CASE REPORT

Syncope: there is more than haemodynamic failure

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SUMMARY

Convulsive syncope may be particularly difficult to differentiate from epileptic seizures. Recurrent syncope are caused by autonomic or non-autonomic failure. In this report, we present a 22-year-old woman who was misdiagnosed as epileptic and whose first symptoms during neurocardiogenic syncope occurred prior to asystole. The patient sensed her aura and reached for the alarm button 2 s before a cardiac asystole was documented in the ECG. We conclude that so far unspecified autonomic mechanisms play a significant role in our patient leading to the symptoms of dizziness and nausea heralding her attacks prior to the occurrence of asystole.

BACKGROUND

This case report documents that symptoms occur prior to asystole in a patient with syncope misdiagnosed as epilepsy. Syncope and some epileptic seizure types share common clinical features. Convulsive syncope may be particularly difficult to differentiate from epileptic seizures. Recurrent syncope are caused by autonomic or non-autonomic failure (neurally mediated syncope or postural tachycardia syndrome).¹ The most common neurally mediated syncope is vasovagal syncope. Provoking factors include standing still, pain, needles, disgust or fear.¹ This observation emphasises that we need to further investigate the mechanisms of symptom generation in syncope. Most likely, not all symptoms are the result of haemodynamic changes related to asystole.

CASE PRESENTATION

We present a 22-year-old female nurse whose medical history was unremarkable except of having had two generalised tonic-clonic seizures at the age of 11. Her physical and cognitive development was normal and her history was unremarkable for trauma, febrile seizures and meningitis. There was no family history of epilepsy. Since the age of 19, she has had episodes of dizziness upto 5–7 times/day which were not triggered by physical or emotional stress and were initially not associated with any impairment of consciousness. However, four attacks were followed by loss of consciousness, falls and jerks of the limbs which were reported by observers. After a diagnosis of epilepsy has been made, levetiracetam, valproic acid, topiramate and lamotrigine were administered but failed to control the attacks. A cranial MRI was normal.

INVESTIGATIONS

During EEG-video-monitoring we recorded three habitual episodes characterised by dizziness and nausea which were associated with atrioventricular

blocks lasting 3–4 s. Interestingly, the patient sensed her aura and reached for the alarm button two seconds before the cardiac asystole was documented in the ECG (figure 1). The video did not reveal any clinical change of the patient before she reached for the alarm button. No interictal or ictal epileptiform discharges were recorded during 11 days of continuous EEG-video recording. The diagnosis of neurocardiogenic syncope was made based on the above-mentioned results of the EEG-video monitoring, Holter ECG (second-degree atrioventricular block) and the results of an abnormal tilt table test.

DIFFERENTIAL DIAGNOSIS

- Epileptic seizures.

TREATMENT

- Pacemaker.

OUTCOME AND FOLLOW-UP

A pacemaker was initially felt to be not indicated by cardiologists. The episodes ceased temporarily with regular endurance training. However, 2 years later attacks recurred with asystoles lasting longer than 7 s which prompted implantation of a pacemaker. Ever since no attacks recurred.

DISCUSSION

We documented clinical symptoms of presyncopal attacks *prior* to the onset of asystole. Thus, the symptoms associated with asystole in our patient were clearly not caused by haemodynamic mechanisms. This is in contrast to common conception that neurological symptoms associated with syncope due to asystole are caused by the haemodynamic failure of blood supply to the brain. However, we cannot exclude a drop of blood pressure prior to asystole which might have caused the initial symptoms prior to the asystole. The mechanism of neurocardiogenic syncope has been described as a response to triggers² which was not the case in our patient as no provoking factors could be identified.

We assume that the two generalised tonic-clonic seizures which our patient experienced during childhood have been a reflection of benign focal epilepsy of childhood. Her prior history of epilepsy may have facilitated to misdiagnose her syncope as drug-resistant focal epilepsy.

We conclude that so far unspecified autonomic mechanisms play a significant role in our patient leading to the symptoms of dizziness and nausea heralding her attacks prior to the occurrence of asystole. Major haemodynamic changes, which in the course of longer lasting cardiac asystoles, may probably be responsible for loss of consciousness, falls and jerking of her limbs can be excluded as the cause of her

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Figure 1 Onset of dizziness/nausea (muscle artefacts caused by movement towards the alarm button=open arrow) preceded onset of asystole (solid arrow) by 2 s.

initial symptoms of dizziness and nausea since their onset clearly preceded the cardiac asystoles. Further polygraphic recordings including blood pressure may help to identify the mechanisms involved in the generation of such sensations prior to asystole.

No similar cases have been published.

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Learning points

- Not haemodynamically clinical symptoms induced in syncope.
- Differential diagnosis syncope versus epileptic seizure.
- Importance of EEG-video-monitoring for establishing the diagnosis.

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