Case Report

Acute Compartment Syndrome Following Delayed Donor Site Necrosis: A Case Report & Literature Review

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ABSTRACT

Fibula free flap donor site morbidity is usually minor, but acute compartment syndrome (ACS) is exceedingly rare. The sequential development of donor site necrosis followed by delayed ACS is scarcely reported. A 61-year-old man underwent mandibular reconstruction using a fibula osteocutaneous free flap. The donor site was closed under tension after a 120 min tourniquet time. By postoperative day 3, progressive edema, bullae, and ecchymosis developed. On postoperative day 10, the patient showed numbness, loss of motor function, nonpalpable pulses, and worsening necrosis. Based on the clinical findings, delayed ACS was diagnosed. Decompression through reopening the lateral incision revealed a markedly tense lateral compartment. The wound was managed with negative-pressure therapy, with planned skin grafting. Persistent foot drop and sensory loss remained. Seventeen relevant articles were identified, including eight donor-site ACS cases. Most presented in a delayed fashion associated with closure under tension, large or distal skin paddles, prolonged tourniquet use, or postoperative hematoma. Delayed diagnosis commonly led to lasting neuromuscular deficits. Donor-site necrosis can mask a developing ACS and delay recognition. Tension-free closure, cautious tourniquet use, and vigilant postoperative monitoring are essential. Early decompression remains critical to prevent irreversible morbidity.

KEYWORDS
Acute compartment syndrome; Necrosis; Case report

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INTRODUCTION

The fibula free flap (FFF) has become the go-to flap for head and neck reconstruction due to its reliable blood supply, long pedicle, versatility for shaping, and low risk of complications at the donor site. While the focus has largely been on the problems at the reconstruction site, issues at the donor site, like minor seromas or sensory deficits, are often considered less critical. However, severe complications, such as compartment syndrome, are rare but can be devastating [1].

Recent studies suggest donor site complication rates following FFF harvest range from 10% to over 35%, depending on skin paddle size, closure method, and patient comorbidities [2]. Acute complications include delayed healing (9–27%), infection (5–8%), and partial skin graft loss

(7–11%). Chronic issues such as paresthesia, ankle stiffness, and great toe contracture occur in up to 40% of patients, particularly when large skin paddles or flexor hallucis longus resection are involved. Despite acceptable limb function in most cases, long-term deficits in balance, proprioception, and gait have been consistently observed in prospective analyses [3, 4]

Acute compartment syndrome (ACS) in the leg after a fibula flap harvest is an extremely rare complication. Most large studies on FFF outcomes do not even mention compartment syndrome as a reported event, which suggests its incidence is well below 1%. However, individual case reports highlight that it can occur under specific conditions. For example, a 2015 report described a patient who developed ACS at the donor site after a large skin paddle was harvested and the wound was closed under tension [5] . A more recent case detailed a delayed ACS due to a hematoma in a patient on anticoagulation therapy [6]. Accordingly, ACS at the donor site can be caused by multiple factors and may develop gradually, making early diagnosis difficult.

The process that leads to donor site necrosis and then ACS likely involves a series of events, including localized blood flow issues, swelling, and poor venous drainage [7]. Skin paddle necrosis, especially in the lower leg, is often blamed on harvesting skin beyond the territory of reliable perforating vessels. While septocutaneous perforators from the peroneal artery are dominant in most people, they can be variable. Extending the flap more than 8 to 10 cm distally can place the skin paddle in a "choke zone" with limited blood supply. This compromised tissue is then susceptible to progressive necrosis, inflammation, and fluid buildup [5]. When this is combined with a tight wound closure or an extended tourniquet time, it can lead to increased pressure within the fascial compartments [7]. In some cases, the inflammatory response or a postoperative hematoma may cause a delayed compartment syndrome days after surgery [6].

This complex process highlights the need for careful flap design, especially regarding the size and location of the skin paddle. Preoperative planning with computed tomography angiography (CTA) or Doppler ultrasound can help identify the best perforators and prevent harvesting skin in poorly perfused areas. Intraoperative indocyanine green (ICG) angiography can also provide real-time feedback on blood flow [8]. While these techniques are becoming more common in high-volume medical centers, they are not yet standard practice everywhere [9].

Several patient and surgery-related factors can increase the risk of donor site ischemia or ACS. Smoking, diabetes, peripheral vascular disease, prior trauma, and anticoagulation therapy have all been linked to a higher risk of wound complications. A recent study of 119 FFF patients found that alcohol use and the method of donor site closure were independent predictors of wound breakdown [10]. The study also noted that using a split-thickness skin graft increased the risk of wound complications by 2.5 times, which underscores the need for individualized closure techniques. Excessive tourniquet pressure or prolonged use can also be a factor, with orthopedic guidelines recommending limiting tourniquet time to under 120 min at the lowest effective pressure.

Diagnosing ACS is a top priority, as delays in treatment can lead to permanent nerve and muscle damage, contractures, or limb loss. Classic symptoms include disproportionate pain, pain with passive movement, numbness or tingling, and tense compartments. However, in patients who are sedated or have just had surgery, these signs may be difficult to detect or be misinterpreted. Measuring the pressure within the compartment can help when the clinical signs are unclear. An absolute pressure over 30 mmHg or a pressure differential (diastolic blood pressure minus compartment pressure) below 30 mmHg are surgical thresholds [7]. However, relying too heavily on these measurements should not delay intervention if there is a strong clinical suspicion.

Emergent four-compartment fasciotomy is the definitive treatment for ACS and should ideally be performed within 6 hours of symptom onset to save the tissue. Post-fasciotomy wound management often involves negative-pressure wound therapy (NPWT) and staged closure or skin grafting. NPWT has been successfully used to manage donor-site tissue loss after fibula flap harvest, as it promotes granulation and helps control bacterial growth [5].

To prevent these complications, surgeons can limit the skin paddle size, avoid closing the wound under tension, use drains to prevent hematoma formation, follow safe tourniquet protocols, and standardize postoperative neurovascular monitoring [2, 5, 7]. Although donor site risks for the FFF are still

relatively low compared to other flaps, the potential for catastrophic outcomes like ACS justifies increased vigilance.

While both donor site necrosis and compartment syndrome are known complications, their sequential presentation has not been well documented.

This case report provides a unique and clinically important account of a rare progression from donor site necrosis to delayed ACS. We describe the case of a patient who underwent fibula osteocutaneous flap harvest for mandibular reconstruction and subsequently developed progressive skin necrosis at the donor site that ultimately led to acute compartment syndrome. The timeline, clinical findings, and interventions highlight the importance of careful surgical planning, early diagnosis, and vigilant postoperative care.

CASE PRESENTATION

The patient was a 61-year-old man with no significant systemic comorbidities. He was not taking antiplatelet/anticoagulant agents, steroids, or vasopressors preoperatively.

On 2nd July, the patient underwent mandibular reconstruction using an osteocutaneous fibula free flap to address an oncologic mandibular defect. This was performed to cover a previously placed reconstruction plate. During the procedure, the fibular head was preserved, and a tourniquet was maintained at 350 mmHg for 120 min. Hemostasis was achieved, and a Hemovac drain was placed, which yielded 250 mL in the initial 24 hours. The donor site fascia and skin were closed under tension without the use of a splint or compressive dressing. While in the ICU, the patient was receiving analgesia and sedation and did not report pain.

Three days post-surgery, the donor leg showed progressive swelling with the development of hemorrhagic and serous bullae and diffuse ecchymosis. A bedside examination revealed a tense limb with nonpalpable pedal pulses, prolonged capillary refill, and a fever of 38 °C, though systemic oxygen saturation remained preserved. Approximately ten days after the initial operation, turbid drainage, which was suspicious for infection, appeared from the incision, prompting a decision for operative decompression (Figure 1).

At the time decompression was considered, approximately ten days post-surgery, the patient's heart rate was 70 beats per minute and blood pressure was 138/87 mmHg. The donor leg exhibited marked edema and ecchymosis with hemorrhagic and serous bullae. Maximal firmness was noted over the lateral compartment. Pedal pulses were not palpable, and no Doppler signals were documented. The patient experienced numbness in the foot and had no voluntary motor activity at the ankle or toes.



Fig. 1. Donor leg on postoperative day 3 showing edema, ecchymosis, and hemorrhagic bullae

Pain on passive stretch was not demonstrable, likely due to the ongoing ICU analgesia. The skin temperature of the limb was perceived as normal. Compartment pressures were not measured.

A preoperative CTA had demonstrated normal arterial anatomy. No post-symptom CTA, ABI, or duplex ultrasound was obtained. Clinical photographs revealed a long lateral staple line with massive edema, widespread ecchymosis, and hemorrhagic and serous bullae at the donor site, consistent with cutaneous ischemic compromise secondary to raised intracompartmental pressure.

The working diagnosis was acute compartment syndrome of the fibula donor leg. The most plausible etiology was closure under tension within a noncompliant compartment, compounded by reperfusion edema following a prolonged high-pressure tourniquet and extensive osteocutaneous dissection. The absence of palpable pulses was attributed to severe edema rather than a proximal arterial occlusion.

The donor limb was decompressed approximately ten days post-surgery by reopening the original lateral incision. Intraoperatively, no hematoma or frank muscle necrosis was reported, though the lateral compartment was noted to be under the highest tension. The wound was left open and managed with modern dressings and a suction drain functioning as negative-pressure therapy. The plan was for a delayed split-thickness skin graft once a healthy granulating bed formed.

Empiric cefazolin 1 g every six hours was prescribed for seven days. Antithrombotic management consisted of heparin 5,000 IU intravenously three times daily for seven days, with a transition to aspirin 80 mg once daily after discharge. Wound cultures were obtained to guide potential antibiotic adjustments, and rehabilitation was scheduled to commence after soft tissue stabilization.

With decompression at this stage, the primary objective was to salvage limb perfusion, control evolving soft tissue compromise, reduce infectious risk, and prepare the wound bed for grafting. Given the delay from the onset of objective signs to fasciotomy, persistent neurological deficits, particularly common peroneal nerve distribution weakness and sensory loss, were anticipated. However, partial recovery with structured rehabilitation and the use of an ankle–foot orthosis was a hoped-for outcome. The expected soft tissue outcome was durable coverage following negative-pressure therapy and a subsequent split-thickness skin graft.

Following decompression, the donor site wound remained open under negative-pressure wound care and required a split-thickness skin graft. The patient has persistent foot drop and sensory loss in the donor limb, and formal rehabilitation has not yet commenced. Screening for deep vein thrombosis and formal exclusion of a superimposed cellulitis or necrotizing infection has not been documented. Further management will focus on completing soft tissue closure, initiating physiotherapy with orthotic support, serially documenting Doppler perfusion, tailoring antibiotics to culture results, and arranging electrodiagnostic testing at three to four weeks to refine the neurologic prognosis and plan for a potential late tendon transfer if dorsiflexion fails to recover.

REVIEW OF LITERATURE

Database searches initially identified 37 records (PubMed: 14, Scopus: 20, Web of Science: 3). After removing 16 duplicates, 21 unique records remained for title and abstract screening. Four records were subsequently excluded for not meeting the inclusion criteria (non-fibula flaps, recipient site–only outcomes, or insufficient donor site data). Consequently, 17 articles proceeded to full-text review and were included in the qualitative synthesis.

A review of the eight donor-site ACS cases identified in this corpus revealed that the onset was most frequently delayed (≥ postoperative day 4) in 5/8 (62.5%) of cases, followed by early (postoperative day 2–3) in 2/8 (25.0%), and immediate (≤ 24 h) in 1/8 (12.5%) [11-18]. The reported delayed presentations occurred as late as postoperative day 7–13 or "around week 1–2," underscoring that progressive edema, tissue necrosis, or hematoma formation, rather than a classic hyperacute presentation, often culminates in a late intracompartmental pressure rise [11, 13, 17, 18].

Etiology and predisposing factors

A recurrent theme in the identified cases was closure under tension, particularly after a skin paddle was harvested or following a tight, layered primary closure. This appeared to precipitate both cutaneous ischemia and an elevation in compartment pressure [11, 12, 16-18]. Large or distally extended skin paddles and

distal third skin incisions were repeatedly implicated in donor site ischemia, which aligns with the known perforator "choke zone" concerns in the lower leg soft tissues [13, 15, 16, 19].

Tourniquet use was documented in most ACS cases, though with heterogeneous settings, including one report of approximately 190 min of ischemia and pressures around 400–450 mmHg. This suggests that excessive pressure or prolonged duration may amplify reperfusion edema within the noncompliant fascial compartments [13, 14, 17]. Conversely, a technique paper advocating for tourniquet-free harvest proposed that minimizing tissue distortion during flap elevation could lower the overall donor site morbidity risk profile, implicitly including the risk of ACS [19]. Furthermore, the explicit presence of hematoma or dark, turbid fluid at the donor site was noted in delayed ACS cases and may serve as a direct driver of the pressure rise and subsequent skin necrosis cascade [6, 17, 18].

Diagnosis and clinical clues

Classical findings associated with ACS such as disproportionate pain, pain on passive stretch, tense compartments, sensory changes, and rising creatine kinase (CK) were variably present. Importantly, analgesia and sedation in several cases masked the pain, leading to atypically subtle or "mild pain" presentations that delayed recognition [11, 14, 18]. Loss of distal pulses was an inconsistent finding and, when observed, was often related to severe edema rather than a proximal arterial occlusion, highlighting the critical importance of evaluating the overall clinical picture during decision-making [15, 17].

Management patterns

The majority of cases required urgent decompression, which was achieved either by reopening the donor incision or performing a formal fasciotomy. This was frequently followed by staged debridement, application of negative pressure wound therapy (NPWT/VAC), and delayed split-thickness skin grafting (STSG) [13, 16-18]. In the reviewed ACS cases, primary closure had been performed in 6/8 patients, STSG at the time of the index closure in 2/8, and the donor site was left open in 1/8 (Note: one case may have been managed by simple incision opening, not formal fasciotomy) [11, 12, 15-17].

This distribution supports the narrative that tight primary closure is a modifiable risk factor. NPWT was reported as a useful adjunct in approximately one-third of ACS cases (3/8), aiding granulation and bacterial control before grafting [13, 17, 18].

Outcomes

Despite successful limb and flap salvage in most reports, persistent neuromuscular deficits were a common outcome, documented in 5/8 total ACS cases. This included foot drop, equinus, dorsiflexion weakness, and sensory loss, occurring especially when the diagnosis or decompression was delayed [11, 14-17]. Good or near-complete functional recovery was achieved only in a minority of patients, typically when timely decompression was coupled with structured wound and rehabilitation protocols [13, 18].

Context from morbidity studies

Beyond the immediate complication of ACS, cross-sectional and cohort data emphasize the existence of substantial long-term donor site morbidity. Neuropathic pain has been reported in approximately 21% of patients, and claw toe deformity in around 25%. These conditions have been linked to ischemic injury of the intrinsic leg muscles and appear to be influenced by the closure strategy and skin paddle geometry [20, 21].

Historical series involving complex upper extremity reconstructions using osteocutaneous fibula flaps reported high flap survival rates but still documented isolated cases requiring donor site decompression, reinforcing that such catastrophic complications, while rare, do occur [22-24]. Technique papers have proposed several risk-reduction measures including limiting distal third skin paddles, avoiding tight primary closure, and considering tourniquet minimization or avoidance to mitigate the ischemia-edema dynamics at the donor site [19, 25].

The sequential progression from donor site necrosis to delayed ACS observed in the present patient is consistent with the delayed timelines and mechanistic interplay reported in the literature. It mirrors the combination of tensioned closure, potential tourniquet related edema, and the possible local effects of fluid or hematoma [6, 11, 13, 14, 17, 18]. This synthesis supports a mechanistic chain that is challenging to detect promptly in the context of postoperative analgesia.

This review reinforces the importance of practical safeguards for future cases: meticulous skin paddle design and positioning, strict avoidance of tight primary closure, disciplined tourniquet settings and minimized ischemia time, early drainage and hematoma control, vigilant neurovascular monitoring despite the use of analgesia, and maintaining a low threshold for decompression when the global clinical picture suggests evolving compartment pressure [15, 16, 19-21].

DISCUSSION

The main finding of this case report is the detailed description of an uncommon yet clinically critical progression from delayed donor site necrosis to ACS following FFF harvest. The timeline, characterized by the onset of edema and bullae by postoperative day 3 and culminating in surgical decompression on day 10, underscores the significant diagnostic hurdles imposed when early warning signs are masked by analgesia or sedation [6, 14, 17].

The pathophysiology observed in this patient aligns with a confluence of established risk factors for donor site complications. The development of ACS in this case reflects the interplay between excessive closure tension, a prolonged tourniquet time (120 minutes at 350 mmHg), and subsequent postoperative reperfusion edema, all contributing to a critical rise in intracompartmental pressure [5, 7, 13]. The lateral skin paddle may have extended beyond reliable perforator zones, placing it in a vascular "choke point" and increasing the risk of necrosis and inflammatory swelling [5, 13, 15]. Sedation likely masked disproportionate pain, typically the earliest warning sign, delaying the clinical suspicion of ACS [11, 18]. Although decompression was ultimately performed, persistent neuromuscular deficits reflect irreversible ischemic damage from the delayed intervention [5, 13, 17].

This case highlights the imperative for rigorous donor site risk mitigation in all fibula free flap procedures. To prevent perfusion deficits caused by "choke zones," skin paddles must remain within zones of reliable vascularity, generally restricted to the area within 8–10 cm from the fibular head [5, 13, 15]. Surgical closure must be performed without tension, an especially critical precaution for patients with limited soft tissue redundancy or comorbidities that impair wound healing [11, 16]. Furthermore, tourniquet protocols should strictly adhere to evidence-based thresholds typically less than 120 min and the lowest effective pressure to effectively reduce the risk of reperfusion edema [13, 14]. In sedated or heavily medicated postoperative patients, pain is an unreliable indicator of evolving complications. Clinicians must instead rely on objective global signs, such as tense compartments, bullae, delayed capillary refill, or absent Doppler signals. When clinical suspicion is high, prompt decompression is mandatory and should not be delayed for compartment pressure measurements [7, 14, 17].

Strengths and limitations

The principal strength of this report is its detailed chronological account of the complication, which aligns closely with previously published mechanisms of ACS. This narrative enhances the external validity of the findings and provides valuable insight into a rarely documented sequence of events (the evolution from skin necrosis to ACS), enabling practitioners to recognize these crucial early warning signs. The report's limitations include the absence of objective compartment pressure data and delayed use of diagnostic imaging, which could have provided more quantitative support for clinical diagnosis. Additionally, long-term functional outcomes remain uncertain at the time of reporting.

Future Research

Prospective studies are needed to evaluate early markers of donor site ischemia, such as the use of intraoperative perfusion imaging or real-time postoperative monitoring with continuous oximetry. Comparative clinical trials focused on closure techniques (e.g., primary closure versus skin grafting) could definitively clarify which strategies best minimize donor site pressure. Moreover, establishing registries to capture rare complications like ACS would be highly valuable for quantifying the true incidence and establishing robust, evidence-based prevention protocols.

CONCLUSION

This case effectively demonstrates how donor-site necrosis can evolve into delayed acute compartment syndrome, particularly when compounded by factors like closure tension and prolonged tourniquet use.

Early recognition, multidisciplinary vigilance, and timely decompression are essential practices for preventing irreversible neuromuscular injury and optimizing limb salvage.

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USE OF AI TOOLS

The authors used ChatGPT (GPT-5.1) solely to improve language fluency (style, paraphrasing, and grammar). All scientific content, data interpretation, and conclusions were developed by the authors. The authors reviewed and edited the AI-assisted text and take full responsibility for the content of this publication.

ETHICS APPROVAL AND INFORMED CONSENT

This case report was prepared in accordance with the principles of the Declaration of Helsinki. Written informed consent was obtained from the patient for publication of the case details and accompanying clinical images.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

REFERENCES

1. Russell J, Pateman K, Batstone M. Donor site morbidity of composite free flaps in head and neck surgery: a systematic review of the prospective literature. *Int J Oral Maxillofac Surg* 2021;**50**(9):1147-55.
2. Sharma M, Mohammad A, Alam Parwaz M, Escandon JM. Donor-site Morbidity following Microvascular Fibular Transfer for Head and Neck Reconstruction. *Plast Reconstr Surg Glob Open* 2022;**10**(10S):88.
3. Chan TG, Rosado A, Goyal S, et al. Long-Term Postoperative Donor Site Musculoskeletal Morbidity after Osseous Free Flap Harvest. *OTO Open* 2025 Jan-Mar;**9**(1):e70069.
4. Rendenbach C, Kohlmeier C, Suling A, et al. Prospective biomechanical analysis of donor-site morbidity after fibula free flap. *J Craniomaxillofac Surg* 2016 Feb;**44**(2):155-9.
5. Topkara A, Ozkan A, Ozcan RH. The treatment of tissue defects related to the compartment syndrome occurring in the fibula osteocutaneous free flap donor area with vacuum assisted closure therapy. *Microsurgery* 2015;**35**(5):417-8.
6. Alahmadi DF, Halawani IR, Alghamdi AM, Shawosh MH, Althobaity AN. Late Donor-Site Complications Following Free Fibular Flap Harvest: A Report of a Rare Case. *Cureus* 2024 Aug 30;**16**(8):e68177.
7. Moon BM, Pae WS. Compartment syndrome as a donor-site complication of anterolateral thigh free flap: a case report. *Arch Hand Microsurg* 2022 Mar;**27**(1):83-7.
8. Zhao Y, Xiang D, Song L, et al. The clinical application and anatomical analysis of proximal peroneal artery perforator in free fibula flap based on CTA. *BMC Oral Health* 2025 Jan 16;**25**(1):77.
9. Nadeem W, Ferrell JK, Taylor CB. Peronea Magna: An Important Anatomic Variant Impacting Fibula-Free Flap Reconstruction. *OTO Open* 2024;**8**(1):e121.
10. Ershadifar S, Colback A, Basmaci UN, Wilson M, Birkeland AC, Silverman DA. Predictors of Donor-Site Wound Complications Following Fibula Free Flap Reconstruction. *OTO Open* 2025;**9**(2):e70126.
11. Berzofsky C, Shin E, Mashkevich G. Leg compartment syndrome after fibula free flap. *Otolaryngol Head Neck Surg* 2013 Jan;**148**(1):172-3.
12. Fodor L, Dinu C, Fodor M, Ciuce C. Severe compartment syndrome following fibula harvesting for mandible reconstruction. *Int J Oral Maxillofac Surg* 2011 Apr;**40**(4):443-5.
13. Hwang J, Kim K, Lee S. A Case of Nonisland Pedicled Foot Fillet Flap for Below-Knee Amputation Stump Wound: Treatment Option for Compartment Syndrome after Fibular Free Flap Surgery. *J Korean Med Sci* 2014 Feb;**29**(2):305-8.
14. Kerrary S, Schouman T, Cox A, Bertolus C, Febrer G, Bertrand JC. Acute compartment syndrome following fibula flap harvest for mandibular reconstruction. *J Craniomaxillofac Surg* 2011 Apr;**39**(3):206-8.
15. Saleem M, Hashim F, Babu Manohar M. Compartment syndrome in a free fibula osteocutaneous flap donor site. *Br J Plast Surg* 1998 Jul;**51**(5):405-7.
16. Sun G, Yang X, Wen J, Wang A, Hu Q, Tang E. Treatment of compartment syndrome in donor site of free fibula flap after mandibular reconstruction surgery. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2009 Nov;**108**(5):e15-8.
17. Wang J, Wang C. Osteofascial compartment

syndrome. *J Craniofac Surg* 2011 May;**22**(3):1100-2.
18. Rubino C, Faenza M, Muzzeddu GP, Massarelli O, Tullio A, Farace F. Compartment syndrome at the fibula flap's donor site and salvage by anterolateral thigh chimeric flap. *Microsurgery* 2012;**32**(8):657-8.
19. Wolff KD, Hölzle F, Kolk A, Hohlweg-Majert B, Steiner T, Kesting MR. Raising the osteocutaneous fibular flap for oral reconstruction with reduced tissue alteration. *J Oral Maxillofac Surg* 2011 Jun;**69**(6):e260-7.
20. de Lange JWD, Hundepool CA, Duraku LS, et al. Neuropathic pain at the donor-site following free fibula flap harvest: A multicenter study. *J Plast Reconstr Aesthet Surg* 2025;**102**:396-403.
21. Ishibashi S, Shinomiya R, Hayashi Y, et al. Factors Associated With Claw Toe Deformity Following Vascularized Fibula Flap Harvesting: A Retrospective Analysis of Potential Risk Factors and Preventive Strategies. *Microsurgery* 2025 Feb;**45**(2):e70031.
22. Kremer T, Bickert B, Germann G, Heitmann C, Sauerbier M. Outcome assessment after reconstruction of complex defects of the forearm and hand with osteocutaneous free flaps. *Plast Reconstr Surg* 2006;**118**(2):443-54.
23. Qureshi SS, Kembhavi S, Bhagat M, et al. Primary non-metastatic Ewing sarcoma of the jaw in children: Results of surgical resection and primary reconstruction. *J Surg Oncol* 2014;**110**(6):689-95.
24. Wei FC, Çelik N, Chen HC, Cheng MH, Huang WC. Combined anterolateral thigh flap and vascularized fibula osteoseptocutaneous flap in reconstruction of extensive composite mandibular defects. *Plast Reconstr Surg* 2002;**109**(1):45-52.
25. Namba Y, Kimata Y, Koshima I, Sugihara S, Sato T. Fibular osteoadiposal flap for treatment of tibial adamantinoma: a case report. *Acta Med Okayama* 2006 Aug;**60**(4):233-6.