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Acute paralytic poliomyelitis presenting as Guillain–Barré syndrome

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Summary

The initial clinical picture and CSF changes in four children with acute ascending paralysis simulated Guillain–Barré syndrome. However, diagnosis of poliomyelitis was confirmed on the basis of isolation of wild poliovirus type 1 and high neutralising antibody to it. The four children had received primary vaccination with live attenuated oral poliomyelitis vaccine. It is postulated that the clinical course of paralytic poliomyelitis may be altered in children who have previously been vaccinated with live oral vaccine.

Introduction

Infection of a susceptible person with poliovirus type 1, 2 or 3 usually results in an inapparent infection or abortive illness.[1] Paralysis affects only 1–2% of cases[1] even fewer among young children.[2] The disease can be prevented by immunisation with Sabin oral live attenuated poliomyelitis vaccine (OPV) or Salk inactivated vaccine (IPV).[2] Paralytic poliomyelitis used to be common in Saudi Arabia until a mass vaccination programme was started by Royal decree in 1979. This linked vaccination with birth registration.[3,4] UNICEF have recently estimated that at least 90% of the Saudi children are vaccinated against poliomyelitis (Ministry of Health Annual Report, 1988). OPV is used for primary and booster vaccinations.

Paralytic poliomyelitis may manifest itself in children as spinal, bulbar, bulbospinal or encephalitic forms. Paralysis is described as patchy and asymmetrical, involving one or more groups of muscles. Involvement of the respiratory muscles is rare. Progression of paralysis is usually rapid but ceases within the first few days of its onset.[1,2]

Acute paralytic poliomyelitis can be difficult to differentiate from Guillain–Barré syndrome (GBS) on clinical features alone. Over the last year (1988–1989) four children were referred to us with a diagnosis of GBS who were subsequently proved to have poliomyelitis. Their clinical features, CSF changes and detailed virological studies form the basis of this report.

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Methods

Patients

Four children with a diagnosis of GBS were referred during a 1-year period to the King Khalid University Hospital (KKUH). Three of the patients were treated initially at other intensive care units in the Kingdom and were sent to us for plasmapheresis and/or virological investigations.

Laboratory investigations

Rectal and throat swabs, blood on each of the four patients and CSF of two cases were sent for virological investigations. At KKUH virus isolation on the rectal and throat swabs and CSF was attempted in two cell lines: vero cells (Flow Laboratories, U.K.) and human embryonic lung fibroblast (King Faisal Specialist Hospital and Research Centre, Riyadh, Saudi Arabia). Blood was tested for neutralising antibodies against a wide range of enteroviruses.

Results

A summary of the relevant clinical features and laboratory data of the four patients is shown in Table I. All four had received three doses of OPV at the same time as DPT vaccine at ages 3, 4 and 5 months. Patients 3 and 4 had the first booster dose of OPV and DPT at 18 months of age. None of the children had been given any injections or any vaccine during the 3 months prior to the onset of the illness.

Clinical assessment

In all cases the disease started as fever and cough for a few days. None had any history of vomiting, diarrhoea or convulsions. All had mild fever (37·5–38 °C) on admission. In every case the paralysis started in the lower limbs, was symmetrical and progressed in an ascending fashion involving upper limbs, chest and abdominal muscles, laryngeal and, lastly, the extraocular muscles. The level of consciousness remained normal throughout the illness in all cases. The paralysis was of lower motor neurone type, flaccid with absent tendon reflexes. There was no sensory loss. It took an average of 4·5 days (range 2–9 days) for the paralysis to become complete and reach its nadir. All needed mechanical ventilation for respiratory failure resulting from chest, abdominal and laryngeal muscle paralysis. One patient died from persistent hypoxia leading to brain death. Motor or sensory nerve conduction velocities were not recorded in any of the patients.

Virological studies

An enterovirus-like cytopathic effect (CPE) could be detected in three specimens (rectal swabs) 5–6 days post-inoculation and in both cell lines, from patients 1, 2 and 4. The isolates were passaged twice more in tissue culture for adaptation and for a high virus yield. From their CPE and electron microscopic appearance (28 nm diameter) they were identified as an enterovirus and as poliovirus type 1 from the neutralisation test using anti-polio 1, 2 and 3 and also by immunoelectron microscopy. The three poliovirus isolates were

Table I *Clinical and laboratory data*

Number	Age (years) and sex	Days to maximum paralysis	Ventilated days	Days to recovery	Functional grading at discharge*	CSF			Poliovirus isolate and titres		
						Days of illness	Cells per cm^3	Protein (mg %)	Days of illness	Type	Titre
1	9 M	5	33	50	2	3	4	44	3	1	256
						50	1	244	—	—	—
2	8 M	2	20	35	2	1	0	40	14	1	256
						6	2	167	—	—	—
						14	2	285	—	—	—
3	4 F	9	17	33	3	9	2	45	10	none	512
4	2 F	2	5	—	6	1	2	40	5	1	512

* Functional grading. Grade 1. Full motor recovery. Grade 2. Able to walk without any support. Grade 3. Able to walk with support. Grade 4. Bed or chair bound (unable to walk). Grade 5. Assisted ventilation required for at least part of the day. Grade 6. Dead.

identified as wild-type poliovirus by using the temperature marker test.[5] Blood from all four patients showed high neutralising antibody titres to the isolated virus. No virus was isolated from throat swabs or CSF specimens.

Discussion

Although all four cases fulfilled the diagnostic criteria of GBS at presentation,[6] the rapid onset of paralysis and the relatively short interval of only 4·5 days to maximum weakness is against the natural history of that disease.[7,8] This prompted us to do virological studies. In poliomyelitis the CSF characteristically shows pleocytosis with normal or slightly raised proteins in the acute stage and raised proteins and decreased cell count subsequently.[1,2] However, several investigators have shown that 0·5–15 % patients with acute poliomyelitis may have normal cell counts and 25–50 % have raised proteins in the CSF.[9] Our four patients had this albuminocytological dissociation, which prompted the initial diagnosis of GBS. The near complete recovery of three of the patients is not the usual outcome in severe cases of poliomyelitis with paralysis. The fourth case died from severe hypoxia before effective mechanical ventilation was established.

The diagnosis of acute paralytic poliomyelitis was based on the clinical picture of acute ascending lower motor neurone paralysis and high antibody titres in all four patients and concurrent isolation from rectal swabs of wild-type 1 poliovirus in three of them. The neutralising antibody test was done on only one blood sample from each patient, but the high titre found (> 256) was strongly suggestive of recent infection. Preliminary results of an investigation into the amounts of neutralising antibody to polioviruses in vaccinated Saudi children show that the titre rarely exceeds 32. This supports the above conclusion (unpublished observation).

In a study of acute paralytic poliomyelitis in immunised and non-immunised children from India, the authors noted that only type 1 virus infection resulted in bulbar or bulbospinal disease in both groups, and that 28 % of children in the immunised group had severe paralysis.[10] According to the WHO poliomyelitis type 1 predominates in communities with low immunisation coverage and types 2 and 3 in areas with high immunisation coverage.[11] Our four cases were due to type 1 poliovirus. Failure of OPV in preventing acute paralytic poliomyelitis has been reported from Saudi Arabia previously,[4] and from Israel where an outbreak of 15 cases of paralytic poliomyelitis due to type 1 poliovirus occurred during 1988. Nine of these had received full vaccination with OPV.[12] These reports and ours cast doubt on the efficacy of OPV in preventing poliomyelitis. Primary vaccination using IPV or a combination using IPV and OPV has been suggested.[13]

Recently in Finland an increased incidence of childhood GBS has been observed following a mass vaccination campaign against poliomyelitis using OPV. The authors conjecture that OPV may increase the occurrence of GBS in children.[14] It is possible that the clinical course of poliomyelitis is modified in children who have previously been vaccinated with OPV.

Plasmapheresis was considered in all our patients in view of the initial diagnosis of GBS. It has been suggested that plasmapheresis and/or high-dose

immunoglobulin therapy may improve the outcome in GBS.[8,15]. However, in the light of our observation, we caution that virological studies be carried out before one embarks on such expensive therapy.

In summary, we present four cases of paralytic poliomyelitis due to type 1 infection, who had been diagnosed initially as GBS. All patients had received primary vaccination against poliomyelitis using OPV. We conclude that acute paralytic poliomyelitis can present as symmetrical ascending paralysis and that the CSF changes may simulate those seen in GBS. Detailed virological studies are warranted in patients with a diagnosis of GBS in the presence of unusual features.

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References

1. Schoub BD. Poliomyelitis. *Med Int* 1984; **2**: 91–96.
2. Christie AB. *Infectious diseases. Epidemiology and clinical practice*. 3rd ed. Edinburgh: Churchill Livingstone, 1980: 572–604.
3. Nofal HS. Paralytic poliomyelitis in Saudi Arabia: a retrospective study on incidence and control. Proceedings of an International symposium on immunization against childhood diseases. Smith Kline-RIT and Ministry of Health, Saudi Arabia. 1981; 59–69.
4. Ramia S, Bakir TMF, Al-Frayh AR, Bahakim HM. Paralytic poliomyelitis and non-polio enteroviruses in Saudi Arabia. *J Trop Pediatr* 1987; **33**: 166–167.
5. Lwoff A, Lwoff M. Remarques sur quelques caractères du développement du virus de la poliomyélite. *Compt Rend* 1959; **248**: 248–250.
6. Asbury AK. Diagnostic considerations in Guillain–Barré syndrome. *Ann Neurol* 1981; **9** (Suppl): 1–5.
7. Evans OB. Guillain–Barré syndrome in children. *Pediatr Rev* 1986; **8**: 69–74.
8. Kleyweg RP, Van Der Meche FGA, Loonen MCB, Jonge JDE, Knip B. The natural history of the Guillain–Barré syndrome in 18 children and 50 adults. *J Neurol Neurosurg Psychiatr* 1989; **52**: 853–856.
9. Witte JJ, Henderson DA. The cerebrospinal fluid in type 3 poliomyelitis. *Am J Epidemiol* 1966; **83**: 189–195.
10. Sen S, Sharma D, Santhanam S. Clinical and serological study of acute paralytic poliomyelitis in immunized and nonimmunized children. *Indian Pediatr* 1985; **22**: 891–897.
11. Assaad F, Ljungars-Esteves K. World overview of poliomyelitis: regional patterns and trends. *Rev Infect Dis* 1984; **6**: 302–307.
12. Slater PE, Orenstein WA, Morag A *et al*. Poliomyelitis outbreak in Israel in 1988: a report with two commentaries. *Lancet* 1990; **335**: 1192–1198.
13. Beale AJ. Polio vaccines: time for a change in immunisation policy? *Lancet* 1990; **335**: 839–842.
14. Uhari M, Rantala H, Niemela M. Cluster of childhood Guillain–Barré cases after an oral poliovaccine campaign. *Lancet* (Letter) 1989; **2**: 440–441.
15. The Guillain–Barré syndrome study group. Plasmapheresis and acute Guillain–Barré syndrome. *Neurology* 1985; **35**: 1096–1104.