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Case Report

Late compromise of the free fibula osteocutaneous flap and its sequela: a case report

Yi Liu, Mardhiah Jeffrey, Wan Azman Wan Sulaiman

Reconstructive Sciences Unit, School of Medical Sciences, Health Campus, Universiti Sains Malaysia, Kelantan, Malaysia

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ABSTRACT

Introduction: Hematoma can adversely affect a flap in several ways, leading to flap compromise. The impact of hematoma on free flap is more complex than previously understood. Free flap compromise due to hematoma most commonly occurs during the early postoperative period.

Aim: This article highlights the late compromise of the free fibula flap and the significance of flap monitoring after a secondary procedure.

Case study: We present a case of late free fibula osteocutaneous flap compromise on postoperative day 13 due to hematoma. A 21-year-old woman with left tibia osteofibrous dysplasia underwent wide resection and left tibia reconstruction using the modified Capanna technique. Postoperatively, she developed a deep surgical site infection with implant exposure, secondary to partial thickness necrosis of the skin paddle caused by hematoma-induced ischemia-reperfusion injury. She was treated with antibiotics, dressings, and debridement. Implant removal was performed at postoperative month 5 following bone union.

Results and discussion: Hematoma can exert pressure on surrounding tissues, leading to vascular pedicle compression and thrombosis. The time dependency of flap salvage rates is associated with the progression of irreversible ischemia-reperfusion injury. Complications such as surgical site infection with implant exposure may occur. Data on timing of implant removal remain limited. Conventional management involves debridement, antibiotics, and hardware removal. The implant may be preserved until bony healing is achieved, provided there is no implant failure, prolonged exposure, or active infection.

Conclusions: Early recognition and prompt re-exploration are crucial for flap salvageability in the presence of hematoma. The removal of an exposed implant is controversial, as clear guidelines are still lacking.

Corresponding author: Yi Liu; Reconstructive Sciences Unit, School of Medical Sciences, Universiti Sains Malaysia, 16150 Kubang Kerian, Kelantan, Malaysia. Tel.: +60178998768.
E-mail address: yi5461@gmail.com

1. INTRODUCTION

Hematoma is believed to adversely affect the flap viability through various mechanisms, ultimately leading to flap compromise. Free flap compromise due to postoperative hematoma most commonly occurs in the early postoperative period. We present a case of late free fibula osteocutaneous flap (FFOCF) compromise secondary to hematoma.

2. AIM

This article highlights the late compromise of the free fibula flap and the significance of flap monitoring after a secondary procedure.

3. CASE STUDY

A 21-year-old woman presented with a painless swelling over the left mid-shin that had been progressively increasing in size for 7 years, without associated functional deficits. She experienced discomfort in the left leg, particularly during prolonged standing and going down the staircase, for the past 5 months. There was no history of trauma or significant family history.

On examination, she was well-built, and we noticed a hard mass with intact overlying skin measuring 13 × 10 cm over the anterior mid-third of the tibia (Figure 1). Tissue biopsy confirmed osteofibrous dysplasia. Magnetic resonance imaging (MRI) revealed a left tibial bone intraosseous mass measuring 3.2 × 3.7 × 12.3 cm over the anteromedial aspect of the proximal and mid-diaphysis of the tibia (Figure 2). There was no periosteal breach or extension into adjacent tissue.

The orthopedic team performed wide resection of the left tibia, resulting in a 17-cm defect. The tibia reconstruction was carried out by the plastic and reconstructive surgery team using the modified Capanna technique, combining a FFOCF and a cadaveric bone allograft. The peroneal artery was anastomosed to the anterior tibial artery, and each of the peroneal veins was anastomosed to the anterior tibial vein and great saphenous vein (GSV), respectively. On postoperative day 13, the flap became congested following delayed primary closure the day before (Figure 3). Emergency flap re-exploration discovered a hematoma beneath the skin paddle without an obvious bleeding point. The GSV was thrombosed and unsalvageable, while the remaining venous and arterial pedicles were patent.

The flap remained viable with areas of partial thickness necrosis. Subsequently, she developed sepsis secondary to left leg deep surgical site infection (SSI) (Figure 3). Modern dressings, debridement, and antibiotic administration were commenced. At the 5-month follow-up, she presented with persistent serous discharge and a worsening wound defect with implant exposure. Implant removal was performed. The intraoperative macroscopic inspection (Figure 3) and X-ray (Figure 4) demonstrated bone union at both ends of the tibia

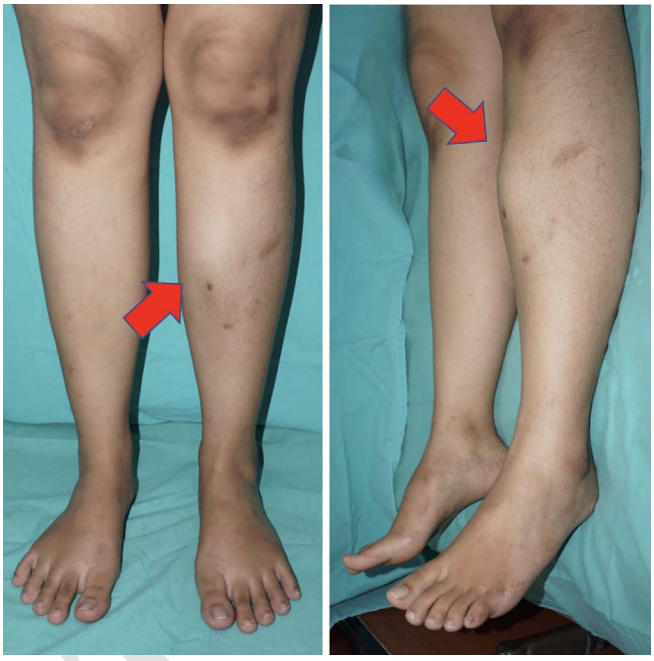

Figure 1. Preoperative photos reveal a hard mass measuring 13 × 10 cm over the anterior middle third of the left tibia (red arrow). The overlying skin was intact.

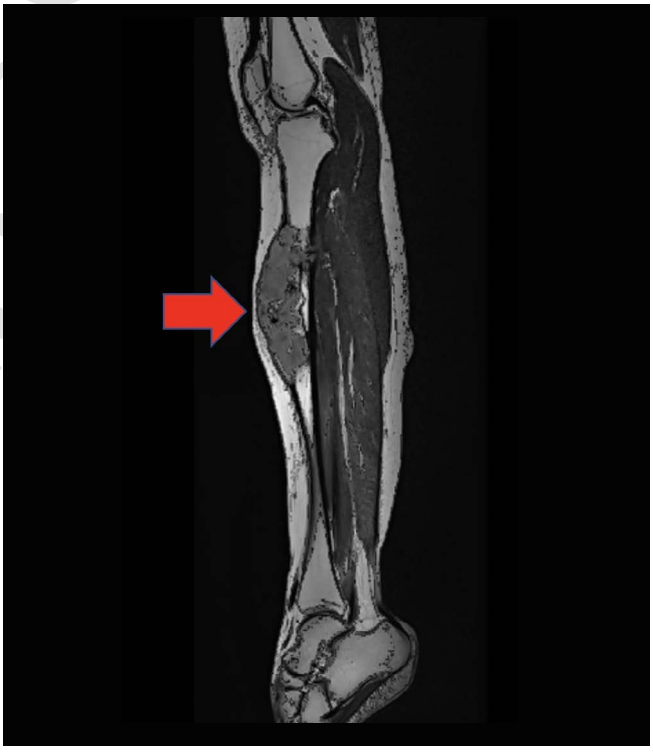

Figure 2. T2-weighted MRI of the left leg reveals an intraosseous heterogeneous hyperintense mass (red arrow), measuring 3.2 × 3.7 × 12.3 cm over the anteromedial aspect of proximal and mid diaphysis of the left tibia. This mass is exophytic and causes cortical destruction at the anterior and medial aspects of the left tibia. However, the mass is completely enclosed by the periosteum of the tibia. No breach of periosteum nor extension into the adjacent tissue or knee joint was identified.

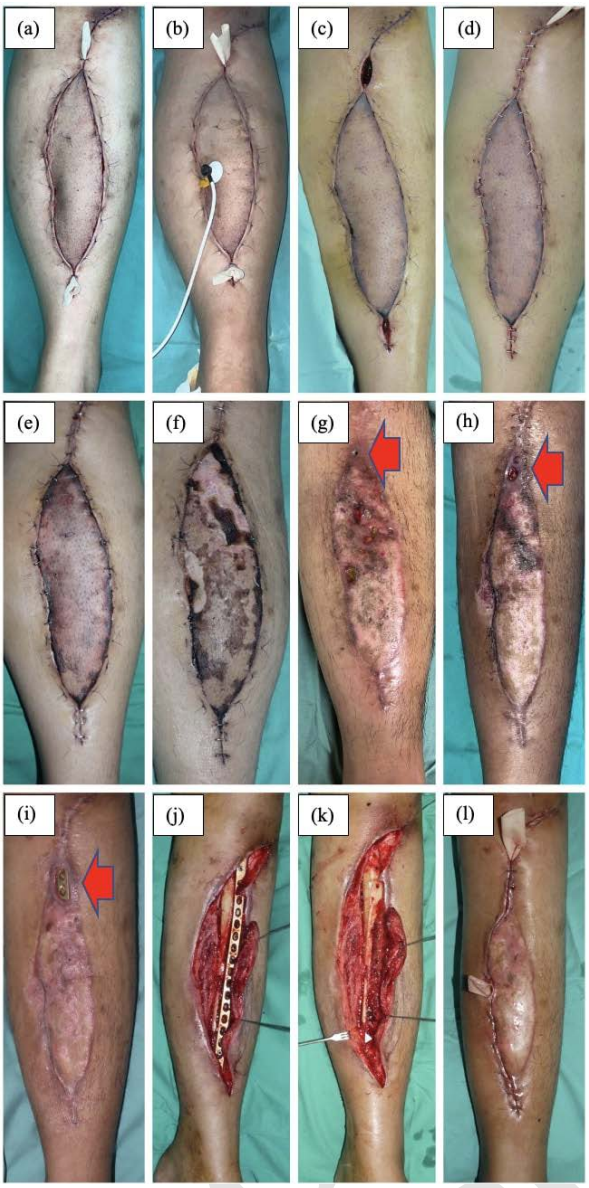

Figure 3. Immediately post-reconstruction (a); Post-reconstruction day 2, flap was pink and supple (b); Post-reconstruction day 13, flap appeared to be dusky and swollen, thus emergency re-exploration was performed (c); Immediately post-exploration and evacuation of clots, the reperfusion was evidenced by flap hyperemia (d); Post-exploration day 5, ischemic reperfusion injury was well evidenced by the blistering and patchy epidermolysis (e); Post-exploration 3 weeks, partial thickness necrosis was well-defined (f); Post-exploration 2 months, the patient developed deep SSI with a sub-centimeter sinus (red arrow) draining pus from the superior pole of the skin paddle (g); Post-exploration 4 months, the sinus (red arrow) enlarged to 1cm as the skin paddle contracted with implant exposure (h); Post-exploration 5 months, the defect (red arrow) over the previous sinus enlarged to 3 cm (i); Post-exploration 5 months, removal of implant was done and revealed no evident of implant failure (j); After implant removal, bone unions were noted at both ends of the allograft (green indicators) with no signs of osteomyelitis (k); Primary wound closure done with a local advancement flap (l).

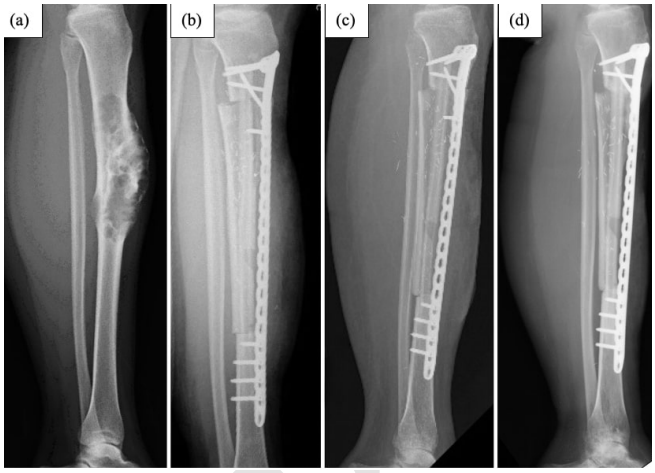

Figure 4. Serial X-rays of left leg: (a) The tumour was occupying the proximal and middle third of the tibia; (b) Immediate post-resection of the tumour and tibia reconstruction with the modified Capanna's technique; (c) Post-reconstruction 3 months, there was promising bone union without signs of implant failure or osteomyelitis despite on-going SSI; (d) Post-reconstruction 5 months, bone unions were noted at both ends of the allograft with thinning of the overlying skin paddle.

allograft and no implant failure or signs of osteomyelitis. The defect was resurfaced with a local advancement flap.

Postoperatively, she developed pes equinus over the recipient leg due to tight Achilles tendon secondary to prolonged immobilization with cast and crutches ambulation. The condition resolved following physiotherapy. At 10 months postoperatively, she was able to ambulate with full weight bearing.

4. DISCUSSION

As the second-largest bone in the human body, the tibia functions primarily to bear weight and facilitate locomotion.[1] The goal of tibia reconstruction is to restore both form and function as closely as possible to the baseline.[1] The modified Capanna technique offers a single-stage solution that provides structural stability for long-segment tibia reconstruction, minimizes size mismatch between the tibia and fibula flap by incorporating a cadaveric bone allograft, while the vascularized fibula enhances biological integration through its strong osteogenic capacity.[1] This technique also facilitates smoother sliding of the vascularized fibula with a larger trough in the allograft and enables flap monitoring through the skin paddle.[1] In contrast, other commonly used techniques, such as distraction osteogenesis, Masquelet's induced membrane technique, and allografts, generally require multiple surgeries and longer treatment duration.[1]

Currently, there is no literature on the optimal timing for definitive closure of the recipient site following FFOCF. In our practice, delayed primary closure of the recipient site is performed approximately two weeks post-reconstruction or when the drain has persistent minimal output, indicat-

ing cessation of bone marrow bleeding. During flap re-exploration, no bleeding point was identified. As the patient was not on anticoagulants, the hematoma likely developed either from injury sustained during the delayed primary closure or slow oozing from the bone marrow that subsequently stopped due to the tamponade effect of the hematoma. Therefore, frequent flap monitoring is crucial after delayed primary closure of the recipient site to ensure early recognition flap compromise.

The thrombosed GSV was not salvaged as the clinical signs of compromise resolved after evacuating the hematoma. Hematoma could exert pressure on the surrounding tissues, leading to vascular pedicle compression and thrombosis.[2] Ahmad et al. demonstrated that 60% of compromised flaps secondary to hematoma exhibited evidence of vascular thrombosis at the time of re-exploration, with isolated venous thrombosis occurred most frequently.[2] Other proposed mechanisms for hematoma-associated flap compromise include oxidative and nitrative stress, release of proinflammatory cytokines, complement activation, cellular inflammatory response, and endothelial dysfunction, leading to tissue injury with or without vascular occlusion.[2]

The time dependency of flap salvage rates is associated with the increasing irreversible ischemia-reperfusion injury (IRI) or the no-reflow phenomenon in cases where vascular patency can be re-established.[2] The presence of thrombosis in the pedicle in cases of hematoma-induced flap compromise, is a strong predictor of decreased flap salvage.[2] The skin paddle is an effective monitor for the bone component.[3] The IRI was evident from the partial thickness necrosis of the skin paddle. Complications such as SSI with implant exposure eventually occurred. There is limited consensus to guide the appropriateness of routine implant removal.[4] Conventional methods typically involve irrigation, debridement, antibiotics, and hardware removal.[5] The implant may be retained until bony healing is achieved, provided that the exposure time is short, infection is controlled, and there is no evidence of implant failure.[5] Jayaramaraju et al. reported the plating removal after bone union was achieved, which usually took five to nine months.[1] Yudistira et al. systematically reviewed implant removal and retention methods for SSI management after spinal instrumentation surgery and found that implant retention was justified if the SSI occurred less than three months post-surgery, while removal is recommended if infection persisted beyond three months.[6]

6. CONCLUSIONS

(1) Early recognition and prompt re-exploration are crucial for flap salvageability in the presence of hematoma.
(2) The removal of an exposed implant is controversial, and clear guidelines are still lacking.
(3) Further study is necessary to compare the effect of implant removal timing and retention on patients' quality of life, pain, and infection recurrence.

Conflict of interest
None declared.

Funding
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