A woman with transient loss of consciousness

Introduction

The differential diagnosis of transient loss of consciousness with spontaneous recovery (TLC) consists of syncope, epilepsy and conversion disorder. The majority of syncopes have a reflex etiology, i.e., vasovagal syncope, micturition syncope or syncope from carotid sinus syndrome.

It is estimated that up to half of the normal population have at least one lifetime episode of TLC; in the vast majority of cases, presumably of reflex origin [9]. Psychiatric and anti-hypertensive medications may contribute to the proneness for reflex syncope [1].

In most cases, history taking will suffice for differentiating the causes mentioned above. Additional tests should only be performed on strict indications. Because of relatively low sensitivity (50%; and specificity, about 80%) of tilt table testing this test should be used conservatively [1].

Interpretation of the medical history

Based on the medical history of our patient, epilepsy is a very unlikely cause of her episodes of TLC. There is no report of synchonic myoclonic jerking, tongue biting or post-ictal confusion.

Almost all clinical clues point to a reflex origin of the reported episodes. The relation to meals (i.e., postprandial hypotension component), sneezing and coughing suggest a reflex mediated etiology. The use of nitroglycerin and standing up as triggers are pathognomonic for such a cause. The use of several anti-hypertensive medications supports this idea.

Some information however is atypical for reflex syncope: the accompanying pain, anxiety — only in case of blood phobia anxiety leads to syncope — and to a lesser extent the frequency of the episodes. Several dozens of episodes in four months is an unusually high frequency for reflex syncope. The pain, anxiety and the frequency are atypical for reflex syncope, but fit comfortably in the picture of a conversion disorder [2]. The patient’s psychiatric history supports this possibility. Based on the history as a whole however we can take reflex syncope as a working diagnosis. One should keep in mind the admittedly less attractive possibility of a combination of two diagnoses.

Interpretation of the physical examination

The patient’s cardiovascular reflex adaptation to standing is normal, with an increased diastolic pressure during orthostatic stress and heart rate increases within normal ranges [10]. Nevertheless there is one relevant finding during the first visit: symptoms of light-headedness while a normal blood pressure (124/86 mmHg) is measured. Light-headedness as a complaint is often very hard to objectify; however with such BP it is most unlikely to be caused by cerebral hypoperfusion. A psychogenic origin emerges. In addition, the low BP (78/45 mmHg) during the second visit should not slip from our diagnostic attention. It would be interesting to know whether the patient experienced at that time symptoms similar to earlier episodes. But also without this, we can make a preliminary diagnosis of iatrogenic vasomotor dysfunction causing reflex syncope. Adapt-
ing the anti-hypertensive medications (i.e. ceasing them) seems indicated.

**Interpretation of additional testing**

From the surplus of additional testing the ECG is probably the most interesting. The normal ECG reduces the risk of a (possibly fatal) arrhythmia to below the population risk for this age and gender. The conclusion based on the medical history that the episodes are most unlikely to be conversion. The TLC observed by the clinicians. The episode starts and hypertension return.

The nutcracker esophagus may explain the retrosternal pain and since such pain may trigger a vasovagal reaction, this favors a syncope diagnosis. However chest pain is a very non-specific complaint and other factors such as anxiety and hyperventilation may also be involved. The amount of patients in whom such pain causes vasovagal syncope is probably very small (a Medline search on “nutcracker esophagus” AND “syncope” gives no literature on the subject). A highly speculative additional explanation for the TLC in combination with the esophagus’ state, would be deglutition syncope. It is thought to be associated with an abnormal vagovagal reflex originating in the esophagus, triggered by swallowing that leads to transient bradycardia [3]. However it is difficult to find a pathophysiological connection between the nutcracker esophagus and deglutition syncope. Moreover, the reported episodes are not typically associated with ingestion of food and, therefore this explanation remains unsatisfactory.

Although there are some arguments for a psychiatric etiology, we preserve the working diagnosis of reflex syncope for the moment, possibly triggered by retrosternal pain. The latter is however speculation.

Now we come to the epicrise of this diagnostic quest: the TLC observed by the clinicians. The episode starts with severe chest pain, anxiety, hyperventilation, eye blinking and arrhythmic muscle tensing. Blood pressure and heart rate increase suggesting high sympathetic state, such as seen during pain or anxiety. Then BP and HR both drop to normal levels (about 160/60 mmHg; 90 bpm) and the patient loses consciousness while still supine. When the patient regains consciousness pain and hypertension return.

**Discussion**

Do these observations support the reflex syncope hypothesis? Fainting while supine is possible, but only occurs during sustained extremely low BP’s, i.e. due to vagal bradycardia’s or cardiac arrests (systolic BP’s < 60 mmHg.; HR < 40 bpm). In our patient neither are present. Although hypocapnia from hyperventilation may contribute to in favororable cardiovascular effects (i.e. decrease in somatic vascular resistance; increase in brain vascular resistance), this cannot explain TLC at a BP of 160/60. There is no reason to assume that our patient has an abnormal cerebral perfusion, or cerebral auto-regulation. Based on these observations and considerations, reflex syncope is excluded from our differential diagnosis.

The observed episode is a textbook example of a conversion reaction leading to TLC. There are “hysterical” prodromal symptoms: anxiety, hyperventilation and arrhythmic muscle tensing and most typically blinking of the eyes [4]. A hypersympathetic state while conscious causes BP and HR to rise to supra-physiological levels. Once the central trigger has been removed when the patient loses consciousness the cardiovascular parameters return to normal values.

There are numerous reports in the literature of similar cases in which conversion reactions were diagnosed during autonomic testing [5–8]. The chest pain could also be of psychiatric origin [4]. However, taking the gastric motility test into account one could speculate that the chest pain is a trigger to the conversion reaction.

But what about the “pathognomonic” symptoms in patient’s history? As remarked earlier, reflex syncope is very common. With her comprehensive anti-hypertensive medication our patient is very likely to have some vasomotor complaints. Therefore some of the episodes in the history will probably have been reflex syncope, i.e. after standing up and use of nitroglycerin. (The combination of reflex syncope and conversion disorder has been described before. [7]) But the episode in the laboratory, and probably the majority of those “in the wild” – considering the frequency – have been psychiatric.

**References**


