Eyelid Necrosis Following Intralosal Corticosteroid Injection for Capillary Hemangioma

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ABSTRACT

The intralosal injection of corticosteroids has been employed successfully in treatment of adnexal neonatal hemangiomas since 1979. This form of treatment is easily administered, is repeatable and free from serious complications. We present an exceptional case in which full-thickness eyelid necrosis ensued following Intralosal injection of corticosteroids in a capillary hemangioma. After eyelid reconstruction the patient’s visual axis has remained unobstructed, and amblyopia has been thus far averted.

Capillary hemangiomas are benign tumors which tend to involute spontaneously after a period of variable growth. They appear shortly after birth and continue to grow until after the first year of life. The usual course is for spontaneous gradual involution over the next four to five years. Treatment is normally not required unless these lesions are located in the upper eyelid where they may cause occlusion or refractive amblyopia. For these situations many treatment modalities have been described. These include surgical excision, radiotherapy, injection of sclerosing agents, radon seed implantation, and the administration of systemic corticosteroids. All of these have well-known serious disadvantages.1 Intralosal use of corticosteroids for infantile adnexal hemangiomas was introduced by Kushner2 in 1979, and to date this mode of treatment has been free of serious complications.3-6 We report the unusual occurrence of eyelid necrosis following the intralosal use of corticosteroids. This complication has not been previously described in the literature.

CASE REPORT

A four-week-old Haitian girl was seen on February 18, 1985, with a one-week history of progressive swelling of the right upper eyelid and forehead. Her birth, growth and development had been normal.

Examination revealed a large blue-red mass involving the right upper eyelid, almost completely occluding the visual axis (Figure 1). She did not fixate well with the right eye. Accurate retinoscopy was difficult due to occlusion of the visual axis by the tumor. The clinical diagnosis of capillary hemangioma was made.

The hemangioma continued to increase in size and completely occluded the visual axis during the next three weeks. On March 13, 1985, 40 mg (1 ml) of triamcinolone suspension and 0 mg (1 ml) of betamethasone sodium phosphate were injected into the lesion.

During the next three weeks there was progressive, full-thickness necrosis of the lateral half of the right upper eyelid following initial tumor regression (Figure 2). On April 10, 1985, the eyelid was reconstructed by a tarsoconjunctival rotation flap and a bilobed skin rotation flap from the medial eyelid. Tissue fragments submitted for histological examination showed pyogenic
granuloma formation, signs of acute and chronic inflammation, and ulceration. Subsequent to the surgery, the infant's visual axis remained unobstructed.

At 4 ½ months of age the patient developed 2 mm of downward globe displacement on the involved side. Computed tomography showed a diffuse vascular anomaly in the lateral and superior aspects of the orbit — compatible with orbital hemangioma (Figure 3). Visual evoked responses in July 1985, showed only a slight difference between the two eyes with predicted visual acuities of 20/30 to 20/50 in the right eye and 20/20 to 20/40 in the left eye. One month later the downward displacement of the globe appeared slightly greater. A course of systemic prednisone (20 mg orally per day) was begun and slowly tapered.

When last examined on February 27, 1986, the visual axis was not obstructed, and her fixation was equal in both eyes. There was a small amount of lagophthalmos with no corneal decompensation. The right globe was no longer displaced downward, and upgaze was not restricted. The lateral aspect of the right upper lid was still moderately deformed. Cycloplegic retinoscopy revealed: right cxc. +4.50 –1.75 at 45; the left cxc. +3.00 sphere.

**DISCUSSION**

In 1979, Kushner\(^2\) successfully treated three of four patients with periorcular capillary hemangiomas with intralesional corticosteroids. He subsequently treated 21 additional patients with good results and minimal complications.\(^3\)\(^-\)\(^4\) Complications in his series were limited to subcutaneous deposits of solid corticosteroid material in several patients which resolved less than two months after the injection. The experience of others\(^5\)\(^-\)\(^7\) with intralesional corticosteroid therapy has been equally gratifying.

Complete necrosis, of the eyelid after intralesional corticosteroid therapy has not been previously reported. In Kushner's series,\(^3\)\(^-\)\(^4\) however, one of his patients had a substantial area of cutaneous necrosis overlying an eyelid hemangioma. This progressed to ulceration as the tumor regressed following intralesional therapy. Kushner did not feel that this process was related to the corticosteroid injection since the cutaneous necrosis existed prior to the injection. Also, this complication did not occur in the 24 other patients he treated. His patient had cutaneous necrosis prior to injection, and our patient had cutaneous necrosis progressing to a full-thickness eyelid necrosis after corticosteroid injection — we employed in our case the same dose of corticosteroid used by Kushner.

Jakobiec and Jones\(^1\) state that the eyelid may be lost in patients with capillary hemangiomas due to thrombosis or necrosis of a lid lesion. Such lesions may spontaneously ulcerate and often heal with scarring.

Review of the dermatologic literature revealed only one case of tissue necrosis following intralesional corticosteroid use. Abdel-Fattah\(^8\) reported complete sloughing of a presternal keloid treated with intralesional triamcinolone acetate. He referred to this complication as being "unexpectedly spectacular and difficult to explain." He postulated that his patient may have had an unusual sensitivity to triamcinolone.

Physicians using facial, intranasal and periorcular corticosteroid injections should be aware that intraarterial injection may cause disastrous consequences. Retinal embolization and blindness have been reported following forceful injections into peripheral
branches of the ophthalmic artery. Similarly, injection of repository corticosteroids into anastomotic branches from the external carotid artery may reach the ocular circulation by retrograde flow. Clinical observation has not documented retinal embolization as a complication in the treatment of periocular hemangiomas; however, precautions should be taken to prevent its occurrence.

While the necrosis of the eyelid of our patient followed the intralensal steroid injection, there also was concomitant progression of the orbital hemangioma. The rapid enlargement of this mass may well have led to spontaneous thrombosis within the lesion with the resultant necrosis of the eyelid. The intralensal injection of the corticosteroids prior to this event could have been coincidental. However, it is possible that the vasoconstrictive effect of the steroids could have enhanced thrombosis already in progress. The basis for this statement can be found in a report by Zweifach, Shore, and Black. They hypothesize that corticosteroids sensitize the vascular bed to vasoconstrictors naturally occurring in the body. Jakobiec and Jones refer to this as "pharmacologic ligation." This vasoconstrictive effect would seem to be more potent in a rapidly expanding capillary hemangioma that is outstripping its blood supply.

Under most circumstances full-thickness eyelid necrosis would be a disastrous complication. With prompt eyelid reconstruction, our patient was afforded an unobstructed visual axis without corneal decompensation. Thus far, she has maintained good visual acuity despite her extensive lesion.

SUMMARY
We report full thickness eyelid necrosis in a six-week-old infant with capillary hemangioma of her upper eyelid after intralensal corticosteroid injection. The possible causes of this complication are discussed.

ADDENDUM
A poster was presented by Merritt at the annual meeting of the American Academy of Ophthalmology, 1985. He showed a case of left upper eyelid necrosis and corneal ulceration three days following two intralensal corticosteroid injections given one week apart. He did not postulate the cause for this event.

REFERENCES