Guillain-Barre Syndrome Presenting as Acute Abdomen

Akut Karın ile Gelen Guillain-Barre Sendromu

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ABSTRACT

Guillain-Barré syndrome (GBS) is the most common cause of acute flaccid paralysis in childhood. Symmetric weakness, headache, respiratory symptom, neuropathic pain, muscle pain, paresthesia, and facial palsy were the most common clinical presentations. We report a 13-year-old boy with GBS who presented with acute abdominal pain. This is the first report, to our knowledge, first presented of acute abdomen of a pediatric patient with GBS.

Key words: Guillain-Barré syndrome, acute abdomen, children

ÖZET


Anahtar kelimeler: Guillain-Barre Sendromu, akut karın, çocuklar

INTRODUCTION

Guillain-Barré syndrome (GBS) is an acute inflammatory polyneuropathy most commonly characterised by rapidly progressive, essentially symmetric weakness and areflexia. Its incidence has been reported 0.4-1.3 / 100,000¹.

Guillain-Barré syndrome has become the most common cause of acute flaccid paralysis in both the developed and developing countries. Guillain-Barré syndrome is frequently associated with a preceding illness, such as upper respiratory infection or acute enterocolitis². The most common symptom at onset in paediatric patients was limb weakness. Affected children also complain of headache, respiratory symptom, urination problem, neuropathic pain, muscle pain or soreness, abdominal pain, paresthesia, facial palsy and dysphagia¹. In literature, abdominal pain associated GBS has been reported in rare patients³,⁴. Here, we report a 13-year-old boy with acute abdomen as a presenting symptom of the GBS.

CASE REPORT

A previously healthy 13-year-old boy was hospitalized for progressive weakness for 5 days. Fifteen days before admission, he received the tetanus-diphtheria toxoid vaccine. Seven days later, the patient first presented to another hospital because of abdominal pain in the right side of his abdomen lasting one day. The pain was initially intermittent, and became worsened. He refused to
walk and has weakness in his legs after one day of abdominal pain started. His past medical and family history were not significant. No history of febrile illness and enterocolitis were reported during the previous month. There were no other symptoms. The patient was operated for acute abdomen by pediatric surgery in that hospital. But, the pathology report of the specimen (appendix) was normal. After the surgery, the patient was transferred to our hospital became flaccid paralysis and respiratory failure.

After admission to the Intensive Care Unit, the patient was entubated and ventilated. Vital signs were stabil. On neurological examination, the pupils reacted normally and there was no facial palsy. There was severe symmetric weakness in the all extremities (strength 0-1/5). There was dysesthesia. Tendon reflexes in the all extremities were absent. The remainder of the examination was unremarkable.

Serologic tests for C. jejuni, human immunodeficiency virus, Epstein-Barr virus, cytomegalovirus, hepatitis A-C virus, Chlamydia pneumoniae were negative. Antibodies of (IgM, G) Mycoplasma pneumoniae in serum were positive. Lumbar puncture was not performed. Nerve conduction and electromyographic examination revealed motor axonal damage. Spinal magnetic resonance imaging was normal.

On the basis of the clinical and laboratory findings, the child was diagnosed as GBS. Supportive treatment included mechanical ventilation, nasogastric tube feeding, and physiotherapy. Plasmapheresis (PE) for 5 days was performed and intravenous immunoglobulins (IVIG) 0.4 g/kg for another 5 days were administered. Clarithromycin treatment was started after serological test of Mycoplasma was positive. Despite treatment, the patient was tetraplegic and not spontaneous breathing.

DISCUSSION

Guillain-Barré syndrome is an acute inflammatory polyneuropathy, characterized by rapidly progressive, symmetrical flaccid paralysis and areflexia. Clinically, motor weakness and sensory loss begin in the lower extremities and progressively ascend to the upper extremities, with cranial neuropathy and autonomic symptoms often combined.

In 50-70% of cases, GBS develops 2-4 weeks after a prodromal gastroenteritic or respiratory illness, or immunization. During the acute phase, disability can be severe and can result in respiratory insufficiency and death. It is believed to be an autoimmune disorder resulting from T-cell activation, with the production of antibodies directed against the proteins of the peripheral nerves. Infectious agents, such as the Epstein-Barr virus or Campylobacter jejuni, as well as immunisation or surgery, may trigger the formation of these autoantibodies. Most of these antibodies are directed at myelin sheath proteins, but they may also be directed against axonal proteins.

In this case, there was an immunisation history, and we showed M. pneumoniae infection in addition.

Limb weakness, especially in the distal part of the lower extremities, was the most initial presentation symptom associated with GBS. Respiratory failure is a rare but important initial presentation of pediatric patients. Distal paresthesia and neuropathic pain are also common in children. Neuropathic pain involving the back, buttocks, or legs, and dysesthesia are also commonly encountered, representing as many as 50% of affected children on initial presentation. This pain is often poorly localized, and may cause irritability, vomiting, headache with meningismus, and apparent encephalopathy.

Lee et al. reported 56 patients who presented as the initial symptom limb weakness in %53.6, paresthesia or muscle pain in 28.5%, facial palsy in 26.8%, ophthalmoplegia in 21.4%, dysphagia or dysarthria in 26.8% patients.

In literature, there have been rare patients who presented abdominal pain with GBS. Wong et al. reported a 19-year-old girl who presented abdominal pain in the right side. Another case has
been reported by Lyons et al., 3 year old girl has developed lower abdominal pain with GBS\(^4\). This is the first report, to our knowledge, first presented of acute abdomen of a pediatric patient with GBS. Acute abdomen is a common complaint in the pediatric emergency department but has not previously been reported in the GBS. Whether this patient's pain was related to sensory nerve and root demyelination, or to gastrointestinal autonomic dysfunction is uncertain, but there were no associated gastrointestinal symptoms\(^3\).

It is, therefore, important to diagnose GBS early, and should be in the differential diagnosis of any child presenting with acute abdominal pain. Once diagnosed it is important to be aware of the possibility of mortality and morbidity and to refer the patient for IVIG and PE treatment without delay.

REFERENCES


