al. [12] reported that the main advantage of *in situ* repair of retrograde avulsion of the skin was the maximal recovery of the anatomical structure and function of the skin. We agree with this view. After debridement, we squeezed the hemorrhage and effusion, reimplanted the fat pad *in situ*, and sutured the anatomical counterpoint exactly and loosely to provide space for the blood circulation to rebuild and to facilitate drainage of the hemorrhage and effusion. The depth of the needle was suitable when it only penetrated the skin, thereby not causing injury to the remaining blood supply. The flat, hard, sterile plastic and hollow nails allowed the fat pad to bear average force and closely attach to the plantar fascia, thus eliminating the dead space and avoiding effusion and hemorrhage; this was favorable for microvascular regeneration of the contralateral organization and re-establishment of the blood circulation. Organized nutrition may enter the injured flap from the healthy part. Jeng and Wei [13] also reported that the injured flap can partly survive by relying on the organized drainage nutrition after a classic immobilizing and compression dressing. Compressed fixation provides a safe environment by preventing the fat pad from sliding and extruding, thus reducing the chance of shearing movements between the fat pad and the plantar fascia. Use of a transparent plastic sheet was advantageous for observing the blood supply. Adjusting the tension of the hollow screw in a timely manner was also important to avoid secondary damage. The vacuum-assisted closure device could efficiently remove the hemorrhage, effusion, and necrotic tissue [14,15], thereby greatly reducing the risk of infection and necrosis. Formation of fresh granulation tissue was rapidly promoted [16]; thus, 12 of 27 feet flaps survived completely during the initial repair period. Partial necrosis of the flap only occurred in 8 feet, but after receiving a full-thickness skin graft on the surface of the fresh granulation tissue, they retained the functions of buffering, bearing, and supporting.

Limitations of this study include its retrospective evaluation of surgical treatment, the small number of patients, and the absence of long-term follow-up for evaluating outcomes. Further prospective studies are required to compare this technique with other treatments. Although our sample size was small compared with those of other studies, our clinical experience was successful.

## Conclusions

Based on our analysis of 54 feet, we think that compressed fixation with vacuum-assisted closure is an effective method for treating injury of the heel fat pad during the first stage of repair. The technique had a high rate (74%) of success and functioned well; however, the therapeutic effect had a close relationship with the mechanism and degree of injury. Although full-thickness skin grafting is controversial in the clinical setting, patients with partial necrosis of the flap returned to normal work and life activities after 1 year. This novel technique of compressing fixation combining with vacuum-assisted closure deserves serious consideration for treating acute injury of the heel fat pad during the first stage of repair.

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