

Intra-orbital fat necrosis following transcaruncular orbitotomy mimicking a surgical site infection: A case report

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Abstract:

Fat necrosis is a benign non-suppurative inflammation of adipose tissue most commonly occurring in breast, subcutaneous tissue or intraabdominal fat post trauma, surgery, radiation. Transcaruncular orbitotomy provides a safe, rapid, and cosmetically pleasing approach to the medial wall and orbital apex. Intraorbital fat necrosis as its complication has not been documented in literature. The authors report the case of an elderly lady who presented with localized pain, swelling following a transcaruncular orbitotomy for excision biopsy of an orbital vascular mass. The etiology, clinical presentation, intraoperative finding, imaging, and possible mechanisms contributing to the pathogenesis of postsurgical orbital fat necrosis has been suggested.

Keywords:

Orbit, fat necrosis, non encapsulated, Transcaruncular orbitotomy

INTRODUCTION

Fat necrosis is a benign nonsuppurative inflammatory process of adipose tissue, most commonly reported in the breast.^[1,2] Existing literature reveals only one case of posttraumatic encapsulated orbital fat necrosis.^[3] Transcaruncular orbitotomy provides a safe, rapid, and cosmetically pleasing surgical approach to the medial orbital wall and orbital apex.^[4] Intraorbital fat necrosis as a complication of transcaruncular orbitotomy has not been reported in the literature. Herein, we report the etiology, clinical presentation, intraoperative finding, imaging, and possible mechanisms contributing to the pathogenesis of nonencapsulated orbital fat necrosis following transcaruncular orbitotomy. Patient consent to publish identifiable photograph was obtained.

CASE REPORT

A 64-year-old female presented with painless gradually progressive proptosis of the left eye since 1 year [Figure 1a]. Best-corrected visual acuity in both eyes was 20/20 along

with normal ocular examination. Computed tomography (CT) of the left orbit showed a well-defined contrast-enhancing homogeneous mass involving mid and posterior orbit in between superior and the medial rectus in close proximity to the superior oblique muscle [Figure 1b and c]. Excision biopsy of the mass through a transcaruncular medial orbitotomy approach revealed it to be a benign vascular malformation on histopathological examination [Figure 1d]. Early postoperative course was uneventful. Two weeks later, she presented with complaints of localized pain with swelling of the medial upper eyelid for 2 days with no visual complaints. Ocular examination showed left superomedial eyelid edema along with localized tenderness. There was conjunctival wound gape with prolapse of pale yellow soft globular mass with no localized conjunctival congestion [Figure 2a]. The rest of the ocular examination was normal. The patient was afebrile. CT scan orbit showed an ill-defined mixed density lesion (-60, +10, +20-30 HU at different areas) along the left medial wall extending from wound area to the orbital apex with mild temporal displacement of the globe [Figure 2b]. Suspecting a surgical site

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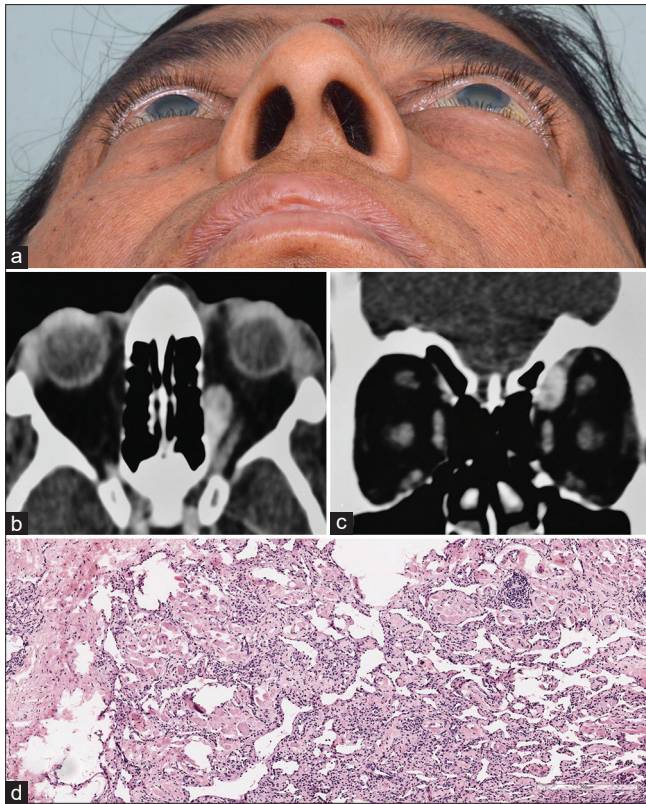


Figure 1: (a) Worm's view showing left eye proptosis; (b and c) Computed tomography scans of orbits (mid axial and coronal sections) showing well-defined homogeneous mass involving the left mid and posterior orbit extending up to the apex, abutting the optic nerve and in close proximity to the left superior oblique muscle; (d) Photomicrographs shows intravascular glomeruloid like endothelial and spindle cell proliferation with bland nuclear morphology (H and E stain, $\times 10$)

infection, intravenous antibiotics were initiated and wound exploration and drainage was planned. Intraoperatively, the whitish-yellow mass seen at the superonasal caruncular incision site appeared to be fat prolapsing through conjunctival wound and not pus. Copious pale fat-like tissue was noted in the medial orbital space along with inflamed muscles and surrounding tissues; however, there was no evidence of any focus of infection [Figure 2c and d]. Microbiological workup of fat did not reveal any organism, and histopathology was consistent with fat necrosis [Figure 2e]. Tapering dose of oral steroids was initiated, and the patient was doing well 1 year postsurgery.

DISCUSSION

Fat necrosis although first described in breast in the early 20th century is known to occur at several sites in the body.^[1,2] The etiology of fat necrosis is not very well elucidated. The different etiological factors reported include trauma, breast surgery (cyst aspiration, biopsy, lumpectomy, mastoplastic, implant removal, breast reconstruction with abdominal flap), fat transfer or tissue flaps, radiotherapy, chemotherapy, anticoagulation therapy, and breast infection. Other rare causes include collagen vascular diseases such as giant cell

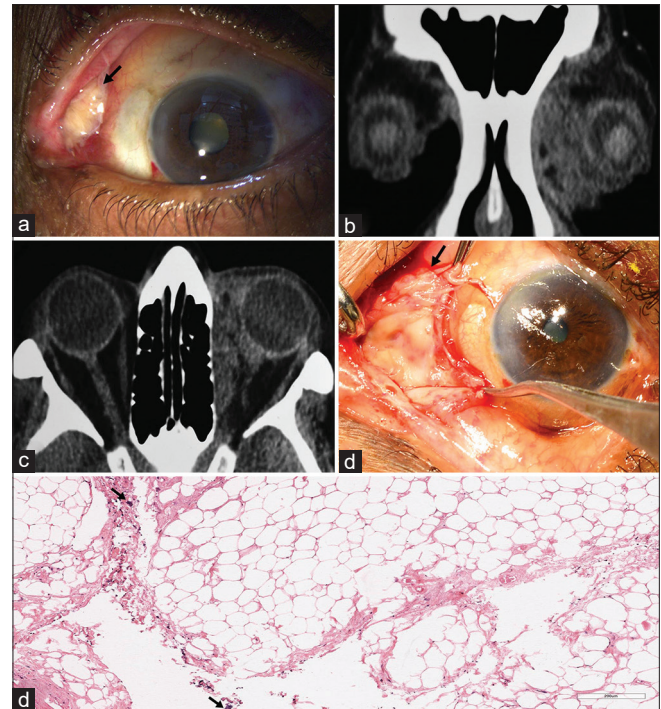


Figure 2: (a) Clinical photograph showing pale yellow mass (black arrow) prolapsing through the conjunctival wound; (b and c) Computed tomography scans of orbits (coronal and midaxial sections) showing ill-defined mixed tissue density along the left medial wall extending up to the orbital apex causing temporal displacement of globe; (d) Per-operative photograph showing pale orbital fat (black arrow) filling the medial orbit space; (e) Photomicrograph shows ghost-like pale staining adipocytes with fibrin thrombi in the necrosed vessels (H and E stain, $\times 10$)

arthritis, granulomatosis with polyangitis, polyarteritis nodosa, Weber-Christian disease, granulomatous angiopanniculitis or systemic lupus erythematosus. In some cases, it may also be idiopathic.^[1,2] A few cases of fat necrosis resulting from repeated insulin injections have also been reported.^[5,6] Tissue ischemia resulting from vascular trauma (physical and chemical) has been considered to be the main etiologic factor.^[6,7] To the best of our knowledge, till date, there is only one report of encapsulated orbital fat necrosis following trauma within the lateral rectus muscle.^[3] The imaging findings in their case closely mimicked a tumor of lipomatous origin, but the histopathology revealed fat necrosis. However, our case did not show any pseudocapsule on gross or microscopic examination.

The pathogenesis of postoperative fat necrosis in our patient can only be speculated. One possible theory is direct surgical trauma causing cellular breakdown because of nonlacerating compressive forces and subsequent activation of enzymes causing fat necrosis.^[8] Leaking of lysosomal contents of necrotic cells may elicit inflammation in the surrounding tissues further aggravating tissue ischemia and initiating a vicious cycle.^[9] The second possibility can be postoperative soft-tissue edema and blood accumulation in the medial orbital space causing tissue ischemia and pressure necrosis of orbital fat. The third hypothesis is thermal and ischemic damage to the fat cells causing necrosis in an attempt to achieve hemostasis

during surgery. However, the latter is extremely unlikely a high frequency radiofrequency unit (3.8 MHz, Ellman Surgitron FFPF EMC, Ellman International Inc.) was utilized for hemostasis which causes minimal thermal alteration compared to conventional electrocautery.

Clinical presentation of fat necrosis of the breast can be extremely variable depending on the site of the lesion. It can range from a painless asymptomatic lump to a painful lump with ecchymosis, erythema, induration, and skin retraction.^[8] Subcutaneous fat necrosis usually presents as painful tender nodules, but pain is not a sine qua non for the diagnosis.^[1,2] In the only case of orbital fat necrosis described in the literature, the sole symptom reported by the patient was diminution of vision following blunt trauma and there were no significant clinical signs.^[3] The presence of localized pain, tenderness in the medial orbit 2 weeks postorbitotomy and ill-defined mixed densities all along the medial orbital wall on CT scan lead to the impression of postoperative surgical site infection in our patient. However, lack of systemic toxic features, absence of purulent discharge, and localized conjunctival congestion near the incision site was not substantiating the diagnosis of orbital infection.

On CT scan, fat is hypodense (black); however, inflamed edematous fat becomes hyperdense^[10] thus giving rise to the variable densities in the medial orbit in our case. The appearance of fat necrosis on magnetic resonance imaging (MRI) has been well described in literature. Tsai *et al.* proposed MRI criteria for the diagnosis posttraumatic fat necrosis which were: Linear alterations in signal intensity, confined to subcutaneous tissue without a well-defined mass.^[8] The lack of a discrete mass is not universal, as presence of mass-like features has also been described.^[10,11] However, MRI was not done in our patient as there was no prior suspicion of fat necrosis. The diagnosis of orbital fat necrosis can be easily missed unless there is a strong suspicion and specific imaging modalities are employed. Burkholz *et al.* reported a case of sub cutaneous fat necrosis which closely mimicked a liposarcoma and warranted a biopsy for confirmation of the diagnosis.^[12]

There is no conclusive management protocol available for the treatment of orbital fat necrosis. For subcutaneous painful lesions, excision is usually warranted while asymptomatic lesions can be observed.^[6] For diffuse orbital fat necrosis like in our case, conservative management with anti-inflammatory therapy should suffice in addressing the symptoms.

In conclusion, this case highlights orbital fat necrosis as a possible complication of transcaruncular orbitotomy and the clinical presentation can closely mimic postoperative surgical site infection. Lack of systemic toxic features, absence of purulent discharge, localized conjunctival congestion, and

MRI orbit can differentiate it from infection. In retrospect, if diagnosis of fat necrosis had been considered with greater confidence preoperatively, a conservative approach could have been taken and surgical intervention avoided in this case. Awareness of this rare possibility and speculation of possible mechanisms will help ophthalmic plastic surgeons be better prepared for this rare complication.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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