Final diagnosis

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

Keywords: aneurysm, aorta, computed tomography


Acute paralytic illness

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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?
**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 immunoglobin 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 electromyelography**

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 Coxackie 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-
resemble infection with poliomyelitis. In one series, residual neurological impairment existed in two-thirds of sufferers several years after the acute illness.\textsuperscript{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,\textsuperscript{19} and this may explain why the paralytical 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.\textsuperscript{7}

**Final diagnosis**

Acute paralytic illness resembling poliomyelitis due to enterovirus infection.

**Keywords:** acute flaccid paralysis, polio, enterovirus

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