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Orbital blowout fractures were described as early as 1844. However, they were not widely recognized until 1957 when Smith and Regan introduced the term "blowout fracture". The authors described a specific type of fracture of the orbital floor without fracture of the orbital rim with entrapment of one or more orbital tissues, which limited ocular motility and caused diplopia and enophthalmos.

The fracture is produced by a blunt non-penetrating force over the orbital region. The strong rim of the orbit helps protect against objects with a radius of curvature greater than 5 cm. The ocular globe, which is surrounded by shock absorbing orbital fat is further protected by this increased intraorbital pressure by the blowout fracture; a safety valve which decompresses the cavity. The force striking the globe and causing the fracture may be relatively moderate as the thin bone of the orbital floor is less than 1 mm.

Fujino and his collaborators have offered and additional method for blowout fractures. They have shown that percussion by weights over the infraorbital rim can transmit the striking force and produce a secondary fracture of the orbital floor by percussion without fracture of the orbital rim.

Diagnosis of the orbital blowout fracture is based on the presence of both clinical and radiological findings. In the typical blowout fracture patient, the patient complains of diplopia in the primary position, which increases in upgaze. When examined during the first hours after fracture, the ocular globe may appear displaced backward and downward and the supratarsal sulcus deepened. However, edema and hematoma may obscure such clinical findings only hours after the injury.

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The test known as the "forced ductions test" or traction test, is considered the pathognomonic sign of a blowout fracture of the orbit indicating mechanical restriction of ocular motility inferiorly in the orbit. In a suspected orbital floor fracture, anesthesia or hypesthesia in the area of the distribution of the infraorbital nerve is suggestive evidence of a blowout fracture involving the infraorbital grove or canal. Its absence, however may only mean that the fracture site is located away from the infraorbital grove or canal.

Radiologic diagnosis is essential and reliable. With adequate technique, blowout fractures of the orbit can be diagnosed in over 90 per cent of cases with conventional radiography. Polytomography has a similar degree of diagnostic accuracy and in addition can delineate the location depth and extent of the fracture site.

Despite our basic understandings of the mechanisms involved in blowout fractures, there has been a host of controversy regarding the proper management of these injuries. In 1957, Converse and Smith first advocated early surgical exploration after the clinical and radiological findings of blowout fracture were present. In a nonrandomized series of 50 patients with blowout fractures referred following unsuccessful treatment, 43 had diplopia and 27 had cosmetically unacceptable enophthalmos.

Converse and Smith emphasized that diplopia occurring in blowout fractures in the presence of positive forced ductions required inmediate surgery. They argued that the test implied permanent muscle entrapement which must be relieved in order to avoid late sequela.

Enophthalmos, the second major complication of fractures was, according to Converse and Smith, the result of a number of causative factors. They hypothesized four causes; 1) the escape of orbital fat from the orbital cavity, 2) enlargement of the orbital cavity without actual loss of fat, 3) Orbital fat necrosis from pressure caused by orbital hematoma or low grade inflammation, 4) maintenance of the ocular globe in a backward position by the entrapped muscle with eventual fibrosis of all the extraocular muscles. They believed that early correction of enophthalmos was necessary to prevent late deformity.

In 1971, Emery, et. al. reported a retrospective series of 159 patients, which contradicted the claims of Converse and Smith. They failed to revealed a significance difference in the incidence of enophthalmos between the surgical and monsurgical groups. Of more importance, was the observation that 24 of 49 patients had persistent diplopia after 3.4 years despite surgical repair in the first

two weeks. They felt that a few days after injury was enough time for these factors to subside and make forced ductions reliable. They recommended surgery in patients with continued diplopia and positive forced ductions after only 14 days. They recommended surgery as well for any patient who demonstrated on x-ray a large floor defect or prolapse of orbital contents despite negative forced ductions and absence of diplopia or enophthalmos.

In 1974, Putterman, Stevens and Urist reported 57 patients with pure blowout fractures who were not operated on and were followed for a variable of time. In all cases, the symptoms improved sufficiently so that no surgery was indicated. They hypothesized that traumatic hemorrhage and edema of the inferior fat pad, with or without prolapse of the maxilary sinus was the basic cause of blowout symptoms and not an entrapped inferior rectus as Converse and Smith had insisted. As a result, in most cases, resolution of edema, absortion of blood and stretching of the prolapse fibrous fat were believed to be responsible for the relief of symptoms after four months or more. They advocated that nonsurgical management of all blowout fractures for 4 to 6 months.

As recently as 1980, Mustarde has recommended inmediate surgery for patients with positive forced ductions or noticeable enophthalmos. His surgical criteria have been continued diplopia, positive forced ductions after 14 days, x-ray evidence of a large fracture of soft tissue prolapse.

Computed tomography has recently proven useful in providing a detailed visualization of soft tissue in cases of orbital blowout fractures. The technique of computed tomography has permitted visualization of soft tissue detail, not possible with conventional plain films or polytomography. In blowout fractures, nonspecific antral soft tissue densities which are visible with conventional radiography and which may represent orbital fat, extraocular muscle hematoma or an unrelated antral retention cyst, are now clearly distinguishable by CT. The accuracy and superiority over conventional tomography has been confirmed in the cadaver model.

In 1985, my colleagues and I asked the question: Can computerized tomography enable us to predict the late outcome of orbital blowout fractures? A prospective study of 19 patients with orbital blowout fractures were followed in a prospective study. All patients underwent conventional radiologic evaluation. Patients with positive radiographic evidence or a strong clinical suspicion of blowout fractures underwent computed axial and coronal tomography within one week of injury. Clinical examination including forced duction testing, motility evaluation and exophthalmometry were performed on

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all patients. Patients were followed for at least one month without surgery to allow for clinical improvement. On the average, patients were followed for 5 months with follow-up as long as 11 months. When possible, the CT scans were obtained during the convalescence period.

CT scans were read by a head and neck radiologists in a masked fashion in accordance with two separate criteria. First, all scans were graded with regard to the degree of orbital volume expansion and soft tissue prolapse. Grade I was defined by minimal orbital expansion and soft tissue herniation, as seen here. Grade II was defined as moderate expansion and prolapse and grade III significant expansion and prolapse. Second, all scns were evaluated with regard to inferior rectus muscle position in relation to the fracture site. Here is a scan where the inferior rectus is free and not in continuity with the fracture site.

Alternatively, here is a CT with the inferior rectus muscle hooked but not trapped. Finally, this is an example of an entrapment of an inferior rectus muscle.

All CT findings were correlated with clinical presentation and outcome. The appearance on CT of our 19 patients in the study is summarized here. Patients are grouped twice, once in each column according to the previously described criteria.

This graph correlates orbital volume expansion and soft tissue prolapse as seen on CT with enophthalmos. Grade I and II patients failed to demonstrate enophthalmos at presentation or follow-up. This is in contrast to grade III patients where one out of seven patients presented with enophthalmos and two more developed it later on.

This graph demonstrates the correlation between muscle position on CT and positive forced duction testing. At presentation, all patients with hooked and entrapped muscles had positive forced ductions. This is in contrast to 2 out of 12 patients with free muscles. Only patients with entrapped muscles on CT continued to have positive forced ductions on follow-up.

Finally, this graph illustrates the correlation between muscle position on CT and diplopia. Initial diplopia occurred in all CT muscle groups. Of note is that there is no correlation between the amount of diplopia and CT muscle position. On follow-up, all patients with hooked and free muscles had resolution of diplopia. In contrast, all 5 patients with entrapment of the inferior rectus muscle on CT showed clinically significant diplopia.

A number of important points can be made from this data. First, orbital volume expansion and soft tissue herniation are key in development of enophthalmos. Second, the CT scan can indicate which patients are not at risk for the development of enophthalmos. Thirdly, positive forced ductions can occur without entrapment of the inferior rectus muscle and need not imply continued diplopia. Finally, the CT scan can predict which patients will have persistent diplopia.

Although coronal CT is very helpful in evaluating orbital blowout fractures, routine CT scans on all patients having or suspected of having blowout fractures are unnecessary. Instead, CT should be used to aid surgical decision making when the clinical picture is unclear. To screen patients suspected of having blowout fractures routine orbital films are appropriate. If no entrapment is clinically evident and only a small fracture is present, a CT scan is unnecessary since no surgery is required. On the other hand, if routine orbital films show a moderate to large fracture, CT scan is advisable to delineate the degree of orbital prolapse. A large amount of prolapse on CT puts the patient at higher risk for enophthalmos; thus surgery is indicated. For patients with clinical evidence of entrapment, (i. e., positive forced ductions) CT should be performed to verify entrapment. Patients with CT proven entrapment are destined for continued diplopia and should undergo surgery to release the entrapped muscle. Finally, in cases of clinical enophthalmos at presentation, surgery is indicated and CT scanning is useful, not for surgical decision, making, but to guide the surgeon as to the depth of exploration.

In the past few years, I have used this approach to blowout fractures with great success. In addition, for those patients which CT has predicated the need for operative intervention, I have found it technically advantageous to operate within the first weed of injury. This has allowed for flexibility of the fracture site and easy release of prolapse or entrapped tissues.

Here is a case of a 35 year old white man who was referred following blunt trauma to the left orbit a few days previously. He had noted left infraorbital hypesthesia but no vertical diplopia. Examination showed his visual acuities to be 20/20 OU. Ductions and versions were full. He had echymosis over the left periorbital area and moderate swelling and hypesthesia along the left lower lid and cheek are. Hertel measurements were symmetrical at 16 mm with a base of 100. Forced duction testing was normal. Retinal evaluation was unremarkable. Slit lamp evaluation was remarkable only for mild iritis. There was no step deformity of the orbital rim. A Water's view demonstrated a large opacification in the left maxillary antrum, as well as depression of the orbital floor. The

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coronal CT scan confirmed significant floor depression on the left. The sinus was filled with blood with no fat present in this location. However, the orbital contents remained elevated with air separating bone from orbital contents. Periosteum was presumed to be intact holding up the orbital contents. The inferior rectus muscle was above the fracture site. Because of minimal orbital prolapse and a free inferior rectus muscle, this patient was observed without surgery. Nine months following injury, this patient had no diplopia, no enophthalmos and no hypesthesia.

Here is the case of a 33 year old woman who was referred following blunt trauma to the right orbit two days previously. She had vertical diplopia in up and down gaze and a right infraorbital hypesthesia. Examination showed that her vertical visual acuity was 20/25 OU. Motility evaluation revealed severe restriction in the right eye in up and down gaze. There was diffuse swelling in the right lower lid area, as well as hypesthesia. Hertel measurements were 15 mm OU with a base of 91. Forced duction testing was positive. There was no palpable step deformity in the inferior orbital rim. The examination was otherwise unremarkable. A water's view seen here revealed opacification of the right sinus with some floor depression. Coronal CT demonstrated entrapment of the inferior rectus muscle, minimal floor depression and prolapse of small amount of orbital fat. Because of CT conformation of muscle entrapment, this patient underwent surgery 8 days after injury. At surgery, the rectus muscle was found to be entraped as seen here and a Teflon implant was placed. Four months after injury the patient had one prism diapter of tropia in up and down gaze that caused no functional difficulty. She had no enophthalmost but does have residual hypesthesia.

Here is a case of a 33 year old woman, who was referred following blunt trauma to the right orbit one week previously. She had noted infraorbital hypesthesia and vertical diplopia. On examination, her visual acuity was 20/20 OU. Motility evaluation revealed 4 prism diapters of tropia in extreme up and down gaze. She had approximately 1 to 2 mm of right upper lid ptosis, secondary, to lid swelling and hypesthesia along the infraorbital nerve. Hertel measurements were symmetrical with no evidence of enophthalmos. Forced duction testing was normal. There was no palpable step along the infraorbital rim. Slit lamp evaluation was remarkable for mild iritis on the right. Retinal evaluation was unremarkable. A Water's view demonstrated a large amount of opacification of the right maxillary sinus, as well as depression of the orbital floor. The coronal CT scan confirmed significant depression of the orbital floor on the right and revealed the sinus opacification to be orbital fat of the grade III type. The muscle was free in the fracture site. The patient underwent surgical

repair eight days after injury. Here is that patient on the operating room table. A subcillary incision was made down to the level of the orbicularis muscle. The orbicularis plane was entered just above the area of the inferior orbital rim. The periostium was incised approximately 1 to 2 mm below the infraorbital rim. Care must be taken to avoid the infraorbital nerve which exits approximately 5 to 6 mm below the orbital rim. In this patient, a large fracture was verified, the fracture was elevated and the prolapse tissue was returned to the orbit and supported with a Teflon implant. The lower lid was then closed with a running interrupted 6-0 nylon closure. Ten months following injury, this patient continued to do well with resolution of diplopia, hypesthesia and no enophthalmos.

In summary, CT scan is extremely useful in predicting the outcome of orbital blowout fractures. It can identify which patients are at risk for enophthalmos or persistent diplopia, thus indicating the need for operative intervention. For patients requiring surgery, I have found it technically advantageous to operate within the first week of injury.