Metastatic endophthalmitis caused by *Clostridium perfringens*

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Abstract
A case of metastatic endophthalmitis due to *Clostridium perfringens* originating from the biliary tract is reported. The grave visual prognosis and the importance of early detection and treatment of the primary source of infection are emphasised.

Case report
A 65-year-old man presented with a 24-hour history of loss of vision in the left eye. The eye had become red and the lids swollen. During the preceding week, he had suffered severe colicky pain in the right hypochondrium. Past medical history was unremarkable.

On examination the patient was aphyreial and appeared well. His right eye was normal with a visual acuity of 6/5. His left eye had no light perception. The lids were swollen and indurated. There was complete ptosis and 5 mm of proptosis. All ocular movements were grossly restricted. The conjunctiva was congested with subconjunctival haemorrhage. The cornea was oedematous with severe Descemet's folds and many large keratic precipitates. The anterior chamber was shallower than in the fellow eye, with cells and flare. The pupil was fixed and dilated. There was no red reflex. The intraocular pressure was 50 mm Hg. He was commenced on intramuscular Magnapen 500 mg four times daily and gentamicin 80 mg three times daily. Oral acetazolamide 250 mg four times daily was given. Gentamicin and methicillin were injected in the subconjunctival space. Topical chloramphenicol hourly, atropine 1% twice daily, and betamethasone four times daily were started.

Investigations were as follows. The ESR was 70 mm/hour. A leucocytosis of 13·2×10⁹/L with 90% neutrophils was noted. Serum electrolytes and liver function tests were normal, as was the chest x-ray.

On the following day proptosis and corneal haze had increased. Details of the anterior chamber were obscured. Cultures from the conjunctiva, urine, and blood were sterile. Ultrasound examination (Figs 1 and 2), performed 24 hours after presentation, showed gross thickening of the choroid, with a large collection of subretinal fluid in the inferotemporal quadrant. Later in the day the globe perforated at the limbus. Evisceration was performed. The evisceration specimen grew a massive culture of *Cl perfringens*, sensitive to metronidazole, penicillin, ampicillin, and cloxacillin. The patient was treated with intravenous benzylpenicillin 600 mg four times daily.

Ultrasound examination of the abdomen confirmed an enlarged gall bladder. A laparotomy performed 9 days after presentation, revealed an acutely inflamed gall bladder. Within the bladder were multiple stones and debris, and the mucosal surface had several ulcers. The bile was sterile, probably due to the antibiotic treatment. The recovery from surgery was uncomplicated.

Discussion
*Cl perfringens* produces a particularly vicious suppurative panophthalmitis. In an extensive review of 56 cases Leavelle described the following signs and symptoms to be typical of gas gangrene panophthalmitis. Pain and rapid loss of vision within 12 hours following injury. Proptosis, hypopyon, ring abscess of the cornea, coffee-coloured discharge, gas bubbles in the anterior chamber, rise in intraocular pressure, immobilisation of the globe, and total loss of fundus reflex, all usually evident by 24 hours. All 56 cases followed penetrating injuries to the eye. In every case there was total loss of vision, and the eye was removed.

Since then two cases have been reported, where the eyes have been saved following anterior chamber infection by *Cl perfringens* following penetrating injury. The first responded to removal of foreign body and systemic antibiotics,
and the second underwent debridement of anterior chamber with vitrectomy instrumentation and systemic antibiotics. In both cases there was no definite evidence of vitreous involvement.

The patient in our report did not suffer a penetrating injury. All findings suggested a metastatic infection arising from the biliary tract. Despite this difference, the course of panophthalmitis was generally similar to that described by Leavelle.1

The only previously reported case of endogenous Cl perfringens panophthalmitis also arose from a biliary infection in a 68-year-old man,4 A gross haemorrhagic retinopathy was visible before the fundal view was lost. The loss of vision was rapid and complete. The gall bladder perforated despite surgical drainage and systemic antibiotic therapy with chloramphenicol, ampicillin, gentamicin, and methylprednisolone given parenterally. Death resulted from clostridial septicemia.

No form of treatment has ever been demonstrated to alter the devastating progression of Cl perfringens infection with vitreous involvement. Systemic and subconjunctival antibiotics have not succeeded.1 Intravitreal penicillin was shown to be beneficial in experimental infection in rabbits3 and should be considered in conjunction with early vitrectomy. The importance of a vitreous tap to aid in the diagnosis, as in any endophthalmitis, cannot be overemphasised. Clinical experience in the efficacy of vitrectomy in clostridial infections is non-existent because of the rarity of its occurrence and the fulminating course. However it should be a serious consideration if the eye can be salvaged. Endogenous endophthalmitis in comparison with exogenous endophthalmitis may be more difficult to salvage even with an early vitrectomy due to the vascular route of spread of Cl perfringens within the eye, and the attendant damage to the vascular and neural tissue of the choroid, retina, and optic nerve caused by its toxins.

Although the prognosis for the eye is poor early recognition of this condition is vital, so that the source of infection can be detected and treatment instituted urgently in this life threatening situation.