

Restless Legs Syndrome Presenting As Acute Form in a Patient with Guillain-Barre Syndrome

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Received: December 15, 2015; **Published:** December 23, 2015

Abstract

Restless Legs Syndrome (RLS) is a sleep-associated movement disorder with sensory and motor components that follows a chronic course. Acute development of RLS has rarely been reported. Our purpose was to raise awareness of the subject by describing a picture of acute RLS in a case of Guillain-Barre syndrome.

Keywords: Restless Legs; Syndrome; Guillain-Barre; RLS

Introduction

Restless legs syndrome (RLS) is a sleep-associated movement disorder characterized by an urge to move the extremities in response to unusual and uncomfortable sensations in the limbs. It is life-long, chronic disease and has two clinical forms, idiopathic and secondary. The idiopathic form shows a genetic predisposition and follows a milder and slower course beginning in early adulthood. The secondary form exhibits no genetic predisposition, begins in late adulthood, follows a moderate or severe clinical course and progresses more rapidly [1].

Development of RLS in acute onset is an unexpected clinical presentation. Only two RLS cases emerging in acute form in cases of GBS have been reported [5]. Our purpose was to contribute to the literature by reporting a case of acute RLS developing in GBS and to compare our cases with the others.

Case Report

A 52-year-old male patient presented to our emergency department with increasing numbness and weakness in extremities beginning three days prior to hospitalization. He had experienced a gastrointestinal system infection two weeks previously. The first physical examination revealed tetraparesis together with a decreased deep tendon reflexes in all extremities. Immediate evaluation with electroneuromyography revealed acute demyelinating sensorimotor polyneuropathy accompanied by conduction blocks. Intravenous immunoglobulin therapy was administered immediately for five days. On the fifth day of symptoms, the patient began complaining of a need to move his legs, particularly at night, accompanying paresthesia that he found it difficult to describe, and that he suffered from sleeplessness as a result. He stated that the symptoms improved with passive exercise and massage performed with the help of relatives, but did not disappear. The patient also stated he had had no similar complaints prior to this clinical picture. A single dose of 0.250 mg pramipexole, a dopamine agonist, was initiated two hours prior to sleep time. Although there was a significant improvement in symptoms on the second night, the dosage was increased due to a worsening of symptoms on subsequent nights (a single nocturnal dose of 0.75 mg/day). But clinical well-being was not as good as that on the first night. Treatment was therefore adjusted to a 600 mg gabapentin tablet (evening dose), and a pronounced improvement in symptoms was reported. Ferritin and vitamin B12 levels and thyroid function tests were within normal limits. Polysomnography (PSG) was recorded for one night to assist diagnosis. The suggested immobilization test was performed

Citation: Vildan Altunayoglu Cakmak, *et al.* "Restless Legs Syndrome Presenting As Acute Form in a Patient with Guillain-Barre Syndrome". *EC Neurology* 2.5 (2015): 245-247.

prior to nocturnal sleep, revealed 156 periodic limb movements and supported the diagnosis of RLS (Figure 1). Symptoms decreased but persisted at check-up one month after the start of clinical treatment, while at six-month check-up we learned that the patient had stopped his medication of his own volition and that symptoms had not recurred.

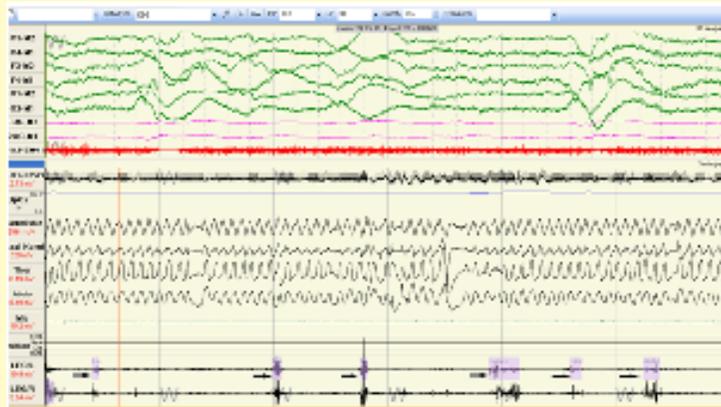


Figure 1: Picture is illustrating a series of periodic leg movements (arrows) recorded by polysomnography during suggested immobilization test.

Discussion

Several studies have noted a greater prevalence of RLS in polyneuropathies involving small fibers and immune-mediated polyneuropathies [7]. The literature contains no clinical studies investigating the presence of RLS in acute polyneuropathies. There are only two cases of RLS associated with GBS with acute onset [5]. Interestingly, a second condition in which acute RLS is reported to develop is multiple sclerosis, an autoimmune inflammatory disease of the central nervous system [2,4]. Recently a review published by Weinstock, *et al.* pointed out the role of immune-mediated mechanisms as well as dopamine dysfunction and iron efficiency in the central nervous system and set out three mechanisms in the relationship between RLS and immunological dysfunction:

1. Inflammation leading to iron deficiency through increased synthesis of hepcidin.
2. Direct effect of humoral and cellular immunological mechanism on the central and peripheral nervous systems.
3. Possible interaction between genetic susceptibility to RLS with inflammatory disorders and immune alterations [8].

The two cases previously reported in the literature and our cases were similar in many aspects. They presented in the acute demyelinating sensorimotor polyneuropathy, exhibited a good response to antiepileptic therapy (carbamazepine, gabapentin) an RLS symptoms resolved and drug therapy was stopped at follow-up visits. Gabapentin, an antiepileptic effective in neuropathic pain, is the most popular preparate in the treatment of RLS after dopamine agonists and may be the first option in the treatment of RLS cases in which sensory and motor symptoms predominate [3].

It can be difficult to differentiate between RLS symptoms and neuropathic pain. However, patients should be asked about features that differentiate RLS from polyneuropathy-associated symptoms such as the need to move, complete or partial relief being obtained from movement and a circadian rhythm characteristic. Identification of dopamine response can help with diagnosis. SIT tests performed before sleep and the observation of periodic leg movements can also support diagnosis [6]. It is also important to investigate other disorders that may cause secondary RLS.

Conclusion

In conclusion, although it is rare, RLS can develop in acute form and then disappear in acute inflammatory diseases. The development of RLS secondary to acute immune-mediated peripheral nerve diseases suggests that peripheric and immunologic mechanisms may contribute to the pathogenesis of RLS.

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Volume 2 Issue 5 December 2015

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