Nocturnal hypoglycaemia presenting as somnambulism

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Abbreviation

NREM Non-rapid eye movement

To the Editor: Somnambulism (sleepwalking) is defined as a parasomnia of non-rapid-eye-movement (NREM) sleep during which movement behaviours that normally only occur in the awake state occur during sleep. Somnambulism is mainly a disease of childhood, but it can occur in adults and may be precipitated by environmental factors such as change in sleep schedule (jet lag) or emotional stress [1]. Use of hypnotics, particularly zolpidem, sedatives, narcoleptics or alcohol can also cause somnambulism. Pathologies such as fever, cardiac arrhythmias, epileptic seizures, asthma, obstructive sleep apnoea and psychiatric conditions have also been reported to precipitate somnambulism [2].

An extensive literature search did not reveal any reports of somnambulism being precipitated by hypoglycaemia, which is surprising as somnambulism is frequently accompanied by ‘night eating’. In this report, I describe a type 1 diabetic patient with somnambulism caused by nocturnal hypoglycaemia that resolved and did not recur when her long-acting insulin was administered twice daily rather than once daily.

A 26-year-old white woman who had had type 1 diabetes since the age of 17 years and who, because of intended pregnancy, had recently attempted to intensify her glycaemic control presented with frequent hypoglycaemia. To achieve better control she had changed from twice daily 70/30 human insulin to a basal-bolus regimen of bedtime insulin glargine (A21Gly,B31Arg,B32Arg human insulin), 0.38 U/kg, and preprandial insulin lispro (B28Lys,B29Pro human insulin), 1 U/10 g carbohydrate. With this regimen she achieved an HbA_{1c} of 6.1%, but she also had frequent but mild hypoglycaemia occurring during waking hours. However, she had a history that suggested unrecognised nocturnal hypoglycaemia: at least once a week she awoke with a headache that was accompanied by nausea. In addition, once or twice a week she would, on awakening, recognise that she had been outside her home during the night as her socks, which were clean on going to bed, were covered with dirt. She had no memory of being out of her home during the night and neither her departure nor her return were witnessed by her ‘heavy-sleeping’ husband. As she had a strong family history of ‘sleepwalking’ she recognised that this was the probable cause. However, she had not previously been known to sleepwalk, even as a child.

Her insulin glargine was reduced to 18 U at bedtime, which resulted in higher morning glucose readings but failed to avoid nocturnal hypoglycaemia, which was now being documented with low home glucose monitor readings at 02:00 hours. When 02:00 hours monitoring was discontinued, the out-of-home sleepwalking spells recurred, but were less frequent (one every 2 to 3 weeks) despite precautions being initiated, such as locking the woman’s bedroom door. When the total insulin glargine dose was split to a twice-daily regimen with the injections being administered 12 h apart, glycaemic control improved. Neither

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nocturnal hypoglycaemia nor somnambulism occurred, even during the pregnancy when HbA_{1c} levels varied between 4.9% and 5.5%.

Somnambulism, which is more common during childhood and rare in adults, is primarily diagnosed from the clinical history as episodes seldom or never occur during a sleep study. New-onset somnambulism in an adult without a childhood history, even in the presence of a strong family history, should raise suspicion of an underlying pathological cause. In this case the cause was hypoglycaemia, the correction of which resulted in long-term resolution of the somnambulism. Because of the strong family history it is likely that, in this patient, hypoglycaemia unmasked a genetic predisposition to somnambulism.

Factors that precipitate somnambulism either increase the duration of slow-wave sleep or are disruptive to this stage of sleeping by heightening the delta wave activity that accompanies NREM sleep. While hypoglycaemia has been shown to activate arousal-related neurons in rats, this has not been demonstrated in humans [3]. While a sleep study of teenagers with type 1 diabetes did not show any change in arousal with hypoglycaemia, a sleep study of adults with hypoglycaemia induced by intravenous insulin did show increased arousal accompanied by increased amplitude of delta waves and sleep fragmentation [4, 5]. During sleep, measures of interstitial glucose have shown that glucose levels increased in the majority of patients during rapid-eye-movement sleep, indicating that hypoglycaemia is the cause rather than the result of somnambulism [6].

In conclusion, I have documented for the first time a case of new-onset somnambulism caused by nocturnal hypoglycaemia. Nocturnal hypoglycaemia should be considered as a pathological and correctable cause of somnambulism, particularly in the insulin-requiring diabetic child,

Duality of interest The author declares that there is no duality of interest associated with this manuscript.

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