 DOI: 10.18231/2455-846X.2016.0009

Salvage of free radial artery forearm flap following reconstruction of lower lateral lip: A case report

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Abstract

The increasing success rate of free flaps along with their reconstructive advantages have got them into regular practice options in Maxillofacial reconstruction. Although associated complications are inevitable in a percentage of patients, it is good postoperative care and monitoring that determines the success or failure of the reconstruction. With early recognition and prompt intervention salvage of compromised flap is possible. The purpose of this article is to emphasise the importance of keeping low threshold for re- exploration in situation of any suspected failing free flap.

Keywords: Free Radial Artery Forearm Flap (FRAFF), Squamous cell carcinoma (SCC), Supra Omohyoid Neck Dissection (SOND), Lymph Nodes (LN), Internal Jugular Vein (IJV)

Pt Report

A 60 year-old male patient presented with an ulcero-proliferative mass measuring around 4cmx3.5 cm which involved the right lip mucosa and had infiltrated the underlying tissue to appear over the skin with central zone of necrosis and surrounding erythematous induration. (Fig. 1) Incisional biopsy from mucosal surface of the lesion revealed well to moderately differentiated squamous cell carcinoma.

Fig. 1: Ulcero-proliferative mass infiltrating the right lip mucosa and underlying tissue with central zone of necrosis and surrounding erythematous induration

The neck was clinically N0 which similarly correlated following contrast CT scan of the neck. The surgical planning for the tumour was wide excision, SOND (Supra Omohyoid neck dissection) and reconstruction of the defect with FRAFF. Wide excision of the primary lesion was carried out along with the SOND on the affected side.

The neck dissection was done with extreme care to preserve the facial vessels when dissecting level IB. Dissection of the submandibular gland was done by safely ligating the submandibular branches of the facial artery to the gland and preserving the whole artery along its course on the posterior aspect of the gland. The right lip commissure involving lateral one third of the lower lip, part of skin in the cheek along with underlying part of cheek musculature, inner buccal and labial mucosa, gingivo-buccal sulcus and gingiva along with the alveolar bone sparing the lower border of the mandible were resected. The size of the primary defect following tumour resection was 5.2x4.8x3.7 cm. The reconstruction of the surgical defect was done with FRAFF. On completion of the reconstruction the flap looked well perfused and healthy. The flap was monitored clinically at an hourly basis. By 4^{th} -5^{th} hours mild ooze from the drain port had become noticeable along with marked oedema in the flap. (Fig. 2) On the 8^{th} hour post operatively there was dusky appearance of the flap, along with oedema. On needle prick there was dark blood ooze from the flap and therefore the decision for re exploration was taken suspecting venous compromise. On re exploration a huge hematoma was noticed under the flap, around the anastomosis and along the IJV. The clots were delicately removed. The adjoining areas were irrigated with warm saline, and venous and arterial anastomoses were evaluated. There were no clots in the vein and normal flow through the artery was seen. Acland's "milking" test was performed. In the test the artery was occluded with a forceps immediately caudal to anastomosis and then with a second forceps artery was occluded distal to the first one. The second forceps was slid over the artery in the direction of the blood flow. Keeping the second forceps closed and release of the first forceps caused instant filling of the emptied segment of the artery thus establishing patent anastomosis. Warm mops were applied to the flap and bleeding points at the flap periphery were evaluated. One to two points of slow oozes were cauterised with bipolar. As the flow through the anastomosis was satisfactory. Flap was re-

inset after establishing a drain. The flap was monitored on regular basis for another week and the recovery was uneventful (Fig. 3).

Fig. 2: 8th hour post operatively, dusky appearance of the flap along with edema

Fig. 3: Post-operative, after re-exploration & removal of hematoma

The histological examination of the surgical specimen reported as keratinising squamous cell carcinoma, grade I of lower right lip. All cervical LNs were negative except one positive LN from level 1B. Salivary gland was free of tumour. There was also perineural invasion. The subject was given radiotherapy sessions and followed. The lip incompetency was planned to be managed with palmaris longus tendon or Fascia Lata sling following completion of radiotherapy.

Discussion

Free flap success rates in most high volume centres are in excess of 95%.[1–6] Majority of flap failures occur within the first 48 hours. Venous thrombosis is more than twice as common as arterial thrombosis among causes of flap failures due to pedicle thrombosis.[6] Compression due to hematoma was the cause for vascular compromise and impending flap failure in our subject. Hidalgo et al[4] identified venous problems (35%) as the most common etiology of flap failure followed by arterial problems (28%), hematoma (26%) and recipient vessel problems (11%). Intravenous Heparin was given during the anastomosis along with the plasma expander. Drawback of intravenous heparin use is the potential for bleeding and hematoma formation. Brown et al[7] reviewed 427 free tissue transfers with 16% requiring return to the operating theatre within seven days for compromised flap or hematoma. Venous compromise (83%) was once again much higher than arterial compromise (8%).[7] Extrinsic compression of the vascular pedicle by tight wound closure, tapes around the neck or wound hematoma may also compromise the flap by obstruction of venous outflow.[8] Late Flap failure were due to infection or mechanical stress around the anastomosis.[4]

The success of microvascular salvage follows an inverse relation to the time between onset of flap ischemia and clinical recognition.[9] The secondary critical ischemia time is the critical period after postoperative flap compromise in which it is still possible to restore flap circulation.[10] If circulation to a failing flap is not re-established within 8 to 12 hours, salvage may become impossible due to development of the no-reflow phenomenon[11] where restoration of blood flow results in initial hyperaemia followed by a gradual decline in perfusion. Reperfusion injury mediated by oxygen free radicals, the complement system, and neutrophils also leads to cellular death and necrosis.[12]

The first step in managing free flap failure is early recognition of a compromised flap. Post operatively, flap monitoring is done at hourly interval for 48 hours, four hourly for the next 120 hours, and daily until discharge. Clinical observation remains the simplest method of identifying vascular compromise.[13] Clinical evaluation by an experience microsurgeon is considered the gold standard for perfusion assessment. To be effective, changes in perfusion need to be recognized quickly to correct any treatable problems. Disruption of perfusion to a flap can result in partial or complete tissue loss.

Important physical signs to observe during flap monitoring include the colour of the flap, temperature, bleeding from the cut edge of transferred tissue, and tissue turgor. Signs of a congested (venous compromised) flap include a bluish color, increased swelling and shortened capillary refill (<2 seconds). In the case of ischemia (arterial compromised flap), the flap will be pale in color, cold, and will have a delayed capillary refill (>3 seconds). If no bleeding is noted, arterial occlusion is implied. If brisk, dark bleeding occurs, venous insufficiency should be considered. Stabbing with a needle or scalpel yields similar information. Several different monitoring devices are in use now for flap monitoring which include: Surface or pencil Doppler, Temperature probe, Laser Doppler probe, Quantitative fluorometer, Implantable Doppler,

near infra-red spectroscopy, Qualitative indocyanine green.

Successful salvage rates range from 28% to over 90%.[3,4,7,14,15] The rates vary depending upon the etiology, timing of salvage and experience of the centre. In a review of 150 cases Hidalgo[8] suggested that attempted salvage of compromised flaps significantly increases flap survival rates and recommended an aggressive approach towards early exploration. In Brown's series[7] 73% of failed free flaps were successfully salvaged. Most of these were within 24 hours of initial operation and salvage rates were significantly higher for radial forearm than for composite flaps.

Hidalgo et al[4] in their series of 716 free flaps reported an eight percent re exploration rate for vascular compromise with a successful salvage rate of 70%. Flap loss was much higher in buried flaps (7%) compared with non-buried flaps (2%) with a longer time to re-exploration in the buried group due to unreliable flap monitoring. Salvage rates with late exploration are generally poor. Mean re-exploration time for salvage cases was 1.3 days compared with 3.9 days for those not salvaged.[16]

Kind et al[17] suggested that a miniature doppler ultrasonic probe attached directly to the outflow vein of the flap may lead to a significant improvement in the salvage rates of free flaps. They identified 20 instances of vascular compromise in 147 free flaps using this technique with a salvage rate of 100%.

Suspicion of vascular compromise one should have a low threshold for return to the operating room for re-exploration.[7] Kubo et al[18] reviewed the management of the flap with venous compromise and suggested that surgical methods should be the first choice as it offers significantly higher salvage rates.

With re-exploration initial attention should be directed at the vascular pedicle. Causes of extrinsic compression such as hematoma, pedicle kinking or misconfiguration are easily identifiable and potentially correctable. The internal jugular vein should also be examined for possible thrombosis[18] the arterial system should be examined under magnification for vascular spasm, for which topical Papaverine may be used. Identification of thrombus should prompt opening the anastomosis and evacuation of the clot with heparinised saline irrigation or a Fogarty catheter prior to careful re anastomosis.[19]

Thrombolytic agents, such as streptokinase, urokinase or tissue plasminogen activator, can be used if a thrombus is identified, particularly in the venous system. Their use has been well documented as a means to salvage vascular insufficiency and theoretically prevent irreversible ischemic reperfusion injury or no reflow phenomenon.[18,20] To avoid systemic effect of these agents subjects should be evaluated on individual basis for possible adverse outcomes.

If thrombosis occurs at the time of re anastomosis the initial recipient vein and/or artery may not be appropriate, in which case another should be chosen.[21]

Dabb[22] described several successful cases of venous congested flaps salvaged by leeches, suggesting that relief of congestion for four to ten days may allow enough time for neo-vascularisation. Neo-vascularization has been reported to occur as early as six days.[23] Leech therapy is primarily used in the management of venous congestion of flaps with a cutaneous portion used for external head and neck skin coverage.

Technical advances in the field of free tissue transfer and increase success rate are making free tissue transfers as a most sought-after options for reconstruction of oral and maxillofacial defects. The success of free tissue transfer relies on good surgical technique and vigilant post-operative monitoring and therefore the team should have low threshold for re-exploration in cases of failing flaps.

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