Intraorbital Subperiosteal Hematoma due to Paranasal Mucocele
—Case Report—

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Abstract

A 68-year-old female was admitted with a rare intraorbital subperiosteal hematoma manifesting as sudden orbital pain, progressive blepharoptosis, and diplopia. Computed tomography showed a biconvex high density mass in the superomedial part of the left orbit, which was recognized to be a subperiosteal hematoma intraoperatively. The eroded orbital roof between the frontal sinus and orbit was a result of mechanical compression by the mucocele. Infection extending into the orbit was important in causing the hemorrhage.

Key words: orbit, subperiosteal hematoma, paranasal mucocele

Introduction

Paranasal mucoceles generally occur in the maxillary sinus, but a paranasal mucocele invading the intraorbital cavity may be the cause of exophthalmos and eye movement disorder. The incidence of paranasal mucocele is high, at 2–18% of total intraorbital lesions. Subperiosteal hematoma associated with mucocele has been reported in only six cases. We described an extremely rare case of paranasal mucocele associated with subperiosteal hematoma that manifested as sudden onset of orbital pain and exophthalmos.

Case Report

A 68-year-old female experienced sudden orbital pain on May 31, 1994. She visited our clinic the next day because of eyelid ptosis and double vision. She had been suffering from a cold for a few days, but had no history of trauma. Her left conjunctiva was red with no pulsation and left ocular movements were limited in all directions, but the eyeball was intact. Funduscopic examination was within the normal range. Blood, urine, and physical examinations found no abnormalities.

Skull radiography showed a cloudy area in the left frontal sinus, extending to the medial side of the orbit. Computed tomography (CT) revealed a relatively high density area that filled the left anterior ethmoidal cells through the frontal sinus and a well-demarcated biconvex mass extending into the orbit via the orbital roof and compressing the left globe inferomedially (Fig. 1). The lesions were not enhanced by contrast medium. T1-weighted magnetic resonance (MR) imaging demonstrated a hypointense mass and T2-weighted MR imaging showed a hyperintense mass in the same area (Fig. 2). Angiography showed no remarkable findings except for downward shift of the left ophthalmic artery.

Conservative therapy was begun under the diagnosis of intraorbital mucocele invading from the frontal sinusitis with sudden deterioration due to hemorrhage. Steroids were effective in improving the eye movement impairment by acute inflammation of oculomotor nerve. However, she suffered from blepharoptosis and exophthalmos even after daily administration of 500 mg dexamethasone for 5 days. CT showed the mass still remained. Surgery was performed on the 10th day after onset. The subfrontal extradural approach was used for removing the an-
terior portion of the orbital roof and preventing subdural infection. The frontal sinus was opened and found to be filled with a dark greyish hematoma. Eroded bone was recognized at the bottom of the sinus with an old clot extending into the intraorbital cavity (Fig. 3). The hematoma in the orbit was removed easily, and was bordered with intraorbital soft tissue by periosteum. Neither tumors nor vascular abnormalities were observed. Mucosa was removed as far as possible. No drainage was set up.

Histological examination showed mucosal tissue under the hematoma. Infiltration of leukocytes and edema established a diagnosis of subperiosteal hematoma associated with chronic sinusitis.

Postoperatively, her exophthalmos and eye movements were improved. She was discharged, with mild blepharoptosis. During the follow-up period of 20 months, she has been uneventful.

Discussion

The frontal sinus is formed by upward growth of the

Neurol Med Chir (Tokyo) 37, August, 1997
anterior ethmoidal cells. The nasofrontal duct is longer than other draining pathways to the nasal cavity and the narrow orifice is easily obstructed, which results in mucocele formation. Increasing content of mucocele gradually destroys the adjacent bony structures. The bottom and anterior wall of the frontal sinus are especially thin. We confirmed intraoperatively that the infection had spread through the eroded partition. Accidental hemorrhage might compress the intraorbital soft tissue, and result in sudden onset of exophthalmos and oculomotor palsy.

The hemorrhage is mainly caused by trauma. Small vessels under the periosteum are mechanically disrupted, resulting in a subperiosteal hematoma. However, hemorrhage may be secondary to paranasal sinusitis, or idiopathic or spontaneous Spontaneous orbital hemorrhage may occur because of extension of phlebitis from the valveless veins in the sinus mucosa to those in the subperiosteal space.

Our patient had no evidence of fever, leukocytosis, or increased erythrocyte sedimentation rate. However, MR imaging revealed acute hemorrhage mixed with another material. The bony defect between the frontal sinus and orbital space demonstrated the presence of mucocele. Therefore, the pre-existing mucocele very probably compressed and destroyed the superior wall of the orbit during growth, and the infection that extended to the subperiosteal veins induced the vessels to rupture, finally resulting in acute hemorrhage.

Mucocele itself can be cured simply by transnasal drainage. However, some refractory cases required repeated aspiration from the sinus. External fronto-ethmoidectomy and lateral orbitotomy are also safer methods. Radical management is required to avoid the possibility of permanent visual deficits, as vascular or neoplastic lesions may be present. We employed the transcranial approach, which was useful for wide exposure of the orbital roof and the identification of abnormalities.

References


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