An Outbreak of Shipyard Eye in a Car Factory—A Diagnostic Dilemma

D. S. CHATTERJEE

Senior Medical Officer, Ford Motor Company, Basildon, Essex

Summary

Nineteen cases of acute conjunctivitis occurring in a factory between June and July 1981 are described. The clinical features of these cases were distinguishable from those observed in typical keratoconjunctivitis. No virus or other pathogenic organism was isolated from conjunctivae and the serology was negative during the acute phase of the illness. The diagnosis was confirmed only from the presence of adenovirus complement fixing antibodies in the convalescent sera.

Introduction

In spite of its popular name as 'Shipyard Eye', the epidemic Keratoconjunctivitis (EKC) has been known to occur in industrial concerns other than shipworks (Anonymous, 1972; Sprague et al., 1973; Anonymous, 1975).

The disease was first described by Fuchs (1889) and later the name 'EKC' was coined by Hogan and Crawford (1942). In its typical form, the infection is caused by adenovirus type 8 (Jawetz, 1959), less commonly by the types 19 and 10 (Darougar et al., 1977; Tullo and Higgins, 1979) and sometimes in sporadic or mild cases by other types such as adenovirus 3 and 7 (Tullo, 1981).

The present article aims to describe an outbreak of acute conjunctivitis in which the observed clinical and laboratory findings were atypical and presented difficulties in reaching an accurate diagnosis.

Materials and methods

Between June and July 1981, thirty-four cases of acute conjunctivitis were reported at the Occupational Health Centre of a car factory. Of these, nineteen patients with ages ranging from 19 to 61 years and a mean age of 37.8 (s.d. =13) years, were available for detailed observations. Immediately after the outbreak was recognized, strict hygiene measures were adopted throughout the Plant, especially in the treatment rooms, canteens, toilets and other amenity areas. The Area Community Health Department was alerted and the local general practitioners were fully informed about the outbreak. An epidemiological search was conducted with the assistance of the Communicable Disease Surveillance Centre at London. The search included tracing of patients' movements within the work place during that period as well as their interpersonal contacts outside the work place and through travel abroad. In addition each patient was asked about the possible occupational exposure to any chemical and physical irritants including foreign bodies, history of wearing contact lenses and taking medication such as Practolol (eraldin) which is known to cause conjunctivitis. The eyes were examined under slit lamp following fluorescein staining and once the clinical diagnosis was made the patients were isolated from work until they were symptom free.

Initially 11 patients within 1 to 8 days of the onset of their illness were referred to the local hospital for microbiological examinations of the conjunctivae and serology. For three of these subjects serological examinations were repeated after 3 weeks. Subsequently, one patient from this group and six others were bled 4 months after the onset of the disease and the convalescent sera were examined at the Public Health Laboratory Services at Bristol for adenovirus antibodies.

Results

The results are based on the analysis of the findings on 19 patients who volunteered for epidemiological, clinical and laboratory investigations.

Epidemiology

The duration of the outbreak extended from 23 June to 17 July and its peak was reached between 30 June and 3 July 1981 (Fig. 1). During the early onset of the disease a janitor, a nurse and a canteen assistant were affected. Apart from these no evidence of any common source for infection was identified.

Clinical Features (Table I)

The predominant symptoms as experienced by the 14 to 15 (73 to 75 per cent) patients were intense pain in eyes,
severe photophobia, blurred vision and profuse lacrimation. In addition, nine (47 per cent) patients had foreign body sensation in eyes and four (21 per cent) complained of frontal headaches. With the exception of three patients with uniconical lesions, the rest (84 per cent) had bilateral conjunctivitis with follicular changes limited to tarsal conjunctive. Among the associated features 16 (84 per cent) patients presented themselves with periorbital oedema and chemosis but no corneal lesion was detected in any one of the subjects examined. The duration of the illness, as defined by the time to return to work by each person, varied from 1 to 3 weeks with a mean of 2-5 weeks.

Microbiology
The conjunctival swabs taken from 11 patients during the acute phase of the illness showed no evidence of pathogenic virus, bacteria or chlamydia; three patients had only a scanty growth of diphtheroid bacilli.

Serology
Blood samples at the early phase of the disease including those taken 3 weeks after the onset showed no evidence of adenovirus antibodies. Subsequent serological tests on seven patients at the convalescent phase showed that complement fixation (CF) adenovirus antibodies were present at a dilution of 1/12 in one patient, 1/4 in three and at 1/32 in two patients. None of the subjects, however, showed any evidence of haemagglutination inhibiting antibodies (HAI) to adenovirus type 8 in the early as well as in the convalescent sera.

Discussion
The results showed that isolation of the patients and aseptic measures taken during the acute phase were the most important steps in controlling the outbreak. Epidemiological search failed to identify any common source of infection, but the involvement of a nurse, a janitor and a canteen assistant was at least partly responsible for the rapid transmission of the disease at the early stage. Similar fortuitous spread has been reported by Dawson and Darrell (1963) during a similar outbreak in the United States. The period of the outbreak between June and July seemed also to be consistent with the findings in other studies (Grist et al., 1971; Barnard et al., 1973).

Clinically, the absence of any conical lesion which is regarded as the hallmark of EKC made the initial assessment very difficult, especially when the microbiology and serology were both negative. Subsequently the diagnosis was confirmed from the presence of adenovirus antibodies in the convalescent sera only.

Although it can be agreed that the levels of antibodies present were too low to reach such a definite conclusion the diagnosis of adenovirus conjunctivitis could be justified for the following reasons.

Firstly, it is usual for virus CF antibodies to fall quite rapidly after acute onset of the disease, and hence it was not uncommon to find such low levels of titre after 4 months.

Secondly, it would be unlikely to find adenovirus antibodies in six out of seven ordinary adults without a virus infection. Lastly, during that period sporadic outbreaks of similar cases were evident in the local community, and in one particular case it was possible to isolate adenovirus from the conjunctiva and from the stool (Dowsett, personal communication).

In conclusion, the study showed that the outbreak of conjunctivitis was due to some form of adenovirus infection, but not from the type 8, as the related HA1 antibodies could not be detected. In addition, it highlights that when the clinical and microbiological evidence is not conclusive, it seems essential that both early as well as convalescent sera are examined to establish a correct diagnosis.

Acknowledgements
I am indebted to Dr E. C. Dowsett, Consultant Microbiologist from Basildon District General Hospital; Dr S. K. R. Clarke, Consultant Virologist from Public Health Laboratories, Bristol; Dr Susan Hall from Communicable Disease Surveillance Centre, London; Dr S. E. Brill for approving this article for publication and Mrs P. Tucker for typing the manuscript.

REFERENCES


Requests for reprints should be addressed to: Dr D. S. Chatterjee, Ford Motor Company, Research & Engineering Centre, Laindon, Basildon, Essex SS15 6EE