Abdominal Pain Followed by Altered Mental Status: A Rare Presentation of Guillain Barré Syndrome

Shekhar Shekhar 1, , Avi Harisingani 1, , Nikita Gupta 1

1. General Medicine, Grant Government Medical College and Sir J.J. Group of Hospitals, Mumbai, IND

Abstract

Guillain Barré syndrome (GBS) is an acute inflammatory polyneuropathy with several variants; it usually presents as acute symmetrical muscle weakness with or without bulbar involvement, making it a neurological emergency. In this report, we describe the case of a 39-year-old male who presented with abdominal pain for three days and whose illness became progressively drowsy on the fifth day. Based on clinical assessment, nerve conduction studies, and biochemical, microbiological, and radiological investigations, other causes were ruled out and it was concluded that the patient had hyponatraemia secondary to syndrome of inappropriate diuretic hormone secretion (SIADH) due to GBS.

Although sensory symptoms like pain or dysesthesias occurring in the back or extremities are common and may precede motor weakness, abdominal pain remains a very rare presentation of GBS. GBS is usually understood as a "pure" peripheral nervous system disorder but central nervous system (CNS) dysfunction may occur due to metabolic abnormalities (like hyponatraemia and CO₂ narcosis) or autonomic dysfunction in GBS, its treatment, or due to GBS itself (Anti-GQ1b disease variant).

Introduction

Guillain Barré syndrome (GBS) is a cause of acute weakness commonly following an infection. There are many variants of GBS, the most common being acute inflammatory demyelination polyneuropathy (AIDP). Other sensorimotor variants are acute motor axonal neuropathy (AMAN), acute motor sensory axonal neuropathy (AMSAN), pure sensory GBS, and Miller Fisher syndrome. MFS presents as ophthalmoplegia, ataxia, and areflexia but sometimes all the symptoms might not be present. Bickerstaff brainstem encephalitis is a type of MFS that includes ataxia, and ophthalmoplegia along with encephalopathy. Both MFS and Bickerstaff brainstem encephalitis have been associated with anti-GQ1b antibodies. Other rare variants include pharyngeal-cervical-brachial weakness, acute pandysautonomia (diarrhea, vomiting, abdominal pain, ileus, and urinary retention), acute bulbar palsy, facial diplegia, and distal limb paresthesia [1]. Our patient presented with abdominal pain followed by altered mental status. Back pain and lower limb pain are common in GBS whereas presentation of GBS with abdominal pain is extremely uncommon [2].

Case Presentation

The patient was a 39-year-old male, Indian Muslim, working as a taxi driver, who presented to the hospital with the chief complaint of abdominal pain for three days and was admitted under general surgery. He described stabbing, intermittent, non-radiating pain in the lower half of the abdomen (more so in the periumbilical region), lasting for minutes to hours, severe enough to wake him up at night, relieved on walking/moving about, without diarrhea, constipation, or vomiting. The patient had not had any fever or gastrointestinal or urogenital symptoms prior to this illness. On examination, he was vitally stable with tenderness in the periumbilical region on deep palpation with no guarding. A digital rectal examination was normal. There was no history of significant weight loss. He did not consume alcohol, tobacco, or recreational drugs. Complete blood count, blood sugar, erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP) were within normal limits. Serum sodium and potassium were 157 meq/L and 4.2 meq/L respectively. Renal function and liver function tests were within normal limits. Stool chemical examination was negative for occult blood. Stool microscopy did not show any parasitic forms, pus cells, red blood cells, eosinophils, or fat globules. Fecal calprotectin and elastase levels were within normal limits. Stool cultures sent on two subsequent days were negative. Ultrasonography of the abdomen did not reveal any abnormality. The patient was given inj. pantoprazole 40 mg I.V. OD, inj. Drotaverine 40 mg I.V. BD, and inj. DNS (5% dextrose + 0.9% normal saline) 500 cc I.V. O.D. during his stay at the general surgery. He had not been taking any medications prior to the admission and denied any comorbidities.

On day five of the illness and since the onset of abdominal pain (that is, day three of admission), the patient...
The presentation of GBS with abdominal pain is extremely uncommon. Abdominal pain can be due to cause radicular pain shooting into the limbs. Thirdly, small fiber involvement can also cause pain in GBS lower back and lower limbs. The mechanism of pain can be multifactorial. Firstly, inflammation of large occurred last in this case). GBS was found to be the underlying cause of his abdominal pain, hyponatremia, and weakness (that suggestive of lead or arsenic toxicity. Altered sensorium was attributed to hyponatremia due to SIADH. Once given the short clinical history. The patient did not have any history of exposure or examination findings niacin, Vitamin E), inflammatory bowel disease, celiac disease, and Whipple’s disease were highly unlikely other causes of abdominal pain with neurological symptoms and porphyria were ruled out as blood sugar, renal function test, and urine porphobilinogen were normal. Metabolic causes of abdominal pain with neurological symptoms like diabetic ketoacidosis, uremia, and porphyria were ruled out as blood sugar, renal function test, and urine porphobilinogen were normal. Other causes of abdominal pain with neurological symptoms [3] like nutritional deficiencies (thiamine, niacin, Vitamin E), inflammatory bowel disease, celiac disease, and Whipple’s disease were highly unlikely given the short clinical history. The patient did not have any history of exposure or examination findings suggestive of lead or arsenic toxicity. Altered sensorium was attributed to hyponatremia due to SIADH. Once sodium was corrected and the patient became oriented, his neurological symptoms were investigated, and GBS was found to be the underlying cause of his abdominal pain, hyponatremia, and weakness (that occurred last in this case).

One-third to two-thirds of patients with GBS experience pain in the acute phase, more frequently in the lower back and lower limbs. The mechanism of pain can be multifactorial. Firstly, inflammation of large myelinated nerve fibers can cause muscle pain in limbs with dysesthesia. Secondly, nerve root pain can cause radicular pain shooting into the limbs. Thirdly, small fiber involvement can also cause pain in GBS [4]. The presentation of GBS with abdominal pain is extremely uncommon. Abdominal pain can be due to sensory nerve inflammation, dorsal nerve root inflammation, or gastrointestinal autonomic dysfunction [2].
Since other autonomic symptoms and signs were not observed in our patient, it was unlikely that autonomic dysfunction was the cause of the abdominal pain.

Central nervous system (CNS) involvement is classically excluded from the spectrum of GBS. However, it is not uncommon for GBS to present with an altered mental state [5]. CNS dysfunction in GBS correlates with more severe disease and the chance of requiring assisted ventilation [6]. Altered mental status in GBS may be due to any of the following factors: neuropsychiatric manifestations of the disease itself, metabolic and autonomic derangements caused by the disease, treatment of the disease, or GQ1b-related disease variants.

Neuropsychiatric manifestations of GBS include vivid dreams, hallucinations, illusions, and delusions—mostly of the paranoid type. These have been found to occur in GBS patients even prior to ICU admission (and hence differing from ICU delirium) in a prospective controlled study [7].

Among metabolic derangements, hyponatremia due to SIADH is common in GBS and develops in roughly half of the patients. This is due to altered hypothalamic osmoreceptors and ADH sensitivity of the renal tubules, both of which could arise from immune-mediated damage [8]. CO₂ narcosis resulting from respiratory paralysis may also cause altered mental status.

Autonomic dysfunction is a characteristic feature of GBS and is seen in about two-thirds of all cases. This can cause CNS dysfunction by blood pressure fluctuation [5]. For example, severe hypertension can lead to posterior reversible encephalopathy syndrome (PRES), a condition that can result in seizures, altered mental status, and visual impairment. Orthostatic hypotension may lead to syncope.

Altered mental status can also result from the treatment of GBS. IVIG, a treatment for GBS, can cause adverse effects like thrombosis and aseptic meningitis. IVIG can also contribute to the development of PRES. Therefore, IVIG can also be a cause of CNS manifestations in GBS by these mechanisms [4]. It is important to note that IVIG causes pseudohyponatremia as serum osmolality remains normal.

Lastly, the GQ1b-related disease variant, namely Bickerstaff brainstem encephalitis, a condition closely related to the MFS variant of GBS, is a possible cause of altered mental state [9].

Conclusions

GBS should be considered in differentials of acute abdomen with neurological symptoms after excluding other possible causes (depending on specific case scenarios) like infective ones (enteric fever, Lyme disease), metabolic disorders (diabetic ketoacidosis, diabetic neuropathy, uremia, porphyria), heavy metal intoxication (lead, arsenic), gastrointestinal disorders (celiac disease, Whipple’s disease), and nutritional deficiencies (thiamine, niacin, vitamin E). Mental status abnormalities should prompt a search for alternative explanations but cannot rule out GBS.

Additional Information

Disclosures

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