

Chapter: 16

RESTLESS LEGS SYNDROME

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A disorder characterized by an almost irresistible urge to move, usually associated with disagreeable leg sensations , worse during inactivity, and often interfering with sleep.

RLS Diagnosis : Essential Criteria(NIH 2002)

- An urge to move the legs occurs, usually accompanied or caused by uncomfortable and unpleasant sensations in the legs.
- The urge to move or unpleasant sensations begin or worsen during periods of rest or inactivity such as lying or sitting.
- The urge to move or unpleasant sensations are partially or totally relieved by movement, such as walking or stretching, at least as long as the activity continues.
- The urge to move or unpleasant sensations are worse in the evening or night than during the day or only occur in the evening or night.

Supportive Clinical Features for Diagnosis

1. Response to dopaminergic therapy
2. Periodic Leg Movements (during wake or sleep)
3. Family history of RLS

Another possible cause of idiopathic RLS is a deficiency in iron transport or storage

Iron deficiency, in turn, might cause a dysfunction of the dopamine system .

Secondary RLS is associated with : Pregnancy, Gastric resection, Peripheral nerve disorders , ADHD, Spinocerebellar atrophy, SCA3,Charcot-Marie-Tooth, Type 2,Diabetes,Parkinson disease, Tourette's syndrome and Rheumatoid arthritis

GENETICS : Familial aggregation of RLS Cases has been Suspected for long (>50% of patients know of affected relative) and a number of large pedigrees have been uncovered

3 case series in the mid-1990s found that more than 60% of RLS patients report affected first degree relatives. Patients with idiopathic RLS more likely to have a definite (42.3%) or possible (12.6%) family history of RLS .Patients with definite family history have earlier age at onset (35.5 vs. 47.2 years) . Three loci showing vulnerability to RLS have been described in French – Canadian and Italian families in chromosomes 12q, 14q and 9q, emphasizing on an autosomal dominant mode of inheritance, These have been labeled RLS1, RLS2 and RLS3, respectively.

ORIGIN OF RLS: To date, we cannot say where RLS originates, but it would appear that a variety of neural centers and elements from the peripheral nerves to the brainstem and cerebellum and perhaps to the cortex show altered function during RLS symptoms and may influence the condition in different ways .

EVALUATION OF RLS: A detailed history ,followed by biochemical evaluation to detect a cause: serum ferritin ,screen for uremia, screening for diabetes, peripheral neuropathy. Polysomnography is not routinely indicated in evaluation of RLS!

Treatment: Listen, support, and validate. Reconsider medications known to exacerbate RLS (Lithium, SSRI's, tricyclics, dopamine antagonists)

Possibly beneficial in some patients: hot baths, delayed sleep time/rise time, exercise, avoid alcohol and nicotine.

Dopamine agonists are the mainstay and first-line therapy. Relief in 70-100% ,

Dopaminergic agents came out as having the best evidence for efficacy in primary RLS The following level A recommendations can be offered: for primary RLS, cabergoline, gabapentin, pergolide, ropinirole, levodopa and rotigotine by transdermal delivery (the latter two for short-term use) are effective in relieving the symptoms. Combination therapy may be required in some.

In the end, the therapeutic plan should be individualized to suit each patient's presentation and needs

CONCLUSION: RLS is a common, treatable, and underdiagnosed disorder. It causes significant impairment of quality of life. The pathophysiology of RLS is unknown, RLS can be both secondary and idiopathic. The diagnosis is made by history. Treatment is mainly pharmacologic with an excellent response.