Severe vasovagal attack during regional anaesthesia for Caesarean section

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A patient experienced a severe vasovagal attack during regional anaesthesia for elective Caesarean section. The combination of vagal over-activity and sympathetic block produced profound hypotension that threatened the life of the mother and infant. The vasovagal syndrome is described, and its prevention and management discussed.

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Case report

A 29-yr-old woman, parity 1, presented at 38 weeks' gestation for elective Caesarean section because of a high fetal head and a history of previous Caesarean section. Her first pregnancy had ended in an emergency Caesarean section for failure to progress, performed under epidural anaesthesia. Pre-anaesthetic assessment was unremarkable, with no medical problems and a healthy pregnancy. She weighed 70 kg and was 158 cm tall. A combined spinal–epidural, which was our standard technique at the time, was discussed with the patient and consent obtained. Routine antacid premedication was prescribed, consisting of cimetidine 200 mg the night before and the morning of surgery, and 0.3 M of sodium citrate 30 ml in the anaesthetic room.

The patient arrived in the anaesthetic room looking and feeling well, accompanied by her husband. Monitoring of ECG, oxygen saturation and arterial pressure was commenced, showing a heart rate of 90 beat min^{-1} and arterial pressure 133/64 mm Hg. After subcutaneous infiltration of 1% lidocaine, a 16-gauge peripheral line was inserted and a fluid preload of Gelofusine 500 ml was given. With the patient in the sitting position and using an aseptic technique, a total of 4 ml of 1% lidocaine was infiltrated at spinal interspaces L2–3 and L3–4. A lumbar epidural catheter was sited at the L2–3 interspace. After a negative aspiration test, lidocaine 1 mg kg^{-1} was injected into the epidural catheter. During injection of this test dose, the patient complained of feeling dizzy and faint, and became pale. It was noted that she had a heart rate of 60 beat min^{-1} and arterial pressure of 70/35 mm Hg. On direct questioning, the patient denied any symptoms (such as peri-oral numbness, tingling, visual or auditory disturbances or a metallic taste) that would suggest the onset of local anaesthetic toxicity associated with i.v. injection.

A second anaesthetist administered ephedrine 6 mg while the epidural catheter was quickly secured with the intention of lying the patient down. However, with the patient still sitting, the symptoms regressed; heart rate increased to 90 beat min^{-1} and arterial pressure to 90/60 mm Hg. One minute later, arterial pressure was 110/65 mm Hg. There was no sign of a rapidly developing motor block or a high sensory block to suggest subarachnoid catheter placement. A diagnosis of vasovagal attack was made, and it was decided to proceed.

An infusion of ephedrine 30 mg in Gelofusine 500 ml was commenced and spinal anaesthesia performed with a 27-gauge Whitacre needle at the L3–4 interspace, again with the patient in the sitting position. Clear cerebrospinal fluid was obtained and 0.5% hyperbaric bupivacaine 15 mg was injected over approximately 30 s.

Immediately after starting the injection, before one would expect to see cardiovascular changes, the patient once again complained of feeling faint. Infusion of ephedrine was turned to maximum flow, the spinal injection completed, and the patient immediately positioned supine with left lateral tilt. Heart rate was 80 beat min^{-1} but the patient looked pale and grey. Oxygen was administered via a Hudson mask. A second infusion of Gelofusine 500 ml was commenced under pressure and further i.v. bolus doses of ephedrine were given. Ten minutes later, Gelofusine 1500 ml, ephedrine 70 mg and atropine 600 μg had succeeded in producing only brief periods of normotension interspersed with severe hypotension and near syncope. It was decided that prompt

delivery of the baby was required for its survival and to remove any contributory aortocaval compression.

The anaesthetic level was T4 to light touch, and Caesarean section proceeded rapidly without complication. A female infant was delivered; she was pale and floppy with a heart rate of 60 beat min^{-1}. Bag and mask resuscitation produced prompt correction of the bradycardia but at 10 min the baby was still floppy and respiratory efforts were laboured. The umbilical cord pH was 6.8. The baby was transferred to the special care baby unit where, over a 2-day admission, she slowly recovered normal muscle tone and respiratory pattern. Postoperative recovery of the patient was unremarkable although she had virtually no memory of the events during the Caesarean section. Three years later, there are no apparent sequelae for mother or baby.

Subsequent careful questioning revealed two relevant episodes in the patient's medical history: one was an unrecorded episode of symptomatic hypotension during the epidural top-up performed immediately before her previous Caesarean section and the other was an episode of profound vasovagal syncope during a hot air balloon ride while several hundred feet above the ground.

Discussion

The differential diagnosis of this haemodynamic collapse includes: i.v. injection of the epidural test dose; high spinal block; or a combination of a vasovagal reflex with spinal-induced sympathectomy. We believe the latter to be the cause because there were no symptoms of local anaesthetic toxicity with the epidural test dose, and the final spinal block was at a level of T4 to touch. The spinal dose of bupivacaine 15 mg was large and is no longer our current practice, but in our view it contributed to the haemodynamic picture rather than being the sole cause.

Vasovagal syncope is a heterogeneous condition. The vagal response can be initiated by external stimuli (e.g. venepuncture or visual input). An alternative mechanism is by stimulation of the Bezold–Jarisch reflex in which low ventricular volumes result in high intraventricular pressures caused by the empty ventricle contracting against itself. This reflex causes parasympathetic activation *and* sympathetic withdrawal resulting in hypotension and bradycardia. The presence of sympathetic withdrawal is supported by the fact that atropine administration may prevent bradycardia but not hypotension.[1] Neurally mediated 'active' vasodilatation may also be involved, as skeletal muscle vasodilatation is greater that that caused by sympathetic withdrawal alone.[1] The Bezold–Jarisch reflex is postulated to be responsible for the condition called 'malignant vasovagal syndrome' or 'neurally mediated syncope' in which patients have recurrent syncope without warning or precipitating stimuli.[2] This condition is often tilt-inducible, and has been labelled 'malignant' because patients can suffer trauma during the sudden unexpected syncopal episodes.[3]

Vasovagal episodes in the anaesthetic room before regional anaesthesia for Caesarean section are not, in our experience, rare. They can occur in either the patient or the partner, and before any procedure has been performed. A vasovagal reaction to venepuncture is common, particularly in the young or in patients with a history of fainting.[4] In this patient, symptoms occurred during placement of the epidural catheter, then appeared to resolve, only to return at the start of subarachnoid injection. The procedure could have been halted at either of these times, but as previous experience indicated that vasovagal activity could be suppressed readily with ephedrine and atropine, it was decided that the best option was to complete the procedure as quickly as possible. In retrospect, this judgement was unwise. The patient may either have suffered a recurrent vasovagal episode or the primary episode may have been prolonged and had only been masked transiently by ephedrine. The outcome was that the combination of vasovagal overactivity and sympathetic block, with aortocaval compression as a likely contributory factor, produced circulatory collapse that was potentially life-threatening to the mother and infant. Just 10 min of this resistant hypotension produced an umbilical cord pH of 6.8 and significant early morbidity in the baby.

How could this woman have been better managed? At the first occurrence of vagal activity, after appropriate treatment with vasoactive drugs, Caesarean section could have been delayed while anticholinergic and sedative premedications were administered. However, anticholinergic premedication may prevent vagally mediated bradycardia, but does not prevent hypotension which is the likely primary event.[5] While there is no rationale to suspect that larger fluid preload might have prevented a vagal reaction, it might have reduced the haemodynamic consequences. Thereafter, a slow onset regional block using the epidural alone or in combination with a much reduced dose of intrathecal local anaesthetic might have been more appropriate. The sitting position should have been abandoned and anaesthesia performed in the operating theatre to facilitate rapid delivery of the baby in the event of haemodynamic collapse. This approach would also be appropriate for an elective case with a history of fainting episodes.

In summary, vasovagal episodes superimposed on regional anaesthesia are a significant hazard. Haemodynamic monitoring and constant vigilance for sudden bradycardia are mandatory during regional anaesthesia, and early and aggressive management of any vasovagal episode is recommended. In the light of this case, it cannot be assumed that anticholinergic drugs alone or in combination with ephedrine will always effectively prevent or treat this problem, and rapid delivery of the infant may be necessary.

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