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Epidemic keratoconjunctivitis in the West of Scotland, 1967–72

By D. REID
Communicable Diseases (Scotland) Unit, Ruchill Hospital, Glasgow G20 9NB
ELEANOR J. BELL
Regional Virus Laboratory, Ruchill Hospital, Glasgow G20 9NB
N. R. GRIST and J. C. TAYLOR*
University Department of Infectious Diseases, Ruchill Hospital, Glasgow G20 9NB
and JEAN R. ELLIS
Glasgow Eye Infirmary

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SUMMARY

Outbreaks of epidemic keratoconjunctivitis have occurred among workers in shipyards and other industrial concerns in the West of Scotland in 1956, 1967 and 1971–72. In the most recent episode 220 persons were known to be affected and those mainly involved were shipyard personnel working on the open decks of ships under construction; only a few non-industrial workers were affected. As in previous outbreaks adenovirus type 8 was shown to be the causal organism. It is likely that spread of the virus was probably facilitated in some of the patients by such procedures as first aid measures to remove foreign bodies from the eye. A survey of the family contacts of those affected in 1971–72 revealed that only 2 % were secondarily infected. This was probably due to propaganda measures to discourage the use of communal face towels, etc.

Despite close virological surveillance over a period of 6 years of patients attending opthalmic clinics in the West of Scotland, there is as yet no clue to the whereabouts of adenovirus type 8 during interepidemic periods. It is suggested that travellers might be responsible for the introduction of the infection into an area.

INTRODUCTION

Keratoconjunctivitis, a disease characterized by unilateral or bilateral inflammation of the conjunctivae, and oedema of the lids and periorbital tissues, and its frequent presentation in an epidemic form, was first described in Austria in 1889 (Fuchs, 1889). Since then many reports of large outbreaks have appeared in the medical literature indicating that the disease has a world-wide distribution

* Present address: St Thomas's Hospital, London.

(Jawetz, 1959). That the disease can cause considerable morbidity was illustrated in 1941 when more than 10,000 cases occurred in Hawaii, at the Naval shipyards of Pearl Harbour and, because of this frequent occurrence of the disease among workers in shipyards, the term 'shipyard eye' was coined. In addition to the descriptions of outbreaks of the disease in shipyards and industrial concerns, several episodes of infection within eye hospitals and clinics have also been described (Wegman, Guinee & Millian, 1970; Barnard, Dean Hart, Marmion & Clarke, 1973). Contaminated instruments and the fingers of medical attendants have often been implicated in such reports.

Keratoconjunctivitis is often a painful and distressing condition causing photophobia, conjunctivitis with severe lacrimation, and discharge. Large follicles may develop on the conjunctiva and corneal opacities may form causing impaired visual acuity often for long periods of time. Although a viral aetiology was suggested in 1930 (Wright, 1930), it was not until 1955 that adenovirus type 8, the most common aetiological agent, was first isolated from a patient (Jawetz *et al.* 1955).

Several outbreaks of epidemic keratoconjunctivitis have occurred in the United Kingdom and at least three – in 1956, 1967 and 1971 – have involved workers in the shipyards and heavy industrial complexes situated in Glasgow and the Clyde Valley (Sommerville, 1958; Taylor, 1967 and Grist, Reid, Bell & Ellis, 1971). The latest outbreak in 1971 presaged three other outbreaks in Britain during the same year – in Bristol in July (Barnard *et al.* 1973), in London and in the Midlands of England in September (Public Health Laboratory Service, 1972).

The activity of adenovirus type 8 in the West of Scotland has been monitored since 1967 during both the epidemic and interepidemic periods to try and study the epidemiological features and reservoir of infection of keratoconjunctivitis. The epidemiological, clinical, and virological findings during this period are reviewed in this paper.

EPIDEMIOLOGICAL FEATURES

Investigation of the outbreaks in 1967 and 1971 was stimulated by reports of cases of keratoconjunctivitis to the Regional Virus Laboratory, Ruchill Hospital, Glasgow, by ophthalmologists and industrial medical officers. Serological tests rapidly established that a member of the adenovirus group was responsible and subsequently type 8 strains were isolated (Bell, Martin & Ross, 1969; Bell, Sneddon & Ellis, 1972).

Outbreaks

The outbreak in 1967 started in August and lasted until December with a peak in September. During that time 382 cases of conjunctivitis were reported from shipyards, engineering and steel works in Glasgow, Clydebank and Motherwell. In one shipyard with 3700 employees 2·2 % were affected and in another 4·7 % of 2390 employees were involved. Platers, caulkers, drillers, welders and labourers accounted for most of the patients from the shipyards.

Several examples of secondary spread of infection were observed including six virologically confirmed patients with no previous ophthalmic condition or

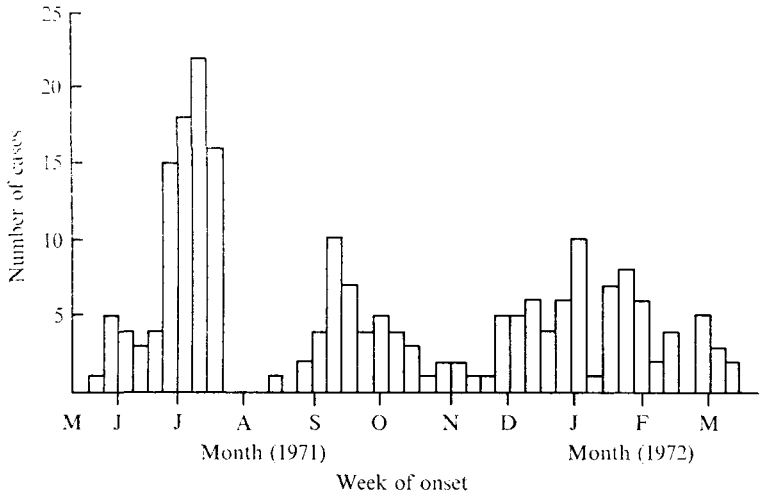

Fig. 1. Number of cases of keratoconjunctivitis occurring in the West of Scotland, May 1971 to March 1972.

industrial contact. They were domestic contacts, within their five families, of hospital-acquired infection in three instances; infection was apparently introduced into the other two families by contact of children with affected playmates. Infection spread to involve all members of two households (two children, both parents and a visiting grandmother in one family; a baby, both parents and a visiting grandmother in the other).

After the cessation of the outbreak in December 1967 very few cases of keratoconjunctivitis occurred until May 1971 when an increasing number of patients with the condition began to be seen at the Eye Infirmary in Glasgow. At first most of the patients came from one of the shipyards in Glasgow which had been involved in the 1967 outbreak and it was only later that workers in other shipyards and industrial concerns in the West of Scotland became infected. The peak of this outbreak was reached at the beginning of July (Fig. 1) The annual vacation taken by industrial concerns in the Glasgow area during the last two weeks of July and first week of August brought a temporary cessation to the number of new cases but, disappointingly, there was a resurgence of infection after the return to work and clinical cases continued to occur until March, 1972.

To try and determine the number of persons involved in the 1971–72 outbreak and collect other epidemiological information, the staffs of ophthalmic clinics and industrial ambulance rooms in the area were asked to collect surveillance data on a standard form which was issued to them. Two hundred and twenty persons presenting with the clinical features of keratoconjunctivitis were recorded.

Of these patients 107 (49%) worked in shipyards (Table 1). The shipyard workers were mostly platers (22%), welders (19%), caulkers (9%), fitters (9%), engineers (7%), electricians (6%), sheet iron workers (6%) and drillers (5%). Among the 113 patients (51%) who did not have a connexion with shipyards, engineers formed the largest group (19% of those affected) followed by clerical workers (8%), housewives (8%) and nurses (6%). Although none of the nurses

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Table 1. *Eye infection according to occupation* (1971–72)

Occupation	Number of cases
Shipyard worker	107
Engineer	22
Clerical worker	9
Housewife	9
Nurse	7
Building worker	6
Retail worker	5
School child	5
Other	50
Total	220

Table 2. *Secondary infections in household*

Age group	Affected	Unaffected
< 15 years	4	216
$\geqslant 15$ years	10	368

were employed in shipyards, two worked at clinics in industrial concerns; the remaining five came from local hospitals.

A definite history of injury was obtained from 50 (23 %) of those affected. The remainder presented at the ambulance room or clinic with the features of keratoconjunctivitis as their initial complaint.

Because secondary spread of infection to family contacts was noted in the 1967 outbreak an effort was made in 1971 to gauge the extent of domestic spread. Of 598 family contacts of the 220 patients surveyed, 14 (2 %) were secondarily infected (Table 2). Four of these were under 15 years of age and 10 were older contacts.

Long-term surveillance

An attempt has been made since the 1967 outbreak in Clydeside to accumulate data during the interepidemic period to determine whether the virus smoulders inconspicuously among industrial populations, or among patients attending ophthalmic clinics or whether, as in Japan (Mitsui *et al.* 1955), the reservoir of infection is in children with mild upper respiratory symptoms. To try and solve this problem, virological surveillance of a sample of patients attending ophthalmic clinics with conjunctivitis and keratoconjunctivitis has been undertaken during the past 6 years. From the 957 conjunctival scrapings examined, 203 adenoviruses were isolated, 128 of these being adenovirus type 8 (Table 3). However, in the interepidemic years of 1968, 1969 and 1970, adenovirus type 8 was isolated on only five occasions, all in 1968, and then only from members of a typical industrial group (Grist, Bell & Gardner, 1970).

Table 3. *Virological surveillance of cases of conjunctivitis and keratoconjunctivitis*

	Adenovirus types									
Year	1	2	3	4	7	8	9	10	Not known	Total examined
1967	—	—	11	—	4	57	—	2	1	187
1968	—	—	4	—	3	5	1	—	—	88
1969	—	—	—	—	—	—	—	—	—	35
1970	—	—	6	5	—	—	—	—	—	105
1971	2	1	8	—	8	66	—	1	—	385
1972	3	—	5	1	3	—	2	1	3	157
Total	5	1	34	6	18	128	3	4	4	957

CLINICAL FEATURES

The onset of the illness was acute and heralded by symptoms of irritation often described as a feeling of 'something in the eye', marked lacrimation and photophobia. This was followed by the rapid development of lid oedema, conjunctival infection, chemosis and often a watery discharge. Prominent follicles of the palpebral conjunctiva of both upper and lower lids were usually apparent at the first visit to the clinic (mostly within 4 days of the onset of symptoms). Preauricular adenitis was found in approximately half the cases. In the first week the cornea usually showed a superficial punctate keratitis which developed into very characteristic sub-epithelial infiltrations usually in the second week. There were very few virologically proved cases in which corneal complications did not appear. Follicles persisted for weeks and often months after infection. The sub-epithelial infiltrations were the last signs of keratoconjunctivitis to disappear and in several instances were still present after 18 months. Visual acuity was depressed during the course of the infection but in most cases recovered to the level of vision present before infection, the exceptions being severe cases in which sub-epithelial infiltrations were numerous and persistent.

VIROLOGICAL FINDINGS

Conjunctival scrapings, collected in virus transport medium (Grist, Ross, Bell & Stott, 1966) were examined using the techniques described by Bell and her colleagues (1969). Paired sera were examined by complement fixation and haem-agglutination-inhibition tests.

Adenovirus type 8 was the predominant organism in both of the outbreaks (Table 3). During 1967 laboratory evidence of adenovirus type 8 was obtained from 57 (31 %), adenovirus type 3 from 11 (6 %) and adenovirus type 7 from 4 (2 %) of the 187 patients examined. During 1971 evidence of adenovirus type 8 was obtained from 66 (17 %), adenovirus type 7 from 8 (2 %) and adenovirus type 3 from 8 (2 %) of the 385 patients examined.

DISCUSSION

The occurrence of epidemic keratoconjunctivitis in the West of Scotland in 1967 and 1971–72 together with the experience in other parts of the United Kingdom during 1971 illustrates the two principal modes of spread of this potentially preventible disease. The outbreaks in Bristol (Barnard *et al.* 1973), London and the Midlands of England (Public Health Laboratory Service, 1972), were all associated with infection within hospitals and related to ophthalmic investigations and treatment. On the other hand, the outbreaks in Glasgow and the West of Scotland occurred mainly in shipyards and industrial concerns and were not primarily related to the passage of the virus from patient to patient as a side effect of medical techniques. Spread of the disease in both outbreaks mostly occurred outside the hospital or shipyard ambulance room as the majority (77 % in the 1971–72 outbreak) of the patients presented themselves for treatment with the condition and had not previously been to a clinic with another eye disorder. Thus it is likely that spread of the virus within a shipyard is probably due to auto-inoculation or amateur first aid procedures to remove foreign bodies. This is supported by the finding that amongst the shipyard workers the majority of those affected were employed as platers, caulkers and welders, i.e. those workers most likely to receive foreign bodies or flashburns in their eyes. That fingers are an important means of transmission of the virus is also suggested by the fact that ambulance room attendants were affected in the 1967 outbreak and nurses in the 1971–72 outbreak. Infection in ophthalmologists also has been reported by Dawson & Darrell (1963).

In the 1967 outbreak attention was drawn to the spread of the disease within families. This was also noted in the Bristol outbreak in 1971 (Barnard *et al.* 1973). Particular attention was paid to this aspect of the disease during the 1971–72 episode and it was estimated that 2 % of the family contacts developed eye symptoms. It is likely that strong propaganda measures (e.g. the use of separate face towels, etc.) which were instituted at the start of this outbreak had a beneficial effect in limiting the extent of family spread.

A feature of eye infections by adenovirus type 8 is the explosive nature of the epidemics which are, in turn, interspersed with prolonged periods of almost complete absence of the virus. Despite close virological surveillance, over a period of 6 years, of patients attending ophthalmic clinics in Glasgow and the West of Scotland, there is as yet no clue to the whereabouts of adenovirus type 8 during the interepidemic periods, and failure to detect this virus during investigations of acute respiratory illnesses of Glasgow children extending over many years does not suggest a local reservoir of infection in this age group as in Japan (Mitsui *et al.* 1955). However, the fact that outbreaks have occurred in seaports such as Bristol, Glasgow, Copenhagen (Mordhorst & Kjer, 1961) and Singapore (Yin-Coggrave & Loh, 1966) suggests the possibility of the virus being periodically imported from other areas or countries, for example, by seamen, transport drivers, or other travellers.

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