Review Article

The restless legs syndrome (Ekbom's syndrome)

R. SANDYK

Summary

The restless legs syndrome is a condition of unknown aetiology characterized by nocturnal paraesthesiae in the lower limbs, an irreversible tendency to move the limbs, pain in the distribution of the paraesthesiae, and psychiatric disturbances. The syndrome may occur at any age and in either sex and usually runs a course over many years with spontaneous improvements and exacerbations. An association with iron deficiency has been suggested but in most cases no apparent cause has been found, and treatment at present is symptomatic with analgesics and sedatives. The differential diagnosis includes phenothiazine-induced akathisia and meralgia paraesthetica.

S Afr Med J 1983; **63:** 701-702.

The restless legs syndrome (RLS) is manifested primarily by the occurrence of ill-defined discomfort in the calves when the legs are in a position of rest. The condition was probably first described in 1685 by Thomas Willis,[1] who gave an accurate description of the main clinical features. Since 1944 Ekbom has made important contributions to the recognition of this syndrome.[2] He coined the term 'restless legs', which is now widely accepted.

Clinical features

RLS consists of the following main clinical features: (*i*) paraesthesiae or creeping sensations in the limbs, mainly in the calves, occurring at rest, especially in the evening or at night; (*ii*) an irresistible tendency to move the limbs, to stand up and walk; (*iii*) insomnia secondary to the above; (*iv*) pain with the same distribution as the paraesthesiae; and (*v*) anxiety, tension or mild depression.

Paraesthesiae or creeping sensations are usually confined to the calves. They are extremely unpleasant and deep-seated in muscles or bones rather than in the skin, mostly affecting the legs between the knee and ankle. The sensations are bilateral and symmetrical and occasionally may predominate on one side. In a few patients these sensations may involve the thighs and even the upper extremities (restless hands or arms). The paraesthesiae occur mainly in the evening and/or at night. The majority of patients have no discomfort during the day, but as the evening approaches they experience unpleasant sensations which usually last from 10 minutes to several hours.

Characteristically the patient experiences an urge to move his legs when paraesthesiae are felt, since these sensations occur when the limbs are at rest and are relieved by movement. To relieve the discomfort the patients move their legs in bed, sit on the edge of the bed swinging their legs in the air, stand up and walk around the room, or sometimes go outside and walk. Some may derive relief from arm movements.

The patients usually suffer from insomnia because the paraesthesiae usually either begin or become worse in bed, particularly during the first few minutes to half an hour after falling asleep, thus forcing the patient to wake up and move his legs. Some may become violent when restrained from walking during an attempt to relieve the pains. Lack of sleep may itself trigger other unpleasant symptoms, such as anxiety and mental exhaustion. The pains are described as a 'deep ache' or like 'toothache', and anxiety, tension and mild depression appear to be very common.

The incidence of the syndrome in the general population is unknown because many patients do not seek medical advice or are not greatly disturbed by the symptoms. In one study, Ekbom[3] found that among 500 healthy subjects 5% had symptoms of RLS. There is no sex predominance and the syndrome may appear at any age from 4 to 82 years, but is more frequent in persons in their sixties. RLS can run a course over many years with spontaneous improvements and exacerbations. It is considered to be benign. Pregnancy may be an aggravating factor, the paraethesiae usually appearing in the second half or in the last trimester and ceasing soon after delivery.

Pathogenesis and aetiology

In most cases no definite cause can be found. It has been suggested in the Swedish literature that iron deficiency and anaemia may be causative factors. Ekbom[4] showed that a quarter of the patients with RLS had decreased serum iron levels; conversely, a quarter of an unselected patient group with decreased blood iron values had RLS. The association of iron deficiency with RLS is encountered only in a limited number of patients.

A high familial incidence with a dominant mode of inheritance has been suggested. Boghen *et al.*[5] described an autosomal dominant transmission in a family in which there had been an association of RLS with myoclonus over 5 generations. A variety of other conditions have been reported in association with RLS, but all were isolated observations. RLS was found in 40 out of 600 patients with Parkinson's disease.[6]

The pathogenesis remains unknown. Some authors favour a peripheral origin while others suggest that the syndrome originates at the level of the central nervous system in the spinal cord or cortex.

Laboratory investigations

At present there is no diagnostic test for RLS, the only tests necessary being aimed at excluding secondary causes. Serum

Department of Medicine, Hillbrow Hospital, Johannesburg
R. SANDYK, M.D. (BONN), ÄRZTLICHE PRÜFUNG (SANKT AUGUSTIN)

Date received: 4 June 1982.

iron and haemoglobin levels should be determined in every patient with RLS. Psychological tests are important and the dismissal of a patient with the label of neurosis or hysteria should be condemned. Sleep EEGs may confirm the chronic insomnia; the electromyogram is usually normal but may occasionally reveal a neurogenic pattern.

Treatment

The majority of patients simply require symptomatic treatment with analgesics, sedatives and hypnotics. Diazepam has been shown to be most useful in a dose reaching 20 mg in the evening. Some patients may be helped by chlorpromazine in a dose of 50-100 mg at night. Because of the benign nature of the condition aggressive treatment with narcotics should be avoided.

Differential diagnosis

The clinical diagnosis is relatively easy, but unfamiliarity with the syndrome may lead to the diagnosis of neurosis, anxiety or hysteria. RLS should primarily be differentiated from phenothiazine-induced akathisia. The latter condition usually follows longstanding phenothiazine administration and manifests itself only during the day. The patient usually develops unpleasant sensations in the entire body, these ceasing when the drug is discontinued.

Meralgia paraesthetica is localized to the lateral aspect of the thigh, felt superficially, and associated with objective findings; it may be relieved by lying down. The syndrome is caused by fibrous tissue constricting the lateral cutaneous nerve, either where it emerges from the pelvis or where it passes through or beneath the inguinal ligament. It may follow the use of a corset and occasionally develops secondary to tilting of the pelvis in patients with lumbar intervertebral disc prolapse.

Paraesthesiae in the foot may occasionally be caused by compression of the peroneal nerve at the neck of the fibula.

REFERENCES

1. Willis T. *The London Practice of Physick.* London: Thomas Basset & William Crook, 1685.
2. Ekbom KA. Restless legs. *JAMA* 1946; **131**: 481-486.
3. Ekbom KA. Restless legs. *Acta Med Scand (Suppl)* 1945; **158**: 1-6.
4. Ekbom KA. Restless legs. In: Vinken PJ, Bruyn GW eds. *Handbook of Clinical Neurology*, vol. 22. Amsterdam: North Holland, 1976: 231-242.
5. Boghen DR, Peyronnard JM, Lamarre Y. Myoclonus in familial restless legs. *Neurology* 1975; **25**: 379-384.
6. Strang RR. The symptom of restless legs. *Med J Aust* 1967; **1**: 1211-1213.