mRNA was significantly higher in the Nell-1–treated group than in the control group, whereas no differences in the expression of aggrecanase or IL-1 mRNA were found between the experimental and the control group. This finding is also in accord with an earlier study in vitro[15] and suggests that in vivo Nell-1 also has a potent ability to increase type II collagen and aggrecan within the ECM to protect the articular cartilage against destruction in TMJOA.

In conclusion, our results showed that intra-articular injection of Nell-1 can protect the articular cartilage from degeneration by stimulating synthesis of type II collagen and aggrecan in TMJOA models induced by partial perforation of the articular disk, which suggests that intra-articular injection of Nell-1 into the TMJ may be a good alternative for the treatment of cartilage degeneration in OA.

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Surgical Management of Posttraumatic Intraorbital Hematoma

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Abstract: Retrobulbar hematoma is a rare condition but represents a diagnostic and therapeutic emergency. It occurs in between 0.3% and 3.5% of facial traumas and can be caused by direct or indirect injury of the orbit; they can be classified into intraorbital and subperiosteal hematoma. We describe 4 different cases of posttraumatic retrobulbar hematoma treated at the Unit of Maxillofacial Surgery of the Novara Major Hospital between January 2005 and December 2009, each different from the others for morphologic aspects, and we

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discuss its diagnosis and management. Surgery decompression of the orbit is recommended when visual deficit arises and when there is no response to pharmacologic therapy. Several techniques for orbital decompression have been proposed. The lateral canthotomy and/or the inferior cantholysis are the 2 techniques most practiced. Anterior-chamber paracentesis is effective, but it is rarely indicated for frequent complications such as cataract formation, herniation of the iris, infection, and trauma to the canal of Schlemm. Other procedures including transantral ethmoidectomy, transantral sphenoidectomy, and transfrontal craniotomy are described.

Key Words: Retrobulbar hematoma, orbital decompression

Retrobulbar hematoma is a rare condition but represents a diagnostic and therapeutic emergency. It occurs in between 0.3% and 3.5% of facial traumas and can be caused by direct or indirect injury of the orbit; they can be classified into intraorbital and subperiosteal hematoma.[1,2]

The pathophysiology of retrobulbar hemorrhage that leads to blindness still remains unclear. The currently accepted theory states that bleeding into a nonyielding space, the orbit, causes an increased intraorbital pressure, which begins a vicious circle? leading to an irreversible injury if not promptly recognized and treated.[3,4]

Bleeding into a nonyielding space, the orbit, causes an increased pressure that impairs intraorbital structures such as (1) ocular bulb, (2) optic nerve, and (3) blood vessels. Scientific literature describes the association between retrobulbar hematoma and intracranial extradural/subdural hematoma.[5–7]

Initially, the increase in intraorbital pressure displaces the globe anteriorly, which is limited by the eyelid apparatus, the orbital septum, and the optic nerve itself. Therefore, the increased intraorbital pressure represents a compartment syndrome with venous compression. The consequent venous stasis contributes to increasing the intraorbital pressure, which damages the arterial system with compression of retinal artery, decreasing perfusion pressure, and subsequently ischemic injury.[8–10]

We describe 4 different cases of posttraumatic retrobulbar hematoma treated at the Unit of Maxillofacial Surgery of the Novara Major Hospital between January 2005 and December 2009, each different from the others for morphologic aspects, and we discuss its diagnosis and management (Tables 1 and 2).

MATERIALS AND METHODS

Patient 1

Patient 1 was a 65-year-old man with facial trauma after accidental fall. On physical examination, the patient showed right eyelid hematoma, right chemosis, and a deep laceration of the right upper eyelid. The pupils were equal in size. Extraocular muscle motility, light-eyelid reflexes, and visual acuity were present. Computed tomography (CT) showed right orbital roof and lateral wall fracture. No associated lesions were present.

Twenty-four hours after the trauma, the patient experienced major decline in visual acuity of the right eye accompanied by right occipital headache. He underwent eye examination that reported loss of vision of the right eye and the absence of direct photomotor reflex.

Therapy was initiated with intravenously administered dexamethasone 1 mg/kg every 6 hours. He underwent magnetic resonance imaging (MRI) of the head and neck that highlighted the presence of subperiosteal intraorbital hematoma in the superior compartment, which displaced the superior rectus muscle inferiorly (Figs. 1A, B). There was no evidence of signs or symptoms of intracranial extradural hematoma.

The patient underwent emergency surgery through a 2-cm supraciliary incision and the evacuation of the hematoma by blunt dissection. Intravenous steroid therapy was maintained for 3 days. The patient showed a progressive improvement of visual acuity. The dose of dexamethasone was reduced to 0.5 mg/kg every 6 hours and continued for another 2 days. Oral therapy was then set with prednisone 25 mg twice daily for 3 days, later reduced to 5 mg twice daily. After 10 days of hospitalization, the patient was discharged with a good functional recovery. Ophthalmologic and maxillofacial monitoring was planned once a week for the first month and then monthly for the next 6 months. The patient showed no signs and symptoms of recurrent retrobulbar hematoma, showing visual function improvement with some residual impairment.

Patient 2

Patient 2 was a 25-year-old woman with craniofacial trauma after a car accident. Head and neck examination revealed left eyelid

TABLE 1. Demographic Characteristics of the Patients Considered in This Study

Patients	Age, y	Sex	Associated Lesions	Type of Fracture	Location of Hematoma
Patient 1	65	Male	No	Orbitomaxillozygomatic	Extraconal, superior compartment
Patient 2	25	Female	No	No	Intraconal
Patient 3	28	Male	Yes	Orbitomaxillozygomatic	Intraconal
Patient 4	10	Male	No	None	Extraconal, superior compartment

TABLE 2. Clinical Characteristics of the Patients Considered in This Study

Patients	Onset Delay	Diagnosis	Medical Treatment	Surgical Treatment	Visual Function Recovery
Patient 1	24 h	CT and MRI	Dexamethasone	Supraciliary, drainage	Partial
Patient 2	3½ h	CT and MRI	Dexamethasone	Supraciliary, drainage	Yes
Patient 3	Immediately	CT	Methylprednisolone	Paralateronasal, drainage	Yes
Patient 4	No	CT and MRI	None	Percutaneous	Yes

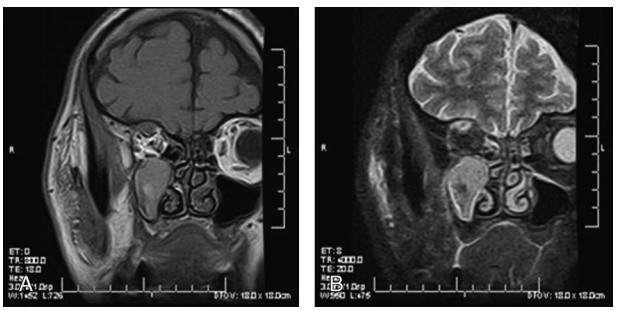

FIGURE 1. A and B, Coronal MRI scan showing intraorbital hematoma in the superior compartment displacing inferiorly the superior rectus muscle.

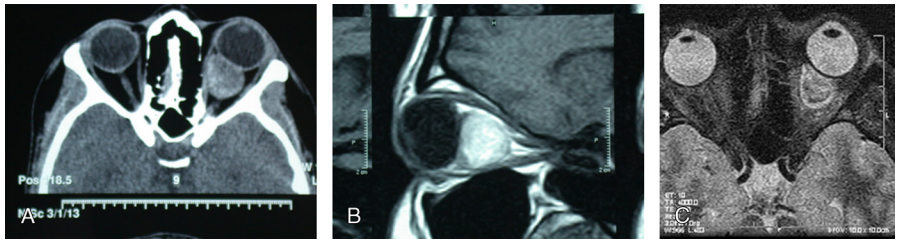

FIGURE 2. A–C, Computed tomography and MRI scans showing left retrobulbar intraconal hematoma.

hematoma, left exophthalmos, and decreased extraocular muscle motility. The direct photomotor reflex and the visual acuity were progressively deteriorated. Computed tomography and MRI showed left retrobulbar hematoma (Figs. 2A–C) without concomitant maxillofacial fracture. Chemotherapy was started with intravenously administered dexamethasone 4 mg every 12 hours for 2 days; the patient underwent immediate decompressive surgery.

Through a left supraciliary medial incision, the medial wall and the roof were approached, the intraconal compartment was exposed by blunt dissection, and the hematoma was evacuated. Intravenous chemotherapy was continued for 3 days after surgery. Three days after surgery, the eye examination showed improvement in ocular motility, and the vision was preserved. The dose of dexamethasone was reduced and continued for another 3 days. Oral therapy was then set with prednisone for 2 weeks.

The patient was discharged with ophthalmologic and maxillofacial checkups weekly. The patient had a complete recovery of visual function without recurrent retrobulbar hematoma.

Patient 3

Patient 3 was a 28-year-old man with facial trauma after a work accident. Clinical examination showed a bruised right eyelid with a massive hematoma, a supraciliary lacerated wound, exophthalmos, and chemosis. The extrinsic ocular motility was limited, and the direct photomotor reflex was absent; the affected eye presented no vision. There were concomitant pneumothorax, hepatic contusion, and hip fracture. Computed tomography ruled out the presence of orbital wall fracture and anterior displacement of right eyeball by hyperdense retrobulbar adipose tissue of hemorrhagic nature.

The patient underwent emergency surgery. Through a 1-cm paralateronasal incision, the blood collection was drained with a blunt cannula, and careful hemostasis was achieved. Intravenous therapy with methylprednisolone 5.4 mg/kg every hour for 24 hours was set and then prednisone 1 mg/kg per day for 11 days.

Two days after surgery, CT scan documented the reduction of the retrobulbar hyperdensity. Eye examination showed clinical improvement with recovery of the visual acuity and the extraocular movements. At 4 days after trauma, the patient was discharged with ophthalmologic and maxillofacial checkups weekly. The patient showed no signs and symptoms of recurrent retrobulbar hematoma.

Patient 4

Patient 4 was a 10-year-old boy with craniofacial trauma after a car accident. Clinical examination showed swelling and bruising of the left eyelid with the absence of any sensory and motor deficits. Computed tomography of the orbits showed intraorbital hematoma above the superior rectus muscle (Fig. 3A).

After 6 hours, MRI of the orbits was performed, and it confirmed the presence of subperiosteal spindle-shaped fluid collection between the roof of the orbit and the superior rectus muscle (Fig. 3B). He underwent emergency surgery through a percutaneous drainage of the hematoma with a blunt cannula.

Steroid therapy was not set because the patient had no evidence of visual impairment. At the third day, postoperative CT scan showed the reduction of the subperiosteal blood collection. After 4 days of hospitalization, the patient was discharged with ophthalmologic and maxillofacial assessment every week for the first month and then monthly for the next 4 months. Three months after surgery, MRI showed complete "restitutio ad integrum" of the superior orbital compartment.

DISCUSSION

Retrobulbar hematoma is a rare complication of blunt periorbital trauma. Hayreh et al[11] demonstrated that, in case of increased intraorbital pressure, the vessels anterior to the lamina cribrosa sclerae were first compressed, followed by the peripapillary choroidal vessels, the retrolaminar vessels of the optic nerve, and finally the retinal central artery. Moreover, they demonstrated that the irreversible retinal injury requires 3 hours or more to set up.

Huang et al[12] injected autologous blood into the retrobulbar space in 24 rabbits to evaluate the effects of retrobulbar hemorrhage. Intraorbital injection of blood immediately produced lid edema, lid ecchymosis, chemosis, mydriasis, and proptosis. Subsequently light-eyelid reflexes disappeared. The vision was preserved because the intraorbital pressure was not maintained long enough to produce irreversible optic nerve or retinal damage.

Rahn[13] stated that intraorbital pressure has to exceed the mean arterial pressure by 60 to 70 mm Hg to result in central artery occlusion. It is known that retinal circulation is usually protected by various compensatory mechanisms, so that the blood flow in the retina is adequately maintained even if the entire system is under stress.

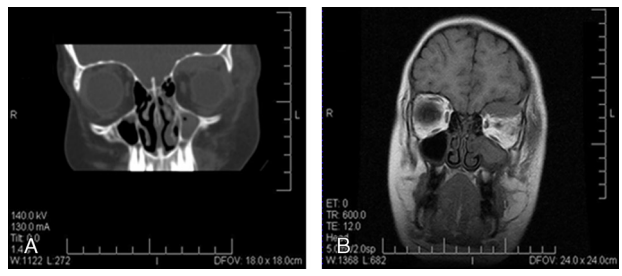

FIGURE 3. A and B, Computed tomography and MRI scans of the orbits showing the presence of subperiosteal spindle-shaped fluid collection between the roof of the orbit and the superior rectus muscle.

The neural tissues are vulnerable to ischemic injury, so that a transient interruption of the blood supply to the retina can cause neural cells to be unresponsive to light stimuli.[14] The optical nerve damage, caused by expensive lesions into the retrobulbar space, is caused by 3 different mechanisms: neurovascular compression, optic nerve stretch, and ischemia.[15]

It is usually self-limiting because the hemorrhage into the retrobulbar space is drained into the paranasal sinus through fractures of orbital walls. Nevertheless, in the absence of fractures or in the presence of linear fractures, the drainage could be absent with arising of retrobulbar pressure and irreversible damage of vision system.[2]

The clinical suspicion of retrobulbar hematoma must be adequately investigated and promptly treated to avoid the onset of irreversible and devastating complications.[6,16] Onset of retrobulbar hematoma can be several days after traumatic episode; therefore, the patient should be observed carefully to individuate the presence of chemosis, proptosis, painful exophthalmos, decreased visual acuity, and extraocular muscle motility.[1] Moreover, the clinical presentation can be variable, and it can be confused with other conditions such as traumatic superior orbital fissure syndrome and carotid-cavernous sinus fistula.

Prevention of devastating sequelae relies on a prompt diagnosis. Magnetic resonance imaging and CT proved to be tightly complementary in the diagnostic assessment, determining (1) orbital wall fractures, (2) extension of any hemorrhagic lesions, and (3) associated intracranial hematomas.[16,17]

Magnetic resonance imaging has several advantages including (1) lack of harmful ionizing radiation, (2) multiplanar imaging, and (3) high soft tissue contrast resolution.[7] It is the modality of choice for characterization of subperiosteal hematomas, which represent the rarest subtype of hemorrhagic intraorbital lesions.[5]

Treatment of this condition should be addressed as soon as signs of retrobulbar hemorrhage appear, avoiding irreversible events. Despite conservative measures including immediate eye massage to reduce intraorbital pressure, bed rest, and elevation of the head, prompt pharmacologic therapy should be started. Pharmacologic therapy includes administration of mannitol, acetazolamide, and high doses of corticosteroids.

Surgery decompression of the orbit is recommended when visual deficit arises and when there is no response to pharmacologic therapy. Several techniques for orbital decompression have been proposed. The lateral canthotomy and/or the inferior cantholysis are the 2 techniques most practiced.

Under local anesthetic, the lateral canthotomy is performed by placing an artery clip between the upper and lower lids and then transecting the lateral canthus. The inferior cantholysis detaches the inferior crus of the lateral tendon, leading to a complete mobile lower eyelid.

Anterior-chamber paracentesis is effective, but it is rarely indicated for frequent complications such as cataract formation, herniation of the iris, infection, and trauma to the canal of Schlemm.

More extended approaches to the internal orbit with a more complex procedure and a higher morbidity are used in the case of extensive retrobulbar hemorrhage and when it is necessary to manage complex fractures or optic nerve decompression. These procedures include transantral ethmoidectomy, transantral sphenoidectomy, and transfrontal craniotomy.[18,19]

Carbonic anhydrase inhibitors such as acetazolamide 500 mg, intravenously administered hydrocortisone 100 mg, and a rapid infusion of 20% mannitol are frequently used. The action of mannitol is immediate, whereas that of acetazolamide is delayed. Steroids reduce inflammation and stabilize cell membranes against ischemic damage. Other nonsurgical treatment methods suggested include the use of timolol maleate eye drops, 0.25% topical solution, which decreases the production of aqueous humor.

The necessity of a rapid diagnosis and management of a retrobulbar hemorrhage has been described in this article. A combined medical and surgical management is the most appropriate treatment option for this condition; this maxillofacial emergency has a variable clinical presentation, and the onset can be delayed. Visual acuity must be monitored in all patients affected by orbital trauma, and CT or MRI assessment should be performed in any suspected case to permit a prompt diagnosis and management, avoiding permanent blindness.

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