Lesson of the Week

Vaccine related poliomyelitis in non-immunised relatives and household contacts

D E BATEMAN, G ELRINGTON, P KENNEDY, M SAUNDERS

The diagnosis of paralytic poliomyelitis is now rarely considered in cases of acute lower motor neurone weakness in patients resident in Britain. Paralytic poliomyelitis still occurs,¹ however, and we report two cases, one of which would not have occurred had the present immunisation procedures been followed. The occurrence of the other case suggests that immunisation procedures should be modified.

Case 1

A 16 year old boy developed a sore throat, nausea and anorexia, and general malaise two weeks after his niece, a household contact, had received oral poliomyelitis vaccine. Four days later he deteriorated with generalised headache and photophobia, and the next day he awoke with a completely paralysed left arm. He was said to have been immunised against poliomyelitis as a child but this could not be confirmed. On admission he had a fever of 38°C, mild neck stiffness and photophobia, and flaccid paralysis of his left arm with absent reflexes in the arm and normal sensation. Results of routine blood tests were negative, as were viral antibody titres. Examination of cerebrospinal fluid yielded 15×10^6 lymphocytes/l, a protein concentration of 0.33 g/l, and a glucose concentration of 3.6 mmol/l (65 mg/100 ml). Serum antibody titres to poliovirus on the day of admission and 14 days and six weeks later were less than 16 to P1 and P3 but showed a fourfold or greater rise to P2 (1/64, 1/256, 1/1024). Stool culture grew a poliovirus type 2 of a vaccine related strain.

The patient's systemic symptoms settled rapidly in two days, but over the next five years there was no appreciable return of neurological function in the left arm.

Case 2

A 23 year old toolmaker developed a sore throat and mild frontal headache 47 days after his son had been immunised against poliomyelitis. Three days later his condition became worse with generalised headache and vomiting and general malaise. A week after his initial symptoms he noticed difficulty swallowing and weakness of his voice and was admitted to hospital. He had never been immunised against poliomyelitis and had had a tonsillectomy. On admission he was feverish (38°C) with mild neck stiffness. Neurological examination showed nystagmus on lateral gaze, severe bilateral facial weakness, and weakness of jaw opening. Palatal movement was absent, though sensation was normal and there was no cough. There was severe weakness of the neck flexors and tongue protrusion. The limbs showed mild weakness of the right deltoid, biceps, and triceps with reduction in these reflexes but no other abnormalities and no sensory loss.

Non-immunised parents and household contacts of children receiving primary immunisation should be immunised against poliomyelitis at the same time as their children.

Initial examination of cerebrospinal fluid yielded 41×10^6 mononuclear cells/l with a protein concentration of 0.5 g/l and glucose concentration 3.5 mmol/l (63 mg/100 ml). Stool cultures from the patient and his son grew a poliovirus type 3 of a Sabin-like strain. Initial serum antibody titres to poliovirus types 1, 2, and 3 were negative but a subsequent specimen after 10 days showed a greater than fourfold rise to P3 only (1/1024). The initial cerebrospinal fluid poliovirus antibody titre was negative but a repeat examination 10 days later showed a titre of 1/32 to P3. Serum antibody titres to other viruses (Coxsackie and ECHO) were negative.

Owing to failure to control secretions the patient required a tracheostomy shortly after admission. He developed no further weakness and made a gradual recovery over the next six weeks, being left with a mild bulbar palsy and weakness of his sternomastoids.

Discussion

Both these patients had clinical illnesses characteristic of paralytic poliomyelitis. Neither had been previously immunised. Though our first patient was thought by his parents to have been immunised, this could not be confirmed and his initial antibody titres showed this not to be the case. Both patients were excreting a vaccine related strain of poliovirus, and antibody titres in blood and cerebrospinal fluid (case 2) showed evidence of recent infection with the appropriate vaccine related strain confirming that they had vaccine related poliomyelitis.

Poliomyelitis is defined as being "contact vaccine associated" if it occurs in a patient within four to 60 days after contact with a recently vaccinated person.^{2,3} In case 2 the period of 47 days after immunisation of the patient's son before the development of paralytic poliomyelitis was clearly well within this definition, and furthermore the son was still excreting a similar Sabin related strain. Vaccine related poliomyelitis in contacts is well described and estimated to occur with a frequency of 0.2-0.4 cases per million doses.^{2,3} The WHO collaborative study showed that in 69% of cases the patients were aged 15 or over and many were young non-immunised parents of children receiving primary immunisation.³ Vaccine associated poliomyelitis in contacts occurs most commonly with strains related to P2 and P3.³ Studies have shown that Sabin polioviruses increase in neurovirulence on passage in the human gut,⁴ and recently this has been found to be associated with a single nucleotide change in a non-coding region of the genome of the Sabin type 3 poliomyelitis vaccine.⁵

Our two patients are therefore typical examples of vaccine associated paralytic poliomyelitis in contacts. Neither had been

Wessex Neurological Centre, Southampton General Hospital, Southampton SO9 4XY

D E BATEMAN, MD, MRCP, senior registrar in neurology

G ELRINGTON, MB, MRCP, registrar in neurology

P KENNEDY, MB, FRCP, consultant neurologist

Department of Neurology, Middlesbrough General Hospital, Middlesbrough TS5 5AZ

M SAUNDERS, MB, FRCP, consultant neurologist

Correspondence to: Dr Bateman.

immunised and they were in contact with relatives receiving primary immunisation; one case was associated with a P2 Sabin like strain and the other with P3.

To prevent vaccine associated poliomyelitis in contacts it is currently recommended that non-immunised parents of infants receiving primary immunisation should be immunised either before or at the same time as their children. Naturally this is often forgotten, and clearly our second patient would not have contracted paralytic poliomyelitis had these recommendations been followed. Case 1 could not have been prevented since the patient was believed to have been immunised and present recommendations regarding immunisation extend only to parents. The occurrence of case 1, however, emphasises that the recommendations should be extended to household contacts of the child being immunised.

Health visitors now interview parents before primary immunisation, and a simple way to prevent further cases would be to include a question about the immunisation state of parents and household contacts and then to ensure that all non-immunised relatives and

household contacts are immunised either before or at the same time as their children.

We thank Dr M Ogilvie for her help with the virological studies in case 2.

References

- 1 Smith JWG, Wherry PJ. Poliomyelitis surveillance in England and Wales 1969-1975. *J Hyg (Camb)* 1978;80:155-67.
- 2 Collingham KE, Pollack TM, Roebuck MO. Paralytic poliomyelitis in England and Wales 1976-1977. *Lancet* 1978;i:976-7.
- 3 WHO Consultative Group. The relation between acute persisting spinal paralysis and poliomyelitis vaccine—results of a ten year enquiry. *Bull WHO* 1982;60:231-42.
- 4 Magrath DI, Boulger LR, Hartley EG. Strains of polio virus from individuals fed Sabin vaccine: studies by in vitro markers and the monkey neurovirulence test. In: *Proceedings of 10th European symposium on poliomyelitis*. Brussels: EAP, 1964:334-5.
- 5 Evans DMA, Dunn G, Minor PD, et al. Increased neurovirulence associated with a single nucleotide change in a non-coding region of the Sabin type 3 poliovaccine genome. *Nature* 1985;314:548-50.

(Accepted 16 October 1986)

PORTRAITS FROM MEMORY

3—Professor J Henry Dible (1890-1971)

Those who did not know Dible well were inclined to regard him as strangely conservative and reactionary and nothing more. This opinion is understandable, but it far from expresses the whole truth. Dible did not welcome change, and it was only after a long and sometimes bitter struggle that he eventually came to accept that the members of the Pathological Society, as well as those of the Association of Clinical Pathologists (ACP), really wanted to unite to found a College of Pathologists. When at last convinced of this, however, he gave much good advice to those concerned with the formation of the college. He was rightly anxious to ensure that the college should be a truly independent offspring of its two parents, the reluctant father (the Pathological Society) and the importunate mother (the ACP)—and so it happily became. In 1967 he was glad to accept an honorary fellowship of the college.

One of Dible's greatest contributions to pathology lay in the wise and perceptive way in which he organised the twice yearly scientific meetings of the Pathological Society. With his alarm clock set to ring after 15 minutes he ensured that speakers never overran their allotted time. Appeals for extensions were rightly and firmly drowned by the continued ringing of the clock, and inexperienced presenters of first papers were often privately and wisely guided by Dible's words, "You are given fifteen minutes; take only twelve." He often recalled the overbusy meeting at which the last few speakers were each given exactly two minutes in which to state only their conclusions. These brief communications produced a series of such lively and successful discussions that the merits of taking less than the permitted 15 minutes should never have needed re-emphasising.

Dible gave good advice also on preparing slides and speaking only from headings. "Say what you did; why you did it; what you found; and what you think it means. Keep to essentials, and you'll get a good discussion in which you can give such further details as may prove to be really wanted." Dismayed by falling standards of presentation, he gave an admirable communication to one meeting in the 1960s, summarising the advice he had so often given privately. He approvingly quoted S L Baker, who complained that "recently we have had too many inaudible soliloquies addressed to invisible tables."

To Dible, the pathologist was "the consultant's consultant." The pathologist should aim at being a man with knowledge and training in the science of medicine superior to that of the clinician. He deplored the tendency of pathologists to become overinvolved with the details of laboratory methods, and, with considerable foresight as early as the 1950s, he urged pathologists to make greater and better use of the aid of skilled technicians, science graduates, and machines.

He was a keen sailor and a member of the Royal Yacht Club. As a host or a guest he was an admirably relaxed and entertaining companion. He was an expert on wines, having discovered the merits of Chateau Pontet Canet long before it became fashionable. He told me how, for the entertainment of an American host, he had tramped the hot, hard sidewalks of New York for hours before landing a few bottles of the chosen claret. He sought to explain to the wine waiter of the hotel exactly how he wished them to be treated. In vain: he was waved aside with a "Leave it to me, sir—in this hotel we do not need to be told how to handle a fine claret." Reluctantly, he left it to the waiter, who duly served the bottles, still with their corks in, but safely packed on ice!—JAMES HOWIE.

What treatment is advised for an elderly woman who is losing her hair?

Firstly, a diagnosis must be made and this will need a full history of the complaint, relevant symptoms and diseases, the family history of hairfall, and then a general examination to assess the distribution of the hairfall, the presence of disease of the scalp and skin disease elsewhere, and signs of anaemia and thyroid disease. If the hairfall is associated with dandruff it may be secondary to psoriasis, pityriasis capitis, or even tinea capitis, but if accompanied by scarring lichen planus or lupus erythematosus may be the diagnosis. If the scalp appears normal and hairfall is diffuse there may be a systemic cause for it such as iron deficiency (the serum iron concentration may be low even with a normal blood count and blood film), thyroid disease, or recent acute illness. If the hairfall is well defined and localised tinea capitis or alopecia areata should be considered. If it is more obvious in the frontal and temple areas then traction due to hairdressing techniques such as back combing and tight rollers may be responsible. Finally, if the hairfall is mostly on the vertex and frontal areas and the scalp appears otherwise quite healthy it might be due to hormonal disease—unlikely in the elderly—or most probably, and this is the commonest type of hairfall in the elderly, the hair follicles have just given up producing hair. We then blame their inheritance because we have no better explanation, a diagnosis usually supported by the family history.

If the cause is remediable once treated the hair should regrow. If not the best remedy is to wear a wig, which can produce a remarkable and welcome transformation. Two are needed—one to wear and one to wash—and the NHS will subsidise wigs for women with obvious hairfall difficult to conceal with simple hairdressing techniques, but wigs cannot be prescribed on NHS prescriptions by general practitioners. There is a wide choice of attractive acrylic wigs, which may be washed at home and, although they may need replacing within two years, in the long run they are cheaper than bespoke human hair wigs and more convenient because they do not need to be regularly dressed by the makers.—ALAN B SHRANK, consultant dermatologist, Shrewsbury.