The Comprehensive Management of Chemosis following Cosmetic Lower Blepharoplasty

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Background: Chemosis can cause persistent discomfort and aggravation in the postoperative period following surgery of the eyelids. This article focuses on chemosis associated with cosmetic lower blepharoplasty. The cause is multifactorial and includes exposure, periorbital edema, and postoperative lymphatic dysfunction.
Methods: A chart review of 312 primary bilateral lower transcutaneous blepharoplasties was performed. Data were collected to identify the incidence of chemosis, define associated etiologic factors, develop a chemosis classification system, and outline a successful treatment algorithm.
Results: The incidence of chemosis was 11.5 percent in this population of lower lid blepharoplasty patients. Chemosis presented intraoperatively or up to 1 week postoperatively. The median duration was 4 weeks, with a range from 1 to 12 weeks. Associated etiologic factors included conjunctival exposure, periorbital and facial edema, and lymphatic dysfunction. The four general patterns of presentation were classified as type 1, acute mild chemosis with complete lid closure; type 2, acute severe chemosis that prohibits complete lid closure (chemosis-induced lagophthalmos); type 3, subchronic chemosis that persists longer than 3 weeks; and type 4, chemosis associated with lower lid malposition. Successful treatment existed along a continuum from liberal lubrication to ophthalmic steroid preparations and ocular decongestants to eye-patching to minor surgical procedures such as drainage conjunctivotomy and temporary tarsorrhaphy. In all cases, chemosis ultimately resolved.
Conclusions: Chemosis is a common complication of lower blepharoplasty. Pharmacologic, mechanical, and surgical therapies may be used alone or in combination for the successful management of chemosis. Prevention by minimization of triggering factors intraoperatively and immediately postoperatively is important. (*Plast. Reconstr. Surg.* 122: 579, 2008.)

Chemosis is defined as transudative edema of the bulbar and/or fornical conjunctiva and is characterized by visible swelling of the conjunctiva. Conjunctival inflammation is frequently present in many cases. Epiphora, irritation, a foreign-body sensation, and mild visual alterations may also be experienced.[1–4]

In the context of lower blepharoplasty, the cause includes the following factors: exposure of the conjunctiva from lagophthalmos, regional edema and lymphatic dysfunction caused by the blepharoplasty dissection, and scarring.[5,6] In most cases, chemosis resolves spontaneously early in the postoperative period, but for some patients, resolution is delayed. There are a number of treatment options including both conservative and surgical management.[4,7,8]

The majority of the literature regarding chemosis in blepharoplasty exists in the form of small case series with suggestions for management.[2,7] One large series of 4395 patients suggests an incidence as low as 1 percent.[6] The purpose of this publication is threefold. First, the goal was to review the incidence of chemosis after lower lid blepharoplasty to define etiologic factors. Second,

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a classification system is presented to facilitate the discussion of the multiple distinct presentations of chemosis following blepharoplasty. Third, an algorithm is presented that proscribes a stepwise treatment regimen.

PATIENTS AND METHODS

A retrospective chart review of 312 primary transcutaneous lower blepharoplasty procedures performed between 1994 and 2005 by a single surgeon (M.A.C.) was undertaken. The surgical technique included a lower lid skin muscle flap with release of the orbitomalar ligament followed by fat removal or fat transposition and lateral canthopexy or canthoplasty for tarsoligamentous laxity.[9] The onset and duration of chemosis were evaluated, as was the treatment.

RESULTS

The incidence of chemosis was 11.5 percent (36 patients). The onset of chemosis was noted intraoperatively in 5 percent of the affected 36 patients. In the remainder of the cases, it presented in the first postoperative week. The median duration of the chemosis was 4 weeks, ranging from 1 week to 3 months. All 312 patients underwent some form of canthal support. Canthopexy was performed in 226 patients and canthoplasty was performed in 86 patients. The incidence of chemosis in the two groups was 11.1 percent and 12.8 percent, respectively; this did not represent a statistically significant difference (chi-square test, 0.182; $p = 0.67$). The predisposing factors were increased periorbital swelling and conjunctival exposure related to increased surgical time from additional procedures and lagophthalmos from upper blepharoplasty and/or brow lift.

The initial presentation consisted of edematous and injected conjunctiva. Often, the edematous tissue had a light yellow hue. The degree of swelling varied from mild (Fig. 1, *above*) to severe (Fig. 1, *center*), even to the extent that lid closure was hindered (chemosis-induced lagophthalmos) by conjunctival tissue prolapsed beyond the blepharal plane (Fig. 1, *below*). Five patients (13.9 percent of patients with chemosis, 1.6 percent of patients undergoing lower blepharoplasty) experienced chemosis greater than 3 weeks and ultimately experienced resolution by 3 months. All patients had normal slit lamp examinations performed by the senior author (M.A.C.), with no corneal ulcers and resolution of chemosis with no permanent sequela. No referrals to an ophthalmologist were required for assistance in management.

Fig. 1. (*Above*) Type 1 chemosis (*Center*) Type 2 chemosis. (*Below*) Type 2 chemosis with demonstration of chemosis-induced lagophthalmos from prolapsed conjunctival tissue.

DISCUSSION

This study demonstrates that chemosis is a frequent complication in cosmetic lower lid bleph-

aroplasty. Knowledge of three basic facts about the histology and anatomy of the conjunctiva will elucidate why this tissue is prone to the profound intraoperative and postoperative edema known as chemosis. First, the conjunctiva is a loosely adherent flexible protective covering over the pericorneal anterior surface of the globe.[5] The conjunctiva has several plicated folds in the fornix and many crypt-like invaginations, increasing its surface area.[1,10] Thus, it is both unrestrained and has significant potential for expansion. Second, the stroma is a fibrovascular connective tissue that contains lymphatic channels, capillaries, and the episcleral venous plexus. The bulbar endothelial cell junctions are not entirely competent, thus permitting extravascular extravasation of fluid in states of inflammation.[11] Third, the stroma contains a population of lymphocytes capable of providing proinflammatory chemotactic signals.[10]

Etiologic Factors

The main etiologic factors associated with chemosis include exposure, edema, and lymphatic dysfunction (Table 1). The scope of this study does not permit comment regarding the relative importance of these factors in the genesis of chemosis. Likewise, the literature does not contain evidence regarding their relative importance.

Exposure

Exposure leads to desiccation. The conjunctiva is prone to desiccation because of its nonkeratinized mucosal epithelial surface.[1] Desiccation of conjunctival tissue results in endothelial dysfunction, fluid extravasation, and edema.[12] Intraoperative exposure occurs because of the absence of lid closure during general anesthesia or reduced blink reflex with topical anesthetics and sedation procedures. Postoperative exposure is attributable in large part to lagophthalmos. This state is more likely with concomitant upper lid blepharoplasty and/or brow lift. Local anesthetics, lid edema, altered neuromuscular relationships, and chemosis itself also hinder complete lid closure.

One other factor to consider in managing postoperative chemosis resulting from exposure is lid malposition and ectropion.[13] Exposure-related chemosis caused by lower lid malposition can be temporized by chemosis management strategies. Definitive treatment entails correction of the malposition.

A cyclic relationship exists between exposure and chemosis (Fig. 2). Lid closure can be hindered by prolapsed conjunctival tissue in moderate to severe chemosis. Incomplete closure results in exposure and desiccation. These in turn worsen chemosis. Thus, exposure (a cause) and chemosis (a pathologic condition) are in a mutually propagating cycle.

Periorbital Edema

There are no lymphatic channels in the orbit deep to the septum orbitale.[14] The conjunctiva is drained ultimately by the deep division of the eyelid lymphatic system. Plexuses are formed along the tarsal borders that drain the conjunctiva. The lateral one-half of the conjunctiva drains to the preauricular node and the medial one-half of the conjunctiva drains to the submandibular node by lymphatic vessels that follow the path of the angular and facial arteries[10,11] (Fig. 3). This anatomy makes evident the fact that periorbital edema and facial edema from facial rejuvenative procedures will cause regional lymphatic stasis that will both trigger and propagate chemosis. In essence, the presence of periorbital and facial edema prevents the conjunctiva from draining effectively.[15]

Lymphatic Dysfunction

Lymphatic dysfunction refers to structural damage to lymphatic channels draining the conjunctiva that occurs as a necessary part of the procedure.[3] It is distinguished from periorbital and facial edema that exists in the absence of direct damage to lymph channels. Lymphatic dysfunction is more likely following lower lid blepharoplasty secondary to the increased dissection in the regions of lymphatic drainage channels to the caudal face.[10] Fat pad manipulation, septal incisions, and skin-muscle flap elevation decrease the total number of patent channels and results in lymphatic dysfunction. The end result of lym-

Table 1. Etiologic Factors

Etiologic Factors	Mechanism	Associations
Exposure	Irritation, inflammation	General anesthesia, Upper blepharoplasty, brow lift, decreased blink, moderate/severe chemosis (cyclic relationship)
Edema	Decreased fluid drainage	Upper blepharoplasty, brow lift, face lift, excessive sodium consumption
Lymphatic dysfunction	Decreased fluid drainage	Dissection, scarring

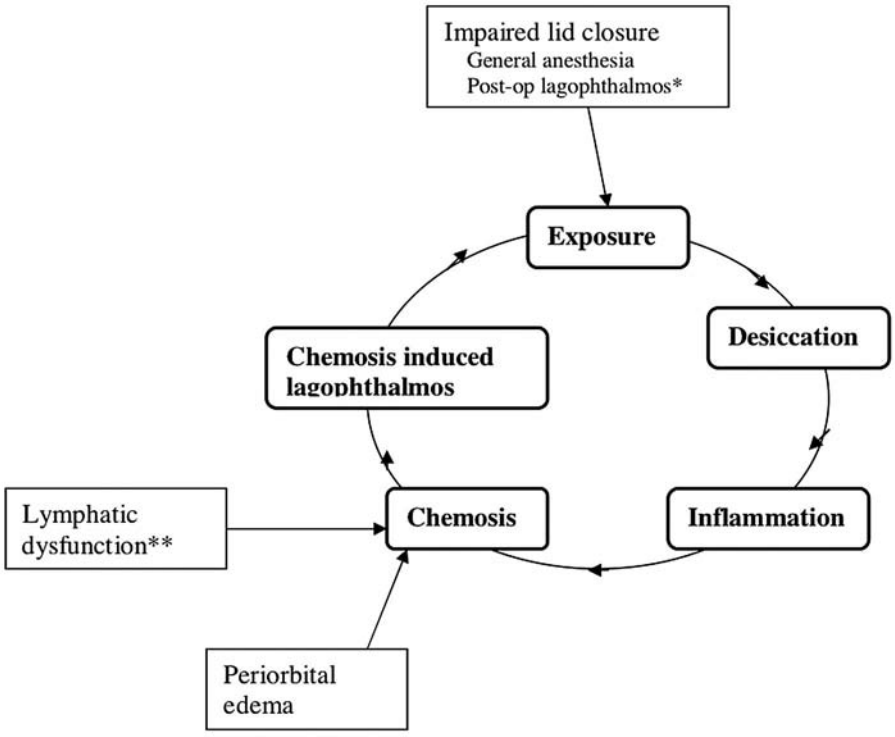

Fig. 2. Mutually propagating cyclic relationship between exposure and chemosis.

phatic dysfunction is edema of the periorbital tissues and conjunctiva. It is possible that different techniques would result in less lymphatic disruption and therefore less chemosis. However, this article only evaluates patients undergoing lower lid blepharoplasty with a skin-muscle flap combined with a canthal support procedure.

Chemosis Classification

The intraoperative and postoperative presentation of chemosis in lower blepharoplasty exists in a spectrum from mild to severe, and the duration is both acute and subacute. Based on our observations of these various presentations and a review of the literature focused on chemosis and lower lid malposition and ectropion, chemosis is classified as type 1, 2, 3, or 4.[2–4,6–8,13,15,16] (Table 2 and Fig. 4).

Type 1 chemosis represents a mild form of acute chemosis. Type 1 chemosis is often noted intraoperatively but may be present postoperatively as well. It is characterized by edema, often with a slight yellow hue and injection. The presence of the hyperemia-associated injection suggests that there is an inflammatory component. One distinguishing feature is the ability, or projected ability if assessing intraoperatively, to completely close the lid.

Type 2 chemosis represents severe acute chemosis. The edematous and injected conjunctiva prolapse out of the palpebral fissure, producing a mass effect. The end result is chemosis-induced lagophthalmos.

Type 3 chemosis represents subchronic edema and inflammation of the conjunctiva lasting up to 6 months in several cases presented in the literature. In our experience, it resolved in 3 months. The cause of type 3 chemosis is likely related to prolonged lymphatic dysfunction caused by division and inadequate reanastomosis of lymph channels within the eyelids and surrounding facial tissues. Lagophthalmos generally is not present.

Type 4 chemosis represents chemosis caused by lower lid malposition and/or ectropion. It is important to distinguish the associated incomplete lid closure of type 4 from the lagophthalmos caused by the mass effect of severe chemosis in type 2. In type 4, both edema and inflammation

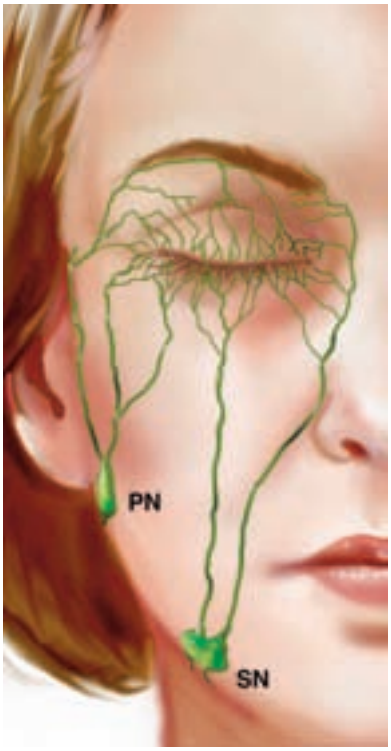

Fig. 3. Lymphatic drainage of the conjunctiva. The lateral conjunctiva drains to the preauricular nodes. The medial conjunctiva drains to the submandibular nodes. Disruption and/or congestion in the channels from blepharoplasty and other facial procedures contribute to conjunctival edema.

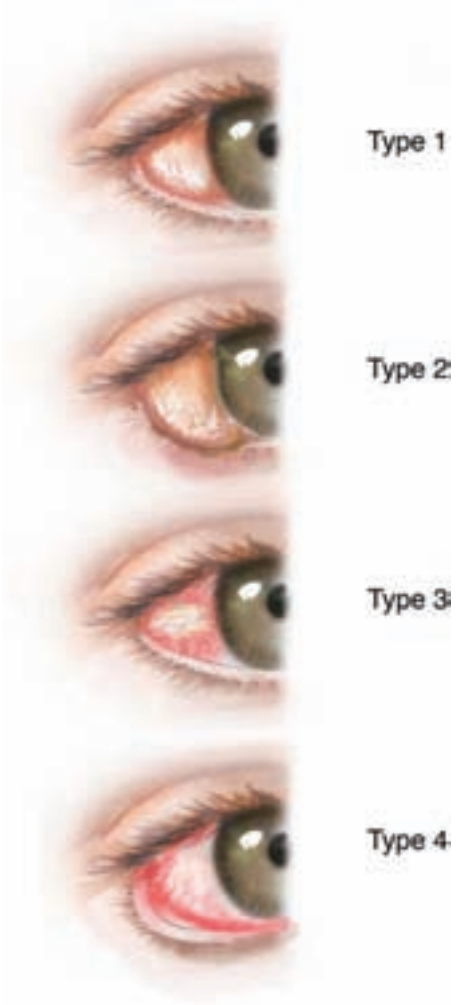

Fig. 4. Chemosis classification system. The four classes are type 1, mild acute edema and inflammation with complete lid closure; type 2, severe acute edema with inflammation that prohibits complete lid closure (chemosis-induced lagophthalmos); type 3, subchronic edema and inflammation that persists longer than 3 weeks; and type 4, chemosis associated with lower lid malposition.

are present and are the result of exposure. Type 4 chemosis will not resolve until lower lid malposition and ectropion are corrected.

Prevention

Prevention of chemosis is initiated in the operating room and is centered on reducing the impact of triggering factors (Table 3). Rinsing the corneal protectors and the globes with balanced saline solution before insertion prevents chemical irritation from preparation solutions. Frequent moisturizing with wetting drops, ophthalmic lubricating ointments, and intermittent forced lid closure prevents exposure-related desiccation when the protectors are not in place and at the end of the case. The use of cold saline-soaked gauze, elevated head position, normotensive an-

Table 2. Chemosis Classification

Class	Edema	Inflammation	Color	Lagophthalmos	Duration
Type 1 (acute mild)	Mild	Mild	Yellow and/or pink	Absent	<3 wk
Type 2 (acute severe)	Severe	Severe	Yellow and/or pink	Present laterally (chemosis hinders closure)	<3 wk
Type 3 (subchronic)	Mild to severe	Chronic	Pink	Absent	>3 wk to 6 mo
Type 4 (subchronic because of lower lid malposition)	Severe	Moderate to severe	Pink	Lower lid malposition and/or ectropion	Until lid malposition is corrected

Table 3. Chemosis Prevention in Cosmetic Lower Lid Blepharoplasty

Intraoperative prevention measures
Normotensive anesthesia
Minimal intravenous fluids
Judicious dissection
Tarsorrhaphy suture ("antichemosis suture")
Intraoperative and postoperative measures
Wetting drops
Ophthalmic lubricating ointment
Iced gauze
Head elevation
Postoperative measures
Limiting sodium consumption

esthesia, and minimal intravenous fluid administration combat excessive edema.

Limiting dissection to only what is necessary in the periorbital region will decrease the potential for lymphatic dysfunction. The placement of a temporary tarsorrhaphy suture (antichemosis suture)[17] is considered in cases where significant dissection has been used (e.g., concomitant cheek lift). It is also considered when the potential for temporary lagophthalmos exists secondary to upper blepharoplasty and brow lift. The intermarginal suture should be placed through the tarsus and at the level of the lateral limbus, or slightly more lateral to it.

Preventative measures are continued in the postoperative period. These include limiting exposure-related desiccation with wetting drops during the day and ophthalmic lubrication ointments at night.[16] The head should remain in an elevated position, and iced gauze or ice packs are applied to the periorbital region for 24 hours. Sodium consumption is limited.

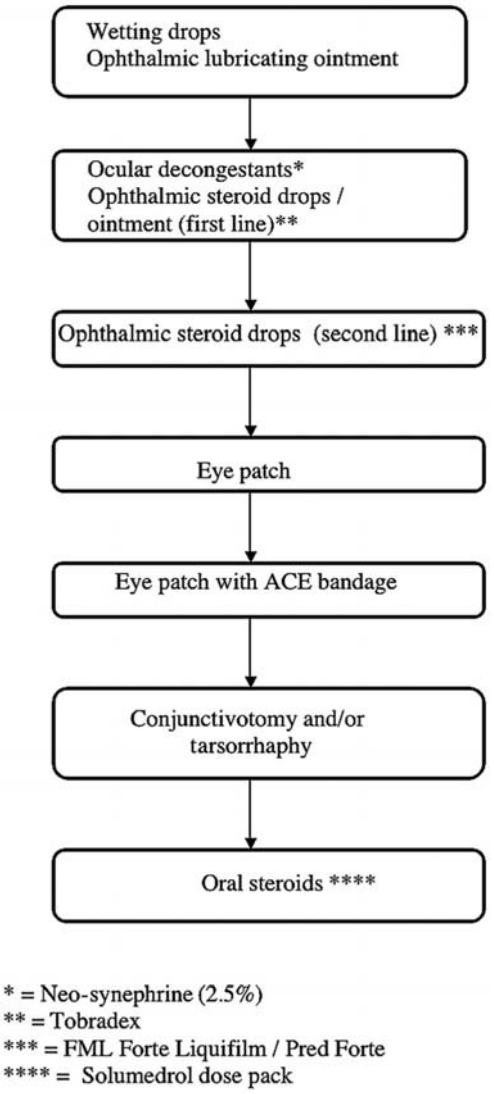

Fig. 5. Treatment algorithm.

Treatment Algorithm

Treatment is initiated on detection and is administered according to an algorithm (Fig. 5). In general, conservative interventions are instituted before invasive measures. The first step in management is the liberal use of wetting drops during the day and ophthalmic lubricating ointment at night. It should be noted that wetting drops and ointment are routinely used postoperatively for 1 week for the prevention of chemosis. Thus, this step refers to instructions given to patients should they report mild chemosis and inquire about therapy before their first follow-up appointment. If the patient presents at the first follow-up visit with type 1, or a less severe variation of type 2 chemosis, the ocular decongestants 2.5% Neo-Synephrine (Sanofi-Sythelabo, Inc., New York, N.Y.) and TobraDex (Alcon Laboratories, Inc., Fort Worth, Texas) drops and ointment are used. If resolution has not occurred by the second follow-up appointment, FML Forte Liquifilm 0.25% (Allergan, Inc., Irvine, Calif.) or Pred Forte (Allergan) is used. The eye may also be patched; ophthalmic ointment should always be placed in the eye when patching is preformed (Fig. 6, *above*). In more severe cases of type 2 chemosis, a circumferential elastic bandage head wrap is applied and used overnight (Fig. 6, *below*). These pharmacologic and noninvasive mechanical interventions may be successful at downgrading the chemosis to a type 1. If chemosis persists beyond 2 weeks, and especially if significant lagophthalmos is present, conjunctivotomy with possible tarsorrhaphy is performed (Fig. 7). Oral steroids (Medrol Dose Pack; Pharmacia & Upjohn, Division of Pfizer, New York, N.Y.) are used at 3 weeks when subchronic

Fig. 6. (*Above*) Patching of the affected eye. Ointment should be placed in the eye before patching. (*Below*) The addition of a circumferential elastic bandage wrap over a patched eye for 24 hours or for several nights provides additional gentle pressure that facilitates the resolution of conjunctival edema.

Fig. 7. Lateral conjunctivotomy for severe chemosis with significant chemosis-induced lagophthalmos and conjunctival prolapse. (*Above*) In the clinic setting, the injection of 1% lidocaine with 1:100,000 epinephrine provides anesthesia and also provides vasoconstriction that can also facilitate resolution. Ophthalmic tetracaine drops used before injection further decrease the discomfort. (*Below*) Westcott scissors are used to incise the lateral conjunctiva. Gentle spreading facilitates drainage of the fluid. Antibiotic ointment is applied and used for at least 3 days after the procedure.

chemosis is present that has not resolved with the aforementioned management strategies.

The above algorithm is used in a stepwise fashion. However, some important considerations may dictate using the interventions in an alternative sequence (Table 4). Lubricating drops and ointment are used throughout management. Tarsorrhaphy can be used at any time as a preventative measure to treat chemosis at any point and any stage, for example, if type 2 is present longer than 2 weeks and the treating surgeon does not wish to opt for conjunctivotomy. Type 2 chemosis presenting intraoperatively can be treated with immediate conjunctivotomy, taking advantage of the operative setting.

CONCLUSIONS

Chemosis is the most common nonsurgical complication of lower lid blepharoplasty, occur-

Table 4. Important Treatment Considerations

Class	Treatment Consideration
Type 1	If present intraoperatively, consider tarsorrhaphy; follow treatment algorithm postoperatively
Type 2	If chemosis-induced lagophthalmos is present intraoperatively or if significant prolapse is present postoperatively, consider conjunctivotomy as the first step in treatment
Type 3	Oral steroids may be helpful if chemosis reaches a chronic phase
Type 4	Lubricate; decrease inflammation with steroid drops; correct lid malposition

ring in 11.5 percent of the patients in this report. It presents as four different types and management depends on the type present. Type 1 is mild acute chemosis. Type 2 is severe acute chemosis-associated lagophthalmos. Type 3 is subchronic chemosis that persists longer than 3 weeks and up to 6 months. Type 4 is chemosis resulting from lid malposition. Treatment ranges from topical preparations of steroids and ocular decongestants to eye patching to conjunctivotomy and tarsorrhaphy. Although many cases are short lived and resolution occurs universally, some cases may require up to 6 months of expectant management. These facts should be a part of the preoperative education and informed consent.

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