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Orbital Compartment Syndrome in Burn Patients

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Purpose: To identify clinical characteristics of burn patients requiring emergent orbital decompression for vision-threatening orbital compartment syndrome.

Methods: A retrospective review of 28 burn patients at a trauma center provided data regarding demographics, physical examination findings, and resuscitation fluid volumes. Patients requiring orbital decompression were compared with those who did not, using *t* tests and Fisher exact test. Linear regression was used to test for an association between peak intraocular pressure and fluid volume. Logistic regression was used to assess associations between need for orbital decompression and fluid volume.

Results: Eight of 28 patients required emergent orbital decompression, which immediately reduced intraocular pressure from 59.4 ± 15.9 mm Hg to 28.6 ± 8.2 mm Hg ($p < 0.001$). There was a positive relationship between fluid volume in the first 24 hours and peak intraocular pressure ($p < 0.001$). Patients who were treated with orbital decompression were resuscitated with a higher fluid volume in the first 24 hours than those who were not (37,218 ± 14,405 ml versus 24,649 ± 12,339 ml, $p = 0.015$). This was no longer statistically significant when adjusted for periocular burns. The relative risk for undergoing orbital decompression in patients receiving ≥8.6 ml/kg/% total body surface area burned in the first 24 hours was 4.4 ($p = 0.03$).

Conclusions: Risk factors for vision-threatening orbital compartment syndrome include fluid volume and periocular burns. Signs of vision-threatening orbital compartment syndrome should be addressed early with orbital decompression.

Patients suffering severe burn injuries who receive aggressive fluid resuscitation may be at an increased risk of orbital compartment syndrome. This association has been documented in 2 retrospective case series.[1,2] Since many burn patients are intubated before or early in their hospital course, they may not be able to relate symptoms of orbital pain or change in vision. For this reason, it is important for ophthalmologists and admitting physicians in this setting to be aware of this potentially blinding problem.

The association between blindness and severe bodily trauma, including burns, has been reported.[3–5] These studies hypothesized but did not prove the mechanisms for vision loss in these patients. Evans reported a small case series of 3 severe burn patients who suffered elevation in intraocular pressure (IOP) after undergoing aggressive fluid resuscitation.[1] These patients were treated with lateral canthotomies, which relieved the elevated pressure immediately. Given the immediate decrease in IOP after orbital decompression, it was concluded that the etiology of the pressure rise was increased intraorbital pressure transmitted to the eye.

The etiology of increased intraorbital pressure is likely multifactorial. Burn injuries cause loss of intravascular fluid and protein in the interstitial space, leading to tissue edema.[6,7] Burn patients also receive high volumes of fluid resuscitation.[8–11] These 2 mechanisms are likely responsible for the well-documented risks of abdominal and extremity compartment syndrome in these patients.[12–17] The orbit, like the abdomen or extremities, is a closed system. Orbital compartment syndrome, with a sufficiently high pressure, could lead to ischemic or compressive optic neuropathy. Elevated IOP could also lead to nerve fiber layer loss and retinal vessel occlusion. These events could occur in a patient unable to communicate if intubated, sedated, or unconscious.

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In the late 1960s, Baxter et al. described using 3.7 ml/kg to 4.3 ml/kg per percentage total body surface area (%TBSA) burned to guide physicians in effectively resuscitating burn patients.[8] Recent research has suggested that the original formula recommended by Baxter et al. is inadequate in many burn patients.[11] Since the 1970s, there has been a trend toward higher volumes of fluid resuscitation.[9,10] This trend is concerning, given the potential systemic side effects of fluid resuscitation, including abdominal and extremity compartment syndrome, pulmonary edema, and acute respiratory distress syndrome.[12–18] Early diagnosis and treatment of extremity and abdominal compartment syndromes has become standard of care in burn patients.[19–21] Similarly, early identification and treatment of orbital compartment syndrome in these patients could prevent ocular sequelae.

The potential for orbital compartment syndrome and blindness in burn patients is described in the literature.[1–5] We define severe orbital compartment syndrome as a rise in intraorbital pressure requiring emergent orbital decompression to preserve vision. The purposes of this study are to define the clinical characteristics for severe orbital compartment syndrome in this patient population, to alert ophthalmologists to this potentially blinding problem, and to promote the early involvement of appropriate ophthalmologic care in these patients when indicated.

METHODS

We began monitoring the IOP in severe burn patients in 2004 after several burn patients were found to have elevated IOP on ophthalmologic consultation. Initially, patients with ≥25% TBSA burned were defined as severely burned and were included. We did include a patient with 22% TBSA burned in this study, as this patient suffered periocular burns and was aggressively hydrated. This patient was followed as the others were. After receiving approval from the University of Washington Human Subjects Committee Institutional Review Board, we retrospectively reviewed the records of 28 patients admitted to Harborview Medical Center from April 2004 to December 2005. Record review and analysis was performed in a HIPAA-compliant fashion.

These patients underwent comprehensive ophthalmologic examination within 24 hours of admission, followed by IOP monitoring on hospital days 2 and 3. This was done with a portable tonometer (Tonopen, Mentor, Santa Barbara, CA, U.S.A.). All IOP measurements were recorded with no more than 5% chance of error. A Desmarre's eyelid retractor was used in patients unable to open their eyelids, without compressing the globe. These patients underwent treatment for eye injuries and evidence of orbital compartment syndrome if indicated. Severe orbital compartment syndrome is defined as a vision-threatening rise in IOP in the setting of periocular tissue edema and in the absence of other etiologies for a rise in pressure. Severe orbital compartment syndrome was treated with emergent bilateral lateral canthotomy with inferior and superior cantholysis under local anesthesia. All patients undergoing orbital decompression also received topical IOP-lowering therapy. All ophthalmologic examinations and interventions were performed by the ophthalmology service at the University of Washington. Preliminary results from this retrospective study (n = 13) were reported by Sullivan et al. in 2006.[2]

Data collected from patient records included age, gender, %TBSA burned, intubation, IOP measurements, comprehensive ophthalmologic examination findings, interventions for elevated IOP, extremity hypoperfusion, abdominal compartment syndrome, IV fluid administered (lactated Ringer's solution) at 24 hours, and mortality. Periocular burns were defined as burns involving the eyelids. Intraabdominal pressures were not routinely measured, and abdominal compartment syndrome was diagnosed by clinical means. Mortality was defined as failure to survive to hospital discharge. Measurement of extraocular motility was not routinely done, as most of these patients were intubated and sedated. No orbital imaging studies were performed.

Statistical analysis was performed after consultation with the Department of Biostatistics at the University of Washington. IOP measurements between right and left eyes were highly correlated, and so these values were averaged at each measurement. IOP and fluid levels were both natural log transformed for analysis. This was done to reduce skewed data and to down-weight large values, given the potential inaccuracy of IOP measurement at large values. Linear regression was used to test for an association between natural log transformed peak IOP, or ln(peak IOP), and the ln(amount of fluid administered at 24 hours). Both total fluid and ml/kg/%TBSA burned were tested in this manner. Demographic and clinical characteristics were compared for patients who developed severe orbital compartment syndrome and those who did not. Continuous variables were compared using a t test (2-sided, allowing for unequal variances) and categorical variables were compared using Fisher exact test (2-sided). Logistic regression was used to compare associations between ln(total fluid) and severe orbital compartment syndrome, adjusting for other characteristics. Fisher exact test was used to test if severe orbital compartment syndrome was associated with receiving fluid levels ≥8.6 ml/kg/%TBSA burned (equal to or greater than twice the value predicted by the Baxter formula), compared with receiving fluid levels <8.6 ml/kg/%TBSA burned. This value was chosen for comparison since many patients exceeded twice the fluid volume predicted by the Baxter formula. As sensitivity analysis, the association between fluid levels and severe orbital compartment syndrome was examined, eliminating from the sample children (age <18 years) and patients without severe orbital compartment syndrome who received topical IOP-lowering treatment. This second sensitivity analysis was performed because a small number of patients without severe orbital compartment syndrome were treated for mild IOP rises. Excel 11.2 (Microsoft, Redmond, WA, U.S.A., 2004) and Stata 8.2 (StataCorp, College Station, TX, U.S.A., 2005) were used for statistical analysis.

RESULTS

Table 1 lists the demographics and clinical characteristics of the 28 patients included in this study. No patients sustained orbital trauma. There was no evidence of direct eye trauma in

TABLE 1. *Comparison of Demographics and Clinical Characteristics of Patients Who Developed Severe Orbital Compartment Syndrome Versus Those Who Did Not*

Characteristic	No Decompression (n = 20)	Severe Orbital Compartment Syndrome (n = 8)	p*
Age (yr)			
Range	4–80	21–68	
Mean ± SD	40.0 ± 20.5	45.2 ± 13.5	0.44
% Male	85	62.5	0.33
Weight (kg)			
Range	23.2–104.8	68–134.4	
Mean ± SD	75.6 ± 23.4	89.9 ± 31.9	0.28
%TBSA burned			
Range	22–88	30–85	
Mean ± SD	45.2 ± 17.1	46.4 ± 19.0	0.88
% With periocular burn	55	87.5	0.11
% Corneal abrasion	15	37.5	0.21
% Mortality	15	62.5	0.022
% Intubated	80	100	0.30
% Extremity hypoperfusion	40	75	0.21
% Abdominal compartment syndrome	5	12.5	0.50
Peak IOP (mm Hg)			
Range	13.5–39.5	38.5–90	
Mean	20.6 ± 6.4	59.4 ± 15.9	<0.001†
Baxter formula, predicted fluid (ml), 24 h			
Range	9,331–20,661	6,215–38,370	
Mean ± SD	14,474 ± 8,256	17,066 ± 6,291	0.38†
Total fluid, 24 h (ml)			
Range	18,576–67,520	11,516–63,161	
Mean ± SD	24,649 ± 12,339	37,218 ± 14,405	0.015†
Fluid, 24 h, ml/kg/%TBSA burned			
Range	3.6–11.3	5.6–14.1	
Mean ± SD	7.7 ± 1.8	9.7 ± 2.8	0.086†

*p from t test or from Fisher exact test.
†p based on natural log transformed values.
IOP, intraocular pressure; TBSA, total body surface area; SD, standard deviation.

any patient, and none of the patients had a hyphema. No patients had a history of glaucoma, nor were any patients taking ocular medications, oral carbonic anhydrase inhibitors, or steroids. There were no other etiologies noted for elevations in IOP in any of the patients. Periocular burns were bilateral in every case. No patients had third-degree burns of the periorbita or developed cicatricial eyelid retraction during admission. All of the patients with evidence for extremity compartment syndrome (50% of the total) underwent decompressive escharotomies. Of the patients with abdominal compartment syndrome (7.1% of the total), 1 underwent decompressive laparotomy and the other truncal escharotomy. There were no patients who developed retinal vascular occlusion.

The mean predicted volume of fluid resuscitation delivered, calculated by the Baxter formula (4.3 ml/kg/%TBSA burned), was 15,214 ± 7,724 ml. The mean total volume delivered in the first 24 hours was 28,240 ± 13,941, or 8.3 ± 2.3 ml/kg/%TBSA burned (approximately 1.9 times the level predicted by the Baxter formula).

Eight patients were defined as having severe orbital compartment syndrome. The mean IOP decreased from 59.4 ± 15.9 mm Hg to 28.6 ± 8.2 mm Hg ($p < 0.001$) after bilateral orbital decompression. Three other patients were treated successfully with topical IOP-lowering medication only.

Linear regression analysis shows a positive relationship between ln(peak IOP) in the first 48 hours and ln(total fluid administered at 24 hours), both in total milliliter ($r^2 = 0.36$, $p < 0.001$), and in ml/kg/%TBSA burn ($r^2 = 0.25$, $p < 0.001$) (Figs. 1 and 2). This was also statistically significant when restricted to adults (≥18 years old, n = 26, $p = 0.006$) and when the 3 patients who received topical medications only were eliminated (n = 25, $p = 0.006$).

There were no statistically significant differences between the groups with respect to age, gender, weight, %TBSA burn, periocular burns, corneal abrasions, intubation, extremity compartment syndrome, or abdominal compartment syndrome. The amount of fluid predicted by the Baxter formula at 24 hours was not statistically significant between the 2 groups (p =

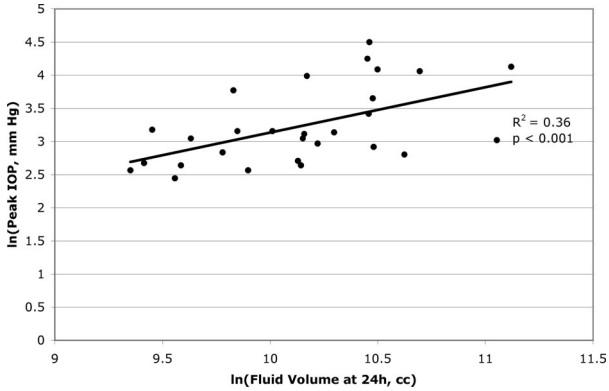

FIG. 1. Peak IOP (mm Hg, natural log) at 48 hours compared with fluid volume administered at 24 hours (ml, natural log) for all patients (n = 28).

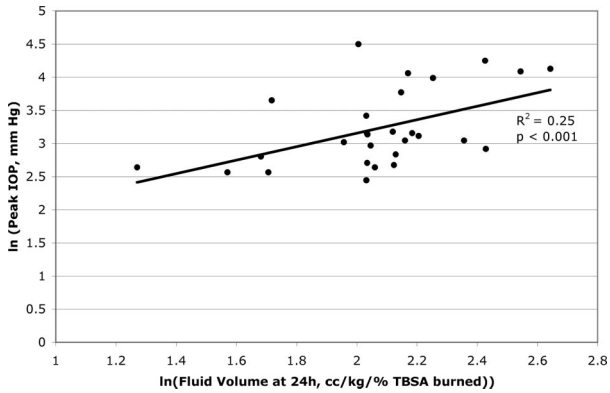

FIG. 2. Peak IOP (mm Hg, natural log) at 48 hours compared with fluid volume administered at 24 hours (ml/kg/%TBSA burned, natural log) for all patients (n = 28).

0.38). The total amount of fluid delivered at 24 hours was higher in the group who developed severe orbital compartment syndrome, 37,218 ± 14,405 ml versus 24,649 ± 12,339 ml ($p = 0.015$) (Fig. 3). This was statistically significant when restricted to adults (≥18 years old, n = 26, $p = 0.031$) and when the 3 patients who received topical medications only were eliminated (n = 25, $p = 0.0055$). The association between fluid level and severe orbital compartment syndrome was not statistically significant when fluid was measured as ml/kg/%TBSA burned ($p = 0.086$). Patients with severe orbital compartment syndrome had a higher mortality rate than those who did not (62.5% versus 15%, $p = 0.022$) (Table 1).

The statistically significant association of higher total fluid administered at 24 hours (ml) in patients with severe orbital compartment syndrome remained significant when adjusted for age, gender, and weight, using logistic regression. It was not statistically significant, however, when adjusted for periocular burns or corneal abrasions (Table 2).

Patients receiving ≥8.6 ml/kg/%TBSA burned (twice the Baxter formula) were 4.4 times as likely to develop severe orbital compartment syndrome than those receiving less than this volume ($p = 0.03$). This was statistically significant when restricted to adults (≥18 years old, n = 26, $p = 0.017$) and when the 3 patients who received topical medications only were eliminated (n = 25, $p = 0.022$).

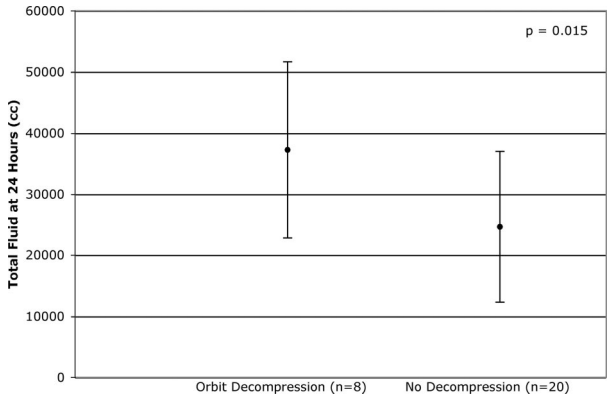

FIG. 3. Comparison of total mean total fluid volume administered at 24 hours in patients who developed severe orbital compartment syndrome versus those who did not.

TABLE 2. *Association Between Total Fluid at 24 Hours (ml, Natural Log) and Severe Orbital Compartment Syndrome, Adjusting for Key Covariates*

Covariate	*p**
Unadjusted	0.015
Age	0.043
Gender	0.023
Weight (kg)	0.046
Gender and weight (kg)	0.038
Periocular burn	0.114
Corneal abrasion	0.064

**p* for from logistic model for ln(fluid) and covariates.

DISCUSSION

Orbital compartment syndrome likely has a similar mechanism as extremity and abdominal compartment syndromes in burn patients. The combination of extravasation of fluid and protein in burn patients and the increasingly aggressive volumes of fluid delivered for resuscitation are probably the main etiologies.[6–11] The orbit, being a closed compartment, could become engorged with fluid in this setting. The subsequent increase in pressure is transmitted to the globe, explaining the high IOP noted in patients in this study. The immediate decrease in IOP after lateral canthotomy with cantholysis, noted both in this study and in previous reports, supports this hypothesis.[1,2]

Fluid volume was a risk factor for developing severe orbital compartment syndrome (Figs. 1–3, Table 1). Sensitivity analysis strengthens the validity of these results. An additional risk may be the presence of periocular burns: 87.5% of patients who developed severe orbital compartment syndrome had periocular burns compared with 55% of those who did not (Table 1). Although these results are not statistically significant, the association of total resuscitation fluid volume to the development of severe orbital compartment syndrome was no longer statistically significant when adjusted for the presence of periocular burns (Table 2). This suggests that periocular burns are potentially an independent factor in the development of severe orbital compartment syndrome. Burns to the eyelids may cause tightening or edema of the periocular tissue. More rigid eyelids would be less likely to elastically expand in response to increased orbital volume, leading to higher orbital pressure. Of note, adjusting for corneal abrasion had the same impact on the data. This is likely because the presence of corneal abrasion is highly correlated with the presence of periocular burns.

We do not conclude that severe orbital compartment syndrome is a risk factor for higher mortality rates noted in these patients (Table 1). The need for orbital decompression likely mirrors the need for higher resuscitation

fluid requirements in more critically unstable patients. Further speculation on mortality in this group of patients is beyond the scope of this study.

These results also suggest possible mechanisms for blindness reported in previous studies.[3–5] Cullinane et al. reported a retrospective study of 350 patients admitted to a major trauma center who received massive fluid resuscitation (33 ± 8 l). Of these, 4 developed bilateral blindness, and 5 developed monocular blindness. Of these 9 patients, none were burn patients. The mechanism in these cases, along with several of the aforementioned case reports, was concluded to be due to anterior ischemic optic neuropathy, although documentation as to the method of arriving at this diagnosis was not reported. It is possible that the patients in these reports suffered hypoperfusion leading to anterior ischemic optic neuropathy, but severe orbital compartment syndrome must now be considered in the differential diagnosis. Reports of blindness in this population suggest that all patients receiving large resuscitation volumes should be studied to evaluate for evidence of orbital compartment syndrome.

The size of this retrospective review is not large enough to perform multivariate analyses to clearly identify independent risk factors. However, the trends in the data allow for some recommendations to be made regarding this patient population. Burn patients who receive fluid volumes in excess of 5.5 ml/kg/%TBSA burned in the first 24 hours of admission should be monitored daily for 72 hours for severe orbital compartment syndrome. This value captures all 8 patients in this study who were treated with orbital decompression. Those who suffer periocular burns should also be followed similarly, as they may be at higher risk. Orbital decompression should be considered in patients whose IOP rises above 30 mm Hg. Although this is not necessarily a dangerous level, patients receiving large volumes of fluid may have continued rises in IOP that may not be diagnosed until the following examination. The results of this study, along with the case reports of blindness in aggressively hydrated patients, warrant early surveillance for severe orbital compartment syndrome in this patient population.

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