Final diagnosis**Keywords:** aneurysm, aorta, computed tomography

Para-anastomotic aortic false aneurysm with extension into the left psoas muscle and consequent compression of the lumbar plexus nerves.

- 1 Haiart DC, Callam MJ, Murie JA, Ruckley CV, Jenkins AM. Reoperations for late complications following abdominal aortic operations. *Br J Surg* 1991; 78: 204–6.
- 2 Allen RC, Schneider J, Longenecker L, Smith RB, Lumsden AB. Paraanastomotic aneurysms of the abdominal aorta. *J Vasc Surg* 1993; 18: 424–31.

- 3 Hilton S, Megibow AJ, Naidich DP, Bosniak MA. Computed tomography of the postoperative abdominal aorta. *Radiology* 1982; 145: 403–7.
- 4 Mark A, Moss AA, Lusby R, Kaiser JA. CT evaluation of abdominal aortic surgery. *Radiology* 1982; 145: 409–14.

Acute paralytic illness

D Kidd, H Manji, D Brown, RS Howard

A 21-year-old man presented with the sudden onset of weakness of the left leg, low back pain and tingling around the lower abdomen. On the following day he developed fever, severe generalised headache, photophobia, breathlessness and urinary hesitancy. One week later he developed dyspnoea, limb weakness, difficulty sitting up and had generalised paraesthesiae, particularly affecting his hands, feet and periorbital regions. He was admitted to hospital and at that time it was noted that he was unable to lift his leg off the bed and had severe difficulty in walking. Cerebrospinal fluid (CSF) protein was 0.2 g/l, white cell count 80 cells/ml (100% lymphocytes). A diagnosis of viral meningitis was made and he was treated with intravenous Acyclovir.

Over the course of the next six weeks the situation remained unchanged; his breathlessness continued, particularly lying flat, and there was weakness of the right arm and left leg. On examination, he walked with a left Trendelenberg gait and right shoulder droop. The cranial nerves were normal. In the upper limbs there was wasting of right triceps, spinati and deltoid. There was severe weakness of these muscles and moderate weakness of biceps, brachioradialis and triceps, and mild distal weakness. The left arm was normal. In the lower limbs there was wasting of the left quadriceps, with severe weakness of abduction, adduction and extension of the left hip and moderate weakness of knee flexion, ankle dorsiflexion, inversion and eversion. The left ankle reflex was absent and the plantar responses were flexor. Sensation was normal. Forced vital capacity was 6.2 l erect and 4.9 l lying; diaphragmatic screening confirmed evidence of mild diaphragmatic weakness.

It was noted that he had never received vaccination against polio in childhood. His son who was seven months old had received two doses of DPT and polio vaccinations, the second of which had been two weeks before the onset of his father's illness. He was in the habit of changing the child's nappy.

Questions

- 1 What is the most likely diagnosis?
- 2 What four further investigations should be performed?
- 3 What is the probable outcome?

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Answers

QUESTION 1

The history is one of acute meningitis followed a short time later by the development of an acute flaccid paralysis due to dysfunction of the lower motor neuron. This is characteristic of acute paralytic poliomyelitis. The differential diagnosis is set out in box 1. The occurrence of sensory symptoms (but no sensory signs) is well known.

QUESTION 2

Blood tests

Blood tests should be carried out to investigate possible sources of infection, vasculitis or autoimmune disease. Full blood count was within normal limits; erythrocyte sedimentation rate 4 mm/first hour. Electrolytes, renal, bone and liver profiles normal. Creatine kinase 19 (24–195) IU/l, plasma protein electrophoresis and immunoglobulin profile normal, thyroid function normal, antibody screen normal. Acetylcholine receptor and anti-GM1 antibodies negative.

Spinal fluid examination

His CSF protein was 0.86 g/l, white cell count 7 cells/ml (100% lymphocytes).

Nerve conduction studies and electromyography

Nerve conduction velocities were normal but F wave latencies were delayed or absent. Electromyography showed evidence in all muscles sampled of denervation and reinnervation (polyphasia and large amplitude positive sharp waves).

Serological tests

Serological tests for recent infection with *Borrelia burgdorferi*, *Mycoplasma pneumoniae*, influenza A and B, adenoviruses, herpes simplex, varicella zoster, cytomegalovirus, Epstein–Barr virus, mumps and measles were negative. Acute and convalescent sera and CSF were all negative (titre <8) for poliovirus 1, 2 and 3 antibodies by serum neutralisation.¹ Coxsackie B IgM titre was detected in both sera by ELISA,² indicating a recent infection.

QUESTION 3

Acute paralytic illnesses due to non-polio virus enteroviruses usually follow a benign course and have an excellent prognosis for recovery. This is mainly due to the low incidence of bulbar weakness and autonomic complications. Infection due to enterovirus (EV) 70 and 71, however, tends to be more severe, and recovery may be incomplete. In acute paralytic poliomyelitis the mortality is much greater and the incidence of residual neurological impairment is higher.³

Clinical course

Slow spontaneous improvement occurred over the next 12 months. After six months there remained wasting of the deltoid and right quadriceps, and mild weakness of those mus-

Acute flaccid paralysis: differential diagnosis

- acute paralytic poliomyelitis
- vaccine-associated poliomyelitis
- acute polio-like illness due to other enteroviruses
- Guillain–Barré syndrome
- *Mycoplasma pneumoniae* or Epstein–Barr virus infection
- Lyme disease
- acute intermittent porphyria
- HIV neuropathy
- diphtheritic neuropathy
- botulism
- triorthocresyl phosphate poisoning
- heavy metal poisoning

cles as well as left hip flexion and abduction; after 12 months the arms were normal and there was only mild weakness of hip flexion. He is now back at work as a maintenance engineer and is able to run. He has no symptoms.

Discussion

The clinical course of a meningitic illness with asymmetrical flaccid paralysis and the electrophysiological findings of denervation with reinnervation (polyphasia and large amplitude positive sharp waves) with delayed or absent F-wave latencies but normal peripheral conduction velocities is typical of a polio-like paralytic illness.^{4,5}

The incidence of paralytic poliomyelitis has been reduced dramatically since the advent of mass vaccination programmes using oral polio vaccine.⁶ In England and Wales over the five years beginning 1985 only 21 cases were reported⁷; the majority of these infections arose either in adults who had not been vaccinated and had come into contact with infants excreting live attenuated vaccine, or in vaccine recipients themselves.^{8–10}

It has also been recognised that a paralytic illness which closely resembles poliomyelitis clinically may develop in association with other enterovirus infections such as Coxsackie virus and Echo virus^{11–14} and EV 70^{15,16} and 71.¹⁷ Between 1976 and 1979 in the US over 50% of all reported cases of paralytic diseases were due to non-polio enterovirus infections.¹⁸

Paralysis due to Coxsackie and Echo viruses tends to be less severe than that due to polio, and bulbar involvement, which has an important association with mortality, is rare.¹⁹ Epidemics of paralysis due to Coxsackie A7 occurred in Russia in the 1950s and in Scotland in 1959 and 1963.¹⁴ Infections due to A9, B1, B2 and B3 as well as numerous Echo viruses have also been known to cause paralytic illness.¹⁴ Paralytic illnesses due to EV 70 and 71 are important since they have been responsible for widespread epidemics in recent years; EV 70 infection is associated with acute haemorrhagic conjunctivitis,^{15,20} and epidemics have occurred in India and Japan, while large epidemics of paralysis due to EV 71 occurred in Bulgaria and Hungary in the 1970s.²¹ These illnesses are more severe and more closely

resemble infection with poliomyelitis. In one series, residual neurological impairment existed in two-thirds of sufferers several years after the acute illness.¹⁵

Coxsackie and Echo viruses appear to cause aseptic meningitis or meningoencephalitis more commonly than paralytic illness; these viruses are thought to enter the central nervous system via the choroid plexuses. EV 70 and 71, in contrast, are thought to enter by a similar mechanism to polio viruses, namely, fast axonal transport from a single site of entry,¹⁹ and this may explain why the paralytic illnesses caused by EV 70 and 71 are more commonly associated with paralytic illness and why this disease tends to be more severe and more frequently associated with residual neurological impairment.

Virological confirmation of an enterovirus infection is best accomplished by virus detection, either by isolation or the polymerase chain reaction from a throat swab, CSF or

stool samples. Serological diagnosis is more problematic since there are more than 60 related viruses which induce cross-reactive immune responses. In this case we were able to exclude poliovirus infection. The Coxsackie B virus IgM response detected indicates a recent enterovirus infection although it is not possible to determine the infecting serotype because of the extensive cross-reactions seen in these types.

This case emphasises that enterovirus infection may occasionally mimic acute paralytic poliomyelitis and highlights the importance of appropriate early virological investigation.⁷

Final diagnosis

Acute paralytic illness resembling poliomyelitis due to enterovirus infection.

Keywords: acute flaccid paralysis, polio, enterovirus

- 1 Day C, Cumming H, Walker J. Enterovirus specific IgM in the diagnosis of meningitis. *J Infect* 1989; **19**: 219–28.
- 2 White PMB, Green J. Prevalence of antibody to poliovirus in England and Wales. *BMJ* 1986; **293**: 1153–5.
- 3 Kidd D, Williams AJ, Howard RS. Classic diseases revisited: poliomyelitis. *Postgrad Med J* 1996; **72**: 641–7.
- 4 Weichers DO, Hubbell SL. Late changes in the motor unit after acute poliomyelitis. *Muscle Nerve* 1981; **4**: 524–8.
- 5 So YT, Olney RK. AAEM case report no. 23: acute paralytic poliomyelitis. *Muscle Nerve* 1991; **14**: 1159–64.
- 6 Hull HF, Ward NA, Hull BP, Milsten JB, de Quadros C. Paralytic poliomyelitis: seasoned strategies, disappearing disease. *Lancet* 1994; **343**: 1331–7.
- 7 Jock R, Wood D, Brown D, Begg N. Paralytic poliomyelitis in England and Wales 1985–1991. *BMJ* 1992; **305**: 78–82.
- 8 Nkowane BM, Wassilaki SGF, Orenski WA, et al. Vaccine-associated paralytic poliomyelitis; United States 1973 through 1984. *JAMA* 1987; **217**: 1335–40.
- 9 Arlazoroff A, Bleicher Z, Klein C, et al. Vaccine-associated contact paralytic poliomyelitis with atypical neurological presentation. *Acta Neurol Scand* 1987; **76**: 210–4.
- 10 Querfurth H, Swanson PD. Vaccine-associated paralytic poliomyelitis: regional case series and review. *Arch Neurol* 1990; **47**: 541–4.
- 11 Grist NR. Isolation of Coxsackie A7 virus in Scotland. *Lancet* 1960; **i**: 1054–5.
- 12 Steigman AJ, Lipton MM. Fatal bulbospinal paralytic poliomyelitis due to Echo 11 virus. *JAMA* 1960; **174**: 178–9.
- 13 Gear JH. Nonpolio causes of polio-like paralytic syndromes. *Rev Infect Dis* 1984; **6**: S379–84.
- 14 Grist NR, Bell EJ. Paralytic poliomyelitis and nonpolio viruses: studies in Scotland. *Rev Infect Dis* 1984; **6**: S385–6.
- 15 Wadia NH, Wadia PN, Katrak SM, Misra VP. Neurological complications of acute haemorrhagic conjunctivitis. *Lancet* 1981; **ii**: 528–9.
- 16 Katiyar BC, Misra S, Singh RB, et al. Adult polio-like syndromes following enterovirus 70 conjunctivitis. *Acta Neurol Scand* 1983; **67**: 263–74.
- 17 Melnick JL. Enterovirus type 71 infections: a varied clinical pattern sometimes mimicking paralytic poliomyelitis. *Rev Infect Dis* 1984; **6**: S387–90.
- 18 Centers for Disease Control. *Enterovirus surveillance report 1970–1979*. Atlanta, Georgia: Centers for Disease Control, 1981.
- 19 Jubelt B, Lipton HL. Enterovirus infections. In: Vinken PJ, Bruyn GW, Klawns HL, McKendall RR, eds. *Handbook of clinical neurology* revised series, vol 56: Viral disease. Amsterdam: Elsevier Science Publishers BV, 1989; pp 319, 328–9.
- 20 Wadia NH, Urani PF, Katrak SM. Neurological complications of a new conjunctivitis. *Lancet* 1972; **ii**: 970–1.
- 21 Chumakov M, Voroshilova M, Shindarov L, et al. Enterovirus 71 isolated from cases of epidemic poliomyelitis-like disease in Bulgaria. *Arch Virol* 1979; **60**: 329–40.