Case Report

Recurrent Guillain-Barre Syndrome- a case report.

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Abstract

Guillain-Barre syndrome (GBS) manifests as acute flaccid weakness of limbs, and is considered a monophasic illness. But recurrences have been reported. Published case studies suggest that 1- 5% of patients who had GBS will have recurrent attacks. We describe a 66-year-old lady who presented with acute onset descending, symmetrical, areflexic, flaccid quadriparesis that progressed to respiratory failure. She had a history of GBS five years back of which she had made a complete recovery with no residual weakness. The diagnosis of recurrent GBS was made.

Key words: Recurrent Guillain-Barre syndrome; chronic inflammatory demyelinating polyradiculoneuropathy

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Introduction

Recurrent GBS is rare but presents a diagnostic challenge. Even though not well described recurrences have been reported (1,2). We report a 66-year-old lady who presented with a second episode of GBS after an interval of five years.

Case presentation

Second episode

Sixty six-year-old house wife from Medirigiriya, was admitted to the local hospital due to generalized weakness in her upper limbs in the morning. The weakness then descended rapidly during the day to involve the lower limbs and was transferred to Teaching Hospital Anuradhapura (THA). On admission to THA in the same day afternoon she complained of difficulty in breathing. During the examination at the Emergency Treatment Unit at THA there was no bladder or bowel involvement. She had no preceding fever, respiratory tract infection or gastroenteritis. She had no exposure to toxins, poison or had no recent vaccination. She had not travelled abroad. She had no loss of appetite or weight loss.

At the THA she was conscious and rational but had flaccid quadriplegia with areflexia and flexure plantar response. There was no associated sensory, ocular or bulbar involvement or fatigability. Cerebellar signs were normal. She had tachycardia (110 beats per minute) and blood pressure of 190/80 mmHg indicating autonomic dysfunction.

Few hours after admission, her cough reflex became poor and the spontaneous vital capacity deteriorated to 150 l/min compared to 260 l/min on admission. Patient was intubated and ventilated, and transferred to the intensive care unitduring the night of the day of admission.

Her cerebrospinal fluid (CSF) protein was 54 mg/dl with albuminocytologic dissociation and the nerve conduction wasreported as acute inflammatory demyelinating polyneuropathy (AIDP). Other investigations were normal including serum electrolytes.

She was treated with intravenous immunoglobulin (IVIG) for five days together with physiotherapy. She recovered gradually over the next three weeks gaining a muscle power of 2 in upper and 3 in lower limbs. She was off ventilator support after one month but could not walk. Six weeks later she died due to pneumonia.

Previous (first) episode

Five years back she was admitted to THA with a history of numbness of toes followed by ascending weakness for four days. Her bladder and bowel functions were preserved.She had flaccidquadriplegia with global areflexia with no sensory, cranial nerve or autonomic involvement.

Her nerve conduction showed an axonal and demyelinating type neuropathy and CSF protein was 80mg/dl with no cells again showing albuminocytological dissociation. Her basic laboratory investigations were all normal.

A diagnosis of GBS had been made. She was treated with IVIG and did not require ventilatory support. She gradually improved over the next three months and made a complete recovery with no residual weakness. The details of the first episode were traced from original bed head ticket and diagnosis card.

Discussion

Guillain-Barre syndrome (GBS) is an acute areflexic polyneuropathy, of autoimmune nature. The weakness reaches its peak within 4 weeks(2,3). GBS is considered to be a monophasic disease and sub-typed neurophysiologically and with anti-ganglioside antibodies as; acute inflammatory demyelinating polyneuropathy, acute motor axonal neuropathy, acute motor sensory axonal neuropathy and Miller-Fisher variant of GBS.

Few patients with GB Shave recurrences (4,5). There is a debate whether it is recurrent GBS or anacute-onset chronic inflammatory demyelinating polyradiculone-uropathy (CIDP) (6).

Most patients with recurrent GBS develop one episode, but patients with two or more episodes are described (7) with recurrence occurring months to years after the first (4).Patients with acute-onset CIDP may initially appear to have GBS. Steroids and other immune-suppressants are indicated in management of CIDP, while not in GBS.

Our patient presented with two episodes of areflexic quadriplegia five years apart,following complete recovery from the first. First episode is unlikely to be CIDP, because patient had complete recovery.In both episodes she reached the peak motor dysfunction within the first week and then gradually improved, which again does not support a diagnosis of CIDP.The second episode was prolonged and patient never recovered fully. The second episode can be considered as a diagnostic challenge because it resembles acute-onset CIDP, and differentiating it from recurrent GBS is difficult. Descending paralysis seen in the second episode does not help to differentiate between AIDP subtype of GBS and acute-onset CIDP. Also there were no treatment related fluctuations. However autonomic dysfunction and inability to walk independently after the second episode supports the diagnosis of recurrent GBS. The nerve conduction also had no features suggestive of CIDP(6).

Accurate diagnosis of GBS and its subtypes may be important as clinical epidemiology of these sub-types may vary. Although our patient was diagnosed as AIDP sub-type electrophysiologically, anti-ganglioside antibodies and serial electrophysiological studies may be needed to correctly sub-type (8).

In a retrospective case-control study that compared recurrent and non-recurrent-GBS, the recurrent-GBS patients was younger, had milder symptoms, and was more likely to have the Miller Fisher syndrome variantduring the first attack (4,9). Also patients with multiple recurring episodes of GBS have an increased tendency for residual neurological deficits (7).

This case highlights that GBS is not a monophasic illness. Further research is needed to understand why only few have recurrences and to identify predictors.

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Competing Interests
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