infiltrated by lymphocytes were noted within the granulomatous inflammation (Fig. 2D).

DISCUSSION

Lupus miliaris disseminatus faciei was first described as disseminated follicular lupus in 1878.[1] However, it has also been referred to by other names such as acne agminata as by Duke-Elder and MacFaul,[4] rosacea-like tuberculid, micropapular tuberculid, lupoid rosacea, and most recently facial idiopathic granulomas with regressive evolution.[1]

Contrary to its name, this condition is not associated with systemic lupus erythematosis or tuberculosis. LMDF was originally thought to be a variant of lupus vulgaris or a tuberculid, but there is no evidence to support this. Others have regarded it as a papular form of rosacea (granulomatous rosacea) or maculopapular sarcoidosis because LMDF's characteristic histopathology may be found in granulomatous rosacea while the clinical features are sometimes similar to cutaneous sarcoidosis.[2]

It generally is regarded that the histopathologic picture of LMDF reveals a variable amount of necrosis surrounded by epithelioid cell granulomas.[1,3] Other variables have been debated such as the relationship with pilosebaceous units[5] and the amount and type of inflammatory cells seen in the vicinity.[3,5] However, there is a spectrum of histologic findings in this condition, evolving from lymphocytic infiltration around hair follicles with epithelioid granuloma (stage 1 disease), to neutrophilic abscesses (stage 2), to caseation necrosis (stage 3), which was described only in 13% to 48% of cases.[1,3,5,6]

Although the pathogenesis of LMDF is still unknown, there are many hypotheses. Several investigators have suggested the pathogenesis of LMDF involves a granulomatous reaction to the breakdown of pilosebaceous apparatuses or epidermal cysts.[3,5] In our cases, degenerated follicular epithelium was in continuity with the central necrosis found within the epithelioid cell granulomas and abscesses with epithelioid cells were adjacent to the follicular epithelium. It is believed that the initial event in LMDF is an attack of hair follicles by lymphocytes.[3] Damage to the follicular wall results in the release of an antigenic substance in the dermis eliciting a granulomatous reaction.[3,5]

The differential diagnosis of LMDF includes the papular form of acne (granulomatous) rosacea, acne vulgaris, perioral dermatitis, lupus vulgaris, papular syphilid, sarcoidosis, papulonecrotic tuberculid, the papular form of granuloma annulare, and chalazion.[1] A chalazion, as initially diagnosed in our first case, can be distinguished from LMDF by involvement of sebaceous glands rather than hair follicles and the presence of lipids.

The etiology and pathogenesis of LMDF remains unclear. Consequently, there have been a variety of treatments including intramuscular triamcinolone, 1450-nm diode laser, minocycline, isotretinoin, and oral corticosteroids with varying effectiveness.[1] Surgical excision is another therapeutic option, primarily used for diagnosis, as was performed in both our patients.

We report 2 cases of LMDF, which posed as diagnostic dilemmas. Our findings were consistent with the hypothesis that granulomas with necrosis develop in response to inflamed and damaged pilosebaceous apparatuses. Thus, we regard LMDF as a separate clinical entity, as do most investigators.

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Primary Ocular Presentation of Sinonasal Undifferentiated Carcinoma

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Abstract: **The authors describe 2 consecutive patients who presented to Vanderbilt University Medical Center with primary orbital presentation of sinonasal undifferentiated carcinoma and were treated from July 2005 to April 2009. The patients were a 39-year-old woman and 54-year-old woman who both presented to the ophthalmology service due to complaints of diplopia. Imaging studies demonstrated large soft tissue masses originating in the sinuses with extension in the orbit in both cases. Both patients were treated with carboplatin, paclitaxel, and dexamethasone as induction chemotherapy followed by concurrent chemoradiation with intensity-modulated radiation therapy. This treatment regimen resulted in significant tumor shrinkage, resolution of symptoms, and no evidence of recurrence while avoiding surgical intervention and allowing orbital preservation.**

The sinonasal tract can give rise to a variety of epithelial and nonepithelial malignant neoplasms. Most of these arise from the maxillary sinus, and it is possible to have invasion in adjacent structures. Sinonasal undifferentiated carcinoma (SNUC) is a rare and aggressive neoplasm arising from the nasal or paranasal sinuses that was first described in 1986 by Fricrson et al.[1] At presentation, there is often locally advanced disease with patients often having involvement of multiple sinuses and even extension in the orbit or intracranial vault.[2,3] The median survival is 4 months (range, 1–41 months).[1]

Common presenting symptoms are secondary to its involvement of the sinuses and less commonly, the orbit. Here, we

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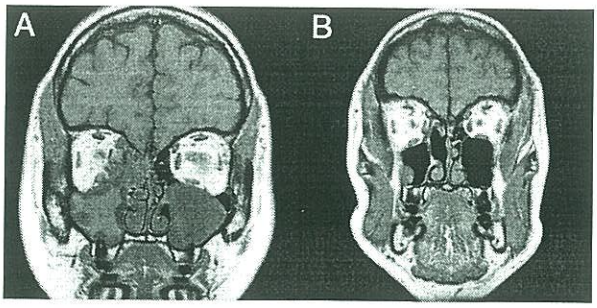

FIG. 1. A, Coronal MRI of patient from Case 1 demonstrating a large soft tissue mass in the bilateral maxillary sinuses and right ethmoid sinus with extension in the extraconal space of the right orbit. B, Coronal MRI of patient from Case 1 following induction chemotherapy followed by concurrent chemoradiation demonstrating significant shrinkage in size of the tumor.

report the clinical, radiologic, and pathologic findings of 2 patients with SNUC. For both of these patients, ocular complaints were one of the prominent reasons for seeking evaluation.

CASE REPORTS

Case 1. A 39-year-old female presented with an 8-week history of decreased vision and double vision in right gaze. On further questioning, she also reported a 2-week history of right cheek numbness. Pertinent abnormal findings on ocular examination included 2 mm of proptosis and hypesthesia along the V2 distribution, and a 2-prism-diopter right hypertropia that built on right gaze and head tilt. CT and MRI revealed bilateral maxillary and ethmoid opacification extending in the extraconal space of the right orbit with probable involvement of the medial rectus muscle and posterior extension in the foramen rotundum (Fig. 1A). The biopsy demonstrated SNUC. Positron emission tomography scan confirmed confined disease of the tumor in the sinuses with extension in the orbit. The patient's disease was unresectable. The patient was treated with induction chemotherapy followed by concurrent chemotherapy consisting of carboplatin, paclitaxel, and dexamethasone with intensity-modulated radiation therapy. She had radiographic near complete response (Fig. 1B) and resolution of diplopia. There has been no evidence of recurrence 56 months after completing chemoradiation.

Case 2. A 54-year-old woman presented to the emergency department with diplopia and right sided headache for 1 week. Her ocular examination was remarkable only for a 15-prism-diopter right exotropia and 12-prism-diopter right hypertropia. A CT scan was performed, which demonstrated a large soft tissue mass involving the right paranasal sinus, right maxillary, and sphenoid sinuses with associated bony destruction, compression of the right medial rectus muscle, and posterior extension in the middle cranial fossa (Fig. 2A). Biopsy demonstrated SNUC. The tumor was felt to be unresectable. This patient was treated with the same therapy as described in Case 1. The patient had a radiographic near complete response (Fig. 2B), and the patient reported resolution of her diplopia after a single cycle. There has been no evidence of recurrence 51 months after completing chemoradiation.

DISCUSSION

It is not uncommon for neoplasms originating from the nasal and paranasal sinuses to involve the orbits.[4] Typical symptoms due to sinus neoplasms include epistaxis, nasal congestion, and vague facial pain. With orbital involvement, signs and symptoms that may occur include proptosis, ocular pain, visual loss from optic neuropathy, diplopia, and chemosis.[2,3] These symptoms are often the reason why patients seek treatment as demonstrated in these 2 patients. It is important for the ophthalmologist to keep nasal and paranasal sinus tumors in mind when evaluating patients with this constellation of ocular signs and symptoms. Given the aggressive nature and poor prognosis of SNUC, it is important to have prompt diagnosis and therapy.

The histologic appearance of the tumors in these 2 patients was similar—a malignant undifferentiated neoplasm with epithelioid and neurodendocrine features having frequent mitotic figures and necrosis (Fig. 3).

There is no standardized treatment plan for SNUC and may require an interdisciplinary approach. Many institutions

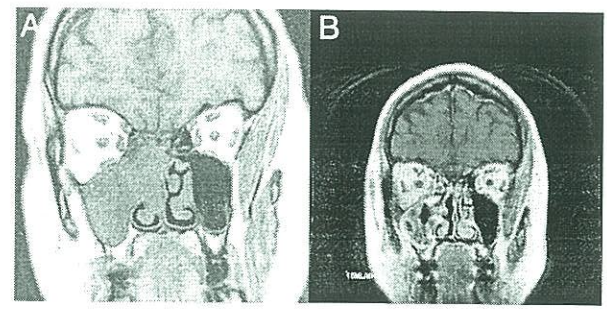

FIG. 2. A, Coronal MRI of patient from Case 2 demonstrating a large soft tissue mass involving the right maxillary and ethmoid sinuses. B, Coronal MRI of patient from Case 2 demonstrating decrease in tumor size following treatment.

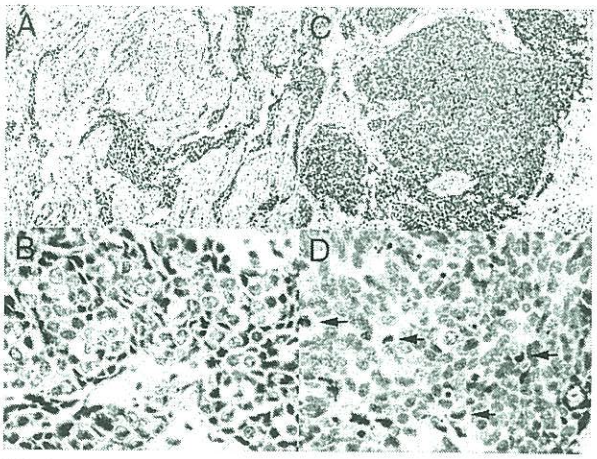

FIG. 3. A, Low-power, hematoxylin-eosin–stained slide (×20) and (B) high-power, hematoxylin-eosin–stained slide (×100) from Case 1 demonstrating a malignant neoplasm growing in nests comprised of medium sized polygonal cells with prominent nucleoli, fine chromatin, and small to moderate amounts of eosinophilic cytoplasm. A lymphocytic infiltrate is present. C, Low-power, hematoxylin-eosin–stained slide (×20) and (D) high-power, hematoxylin-eosin–stained slide (×100) from Case 2 demonstrating a mitotically active (arrows) malignant neoplasm comprised of sheets of undifferentiated cells with coarse chromatin, occasionally prominent nucleoli, and moderate amounts of eosinophilic cytoplasm with a background of karyorrhectic debris.

offer patients aggressive multimodal treatment to control local disease.[2,3,5] Rischin et al.[6] recently demonstrated that induction chemotherapy followed by concurrent chemoradiation may be a promising treatment approach in patients with locoregionally advanced SNUC. At their institution, the regimen consisted of 3 cycles of induction cisplatin or carboplatin and 5-FU chemotherapy followed by concurrent platinum chemotherapy and radiation. Surgical resection was reserved for patients with residual disease. A similar treatment philosophy was used in the management of the 2 patients discussed in this case series. In both patients, there has been a significant decrease in tumor size, resolution of symptoms, and no evidence of recurrence without surgical intervention.

In conclusion, it is important to recognize ocular complaints consistent with the presenting signs of nasal and paranasal sinus tumors. Prompt diagnosis is essential in SNUC given the aggressive nature of the neoplasm and dismal prognosis. An interdisciplinary approach with involvement from other services is important in developing a treatment plan for these patients.

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Ultratine Breakage

Aaron Savar, M.D.†, and John Shore, M.D.†*

Abstract: **Both Endotines and Ultratines are made of polymers of polylactic acid and polyglycolic acid and are used in brow elevation procedures. Ultratines appear to be much more likely to break during loading and insertion than Endotines. The authors report our experience with these devices and offer suggestions to decrease the likelihood of breakage.**

With the introduction of multipoint fixation, brow lifting has changed substantially. The Endotine device (Coapt Systems Inc., Palo Alto, CA) was introduced in 2003. It is made of a polymer of polylactic acid and polyglycolic acid and allows fixation of the forehead tissues to the underlying skull via the "hooks." There are 5 hooks on each Endotine. Normally, 2 Endotines are used in a patient to achieve symmetrical fixation and elevation. Implantation is performed via an open or endoscopic approach and has been reported to be safe and effective in several small series.[1–7]

The Ultratine was introduced by Coapt as a faster absorbing alternative to the Endotine. The Ultratine configuration is the same as that of the Endotine. Experience with this device has been reported in 2 small series.[8,9] We have used the Ultratine successfully in 60 patients. Recently, we have noticed that several Ultratines have broken during surgery. Because of our observation that the Ultratine is more delicate than the Endotine, we reviewed our experience with each device.

MATERIALS AND METHODS

We retrospectively reviewed all cases of Endotine or Ultratine implantation between December 1, 2003, and April 30, 2009, at the Center for Aesthetic and Reconstructive Eyelid and Oculoplastic Surgery in Austin, TX. The number of devices implanted each year was recorded. Additionally, all broken devices were recorded and noted whether the device broke during loading or insertion.

RESULTS

One hundred seventy-six Endotines were inserted in 90 patients (86 bilateral and 4 unilateral) between December 2003 and March 2007. None were reported broken. From April 2007 to April 2009, 116 Ultratines were inserted in 60 patients (56 bilateral and 4 unilateral). During this period, 6 were broken and returned to Coapt. The breakdown by year can be seen in Table. Two Ultratines were broken while loading in the inserter. Four Ultratines were broken during implantation. One Ultratine was broken for every 19.5 successfully implanted, while 178 Endotines were implanted successfully without breaking any.

A cluster of 3 Ultratines were broken during 2009. Two of these were found to be from a lot with a mislabeled expiration date, though none were expired at the time of implantation. This lot was noted to have an increased number of broken Ultratines according to Coapt, but has not been recalled. Two Ultratines that were implanted in one patient in 2008 required subsequent removal due to painful cyst formation at the site of the Ultratines.[10]

DISCUSSION

Both Endotines and Ultratines have been reported to be safe and effective in a number of series. Both devices have been approved by the FDA as a fixation device for endoforehead lifting. The complication rates are lower than other methods used for forehead fixation,[1] and the results are more predictable than older single point fixation methods.[7] Although we did not systematically review the outcomes with each device, we have

Number of Ultratines and Endtotines implanted by year

Year	No. Ultratines implanted/No. patients (number broken)	No. Endotines implanted/No. patients (number broken)
2003 (beginning December 2003)		8/5 (0)
2004		50/26 (0)
2005		42/21 (0)
2006		70/35 (0)
2007	52/27 (1)	6/3 (0)
2008	50/26 (2)	
2009 (through April 2009)	14/7 (3)	

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noticed good results with both. Based on our data, the Ultratine appears to be more delicate than the Endotine, however, we prefer the Ultratine because of its more rapid degradation and disappearance from the surgical site.

Our experience has taught us that when handling these devices, the Ultratine is more brittle than the Endotine. The Endotine is known to be made of a polymer consisting of 82% polylactic acid and 18% polyglycolic acid. According to Coapt, the polymer is altered in the Ultratine; however, Coapt would not provide further information on the differences.

Several strategies can be used to reduce the likelihood of breakage of Ultratines. The device must be handled with care. When loading it in the inserter, one must ensure that it is loaded properly. The inserter should not be clamped too tightly on the Ultratine. Care should be taken to avoid allowing the device to become caught on surrounding tissues during insertion. This can be facilitated by making sure the incision is long enough to allow placement without undue pressure being exerted. A 2-cm incision allows easy passage of the Ultratine through the skin in the subperiosteal space. When snapping the device in the drilled hole, force should be applied directly perpendicular to the bone. Twisting should be avoided. Additional Ultratines should be available in the event that one is broken.

If the Ultratine is broken during loading, a new Ultratine should be placed. Coapt has been willing to replace broken Ultratines for us if the damaged device is returned to them. If the break is noted after insertion, an attempt should be made to remove the Ultratine. This is not always easy to accomplish if the stalk is already in place in the bone at the time breakage occurs. In these situations, it may be necessary to leave the stalk in place, contour the protruding stalk to conform to the frontal bone, then drill a new hole and place a new Ultratine.

Although the Ultratine does have the advantage of faster absorption compared with the Endotine, the Ultratine is much more likely to break than the Endotine. Surgeons and their operative staff should be aware of this.

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Idiopathic Inflammatory Pseudotumor of the Eyelid

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Abstract: **A 40-year-old male presented with swelling and erythema in his right upper eyelid that began 4 months prior. Clinical evaluation, rheumatologic serology, sedimentation rate of erythrocyte, and C-reactive protein results were all normal. The patient also did not respond to local and systemic antibiotic therapy. Incisional biopsy revealed a pseudotumor. Oral prednisolone was initiated, and the drug dose was slowly tapered according to the patient's clinical response. After 6 months of follow-up, the eyelid lesion disappeared entirely.**

Nonspecific orbital inflammation is the third leading cause of orbital diseases after thyroid ophthalmopathy and lymphoproliferative disorders and constitutes 5% of all disorders in the orbital region. The characteristic clinical feature of nonspecific orbital inflammation is inflammation of the orbit without any apparent local or systemic cause. Clinical findings, imaging, and pathologic features of the disease have been described previously.[1–3] However, due to the variable presentation of the disease, a diagnosis should be considered after ruling out other possible conditions. Orbital inflammatory syndrome can affect any tissue in the orbit.[1–6] Eyelid involvement is almost always accompanied by involvement of deeper tissues in the region, and it is rare to have the disease isolated to the eyelid.[7–10] Here, we report a case of isolated idiopathic inflammation of the eyelid and discuss the possible diagnostic and therapeutic challenges with regard to the disease.

CASE REPORT

A 40-year-old man was referred to occuloplastic clinic in Labbafinezhad hospital for edema and erythema of the right upper eyelid of 4 months duration. The patient reported several previous episodes of chalazia, which improved without any specific therapy.

The patient had multiple prior ophthalmologic visits and received administration of various topical and parenteral antibiotics. Initial eye examination revealed a 20/30 vision in the right eye (with 0.25–1.5 × 90) and 20/20 in the left eye. Results of slitlamp biomicroscopy and fundoscopy of both eyes were all within normal ranges. Ocular and pupil motility were normal, and relative afferent pupillary defect was negative.

External examination revealed a mass in the right upper eyelid with a macerated center over hanging the eyelid margin and was surrounded by erythema and edema (Fig. 1A). Laboratory results revealed normal urine analysis, peripheral blood smear, sedimentation rate of erythrocyte, C-reactive protein, and uric acid levels. Serologic tests for rheumatologic disease such as rheumatoid factor, Venereal Disease Research Laboratory, antinuclear antibody, antinuclear cytoplasmic antibody, and angiotensin converting enzyme were all negative.

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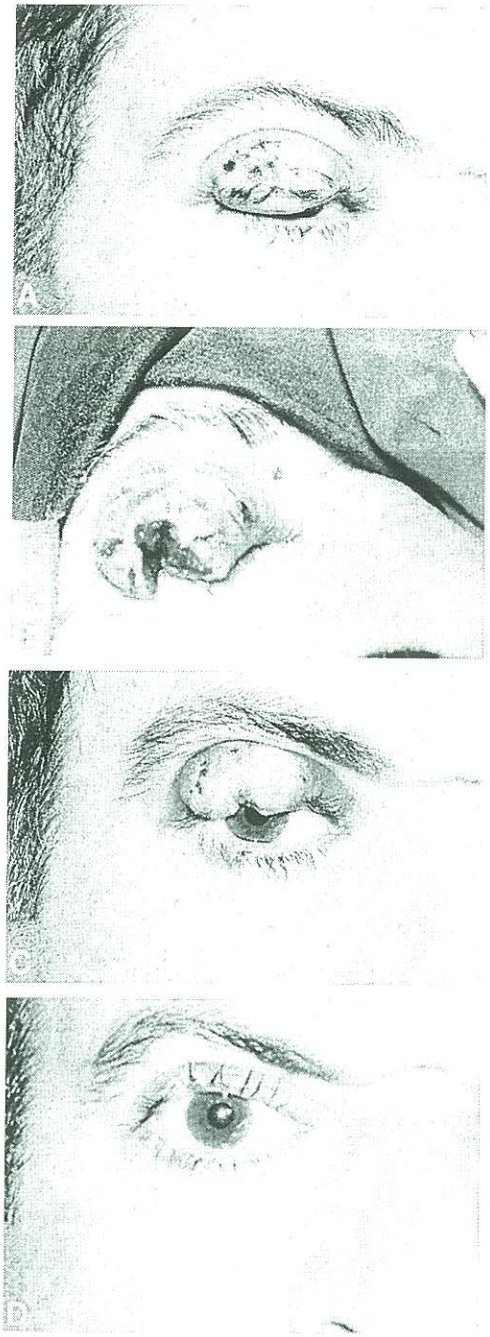

FIG. 1. A, Right upper eyelid showing erythema and swelling, with necrosis in the central region. B, Site of eyelid biopsy showing fish flesh appearance. C, The lesion size was reduced, along with decreases in inflammation 3 weeks after corticosteroid initiation. D, Complete improvement of inflammation (the biopsy site has been repaired).

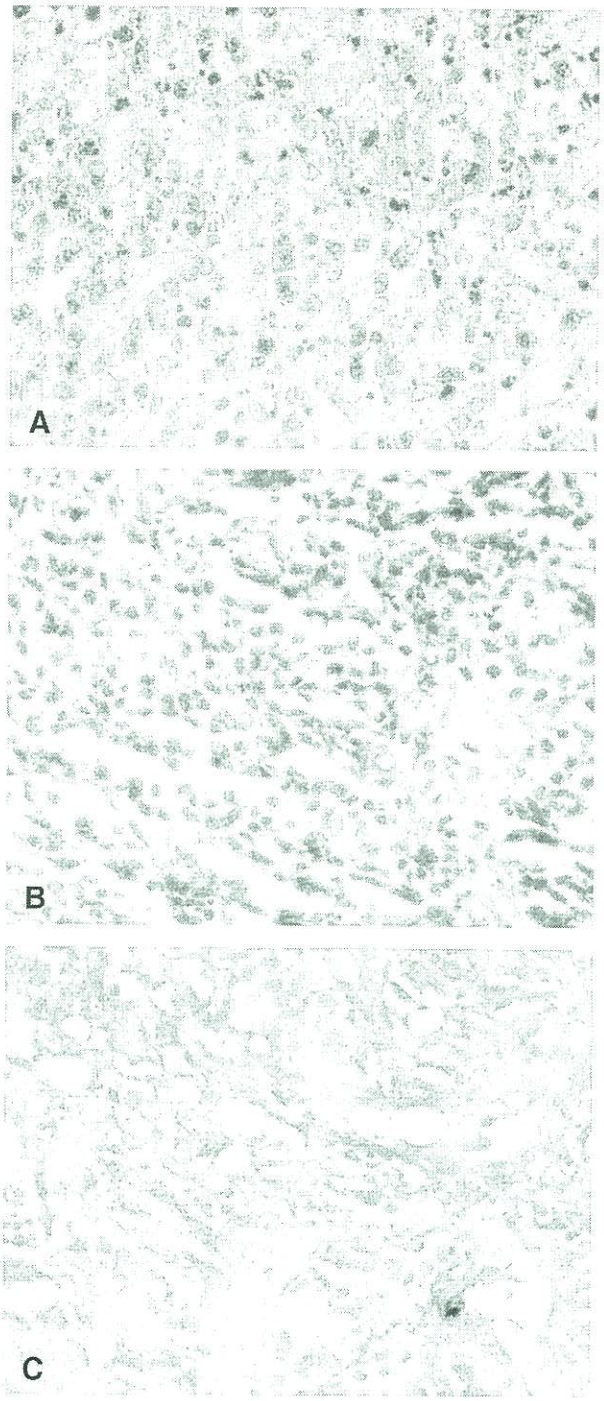

FIG. 2. A, Severe tissue infiltration of inflammatory cells including neutrophils, plasma cells, and atypical lymphocytes, with irregular nuclei in the dermis and hypodermis (hematoxylin-eosin, ×400). B, Periodic acid Schiff staining negative for fungi (×400). C, Ziehl–Neelsen stain negative for mycobacteria (×400).

The patient was admitted in the hospital, and a primary diagnosis of eyelid malignancy and secondary preseptal cellulitis was considered based on the patient's eyelid features. Intravenous antibiotic was subsequently administered to the patient.

Incisional biopsy was performed in a wedge-shaped manner, and a full-thickness section of the right upper eyelid, which included the necrotic mass, was sent to 2 different pathology centers (Fig. 1B). Primary repair of the eyelid was not possible due to fragility of the margins of the wound. Smear and culture of the tissue specimen revealed no significant microbiological organisms.

Pathologic evaluation of the specimen revealed severe infiltration of inflammatory cells to both the dermis and fat tissues. A predominance of neutrophils, plasma cells, and atypical lymphocytes (with irregular nuclei) were reported, and a lymphoproliferative disorder was considered. There was no evidence of mycobacterium or fungal infection (Fig. 2). Immunohistochemistry (for CD3, CD19 and CD20 antigens) confirmed active chronic inflammation and pseudoepitheliomatosis hyperplasia.

Given the diagnosis, 1 mg/kg of oral prednisolone was started, and parenteral antibiotic was discontinued. The eyelid lesion started to shrink with therapy, resulting in decreased inflammation and reduced size of the eyelid mass after 3 weeks on a corticosteroid (Fig. 1C). Drug dosage was then slowly tapered down to 5 mg/day after 2 months, and this regimen was continued until 6 months to prevent any relapse. Steroid was discontinued after 6 months, and in 2 years of follow–up, no further recurrence of the tumor was seen, so at that time the site of eyelid biopsy repaired (Fig. 1D).

DISCUSSION

Eyelid masses can have an inflammatory, infectious, or malignant origin, and these can be diagnosed according to the appearance, anatomic location, patient's age, and other clinical and laboratory examinations. In some cases, particularly in patients suspected of developing a malignancy, biopsy should be performed. Smears and cultures are also indicated for those with unusual appearance and clinical course.[1–3] In most cases, inflammatory eyelid lesions present as a localized swelling that is often accompanied by erythema with or without conjunctival involvement. Furthermore, these lesions can also present as a painless ulcerative or necrotic mass.

Other differential diagnoses for eyelid necrosis include bacterial infections (B hemolytic Streptococci) tumor or trauma; these diagnoses must be ruled out quickly.[1–5]

Given the long duration of the disease, the clinical appearance of the lesion, and the inappropriate response to antibiotics in our case, we initially considered possible malignancy. However, the diagnosis of pseudotumor of the eyelid was established after pathologic evaluations of the eyelid specimen, and the malignancy was ruled out. The most probable vasculitic involvement of the orbit that copes with this clinical picture is Wegener granulomatosis. Even though antinuclear cytoplasmic antibody may be negative in some of these cases, clinical findings, including respiratory and urinary involvements, were not present, and pathologic findings in benefit of this disease like necrosis and giant cells were negative.[11]

Orbital pseudotumor is a disease of unknown cause. However, it may be the result of an autoimmune response by the orbital tissues to unknown immune triggers like viral or bacterial infections.[1–6] Nonetheless, isolated involvement of the pseudotumor to the eyelid is quite rare.[7–10]

A previous study reported 3 cases of idiopathic inflammatory pseudotumor limited to the eyelid. There are also 2 other similar reports that described older cases of idiopathic inflammatory eyelid pseudotumor.[8,9] The last known report of idiopathic inflammatory eyelid pseudotumor is a retrospective study that examined 49 cases of inflammatory pseudotumor during a 10-year period. Out of the 49 cases, 11 (22.4%) exhibited eyelid involvement, but the exact number of idiopathic inflammatory lesions limited to the eyelid was not determined.[10]

CONCLUSION

Isolated pseudotumor involvement to the eyelid is a rare presentation. However, one should be mindful of the disease in listing the possible differential diagnoses for unusual masses in the eyelid. The disease can be confirmed via biopsy and histochemical evaluations.

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