Eye (1990) **4,** 861–864

Letters to the Journal

Sir,

Meningococcal Conjunctivitis

There are a few reported cases of primary meningococcal conjunctivitis. Most cases of meningococcal conjunctivitis occur as part of an endogenous systemic infection with *Neisseria meningitidis,* usually during, or occasionally preceding, meningococcal meningitis.[1] Most of the reported cases of primary meningococcal conjunctivitis occurred in the first half of this century.[2,3,4,5] There have been sporadic cases reported since then, the most recent in the U.K being in a five day old child in 1972.[6]

Diagnosis of a primary meningococcal conjunctivitis is important, as not only may local and ocular complications develop, such as a cellulitis[4] or corneal ulceration[1,5] but systemic infection may ensue in 10% of cases.[4,7] Conversely ocular involvement has been reported in 0.9% of cases of systemic meningococcal infection.[8] Identical meningococci may be found among family members and other contacts of the index case.[4,9]

We report on a case of a meningococcal conjunctivitis occurring in an eight year old girl and of the isolation of a separate strain of *N. meningitidis* from the nasopharynx of her father.

Case Report

An eight year old girl presented with a three day history of a unilateral mucopurulent conjunctivitis. Examination of the right eye revealed a hyperaemic conjunctiva with a mixed papillary and follicular reaction. A coarse punctate epithelial keratopathy was present together with a mild flare in the anterior chamber and an enlarged preauricular lymph node. Visual acuity was 6/36 right and 6/6 left. No other ocular abnormality was present. The left eye was normal. Systemic examination showed no abnormality. There was no history of contact with an individual suffering from an eye infection or febrile illness. Treatment was commenced with topical chloramphenicol and acyclovir. Conjunctival swabs taken for herpes simplex virus (HSV), adenovirus (AV) and chlamydia were negative. *N. meningitidis* type C 14 P1.9 was isolated from the right conjunctiva. A posterior pharyngeal swab was clear. The patient was placed on oral rifampicin in addition to the topical chloramphenicol. The acyclovir was stopped. The conjunctivitis cleared during the following week and subsequent posterior pharyngeal and conjunctival swabs were clear. Two weeks following presentation the corneal keratopathy had resolved and the visual acuity was 6/6 either eye. Viral serology showed no rise in titres to AV, HSV or the enteroviral group. Counterimmuno-electrophoresis indicated a rise (doubling) in antibody titre to *N. meningitidis* group C.

A *N. meningitidis* type B.1 was isolated from a posterior pharyngeal swab of the patient's father, but not from any other member of the family. All members of the family were placed on rifampicin.

Comment

Meningococcal conjunctivitis is described as an acute bilateral purulent conjunctivitis[1] usually with marked lid oedema.[4,5,7]

Multiple punctate corneal epithelial erosions and yellowish maculae may occur in the superficial stroma with the occasional development of a corneal ulcer.[1] A coarse epithelial keratopathy may occur in various viral infections. The absence however, of a viral isolation from the conjunctiva with no rise in titres to HSV, adenovirus and the enteroviruses makes an associated viral infection unlikely, and reaffirms that in this case, *N. meningitidis* was the cause of the conjunctivitis. Exclusion of a viral aetiology is important as the isolation a *N. meningitidis* from a mild mucoid conjunctivitis has been ascribed to represent a carrier state.[9]

The contacts of individuals with meningo-

coccal disease have up to an 800 times greater risk of developing meningococcal disease, such as septicaemia, than those of the general population.[10] The systemic treatment of a person with a meningococcal conjunctivitis and the screening and treatment of their contacts with prophylactic antimicrobials is thus important. This case reaffirms the necessary practise of microbiologically investigating patients with conjunctivitis.

S. B. Kaye
E. M. Molyneux
D. Zala
C. A. Hart
Department of Medical Microbiology,
University of Liverpool,
Duncan Building,
Royal Liverpool Hospital,
Liverpool L69 3BX and
The Royal Liverpool Children's Hospital,
Eaton Road,
Liverpool L12 2AP.

References

[1] Duke-Elder S. Diseases of the Outer Eye: In: System of Ophthalmology. Ed. Duke-Elder S. London: Kimpton 1965, **8:** 174–5.
[2] Thygeson P: Primary meningococcal conjunctivitis treated by sulfadiazine. *Am J Ophthalmol* 1944, **27:** 400–1.
[3] Odegaard K: Conjunctivitis purulenta with keratitis caused by Neisseria intracellularis (meningococcus) *Acta Ophthalmol* 1943, **21:** 295–302.
[4] Newton DA and Wilson WG: Primary meningococcal conjunctivitis. *Pediatrics* 1977, **60:** 104–6.
[5] Stuart RD and McWalter D: Primary meningococcal conjunctivitis in children. *Lancet* 1948, **1:** 246.
[6] Hansman D: Neonatal meningococcal conjunctivitis. *Br Med J* 1972, **1:** 748.
[7] Odegaard A: Primary meningococcal conjunctivitis followed by meninigitis and septicaemia. *NIPH ANNALS* 1983, **6:** 55–7.
[8] Williams DN and Geddes AM: Meningococcal meningitis complicated by pericarditis, panophthalmitis and arthritis. *Br Med J* 1970, **2:** 93.
[9] Brook I, Bateman JB, Pettit TH: Meningococcal conjunctivitis. *Arch Ophthalmol* 1979, **97:** 890–1.
[10] The Meningococcal disease surveillance group: Analysis of endemic meningococcal disease by serogroup and evaluation of chemoprophylaxis *J Infect Dis* 1976; **134:** 201.

Sir

Retinal Signs in Sickle Cell Anaemia

Patients with sickle cell anaemia may have retinal abnormalities ranging in severity from simple arteriolar occlusion to proliferative sickle cell retinopathy (PSR) with visual loss.[1,2] To date, studies examining haematological factors in relation to the severity of sickle cell retinopathy have yielded negative or conflicting results.[3,4] Goldberg[5] has suggested that intravascular red blood cell sickling and increased blood viscosity may be the cause of this retinal vascular occlusion. However, it might then be expected that such sickling would occur at that point in the circulation where oxygen is the lowest, i.e. on the venular end of the capillary or in the venule itself.

It is now known that erythrocytes of sickle cell patients contain varying amounts of polymerised sickle haemoglobin (Hbs) at arteriolar saturation values.[6] The tendency of HbS to polymerise is determined by the intracellular haemoglobin concentration and composition.[6] The resulting heterogeneity in red cell haemoglobin concentration can be assessed by using the calibrated phthalate ester technique.[7] The middle 60% density range (R60 values) serves as an indicator of the heterogeneity of the density of the red cells. We used the phthalate ester technique and red cell indices to examine whether HbS is the primary determinant of the retinal lesions found in sickle cell patients.

We studied a consecutive series of 30 patients, nine women and 21 men, with stable sickle cell anaemia admitted for routine follow-up to the National Institutes of Health. Their mean age was 30.0 + 8.1 years. The diagnosis of sickle cell anaemia was made on the basis of red cell haemoglobin electrophoresis, DNA analysis of bone marrow aspirates and peripheral blood examination. No patient was included who had a sickle cell crisis during either the month before or after the study, who had received a blood transfusion in the four months before the study or who was taking long term medication other than folic acid.

A detailed retinal examination was performed using Goldmann 3 mirror contact lens, and recorded on a fundus drawing. Fundus photographs were obtained in 29 of the 30 patients and fluorescein angiography (FA) in 19 patients. Severity of the sickle cell retinopathy was graded using Goldberg's classification.[1]