Simon Kelley MRCS, Senior House Officer in Plastic Surgery

176 Stainbeck Lane, Leeds LS7 2EA, UK

Simon P.J. Kay FRCS, FRCS(Plast), Consultant Plastic Surgeon

Department of Plastic, Reconstructive and Hand Surgery, St James's University Hospital, Beckett Street, Leeds LS9 7TF, UK

Correspondence to Dr Douglas Ross, St Joseph's Health Care, 268 Grosvenor Street, London, Ont. N6A 4L6, Canada

Paper received 26 September 2002.
Accepted 19 November 2002.

British Journal of Plastic Surgery (2003), **56**

doi:10.1016/S0007-1226(02)00480-0

Late salvage of a free TRAM flap

R. Tse, D. Ross and B. S. Gan

Division of Plastic and Reconstructive Surgery, Department of Surgery, University of Western Ontario, London, Ont., Canada

SUMMARY. We present the case of a patient with arterial thrombosis of a free TRAM flap 11 days after surgery. Initial salvage involved thrombectomy through an arteriotomy using a Fogarty catheter. Subsequent return to the operating theatre was necessary because of further vascular compromise. Thrombectomy was combined with flap thrombolysis and anticoagulation, and the flap remained viable at 6 months. Although rates of successful salvage vary, the literature indicates that flaps are rarely saved if signs of compromise present later than 2 days postoperatively. This case demonstrates that aggressive salvage may be successful even in cases of 'late' vascular compromise. © 2003 The British Association of Plastics Surgeons. Published by Elsevier Science Ltd. All rights reserved.

Keywords: microsurgery, TRAM flap, thrombosis, salvage.

Although microvascular free tissue transfer has gained acceptance as a reliable reconstructive technique, with reported success rates of between 85 and 98%,[1–7] flaps continue to be lost to vascular compromise. Re-exploration rates as high as 6–15% have been reported,[1–6,8] with successful salvage in between 19 and 100% of cases.[1–4,6,8] Early recognition of compromise and urgent re-exploration are imperative for success, and the majority of successful salvages occur within the first 12–24 h postoperatively.[6,7,9,10] Weinzweig and Gonzalez describe 10 cases of progressive failure in six flaps later than the second postoperative day.[11] Tsai et al report an unsuccessful salvage attempt 4 days after primary surgery,[5] and Kroll et al report unsuccessful salvage of 10 flaps after 2 days.[7] Betancourt et al examined the timing of critical thrombosis after digital replantation: none of the 21 digits with critical thrombosis beyond 3 days after replantation survived.[10] The latest salvage of a digital replantation, to our knowledge, was that performed by Parkhouse and Smith 19 days postoperatively.[12] A review of the literature failed to find a case of successful free flap salvage more than 6 days postoperatively: Hidalgo and Jones report a recipient artery atherosclerotic obstruction that was reversed on the sixth postoperative day,[6] and Serletti et al report successful reversals of venous thromboses in two flaps on the fifth and sixth postoperative days, respectively.[13] We report another case of 'late' salvage of a free tissue transfer, 11 days postoperatively.

Case report

A 27-year-old female underwent a right modified mastectomy for infiltrating ductal carcinoma and a left simple mastectomy for prophylaxis. Immediate bilateral free TRAM flap reconstruction was performed using the subscapular vascular tree as the recipient vessels; the ischaemia time was less than 2 h.

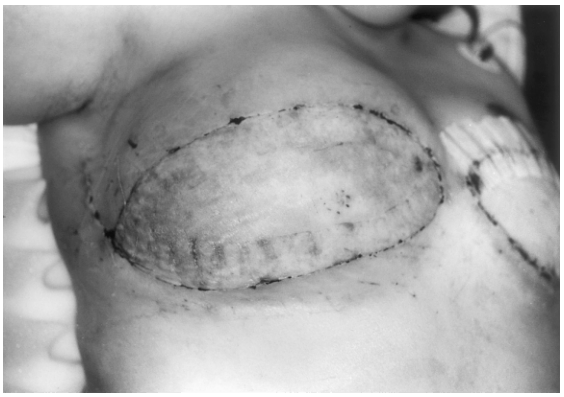

Figure 1—Appearance of the flap at presentation 11 days postoperatively, showing vascular compromise.

Shortly after releasing the microvascular clamps, we observed bilateral arterial thrombosis, which required revision of the arterial anastomoses, with the removal of platelet plugs from the arterial anastomotic sites and flushing with heparinised (10 U ml^{-1}) Ringer's lactate solution. A 5000 U intravenous bolus of heparin and 30 mg of intravenous ketorolac were given during the revision, and a drip of 10% dextran-40 solution with 10 U ml^{-1} of heparin at 20 ml h^{-1} was started. No further anastomotic problems occurred, and the flaps were subsequently well perfused. Laser Doppler was used for postoperative monitoring. The patient was started on 325 mg of aspirin daily, and the dextran infusion was continued for 5 days. The patient was discharged on no medication after an uneventful 7 days in hospital.

On the 11th day after surgery, the patient presented urgently to clinic with a cool violaceous right flap (Fig. 1). Warm ischaemia time was estimated to be within 5–6 h. She denied any trauma, and inquiry revealed only that she had been smoking during the preceding 3 days.

The patient was immediately brought to the operating theatre, where the right axilla was re-opened and the pedicle was carefully dissected. The venous anastomosis was patent, but the arterial anastomosis had thrombosed, with proximal extension to the axillary artery. The clot was removed using a Fogarty catheter passed through an arteriotomy, the anastomosis was redone and the patient was given a bolus of 3000 U of intravenous heparin. Unfortunately, shortly after repair, the thrombus recurred, despite systemic anticoagulation, and a second arteriotomy and thrombectomy was required. Patency was observed, and the pedicle was irrigated with 2% lignocaine prior to wound closure. The procedure lasted 5 h. A dextran-heparin infusion was started, and 325 mg of aspirin was given (Fig. 2).

Poor perfusion of the same flap was detected by laser Doppler 6 h postoperatively, and the patient was again returned to the operating theatre. Secondary re-exploration and thrombectomy was performed. Interestingly, the pedicle was patent, but the appearance of the flap remained unsatisfactory. A total of 30,000 U streptokinase was infused into the flap arterial system via a 30 G needle inserted proximal to the anastomosis. At the same time, the pedicle was filled with heparinised Ringer's lactate and clamped. Adequate bleeding was demonstrated shortly afterwards, and the wound was closed once again. The procedure lasted 1.5 h. The patient was fully anticoagulated using an intravenous heparin drip (1000 U h^{-1}) and restarted on 325 mg of aspirin daily. After an uneventful 8 days stay in hospital, she was discharged with a 6 week course of coumadin anticoagulation.

One year after salvage, the patient has suffered partial necrosis of the lateral aspect of the right flap (Fig. 3) and approximately 20% loss of flap volume. The flap has remained otherwise well, with good wound healing despite chemotherapy (Fig. 4). The patient has recently been diagnosed with pulmonary metastatic disease.

Discussion

Free flap failure is generally recognised as an 'early' complication, occurring within 4–5 days of surgery. Causes are thought to include anastomotic flaws, side-branch injury, vessel spasm, pedicle kinking, haematoma and tight wound closure.[3–5,9] Weinzweig and Gonzalez describe a case series where vascular compromise occurred as late as 6 weeks postoperatively.[11] Duffy et al have also described two cases of digital-replantation failure occurring 14 and 16 days, respectively, after surgery.[14] This case adds to the literature describing late vascular compromise as a complication of free tissue transfer.

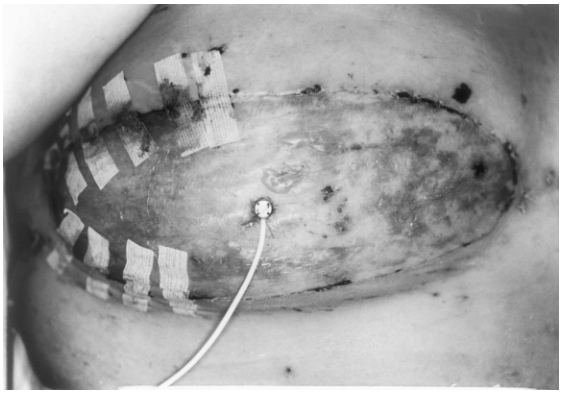

Figure 2—Appearance of the flap after the initial salvage operation.

Through the work of Acland et al, a model of microsurgical thrombosis has been described in which the anastomotic site (zone I) and the downstream microvasculature (zone II) have distinct pathophysiology.[15–17] Primary thrombosis in zone I has been vigorously studied, with the focus on vessel-wall injury, subendothelial exposure, platelet adhesion and activation, suture material, flow disturbances and vessel spasm.[18] Previous surgery and the use of vein grafts have been identified as risk factors for failure.[2] Other presumed factors, including extremes of age, radiotherapy, a history of smoking or alcoholism, high ASA grade and ablative surgery, remain disputed.[2,3] Secondary thrombosis in zone II is not as well recognised and studied. Hypoperfusion is thought to occur as a result of either embolic showering from the anastomosis or vasospasm secondary to the release of platelet metabolic products, such as thromboxane A2.[17] Cases thought to be consistent with the latter mechanism have been described as progressive partial free flap failure and occur between 4 h and 6 weeks after surgery.[11] Identified risk factors include delayed reconstruction of traumatic extremity wounds, established infection, use of vein grafts,

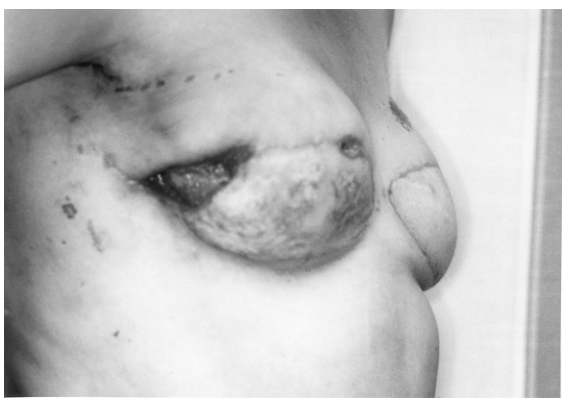

Figure 3—Mild necrosis of the lateral flap margin was treated with serial debridement.

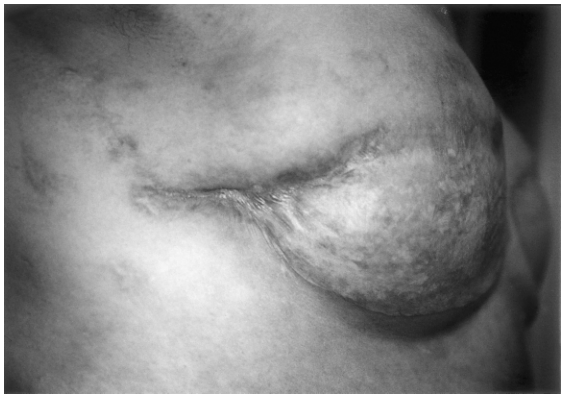

Figure 4—Appearance of the flap following serial debridement, chemotherapy and wound healing.

refractory vasospasm, hypercoagulable states and intravenous drug use.

The pathophysiology of late arterial thrombosis is not understood, and the aetiology of the vascular compromise in our case is unknown. On initial exploration of the flap, no extrinsic factor was identified, and venous outflow was patent. Given that the flap remained perfused for 11 days postoperatively, thrombosis is unlikely to have been caused by a technical error. The patient gave a history of smoking several days before presentation. Although the role of tobacco in this setting remains unclear, smoking could have induced vasospasm, consistent with the findings of Betancourt et al, in which smoking was associated with late thrombosis of a digital replantation.[10] Smoking has been shown to cause tissue ischaemia by vasoconstriction, carbon monoxide poisoning and increased carboxyhaemoglobin, and has been associated with platelet dysfunction. There was no evidence of active infection, but subclinical microbial contamination cannot be ruled out.

The flap was successfully perfused following zone I thrombectomy. On subsequent re-exploration, instigated by a decrease in the Doppler monitoring signal, however, the flap remained compromised despite a patent pedicle. That perfusion was only established after streptokinase thrombolysis of the flap microvasculature is consistent with the concept of secondary zone II thrombosis from proximal clot showering and platelet-mediated vasospasm. In retrospect, a hypercoagulability screen could have helped establish the reason for recurrent thrombosis. None the less, the flap was salvaged by removing the thrombus from zone I, thrombolysis in zone II and maintenance of anticoagulation, postoperatively. A combination of modalities was required for correction.

The finding of pulmonary metastatic disease 1 year after her initial surgery indicates persistent disease. A malignancy may lead to unexplained hypercoagulability.

Regardless of the pathophysiology of flap failure, several management options are available after 11 days. Flap survival via neovascularisation has been described following pedicle division 10 days after tissue transfer,[19–22] so conservative management of the flap was a possibility. Weinzweig and Gonzalez[11] and Duffy et al[14] used this approach in cases of late vascular compromise, achieving partial flap survival. However, in our case complete flap necrosis seemed imminent. More aggressive management involves surgical re-exploration of the pedicle and revision of the anastomosis. Wheatley and Meltzer report using Fogarty catheter thrombectomy successfully in six out of seven cases and with partial salvage in the seventh case.[9] Other approaches use pharmacologic agents, including heparin,[8,23] streptokinase,[12,13,24] urokinase,[25] recombinant tissue plasminogen activator (rt-PA)[26] and prostacyclin.[27] These may be administered systemically or infused to achieve high local concentrations while avoiding systemic complications. Repeated thrombectomy in our case eventually achieved vessel patency despite initial recurrence of thrombosis. The subsequent use of streptokinase thrombolysis and prolonged anticoagulation maintained flap viability with no evidence of significant haematoma formation. In contrast, Wheatley and Meltzer reported that in three out of six cases thrombectomy combined with anticoagulation resulted in a clinically significant haematoma requiring drainage.[9]

Although a multitude of techniques and pharmacologic agents are available to salvage flaps, rapid recognition and re-exploration is widely recognised as a major factor in successful salvage. Hidalgo and Jones achieved a 100% salvage success rate by returning to the operating theatre an average of 1.5 h after identifying compromise.[6] Animal models of secondary ischaemia have demonstrated that survival decreases with time.[28] A period of 7.2 h was found to be associated with a 50% rate of complete flap failure. Although in our case reoperation did not occur until 6 h after the first discovery of compromise, neovascularisation during the 11 days following surgery is likely to have been an important factor in tissue tolerance to ischaemia.

This is one of the few reports of successful salvage of late free tissue transfer compromise. The clinical course was consistent with the phenomenon of secondary thrombosis, and aggressive salvage was achieved without significant haematoma formation. Although, in a review of 990 consecutive free flaps, Kroll et al report that no salvage was achieved at later than 2 days,[7] we report successful salvage on the eleventh postoperative day. Aggressive attempts at free flap salvage may be successful despite a prolonged interval between the primary procedure and flap failure.

References

1. Irons GB, Wood MB, Schmitt EH. Experience with one hundred consecutive free flaps. Ann Plast Surg 1987;18:17–23.
2. Schusterman MA, Miller MJ, Reece GP, Kroll SS, Marchi M, Goepfert H. A single center's experience with 308 free flaps for repair of head and neck cancer. Plast Reconstr Surg 1994;93:472–80.
3. Urken ML, Weinberg H, Buchbinder D, et al. Microvascular free flaps in head and neck reconstruction. Arch Otolaryngol Head Neck Surg 1994;120:633–40.
4. Harashina T. Analysis of 200 free flaps. Br J Plast Surg 1988;41:33–6.
5. Tsai TM, Bennett DL, Pederson WC, Matiko J. Complications and vascular salvage of free-tissue transfers to the extremities. Plast Reconstr Surg 1988;82:1022–6.
6. Hidalgo DA, Jones CS. The role of emergent exploration in free-tissue transfer: a review of 150 consecutive cases. Plast Reconstr Surg 1990;86:492–8.

7. Kroll SS, Schusterman MA, Reece GP, et al. Timing of pedicle thrombosis and flap loss after free-tissue transfer. Plast Reconstr Surg 1996;98:1230–3.
8. Yajima H, Tamai S, Mizumoto S, Ono H, Fukui A. Vascular complications of vascularized composite tissue transfer: outcome and salvage techniques. Microsurgery 1993;14:473–8.
9. Wheatley MJ, Meltzer TR. The role of vascular pedicle thrombectomy in the management of compromised free tissue transfers. Ann Plast Surg 1996;36:360–4.
10. Betancourt FM, Mah ET, McCabe SJ. Timing of critical thrombosis after replantation surgery of the digits. J Reconstr Microsurg 1998;14:313–6.
11. Weinzweig N, Gonzalez M. Free tissue failure is not an all-or-none phenomenon. Plast Reconstr Surg 1995;96:648–60.
12. Parkhouse N, Smith PJ. The use of streptokinase in replant salvage. J Hand Surg 1991;16B:53–5.
13. Serletti JM, Moran SL, Orlando GS, O'Connor T, Herrera HR. Urokinase protocol for free-flap salvage following prolonged venous thrombosis. Plast Reconstr Surg 1998;102:1947–53.
14. Duffy FJ, Concannon MJ, Gan BS, May JW. Late digital replantation failure: pathophysiology and risk factors. Ann Plast Surg 1998;40:538–41.
15. Acland RD, Anderson G, Siemionow M, McCabe S. Direct in vivo observations of embolic events in the microcirculation distal to a small-vessel anastomosis. Plast Reconstr Surg 1989;84:280–9.
16. Barker JH, Acland RD, Anderson GL, Patel J. Microcirculatory disturbances following the passage of emboli in an experimental free-flap model. Plast Reconstr Surg 1992;90:95–102.
17. Barker JH, Gu JM, Anderson GL, et al. The effects of heparin and dietary fish oil on embolic events and the microcirculation downstream from a small artery repair. Plast Reconstr Surg 1993;91:335–43.
18. Johnson PC, Baker JH. Thrombosis and antithrombotic therapy in microvascular surgery. Clin Plast Surg 1992;19:799–807.
19. Serafin D, Shearin JC, Georgiade G. The vascularization of free flaps. Plast Reconstr Surg 1977;60:233–41.
20. Khoo CTK, Bailey BN. The behavior of free muscle and musculocutaneous flaps after early loss of axial blood supply. Br J Plast Surg 1982;35:43–6.
21. Nakajima T. How soon do venous drainage channels develop at the periphery of a free flap? A study in rats. Br J Plast Surg 1978;31:300–8.
22. Black MJM, Chait L, O'Brien BM, et al. How soon may the axial vessels of a surviving free flap be safely ligated: a study in pigs. Br J Plast Surg 1978;31:295–9.
23. May JW, Rothkopf DM. Salvage of a failing microvascular free muscle flap by direct continuous intravascular infusion of heparin: a case report. Plast Reconstr Surg 1989;83:1045–8.
24. Schubert W, Hunter DW, Guzman-Stein G, et al. Use of streptokinase for the salvage of a free flap: case report and review of the use of thrombolytic therapy. Microsurgery 1987;8:117–21.
25. Lipton HA, Jupiter JB. Streptokinase salvage of a free-tissue transfer: case report and review of the literature. Plast Reconstr Surg 1987;79:977–81.
26. Stassen JM, Lü G, Andréen O, Nyström E, Nyström Å. Intraoperative thrombolytic treatment of microarterial occlusion by selective rt-PA infusion. Plast Reconstr Surg 1995;96:1215–7.
27. Renaud F, Succo E, Alessi C, Legre R, Juhan-Vague I. Iloprost and salvage of a free flap. Br J Plast Surg 1996;49:245–8.
28. Kerrigan CL, Zelt RG, Daniel RK. Secondary critical ischemia time of experimental skin flaps. Plast Reconstr Surg 1984;74:522–4.

The Authors

Raymond Tse BSc, MD
Douglas Ross MD, FRCSC, Chair
Bing Siang Gan MD, PhD, FRCSC, FACS

Division of Plastic and Reconstructive Surgery, Department of Surgery, University of Western Ontario, St Joseph's Health Care, 268 Grosvenor Street, London, Ont. N6A 4L6, Canada.

Correspondence to Dr Douglas Ross, St Joseph's Health Care, 268 Grosvenor Street, London, Ont. N6A 4L6, Canada

Paper received 26 September 2002.
Accepted 19 November 2002.

British Journal of Plastic Surgery (2003), **56**

doi:10.1016/S0007-1226(02)00478-2

Castleman's disease presenting as a midline neck mass

S.E. Bond, N.R. Saeed, I. Palka* and F.P. Carls

*Departments of Oral and Maxillofacial Surgery, and *Histopathology, The John Radcliffe Hospital, Oxford, UK*

SUMMARY. We report the case of a 13-year-old girl who presented with a painless midline submental mass. Excision biopsy confirmed Castleman's disease of the hyaline-vascular type. This unusual condition needs to be considered in the differential diagnosis of masses arising in the neck. © 2003 The British Association of Plastics Surgeons. Published by Elsevier Science Ltd. All rights reserved.

Keywords: castleman's disease, midline neck mass.

Castleman's disease is a rare benign condition of unknown aetiology that can present as a localised mass or in a more aggressive multicentric form. The localised form of the disease tends to present as progressive painless slow-growing lymph-node enlargement. The majority of cases involve the mediastinum, but the head and neck is the second most common site of occurrence, where it can pose a diagnostic challenge. Diagnosis is possible only by histological examination. Complete excision is the treatment of choice and tends to be regarded as curative for the localised form of the disease.