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Internal Medicine

RECURRENT GUILLIAN BARRE SYNDROME- A CASE REPORT AND REVIEW OF LITERATURE

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ABSTRACT Recurrence in GBS is very uncommon with a reported incidence of 1-6%. Here we have a 26 year old male presented with progressively increasing weakness of bilateral upper and lower limb of 3 week duration with 2 episodes of past history suggesting of GBS. He completely recovered from last 2 attacks. NCS and Cerebrospinal fluid analysis also pointed to GBS in the form of demyelination and Albumino-Cytological dissociation respectively. Treated with intravenous immunoglobulin and improved symptomatically. Recurrences are higher in those with Miller Fisher variant and in children & younger age. It was also seen that they had more residual neurological deficits with each successive episode of GBS. Response to Plasmapheresis or IVIG may not be as good as first attack on recurrence.

KEYWORDS : Recurrence In Guillian Barre Syndrome

INTRODUCTION

Guillian Barre syndrome(GBS) is an acute onset, rapidly progressive autoimmune syndrome that affects the peripheral nervous system causing ascending paralysis. It often fo llows an episode of viral or bacterial gastroenteritis or respiratory tract infection. Common organisms that have been identified include Campylobacter jejuni, Ebstein Barr virus, Cytomegalovirus, Mycoplasma pneumonia.[1] It is an uncommon disease with a reported incidence of 1.3/100000 population.[2] Men are affected more often than women.[2] Recurrences are very uncommon in Guillian Barre syndrome with a reported incidence of 1-6%.[3] Here we report a case of a 29 year patient who presented with the third episode of GBS and review published literature on recurrent GBS

CASE REPORT

26 year old male presented with progressively increasing bilateral upper and lower limb weakness over a 3 week period. He also had decreased sensation over both lower limbs for the same duration. He developed the weakness after an episode of fever with cough and cold lasting for 2 days 3 weeks ago. He had no history of cranial nerve involvement. There was no swaying. No history of back pain, neck pain or pain radiating down the limbs. No history of bladder or bowel involvement. He had similar history of progressive weakness involving all 4 limbs at the age of 5, when he was diagnosed to have GBS on the basis of nerve conduction study(NCS) showing demyelinating motor radiculoneuropathy involving all 4 limbs. He was treated with weight based intravenous immunoglobulin therapy following which he recovered fully. He developed a second episode of weakness at the age of 20 which was milder in intensity and resolved within 2 weeks. NCS done then revealed both axonal and demyelinating motor and sensory neuropathy. Current episode is the third. He has been completely normal in between episodes. There is no significant family history. No history of substance abuse. Each episode was preceded by a short acute febrile illness. On examination , he was afebrile. His vital parameters were stable. Nervous system examination revealed flaccidity and hypotonia of all 4 limbs more so in the lower limbs. Muscle power was grade 3 in the lower limbs and grade 4 in the upper limbs across all joints. All deep tendon reflexes were absent and plantars were down going. Sensation was reduced below Thoracic 10 dermatome level. Other system examination was normal. Investigations revealed normal blood counts, sugars, electrolyes, renal and liver functions. Magnetic resonance imaging of the brain was normal while imaging of the spinal cord showed enhancement of lumbar nerve roots and perineural thickening of the filum terminale. NCS revealed demyelinating polyradiculo neuropathy of all 4 limbs and sensory neuropathy of upper limbs. Cerebrospinal fluid analysis showed a cell count of 0 and protein of 117 mg/dl suggestive of albumin-cytological disassociation. He was diagnosed to have recurrent GBS and started on 2g/kg dose of intravenous immunoglobulin given over 5 days. He tolerated the therapy well with no adverse reactions. Following this, his muscle power improved to grade 4 in the lower limbs and 5 in the upper limbs and sensory symptoms disappeared.

DISCUSSION

Georges Guillian, Jean Alexandre Barre and Andre Stroll first presented a patient with weakness in 1916 and the term GBS was first used in 1927.[4] There have been several diagnostic criteria used to diagnose this condition. The latest, Brighton Collaboration, uses three clinical criteria-bilateral flaccid limb weakness, decreased or absent deep tendon reflexes in weak limbs and a time course between onset to peak weakness of less than 28 days.[5] Laboratory criteria include CSF cell count of <50/microl and protein above normal values. Nerve conduction studies should be consistent with any one subtype of GBS.[5] All these criteria were fulfilled by our patient.Earlier, demyelination was the only pattern described and it was in the 1980s that acute motor axonal and acute motor sensory axonal patterns were described. Acute inflammatory demyelinating poly neuropathy continues to be the most common pattern affecting 65-70% of cases.[6] Miller Fisher variant presents with opthalmoplegia, ataxia and areflexia but does not have motor weakness and this is also seen more often in males(2:1 ratio).[2]

Plasma exchange as a modality of treatment was introduced in 1978 and its benefits were proven in the 1980s. Intravenous immunoglobulin started being used from 1988 onwards and in now the recommended treatment in GBS.[2]

With these modalities of therapy, mortality rates are around 6-8% and usually due to respiratory muscle involvement with need for mechanical ventilation or autonomic dysfunction .

Recurrent GBS has been described in 1-6% of cases. Recurrences have been described as episodes of GBS that satisfy clinical and laboratory criteria with a time interval of 2 months between episodes (if patient has recovered completely) or 4 months (if recovery is incomplete).[3] Analysis of patients have revealed a higher frequency of recurrences in those with Miller Fisher variant and in children and younger age.[3,8] Severity of disease was milder in those with recurrent GBS and they had sensory involvement more often.[3] Time interval between episodes was variable but tended to become shorter with each successive episode. Antecedent infections could be either similar or different and unusual triggers including vaccinations(influenza) have been reported. NCS patterns could be either similar or different during different episodes as was also seen in our patient.[7] It was also seen that they had more residual neurological deficits with each successive episode of GBS.8 Plasmapheresis, IVIg and immunosuppressive therapy were tried for relapses but responses were not as good as with first episode.8 Nerve biopsy showed onion bulbs in those with several recurrences.[9]

CONCLUSION

Recurrent GBS is an uncommon clinical syndrome that should be considered in patients who develop first episode at an early age, have mild symptoms or Miller Fisher variant. Early evaluation and treatment is needed. Response may be incomplete.

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