they lack an epithelial lining.[1] In our case, keratinising corneal epithelium covered the lesion rather than lining it, thus confirming it as a pseudocyst.

Corneal epithelial pseudocysts are a rare condition. They are the late consequence of inadequate healing and repair within the cornea following a variety of pathologic insults.[2]

A more common cause for pseudocyst formation is surgically induced trauma after cataract surgery. It has been well established that corneal endothelial decompensation leading to bullous keratopathy is a common cause of corneal epithelial bulla — a form of pseudocyst.[3]

A large pseudocyst following acute hydrops was described by Margo and Mosteller.[1]

Pseudocyst formation is the structural manifestation of a failed reparative response to a specific injury. There seems to be a requirement for the initiation of bulla formation (eg corneal decompensation, raised IOP); injury to corneal endothelium leads to loss of normal fluid homeostasis within the corneal stroma. If the injury to the endothelium is severe enough, the accumulation of extracellular fluid continues until pockets of fluid form within the corneal stroma, with hydrostatic forces pushing aqueous fluid into the subepithelial space. The coexistence of a fibrovascular response (pannus) also seems to play an important role, reinforcing the superficial wall, therefore allowing enlargment to such a degree as is seen in our case.

The response of these lesions to treatment is limited. Simple drainage procedures or aspirations have no permanent benefit. Excision of at least a portion of the anterior wall with chemical cautery seems to be successful in some cases,[4] but such treatments carry potential adverse visual consequences. An attempt to minimise these negative outcomes includes placement of lamellar grafts after excision of the anterior wall.[5] Penetrating keratoplasty may be required when the pseudocyst involves the visual axis.[6]

To our knowledge, this is the first report of recurrent corneal pseudocyst following chronic corneal oedema secondary to pseudophakic bullous keratopathy. In these persistent lesions, if there is underlying corneal disease that would benefit from penetrating keratoplasty, combining this with excision of the lesion may result in satisfactory clearance.

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Sir,

Endogenous endophthalmitis secondary to Lemierre's syndrome

Endogenous bacterial endophthalmitis is a rare condition with a prevalence of approximately 2–8% of all cases of endophthalmitis. The organisms commonly implicated are Streptococci, Staphylococci, *Escherichia coli*, *klebsiella*, *Bacillus cereus*, and *Listeria*. *Fusobacterium necrophorum* is a gram-negative anaerobic bacillus, which is a normal inhabitant of the human alimentary tract. The organism is an opportunistic pathogen, which causes a rare disease known as Lemierre's syndrome,[1] characterized by septic thrombosis of the internal jugular vein after oropharyngeal infection. *Fusobacterium necrophorum* has never been implicated as a cause of endogenous endophthalmitis. We report a first case of endogenous endophthalmitis in a patient with Lemierre's syndrome due to *Fusobacterium necrophorum*, which resolved without the need of intravitreal antibiotics.

Case report

A 53-year-old man was admitted in the chest medicine ward with a 5-day history of malaise, sore throat, and left-sided chest pain. On presentation, he was confused, febrile, hypoxic (oxygen saturation was 77% on air), and

had clinical and radiological (chest radiograph) features of bilateral lower-lobe pneumonia. After 3 days, his condition deteriorated and he was transferred to the intensive care unit for ventilatory support and drainage of the pleural fluid. A computerized tomogram (CT) scan of the head and neck with contrast revealed left internal jugular vein thrombosis (Figure 1). During the same time, it was noticed that the right eye was slightly injected. Blood culture grew *Fusobacterium necrophorum*, which was sensitive to penicillin and metronidazole and antibiotics were changed accordingly. He made good recovery and was transferred to the medical ward. However, he complained of decreased vision in the right eye, so urgent ophthalmic assessment was requested and bedside ophthalmic examination was performed. At that time, visual acuity was hand movement for the right eye and N/6 for near left eye. Right-eye examination showed anterior uveitis and few posterior synechiae, but no hypopyon. There was a big white fluffy mass in vitreous, which prevented any fundus examination. The left-eye examination was completely normal. A diagnosis of right endogenous endophthalmitis was made and he was started on topical steroids every 2 h and atropine 1% twice a day. Taking into consideration the patient's health, it was decided to perform intravitreal tap and antibiotics at bedside 2 days later. When the patient was reviewed 2 days later, his general health was better and his visual acuity in the right eye had improved to counting fingers 1 ft. This time fundus examination was possible, although hazy, it did not reveal any gross abnormality. Because of dramatic improvement, intravitreal antibiotic option was postponed. By 4 weeks, the visual acuity in the right eye had improved to 6/36. The anterior segment examination was normal. The vitreous of the right eye still showed fluffy opacity (Figure 2; right) but this had decreased considerably in size and intensity. The fundus view of the right eye was still hazy, but apart from a few haemorrhages, nothing abnormal was detected. There was no evidence of retinal or choroidal infiltrates. Ultrasonic B scan of the eye was quite normal. After 2 weeks, his visual acuity improved to 6/12, the vitreous opacity further shrunk, and the

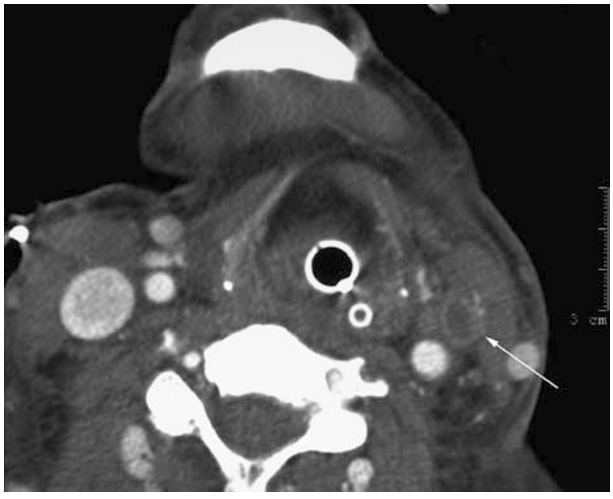

Figure 1 Axial view of CT scan with contrast, of neck showing left internal jugular vein thrombosis (white arrow).

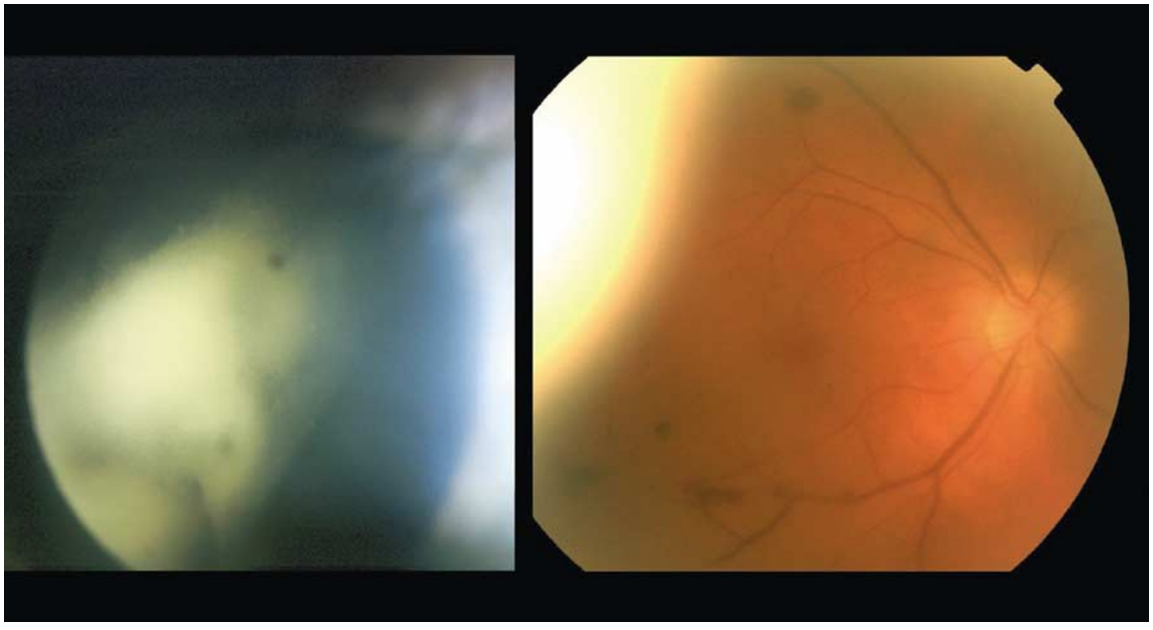

Figure 2 (Right) Anterior segment photograph of the right eye showing a white fluffy mass in the anterior vitreous. (Left) Fundus photograph of the same eye 2 weeks later.

fundus view was much clearer (Figure 2; left). At that time, all systemic antibiotics and topical steroids were stopped. At 3 months follow-up, his visual acuity remained 6/12 in the right eye, and the fundus examination was normal. There was slight vitreous debris but no active inflammation.

Comments

Lemierre's syndrome is a rare condition, characterized by thrombosis of the internal jugular vein that develops following oropharyngeal infection.[2] Sepsis and septic metastasis frequently ensue, and commonly affect the lungs, musculo-skeletal system, and on occasions liver. The classic Lemierre's syndrome is characterized by (i) primary infection in the oropharynx, (ii) septicaemia documented by at least one positive blood culture, (iii) clinical or radiographic evidence of internal jugular vein thrombosis, and (iv) at least one metastatic focus. Fusobacterium necrophorum is the most common pathogen isolated from the blood cultures of these patients.[1]

The metastatic septic emboli commonly involve lungs, liver, long bones and extremity joints, gluteal region, and sternum. There are cases of meningitis, cranial nerve palsy, and cranial vault involvement as well, but there is no case report of endophthalmitis in Lemierre's syndrome in the medical literature.

Our patient developed all the classic signs and symptoms of Lemierre's syndrome. He presented with oropharyngeal infection and empyema. Blood cultures were positive for *Fusobacterium necrophorum* and, during the course of disease, he developed internal jugular vein thrombosis and metastatic bacterial endophthalmitis. The patient probably developed endophthalmitis 1 week after the onset of disease. Four days after he was started on the antibiotics, to which the bacterium was sensitive, his ocular condition improved. He remained on systemic antibiotics for 8 weeks, and during that time, the endophthalmitis resolved.

Prompt administration of antibiotic therapy is key in the acute management of endogenous endophthalmitis. The condition is particularly responsive to intravenous antibiotics, while in exogenous endophthalmitis intravenous antibiotics are not deemed necessary.[3,4] Systemic antibiotics also treat the foci of infection at other sites and help prevent continued bacteraemia, thereby reducing possible infection of the other eye. Intravitreal antibiotic injections have revolutionized the treatment of exogenous endophthalmitis, but their usefulness in endogenous cases is unproven.[4–6] In endogenous bacterial endophthalmitis, the final visual outcome depends on the virulence of the organism, age of the patient, and underlying disease.[7] Enteric gram-negative rods, older age, and underlying factors like diabetes, renal failure, cancer, and immunocompromised states, are all associated with poor visual outcome. Final visual outcome is shown to be unrelated to the use of vitrectomy in the management of endogenous endophthalmitis.[7,8] Our patient was a middle-aged immunocompetent man with no underlying predisposing disease, who responded very well to intravenous treatment alone.

In summary, this is a first reported case of endogenous endophthalmitis, caused by Fusobacterium necrophorum in a patient with Lemierre's syndrome.

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