Orbital blood cyst

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SUMMARY A blood cyst of the orbit is an unusual cause of proptosis and most likely arises in a pre-existing haemangioma. If the cyst occurs at the apex of the orbit the blood should be aspirated. This is followed by excision of the cyst wall, the dissection being done with the aid of an operating microscope. If there is evidence of intracranial connection of the blood cyst, namely, splayed superior orbital fissure or cerebrospinal fluid leak, then the patient should be treated with an antibiotic to prevent intracranial infection.

An orbital blood cyst is an unusual cause of unilateral proptosis (Henderson, 1973). From 1950 to 1977, 4 histologically proved cases have been described, Wolter *et al.* (1966) 1 case; Mortada (1969) 2 cases; Henderson (1973) 1 case. Confusion exists on the origin of orbital blood cysts. The purpose of this communication is to illustrate that the cyst may arise in a pre-existing haemangioma.

Case report

A mulatto child aged 3 years, presented to the department of ophthalmology at Groote Schuur Hospital on 14 March 1977 with a 3-month history of pain in the right eye associated with proptosis. There was a vague history from the mother that the child had bumped the right side of the head approximately 3 to 4 months before the development of proptosis.

The visual acuity of both eyes was 6/6 (Sheridan Gardiner chart). Both eyes were emmetropic. The right eye was proptosed (right eye 15 mm and left eye 12 mm). There was no ecchymosis of the lids, and no subconjunctival haemorrhage was noted (Henderson, 1973). The proptosis was axial, and extraocular movements were full in all directions of gaze.

Examination of the anterior segment of the right eye showed no lesion. There was no papilloedema, the retinal veins were minimally dilated, and the retina was not oedematous. No tension striae were seen in the retina. No masses were palpable in the orbit and the proptosis was not reducible. No bruit was heard over the right eye. Examination of the

left eye showed no abnormality. Peripheral and central fields could not be assessed because of the patient's immaturity.

General examination added nothing of importance. There were small non-tender submandibular lymph nodes. The tonsils were slightly enlarged but not injected. Occasional scattered rhonchi were heard in both lung fields. There was no abdominal visceromegaly, and examination of the cardiovascular system was normal in all respects. There was no systemic evidence of scurvy (Duke-Elder, 1974).

The patient was examined by an otolaryngologist, and no abnormality was found in the nose, nasopharynx, paranasal sinuses, or ears.

A full blood count showed the haemoglobin was 13 g/dl, MCV was $76 \mu\text{m}^3$, and the sedimentation rate was 28 mm in 1 hour. The white blood count was $14\,000/\mu\text{l}$ ($14 \times 10^9/\text{l}$); the differential count was neutrophils 35%, lymphocytes 40%, eosinophils 17%, band cells 7%, monocytes 7%, and platelets $386\,000/\mu\text{l}$ ($386 \times 10^9/\text{l}$).

X-ray of the chest showed consolidation of the right inferior lobe commensurate with a diagnosis of bronchopneumonia. X-ray of the skull showed no abnormalities. X-ray of the orbit showed a right enlarged superior orbital fissure (Fig. 1). The edges of the orbital fissure were well defined, and the bony strut between the optic canal and the orbital fissure was not eroded. Tomograms of the orbit and optic canal of the right side showed no abnormality apart from expansion of the superior orbital fissure. Tomography of the left superior orbital fissure and optic canal was normal.

Computerised axial tomography (with and without contrast medium, Conray 420) showed the right globe to be displaced anteriorly. A heterogeneous (density 10 to 15) avascular mass occupied the lateral

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Fig. 1 The right superior orbital fissure (arrowed) is enlarged

Fig. 2 Computerised axial tomography. The right eye is proptosed and a heterogeneous mass occupies the temporal side of the orbit, causing medial bowing of the optic nerve (arrowed)

aspect of the orbit and extended from the posterior pole of the eye to the apex of the orbit (Fig. 2). No intracranial extension of the mass could be detected. The lateral ventricles were normal.

Ultrasonography showed a mass at the apex of the orbit. The liver scan was normal. The bone marrow aspirant and trephined biopsy were normal in all respects.

On 28 April 1977 a standard lateral orbitotomy was done. The periorbita was divided in the long axis of the orbit and a large blue cystic mass was palpated within the orbit. The cystic mass was approximately 25 to 30 mm in diameter and lay within the muscle cone between the optic nerve and the lateral rectus muscle, extending from the posterior aspect of the eye to the apex of the orbit. An attempt was made to dissect this mass (Fig. 3), but it was intimately connected with the surrounding orbital tissue. Further dissection may have caused damage to the origin of the lateral rectus muscle, to the blood supply of the optic nerve, or to the nerves and vessels traversing the superior and inferior orbital fissure.

A fine-bore needle (25 gauge) was therefore inserted into the cyst and approximately 15 ml of dark blood was aspirated. Serosanguinous fluid followed and then clear cerebrospinal fluid. The fluid was placed in a test-tube and, after an hour, did not clot.

The entire cyst collapsed and the question arose whether or not the communication between the blood cyst and the subarachnoid space should be

Fig. 3 Partially dissected blood cyst

closed. A neurosurgeon (Professor J. de Villiers, Department of Neurosurgery, Groote Schuur Hospital) was called, and he considered there was no indication at that stage to do an intracranial exploration.

The cyst wall was excised with the aid of an operating microscope, care being taken to confine the dissection to the plane of cleavage between the

cyst wall and the surrounding orbital tissue. On completion of the excision, a penicillin powder was placed in the cavity. The periorbital was closed with interrupted 5/0 chromic catgut and the bone replaced. A suction drain was inserted.

The patient was treated for 2 weeks with sulphadiazine 250 mg orally 4 times a day, penicillin 250 mg orally 4 times a day, and chloromycetin 250 mg orally 4 times a day as a prophylaxis against post-operative intracranial infection.

The drain was removed after 48 hours.

Postoperatively the patient did not develop proptosis, and the extraocular movements and visual acuity of the right eye remained unimpaired. The right pupil reacted to light stimulation, and fundal examination showed no abnormality of the optic nerve head; the retinal vasculature was normal, and there was no retinal or macular oedema.

Histopathology of the cyst wall showed fibroblastic proliferation, organised haemorrhage with degenerate blood pigment, and cholesterol crystal clefts (Fig. 4). Foreign body giant cells, pigment-laden macrophages, and foamy cells containing lipid were present. In addition there was a compressed cavernous haemangioma in the cyst wall (Figs. 5-6).

Discussion

Orbital blood cysts may occur at any age (Henderson, 1973) and cause a slowly developing unilateral proptosis. A history of trauma to the orbit is not consistently obtained. The blood cyst may cause enlargement of the orbit and splaying of the superior orbital fissure. The location of the cyst determines the symptomatology.

In the past it has been suggested that orbital blood cyst may arise from a retrobulbar haemorrhage following trauma, haemorrhage associated with the blood dyscrasia (Henderson, 1973; Duke-Elder, 1974), or a retrobulbar injection (Duke-Elder, 1974). Usually the onset of proptosis due to retrobulbar haemorrhage is sudden and the orbital tissues become infiltrated with blood. During the period 1950 to 1977, 22 patients with proptosis associated with retrobulbar haemorrhage were admitted to the Department of Ophthalmology at Groote Schuur Hospital. The trauma was usually of a severe nature and often associated with an orbital fracture. In this period no blood cysts developed. Furthermore, if blood cyst of the orbit commonly followed orbital haemorrhage of traumatic origin, it would be expected that more cases would have been reported (see above).

During 1950 to 1977, 4 patients presented with an orbital haemorrhage associated with blood dyscrasia. Again none of these developed a blood cyst.

Fig. 4 Cholesterol clefts surrounded by granulomatous reaction in the wall of the cyst (H and E $\times 160$)

Fig. 5 Haemangiomatous malformation (arrowed) in wall of cyst adjacent to cholesterol clefts (H and E $\times 410$)

Fig. 6 Haemorrhage, cholesterol clefts, and haemangioma (arrowed) in wall of blood cyst (H and E $\times 160$)

It has been suggested that a blood cyst may occur in an area of fat necrosis. Orbital fat necrosis follows injury of the orbit (Mortada, 1969; Duke-Elder, 1974). The result is a solid mass characterised histologically by a granulomatous reaction surrounding areas of fat necrosis. During 1950 to 1977, 2 cases of proptosis and histologically proved fat necrosis were observed at Groote Schuur Hospital. Again an orbital blood cyst was not found.

Jones (1959) and Mortada (1961, 1969) described blood cysts originating from a haemangioma or lymphangioma. Both described the blood cyst lined by endothelium. In the present case the haemangiomatic malformation found adjacent to the blood-filled cavity indicates that the primary pathology of the orbital blood cyst was a haemangioma. Bleeding may have occurred in the haemangioma or thrombosis in one of the major feeding vessels, and as a result a haemorrhage followed, causing compression of the tumour. Because of recurrent haemorrhage, the wall of the cyst became haemosiderin-laden and cholesterol clefts formed. The latter evoked a granulomatous reaction.

The orbital fissure may be enlarged because of the siting of the tumour at the apex of the orbit (Mortada, 1961, 1969). The exudation of sero-

sanguinous fluid followed by frank cerebrospinal fluid in the present case indicated that there was some communication between the blood cyst and the subarachnoid space. This communication occurred in relation to the superior orbital fissure. Because of the intracranial communication and cerebrospinal fluid leak the patient was treated intensively with antibiotics to prevent intracranial infection.

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