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Case Report A SCITECHNOL JOURNAL

Primary Restless Leg Syndrome in a Patient with Guillain-Barre Syndrome

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Abstract

Secondary restless leg syndrome has been described with Guillain-Barre syndrome. However primary RLS association have never been reported. We report a patient with GBS patient who developed features of restless leg syndrome consistent with primary RLS and positive family history. This patient suggests an association between primary RLS with GBS.

Keywords

Restless leg syndrome (RLS); Guillain-Barre syndrome (GBS); Acute primary RLS and GBS association

Introduction

Guillain-Barre syndrome (GBS) is an immune mediated inflammatory polyradiculo-neuropathy. While motor symptoms are more disabling, it is the sensory symptoms which frequently mark the onset of disease in the form of tingling paraesthesia in the limbs or loss of vibrations or proprioception occurring in 68% to 72% of cases [1]. These pain syndromes are attributed to radiculopathy and RLS. Few case reports suggested association of RLS with GBS, however the RLS here was thought to be secondary [2,3]. We did a computer search for primary RLS with GBS, association of primary RLS with GBS but could not find anything pertaining to our search. Restless leg syndrome, has also been associated with GB syndrome in its secondary form and whether this association is a causality or its an etiological link is not clear. We managed a patient of GBS who had RLS and on detailed history and examination we found that it was primary RLS, hence we want to report this case showing association of primary RLS with GBS.

Case Report

A 27 year old man, presented with acute onset rapidly progressive ascending flaccid quadriparessis. The patient complained of tingling and numbness in hands and feet two days prior to onset of weakness. There was no history of bladder or bowel involvement but patient had difficulty in breathing and swallowing. His muscle power 1/5 in both upper and lower limbs with hypotonia and areflexia. His sensory examination showed normal pin prick but impaired joint position sense in feet and hands. Blood counts and serum chemistry were normal. Nerve conduction study revealed decreased amplitude of compound muscle action potentials (CMAP) with normal distal latencies, and sensory potentials consistent with acute motor axonal variant of GBS. CSF showed raised protein 70 mg/dl without raised cell count, suggestive of albumin-cytological dissociation. He was given intravenous immunoglobulin for five days. His muscle power improved to 2/5 noted in all four limbs within 5 days of treatment.

By day 7 of admission, the patient complained of severe pain in both his lower limbs with an unpleasant sensation in his calves. Pain was more severe in left lower limb and over a period of 24 hours also progressed to involve the ipsilateral trunk and upper limb. Symptoms were worse during night. He would want his limbs to be moved or massaged repeatedly for relief. The patient was diagnosed as RLS according to IRLS diagnostic criteria, in form of having urge to move limbs, unpleasant sensation in limbs, urge to move limbs with unpleasant sensation getting worse at rest and relieve by massage and worsening of symptoms during evening/night. He scored 26 on the disease severity rating scale. His mother also suffered from RLS but was not specifically treated. With a provisional diagnosis of primary RLS, the patient was prescribed ropinirole 0.5 mg two hours before sleep time following which he had complete disappearance of symptoms.

Discussion and Conclusion

This patient of AMSAN variant of GBS developed sensory paraesthesia almost a week later after the disease onset. This would have been classified as either part of GBS or secondary RLS. However, here we had a young patient with family history of similar RLS symptoms in mother along, asymmetry of symptoms, involvement of trunk and upper limb, intact pin prick sensation as well as good response to ropark. These features are consistent with a diagnosis of primary RLS and not secondary/neuropathic RLS which presents with older age of onset, symmetrical symptoms and absence of family history and painful dysthetic symptoms [4]. Pathogenesis of RLS in presence of neuropathy is peripherally disrupted sensory modulation as well as type C and A delta fibre impaired inputs which presents clinically as impaired pin prick sensation whereas activation of spinal generators of RLS due to impaired descending dopaminergic pathways in primary RLS [5].

The association of GBS and RLS have been described in case reports and case series but all of these patients developed acute RLS [2,3]. Both these cases were attributed to secondary RLS as detailed family history and sensory testing were not considered. Since CIDP and many other genetic neuropathies also present with RLS where it is usually considered secondary to neuropathy but whether this is an association only or causality, is an enigma. So we concluded that this is a unique case where primary RLS was associated with GBS rather than secondary to GBS. Therefore, careful family history and meticulous examination should be done for categorising RLS.

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