Bladder Outlet Obstruction as a Cause for Late Total Flap Failure in Pelvic Reconstruction with a VRAM

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Abstract

Keywords

- ▶ pelvic reconstruction
- ▶ perineal reconstruction
- ▶ pelvic exenteration
- ▶ VRAM
- ▶ abdominoperineal resection
- ▶ flap failure

Background A 67-year-old man presented with abrupt failure of a pedicled vertical rectus abdominus myocutaneous (VRAM) flap 13 days postoperatively.

Methods The patient underwent pelvic reconstruction with a pedicled VRAM flap following sacral chordoma and abdominoperineal resection. The flap remained well perfused and viable until postoperative day 13, at which point the patient was noted to become systemically unwell with fever, chills, and abdominal pain. This clinically coincided with prompt arterial and venous insufficiency of the VRAM flap.

Results Computed tomography of the abdomen was ordered to rule out a pelvic collection and revealed an inflated Foley catheter in the bulbar urethra. This was associated with marked distention of the bladder and bilateral hydronephrosis. Direct compression of the deep inferior epigastric pedicle by the bladder neck was noted.

Conclusion The case highlights the importance of considering bladder outlet obstruction and subsequent distention as a cause of pedicle compression and VRAM flap failure following pelvic reconstruction.

Pelvic reconstruction following abdominoperineal resection and pelvic exenteration remains a significant challenge. Perineal wound complications occur in up to 60%^{1–3} of patients with primary closure. The high morbidity associated with primary closure is attributable to the large pelvic dead space, which can lead to fluid collection and superinfection, the high tension skin closure, and the presence of irradiated and poorly vascularized tissues.^{4–6} The vertical rectus abdominus myocutaneous (VRAM) flap has emerged as the workhorse flap in pelvic reconstruction, associated with significant reductions in perineal morbidity compared to primary closure alone.⁷ The advantages of flap reconstruction in this context are the obliteration of pelvic dead space, interposition of well-vascularized tissue into an irradiated wound bed, a decrease in closure tension, and resistance of infection.^{8–12} Despite the purported benefits, the VRAM flap is associated with the inherent risks of any pedicled flap reconstruction, namely, donor site morbidity and the risk of partial or complete flap failure. The rate of complete flap loss following VRAM-based pelvic reconstruction is less than

5%,^{13–17} with the vast majority of failures occurring within the first 72 hours. The rates and etiology of late flap failure, or those occurring after postoperative day 7, however, are poorly elucidated. Herein, we propose one such mechanism of late flap failure, occurring 13 days postoperatively due to pedicle compression secondary to bladder distention. To the best of our knowledge, this is the first reported case of VRAM flap loss via this mechanism.

Case Report

A 67-year-old man with a history of multiple cerebrovascular events, a myocardial infarction, hypertension, and a 30 pack-year smoking history presented with dull 5/10 sacral pain, prompting a pelvic magnetic resonance imaging (MRI) examination. Imaging revealed a large, well circumscribed, heterogeneously enhancing mass through the mid body of the sacrum. Subsequent core biopsy demonstrated a sacral chordoma.

The patient underwent preoperative external beam radiation, followed by sacrectomy and abdominoperineal resection.

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Fig. 1 Intraoperative photograph.

The colostomy was exteriorized through the left rectus abdominus muscle. The resultant pelvic and perineal defect was reconstructed using a right VRAM flap (►Fig. 1).

The patient did well postoperatively. Following an unremarkable 5-day admission to the intensive care unit, the

patient was transferred to the orthopedic surgery unit. Regular flap checks continued to be performed and the flap was consistently well perfused. On postoperative day 13, the patient became systemically unwell with fever, tachycardia, and significant abdominal discomfort. Laboratory investigations demonstrated an increase in white blood cell count and a septic workup was initiated. During this time, the flap demonstrated signs of both venous and arterial insufficiency (►Fig. 2). Computed tomography of the pelvis was ordered to assess for intrapelvic collection and revealed a markedly distended bladder with bilateral hydronephrosis. The deep inferior epigastric pedicle could be visualized and was noted to be occluded by the distended bladder, preventing arterial inflow and venous outflow (►Fig. 3). The bladder was promptly decompressed, resulting in improvement in the patient's clinical status. The perfusion to the flap, however, did not improve, ultimately resulting in total flap loss.

The patient was brought back to the operating room 3 weeks postoperatively for debridement of the VRAM flap. Significant thrombosis of the deep inferior epigastric pedicle could be appreciated in the context of a frankly necrotic flap. Following flap debridement, bilateral advancement flaps were elevated for closure. The patient was discharged from hospital postoperative day 40 and the wound was completely healed by postoperative day 60.

Fig. 2 (A) Postoperative day 13. (B) Postoperative day 14.

Fig. 3 (A and B) Computed tomography of pelvis showing markedly distended bladder compressing VRAM pedicle. VRAM, vertical rectus abdominus myocutaneous.

Discussion

Flap failure in either free or pedicled flap reconstruction typically occurs within the first 72 hours. This is often the result of intrinsic pedicle thrombosis (arterial or venous) or extrinsic compression, eventually leading to thrombosis. In the immediate postoperative period, traction or kinking of the pedicle is the most likely culprit for flap failure. The incidence of and mechanisms for late flap failure, however, remain poorly understood. The described case demonstrates clearly that pedicle compression from a distended bladder is a potential mechanism for late flap failure and should be considered on the differential when assessing a flap with clinical signs of vascular compromise. Furthermore, it illustrates the utility of cross-sectional imaging in helping to elucidate the mechanisms of late flap failure due to pedicle compression.

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Conflict of Interest

None.

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