Post-Traumatic Tension Pneumoorbitus Case Report

M. Akif Bayar, Fatih Kökeş, Cevdet Gökçek, Saffet Doğanay, Nurullah Edebali, Zeki Buharali

Neurosurgical Clinic, Ministry of Health Ankara Hospital (MAB, CG, SD, NE, ZB), Sevgi Hospital (FK), Ankara - Türkiye

Abstract : Post-traumatic pneumoorbitus is generally a benign, self-limiting condition. However, if a fracture produces ball-valve effect allowing air to enter but not leave the orbit, then extremely high intraorbital pressure and visual loss are possible. A 30- year-old man with loss of vision because of tension pneumoorbitus who was successfully treated by craniotomy and in whom the defect in lamina papyracea was repaired by muscle packing is presented here.

Key Words : Orbital trauma, pneumoorbitus, central retinal artery occlusion

INTRODUCTION

Pneumoorbitus is usually the result of a fracture of the lamina papyracea or maxillary roof allowing air passage from the sinuses into the orbit. Although there is almost always a history of antecedent trauma, spontaneous pneumoorbitus following violent nose blowing or sneezing has also been reported (1). The diagnosis of pneumoorbitus is not difficult. Palpation of the eyelids produces pathognomonic crepitation and crackling. Although radiological evidence of pneumoorbitus has been reported in 50 % of cases with orbital fracture, the condition is generally a benign process (4). Three cases of central retinal artery occlusion as a result of pneumoorbitus have been previously reported (2,3). We report herein a fourth case in which, via a craniotomy, the loss of vision was reversed by decompression of the orbit and the defect of the ethmoid sinus wall was repaired by "muscle packing".

CASE REPORT

A 30-year old man was beaten and kicked in the left periorbital region. Twenty-four hours later, he was admitted to our clinic with severe pain and impaired vision of the left eye. On physical examination, there were proptosis, periorbital ecchymosis and oedema on the left side. Motility of the left eye was severely restricted in all directions. The left pupil was mid-dilated. There was significant loss of vision with only light perception in the left eye. On fundus examination, optic disc pallor and stasis within the retinal veins were noted on the left.

Orbital computed tomografic scan showed a defect of the lamina papyracea on the left orbital wall and massive intraorbital air (Fig. 1).

The patient was taken for emergency operation. Removing the left orbital roof by left frontal craniotomy, the defect in the lamina papyracea of the

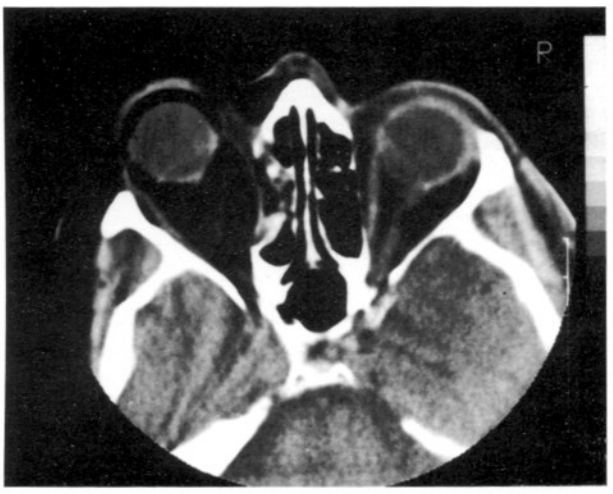

Fig. 1 : Orbital CT scan showing the defect of lamina papyrecea on the left orbital wall and massive intraorbital air.

orbital medial wall was exposed. This was repaired by "muscle packing". At the sixth post-operative hour, the patient's vision returned to normal completely. The patient was discharged on the eighth post-operative day. No complaints or abnormal physical examination findings were reported in the follow- up visit at the end of the first month.

DISCUSSION

It is reported that, 50 % of cases with traumatic orbital fracture show pneumoorbitus radiologically (4). However, this pathology is usually asymptomatic and benign, and no specific treatment is needed. But in some cases the pneumoorbitus may become enlarge, causing occlusion of the central retinal artery by increasing the intraorbital pressure. This process may lead to proptosis and loss of vision. This is possible only when: 1. There is a fracture in one of the orbital walls; 2. The mucosal layer of the paranasal sinus adjacent to the fracture is destroyed, allowing air passage between the sinus and the orbit; 3. There is a ball-valve mechanism in the fracture region that lets air pass through the sinus to the orbit but does not let it out. To sustain the pressure rise also requies that the orbital septum remain intact. Thus for pneumoorbitus to cause visual loss, two conditions must be met. First, an orbital wall fracture into a neighboring paranasal sinus must be present with a configuration of tissues at the fracture site such that in central retinal artery is allowed to enter but not exit the orbit. Second the orbital septum must remain intact even when intraorbital pressure is markedly increased. In the case described herein the increase in intraorbital presure probably obstructed retinal circulation, producing pallor of the optic disc and stasis in the central retinal vein. The clinical, radiological and intraoperative findings of the case we present confirm this process.

Emergency treatment of high intraorbital pressure is of primary importance for the management of these cases. The defect causing the ball-valve mechanism should also be repaired. In one of the three reported cases, only direct needle aspiration was performed (3). Lateral canthotomy and cantholysis were performed in the other two (2). The defect in the orbital wall was not repaired in those cases. In the patient we present here, intraorbital air was released by left frontal craniotomy, and the defective area was repaired by "muscle packing" to prevent a recurence of tension pneumoorbitus. This way, the air passage between the sinus and the orbit was eliminated. This procedure is thought to be a more radical surgical approach.

Correspondence : M. Akif Bayar
Gençlik Caddesi, Döngel Sokak, No: 12/6
Maltepe, Ankara - Türkiye

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