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Noninfectious Inflammatory Response to Gold Weight Eyelid Implants

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Summary: We describe three patients with noninfectious inflammatory reactions to gold weight eyelid implants, a complication not previously reported. Eyelid edema and erythema developed gradually in each patient, and maximal inflammation that prompted treatment was present at 12, 3, and 5 weeks, respectively, after surgery in the three patients. Management involved removal of the implant in the first patient, oral corticosteroids followed by replacement of the implant by a platinum weight in the second patient, and a local corticosteroid injection with retention of the implant in the third. Histopathological features included a thick eosinophilic coagulum at the tissue-gold interface and an intense, predominantly lymphocytic infiltrate in the collagenous capsule that surrounded the implants. Gold weight eyelid implants can elicit a gradually progressive inflammatory response. In at least some cases, local corticosteroid injection may suppress the inflammation and permit retention of the implant. **Key Words:** Gold lid implant—Inflammation—Corticosteroid.

The gold weight implant has been useful for correcting exposure keratitis in patients with seventh nerve paralysis (1–5). Success rates, defined as long-term retention of implants and improved corneal protection, have exceeded 90% in several recent studies (3–5). Complications requiring removal or replacement with smaller implants were generally limited to induced astigmatism or unacceptable ptosis. Extrusion was reported in one instance and infection was not described.

We report three patients with noninfectious inflammatory reactions to gold weight implants—a complication not previously documented. Therapeutic options for the management of this problem are presented.

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CASE REPORTS

Patient 1

A 47-year-old woman had left facial weakness following resection of an acoustic neuroma 6 years before referral. Prior treatments had included topical emollients, tarsorrhaphy, peripheral neuromuscular stimulation, and botulinum injections for synkinetic twitching. Examination revealed a tarsorrhaphy involving the lateral two thirds of the palpebral fissure, a moderately good Bell's phenomenon, and a clear cornea. Basal tear secretion in the left eye measured <1 mm at 5 min, but was normal in the right eye. Left corneal sensation was diminished. To restore a full visual field and improve cosmesis, the patient elected gold weight loading of the upper eyelid as an alternative to tarsorrhaphy. One week after severing the tarsorrhaphy, the patient was judged to have 60% orbicularis oculi function, and a 1.0-g weight was selected.

The surgical procedure included mechanical cleaning of the sterilized implant with a gauze

FIG. 1. Patient 1. Edema and erythema of the left upper eyelid 3½ months after implantation of a gold weight. The inflammation did not respond to oral antibiotics, but resolved rapidly after implant removal.

sponge, soaking in gentamicin solution followed by saline irrigation, implantation posterior to orbicularis muscle, suturing inferiorly to tarsus and superiorly to the septum at its junction with the levator aponeurosis with 5-0 polypropylene suture, and separate closure of orbicularis muscle (7-0 vicryl suture) and skin (7-0 nylon suture). The early postoperative course was unremarkable, and the corneal integrity was maintained with artificial tears four times daily. Substantial edema and erythema of the left upper eyelid were noted 3½ months after surgery (Fig. 1), but low-grade, very slowly progressing inflammatory changes may have been present as early as the third or fourth postoperative week. Cephalexin, 250 mg four times daily, was administered for 2 weeks without response. The gold weight was surgically removed, and the surrounding tissues were found to be boggy and edematous, without purulence or frank necrosis. Cultures of the site yielded light growth of coagulase-negative *Staphylococcus* and *Peptostreptococcus magnus*, both sensitive to cephalexin. Clinical signs of inflammation resolved immediately after implant removal.

Patient 2

A 62-year-old woman had complete left-sided facial nerve paralysis after resection of an acoustic neuroma. Periocular rehabilitation included repair of paralytic ectropion and placement of a 1.2-g gold weight in the left upper eyelid using a surgical procedure identical to that in patient 1. Three weeks after surgery, following complete resolution of her operative ecchymosis and edema, mild erythema and moderate edema of the left upper eyelid were noted (Fig. 2). Despite the administration of a topical antibiotic-corticosteroid ointment, the edema progressed, and an inflammatory reaction to the gold weight was suspected. At that point, the patient revealed an inability to wear gold jewelry because of "gold allergy."

Dexamethasone, 1.5 mg twice daily, was administered orally, with prompt resolution of the eyelid edema. However, inflammatory signs recurred whenever the corticosteroids were tapered. An s.c. injection of corticosteroids was offered but refused. Due to oral corticosteroid dependence, removal of the gold weight was considered. The patient was pleased with the function of the implant and resistant to its removal. After consultation with Dr. Richard Jobe, medical director of Meddev Corporation, a 1.2-g platinum lid load was manufactured. The gold weight was surgically removed along with its pseudocapsule, and the platinum weight was implanted using the previously described surgical technique. There has been no recurrence of eyelid inflammation. The explanted gold weight was applied to the patient's inner forearm for 24 h, with no cutaneous reaction.

Patient 3

An 80-year-old woman had left facial paralysis of 4 months' duration attributed to Bell's palsy by otolaryngology consultants. Seventh nerve stimulation on two occasions showed no response, and the prognosis for full recovery was determined to be poor. The patient complained of external irritation and excess lacrimation. Examination revealed a good Bell's phenomenon, but there was exposure keratitis of the lower fourth of the cornea because of lagophthalmos and lower eyelid laxity. Surgical repair included lateral tarsal strip and Tse (6) pro-

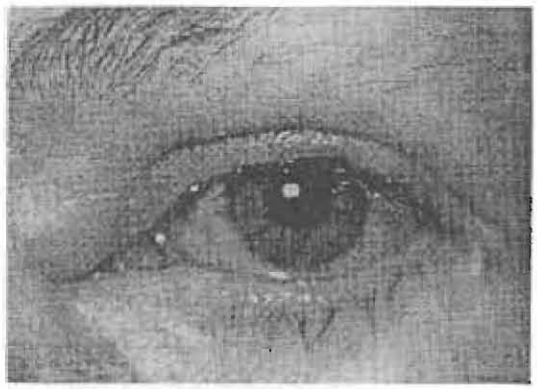

FIG. 2. Patient 2. Moderate edema of the left upper eyelid 3 weeks following gold weight implantation. The inflammatory signs responded to oral corticosteroids, but recurred on withdrawal. The gold weight was replaced with a platinum weight.

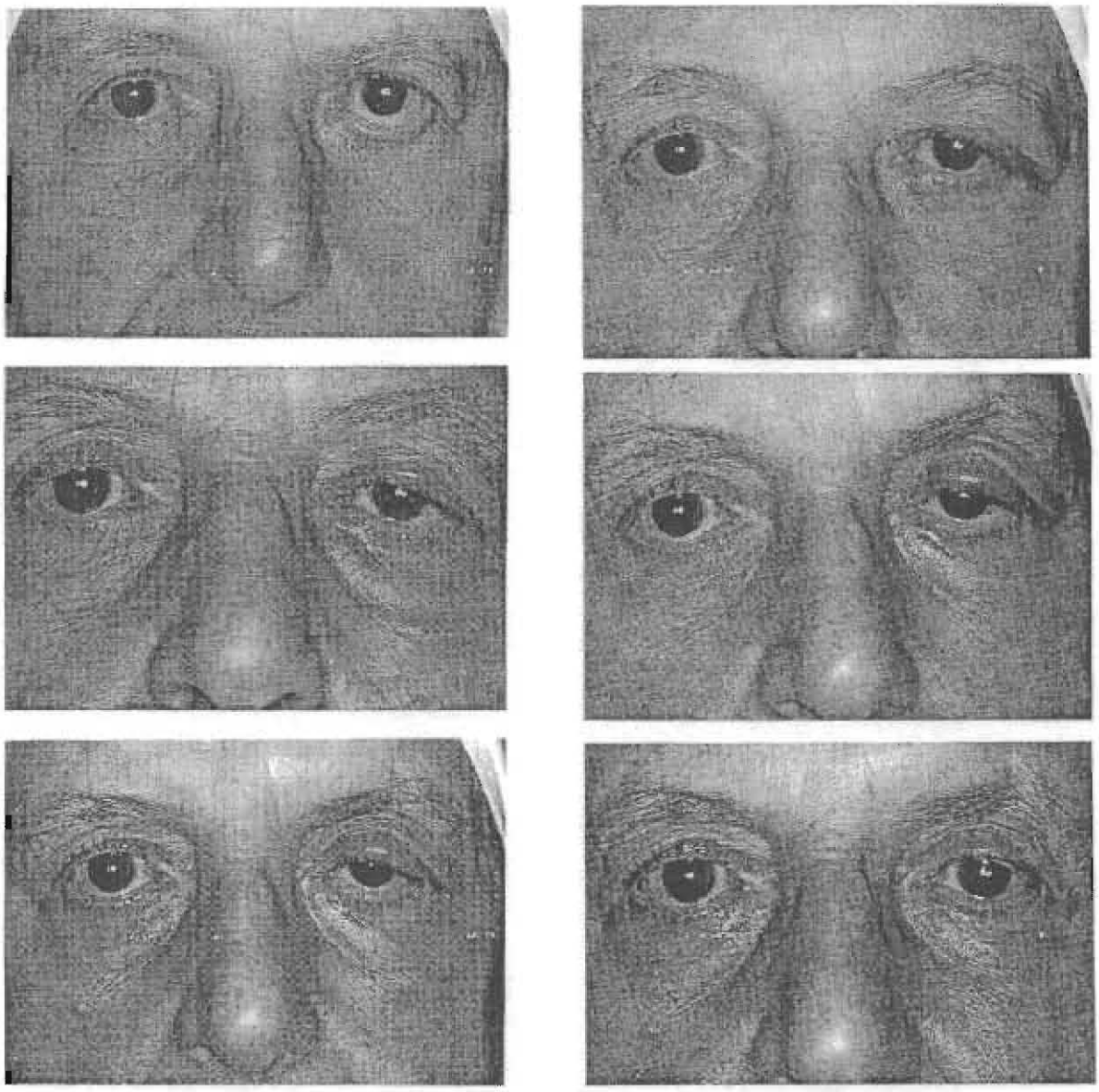

FIG. 3. Patient 3. Sequence shows insidious onset and gradual progression of left upper eyelid inflammation associated with gold weight implant. **Top left,** the patient preoperatively. **Top right,** 1 week after surgery. **Middle left,** 2½ weeks after surgery. **Middle right,** 5 weeks after surgery. **Bottom left,** inflammation did not respond to oral antibiotics administered for 1 week. **Bottom right,** 2 months following a single injection of corticosteroid.

cedures of the lower eyelid and implantation of a 1.6-g gold weight in the upper eyelid, using the techniques described in patient 1. Mild edema 1 week later was attributed to surgery, but slowly progressing inflammation was noted during the ensuing month. The inflammatory signs did not respond to cephalexin, 250 mg four times daily, administered for 1 week. Because a sterile inflammatory reaction to the gold weight was suspected, 0.5 cc of triamcinolone, 40 mg/cc, was injected into the left upper eyelid around the implant. Inflammatory signs resolved in the ensuing 5 to 10 days, and there has been no recurrence of inflammation in an additional 9 months of follow-up (Fig. 3).

Patient 4

A 71-year-old woman underwent gold weight loading of the right upper eyelid for seventh nerve paralysis related to resection of an acoustic neuroma. There were no clinical signs of inflammation, but the implant was exchanged (0.8 g for 1.2 g) 9 months after surgery because of induced ptosis. The pseudocapsule was submitted for histopathological examination.

PATHOLOGIC FINDINGS

In patient 4, without clinical signs of inflammation, the specimen consisted of a capsule composed of mature collagenous fibers and infiltrated by small numbers of lymphocytes arrayed between the fibers. At the tissue-prosthesis interface was an amorphous eosinophilic coagulum containing small numbers of inflammatory cells (Fig. 4).

In patients 1 and 2, with clinical inflammation, the eosinophilic coagulum was much thicker. There was an intense chronic inflammatory infiltrate into the periprosthetic tissues, which was predominantly lymphocytic. Phenotypically, the majority of lymphocytes were T cells. Admixed with the lymphocytes were small numbers of plasma cells and histiocytes. Capillaries had prominent endothelial cells (Fig. 5).

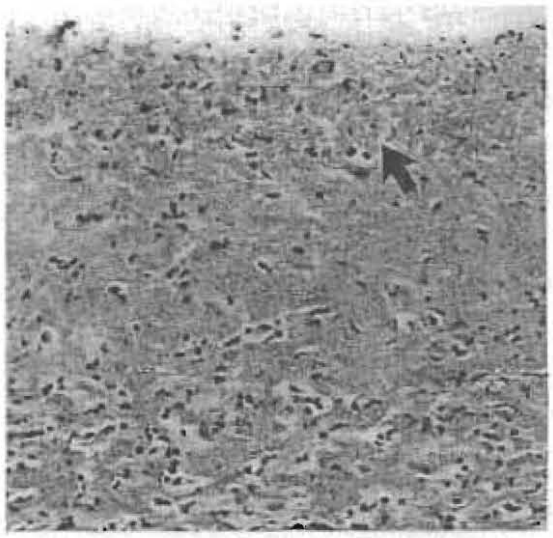

FIG. 4. Patient 4. Capsule of gold weight implant from a patient without clinical signs of inflammation. The capsule consists of mature collagenous fibers and small numbers of lymphocytes. There is a narrow eosinophilic coagulum at the gold-tissue interface (arrow).

DISCUSSION

Gold weight implantation has been successful in correcting exposure keratitis in cases of severe lagophthalmos secondary to seventh nerve paralysis. The procedure is usually performed under local anesthesia with relatively simple techniques. Gold provides a good color match and an acceptable cosmetic result under thin eyelid skin. Although the procedure is usually reserved for permanent paralysis, the weight can be easily explanted if orbicularis function returns. Infection has not been a significant complication, and exposure is rare if the weight is placed behind orbicularis muscle and sutured to tarsus (3–5,7). Ptosis may occur, but can usually be minimized by selecting an appropriate weight preoperatively, based on external application of implants to benzoin-prepared eyelid skin. Astigmatism caused by the weight of the implant on the cornea has been one of the few reasons for implant removal.

We have described three patients with moderate eyelid edema and erythema in response to gold weight implants. Because of insidious onset and very gradual progression, the interval between implantation and the inflammation was difficult to pinpoint. The onset may have even dovetailed with the resolution of operative edema. Very obvious changes, which prompted treatment, were present by 3 to 12 weeks. Although cultures in patient 1 were mildly positive, we suspect contamination rather than clinically significant infection: inflammation had remained very mild for several weeks before any treatment; both organisms were sensitive in vitro to the administered antibiotic; and the histopathological findings were not typical for bacterial infection. Based on the experience with patient 1, patients 2 and 3 were treated with corticosteroids, and the prompt resolution of inflammatory signs also militated against infection.

The histopathological features in patients 1 and 2 are of interest, particularly in comparison to the findings in patient 4, who had no clinical inflammation. The "normal" response to gold weight implantation in the eyelid appears to be low-grade lymphocytic infiltration and fibrosis (Fig. 4), a finding previously noted (5). In the cases with clinical inflammation, however, a much more exuberant lymphocytic infiltrate was observed (Fig. 5). Plasma cells were occasionally noted, but were not prominent. Foreign body giant cells were focally identified in one of the two cases, but may have

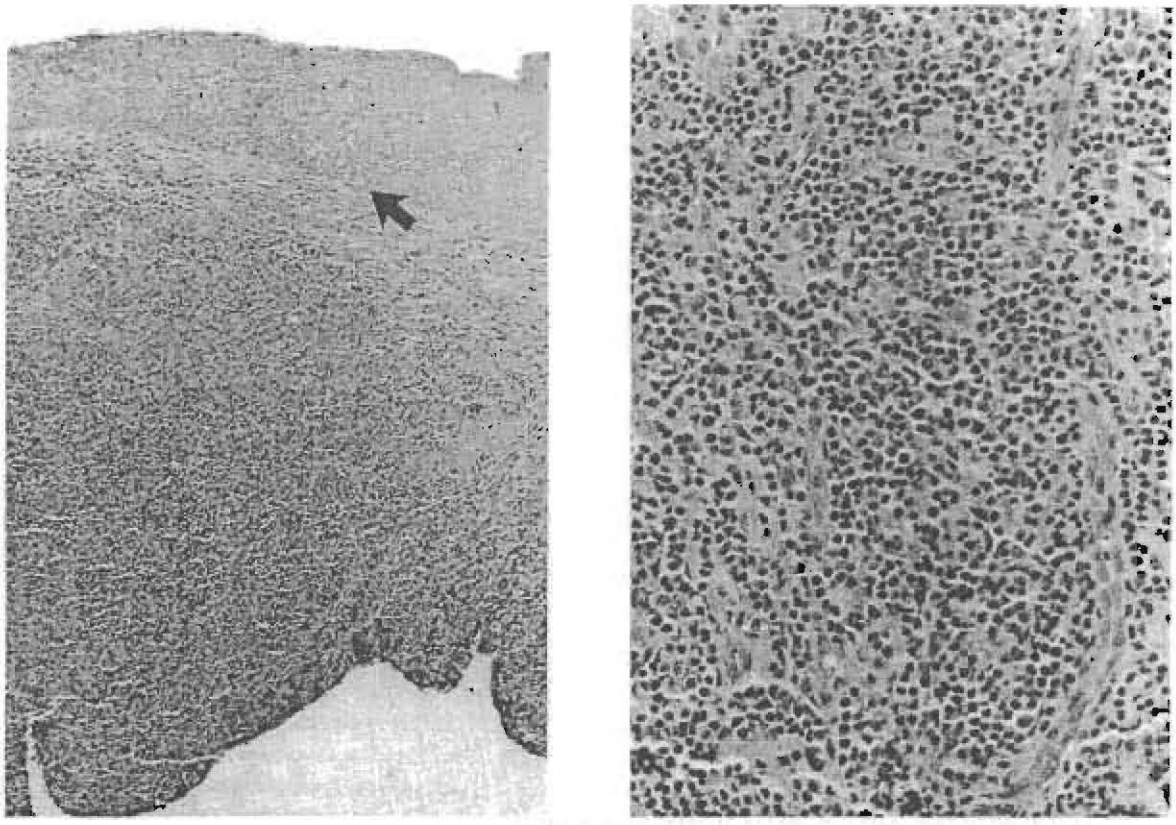

FIG. 5. Patient 2. Tissue adjacent to prosthesis in patient with clinical inflammatory response. Left, interface coagulum (arrow) is relatively thick. There is heavy infiltration by chronic inflammatory cells. ×100. Right, higher magnification shows the inflammatory cells to be mostly small lymphocytes with small numbers of histiocytes. Capillaries show plump reactive endothelial cells. ×250.

been a response to suture material rather than the implant.

It is unclear whether these clinical reactions represent allergy in constitutionally sensitized patients. Only one of our patients gave a history of cutaneous gold allergy. In general, allergy to pure metallic gold is rare because of lack of solubility (8–10). Histologically, these reactions (patients 1 and 2) appear to be an exaggeration of a lymphocytic response that might he elicited by all gold weight implants (patient 4). Perhaps the implants in our three patients were somehow rendered more soluble or toxic. Nevertheless, known gold allergy should be considered in the preoperative history.

Based on the findings of this study and the lack of documented infections in earlier reports, we recommend the following approach. If rapidly progressive inflammation is noted within the first postoperative week, or later if associated with implant exposure, bacterial infection should be suspected and treated with appropriate antibiotics. If inflammatory signs develop gradually during several weeks following surgery, a noninfectious reaction should be suspected. Although removal of the gold implant and substitution with an alternative material, such as platinum, may ultimately be necessary, suppression of the inflammation may also be possible with local corticosteroid injection. In equivocal cases of bacterial infection that cannot be definitely excluded, we recommend a 1- to 2-week course of oral antibiotics before corticosteroid administration.

Note added in proof:

Since acceptance of our article, we have been informed of three additional patients with inflammatory reactions to gold weight eyelid implants (G. B. Bartley, personal communication).

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