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Circulatory collapse following epidural bolus for Caesarean section a profound vasovagal reaction? A case report

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ABSTRACT

INTRODUCTION: Reduced blood pressure is commonly seen associated to spinal anaesthesia for Caesarean section and efforts to reduce its occurrence and its magnitude is common practice. Cardiovascular collapse requiring cardio-pulmonary resuscitation after putting the spinal/epidural block for Caesarean section is however a rare but most dramatic event.

PRESENTATION OF CASE: We describe a case with sudden short loss of circulation, circulatory collapse, short after start of emergency Caesarean section in top up epidural anaesthesia (3 + 12 ml ropivaciane 7.5 mg/ml), requiring CPR. The neonate was delivered during CPR with Apgar 1, 10, 10 at 1, 5 and 10 min. Circulation was restored following 60–90 s of CPR and administration of 0.5 mg adrenaline. No cardioversion was administered sinus rhythm was regained spontaneously. The mother and child had a further uncomplicated course. No signs of cardiac damage/anomaly, emboli, septicaemia, pereclampisa or local anaesthetic toxicity was found. The patient had prior to the decision about Caesarean section had fever and was subsequently relatively dehydrated.

DISCUSSION: The patient had a fast return of sinus rhythm following birth of the child, without cardioversion. None of common causes for cardiac arrest was found and the patient an uncomplicated post Caesarean section course. The combination of epidural induced sympathetic block and reduced preload possibly triggered a Bezold-Jarisch reflex with a profound vasovagal reaction.

CONCLUIOSN: A structured plan for the handling of cardiovascular crisis must be available wherever Caesarean section are performed. Adequate volume loading, left tilt and vigilant control of circulation following regional block performance is of outmost importance.

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1. Introduction

Cardiac arrest may be seen on rare occasions during pregnancy associated to cardiac disease, severe bleeding, pulmonary embolism and amnion fluid emboli. Cardiac arrest requiring CPR associated to spinal or epidural anaesthesia is also a known but likewise a most uncommon event. Circulatory collapse following spinal or epidural block for Caesarean section is a rare and most dramatic event putting mother and child at risk.

2. Case presentation

The patient described has provided explicit informed consent for this presentation. A 37-year old healthy Asian female, BMI 32 before pregnancy (weight 80 kg, length 158 cm), nulli-parous was admitted for induction of vaginal delivery in gestation week 42+ 0 two days. Several doses of misoprostol, dinoprostin and oxytocin were administered with limited effect. The patient was instructed not to eat or drink from the second day of induction. She received a well functioning epidural analgesia. At the same time a temperature of 39.2C was detected and antibiotics were administered. Three hours later Caesarean section was decided due to slow progress. The temperature had declined and the patient was brought to the operating theatre. An infusion with Ringer lactate 1 000 ml was started and infused rapidly, an epidural top up bolus of 3 + 12 ml ropivacaine 7.5 mg/ml with 50 μg fentanyl added was administered. Patient was placed in supine position and tilted to the left side. Heart rate and blood pressure was recorded each minute. Heart rate was around 120 beats/min. Repeated doses of phenylephrine 0.05 mg were provided as blood pressure was instable and systolic pressure was hard to maintain above 100 mmHg. Approximately 20 min after top up of the epidural the block reached Th 3–4. The WHO checklist was done and operation started. The patient complained about hard to breathe. Bradycardia was seen and Atropine 0.5 mg was given at a heart rate of 43 beats/min but heart rate continued

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to fall accompanied by a sudden drop in blood pressure. The patient became unresponsive, ECG and pulsoximetry showed no pulse. CPR was rapidly initiated, patient was intubated, 0.5 mg adrenalin i.v. was given and the baby was delivered instantaneously. Heart beats and blood pressure was restored within 60–90 s, return of circulation was confirmed 90 s after the pulse arrest. Sinus rhythm was spontaneously regained without further interventions, no cardioversion. A high initial heart rate around 160 beats per minute and systolic blood pressure first measured 166 mmHg and oxygen saturation 97% was observed. The neonate had Apgar scores 1, 10, 10 at 1, 5 and 10 min. No signs of increased bleed (peroperative bleed was 800 ml) or intra-uterine abnormalities were observed. Twelve lead ECG and blood tests showed no signs of myocardial damage and echocardiography tight after the section was normal. CT scan performed right after end of surgery did not show any signs of pulmonary embolism or aortic dissection. No septicaemia was found, blood cultures were negative, the infection was assessed as an endometritis and antibiotics continued. No signs of local anaesthetic toxicity was noticed. Mother and child had an uncomplicated postoperative course.

3. Discussion

US figures describing cardiac arrest during in-patient care for delivery being 1 in 12000 admissions and that there is a slow increase. Globally it is estimated that there are some 800 maternal death daily. The American Heart Association has recently issued an update around the handling of the pregnant mother experiencing cardiac arrest [1]. Montufar-Rueda and Gei published a comprehensive review around cardiac arrest in pregnancy in 2015 stating that most common causes were associated to profound bleed, eclampsia and infection, sepsis [2]. Indeed common causes for sudden cardiac arrest associated to pregnancy and delivery should be early and systematically considered as stated in Advanced Life Support resuscitation guide lines, Reversible Causes of Collapse the 4H's and the 4T's) [3].

There is a study from Thailand reporting the incidence of cardiac arrest associated to spinal anaesthesia. There were 11 cardiac arrests among 40,271 cases of spinal anaesthesia corresponding to an incidence of 2.73 per 10,000 anaesthetics. Among 11 patients who arrested, there were 5 cases of caesarean delivery; 5 cardiac arrest among 11,291 spinals for caesarean section [4]. All blocks where performed with bupivacaine and 3 of 5 to a level of Th 4, 1 to Th 2 and 1 to an unknown height. There are also several case reports around circulatory collapse during regional block for caesarean section. In 1996 Scull and Carli presented a case where a 31-year-old Afro-Caribbean woman (weight 76 kg, height 1.68 m), ASA I, presented at term in spontaneous labour [5]. There was a slow progress and the child showed 2 episodes of distress and it was decided to have a Caesarean section. She had a spinal block with 0.75% hyperbaric bupivacaine 2 ml and preservative-free morphine 0.25 mg providing a sensory block to temperature from T2 to S5. After an uncomplicated procedure and delivery of a healthy neonate perioperative bleed 700 ml she became unresponsive and without pulse on arrival in the recovery room. She was rapidly and successfully resuscitated. ECG and lab tests where normal. The authors comment that cardiac arrest may occur following regional anaesthesia. The combination of sympathetic block and reduced venous return may activate a so called Benzold–Jarish reflex with resultant profound bradycardia and circulatory collapse. Ou et al. described a similar case, a 35-year-old parturient with gestational diabetes mellitus history that developed a circulatory collapse, sustained nearly fatal Bezold-Jarisch reflex during Caesarean section under spinal anaesthesia [6].

Jang et al. describes a 39-year-old pregnant woman with episodes of non-sustained ventricular arrhythmia and newly developed vasovagal syncope during pregnancy. She was undergoing section in spinal block in week 38 and immediately after the placental expulsion, a sudden severe bradycardia, followed by a cardiac arrest occurred. The patient fully recovered after prompt cardiopulmonary resuscitation with chest compression, manual ventilation with oxygen, rapid injection of epinephrine and hydration. The event was assessed as vasovagal reaction caused by combination of spinal sympathetic inhibition and the placenta expulsion [7].

Our patient had rapid return of circulation following delivery of the neonate and thus restoration of preload. Adrenalin was also provided although small dose. There was a direct return of sinus rhythm and no need for defibrillation. Ecocardiography, ECG and blood tests where unremarkable. No signs of myocardial damage, arrhythmias, pulmonary emboli, amnion fluid emboli or sepsis were identified. There was no excessive blood loss, no signs of preeclampsia or allergic reaction, thus common causes for cardiac arrest were excluded [8]. There were no signs of local anaesthetic toxicity, no involuntary movements of signs of seizures. Thus most common causes were excluded and taking in consideration the rapid restoration of sinus rhythm and circulation made us suspect a profound vaso-vagal reaction.

The Bezold reflex has a long history, described initially already in 1867 by von Bezold in 1867 and was later revived by Jarisch [9]. Kinsella and Tuckey provided a comprehensive review around Perioperative bradycardia and asystole in 2001 [10]. Reflex cardiovascular depression with vasodilation and bradycardia has been variously termed vasovagal syncope, the Bezold-Jarisch reflex and neurocardiogenic syncope. The circulatory response changes from the normal maintenance of arterial pressure, to parasympathetic activation and sympathetic inhibition, causing hypotension. This change is triggered by reduced cardiac venous return as well as through affective mechanisms such as pain or fear. It is probably mediated in part via afferent nerves from the heart, but also by various non-cardiac baroreceptors which may become paradoxically active. This response may occur during regional anaesthesia, haemorrhage or supine inferior vena cava compression in pregnancy; these factors are additive when combined. Also Tsai et al. describes pathophysiology, mechanism of action and treatment in a review from 2006 [11]. Regional anesthesia, decreased venous return, hemorrhage and abnormal fetal presentation cumulatively increase the risk of vasovagal syncope in cesarean section patients. When a vasovagal response occurs, ephedrine is the drug of first choice because of its combined action on the heart and peripheral blood vessels. Epinephrine must be used early in established cardiac arrest, especially after high regional anesthesia.

Fluid loading and vasoconstriction for prevention and treatment of low blood pressure is standard of care [12]. There is clear evidence in favour for phenylephrine for the management of hypotension associated to Caesarean section spinal anaesthesia by means of better foetal pH and less increase in Base Excess with similar or better blood pressure control [13]. However ephedrine is associated to less bradycardia [14]. One may argue whether general anaesthesia should have been preferred technique because of the fever. Top-up epidural was however considered adequate as the fever had settled. The volume of epidural bolus dose can be argued, considered huge and have without doubt contributed to the sympathetic block.

We consider a reasonable cause for the short lasting but dramatic circulatory collapse in our patient to be a profound vasovagal response elicited by the combination of the epidural bolus sympathetic blockade in combination hypovolemia. Hypovolemic due to fever and reduced preload associated to her supine position, even though left lateral tilt. A vasovagal reaction caused by a Bezold-Jarisch reflex. It seems of importance to have in mind whenever

regional blocks are provided in the pregnant female that adequate preload should be secured by thoroughly left tilt and adequate volume load. Phenylephrine should be administered to maintain a systolic blood pressure above 90 mmHg. Ephedrine and atropine should readily available and early administered when signs of hypotension and bradycardia is seen.

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Conflicts of interest

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Ethical approval

Not applicable, the patient has been informed and provided explicit consent to the presentation of this case report.

Consent

The patient has provided consent for an anonymous presentation of the event.

Author contribution

All authors have contributed equal to the writing/preparation of this case report.

Guarantor

None.

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