

CASE REPORT

REM sleep behaviour disorder: a treatable cause of falls in elderly people

LITSA MORFIS, RAYMOND S. SCHWARTZ, PETER A. CISTULLI¹

Department of Aged Care and ¹Sleep Disorders Centre, St George Hospital, University of New South Wales, Gray Street, Kogarah, NSW 2217, Australia

Address correspondence to: L. Morfis. Fax: (+612) 9 350 3979

Keywords: *elderly, falls, REM sleep behaviour disorder*

Introduction

Reports of falls occurring exclusively at night have usually been in the context of a hospital setting (attempting to climb out of bed or during sleep) rather than in people living at home [1]. We report a case of recurrent falls in an elderly woman, occurring exclusively at night and caused by rapid eye movement (REM) sleep behaviour disorder.

In this condition there is a loss of the normal atonia of postural muscles that usually occurs in REM sleep [2]. It results in complex, vigorous, dream-enacting behaviours during REM sleep [3]. These typical behaviours have the potential to cause serious injury at night, particularly from falls. This disorder appears to have been overlooked in the geriatric literature as a potential source of falls and injury at night.

Case history

A 77-year-old woman was admitted to hospital after an acute fall, preceded by a 9-month history of recurrent falls at night. These episodes were occurring weekly, and always after midnight. On the day of presentation she dreamt she was 'standing in a garden and leant forward to pat a child on the head'. She was actually standing up in bed and as she reached over to 'pat the child', she fell to the ground. She awoke on the floor, having sustained a laceration to her forehead. Her husband reported that she was a snorer, but she did not complain of significant daytime hypersomnolence. There was no history of epilepsy.

Her intercurrent problems included type II diabetes mellitus of 2 years duration for which she required

insulin. She had a 5-year history of hypertension treated with metoprolol 100 mg bd and felodipine 5 mg daily. Her background history included a coronary artery bypass graft in 1990, complicated by a post-operative stroke causing left-hand paraesthesia. She also had hypercholesterolaemia, treated with simvastatin 10 mg daily, and glaucoma, for which she applied 1% timoptol eye drops. She was married, independent and active. She was a non-smoker and non-drinker.

Clinical examination revealed an alert and oriented lady. She was mildly obese. Her blood pressure was 150/70 mmHg lying and 140/65 mmHg standing. She was in sinus rhythm with a pulse rate of 80 per min. Cognition was normal, with a Folstein mini-mental examination score of 30/30. Neurological examination was normal: in particular there was no ataxia. Visual fields and visual acuity were normal. There was a superficial laceration across the head anteriorly, requiring sutures. The rest of the physical examination was unremarkable.

Routine biochemistry, including blood glucose and haematology tests were all normal. An awake electroencephalogram was normal. A cerebral CAT scan was also normal. In consultation with the Sleep Disorders Centre, a provisional diagnosis of REM sleep behaviour disorder was made. Standard nocturnal polysomnography was performed [4]; in addition, electromyographic activity of all four limbs was monitored and the patient videotaped throughout the night. This study revealed loss of REM atonia, and movements of the right arm during REM sleep, thereby confirming the diagnosis. In addition there was evidence of moderate obstructive sleep apnoea, with frequent obstructive apnoeas and hypopnoeas (16 per hour) and maximal oxygen

desaturation to 67%. A cerebral MRI scan was then performed to look specifically at midbrain structures and revealed cerebral atrophy and widespread hyperintense foci in the deep cerebral white matter, right thalamus and bilateral pons.

Treatment with clonazepam was commenced at an initial dose of 0.5 mg at night. There was an immediate reduction in motor activity at night, with no further falls. The dose of clonazepam was increased to 1 mg after several weeks of treatment, with complete resolution of abnormal motor activity. She was supervised closely as it was felt that clonazepam might exacerbate the obstructive sleep apnoea. She refused treatment with nasal continuous positive airway pressure, despite the severity of obstructive sleep apnoea demonstrated on polysomnography, as she was asymptomatic. She has remained well at follow-up, now more than 1 year.

Discussion

REM sleep behaviour disorder is characterized by dream-enacting behaviours during REM sleep, resulting from a loss of the usual atonia of REM sleep [3]. The true prevalence of REM sleep behaviour disorder is not known but is likely to be under-recognized. It is more common in older men and may have a preceding prodromal phase. The diagnosis can be made on history and is usually confirmed by polysomnography [2]. This disorder should be of interest to the geriatrician as it usually presents in later life [3, 5]. The literature reports up to 77.1% of patients with REM sleep behaviour disorder presenting with sleep-related injuries, including bruises, lacerations, dislocations and fractures [3]. One recent report describes subdural haemorrhage [6] arising from a fall at night during REM sleep behaviour disorder nocturnal motor activity.

The exact aetiology of this disorder is as yet undefined, although it is clearly due to a failure of the mechanisms that usually suppress motor function during dreaming. An association with pontine pathology has been described in cat experiments, where lesions of the pontine tegmentum reproduced stereotypical behaviours [7]. Experimental lesions in the medial medulla of the cat have also been shown to cause REM sleep without atonia [8]. It has been proposed that REM sleep behaviour disorder may arise in humans following appropriate brain stem lesions, such as occurs with structural damage to the pontine tegmentum from vascular lesions [9]. The characteristic older age of onset of this condition also suggests an 'organic brain factor' in its aetiology [10]. The cerebral MRI finding of vascular lesions in the pontine and thalamic regions in our case lends further support to the belief that these lesions may be the cause of the disorder.

Clonazepam is highly effective in the treatment of REM sleep behaviour disorder. Long-term studies have

demonstrated a 77% complete response and 12% partial response, without the development of tolerance. It has been suggested that the effectiveness of this benzodiazepine agent may be through its serotonergic properties [3].

Our case describes an unusual cause of nocturnal falls in the elderly, and highlights the importance of a comprehensive sleep history in general history taking in the elderly. In cases of falls occurring only at night this diagnosis certainly needs consideration. Although REM sleep behaviour disorder is not a common modifiable risk factor for falls, recognition and treatment of this disorder will help reduce the consequent disability arising from this potentially treatable parasomnia, and avoid its misdiagnosis as nocturnal syncope or epilepsy.

Key points

- It is important to include a sleep history in general history taking of geriatric patients.
 - Consider REM sleep behaviour disorder when falls occur exclusively during sleep.
-

References

1. Catchen H. Repeaters: inpatient accidents among the hospitalised elderly. *Gerontologist* 1983; 23: 273-6.
2. Diagnostic Classification Steering Committee (Thorpy MJ, chairman). *International Classification of Sleep Disorders: Diagnostic and Coding Manual*. Rochester, MN: American Sleep Disorders Association, 1990; 177-80.
3. Mahowald MW, Schenck CH. REM sleep behavior disorder. In: Kryger MH, Roth T, Dement WC (eds): *Principles and Practice of Sleep Medicine*. 2nd ed. Philadelphia: WB Saunders, 1994.
4. Keenan SA. Polysomnography: technical aspects in adolescents and adults. *J Clin Neurophysiol* 1992; 9: 21-31.
5. Feinsilver SH, Hertz G. Sleep in the elderly patient. *Clin Chest Med* 1993; 14: 405-11.
6. Dyken ME, Lin-Dyken DC, Seaba P, Yamada T. Violent sleep related behaviour leading to subdural haemorrhage. *Arch Neurol* 1995; 52: 318-21.
7. Hendricks JC, Morrison AR, Mann GL. Different behaviours during paradoxical sleep without atonia depend on pontine lesion site. *Brain Res* 1982; 239: 81-105.
8. Lai Y, Siegel JM. Medullary regions mediating atonia. *J Neurosci* 1988; 8: 4790-6.
9. Culebras A. Update on disorders of sleep and the sleep-wake cycle. *Psychiatr Clin North Am* 1992; 15: 467-89.
10. Culebras A, Moore JT. Magnetic resonance findings in REM sleep behavior disorder. *Neurology* 1989; 39: 1519-23.

Received 18 October 1996