

Guillain-Barré syndrome following meningococcal meningitis

Vinod Puri, Anita Khalil, Vineet Suri

Summary

A case of Guillain-Barré syndrome following meningococcal meningitis is reported. The diagnosis was made on clinical grounds and the results of electrophysiological studies. The patient recovered spontaneously. Guillain-Barré syndrome following meningococcal infection has not to our knowledge been reported previously.

Keywords: Guillain-Barré syndrome, meningococcal meningitis

Introduction

Meningococcal meningitis accounts for up to 40% of all cases of purulent meningitis.¹ The neurological complications of this variety of meningitis are various (see box).²⁻⁵ About two-thirds of patients with Guillain-Barré syndrome (GBS) have a preceding infection or an antecedent event a few weeks prior to neuropathy. There are scattered reports of GBS associated with Gram-negative bacterial infections.⁶ To the best of our knowledge there is no published report of GBS after meningococcal infection. We now report such a case.

Case

An 11-year-old boy was referred with fever, headache, vomiting and altered sensorium of one day's duration. By the same evening the patient was unconscious. The next morning he had an erythematous maculopapular rash on the right upper limb, trunk and both thighs. On examination he had grade II coma, blood pressure 100/50 mmHg, pulse 110 beats/min. He had no cranial nerve palsy. Both fundi were normal. Plantar reflexes were flexor and deep tendon jerks normally elicitable on both sides.

Neurological complications of meningococcal meningitis

- encephalitis
- encephalomyelitis
- cranial nerve palsies
- subarachnoid haemorrhage
- acute cerebellar ataxia
- causalgia
- conus medularis

He had neck stiffness and a positive Kernig sign. Biochemical investigation revealed sodium 118 mmol/l, potassium 3.5 mmol/l and normal blood sugar, urea and creatinine, haemoglobin was 11 g/dl, total blood count $18 \times 10^9/l$ with 80% polymorphs. His cerebrospinal fluid (CSF) was turbid with protein 1.8 g/l, sugar 1.38 mmol/l (corresponding blood sugar 4.4 mmol/l) with $0.91 \times 10^9/l$ polymorphs. On Gram staining intra- and extracellular Gram negative diplococci were seen. CSF and blood culture grew *Neisseria meningitidis*. He received parenteral crystalline penicillin (18 million units/day) and chloramphenicol (4 g/day) for 10 days. Repeat CSF on the third day of treatment showed protein 0.8 g/l, sugar 2.22 mmol/l, $0.03 \times 10^9/l$ polymorphs, and was negative for any growth on culture. He had an unremarkable recovery until the tenth day of illness when he had difficulty in moving his legs in bed. Power in the lower limbs was grade 4/5 with diminished knee and ankle jerks. Plantars were flexor and there was no sensory deficit. After another three days he could move his upper limbs but not his legs. There was no respiratory involvement. Neurological examination revealed hypotonia in all four extremities with power grade 2/5 in lower and 4/5 in upper limbs. Plantars were flexor and deep tendon jerks not elicitable. There was no sensory impairment. Cranial nerves were normal. There was no sign of meningeal irritation. His haemogram, biochemical profile for blood sugar, urea and serum electrolytes were non-contributory. CSF examination revealed protein 1.17 g/l, sugar 2.66 mmol/l with 14 cells/cc (all lymphocytes). Blood levels of immunoglobulins and antinuclear factor were normal. On electrophysiology, sensory nerve conduction studies as well as needle electromyogram were normal. The motor nerve conduction studies (table) revealed conduction block, prolonged distal latencies, delayed conduction and prolonged F-wave latencies. The patient started spontaneously improving after about two weeks and was able to walk unaided in another five weeks.

Discussion

The development of peripheral neuropathy after 10 days of meningococcal infection in the present case may have been drug-induced, due to critically ill polyneuropathy, meningococcal-related, or mere coincidence. Penicillin

Department of
Neurology and
Pediatrics, GB Pant
Hospital, Maulana
Azad Medical College,
New Delhi 110002,
India
V Puri
A Khalil
V Suri

Correspondence to:
Vinod Puri, 104-B, LIG
Flats, Mayapuri, New
Delhi-110064, India

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Table Electrophysiological data for motor nerves

Nerve studied		Distal latency (ms)	Amplitude of CMAP on distal/proximal stimulation (mv)	Conduction velocity (m/s)	Minimum F-wave response latency (ms)
Upper limb (wrist to elbow)					
Right	Median	4.2 (3.31 ± 0.4)	4.6/3.8 (8.0 ± 4.6)	36.0 (57.6 ± 4.8)	48.2 (22.1 ± 2.3)
	Ulnar	5.6 (2.58 ± 0.43)	2.12/1.59 (6.14 ± 1.90)	25.0 (61.8 ± 5.0)	46.0 (21.8 ± 1.2)
Left	Median	6.2 (3.31 ± 0.38)	3.8/3.1 (8.0 ± 4.6)	31.0 (57.6 ± 4.6)	58.0 (22.1 ± 2.3)
	Ulnar	7.2 (2.56 ± 0.43)	1.52/0.89 (6.14 ± 2.0)	28.0 (60.3 ± 5.0)	50.2 (21.8 ± 1.1)
Lower limb (ankle to knee)					
Right	Common peroneal	8.4 (3.77 ± 0.86)	1.38/0.64 (5.1 ± 2.3)	37.5 (48.3 ± 3.9)	56.8 (51.3 ± 4.7)
	Posterior tibial	8.7 (3.96 ± 1.0)	1.29/0.29 (5.8 ± 1.9)	27.6 (48.5 ± 3.6)	54.8 (52.3 ± 4.8)
Left	Common peroneal	9.0 (3.77 ± 0.84)	2.1/0.76 (5.1 ± 2.2)	32.0 (48.3 ± 3.9)	53.2 (51.3 ± 4.7)
	Posterior tibial	8.4 (3.98 ± 1.0)	1.10/0.29 (5.8 ± 1.9)	32.3 (48.5 ± 3.4)	62.1 (52.3 ± 4.7)

Figures in parentheses are ranges.

can cause mononeuropathy or brachial plexopathy. However, chloramphenicol produces painful sensorimotor polyneuropathy usually accompanied by optic neuropathy and only after prolonged and high dosage.⁸ Although both penicillin and chloramphenicol were used in this case, the pattern of neuropathy was quite different from the ones described with these drugs.

Critically ill polyneuropathy is a sensorimotor polyneuropathy with an onset in the fifth decade and occurrence at the peak of critical illness.⁹ The electrophysiological characteristics of this type of neuropathy are of the axonal type, ie, reduced amplitude of compound motor and sensory nerve action potential and near normal motor and sensory conduction velocities, along with normal F-wave latency and denervation pattern on needle electromyogram. The patient was seriously ill but did not meet the criteria, either clinical or electrophysiological, described for critically ill polyneuropathy.

The electrophysiological findings in this case were as described for peripheral demyelinating neuropathy.¹⁰ Up to 90% of individuals with GBS may have some electrophysiological abnormality within the first two weeks of illness, eg, partial conduction block or decreased amplitude of compound motor action potential or both (75%), prolonged distal latency (33%), slowed conduction velocity (20%), and temporal dispersion (20%), while by three weeks 96% have abnor-

malities. Abnormal F response either absent or prolonged, reflecting involvement of proximal nerve segment may occur in up to 46% of patients studied in the first month.¹¹ Pathophysiological correlations in GBS have revealed conduction block as an early abnormality, usually occurring before slowing of nerve conduction.¹² The clinical weakness is directly related to the number of nerve fibres showing conduction block. The slowed conduction velocity which is thought to be a feature of demyelination may perhaps be due to remyelination. In GBS, especially during the first week of illness when the demyelinating process is at a peak, the conduction velocity may be within normal limits, whereas in hereditary and chronic demyelinating neuropathies when the clinical weakness is improving the conduction velocity may be markedly slowed. Our patient had both clinical weakness and altered electrophysiology of motor nerves. The clinical profile was that described for GBS.¹¹

Various meningococcal-induced autoimmune disorders have multi-organ immune complex deposition.¹³ The occurrence of peripheral neuropathy within two weeks of meningococcal infection points toward there being a relationship between these events rather than a mere coincidence. This latent period is perhaps due to the time taken for the immune process initiation and manifestation. Thus, this patient appears unique in having GBS following meningococcal meningitis.

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