Ptosis and Blepharoplasty Surgery

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Acquired senile ptosis of the upper eyelid is a common condition in the elderly population and is often accompanied by varying amounts of dermatochalasis, which may mask its presence. Blepharoplasty of the ptotic lid, without repair of the ptosis, may result in exaggeration of the drooping eyelid postoperatively. Acquired senile ptosis is produced by dehiscence or disinsertion of the levator aponeurosis and is characterized by a high or absent eyelid crease, thinning of the upper eyelid tissue, and normal levator function. Frequent causes of acquired ptosis include dermatochalasis, eyelid edema from trauma or allergies, previous ocular surgery, or use of contact lenses. When discovered, repair of the ptosis should be performed at the time of blepharoplasty to produce the best functional and cosmetic result. It is important for every surgeon who performs blepharoplasties to be aware of the presentation and management of acquired senile ptosis for consistent surgical results.


Acquired upper lid ptosis is a common condition in the elderly population, and is often accompanied by varying amounts of dermatochalasis. Blepharoplasty of the ptotic lid may result in exaggeration of the drooping eyelid postoperatively, with a poor cosmetic result. It is important for every surgeon who performs blepharoplasties to be able to recognize this problem and manage it appropriately. To that end, important aspects of acquired senile ptosis, including etiology, pathophysiology, physical diagnosis, and surgical repair, will be discussed. Relevant anatomy of the eyelid will also be presented.

ANATOMY AND PHYSIOLOGY

The anatomy of the upper eyelid is the real key to understanding ptosis and ptosis surgery (Fig 1).1,2 The upper eyelid consists of several layers of tissue that are all distinct structures. The outermost layer of the eyelid is the skin, which is unique in that it is the thinnest skin in the body, with essentially no subcutaneous fat. The orbicularis oculi muscle lies just below the skin and is divided into a preタルサル and preseptal portion. The lower third of the preタルサル segment is firmly adherent to the lower third of the tarsus, while the remainder is attached to the levator aponeurosis. The orbital septum, found just beneath the preseptal portion of the orbicularis oculi muscle, is a fibrous layer of tissue that originates from the periorbital peristeme and extends downward to fuse with the levator aponeurosis, about 2 to 3 mm above the upper border of the tarsus. The next structure identified is the preaponeurotic fat pad of the upper eyelid, which is found in a pocket beneath the orbital septum and lying atop the levator aponeurosis. This fat pad is a key anatomic structure in distinguishing the orbital septum from the levator aponeurosis, two structures that may have a similar appearance at surgery. The levator palpebrae muscle and its aponeurosis are identified with upward retraction of the pad. The muscle originates from the lesser wing of the sphenoid at the orbital apex and extends forward for 40 mm, with its aponeurosis continuing for another 15 mm. At approximately 10 mm above the tarsus, the muscle divides into an anterior lamella, which becomes the fibrous levator aponeurosis, and a posterior lamella. The aponeurosis inserts on the anterior interior two-thirds of the tarsus as well as the preタルサル orbicularis oculi muscle and skin. The posterior lamella contains nonstriated muscle fibers, Müller's muscle, and inserts on the superior tarsal border. The innermost layer of the eyelid is conjunctiva, which is closely adherent to Müller's muscle. The last major structure of the upper eyelid is the tarsus, a plate of dense fibrous and elastic tissue, which provides support for the upper eyelid. It measures 10 mm in height and provides attachments for the levator aponeurosis, Müller's muscle, and conjunctiva.

Innervation of the eyelid includes the seventh nerve, which supplies the orbicularis oculi muscle, and the third nerve, which supplies the levator muscle. Müller's muscle is under sympathetic control and has a primary function of maintaining eyelid tone.

ETIOLOGY

There are many etiologies of acquired ptosis, but the list is greatly reduced when considering the otherwise healthy elderly population. In patients with good levator function, the usual cause of acquired ptosis is thinning and stretching or disruption of the levator aponeurosis, which occurs when the minimal attachment of the aponeurosis to the tarsal plate is insufficient to withstand any excessive pull. There are many common

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conditions that may cause a levator dehiscence or disinsertion. The added weight of excessive skin in dermatochalasis may produce a levator dehiscence and is probably the most common cause of acquired senile ptosis. Marked eyelid edema from trauma, surgery, or allergies may also result in a levator disinsertion. Mechanical trauma to the eyelids from the use of contact lenses may produce a levator disinsertion at any age. Myasthenia gravis should always be considered in the differential diagnosis of acquired ptosis, particularly in the patient with severe ptosis bilaterally. Regardless of the etiology of levator dehiscence or disinsertion, surgery can usually produce good, reliable results.

EXAMINATION OF THE PATIENT WITH PTOSIS

A careful history should be obtained and a physical examination should be performed when a ptotic lid is discovered. The history should include the time of onset, as a congenital ptosis usually involves a dysfunctional levator muscle and is treated differently from an acquired ptosis. A history of other coexistent symptoms, particularly neurologic or muscular, should be obtained to rule out more serious disease processes, such as myasthenia gravis, tumors, or other central nervous system disorders. A history of eyelid edema, surgery, or the use of contact lenses is suggestive of levator aponeurosis disinsertion.

Once a history of benign acquired ptosis is established the physical examination should take place. There are several characteristics that define an acquired senile ptosis on physical examination. There should be a high or absent eyelid crease and thinning of the upper eyelid tissue to the extent that the iris may be visible through the closed eyelid. It is necessary that the levator muscle function normally to diagnose a case as one of acquired senile ptosis. Eyes should be examined in upward and downward gaze, as well as in the primary position. In acquired senile ptosis, the amount of ptosis remains the same in relation to the normal eyelid in all levels of gaze. The amount of ptosis should be documented either by direct measurement or by estimation. For example, the normal eyelid covers the superior limbus by 1 to 2 mm, and the distance from the superior limbus to the midpupillary point is 5.5 mm, so an eyelid at the midpupillary line would represent a ptosis of 3.5 to 4.5 mm. A ptosis of 1 to 2 mm is considered mild; 3 mm, moderate; and 4 mm or greater, severe. Levator muscle function should be measured in all patients with lid ptosis. To evaluate function of the levator muscle, pressure is first placed over the brow with the thumb or finger to prevent participation of the frontalis muscle in lifting the eye.
The patient is then asked to look in an extreme downward, and then upward, gaze. The distance the eyelid travels between these two positions is measured in millimeters and represents the function of the levator muscle. Normal excursion, which is found in acquired senile ptosis, is 15 mm and is considered excellent levator function. Less than 4 mm is regarded as poor levator function; 5 to 7 mm, fair function; and greater than 8 mm, good function. Last, photographic documentation should be obtained of the eyelids in primary, upward, and downward gaze.

Ophthalmologic consultation is recommended in the initial workup of all patients with ptosis. In this way, the degree and type of ptosis may be redocumented, and any other ocular abnormalities may also be noted.

**TREATMENT OF Ptosis**

The aponeurotic repair for levator dehiscence and disinsertion was developed by Jones et al. and popularized by Anderson and Dixon, as well as other authors. We have been able to easily combine this technique with routine blepharoplasty, producing reliable results.

The procedure should be performed under local anesthesia, as patient cooperation is necessary to judge the appropriate amount of repair. The procedure is begun as a routine blepharoplasty with excision of skin and orbicularis oculi muscle. At this point, orbital septum is exposed and should also be incised in routine fashion. The preaponeurotic fat pad may then be excised as needed and retracted upward to expose the levator aponeurosis. When the aponeurosis is totally disinserted, it may appear as a thickened white band of tissue lying over Müller’s muscle. Pretarsal skin and muscle should then be gently freed from the upper portion of the tarsus. Care should be taken not to dissect too far inferiorly, as damage to the hair follicles of the eyelashes may result. With adequate exposure of both tarsus and levator aponeurosis, a horizontal mattress suture is then placed through the edge of the levator aponeurosis down to the upper portion of the tarsus in the midpupillary line (Fig 2). A variety of sutures may be used, but we prefer an absorbable 5-0 polyglactin 910 (Vicryl) suture. One throw is placed, and the patient is then asked to open the eye to check the lid position. Adjustments are made as necessary, and several more sutures are placed in a similar fashion, being careful to produce an even eyelid contour and avoid any notching of the eyelid margin. The level of the eyelid should be overcorrected by 1 to 2 mm, as it will slightly relax in the first months after surgery. The skin incision is then closed in a routine fashion; we use a 6-0 fast-absorbing gut suture (Ethicon), which eliminates postoperative suture removal. We have found this method of aponeurotic repair to be both safe and reliable in treating acquired senile ptosis (Figs 3 and 4).
COMPLICATIONS

Aponeurotic surgery, when performed correctly, is a very predictable procedure with few complications. Overcorrection may occur, although suture placement with patient cooperation should help prevent this problem. Mild overcorrection may be treated by massaging the eyelid in a downward direction. This technique should be continued for approximately one month before another operation is considered. Poor eyelid contour with notching of the eyelid may occur from uneven suture placement and, again, should be treated initially with massage. Recurrence of senile ptosis may be noted in a few patients one to two years after surgery from continued stretching of the aponeurosis. These patients require further surgery for repair of this recurrent ptosis and may need to undergo a more radical procedure than simple aponeurotic repair. Other complications, such as undercorrection, lagophthalmos, and loss of eyelashes, are extremely uncommon with aponeurotic surgery.

The opposite eyelid can also be the source of a very frustrating postoperative result. Because of the equal innervation of both levator muscles, a severe ptosis will cause overstimulation of the muscles and mask a milder ptosis on the opposite eyelid. After correction of the severe ptosis, the levator muscles relax, revealing the milder ptosis of the opposite eyelid. Thus, the patient may begin with ptosis in one eye and, postoperatively, present with ptosis in the opposite eye. At this point, treatment should consist of surgery to correct the ptosis of the second eyelid so that it will match the eyelid already corrected.

COMMENT

Acquired ptosis caused by levator aponeurosis dehiscence or disinsertion is a common condition in the patient population seeking blepharoplasty. Surgeons should recognize the characteristics of the ptotic lid and evaluate it appropriately. Repair may be performed at the time of blepharoplasty with good cosmetic and functional results.

References