Lesson of the Week

Transient hemiparetic attacks due to unrecognised nocturnal hypoglycaemia

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Hypoglycaemia may present with focal neurological signs[1] and rarely as hemiplegia.[2] Recently it has been emphasised that many attacks of nocturnal hypoglycaemia may be unrecognised because they may be followed by "rebound hyperglycaemia."[3] We report on a patient who was treated for transient cerebral ischaemic attacks because of recurrent hemiparesis due to hypoglycaemia which was not recognised by the patient or his doctors.

Nocturnal hypoglycaemia is hard to recognise

Case report

A 40-year-old male nursing officer was admitted in 1977 after a transient right hemiparesis had wakened him at 2.30 am but resolved spontaneously within four hours. He had been a diabetic since 1961 and was receiving Lente insulin 60-70 units daily. On admission he had only minimal right-sided weakness and an equivocal plantar response, but these resolved the next day. His blood sugar concentration was 8·9 mmol/l (160 mg/100 ml), and urea, electrolyte, and calcium concentrations were normal. A blood sugar profile the following day was 14·2 mmol/l (256 mg/100 ml) fasting, and 19·2 mmol/l (346 mg/100 ml) and 14·2 mmol/l (256 mg/100 ml) at 1100 and 1500 hours respectively. The platelet aggregation ratio was 0·67 (normal range 0·75-1·20); electrocardiogram, echocardiograph, and left carotid angiogram were normal. It was assumed that he had had a transient ischaemic attack associated with spontaneous platelet aggregation, and he was treated with aspirin and dipyridamole.

During follow-up the patient refused to change to a twice-daily insulin regimen in spite of persistent glycosuria and high concentrations of blood sugar. Some years earlier he had experienced typical hypoglycaemic symptoms (psychic phenomena and sweating) and would not risk a similar episode. He remained well apart from one further transient right hemiparesis on waking.

In 1980, however, he was admitted after six recurrent attacks of transient right hemiparesis on waking. He attributed recovery from these attacks to taking insulin, but this was invariably accompanied by a drink of tea. On admission he was conscious; he had a dense right hemiplegia associated with spasticity, ankle clonus, and an extensor plantar response. There was associated sensory loss but no contralateral signs. The pulse rate was 75/min, blood pressure 130/90 mm Hg, and he was not sweating. The blood glucose concentration was 1·5 mmol/l (27 mg/100 ml) and his signs completely resolved within four minutes after receiving 20 ml of 50% dextrose intravenously. Autonomic function tests were normal. Aspirin and dipyridamole were discontinued. The diabetes had since been controlled with Leoretard insulin, 48 units in the morning, 24 units in the evening. Although he has had one mild hypoglycaemic attack during the day, this was not associated with neurological symptoms.

Discussion

Hypoglycaemia may be misdiagnosed when the presentation is unusual. Lawrence[4] suggested that structural cerebrovascular disease may explain the uncommon predilection to symptoms on one side of the body only during hypoglycaemia; his own left hemiparesis progressed to complete paralysis during attacks of hypoglycaemia. Because a structural disorder was excluded in this patient, however, symptoms may be explained by vascular spasm since attacks of migraine occasionally complicate hypoglycaemia.[5]

Nocturnal hypoglycaemia is often unrecognised because blood sugar concentrations tend to be high. Although this problem may be overcome in some patients by home glucose monitoring most patients have hypoglycaemia between 1 am and 3 am but normal or high concentrations by 7 am.[3] Clinically the condition should be suspected in patients whose diabetes is not well controlled despite moderate insulin doses ($\geq$ 1 U/kg a day) and in those with chronic symptoms. Lassitude, depression, night sweats, difficulty waking, and early morning headaches are common complaints, and less often nocturnal fits and hepatomegaly.[3] The diagnosis may be supported by finding a high cortisol:creatinine ratio ($>55 \times 10^{-6}$) in urine collected between 10 pm and 7 am.[6] Nocturnal hypoglycaemia may be common, as was shown in 21% to 56% of poorly controlled insulin-dependent diabetic patients selected for investigation,[3 7] but its diagnosis requires a high index of suspicion.

When dietary indiscretion has not precipitated nocturnal hypoglycaemia it is most likely to be associated with the use of long-acting preparations taken once a day. Protamine-zinc insulin is virtually obsolete, but Ultralente and Ultratard MC may also have a peak effect as late as 20 hours after administration.[8] The effects of Lente insulin often persist for 24 hours and may be associated with nocturnal hypoglycaemia, particularly when the dose is increased in response to raised mid-morning blood sugar concentrations. Nocturnal hypoglycaemia is more likely to occur with large concentrations and volumes of insulin since these reduce the rate of insulin absorption.[9] Therefore most patients now receive a twice-daily regimen of an inter-

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mediate preparation such as isophane alone or in combination with soluble insulin. Because there is wide inter-subject variation in the duration of action titration of both the dose and the preparation to suit each patient's requirements is necessary. Nevertheless, in two recent series most patients with nocturnal hypoglycaemia had been receiving twice-daily regimens and ultimately responded to an average reduction of about 25% in total insulin dose and 53% in the long-acting insulin dose.[3 7] Thus the smallest evening dose that will control the fasting blood sugar should be prescribed. Intra-subject variability in the rate of insulin absorption may also lead to unexpected hypoglycaemia. Insulin injected subcutaneously is absorbed more slowly from the thigh than from the arm, and is absorbed fastest from the abdomen.[9] Patients should therefore be told to inject a particular dose into the same part of the body.

Requests for reprints should be addressed to JHS.

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Reading for Pleasure

Listen to me

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Many medical schools seem to produce a small but steady supply of doctors who desert Hippocrates to follow the muse of Drama, and the fact that many actors stay in the medical profession is amply attested by those of our teachers who perform rather than lecture, using to the best advantage their clear voices and their twice-told tales. For many years now I have more or less modestly admitted to deriving pleasure from showing off.

Fortunately, I was educated in a medical school whose Christmas pantomime was one of the highlights of the academic year, and three consecutive Dame parts (in *Robinson Crusoe*, *Ali Baba*, and *The Babes in the Wood*) whetted an appetite for the extrovert that remains keen to this day. Although two years in the RNVR afforded me only one chance to be a murderer (Mr Small in *Saloon Bar*), a decision during my service to become a dermatologist and so "never get called at night" allowed me to maintain a connection with the performing arts. It was not until I was about 30 that a charming lady told me I had a beautiful voice, and that compliment directly led to my present hobby—I not only read for pleasure but I do it out loud and in public.

Having many years ago adopted a stage name in order to prevent some ethical pedant from accusing me of seeking professional publicity, I have read poetry out loud in halls, homes, and even hospitals from Beirut to Hong Kong going East, and from London to Los Angeles going West. This almost invariably gives me pleasure and I hope the audiences enjoy it as well, but of course the foundations of my enjoyment come from the considerable amount of reading that has to be done during the construction of a programme.

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Choosing a programme

If I am asked to read to an audience I have not previously met I usually start with a basic programme entitled "English Verse—English Voice." First a few early ballads, *A Lyke-Wake Dirge*, *Lord Randall*, and *Barbara Allen's Cruelty*. The hypnotising repetition of the second and fourth lines in the dirge seems to instill a quiet receptivity even in hearers who have only come for reasons of politeness. I follow the ballads with a group of Shakespeare's sonnets; such masterpieces as *Shall I compare thee to a summer's day?* (No 18) and *Let me not to the marriage of true minds admit impediments* (No 116) are not among the most difficult poems to read or understand, but many people seem surprised both by their simplicity and beauty and find they gain more from hearing than actually reading such verses. I usually include No 130, which is probably the only truly funny sonnet in the series, and I rather like to imagine that one day Shakespeare and his surrounding poetasters were vying with one another in the local inn with woeful ballads to their mistress's eyebrows when they were suddenly punctured by the pot-boy's assertion that his mistress's eyes were "nothing like the sun" and yet, by Heaven, he thought his love "as rare as any she belied by false compare."

There is always a bardolator around who believes that poetry died in Stratford upon Avon early in the seventeenth century, but a short sharp dose of Donne broadens most peoples minds; *Goe, and catch a falling star* and certain of the love poems provide startling evidence that the liberal-minded churchman is not an invention of the twentieth century. If I have done without Donne, the church is represented by Dean Swift's adventures on the way to Ireland—*An Excellent NEW Ballad or The true English Dean to be hanged for a rape*. John Gilpin's adventurous ride in North London ends the first half and shows that good humour and a good rhythm are acceptable throughout the world.

The second half often starts with one of the eighteenth-