Systemic Nicardipine as an Adjunct to Combat Vasospasm after Prior Flap Failure

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Summary: Unrecognized or untreated vasospasm in microsurgery can lead to flap hypoperfusion and failure. Numerous strategies have been explored for their efficacy in potentiating vasodilation. We present a case of unrecognized vasospasm leading to flap failure followed by a second free flap reconstruction in which severe vasospasm was treated with systemic nicardipine used as an adjunct to other more commonly employed antispasmodics. Although the literature investigating the use of systemic calcium channel blockade in microsurgery is limited, it should be considered an alternative when addressing arterial vasospasm. (Plast Reconstr Surg Glob Open 2013;1:e54; doi:10.1097/GOX.0b013e3182aa8747; Published online 18 October 2013.)

CASE REPORT

The patient is a 58-year-old white man with a history of a facial basal cell carcinoma for which he underwent resection resulting in a right hemirhinectomy, loss of medial cheek, right upper lip, and the anterior portion of the right maxillary sinus including intrasinus mucosa (Fig. 1). We recommended a staged approach with initial free flap reconstruction followed by nasal reconstruction.

Due to radial-dominant blood flow to his left, nondominant hand, and his profession of dismantling and rebuilding tractors, we avoided the free radial forearm flap, and instead, we chose the rectus abdominis muscle flap, due to its overall tissue profile and reasonable pedicle length.

Using the facial artery and vein as recipient vessels, a rectus abdominis free muscle flap, based on properties. When administered systemically, there is a theoretical risk of hypotension and hypoperfusion; however, there also exists the benefit of blocking calcium-dependent platelet aggregation and potentially reducing the risk of thrombus formation.5

Other techniques for free flap salvage have been described, such as administration of continuous intravenous heparin, use of a Fogarty catheter, flap washing with streptokinase, and adventitial stripping.5

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the deep inferior epigastric artery and associated venae, was raised and transferred to the face for microsurgical anastomosis. End-to-end anastomosis was performed from facial vein to flap vein using a 2.5-mm coupler. End-to-end anastomosis of the facial artery to flap artery was performed using a 9-0 nylon interrupted sutures. Clamps were removed with noted bleeding from the flap surface, appropriate color change, and adequate tissue turgor.

Following approximately 10 minutes, the flap lost arterial perfusion. Anastomoses were inspected, topical lidocaine 2% was applied, and an Acland test noted excellent venous flow past the anastomosis. No significant flow was noted distal to the arterial anastomosis, and for this reason, the anastomosis was resected and revised. Repeat end-to-end anastomosis was performed with initial reperfusion followed by cessation of the arterial flow. The subcutaneous tunnel encompassing the vascular pedicle was again inspected with no noted compression points or tension on the vascular pedicle. The assumption was made that intraluminal thrombosis was occurring within the deep inferior epigastric artery of the flap.

The decision was made to attempt venous bypass grafting. Despite extensive dissection in the medial left thigh, the saphenous vein was not identified. Intraoperative ultrasound revealed that the saphenous vein was not present, and therefore, this wound was closed without successful vein graft harvest.

After the fourth anastomotic revision, with no significant thrombus detected, ischemic time of the muscle flap had been approximately 6 hours, and therefore, the flap was discarded.

The patient agreed to return to the operating room to attempt an anterolateral thigh flap reconstruction of the facial defect. Arterial and venous anastomoses were performed without difficulty. The flap became reperfused initially with subsequent loss of arterial perfusion. An Acland test demonstrated no arterial flow; therefore, the anastomosis was resected, revealing minor fibrinous debris. After anastomotic revision, we noticed significant vasospasm of both the superior thyroid and flap arteries. At this time, a 5000 U intravenous bolus of heparin was given followed by a continuous infusion of 750 units per hour.

We then used a 2-mm Fogarty catheter threaded proximally to the base of the superior thyroid artery with subsequent dilation and pulsatile bleeding upon withdrawal of the catheter. The Fogarty catheter was then passed into distal end of the visible portion of the flap artery, inflated, and retracted dilating that segment as well.

At this point, the patient was started on a systemic nicardipine infusion in an attempt to minimize any further vasospasm. The flap remained perfused for the remainder of the case, and the nicardipine infusion was maintained for 48 hours postoperatively without resulting in hypotension. There were no further anastomotic complications, and the patient was discharged postoperative day 4 (Fig. 2).
DISCUSSION

Both in vitro and in vivo studies have demonstrated the efficacy of antispasmodic agents and, in particular, the superiority of papaverine and calcium channel blockers over lidocaine. Topical administration of calcitonin gene-related peptide has also been successful in increasing flap perfusion in mechanically induced vasospasm, although this agent has not achieved the widespread clinical acceptance of the others described.7,8

When faced with flap failure due to arterial hypoperfusion, there are other alternatives to pharmacologic vasodilation, including the use of a Fogarty catheter to disrupt vasospasm. In this case, we lost a free rectus muscle flap likely secondary to under-appreciated vasospasm. The absence of papaverine at our institution and the possible dose-dependent vasoconstrictive effect of 2% lidocaine may have contributed to vasospasm-related hypoperfusion and eventual flap loss. Because arterial anastomotic failures are most commonly due to technique, multiple attempts were made to revise the anastomosis, each time with an initial period of perfusion followed by eventual slowing and ultimately cessation of blood flow.

We remind readers of several options for treating anastomotic vasospasm including anastomotic revision, adventitial stripping, thrombectomy, vein grafting, topical pharmacologic antispasmodic agents, Fogarty catheter-driven spasmylosis, and systemic calcium channel blockade.

CONCLUSIONS

Although not well studied in microsurgery, systemic calcium channel blockade should be considered an alternative when treating arterial vasospasm. The initial flap in this scenario may have been salvaged if the magnitude of vasospasm had been appreciated sooner and appropriate measures taken to combat these effects.

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