dysthyroid ophthalmopathy.[9] Muscle thickening due to myositis usually does involve the tendon, but the lack of pain is inconsistent with this diagnosis since the condition is usually of acute onset and painful.[8] Extraocular muscles involved by tumours usually have irregular borders or areas of focal thickening on CT.[9,10]

Tumours of the lung are the second most common cause of metastasis to the eye and orbit, the most common being breast.[10–12] In previous reports[1–5] the orbital masses appeared to involve other orbital tissues, but in our case there was discrete infiltration of the lateral rectus muscle, as visualised at surgery. Biopsy of the involved muscle under local anaesthesia was straightforward, and considered to carry less morbidity than liver biopsy. The 'smudged' appearance of the biopsy on histology (Fig. 2) is typical of oat cell carcinoma. Sputum cytology was confirmatory, although the chest radiograph was still considered normal on review.

Metastatic carcinoma to the orbit is probably more common than the literature would indicate,[7] although discrete metastasis to an extraocular muscle is rare. This is the first report of oat cell carcinoma of the bronchus associated with a discrete metastasis to an extraocular muscle.

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Sir,

Acute Haemorrhagic Conjunctivitis Epidemic in the Dominican Republic

Between 6 and 8 August 1992 we examined 39 patients with acute haemorrhagic conjunctivitis (AHC)[1] in Santo Domingo, San Cristobal and Bani, Dominican Republic. The initial symptom of the conjunctivitis, which usually occurred 12–24 hours after coming into contact with an affected person, was described as a foreign body sensation. Within a few hours the conjunctival blood vessels were dilated and the eye was noticeably red, swollen, and tearing profusely. Bilateral eye infections were common (61%) in patients seen 0–3 days after the onset of AHC, and most (67%) reported initial infection of the left eye. Petechiae to consolidated blotches of intra- and subconjunctival haemorrhage were observed in the superior temporal half of the bulbar conjunctiva in most (77%) of the patients (Fig. 1). Frequently, the patients reported conjunctivitis in other family members (86%). Of the patients examined, only one had rhinitis and a mild sore throat in association with the AHC. A patient had secondary ocular complications after applying soap to the affected eye. None of the patients presented with or reported neurological abnormalities.

The first cases of the AHC epidemic were detected in the central region of the Dominican Republic at Bonao in the middle of June 1992. Subsequently patients with conjunctivitis were seen from Puerto Plata in the north to El Limonar and Santo Domingo in the south. Hundreds of patients with AHC were seen at the rural clinic of Dr. Elias Santana in Los Al Carrizos and in prisons north of Santo Domingo during July.

The outbreaks of haemorrhagic conjunctivitis in Panama in May, the explosive epidemic in the Dominican Republic in June and the similarity in signs and symptoms of the conjunctivitis in patients in each epidemic suggests that the AHC may have been transported from Panama. AHC is caused either by enterovirus type 70 (EV70) or a variant of coxsackievirus type A24 (CA24v). The eye disease appeared to be similar to AHC caused by EV70. This clinical impression is based upon the absence of upper respiratory tract symptoms that are usually observed in patients with AHC caused by CA24v. Further, our difficulty in isolating virus from tears is consistent with pre-

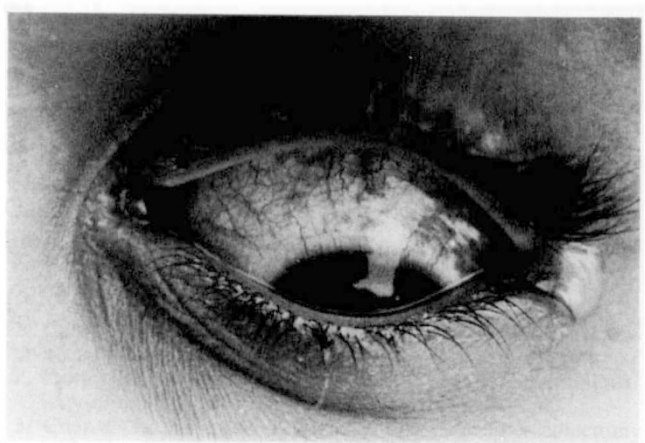

Fig. 1. *The left eye of a patient showing signs of AHC about 24 hours after infection. Note the dilation of the bulbar conjunctival blood vessels, the petechial haemorrhages associated with the blood vessels in the bulbar conjunctiva and the blotch of haemorrhage in the temporal quadrant of the superior bulbar conjunctiva. Note also the abundant tear fluid and mucus discharge.*

vious experience with EV70. Thus, EV70 is probably the aetiological agent of the 1992 AHC epidemic in the Dominican Republic. EV70 caused the AHC epidemics in 1981,[2] while CA24v caused the epidemics of AHC in Central America in 1987.[3] Blood for serological studies is being collected.

Because of the potential for spread of EV70 and CA24v into other countries, primary care physicians should be alert to the growing prevalence of AHC caused by EV70 or CA24v and paralytic disease caused by EV70.[4,5] While there is no recommended treatment for AHC, cold compresses may be of benefit in alleviating pain and swelling.[3] AHC is usually benign and patients recover in 7–14 days without sequelae. The use of home remedies and sharing of eye medications should be discouraged to prevent complications and spread. Rarely (1 in 10 000 cases) EV70 causes paralysis that involves the extremities and/or face as late as 2 months after AHC.[4] Paralytic disease due to EV70 infection of the central nervous system can occur in the absence of AHC.[5] Paralysis resolves gradually, usually without permanent disability.

The reservoir of AHC viruses between epidemics is not known, but virus is detectable in tears collected from 6 hours prior to symptoms up to 4 days after the onset of symptoms of AHC.[6,7] The viruses are spread by hand-to-eye contact. AHC viruses retain infectivity on cool, moist tear-contaminated materials. Contaminated hands and materials can be disinfected with detergent and 1% chloride solution (5000 ppm). Thus, patients and physicians should use good hygiene and sanitation to prevent transmission.

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