OUTCOME OF LEVATOR REATTACHMENT SURGERY FOR BLEPHAROPTOSIS IN BLEPHAROCHALASIS
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ABSTRACT

OBJECTIVE
To report clinical profile of blepharochalasis and assess the surgical outcomes of levator reattachment in chronic blepharochalasis.

METHODOLOGY
A retrospective chart review of 13 patients with acute and chronic blepharochalasis was made and the diagnosis was made based on the typical clinical findings. Patients with acute blepharochalasis were managed conservatively those with chronic blepharochalasis underwent levator reattachment surgery for the correction of blepharoptosis.

RESULTS
Age at presentation varied from 8 years to 13 years with no sex predilection. 4 patients presented in the acute stage and 9 patients presented in the chronic stage. All patients in the chronic stage had blepharoptosis along with other features of connective tissue laxity and underwent levator reattachment surgery with good postoperative outcomes.

CONCLUSION
In the acute stages, blepharochalasis is managed conservatively. However, in the chronic stages, management is essentially surgical. Levator reattachment is an excellent surgical procedure, which provides satisfactory postoperative outcomes.

KEYWORDS
Blepharochalasis, Levator Reattachment.

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INTRODUCTION: Blepharochalasis is an unusual, but a distinct entity seen in the oculoplastic clinic. First described by Beer in 1807, the term blepharochalasis was coined by Fuchs in 1896. The term blepharochalasis literally means ‘eyelid slackening’ and as the name suggests, the disease entity is characterised by some very typical features in the chronic stages. Two clinical presentations of blepharochalasis are known-an acute presentation and a chronic presentation. Acute attacks usually begin during puberty and they reduce in frequency as the patient ages. The patient in the acute stage presents with recurrent episodes of upper lid oedema (which occasionally involves both lids), which is painless, non-erythematous, non-pitting, and transient. An acute attack usually lasts 24 to 48 hours and disappears thereafter.

Over a period of time, which may range for over two to several years, the frequency of acute attacks reduce and a stage of chronicity sets in. The chronic stage as the name suggests is characterised by certain distinct features of slackening of the eyelid tissue and its anchoring tendons.

The chronic phase is further subdivided into a hypertrophic form wherein there is fat prolapse due to laxity of orbital septum and an atrophic form showing fat atrophy. The pathogenesis of blepharochalasis is not clear. Histopathological examination shows the presence of perivascular and interstitial infiltration of the dermis with marked decrease in elastic fibres on van Gieson staining. An immunoglobulin A mediated mechanism has been proposed for the degradation of elastic fibres. While the management in the acute stages is conservative, the management in the chronic stages is essentially surgical. There are no set surgical protocols in the management of chronic blepharochalasis. Various studies have reported satisfactory postoperative surgical outcomes with levator reattachment and blepharoplasty.

MATERIAL AND METHODS: A retrospective chart review of 13 patients diagnosed with acute and chronic blepharochalasis was made. Patients who presented to Minto Ophthalmic Hospital between January 2009 and June
2015 were part of this study. Diagnosis of both acute and chronic blepharochalasis was made clinically. A diagnosis of acute blepharochalasis was made in patients presenting with recurrent episodes of painless, non-pitting, non-erythematous lid oedema, which disappeared in 48 to 72 hours (Fig 1a, 1b). A diagnosis of chronic blepharochalasis was made in patients presenting with cigarette paper wrinkling of skin, blepharoptosis, hollowing of the superior sulcus, nasal fat pad atrophy, fat prolapse, pseudoepicanthal folds, and rounding of the lateral canthus (Fig. 2a, 2b).

All patients with blepharoptosis underwent levator reattachment surgery in the chronic stage after more than 6 months of an acute attack. Under local anaesthesia, an anterior approach was used. Lid crease incision was made. In patients in whom the lid crease could not be identified, the skin incision was made 8 mm above the lid margin. A horizontal incision in the lid crease was made in the skin-ocularis muscle layer parallel to the muscle fibres onto the tarsal plate and the orbital septum was identified. The orbital septum was opened and preaponeurotic fat was dissected to identify the levator aponeurosis. Any thinning of the aponeurosis, dehiscence, and disinsertion of aponeurosis was noted on table. Dehiscence of the aponeurosis was repaired using 6-0 Vicryl suture. Using a double armed 6-0 Vicryl suture on a spatulated needle, levator reattachment was performed. Partial thickness bites about 2 mm inferior to the anterior surface of the superior border of the tarsus was taken and both the ends of the double arm suture were then passed through the levator aponeurosis. A temporary tie was placed and the lid height was titrated on table. After titration of lid height, a permanent tie was placed. Two similar sutures were then placed-nasal to and temporal to the first suture. The lid contour was noted for any notching and corrected intraoperatively. Any excess redundant skin was excised. Dog earing at the edges of the wound was corrected. The new lid crease was reformed as the skin incision was closed with a 6-0 Vicryl suture using continuous suturing technique-this was performed by incorporating the edge of the levator aponeurosis in the skin incision. Antibiotic ointment was applied and the skin sutures were removed one week postoperatively and the patient followed up regularly thereafter.

RESULTS: Of the 13 patients seen in our clinic, 4 patients presented in the acute stage and 9 in the chronic stage (Fig. 3). 7 of our patients were male and 6 female (Fig. 4). Unilateral presentation was seen in 8 of our patients with the remaining 5 of our patients showing bilateral symptoms and signs (Fig. 5a, 5b). Amongst those with unilateral presentation, 7 patients had involvement of the left eye.
The age at presentation in both stages varied between 8 to 52 years. Patients in the acute stage had an earlier presentation with mean age at presentation at 17.5 years and those in the chronic stage presented later on with mean age at presentation at 32 years. All of the 4 patients who presented in the acute stage were treated conservatively with cold compresses and oral antihistaminic drugs. Lid swelling was seen to subside in all patients within 24-48 hours of onset of symptoms. All patients in the chronic stage presented with moderate (n=3) to severe degree of (n=6) of blepharoptosis, which was corrected by levator reattachment surgery. As described in detail previously, under local anaesthesia, incision was made along the lid crease (or 8 mm from the lid margin in cases where the crease was higher up). Dissection was performed through the orbicularis, septum identified and opened, preaponeurotic fat retracted, and the levator aponeurosis was identified. Intraoperatively, thinning of the levator aponeurosis was noted in all cases (Fig 6). The lid height was titrated intraoperatively. Excess lax skin was excised and lid crease was reformed at the time of wound closure. All patients were evaluated on first postoperative day, one week and one month postoperatively (Fig 7). Satisfactory postoperative outcomes in terms of lid height and contour was obtained irrespective of the preoperative severity of blepharoptosis. However, one patient presented with recurrent lid oedema in the operated eye suggestive of an acute attack (Fig 8). She was managed conservatively. No subsequent attacks were documented later in her.
Blepharochalasis from other causes can be differentiated by past history of systemic features. Although a plethora of trigger factors have been reported, the aetiopathogenesis in most cases is usually obscure. A differential diagnosis of acute blepharochalasis should be kept in mind while evaluating any patient presenting with recurrent eyelid oedema. Even when left untreated, an acute episode resolves in 24–48 hours. Histopathological examination reveals dilated blood vessels within the dermis, which are leaky. Therefore, treatment in the acute stage reduces the amount of oedema and subsequent reduction in elastic tissue damage within the dermis. Studies have reported that treatment with oral acetazolamide may reduce the frequency of acute attacks.

It is proposed that oral acetazolamide regulates the size of the fenestrae of certain capillary beds, thus reducing leakage. However, none of our patients were treated with acetazolamide. All our patients who presented in the acute stage were treated with cold compresses and oral antihistaminics. The symptoms subsided within 24–48 hours of onset with the foresaid management. The diagnosis in the chronic stage is straightforward in view of distinct clinical findings of cigarette paper wrinkling of the eyelid skin, blepharoptosis with deepening of the superior tarsal sulcus, blepharophimosis, laxity of the canthal tendons, atrophy of the nasal fat pads, and lacrimal gland prolapse. All the aforementioned features of eyelid slackening are attributed to damage of the elastic fibres within the connective tissues with recurrent episodes of swelling. Definitive management in the chronic stage is essentially surgical.

While planning surgery in chronic cases, timing is crucial to prevent acute attacks postoperatively, which may compromise surgical outcomes. Ideal timing for surgery would be after 6 months to a year of an acute attack. Patients with chronic blepharochalasis in our study were therefore posted for blepharoptosis correction at least 6 months following an acute attack. Blepharoptosis with good levator function is an almost consistent finding seen in all patients in the chronic stages. It occurs due to dehiscences within the levator aponeurosis. In addition, the laxity and disinsertion of the canthal tendons gives rise to acquired blepharophimosis. There is also laxity of the orbital septum, which accounts for fat prolapse and lacrimal gland prolapse. Atrophy of the nasal fat pad maybe seen giving rise to pseudoepicanthal folds. Levator reattachment surgery was performed in all our patients who presented with blepharoptosis in the chronic stages. Good postoperative outcomes in terms of lid height and contour were obtained in our study consistent with the findings of previous studies.

The advantage of levator reattachment lies in the fact that it is possible to titrate the lid position intraoperatively. In addition, as it is done through an anterior approach, ptosis correction can be combined with an upper lid blepharoplasty. The anterior approach also helps to repair lacrimal gland prolapse, which can be done through the same incision. Studies have reported over correction with levator resection surgery. The over correction might occur due to the fact that the lower edge of the levator aponeurosis may be damaged due to recurrent bouts of swelling and stretching and it might be difficult to identify the true edge of aponeurotic disinsertion. It is important to clinically differentiate chronic blepharochalasis from other causes of blepharoptosis, which may present in similar demographic populations as it has an impact on management and prognostication. An important sinister cause of acquired blepharoptosis in the elderly is myasthenia gravis. Clinically, ocular myasthenia can be differentiated from blepharochalasis by the presence of fatigable and variable amounts of unilateral or bilateral ptosis, which may be associated with diplopia, Cogan’s twitch sign, and systemic features.

The diagnosis of myasthenia can be confirmed by performing ice pack test, edrophonium test, repetitive nerve stimulation test, single fibre EMG, and estimating levels of anti-acetylcholine receptor antibodies in serum. Another cause of blepharoptosis, which may mimic chronic blepharochalasis is senile aponeurotic ptosis. Chronic blepharochalasis can be differentiated by past history of recurrent acute episodes and by the typical changes described in the chronic stages along with earlier age at presentation compared to patients presenting with senile aponeurotic ptosis.

CONCLUSION: The general ophthalmologist should be aware of blepharochalasis, which is a distinct clinical entity with typical clinical presentations in the acute and chronic stages of the disease process. The hallmark of the acute stage is the recurrent bouts of painless lid oedema, which
usually resolves with or without treatment in 24 to 48 hours. The reason for treatment in the acute stages is to try and reduce the increased permeability, which may lead to elastic tissue damage and eventual progression to the chronic stages. It is important to be aware of this entity to unnecessarily avoid treating a patient with acute blepharochalasis with steroids. The chronic stage of blepharochalasis is identified by the typical clinical features described. The management in the chronic stages is essentially surgical. Levator reattachment surgery through an anterior approach is an excellent procedure for the correction of blepharoptosis. In addition to correction of ptosis, excess redundant skin can also be excised. However, in all cases of chronic blepharochalasis, the timing of surgery is crucial; ideal timing would be 6 months to a year after an acute attack to prevent recurrent acute attacks postoperatively, which would otherwise compromise surgical outcomes.

REFERENCES