

gene which, unlike high level resistance mediated by the *van A* gene, is difficult to detect.

Efforts to contain VRE and prevent its spread to others is necessary for the management of patients colonised with this organism. Affected patients should be isolated and standard infection control principles adhered to. Particular attention should be paid to the decontamination and disinfection of the environment around the patient. The patient should remain in isolation while colonised with VRE or if readmitted without VRE 'clearance'. The patient may be 'delisted' if rectal and lesion swabs for VRE are persistently negative (three cultures on consecutive weeks) in hospital.

A record of VRE cases should be kept for the epidemiological tracking of cases including their location, antibiotic history and risk factors. Vancomycin use should be re-

served for specific conditions and hospitals should develop guidelines for the proper use of vancomycin⁵.

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ACUTE FLACCID PARALYSIS SURVEILLANCE IN AUSTRALIA: THE FIRST YEAR

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Abstract

Surveillance for acute flaccid paralysis commenced through the Australian Paediatric Surveillance Unit in March 1995. Thirty-five cases were reported in the first year, giving an estimated incidence of 0.90 cases per 100,000 children under the age of 15 years. Nearly half the cases were Guillain-Barre syndrome. No cases of poliomyelitis were identified. This surveillance scheme will assist in the process of certification of the eradication of poliomyelitis in Australia and the World Health Organization Western Pacific Region. *Comm Dis Intell* 1996;20:403-405.

Introduction

The World Health Organization (WHO) aims to eradicate poliomyelitis from the world by the year 2000¹. Poliomyelitis has already been eradicated from the Americas². For a country to be declared polio free it needs to meet a number of requirements, including polio vaccination coverage of more than 80%, no confirmed poliomyelitis cases for three years and adequate surveillance and investigation of suspected poliomyelitis cases.

Australia has not had any poliomyelitis cases reported through the National Notifiable Diseases Surveillance System since one case was reported in 1986, one case in 1978 and two cases in 1977³. The WHO however considers the detection and investigation of all cases of acute flaccid paralysis (AFP) as an essential and sensitive method of detecting wild poliovirus.

The differential diagnosis of acute flaccid paralysis includes Guillain-Barre syndrome, transverse myelitis and traumatic paralysis⁴. Other viruses (for example enterovirus types 70 and 71) may mimic polio. All these

events are rare and little is known about the incidence, clinical course and outcomes of AFP in Australia.

In March 1995, surveillance of acute flaccid paralysis commenced through the Australian Paediatric Surveillance Unit (APSU). The aims of the study were to describe the incidence, causes and clinical picture of AFP cases in Australia and to determine whether any cases of AFP are caused by paralytic 'wild' poliovirus.

Methods

A case of acute flaccid paralysis was defined as a child aged less than 16 years with:

- acute onset of flaccid paralysis in one or more limbs
- or
- acute onset of bulbar paralysis.

The Australian Paediatric Surveillance Unit (a unit of the Australian College of Paediatrics) conducts active, prospective national surveillance of selected rare paediatric

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conditions by sending a reply-paid report card each month to over 900 paediatricians in Australia. Paediatricians are asked whether or not they have seen any of a number of conditions listed on the card. Over 90 per cent of paediatricians return the card each month. Acute flaccid paralysis was included on the APSU card from March 1995.

In addition to returning the APSU card, paediatricians were asked to report cases by telephone to the principal investigator. This was so that paediatricians could be reminded of the WHO requirements for stool testing and asked to comply with them.

Paediatricians were asked to collect two stool specimens from the case, preferably 24 hours apart, within 14 days of the onset of paralysis. Specimens were then sent for viral culture, in particular looking for poliovirus. If poliovirus was isolated, it was sent to the National Polio Reference Laboratory in Melbourne for characterisation as wild or vaccine-like. This protocol has recently changed slightly (see Box).

A two-page questionnaire on the clinical features, laboratory investigations and final diagnosis of the case was sent to reporting paediatricians and was followed by a second questionnaire at 60 days asking about any residual paralysis.

Data analysis was performed using Epi Info version 6⁵.

Cases were classified according to WHO criteria⁶ as:

- Poliomyelitis: an AFP case with wild poliovirus isolation.
- Non-polio AFP: an AFP case with adequate stool specimen testing negative, or with no residual paralysis except if wild virus is isolated, or
- Polio-compatible: an AFP case with residual paralysis or who died or was lost to follow-up and for whom stool specimens were either not taken or were inadequate.

Results

There were 35 cases of AFP with onset dates between March 1995 and February 1996, reported by 49 paediatricians. The 35 cases in 12 months corresponded to an

Table 1. Acute flaccid paralysis cases reported by State and Territory, March 1995 - February 1996

State/Territory	Number of cases
ACT	0
NSW	14
NT	1
Qld	9
SA	2
Tas	2
Vic	3
WA	4

incidence of 0.90 per 100,000 children under the age of 15 years.

Nineteen initial reports (54%) were by telephone. Completed questionnaires containing clinical information were provided for 29 cases. The diagnosis alone was provided for four cases and no information at all was available for two cases. Reports were for cases from all States and Territories except the Australian Capital Territory (Table 1).

Of the 33 cases for which information was provided, 18 cases were male and 15 female. Their ages ranged from two months to 12 years. There was no seasonal distribution of cases. Their reported diagnoses are described in Table 2. Forty-eight per cent of cases were diagnosed as having Guillain-Barre syndrome. No cases of poliomyelitis were identified.

One case with a diagnosis of transverse myelitis had been vaccinated with oral polio vaccine seven days prior to developing paralysis; poliovirus type 3 (Sabin-like) was identified from his stool. In addition, the case had a demonstrated seroconversion to poliovirus type 3.

Thirty-two cases were hospitalised and 11 required intensive care admission. Nineteen cases had paralysis of all four limbs, six had paralysis of two limbs, one had paralysis of one limb and no information was reported for seven cases. Six cases had bulbar paralysis and six had respiratory depression. Nine cases had cranial nerve involvement. Eighteen cases had residual paralysis at 60 days. There were no deaths.

Only eight cases had two stool samples taken and tested according to WHO criteria. A further four cases had one stool sample taken. As a result, of the 35 total cases, 17 were classified as non-polio and 18 were classified as polio compatible.

Discussion

The first year of surveillance of acute flaccid paralysis in Australia has supported the presumption that Australia is free of wild poliomyelitis. The estimated incidence of 0.90 cases of AFP per 100,000 children under the age of 15 years is close to the one case per 100,000 expected for a poliomyelitis-free country. Surveillance of AFP in the Americas found an annual incidence of 1.4 cases per 100,000 children

Table 2. Diagnosis for 33 cases of acute flaccid paralysis, Australia, March 1995 - February 1996

Diagnosis	Number of cases
Guillain-Barre syndrome	16
Transverse myelitis	5
Demyelination	3
Encephalomyelitis	2
Trauma	2
Hypotensive brainstem necrosis or demyelination	1
Lumbar radiculopathy	1
Post drug polyneuropathy	1
Infant botulism	1

under the age of 15 in 1991, the year in which the last case of polio occurred⁷. In the United Kingdom, the rate is approximately one case per 100,000 children under the age of 16 years⁸. The three cases of AFP identified in Victoria are fewer than the expected nine cases for that population and indicate there may be under-reporting in that State. In other States and Territories, the numbers of cases were as expected.

No cases of poliomyelitis were identified by this surveillance system. Although according to WHO criteria, 18 of the cases of AFP would be classified as polio compatible, most cases had multiple investigations and were treated by paediatric neurologists. In these circumstances it is unlikely that the diagnoses reported are incorrect. Paediatricians are encouraged however to continue stool testing to exclude not only poliomyelitis but other viral causes of paralysis. In one case during this study the testing identified an echovirus type 9 as the probable cause of the illness.

The causes of AFP identified in this study are consistent with the experience of similar surveillance in the United Kingdom, with 48% of cases being Guillain-Barre syndrome and 15% transverse myelitis⁸. While no definite vaccine-associated paralysis was identified, one case with a diagnosis of transverse myelitis did have Sabin-like poliovirus type 3 identified from his stool and documented seroconversion to the same virus. It is estimated that one case of vaccine-associated paralysis will occur with every 2.5 million doses of oral polio vaccine distributed⁹. Surveillance of AFP would be expected to occasionally identify these cases.

Australia will shortly be undergoing scrutiny to determine whether wild poliovirus exists in this country, as part of the process of certification of poliomyelitis-free status in the Western Pacific Region of the World Health Organization. A Regional Commission for the Certification of Poliomyelitis Eradication met for the first time in April 1996. Surveillance of cases of AFP will provide information to assist in certifying Australia and the Region polio-free.

While it is unlikely that Australia does have indigenous wild poliovirus, the potential for importation of the virus from other countries is still high. In 1992 in the Netherlands a wild poliovirus type 3 strain introduced from India was responsible for a large outbreak of poliomyelitis in a community opposed to immunisation¹⁰. In 1993 the same virus was identified in Canada through active surveillance in a linked community¹¹. Continued high rates of routine vaccination against polio are still required in Australia to prevent similar importation. Surveillance for poliomyelitis is also important so that if imported poliomyelitis does occur, an immediate public health response can be commenced.

Acknowledgements

The APSU is funded by the Financial Markets Foundation for Children and the Department of Health and Family Services. The National Polio Reference Laboratory receives funding from the National Centre for Disease

Control of the Department of Health and Family Services, and the Victorian Department of Human Services. We would also like to thank the individual paediatricians and laboratories who have contributed to this study.

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Reporting cases of acute flaccid paralysis

Paediatricians should report cases of acute flaccid paralysis to the Australian Paediatric Surveillance Unit (APSU), and by telephone to Dr Ana Herceg on (06) 289 8638.

Stool specimen testing

Two stool specimens from every case of AFP should be collected, preferably 24 hours apart, within 14 days of the onset of paralysis. Following a World Health Organization Technical Advisory Group meeting in Canberra in April 1996, the laboratory protocol for testing stool specimens for AFP has changed. The WHO states that specimens should be tested in a WHO certified laboratory. In Australia, only the National Polio Reference Laboratory is certified.

All stool specimens should now be sent directly to the National Polio Reference Laboratory in Melbourne, not to the State or Territory virology laboratory as previously requested. Laboratories can obtain information on specimen transport from Mrs Margery Kennett on (03) 9280 2397. The National Polio Reference Laboratory will pay for specimen transport.