CASE REPORT

A rare case of Guillain-Barré syndrome presenting with abdominal pain

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Abstract

Background: Guillain-Barré syndrome (GBS) is a heterogeneous condition that encompasses acute immune-mediated polyneuropathies. GBS is the most frequent cause of acute neuromuscular paralysis worldwide and constitutes one of the most serious emergencies in neurology.

Description of case: As it presents extremely rarely with the first symptom being abdominal pain, herein we report the case of a 48-year-old man who presented with acute abdominal pain and diagnosed with GBS. The patient required mechanical ventilation for two weeks and was discharged one month later, after having had a tracheostomy and developed tetraplegia.

Conclusion: GBS should be included in the differential diagnosis of acute abdominal pain when other medical or surgical causes have been excluded. Hippokratia 2015; 19 (4): 374-375.

Keywords: Guillain-Barré, abdominal pain, mechanical ventilation

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Introduction

Guillain-Barré syndrome (GBS), an important cause of acute neuromuscular paralysis[1], is a heterogeneous condition with several variant forms (acute immune-mediated polyneuropathies). The most common forms are the acute inflammatory demyelinating polyneuropathy (AIDP), the acute motor axonal neuropathy (AMAN), the acute sensorimotor axonal neuropathy (AMSAN), and the Miller-Fisher syndrome (MFS)[2]. The common characteristic is the rapidly progressive polyneuropathy, usually provoked by a preceding respiratory or gastrointestinal infection. The most frequent antecedent infection is *Campylobacter jejuni* infection, which is associated with 30% of cases of GBS[3]. One plausible underlying mechanism is that the antecedent infection causes an immune response, which successively cross-reacts with peripheral nerve components because of molecular mimicry[3].

In the present case report, a 48-year-old man developed GBS, ten days following a respiratory tract infection. The patient's initial symptom and main complaint was acute abdominal pain.

Case report

A 48-year-old man presented to the accident and emergency department (A&E) complaining of acute abdominal pain which was characterized by exacerbations and remissions, and nausea. The patient was able bodied. He was not a smoker neither mentioned any previous health problems. On examination, the chest was clear, but the lower half of his abdomen was tender to deep palpation. The patient did not have a neurologic examination upon arrival to the A&E, since the primary focus at the time was his acute abdominal pain. Blood pressure was 140/90 mmHg, and heart and respiratory rate were 95 and 16/min, respectively. Temperature, SpO_2, and blood gasses were normal. Laboratory tests showed a mild leukocytosis with polymorphonuclear type. Erythrocyte sedimentation rate and C-reactive protein were within normal limits. Blood glucose, renal and liver function tests, and urine examination were normal. Blood and urine amylase were negative for pathological findings. Electrocardiography, chest x-ray, ultrasound and computed tomography (CT) of the abdomen were normal. The CT angiogram that was performed to exclude a partition aneurysm was also normal. The patient was admitted to the surgical department for further investigation of his abdominal pain.

On the day after admission, the patient developed weakness and inability to walk and stand. On clinical examination, he looked frail, had tachypnea and was heavily perspiring. His consciousness level was excellent. The upper extremities were normal, but there was numbness and severe weakness in both lower extremities while tendon reflexes were not present. The patient was unable to walk unaided. When asked about any recent infections, he stated that ten days before he had an upper respiratory tract infection, and fever up to 38.5°C that lasted 48

hours. The head CT was normal, and the lumbar puncture was negative for the presence of either protein (35 mg/dl) or cells (3/mm^3). The likelihood of GBS was raised.

The patient was urgently transferred to the neurology department of a tertiary hospital in Athens. During the transport, he presented weakness of both upper extremities and urinary retention. Within a few hours after his arrival, he developed acute breathing difficulty, was intubated, and required mechanical ventilation for two weeks. The further diagnostic tests (repeat lumbar puncture, nerve conduction studies, electromyography) performed corroborated the diagnosis of GBS (acute axonal neuropathy; consistent with the rapid clinical deterioration of the patient).

The patient was treated with a combination of plasma exchange followed by a course of intravenous immune globulin (IVIG). During his hospitalization, he had a tracheostomy and developed tetraplegia. He was discharged one month later and had to remain in a rehabilitation center for four months.

Discussion

The reported incidence of GBS in Western countries ranges from 0.89 to 1.89 cases per 100,000 person-years, although an increase of approximately 20% is observed with every 10-year raise in age after the first decade of life[3]. Males are 1.8-times more prone to be affected than females[3].

The main clinical features of GBS are progressive, mostly symmetric muscle weakness and absent or depressed deep tendon reflexes[4]. The weakness can vary from mild difficulty when walking to nearly complete paralysis of all extremity, facial, bulbar, and respiratory muscles[4]. Ten to 30% of the patients develop severe respiratory muscle weakness that requires ventilatory support[5], as was the case in our patient. In addition, severe autonomic dysfunction occurs in about 20% of the cases warranting intensive care unit monitoring, while it may occasionally be associated with sudden death[6].

The therapeutic mainstays for GBS are the administration of IVIG and plasmapheresis. The treatments are equivalent and improve outcome[7].

Pain, in the form of dysesthesia or muscular, radicular, arthralgic, and meningitic pain, precedes weakness in one third of patients with GBS[2,8]. Furthermore, pain, usually back and extremities pain, can be the primary presenting symptom and is reported by up to 66% of patients with GBS[8,9]. The origin of pain is postulated to be multifactorial[1]. Pain in the acute phase of GBS might be of nociceptive origin due to inflammation, whereas later in the course of the disease, non-nociceptive neuropathic pain could result from degeneration and maybe regeneration of sensory nerve fibers[1]. Simple analgesics or nonsteroidal anti-inflammatory drugs (NSAIDS) may be administered, but often do not provide adequate pain relief. Alternatively one can utilize appropriate narcotic analgesics, but these require careful monitoring[8,9]. Although abdominal pain is a common complaint in the A&E because of surgical and non-surgical (e.g. diabetic ketoacidosis, Henoch-Schönlein purpura, etc.) conditions, in only extremely rare cases GBS presents with abdominal pain as the first symptom[10]. However, one should also consider the differential diagnosis of peripheral neuropathy of acute onset and abdominal pain[11]. In this context, the following pathologic conditions might be included: hereditary diseases such as acute intermittent porphyria (history of precedent attacks) and Fabry disease (only in the early age spectrum), poisoning with heavy metals (arsenic, lead), toxins (ethylene glycol), and drugs (thallium, fluoroquinolones). Lastly, in the differential diagnosis various infections should also be considered, such as Lyme disease (neuroborreliosis), poliovirus, and West Nile virus.

In conclusion, diagnosis of GBS should be included in the differential diagnosis of acute abdominal pain. This is, to our knowledge, the second case report ever reported.

Conflict of interest

The authors have nothing to disclose.

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