Case Report

Guillain-Barré syndrome associated with *Mycoplasma pneumoniae* infection: a case report

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Abstract
Guillain-Barré syndrome (GBS) is an immune mediated polyradiculoneuropathy. GBS manifests as a rapidly evolving areflexic ascending type flaccid motor paralysis. Mycoplasma infection is known to cause GBS, but serologically confirmed cases have not been reported in Sri Lanka. We report a case of Guillain-Barré syndrome presenting in association with *Mycoplasma pneumoniae* infection in a 27-year-old previously healthy male.

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Introduction

Guillain-Barré syndrome (GBS) is described in association with antecedent events like viral, bacterial, mycoplasma infections or vaccinations. We describe a case of Guillain-Barré syndrome presenting in association with *M. pneumoniae* infection.

Case presentation

A 27 year old male presented with mild intermittent fever and a dry cough with whitish sputum for one week. He also had malaise and headache. He developed lower limb weakness on the seventh day of illness. The weakness progressed rapidly during the day to involve the whole of lower limbs. But upper limbs were not involved. There was no bladder or bowel involvement or difficulty in breathing. He did not have postural dizziness or palpitations. There was no significant past medical history of note. The illness was not preceded by vaccination or diarrheal illness.

On examination, he was febrile but did not have dyspnoea. His pharynx was erythematous with no exudates. He was mildly icteric but not pale. There were no skin rashes. His lungs were clear with good respiratory effort. His cranial nerves were intact and upper limb examination was unremarkable. Lower limb muscle power was reduced (3/5) in both proximal and distal muscle groups with absent deep tendon reflexes of knees and ankles. His sensory and cerebellar system examination were normal.

His investigations on admission showed a normal full blood count and a normal chest X-ray. His ESR was 40 mm in the first hour. There was indirect hyperbilirubinemia [total bilirubin 40 micromole per liter (normal 3-20µmol/l) with an indirect fraction of 27 [normal < 12µmol/l] with mildly elevated liver enzymes [AST 92 IU/l (10-40 IU/l), ALT 154 IU/l (7-56 IU/l) respectively] with a reticulocyte count of 4 percent. Titer of his serum for *M. pneumonia* specific IgM was 1:360 on presentation (normal < 1: 40). His blood picture did not reveal any cold agglutinins but direct coombs test was positive. Nerve conduction studies done on day 5 reveled demyelinating type of polyneuropathy in lower limbs with reduced amplitude of compound muscle action potentials (CMAPs) and elevated distal motor latencies with prolonged f waves. Cerebrospinal fluid on day 10 reveled mildly elevated proteins [80 mg/dl (normal 20-40mg/dl)] with 2 white cells (100 percent being lymphocytes) and normal sugar levels. The diagnosis of GBS associated with recent *M. pneumoniae* infection was made.

He was treated with Intravenous high dose immunoglobulin administration from day one with physiotherapy and intravenous clarithromycin. His lower limb weakness started resolving and he could walk with assistance on day nine. Two months after the onset of his symptoms, there were no residual neurological abnormalities, and his *M. pneumoniae* specific IgM titer dropped to 1:80 after one month.

Discussion

The Guillain-Barré syndrome is characterized by acute areflexic paralysis with albumin cytologic dissociation. The pathological spectrum of GBS consists of acute inflammatory demyelinating polyneuropathy (AIDP), acute motor axonal neuropathy (AMAN) and acute motor sensory axonal neuropathy (AMSAN) and Miller Fisher variant.

After eradication of poliomyelitis, GBS is the commonest cause of acute flaccid paralysis in Sri Lanka. The exact incidence of GBS in Sri Lanka and many South Asian countries is not known. Seasonal variation of GBS is also observed by some clinicians in Sri Lanka (1).

Antecedent events such as infections precede GBS by 1 to 3 weeks. Examples of such infections are due to viruses like influenza, human herpes virus, cytomegalovirus or Epstein-Barr virus. Among bacterial infections *Campylobacter* and *M. pneumoniae* infections are known to cause GBS. Vaccines like Influenza, older-type rabies vaccine precedes 50%-75% of the GBS cases. *Campylobacter* infection is known as the single most identifiable antecedent infection associated with the development of GBS (2). *M. pneumoniae* infections occurred more often in GBS patients (5%) than in controls (3).

Our patient presented with upper respiratory tract infection along with jaundice and Guillain-Barré syndrome. *M. pneumoniae* was confirmed by a high titer of IgM antibodies. The titer of IgM more than 1:128 is considered positive (4,5). Co-existing GBS was confirmed by CSF analysis and nerve conduction studies. The presence of reduced amplitude of compound muscle action potentials (CMAPs) and elevated distal motor latencies with prolonged f waves was compatible with demylinating type of neuropathy. Guillain-Barré syndrome associated with *M. pneumoniae* infection include both demyelinating and axonal neuropathies (6,7). Central nervous system manifestations of *Mycoplasma* infection has been studied in Sri Lankan paediatric age group (7) but GBS with confirmed *M. pneumoniae* infections have not been reported previously.

In conclusion, when a patient presents with ascending limb paralysis with cough and fever, GBS caused by *M. pneumoniae* should first be considered. This infection can be effectively treated with macrolide group of antibiotics. This is particularly important because if the patient goes into respiratory paralysis with GBS, the co-existing lung infection could lead to a poor prognosis.

Consent

Written informed consent was obtained from the patient for publication of this case report. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

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