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An outbreak of epidemic keratoconjunctivitis in a regional ophthalmology clinic in New South Wales

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SUMMARY

The objective of the study was to identify the extent and cause of an outbreak of epidemic keratoconjunctivitis (EKC). The study design was active case finding and a case-control study of clinic patients who developed symptoms of EKC between 31 December 2005 and 31 March 2006. The main outcome measures were clinical procedures carried out and clinicians seen during clinic visit. Significantly more cases than controls had tonometry with instillation of anaesthetic drops (OR 16·5, 95% CI 3·9–145·1, $P<0·01$), optical coherence tomography (OR 4·7, 95% CI 1·2–21·9, $P=0·01$), or instillation of dilating drops by an orthoptist (OR 2·3, 95% CI 1·1–4·7, $P=0·01$). Significantly more cases than controls were seen by one orthoptist (OR 21·8, 95% CI 8·2–60·0, $P<0·01$). Transmission of EKC within the clinic was probably due to contamination of either or both the anaesthetic drops and the tonometer head in the room used by an orthoptist. A comprehensive suite of strategies is required to prevent healthcare-associated EKC.

INTRODUCTION

Epidemic keratoconjunctivitis (EKC) is an acute viral disease of the eye and is typically caused by adenovirus types 8, 19 and 37 [1]. The reservoir for infection is humans, and it is transmitted through direct contact with eye secretions of an infected person or indirectly via contact with contaminated surfaces, instruments or solutions [1]. Infection causes unilateral or bilateral inflammation of conjunctivae, and oedema of the eyelids and periorbital tissue. Symptoms include redness, pain, watery ocular discharge, photophobia, foreign-body sensation, blurred vision and occasionally low-grade fever, headache, malaise and lymphadenopathy [2–4]. Sub-epithelial corneal infiltrates can develop, may persist for up to 2 years [5] and can result in permanent scarring [1]. The incubation period is between 4 and 10 days, and the period of

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communicability is from day of onset of symptoms to 12 days following [6]. Outbreaks caused by several adenovirus types have been associated with a variety of eye-care institutions in several countries [2, 5, 7–9].

On 14 March 2006, a private ophthalmology clinic in regional New South Wales (NSW) (the clinic) reported to the local public health unit that 18 patients had presented to the clinic with a provisional diagnosis of viral EKC on the previous day. All patients had visited the clinic in the preceding 3 weeks.

The clinic was the sole ophthalmology practice in the region and served a population of about 170 000 people including a major urban centre of 47 000 people and surrounding rural areas with a population of 88 000 people [10]. In NSW, eight public health units across the state are responsible for disease surveillance and control.

We undertook an epidemiological investigation to identify the extent of the apparent outbreak and the risk factors associated with EKC transmission within the clinic.

METHODS

Case finding

We defined a probable case as a person in the local region who developed symptoms of EKC (i.e. foreign-body sensation, watery ocular discharge, redness and photophobia) between 31 December 2005 and 31 March 2006. Cases were classified as confirmed if a medical practitioner made the diagnosis, and/or there was laboratory confirmation of adenovirus.

To identify cases of EKC between 31 December 2005 and 31 March 2006, we:

(1) Faxed and phoned general practitioners in the local region.
(2) Reviewed local hospital emergency department records against International Classification of Diseases, ninth edition, codes 327 and 077 (acute conjunctivitis and other diseases of conjunctiva due to viruses and chlamydiae).
(3) Asked the principal ophthalmologist at the clinic to identify cases among patients by reviewing patient records.
(4) Interviewed a random sample of patients attending the clinic on each day where staff identified that at least one case had attended the clinic.
(5) Asked cases whether other household members had symptoms of EKC.

Using a standardized questionnaire, we interviewed all probable cases by telephone. The questionnaire collected information on patient demographics, clinical characteristics of the illness, contact with other cases and time spent in the clinic waiting room. Information on the main ocular conditions, date and time of visits after 31 December and prior to symptom onset, procedures undergone during the visit, and the orthoptist and ophthalmologist who treated the patient was extracted from patients' records by clinic staff.

To examine risks associated with illness at the clinic we undertook a case-control study of clinic patients. We included all clinic-associated cases and a sample of clinic patients who did not have EKC in the case-control study.

Case-control study

Cases that developed symptoms of EKC after a visit to the clinic were defined as clinic-associated cases and were enrolled into the case-control study. To enrol controls in the study we generated random lists of patients attending on each day of a case and randomly selected up to three potential controls for each case attending on that day. A maximum of five attempts were made to contact cases and controls for interview between 08:00 and 20:00 hours. Where people selected as controls reported symptoms consistent with EKC (i.e. foreign-body sensation, watery ocular discharge, redness and photophobia) they were included as a case and the next control on the random list of potential controls was selected. The case questionnaire (omitting questions related to onset and duration of symptoms) was administered to controls.

Environmental investigation

Staff at the clinic included five ophthalmologists (designated ophthalmologists 1–5), three orthoptists (designated orthoptists 1–3), nurses and support personnel. We interviewed clinic staff and assessed clinic premises to identify the range of procedures carried out, infection control practices, and their clinical context.

Laboratory investigation

Case confirmation

Conjunctival cells were removed from swabs collected from probable cases using viral transport

swabs (Copan Italia, Brescia, Italy) and transported to the laboratory at 4° C, and placed on glass slides before fixation with cold acetone. Adenovirus-infected cells were then detected using indirect immunofluorescence (IF) using a monoclonal antibody specific for the hexon protein common to all adenovirus serotypes (Chemicon Pty Ltd, Temecula, CA, USA, no. MAB8051). Where the specimen quality was adequate, cells were placed in tissue culture with HELF (human embryonic lung fibroblast) and HEp-2 (human epidermoid) cells, and were observed for 14 days after inoculation. If characteristic adenovirus cytopathic effect was observed, cells were placed on glass slides and stained with the same monoclonal antibody used for direct IF detection.

Identification of the culture isolates was achieved by a neutralization test using prototypic antisera prepared by the National Institutes of Health, Bethesda, MD, USA. Antisera to common local circulating adenovirus serotypes and serotypes associated with EKC (1–10, 19, 35, 36, 37) were used and all isolates were cleanly neutralized with adenovirus type 8 antiserum [11].

Adenovirus survival

To determine the survival of adenovirus serotype 8 in the ophthalmic solutions Tropicamide (dilating agent) and Alcaine (anaesthetic agent), a titred volume of a clinical isolate of adenovirus serotype 8 was inoculated into three volumes each of the dilating drops and local anaesthetic and maintained at 4 °C. The solutions were inoculated into HEp-2 and MRC5 cell monolayers at time intervals over 28 days. The cultures were observed for 14 days for cytopathic effect.

Analysis

Data were analysed using Epi-Info version 3.2 (Centers for Disease Control and Prevention, Atlanta, GA, USA, and World Health Organization, Geneva, Switzerland). We calculated odds ratios for categorical variables and tested for significance using the Mantel–Haenszel method or Fisher's exact test for cells with a value <5. A P value of $<0{\cdot}05$ was considered significant on two-tailed testing. For clinic-associated cases the incubation period was the number of days between date of last clinic visit and date of symptom onset.

RESULTS

Case finding

We identified 68 probable cases. A general practitioner identified one case, two cases were identified through review of local hospital emergency department records, 52 cases by review of clinic patient records, and 13 cases in the course of control interviews. Nine (16%) cases reported secondary spread among household contacts.

Eye swabs were taken from three cases on 15 March and two cases on 23 March. Four were positive for adenovirus by IF and two isolates were serotyped as adenovirus 8.

The epidemic curve shows that the first case had an onset of disease on 16 January (Fig. 1). There was a relatively low incidence of cases during January and February with a rise and tight clustering of cases in time from 7 to 13 March (Fig. 1).

Clinical characteristics

In addition to ocular redness that all patients experience with conjunctivitis, the most frequent additional symptoms reported by the 68 cases were photophobia (99%), tearing/ocular discharge (99%), ocular pain (99%), periorbital swelling (97%), and foreign-body sensation (89%). A minimum of 20% of cases reported impaired vision to the extent that they could not drive or read for at least 2 weeks.

Case-control study

We defined 56 cases as being clinic associated. These 56 cases attended the clinic on seven separate days over a period of 5 weeks. A total of 84% of clinic-associated cases attended the clinic on 2 days in late February and early March, although sporadic cases were identified in January and February (Fig. 2). A total of 709 patients attended the clinic during these 5 days and the overall incidence rate of clinic-associated EKC was 8%. Incidence rates varied between days: 3% on 30 January, 2% on 31 January, 1% on 13 February, 1% on 20 February, 1% on 27 February, 30% on 28 February and 9% on 2 March.

The mean incubation period for cases was 8·7 days (range 1·0–17·0 days). Eye swabs were taken from three clinic-associated cases; two were positive for adenovirus by IF and one was serotyped as adenovirus 8.

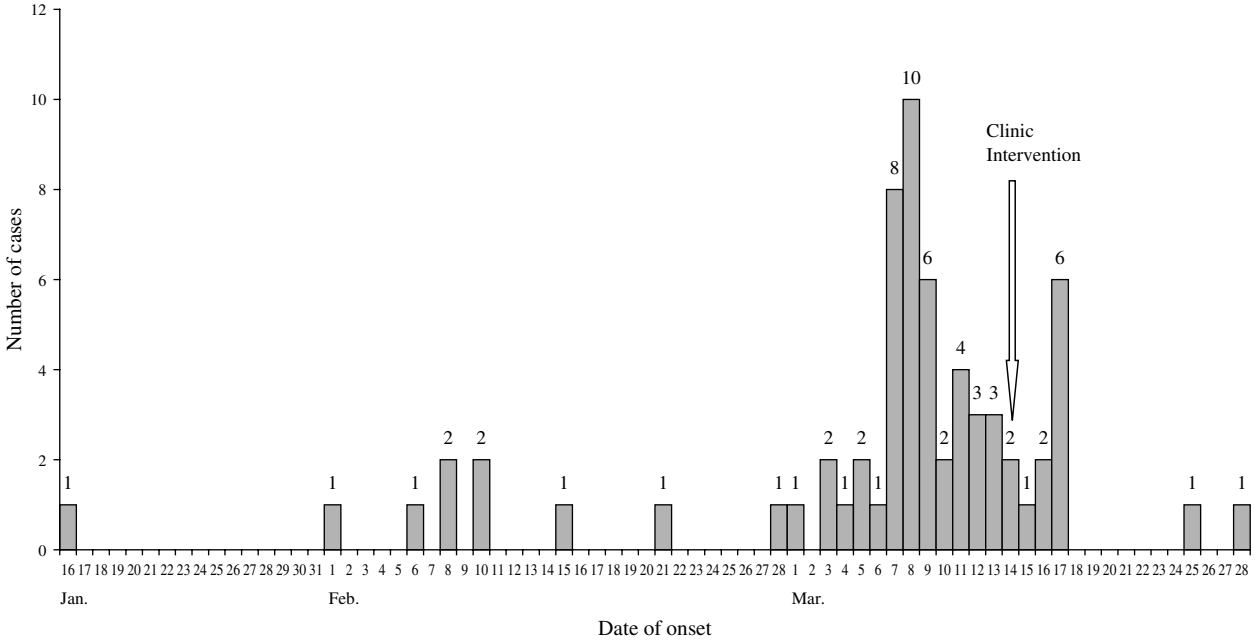

Fig. 1. Suspected and confirmed cases of epidemic keratoconjunctivitis in the local region by onset date, 16 January to 28 March 2006.

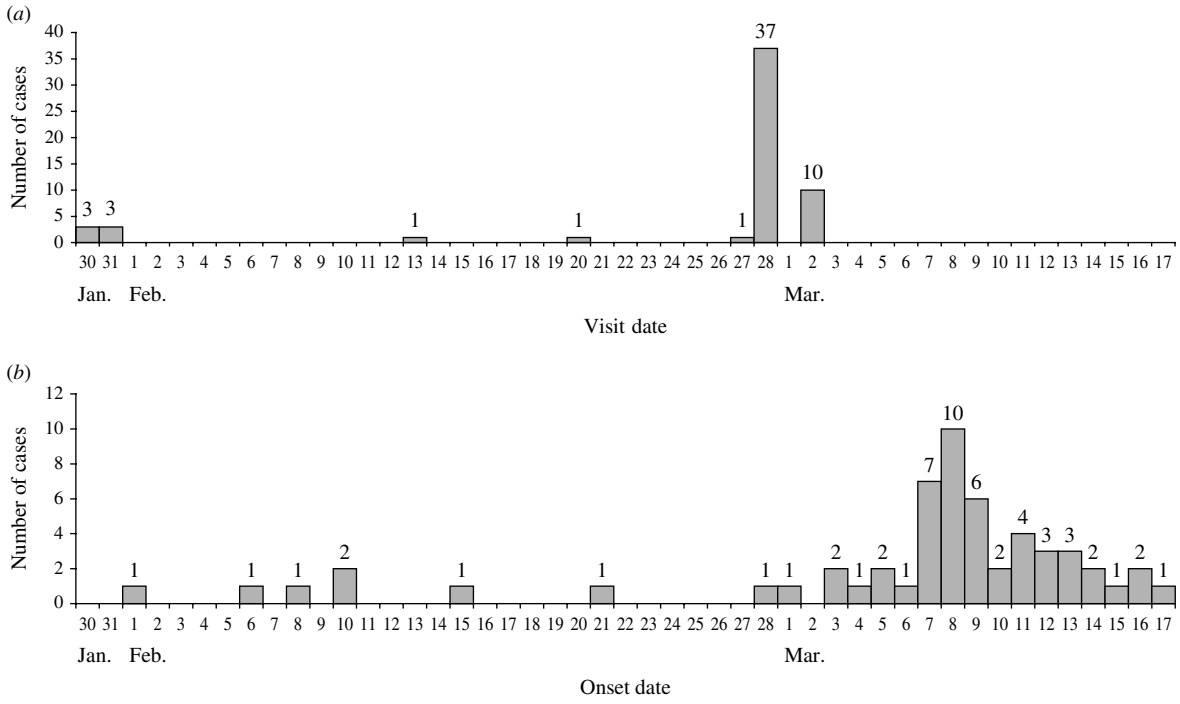

Fig. 2. Clinic-associated cases of epidemic keratoconjunctivitis by (*a*) clinic visit date and (*b*) disease onset date, 30 January to 17 March 2006.

All 56 clinic-associated cases were included in the case-control study and agreed to be interviewed. We attempted to contact 134 potential controls. Four were identified as probable cases. Three controls refused to be interviewed, two could not be interviewed due to illness and interstate travel, 20 could not be contacted and two were interviewed but provided incomplete data. Complete data was collected for 103, giving a response rate of 79%.

Table 1. *Sex, country of birth, age and residence for all reported cases of epidemic keratoconjunctivitis (EKC), and case-control study population from the clinic, 2006*

	All EKC cases ($N=68$) *n* (%)	Case-control study population				
		Cases ($N=56$) *n* (%)	Control ($N=103$) *n* (%)	OR (cases *vs.* controls)	95% CI	*P* value
Sex						
Male	29 (43)	26 (46·4)	47 (46)	1·03	0·51–2·09	0·92
Female	39 (57)	30 (53·6)	56 (54)			
Country of birth						
Australia	55 (80·9)	45 (80·4)	88 (85·4)	0·68	0·7–1·79	0·4
Other	13 (19·1)	11 (19·6)	15 (14·6)			
Age group (yr)				1·77*	0·86–3·63	0·09
0–9	0 (0)	0 (0)	5 (4·8)			
10–19	0 (0)	0 (0)	1 (0·9)			
20–29	1 (1·5)	0 (0)	0 (0)			
30–39	0 (0)	0 (0)	2 (1·9)			
40–49	5 (7·4)	1 (1·8)	5 (4·8)			
50–59	13 (19·1)	9 (16·1)	16 (15·5)			
60–69	12 (17·6)	10 (17·9)	16 (15·5)			
70–79	22 (32·4)	22 (39·3)	31 (30·1)			
80–89	12 (17·6)	12 (21·4)	27 (26·2)			
⩾90	2 (2·9)	2 (3·6)	0 (0)			
Missing data	1 (1·5)					

OR, Odds ratio; CI, confidence interval.
* Calculated on the proportion above the median age of 73 years.

Table 2. *Main ocular conditions for reported cases of epidemic keratoconjunctivitis and controls from the clinic, 2006 (not mutually exclusive)*

Main ocular condition	Cases ($N=56$) *n* (%)	Controls ($N=103$) *n* (%)	OR	95% CI	*P* value
Cataracts	22 (39·3)	36 (35)	1·2	0·58–2·49	0·58*
Corneal disease	1 (1·8)	4 (3·9)	0·45	0·01–4·72	0·66†
Diabetic retinopathy	7 (12·5)	17 (16·5)	0·72	0·25–2·02	0·5*
Glaucoma	13 (23·2)	11 (10·7)	2·53	0·97–6·66	0·04*
Macular degeneration	4 (7·1)	7 (6·8)	1·05	0·22–4·38	1†
Other‡	12 (21·4)	40 (38·8)	0·43	0·19–0·96	0·03*
Total	59	115			

OR, Odds ratio; CI, confidence interval.
* Mantel–Haenszel.
† Fisher's two-tailed.
‡ Other diagnoses reported included cholazion, floaters, photopsia, retinal vein occlusion, ptosis, pterygium and squint, amongst others.

Cases and controls reported similar demographic characteristics, although cases tended to be slightly older than controls (Table 1). Similar proportions of cases and controls were reported as having the ocular conditions of cataracts, macular degeneration, corneal disease and diabetic retinopathy (Table 2). More cases than controls had glaucoma (Table 2).

On their day of visit to the clinic, similar proportions of cases and controls underwent A-scan (measurement of the dimension of the eye) or visual-field testing by an orthoptist; had a diagnostic lens applied directly to the eye, underwent a surgical procedure, or had tonometry with instillation of anaesthetic drops by an ophthalmologist (Table 3).

Table 3. *Frequency, odds ratio and 95% confidence intervals of exposures in case-control study population from the clinic, 2006*

Exposure	Cases ($N=56$) *n* (%)	Controls ($N=103$) *n* (%)	OR	95% CI	*P* value
By orthoptist					
Anaesthetic drops§	54 (96·4)	64 (62·0)	16·45	3·89–145·11	<0·01*
Tonometry§	54 (96·4)	64 (62·0)	16·45	3·89–145·11	<0·01*
Optical coherence tomography	9 (16·1)	4 (3·9)	4·74	1·23–21·93‡	0·01†
Corneal pachymetry	0 (0)	0 (0)	n.a.	n.a.	n.a.
A-scan	1 (1·8)	2 (1·9)	0·92	0·02–18·02‡	1†
Visual field	2 (3·6)	3 (2·9)	1·23	0·11–11·11‡	1†
Dilating drops	31 (55·4)	36 (35)	2·31	1·13–4·74	0·01*
By ophthalmologist					
Diagnostic lens applied direct to eye	0 (0)	1 (1)	0	0–71·73‡	1†
Surgical procedure	1 (1·8)	3 (2·9)	0·61	0·01–7·78‡	1†
Tonometry	1 (1·8)	5 (4·9)	0·36	0·01–3·32‡	0·67†
Dilating drops	0 (0)	0 (0)	n.a.	n.a.	n.a.
Anaesthetic drops	1 (1·8)	6 (5·7)	0·29	0·01–2·54‡	0·42†
Orthoptist					
1	1 (1·8)	45 (43·7)	0·02	0–0·15‡	<0·01*
2	49 (87·5)	25 (24·3)	21·84	8·18–60·0	<0·01*
3	6 (10·7)	3 (2·9)	4	0·81–21·5‡	0·07†
Did not see	0 (0)	29 (28·2)	0	0–0·19‡	<0·01*
Missing	0 (0)	1 (0·9)	n.a.	n.a.	n.a.
Ophthalmologist					
1	3 (5·3)	1 (1)	5·77	0·45–305·99‡	0·13†
2	0 (0)	2 (1·9)	0	0–9·81‡	0·54†
3	16 (28·6)	22 (21·4)	1·47	0·65–3·31	0·31*
4	15 (26·8)	31 (30·1)	0·85	0·39–1·86	0·66*
5	1 (1·8)	16 (15·5)	0·1	0–0·68‡	<0·01*
6	21 (37·5)	29 (28·2)	1·53	0·72–3·23	0·23*
Did not see	0 (0)	2 (1·9)	0	0–9·81‡	0·54†

OR, Odds ratio; CI, confidence interval; n.a., not available.
* Mantel–Haenszel.
† Fisher's two-tailed.
‡ Exact CI.
§ While these factors have been separated in analysis, clinical advice is that tonometry requires prior instillation of anaesthetic drops.

However, significantly more cases than controls had tonometry with instillation of anaesthetic drops, optical coherence tomography, or instillation of dilating drops by an orthoptist (Table 3).

Significantly more cases than controls were seen by orthoptist 2 (Table 3). When cases and controls seen by orthoptist 2 (49 cases, 25 controls) were analysed separately, cases were significantly more likely to have had tonometry with the instillation of anaesthetic drops (Table 4). However, orthoptist 1 and ophthalmologist 5 saw significantly fewer cases (Table 3). There were two cases who did not have tonometry with the instillation of anaesthetic drops by an orthoptist, both of whom were seen by orthoptist 2, and both had dilating drops instilled. Among the cases and controls not seen by orthoptist 2 (7 cases, 77 controls), cases were significantly more likely to have had dilating drops instilled (OR 10·5, 95% CI 1·2–492·5, $P=0·02$).

In 70% of cases, the treating orthoptist had treated a person with infectious EKC earlier in the day and had treated them with anaesthetic drops and tonometry, or dilating drops. An additional 27% of cases were treated by an orthoptist who had treated a person with infectious EKC within the previous 2 days, performing instillation of anaesthetic drops

Table 4. *Analysis of procedures carried out by orthoptist 2: frequency, odds ratio and 95% confidence intervals of exposures*

Exposure	Cases ($N=49$) n (%)	Controls ($N=25$) n (%)	OR	95% CI	P value
Anaesthetic drops§	47 (95·9)	18 (72)	9·14	1·50–94·92‡	0·01†
Tonometry§	47 (95·9)	18 (72)	9·14	1·50–94·92‡	0·01†
Optical coherence tomography	8 (16·3)	1 (4)	4·68	0·56–216·3‡	0·26†
Corneal pachymetry	0	0	n.a.	n.a.	n.a.
A-scan	1 (2)	1 (4)	0·5	0·01–40·87	1†
Visual field	2 (4·1)	1 (4)	1·02	0·05–62·73‡	1†
Dilating drops	25 (51)	9 (36)	1·85	0·62–5·62	0·22*

OR, Odds ratio; CI, confidence interval; n.a., not available.
* Mantel–Haenszel.
† Fisher's two-tailed.
‡ Exact CI.
§ While these factors have been separated in analysis, clinical advice is that tonometry requires prior instillation of anaesthetic drops.

with tonometry, or instillation of dilating drops on the infectious case. Of the remaining 3% of patients (two), both were treated by one orthoptist and were treated on two separate days. There did not appear to be ongoing transmission of EKC from these patients.

Environmental investigation

The clinic provided about 600 episodes of patient care per week.

Each ophthalmologist and orthoptist reported consistently using a designated consultation room during the study period. A patient episode of care generally comprised consultation with both an orthoptist (for procedures such as the instillation of anaesthetic drops or dilating drops and subsequent measurement of ocular pressure using a Goldmann applanation tonometer) followed by consultation with an ophthalmologist. Examination equipment and medications such as eye drops were reported to be specific to each consultation room. Hand-washing facilities were available in two of the seven consultation rooms, or in the clinic bathroom.

Laboratory study

Adenovirus serotype 8 was recovered from the dilating drops up to 21 days post-inoculation with no change in the initial virus titre. Adenovirus was not able to be recovered from the anaesthetic drops due to the toxic effect of Alcaine in the cell cultures used.

Interventions

Clinic staff reported that prior to March 13, staff had used 15-ml multi-dose bottles of anaesthetic and dilating drops. Clinic staff only performed hand washing between patient examinations if the patient had clinical evidence of an eye infection (i.e. a red eye). Alcohol-based (66%) hand cleanser was provided in all consultation rooms and was used if patients had clinical evidence of an eye infection. Tonometer heads were cleaned with an alcohol wipe between patients; alcohol contact time could not be ascertained. Tonometer heads were soaked in 0·05% hypochlorite solution at the end of each day for 10 min, rinsed in tap water and left to air dry before re-use. In addition, bacteriostatic detergent spray was provided at a central point for surface cleaning of equipment between patients. However, cleaning of examination equipment was not part of the routine cleaning schedule.

Clinic staff implemented a number of infection control interventions immediately after 13 March 2006. Clinic staff reduced caseload for a period of four consecutive weekdays (emergency services were maintained), and 15-ml multi-dose bottles of anaesthetic and dilating drops were replaced by smaller volume vials (minims). Patients with a potential diagnosis of EKC were asked to wait in a separate waiting area and all consultation rooms were supplied with a bacteriostatic detergent spray. Clinicians' hand hygiene between patients was promoted by infection control and public health unit staff.

No cases of EKC attributable to a clinic visit after 13 March 2006 were reported to the clinic subsequent to these interventions.

DISCUSSION

We investigated a large outbreak of EKC in regional NSW that was associated with attendance at a private ophthalmology clinic. The case-control study found a significant association between the development of EKC and the instillation of anaesthetic eye drops, the use of tonometry and the instillation of dilating drops (by one orthoptist). There was evidence of ongoing transmission of EKC within households and of EKC circulating within the community.

EKC was probably transmitted in the clinic primarily by anaesthetic drops. These were probably contaminated during treatment of a person with infectious EKC, which then spread disease to subsequent patients. On one particular day 35 cases were preceded by an infectious case and all cases saw the same orthoptist and underwent similar procedures. Applanation tonometry may have also played a role in transmission within the clinic, but if this was the case we would expect that on the day that an infectious EKC case visited the clinic and 35 patients were subsequently infected, transmission would not occur evenly throughout the day as tonometer heads are wiped with an alcohol swab between each patient. This was not the case, and patients who visited subsequent to an infectious case developed infection throughout the whole day. The infection of 15 patients up to 2 days after the treatment of an infected patient further suggests the independent contribution of contaminated eye drops from multi-dose bottles as tonometer heads were reprocessed at the end of each day. A study has demonstrated that multi-dose bottles used on infected patients can serve as vectors for transmission for as long as 9 weeks [12]. In addition, other infection control practices which probably contributed to ongoing transmission of EKC within the clinic included the continued use of tonometer heads after a patient with red eye had visited, no documented triage protocol for patients presenting with a red eye, and variable compliance with hand hygiene.

Multiple studies have evaluated risk factors for transmission of EKC within the clinical setting. This study's findings of the association between EKC infection and instillation of anaesthetic drops, the use of tonometry and the instillation of dilating drops is consistent with other outbreak investigations in ophthalmic and clinic settings, which have implicated contaminated eye drops or tonometer heads [8, 9, 13, 14] and contact with particular uninfected healthcare workers [2, 4].

Other risk factors for clinic-associated EKC transmission have included direct application of a diagnostic lens to the eye [2], hand-to-eye contact [5, 8, 12, 15, 16], foreign-body removal [14, 17], invasive ophthalmic procedures [9, 17], infected heathcare workers [8, 13, 16, 18], pneumotonometry [4, 5, 18, 19] and contaminated environmental surfaces [20]. The contribution of environmental contamination is considered to be uncertain in this outbreak, although the absence of EKC infection among clinic staff suggests that transmission via fomites did not play an important role.

A greater proportion of clinic-associated cases than controls had glaucoma in this outbreak. Patients with glaucoma were more likely to require ocular pressure measurement using anaesthetic drops and tonometry, and this may have placed them at increased risk of EKC infection in this outbreak.

The reported incubation period for EKC is 4–10 days [6]. The incubation period in our study however, ranged from 1 to 17 days with a mean of 8·7 days. Recall bias may explain a relatively short incubation period in some cases. However, our study's findings regarding the shorter incubation period than that described in the literature may require further investigation.

There are a number of limitations to our investigation. First, the independent effects of anaesthetic drops and tonometry could not be tested statistically in this study as no tonometry had been undertaken without prior instillation of anaesthetic drops. However, the transmission of EKC to clinic patients on the first or second day after the treatment of a person with EKC suggests the anaesthetic drops were contaminated and made an independent contribution to the spread of disease. Second, the reduced risk associated with ophthalmologist 5 may be confounded by a pattern of association between individual orthoptists and ophthalmologists. Third, the role of hand-to-eye contact in this outbreak was not determined, but was considered to have potentially contributed to the spread of disease. Hand-to-eye contact arising in the course of treatment was not measured in this study on advice from clinic staff that the reporting of these practices would be of uncertain reliability and validity. Fourth, possible incomplete ascertainment of cases

may result in an underestimate of the incidence rate in the clinic. However, most cases were associated with attendance at the clinic on two particular days and the incidence rates on the remaining five days were low, suggesting that any under-ascertainment was small. Fifth, while the public health unit recommended that the clinic collect swabs, only five were taken at two points in the outbreak. Nonetheless, the strong epidemiological evidence and the consistency of clinical symptoms indicate a single source of infection.

The strong odds ratios found for tonometry with instillation of anaesthetic drops undertaken by orthoptist 2 suggest that these limitations are unlikely to have affected the findings that transmission of EKC in the clinic was probably due to contamination of either or both the anaesthetic drops and the tonometer head in the room used by orthoptist 2.

Further, the laboratory study highlights the potential risk that multi-dose ophthalmic solutions contaminated with adenovirus can present to the initiation of an outbreak of EKC.

In this study, the stability of adenoviruses in Tropicamide indicates the potential role of this eye-drop solution as a vector of adenoviral transmission. Similarly, the toxic effect of Alcaine on the cell cultures used in the laboratory study does not discount its potential role as a vector of adenoviral transmission.

A comprehensive suite of strategies is required to prevent healthcare-associated EKC infection including: triaging of suspected cases to separate waiting and treatment areas, reprocessing of equipment used on suspected cases prior to use on another patient, the use of single-dose vials of anaesthetic and dilating drops discarded after each patient, rigorous hand hygiene between patients and cleaning of all equipment surfaces with a neutral detergent after each patient consultation. In addition, leave of absence would be required for clinical staff presenting with symptoms suggestive of disease.

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DECLARATION OF INTEREST

None.

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