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CASE REPORT

An obstetric patient with neurocadiogenic syncope

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ABSTRACT
We report the peripartum management of a 29-year-old primigravid patient with neurocardiogenic syncope, which had been diagnosed six years previously on tilt-table testing. General principles were applied to minimise the risk of precipitating syncopal episodes. She had an uneventful ventouse-assisted vaginal delivery under epidural anaesthesia in our obstetric high dependency unit. The optimum management of these patients has yet to be established.

Keywords: Neurocardiogenic syncope; Obstetric anaesthesia

Introduction

Syncope is a common problem with multiple aetiologies. It is defined as a transient, self limiting loss of consciousness usually leading to collapse. The onset of syncope is relatively rapid. Recovery is spontaneous, complete and usually prompt. The underlying mechanism is transient global cerebral hypoperfusion.[1]

Neurocardiogenic syncope, also known as vasovagal syncope, vasodepressor syncope, neurally mediated hypotension and bradycardia syndrome,[1–3] is a common cause of syncope.[4] However, only a minority of individuals suffer severe and/or repeated symptoms. Catastrophic events including prolonged periods of hypotension and cardiac arrest have been reported.[5–7] This heterogeneous syndrome is characterised by inappropriate vasodilatation and relative or absolute bradycardia or asystole, resulting in inadequate blood pressure to maintain cerebral perfusion.[8] Episodes are triggered by increases in catecholamines or reductions in preload.[9]

Obstetric patients with neurocardiogenic syncope present an anaesthetic challenge, as there are no clear guidelines regarding their management. We found only two previous case reports on the planned anaesthetic management of these patients.[10] We aim to highlight the anaesthetic considerations relevant to this underreported condition.

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Case report

A 29-year-old primigravid woman presented at 38 weeks of gestation to our obstetric unit for anaesthetic review and formulation of a delivery plan. She had experienced fainting episodes for the last seven years. A cardiologist had diagnosed neurocardiogenic syncope by tilt-table testing six years previously. Her symptoms were precipitated by standing still for periods of time (about ten minutes while pregnant), anxiety and hot baths. A brief prodromal phase of feeling faint usually preceded syncopal episodes, during which she lay still with a vacant gaze. Consciousness was typically regained within seconds, although episodes lasting several minutes had occurred. Management recommended by the cardiologist included advice on avoiding precipitating situations, maintaining a high fluid intake, adopting a sitting or horizontal posture if feeling faint and prescription of the β blocker bisoprolol. Her syncopal episodes had reduced in frequency with this treatment. Bisoprolol was discontinued in early pregnancy due to a risk of intrauterine growth restriction. However, the plan was to resume the bisoprolol if symptoms became problematic. Fortunately, she managed reasonably well with conservative measures, having experienced only two syncopal episodes in pregnancy, with the last nearly two months previously. She was otherwise fit and healthy and there had been no complications in the pregnancy. An anaesthetic plan was made incorporating certain general principles, including minimising pain and anxiety, hunger and dehydration. Aortocaval compression should be avoided as should prolonged standing leading to venous pooling in the lower limbs. Vasodilating drugs should be avoided and prolonged Valsalva manoeuvres kept to a

minimum. Blood loss should be anticipated and corrected promptly.

Delivery was planned to take place in our high dependency room on delivery suite, staffed by specialist midwives trained in obstetric high dependency care. Facilities included immediate access to invasive cardiovascular monitoring (central venous and arterial), in conjunction with a range of standard vasoactive drugs and external cardiac pacing.

Our patient presented in early labour at 39^{+5} weeks of gestation. Her cervix was 2 cm dilated, and the membranes intact. She was not thought to be in established labour, but was admitted for observation. A calm atmosphere with dimmed lights was maintained and the need for analgesia was assessed regularly. She was encouraged to eat and drink freely to avoid hunger and dehydration. When lying in bed, a full left or right lateral position was maintained to prevent aortocaval compression. Prolonged standing was avoided. Graduated compression stockings were fitted to improve venous return.

Labour became established with progressively stronger contractions, requiring Entonox. Thereafter her oral intake was restricted to water. Regular antacid prophylaxis of oral ranitidine 150 mg every 6 h was prescribed. Monitoring was established with continuous electrocardiogram (ECG), oxygen saturation and non-invasive blood pressure measurement. Although the designated midwife was not expected to interpret the ECG, it was important that it was immediately available for medical staff to interpret, if needed.

Over the next 3.5 h, her cervix dilated to 7 cm. Although early epidural analgesia had been agreed, she continued to decline this as her pain was well controlled with Entonox and she remained asymptomatic. As she was no longer maintaining her oral fluid intake, she was given an infusion of Hartmann's solution at 200 mL/h through a 16-gauge peripheral venous cannula inserted under local anaesthesia. She progressed well until the second stage of labour, without any symptoms of pre-syncope, and continued to decline epidural analgesia. However, 45 min after full cervical dilatation, she felt unable to continue pushing without feeling faint. She then requested epidural analgesia. Following a 500-mL bolus of Hartmann's solution given over 20 min, the epidural cannula was sited easily in the left lateral position. She was given a 5-mL bolus of 0.125% levobupivacaine, followed 10 min later by a 10-mL top-up of 0.125% levobupivacaine plus fentanyl 100 μg. Her prodromal symptoms were much improved with epidural analgesia and she was able actively to push again. As blood pressure, heart rate and oxygen saturation remained stable, invasive monitoring was not deemed necessary at this point. However, an intravenous oxytocin infusion for augmentation of uterine contraction was required. In accordance with our standard unit guidelines, oxytocin 10 units was added to 500 mL of 0.9% saline. The infusion was initiated at 1 mU/min (3 mL/h), and increased over the next hour and a half to 4 milliunits/min (12 mL/h). Three hours after epidural insertion, maternal fatigue prompted an assisted ventouse delivery, which resulted in the uncomplicated delivery of a baby boy with Apgar scores of 9 and 10, at 1 and 5 min respectively. Good analgesia for the delivery was achieved with one 5-mL bolus of 0.25% levobupivacaine. The estimated blood loss was 300 mL. The oxytocin infusion rate was then increased to 125 mL/h.

After delivery, she remained cardiovascularly stable and asymptomatic with non-invasive monitoring for some four hours after delivery. She was subsequently discharged to our post natal ward. Having remained stable, she was discharged home 5 h later. There were no postpartum complications.

Discussion

We found only two previously published case reports on the planned anaesthetic management of obstetric patients with neurocardiogenic syncope.[10] Both patients delivered in the same obstetric unit in the UK. Antenatally, one had been managed conservatively in the community, whereas the other had an atrial-based pacemaker in situ. Both had invasive blood pressure monitoring and continuous ECG monitoring in labour. Epidural analgesia was established in both. The first patient required caesarean section, performed under epidural anaesthesia, after a failed attempt at ventouse delivery. Epidural analgesia had the added benefit of controlling blood pressure in the second patient, who had preeclampsia. This woman went on to have an uncomplicated ventouse-assisted vaginal delivery.

Neurocardiogenic syncope may be precipitated by central or peripheral stimuli,[9] but many episodes have no specific provocation.[11] Central triggers include those that increase catecholamine secretion such as emotional stress, pain and unpleasant sights, sounds and smells.[3,9] Peripheral triggering occurs due to a reduction of venous return to the heart, as commonly occurs on standing, when 300-800 mL of blood are displaced to the veins in the lower body.[3,9] Central transmission of the afferent stimuli is followed by the vasomotor centre responses of paradoxical withdrawal of peripheral sympathetic tone and a surge in parasympathetic tone, which, in turn, causes vasodilatation and bradycardia.[3,9]

The exact pathophysiology is unclear and complex. A variant of the Bezold-Jarisch reflex has been widely proposed as a means by which a reduction in venous return may provoke neurocardiogenic syncope. In this reflex pathway the reduction in preload leads to reduced ventricular volume. Subsequent baroreceptor-mediated sympathetic stimulation leads to increased ventricular inotropy.[11,12] Vigorous contraction of the