

Medical device alternatives to Restless Legs Syndrome (RLS) dopaminergic drugs

Helly Simmons

Simmons H. Medical device alternatives to Restless Legs Syndrome (RLS) dopaminergic drugs. *J Neurol Clin Neurosci* 2022;6(1): 01-02.

ABSTRACT

RLS (Restless Legs Syndrome) is a prevalent neurological condition that affects 2.5%-15% of the Western population. Sleep loss impairs everyday function for one-third to one-half of RLS sufferers. Patients with RLS dread the start of each RLS attack because they are so unpleasant. The

most generally established treatment for RLS is drugs that pass the blood-brain barrier. Ergot and non-ergot derived dopaminergic medications were proposed as therapies for RLS in the 1980s. However, reports of severe mid- and long-term complications following dopaminergic medication therapy of RLS began to emerge in the 1970s, including tardive dyskinesia as a result of widespread usage of antipsychotic medicines.

Key Words: *Restless legs syndrome; Medical devices; Meta-analysis; Clinical trials; Sham effect; Placebo effect*

INTRODUCTION

The most prevalent dopaminergic medication adverse effect is known as “augmentation,” a mild-sounding term for a highly unpleasant phenomenon: worsening of RLS severity with time. RLS severity is worsening, with RLS dysphoria spreading from the legs to the arms and RLS symptoms appearing sooner and earlier in the day. At the 2016 SLEEP summit in Denver, CO, a half-day postgraduate session on augmentation mitigation was launched. Medical device therapies for RLS have sometimes been ignored out of hand because first dopaminergic medication treatment of RLS seemed so promising. Because devices do not have long-term, drug-like adverse effects, they must be carefully assessed [1]. RLS has been treated using a variety of technologies. The majority of researchers believe leg anomalies are the cause of RLS, and their device is designed to help with this. We'll give a brief history of RLS pharmacological therapy, a summary of RLS epidemiology, and an assessment of RLS disease modulators before getting into the details of RLS devices. Following that, there will be a restricted assessment of RLS therapy devices that have already been released [2].

RLS history

Restless Legs Syndrome (RLS), also known as Willis-Ekbom Disease (WED), was first reported by English physician Thomas Willis in 1672 and 1685. During RLS bouts, he noticed strange motions and decided that RLS was quite uncomfortable. He didn't seem to realise, however, that during an RLS attack, patients chose to move around in strange ways to alleviate RLS discomfort. Stretching to relieve a leg cramp is comparable to the motions associated with RLS. Leg cramps can be alleviated by consciously stretching the constricted muscles. RLS motions, like stretching, are voluntary movements done by RLS sufferers to alleviate RLS discomfort. Willis suggested a tincture of opium, a well-known pain reliever at the time, to treat RLS. As a result, he paved the way for drug-assisted treatment of RLS.

Patients are obliged to keep moving their legs as long as the sensations persist, which provides some comfort.” It was reported that patient-generated counter-stimulation without using the word. RLS dysphoria is briefly reduced by patient leg movement, which acts as a counter-stimulus to the dysphoria [3]. It was presented an unofficial, placebo-controlled trial that compared the improvement of patients who got placebo pills to those who received vasodilators. Drugs worked substantially better firmly confirming RLS as a problem that can be treated with medication.

RLS epidemiology

To present, epidemiology studies of RLS have only succeeded in determining the disease's burden within and within different populations, as well as identifying RLS's general characteristics. Unlike epidemiology research that linked cigarette smoking to lung cancer or a mosquito-borne

virus infection to yellow fever, these studies have yielded no definitive RLS aetiology. Despite much research, the cause of RLS remains unknown. The prevalence of RLS in the general population is believed to be between 2.5% and 15%. RLS prevalence appears to be a function of age, gender, and, maybe, race in numerous studies.

RLS is age-dependent and can begin as early as childhood. The majority of studies show that RLS prevalence rises with age 11-15, with no increase after the age of 50. Women are reported to have a prevalence of over two times that of men. It's uncertain how much RLS prevalence varies by race, according to studies that utilised the same diagnostic criteria and identified racial disparities. In another study, however, no racial differences were found among RLS patients of various races living in the same city [4].

Disease modulators for RLS

During simulated RLS attacks, the effect of external sensory stimuli was studied. When an external sensory input (a counter-stimulus) was applied during a simulated RLS episode, reduced RLS limb discomfort was seen.

Genetics

Within the RLS population, there is significant genetic variation. A 50% family history of RLS has been frequently recorded, with family history being linked to an earlier age of RLS onset and greater RLS severity. It has been discovered that autosomal dominant genetics exists. Six RLS gene candidates have been identified as potential RLS genes.

Effects of the day, season, and latitude

Diurnal, seasonal, and latitudinal variations are also RLS modulators, with increased incidence at night, in the summer, and at more northerly latitudes.

RLS (Primary)

There is no concomitant condition or illness in the great majority of RLS patients to explain the occurrence of RLS. The anatomic place of origin appears to be in the central nervous system, above the level of the spinal cord and below the level of the cerebral cortex, in patients with primary RLS. It's thought that the source of the problem is in the subcortical area, possibly in the thalamus or cerebellum [5].

RLS (secondary)

RLS has been linked to a wide range of illnesses and diseases due to its widespread prevalence. Some have interpreted these correlations as causative relationships, but such interpretations should be approached with caution. General population studies are less prone to bias and have demonstrated a strong link between RLS and a small number of variables, including

Editorial Office, *Journal of Neurology and Clinical Neuroscience*, Windsor Berkshire, England

Correspondence: Helly Simmons, Editorial Office, *Journal of Neurology and Clinical Neuroscience*, Windsor Berkshire, England, E-mail neurosci@jneurologyandclinicalneuroscience.org
 Received: 03-Jan-2022, Manuscript No. PULJNCN-22-4121; Editor assigned: 06-Jan-2022, PreQC No. PULJNCN-22-4121(PQ); Reviewed: 12-Jan-2022, QC No. PULJNCN-22-4121; Revised: 20-Jan-2022, Manuscript No. PULJNCN-22-4121(R); Published: 24-Jan-2022, DOI: 10.37532/2632-251X.2021.6(1).102



This open-access article is distributed under the terms of the Creative Commons Attribution Non-Commercial License (CC BY-NC) (<http://creativecommons.org/licenses/by-nc/4.0/>), which permits reuse, distribution and reproduction of the article, provided that the original work is properly cited and the reuse is restricted to noncommercial purposes. For commercial reuse, contact reprints@pulsus.com

pregnancy, kidney illness, and iron metabolism. The underlying condition is addressed as the first step in treating secondary RLS. Pregnancy, unlike kidney illness and iron metabolism problems, is a self-resolving condition [6-11].

Kidney disease is a condition that affects the kidneys

RLS is more common in chronic hemodialysis users than in the general population. RLS puts hemodialysis patients at risk of lengthier or incomplete dialysis sessions. These people would benefit from effective RLS treatment while on hemodialysis.

RLS has been connected to the following: with headaches, social isolation, depression, reduced libido, hypertension, and heart problems; with obesity, hypertension, loud snoring, drinking alcohol, and smoking; with hypertension, arthritis, gastroesophageal reflux, depression, anxiety, diabetes, and sleep apnea; and with psychiatric disorders [12-15].

CONCLUSION

RLS is a common sleep disorder for which medicines are currently the gold standard of treatment. RLS medication benefits are low whether examined graphically or as standardised effect sizes, whereas pharmacological side effects can be significant. Leg heating to increase perfusion, whole body vibration to increase blood flow, enhanced external counterpulsation to increase cardiac output, venous sclerotherapy to treat incompetent veins, pneumatic compression stockings to reduce venous and/or lymphatic stasis, and acupuncture and foot compression to modify nerve impulses in the legs have all been proposed as medical device treatments for RLS. Despite the fact that some of these treatments have showed promise, the FDA has yet to approve them as a Class II medical device.

The Relaxis system is a prescription-only device that has been approved by the FDA. It includes a vibrating pad as well as a digital controller. The Relaxis system has been shown in two clinical trials to produce significant sleep improvement, with the degree of improvement comparable to that reported in RLS medication trials. The Relaxis system may be beneficial to some RLS patients.

The causes of RLS and how to treat it

Any RLS treatment proponent believes that their treatment targets the underlying cause of RLS or at the very least a significant contributing element. As a result, the aetiology and therapy of RLS are linked, and they will be discussed simultaneously. RLS is widely thought to be a brain problem, while not everyone agrees—especially medical device developers who feel RLS is caused by a leg anomaly.

RLS Drugs: Imbalance of a specific neurotransmitter

Drugs have long been considered the “gold standard” of RLS treatment. The majority of RLS medications are thought to work by correcting imbalances in specific brain chemicals. Amino acids, amines, amides, peptides, soluble gases, endogenous opioids, and other neurotransmitter families occur in the brain. In a recent meta-analysis of RLS medication therapies, a wide spectrum of these neurotransmitters was investigated. Dopamine-related (pramipexole, ropinirole, levodopa with dopa decarboxylase inhibitor), opioid-related (methadone), amino acid-related (gabapentin, enacarbil, cabergoline, gabapentin, pregabalin, carbamazepine), and amine-related (clonidine) pharmaceuticals were among the treatments studied. The fact that there are so many different drug families or drug classes available to treat RLS shows that no “magic bullet” pharmacological treatment has yet been discovered.

RLS medicine side effects that is troubling

Excessive drowsiness, impaired blood pressure regulation, rebound (worsening of RLS symptoms in the morning), augmentation (greater RLS severity than prior to drug therapy, earlier onset of RLS symptoms in the

evening, and spreading of RLS symptoms to the arms), compulsive activities such as compulsive gambling and compulsive sexual behaviour, and more are all serious side effects of RLS drugs [12]. Medical gadgets for the treatment of RLS should be considered because they do not have any negative side effects [13]. We'll look into medical device RLS remedies after a quick overview of RLS pharmacological therapy, which is the gold standard of RLS treatment. As previously stated, we will utilise sham effects size measures as estimates of the quality of clinical trial blinding in the studies below when they are available. A small sham effect size indicates insufficient blinding, while a higher sham effect size indicates adequate blinding. Due to a lack of blinding, it is more likely that the treatment will be reported as superior to the control.

REFERENCES

1. Reed W, Agramonte A. The etiology of yellow fever. An additional note. By Walter Reed, Jas. Carroll and Aristides Agramonte. *Jama*. 1983;250(5):649-658.
2. Zucconi M, FeriniStrambi L. Epidemiology and clinical findings of restless legs syndrome. *Sleep Med*. 2004;5(3):293-299.
3. Yeh P, Walters AS, Tsuang JW. Restless legs syndrome: A comprehensive overview on its epidemiology, risk factors, and treatment. *Sleep Breath*. 2012;16(4):987-1007.
4. Picchietti D, Allen RP, Walters AS, et al. Restless legs syndrome: prevalence and impact in children and adolescents—the Peds REST study. *Pediatrics*. 2007;120(2):253-266.
5. Phillips B, Young T, Finn L, et al. Epidemiology of restless legs symptoms in adults. *Arch Intern Med*. 2000;160(14):2137-2141.
6. Ulfberg J, Nystrom B, Carter N, et al. Prevalence of restless legs syndrome among men aged 18 to 64 years: an association with somatic disease and neuropsychiatric symptoms. *Mov Disord*. 2001;16(6):1159-1163.
7. Ohayon MM, Roth T. Prevalence of restless legs syndrome and periodic limb movement disorder in the general population. *J Psychosom Res*. 2012;53(1):547-554.
8. Tison F, Crochard A, Leger D, et al. Epidemiology of restless legs syndrome in French adults: a nationwide survey: the INSTANT Study. *Neurology*. 2005;65(2):239-246.
9. Allen RP, Picchietti D, Hening WA, et al. Restless legs syndrome: diagnostic criteria, special considerations, and epidemiology. A report from the restless legs syndrome diagnosis and epidemiology workshop at the National Institutes of Health. *Sleep Med*. 2003;4(2):101-119.
10. Lee HB, Hening WA, Allen RP, et al. Race and restless legs syndrome symptoms in an adult community sample in east Baltimore. *Sleep Med*. 2006;7(8):642-645.
11. Rozeman AD, Ottolini T, Grootendorst DC, et al. Effect of sensory stimuli on restless legs syndrome: a randomized crossover study. *J Clin Sleep Med*. 2014;10(8):893-896.
12. Winkelmann J, Wetter TC, Collado-Seidel V, et al. Clinical characteristics and frequency of the hereditary restless legs syndrome in a population of 300 patients. *Sleep*. 2000;23(5):597-602.
13. Hanson M, Honour M, Singleton A, et al. Analysis of familial and sporadic restless legs syndrome in age of onset, gender, and severity features. *J Neurol*. 2004;251(11):1398-1401.
14. Walters AS, Picchietti DL, Ehrenberg BL, et al. Restless legs syndrome in childhood and adolescence. *Pediatr Neurol*. 1994;11(3):241-245.
15. Quijano MC, Arango JC, Cuervo MT, et al. Traumatic brain injury neuropsychology in Cali, Colombia. *Revista Ciencias de la Salud*. 2012;10: 21-31.