CASE REPORT

A case of nocturnal fainting: supine vasovagal syncope

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Supine loss of consciousness is a relatively rare occurrence prompting investigations for underlying causes as diverse as cardiac arrhythmia, hypoglycaemia and nocturnal epilepsy. Neurally mediated syncope is rarely implicated as the cause of symptoms in supine loss of consciousness because of the absence of orthostatic stress and gravitational relative preservation of cerebral perfusion, but we report here on a case of recurrent, atypical and troublesome vasovagal syncope occurring at night while supine. Diagnosis aided by head-up tilt table testing and conservative management brought about complete resolution of symptoms.

KEYWORDS
Vasovagal syncope; Neurally mediated syncope; Supine; Sleep

Introduction

Supine syncope is an uncommon symptom seen most frequently (in the context of cardiovascular disorders) in association with structural heart disease and arrhythmia.1 Vasovagal syncope while supine is a rare occurrence due to the absence of orthostatic stress and the presence of gravitational effects maintaining adequate cerebral perfusion.2 Here we report the case of a 65-year-old man with recurrent nocturnal supine pre-syncope and syncope secondary to an unusual presentation of vasovagal syncope.

The case

A 65-year-old man was referred for investigation of recurrent pre-syncope and syncope. He described pre-syncope on many occasions since 1986, almost invariably occurring while lying flat in bed at night following consumption of a late meal, particularly a fatty or spicy meal. On three of these occasions his symptoms proceeded to syncope. During an episode he felt sweaty, clammy and nauseous, with severe dyspepsia. On recovery he would often vomit and experience further central chest pain. The latter symptom precipitated three admissions into hospital. Surface electrocardiograms revealed no ischaemic changes and cardiac enzymes were not elevated at any time. The patient had also experienced four episodes of erect syncope following prolonged standing in church. The patient had a complex past medical history including atrial fibrillation (warfarinsed and digitalized), hypertension, (controlled by bendrofluazide), hypogonadism, osteoporosis, and arthritis.

Ambulatory ECG and echocardiogram were unhelpful in diagnosis, showing atrial fibrillation with a controlled ventricular rate on the former and normal left ventricular function on the latter. We then proceeded to 70° head up tilt table testing. Eleven minutes into our standard Italian protocol3 he developed his usual pre-syncopal symptoms. Over the next two minutes blood pressure fell before heart rate, with syncope supervening with a minimum heart rate of 26 beats per minute and an unrecordable blood pressure.4 Consciousness was promptly recovered on assumption of the supine position.

Given the length of history and positive tilt test, vasovagal syncope was thought to be the most likely diagnosis. The two atypical features for vasovagal syncope in this instance were that of the supine nature of events and the chest pain. It was hypothesized that the chest discomfort was a manifestation of gastro-oesophageal reflux precipitated by consumption of food late at night, with the resultant pain causing psychic distress sufficient to induce a vasovagal event. Conservative advice was given regarding prevention and management of vasovagal syncope. The patient modified his diet and eating patterns and symptoms resolved without the need for further pharmacological intervention.

Discussion

When a patient presents with a spell of unconsciousness at night, epilepsy, cardiac arrhythmias, sleep disorders, hyperventilation attacks, and hypoglycaemia are all diagnoses usually considered.5 What is not often contemplated as a...
potential cause for nocturnal collapse is vasovagal syncope. For the most part, syncope occurring while the patient is supine tends to argue against most forms of neurally mediated syncope (carotid sinus syndrome possibly being an exception).² Ours is one of only a few cases of supine vasovagal syncope ever reported.

Krediet et al.⁶ report a series of 13 patients, with a mean age 45 years, with recurrent vasovagal syncope interrupting sleep. The patients had histories consistent with vasovagal syncope and most had experienced daytime vasovagal episodes in response to common triggers. Seven patients had positive unprovoked head up tilt tests with typical prodromal symptoms. Interictal electroencephalogram was also performed in seven patients, only one of which showed epileptiform activity. Iskos et al.⁴ report two cases of supine syncope. In both instances syncope occurred just after assuming the supine position, especially at night. One of the patients maintained stable heart rate and blood pressure during a drug free head up tilt table study but upon return to the horizontal position developed a two second pause and concomitant hypotension associated with typical prodromal symptoms. It is suggested that ‘reverse tilt’ (i.e. moving the patient from upright posture to the supine position) may be a useful confirmatory diagnostic test when faced with this entity.

The mechanism of vasovagal syncope is still contentious. Abnormal autonomic responses to various stimuli cause reflex mediated changes in heart rate or vascular tone, to the extent that cerebral perfusion cannot be maintained adequately. One of the precipitants for vasovagal syncope can be reduced cardiac pre-load secondary to peripheral venous pooling.⁷,⁸ In the case of supine syncope this mechanism is unlikely to apply. It is also known that unpleasant emotion stimuli by themselves may, in susceptible people, induce a vasovagal reaction, even in the absence of a gravitational stress.⁹ Situational syncope, for example defecation or cough syncope, is thought to be mediated by stimulation of the medullary vasodepressor region of the brain stem via sudden activation of mechanoreceptors present through out the body (heart, lungs, gut, and bladder).⁷,⁸ In regards to the case presented above, the most likely explanation for syncope is activation of the pain stimulus presumed secondary to gastro-oesophageal reflux, via modulation of autonomic reflexes. It is also possible that stretch receptors in the oesophagus may have been activated.

It is important to remember that neurally mediated syncope can present atypically, as demonstrated by our case. Vasovagal syncope should be considered as a cause of nocturnal syncope after exclusion of more serious diagnoses such as structural heart disease, cardiac arrhythmias, and epilepsy. The misdiagnosis of attacks can have significant consequences for the patient, in particular in regards to driving and inappropriate and invasive investigation and potentially toxic medication.² Vasovagal syncope is an important diagnosis to establish as the condition is benign and the patient can be reassured. Careful history taking including witness history if available and examination is important in establishing the diagnosis. A history of daytime syncope in response to common triggers and a positive tilt test can aid diagnosis.⁶

Conflict of interest: none declared

References