Case Report

Transient Traumatic Neurogenic Ptosis In An 8-Year-Old Boy: A Case Report

Dohvoma Viola Andin 1,2, Ebana Mvogo Stève Robert 1, Nguena Marie Blanche 2, Gouking Patricia 1, Owono Didier 1,2, Epee Emilienne 1,2, Ebana Mvogo Côme 1,2.

INTRODUCTION

Drooping of the upper eyelid known as ptosis can be congenital or acquired. Based on the aetiology, ptosis may be myogenic, aponeurotic, or neurogenic [1]. Post-traumatic ptosis may be classified as follows: ptosis occurring during lid avulsion, ptosis associated with fractures of the orbital roof and with foreign bodies, post-contusional ptosis, cicatricial ptosis, neurogenic ptosis and post-surgical ptosis [2]. Transient traumatic neurogenic ptosis, which is uncommon, is a self-limited ptosis which recovers after a certain period of time with conservative management or observation.

CASE REPORT

An 8-year old boy, with a non-contributory past history, was seen 24 hours following contusion to the right upper eyelid. Eyelid contusion resulted from the boy hitting the eyelid against the handle of a door during play at home with friends. Visual acuity in the right eye was 0.2 (eyelid was lifted) and was 1.0 in the left eye. Upon inspection, the following were observed: oedema of the right upper eyelid with no visible palpebral aperture (figure 1), normal extraocular motility and pupillary responses without anisocoria. Palpation of the orbital rims was unremarkable. Slit lamp examination of the anterior segments of both eyes was normal. Intraocular pressure (IOP) was 26mmHg OD and 17mmHg OS. Fundus examination was normal in both eyes. Maxillofacial examination including orbital CT scan was normal.

An initial diagnosis of oculo-palpebral trauma associating post contusive ptosis and raised IOP was made. Treatment comprised oral non-steroidal anti-inflammatory drugs (NSAID), topical timolol 0.5% eye drops and topical antiseptic eyedrops. One week later, the IOP was normal and the oedema had resolved completely. Complete ptosis was present with no levator function, consistent with loss of innervation (figure 2). Vision was 1.0 with eyelid lifted. Timolol and other medication were discontinued.

IOP remained normal at week 2 with persistent complete ptosis. At week three, ptosis improvement was noticed (figure 3). Post traumatic neurogenic ptosis was considered in the presence of spontaneous improvement, hence patching of the left eye recommended. Complete recovery was present after the 4th week (figure 4).
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DISCUSSION

Transient traumatic neurogenic ptosis is a rare entity for which there is no consensual definition in the literature. According to Li et al, it has two characteristics: occurs secondary to direct or indirect injury of the upper eyelid and its innervation; and is self-limited with recovery after a certain period of time with conservative treatments or just observation [3]. There are reports of isolated ptosis after medial wall reconstruction of the orbit [4,5]. Our patient had undergone a direct trauma to the upper eyelid. Other cases with direct trauma to the eyelid have been reported [6-8].

Transient traumatic neurogenic ptosis is caused by injury of the terminal branch of the superior division of the oculomotor nerve that supply the levator palpebrae superioris (LPS) muscle. The mechanism could be mechanical from stretching occurring in direct trauma or indirect trauma [3,6]; or ischemic occurring during orbital wall reconstruction [4]. The injury results in neurapraxia, which temporarily interrupts conduction of nerve impulses without axonal degeneration.

It is important to initially observe patients with traumatic ptosis without surgical intervention, because acute factors such as haemorrhage, neurapraxia, and oedema are temporary. Spontaneous resolution or improvement often occurs in these patients over the course of 6 months. In our patient, ptosis persisted despite complete regression of oedema; it later resolved spontaneously, suggesting neurapraxia to terminal fibres of the superior division of the third cranial nerve as the mechanism of insult.

Ptosis occurring aponeurotic disinsertion, LPS rupture and third nerve compression from sphenoid fracture will not improve over time. Sphenoid fracture might be one of the potential mechanisms involved in traumatic isolated oculomotor nerve palsy after mild head injury. Surgical decompression should be considered when there is evidence of bone compression of the superior orbital fissure [9]. Bone compression of the third cranial nerve should be suspected clinically in the presence of associated ophthalmoplegia (internal and/or external).

Most cases of transient traumatic neurogenic ptosis do not require specific treatment [3,6]. Corticosteroid administration has also been used by some authors [5].

CONCLUSION

Transient traumatic neurogenic ptosis is an entity which usually recovers fully within weeks. It is important to initially observe all patients with traumatic ptosis without surgical intervention. Spontaneous resolution or improvement often occurs over the course of 6 months. Surgical repair of patients with non-resolving traumatic ptosis should be done only after at least 6 months of observation.

DISCLOSURE

The authors report no conflicts of interest in this work.
REFERENCES