Case Report

Sudden shrinkage of free rectus abdominis musculocutaneous flap 15 years after maxilla reconstruction

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ABSTRACT

A 60-year-old male displayed sudden shrinkage of a left free rectus abdominis musculocutaneous flap, which had been grafted to his left maxilla 15 years previously. No post-reconstructive irradiation had been performed, and no late occlusion of the vascular anastomosis, local infection, recurrence of the maxillary cancer, or body weight loss was observed. However, the shrinkage amounted to approximately 50%. This is considerably more than previously reported cases of shrinkage of various free flaps, which ranged between 10% and 25%. The resultant depression was successfully augmented with a right free deep inferior epigastric artery perforator flap. The residual fat volume of the previously grafted shrunken flap was revealed to be compatible with that of the newly harvested contralateral donor site, although the mechanism of sudden flap shrinkage is unclear.

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Introduction

In free flap reconstruction, postoperative atrophy of the graft may occur. It is well known that muscular component atrophy occurs extensively within several months. In addition, fat tissue of the flap is also reported to potentially display a volume loss of 10%–25%. However, it is generally believed that the flap maintains its volume after the fat loss if there is no major body weight loss.

Here, we present a case of sudden massive shrinkage of a previously grafted free flap, 15 years after the patient had undergone free rectus abdominis musculocutaneous (RAMC) flap reconstruction for maxillary cancer. Although the cause of the sudden shrinkage was undetermined, the resulting deformity was successfully repaired with a free deep inferior epigastric artery perforator (DIEP) flap.

Case report

A 44-year-old male underwent left maxillectomy for maxillary cancer (T3N1M0), left neck dissection, and immediate reconstruction with a left free RAMC flap in April 2000. He received adjuvant radiotherapy (34 Gy) and arterial injection chemotherapy preoperatively. No post-reconstructive irradiation was administered. The postoperative course was uneventful, and follow-up was completed 5 years postoperatively (Figure 1).

However, in December 2014, the patient developed exercise-induced dyspnea. Furthermore, in November 2015, he noticed transient hematuria and edema of the lower extremities, and felt a rapid shrinkage of his left cheek as well as his oral lining. This had a negative impact on his speech, causing him to have to re-order new dentures. There was no loss of body weight; on the contrary, he

Figure 1. Five years after the primary reconstruction using the left RAMC flap, showing an adequate volume and a good contour of the left cheek.
experienced slight weight gain. When he consulted an otorhinolaryngologist, computed tomographic imaging showed massive flap atrophy and no evidence of flap necrosis, infection, or tumor recurrence (Figure 2a). As he had suffered slight exertional dyspnea for several months, chest computed tomography was performed. The images showed pleural effusion and he was referred to several other departments, including urology. Although slight liver cirrhosis with splenomegaly and thrombopenia was found, pericarditis was finally identified as the cause of his pleural effusion, accompanied with some ascites. Oral administration of diuretics resolved his dyspnea.

He was then referred to our department for re-augmentation of the left cheek (Figure 2b). The approximate volume loss of the graft amounted to 50%. However, the vascular pedicle of the graft, which had been anastomosed to the left facial system, was revealed to be palpable, as well as audible with handheld Doppler ultrasonography. Thus, re-augmentation for massive graft shrinkage due to unknown causes was planned. However, because the subcutaneous tissue was very thin in contrast to the required volume for the large depression, structural fat grafting seemed insufficient. Therefore, a free perforator flap with dermal and fat tissue was employed. A de-epithelialized right DIEP flap (19 x 7 cm, Figure 3) was harvested, trimmed, and grafted to the undermined depression. The vascular pedicle was then anastomosed to the left superficial temporal system. Three months postoperatively, sufficient augmentation was achieved, with a slight overcorrection (Figure 4). The necessity for further procedures will be assessed in future follow-ups.

Figure 2. a) A computed tomographic image at re-consultation 15 years after the primary reconstruction. Nearly 50% reduction of the flap was approximated by corresponding horizontal slices. b) Appearance at re-consultation. Severe depression of the left cheek is prominent.

Figure 3. Intraoperative view of the re-augmentation. a) Flap elevation. Note the extremely thin skin paddle. b) Flap insetting. Sufficient flap volume was supplied.
Discussion

Gradual free flap atrophy within months to several years has been well documented. A residual volume of 82.2% in RAMC or anterolateral thigh (ALT) flaps (median post-reconstructive period of 28.9 months), or 76.9% at 12 months in RAMC flaps have been reported. Another literature indicates a reduced volume of 20.4%, 30.3%, and 42.7% within 1, 3, and 5 years, respectively, in radial forearm flaps.

However, to our knowledge, this is the first report of acute massive volume loss of up to half of the initial flap volume.

There are multiple factors that are suspected to cause flap atrophy. The influence of perioperative radiation is controversial. Regarding the final flap volume difference between irradiated and non-irradiated patients, the conclusions of previous literature include both significant and non-significant findings. Thus, although our patient had received irradiation preoperatively, this might not be enough to explain the sudden massive shrinkage. Weak correlations between smoking (p = 0.051) or drinking (p = 0.069) and flap atrophy have also been reported. Our patient smoked five cigarettes a day for 40 years and drank 180 ml of spirits twice a week, which was not likely to have had any significant effect on the flap volume change.

Another problem was the sudden onset of atrophy in the previously-grafted flap. It has been reported that late obstruction of the vascular pedicle may cause late-onset flap shrinkage. However, the vascular pedicle in the present case was well-traced with handheld Doppler ultrasonography.

Although body mass index (BMI) is thought to directly influence flap fat volume, our patient claimed that his body weight did not decrease around the time of the flap shrinkage. However, it was confirmed intraoperatively that the skin paddle of the left DIEP flap was much thinner than that of the previously elevated right RAMC flap. This finding suggests that the sudden shrinkage of the RAMC flap might have reflected acute thinning of the patient’s subcutaneous fat. Indeed, Fujioka et al. claimed that fat thickness is a more appropriate indicator of the flap volume than body weight or BMI.

The possible mechanism of sudden thinning of subcutaneous fat tissue due to pericarditis may be explained, to an extent, by the concept of ‘cardio-renal cachexia’. In patients with cardio-renal cachexia, the coexistence of mild heart failure, transient hematuria, and lower extremity edema, in addition to low body fat percentage, is observed, as in the present case. On the other hand, the criteria for cachexia include chronic inflammation (elevated levels of C-reactive protein, interleukin–6, or low hemoglobin / serum albumin), however, our patient did not show any signs of such inflammation. Additionally, cachexic patients generally have poor survival rates of <50% within the first 18 months, which was unlikely in our patient. Thus, the exact mechanisms of the sudden onset of massive shrinkage in the present case remain unclear.

Figure 4. Appearance 3 months postoperatively. Sufficient augmentation was achieved.
Conflict of interest

There are no financial or personal relationships with other people or organizations that could inappropriately influence this work.

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