Facial Fat Necrosis Following Autologous Fat Transfer and its Management

Sweta Rai, Alexander M Marsland[1], Vishal Madan[2]

Procedural Dermatology Fellow, Mount Sinai Hospital, New York City, New York, USA, [1]University of Manchester and Salford Royal Hospital, [2]Dermatology Centre, Salford Royal Hospital, Salford, Manchester, United Kingdom

Address for correspondence: Dr. Sweta Rai, Salford Royal NHS Foundation Trust, Stott Lane, M68HD, Manchester, United Kingdom.
E-mail: swetadoc@yahoo.co.uk

ABSTRACT

Autologous fat transfer (AFT) is an increasingly popular cosmetic procedure practiced by dermatologic surgeons worldwide. As this is an office based procedure performed under local or tumescent anaesthesia with fat transferred within the same individual and limited associated down time its is considered relatively safe and risk free in the cosmetic surgery arena. We describe a case of AFT related fat necrosis causing significant facial dysmorphia and psychosocial distress. We also discuss the benefits and risks of AFT highlighting common causes of fat graft failure.

KEYWORDS: Autologous fat, dermatologic surgery, fat necrosis, fat transfer

INTRODUCTION

Autologous fat transfer (AFT) is gaining popularity amongst dermatologic surgeons worldwide mostly due to the fact that fat in many ways is an ideal filler and the procedure is relatively low risk in trained hands. Autologous fat is thought to be less immunogenic than other filling substances due to its transferability within the same individual from sites of low lipolytic activity such as the buttocks, hips and lower abdomen to high lipolytic sites including the face, arms and upper torso. Given that fat is also naturally found in abundance and is biodegradable there has been an assumption that this procedure is risk free. We describe a rare case of facial fat necrosis following autologous fat transfer and the application of sound dermatological surgical technique in the treatment of this adverse event. We also discuss the risks associated with autologous fat transfer procedures and how adverse events can be avoided.

CASE REPORT

A 36-year-old Syrian woman originally presented to the emergency department with acute onset of bilateral periorbital swelling [Figure 1a and b]. An initial diagnosis of periorbital cellulitis was made and treated with oral antibiotic chemotherapy. In spite of this the swelling extended down both cheeks over the following 2 weeks. Spontaneous resolution of the periorbital swelling occurred after 4 weeks. Routine investigations to exclude lupus erythematosus, dermatomyositis/polymyositis and acquired C1 esterase inhibitor deficiency including autoimmune profile, inflammatory markers and a C1 esterase inhibitor level were within normal limits. Specific autoantibodies for lupus erythematosus and dermatomyositis/polymyositis were also negative and a raised eosinophil count of 2.5 U/l was observed. The initial inflammatory response subsided with a resultant sunken appearance of her cheeks, [Figure 1c] which was diagnosed as post inflammatory fat atrophy. The cosmetic implications had an impact on her psychological well-being. She sought opinion from an oculoplastic surgeon and was offered autologous fat transfer (AFT) to fill the cheeks.

The initial AFT was successfully performed however a second procedure was required 4 months later to provide an optimal cosmetic result. Twenty four to 48 hours after the second AFT the patient noted pain and tenderness on both cheeks. This was subsequently followed by erythema and apparent extrusion of fat from the inflamed cheeks [Figure 1d]. She was referred to the department and a skin

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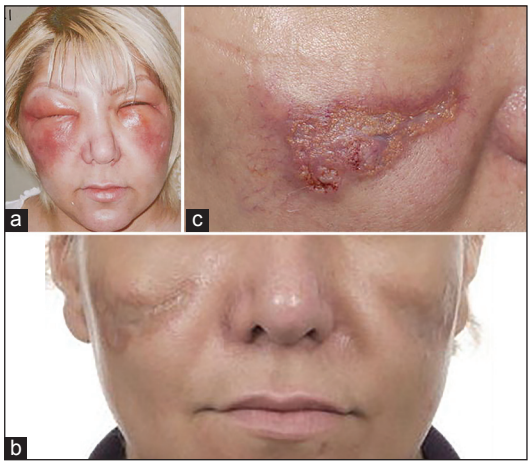

Figure 1: (a) Acute onset periorbital swelling. Close-up image representing limited eye opening; (b) Resultant post inflammatory subcutaneous fat atrophy with bilateral sunken appearance of both cheeks; (c) Post 2nd autologous fat transfer site erythema and ulceration from where fat extrusion was noted

biopsy confirmed the diagnosis of fat necrosis amidst a mixed septal and lobular panniculitis with inflammatory infiltrate extending down to the dermis [Figure 2a and b].

At this point she was commenced on a reducing course of oral prednisolone over 4-6 weeks with no improvement. A special formulation of 0.3% topical tacrolimus in clobetasol proprionate 0.05% ointment[1] applied for 4 weeks resulted in some reduction of inflammation. Subsequently three, four weekly intralesional triamcinolone injections to the affected sites led to complete resolution of inflammatory symptoms. Two large indurated yellow-red plaques on both cheeks remained [Figure 3a], however.

She was then referred to the dermatological laser surgeon for consideration of resurfacing of the resultant scarring. Due to the extensive scarring and fat atrophy, staged serial excisions of the scar tissue were considered a more appropriate option.

Two-staged serial excision of the cheek scars spaced 4 months apart was performed. She was assessed 6 weeks and 3 months post operatively on both occasions and photographic documentation was recorded 6 weeks after the final scar excision [Figure 3b (i-iii)] and 3 months post operatively [Figure 3c (i and ii)]. She finds the resulting curvilinear scars acceptable and is able to use cosmetic camouflage with good effect.

DISCUSSION

Our case demonstrates a rare though possible complication of autologous fat transfer. Fat necrosis of

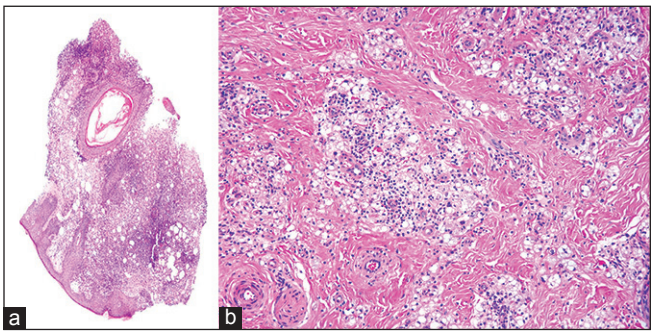

Figure 2: (a) Skin biopsy demonstrating a mixed lobular and septal panniculitis with inflammatory cells extending down to the dermis. Haematoxylin and eosin staining 10×10 magnification; (b) Fat necrosis evident on skin biopsy. Haematoxylin and eosin staining 40×40 magnification

the newborn is recognised as an entity in literature and fat necrosis post autologous fat transfer to the breast has also been described.[2] Fat necrosis resulting from AFT performed for facial filling has however been less extensively reported and is noteworthy of recognition especially due to the potential of permanent scarring.

There may be several factors relevant to the causation of fat necrosis post AFT which include preoperative patient selection, intra operative and post operative risk factors. Special consideration must be given to the identification of any underlying inflammatory dermatoses prior to the performance of AFT and ensuring correct patient selection for this procedure. An underlying active inflammatory dermatosis is likely to accelerate lipolysis of the transferred fat due to the body's inherent defence response to areas of infection or inflammation.

Autologous fat transfer as a procedure requires operator skill as well as practise and precision. This sets this filling procedure apart from other intradermal fillers as a high level of skill is involved in the harvesting, the preparation and the replacement of the fat. Factors that have been reported to improve graft survival include:

1. Low vascularity of the donor site.
2. High vascularity of the recipient site.
3. A low pressure technique used to aspirate the fat.
4. Skilful washing and preparation of fat.
5. A sufficiently large cannula.
6. The use of a multilayered approach to replace the fat.

Overfilling the defect may also impair graft survival.[3]

It is possible that a delayed hypersensitivity response to the prepared fat may also be a cause for graft failure. This may be compounded if the fat is not replaced in its pure form, i.e., after washing off any residual blood and tumescent anaesthesia collected at the time of

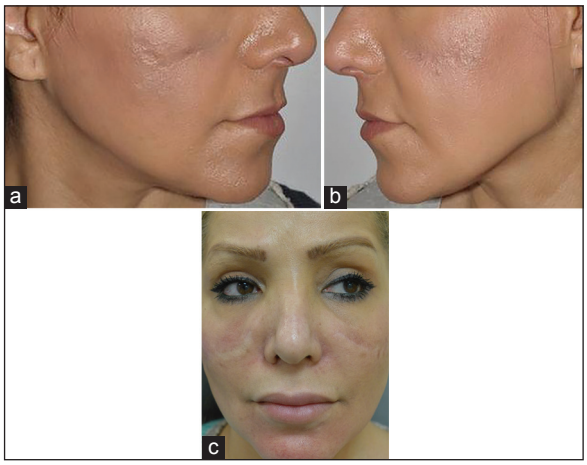

Figure 3: (a-b) Side profile view of resultant scarring 3 months post operatively (staged serial excision of scarring pos 2nd AFT); (c) Head on profile view of resultant scarring after the staged serial excision. This image demonstrates her ability to cover the defects with make up

harvesting. Effective centrifugation of the harvested fat may minimise the risk of immunogenicity.

In this case we postulate the primary cause of graft failure or fat necrosis was an as yet unrecognised and untreated underlying inflammatory dermatosis as the surgeon performing the procedure was both experienced in this technique and performed a previous successful AFT on the same patient in the past. It is also a possibility that this may have been a delayed hypersensitivity reaction to the prepared autologous fat after sensitisation during the initial procedure.

Autologous fat transfer may lead to complications other than fat necrosis. The early recognition and treatment of these enhances a clinician's procedure prowess.

Some other complications include:[4]
Rapid absorption of the replaced fat. This is exacerbated by the presence of blood; damaged fat cells and high pressure technique.

Infection presenting pre, intra or post operatively may affect graft survival. Viral infections or warty growths have also been reported at the site of cannula entry.

Fat embolism, particularly if injected intravascularly.

Blindness may result as a direct consequence of needle puncture and intracapsular trauma or pressure related retinal artery thrombosis.

Cyst formation is more common when a large amount of fat is transferred.

Calcification of fat post transfer has been reported particularly when used for breast augmentation and may present as breast lumps.[5] Calcification may lead to ossification over time.

Skin necrosis and sinus formation may result from overfilling or over augmentation of the desired site.

Compression atrophy may result from over augmentation with associated avascular necrosis due to excessive pressure on vasculature which can result in arterial or venous thrombosis.

Haematoma or seroma formation may occur more commonly if blunt cannulae are not used and when the patient is on other anticoagulant medication or supplements providing an anticoagulant effect.

Iatrogenic injury to the nerves and blood vessels of the face may also be seen.

A rare but recognised side effect is seen if fat is transferred to the face from areas such as the buffalo hump to the face in the form of facial fat hypertrophy or "Hamster syndrome" due the resultant swollen facial appearance resembling a hamster.

This case of fat necrosis was controlled by a special formulation of topical 0.3% tacrolimus in clobetasol proprionate 0.05% ointment and intralesional triamcinolone injections. Post AFT fat necrosis-related scarring was then further treated with serial staged excisions of the affected areas by applying basic principles of dermatologic surgery to the treatment of an unusual cause of scarring. To the best of our knowledge this is the only reported case of fat necrosis resulting from facial autologous fat transfer treated successfully with potent topical and intralesional steroids and serial staged excisions of the resultant scarring.

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