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Severe Hypertension after Spinal Anaesthesia: A Case Report

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Abstract: *Patient with known antiphospholipid syndrome and controlled hypothyroidism suffered an anal sphincter rupture during labour. Spinal anaesthesia was given for the surgical repair procedure. Patient showed bradycardia, hypertension (>180/120mmHg) and complained of severe headache shortly after administration of spinal anaesthetics. Intracranial hematoma or cerebral venous sinus thrombosis was excluded with immediate CT-scan. Hypertension was treated with beta-blockers in the ICU, where the urinary bladder was scanned revealing a full bladder and 1900 ml urine was collected. The symptoms subsequently subsided.*

Patient comorbidities presented diagnostic and therapeutic challenges. The event presents an important phenomenon of paradoxical autonomic presentation during spinal anaesthesia.

Keywords: *Hypertension, Spinal Anaesthesia, Urinary bladder distension.*

1. Introduction

Perioperative hemodynamic instability and post-operative headache are common observations during spinal anaesthesia. In the absence of medical comorbidities, the side effects are usually mild and respond rapidly to treatment. However, in the presence of severe or rare comorbidities, paradoxical presentation may be witnessed. We present an incidence of severe hypertension (HT) and headache immediately after spinal anaesthesia in a patient with known antiphospholipid antibody syndrome (APS).

2. Case Report

Thirty seven year old female with known antiphospholipid syndrome, had grade 3 sphincter rupture after vaginal delivery. She had history of multiple abortions and well treated hypothyroidism, but no history of phaeochromocytoma, neuroendocrine neoplasm and hypertension before pregnancy or preeclampsia. The APS was controlled by regular anticoagulant therapy. As per the Danish national guidelines, the patient received low molecular weight (LMW) heparin during the pregnancy, which was stopped 36 hours before the event as the patient went in labour. Patient was posted for repair of sphincter repair. During pre-anaesthesia visit the neurological examination was normal. On arrival in the theatre, routine monitors (ECG, pulse oximeter and non-invasive-blood pressure) were initiated. Her vital parameters were heart rate (HR) 90/min, blood pressure (BP) 110/70 mmHg and a respiratory rate of 20 per minute.

Spinal anaesthesia (SA) was performed in sitting position, after asepsis with 0.5% alcoholic chlorhexidine and single puncture in the L3-4 interspace with a 27G pencil point, followed by 0.5% (15 mg) hyperbaric bupivacaine. The onset of sensory and motor blockade was assessed in the supine position. After 5 minutes the patient developed bradycardia with a HR of 40, which was settled to 90 per minute after 0.6 mg Atropine. Within few minutes the patient complained of severe headache and the BP increased to 180/120 mmHg, but without other neurological symptoms apart from the motor and sensory blockade following the spinal anaesthesia. Intravenous metoprolol was administered in incremental doses to attenuate BP, and the patient was shifted to ICU for further stabilization. To exclude a cerebral thromboembolic event the patient was taken to a CT-scan with contrast, which did not reveal any signs of intracranial event.

After returning to ICU the urinary bladder was scanned and revealed a full bladder. With the following catheterisation 1900 ml of urine was collected. The patients headache complaints

slowly subsided and the BP stabilized to 130/95mmHg with a heart rate of 85/min and the surgical procedure was carried out as the effect of sensory and motor block still was present. After the recovery, the patient had no neurological deficit and was discharged from the hospital on the 4th postoperative day.

3. DISCUSSION

The case is unique as HT after spinal anaesthesia is a relatively rare observation. Additionally, sudden onset of severe HT and headache generated a speculation of intracranial thromboembolic event as the patient´s clinical history made room for several diagnostic and therapeutic challenges. The first presenting symptom in our case was bradycardia, which is not in frequent after SA and therefore was registered as a usual finding. However, the abrupt increase in BP simultaneous with precipitous onset of headache especially in the absence of common triggers of perioperative hypertensive emergencies was a challenging observation. Post dura-puncture headache appeared less relevant. Its immediate presentation is rare as the usual onset time is 24-48 hours.

Although, the headache in this situation may appear to be a reflection of the rise in BP, its appearance simultaneous with HT and bradycardia raises suspicion of intracranial hypertension secondary to intracerebral hematoma or acute central venous sinus thrombosis (CVST). CVST may be relevant in our case. Headache is the most common (88%) and usually the first symptom and approximately 10% of cases have a thunderclap outbreak, mimicking a subarachnoid haemorrhage[1]. Although the symptoms are variable and non-specific and the diagnosis often delayed (median of 7 days) from the onset of clinical manifestations, we speculated that the patient´s hypercoagulable state related to APS and the pause of the anticoagulant medicine for 48 hours theoretically could have created an asymptomatic, subtotal occlusion of the cerebral venous and dural sinuses. During anaesthesia and positioning for surgery, aggravation to total occlusion leading to symptoms could therefore not be fully refuted. However, the subsequent CT scan examination revealed no signs of CVST and SDH. Although an encouraging finding, it also increased the therapeutic challenge about the symptom triad i.e. bradycardia, hypertension and headache.

The ICU finding of production of 1900 ml urine from the urinary bladder and subsequent stabilization of symptoms fortified the consideration of association of bladder distension to the triad of symptoms. The urinary bladder tone gets reduced during pregnancy to accommodate increased urine production. The bladder hypotonia persists in the immediate postpartum period making it prone to over distension, compounded by a physiological postpartum diuresis. The patients are usually encouraged to urinate at least every 2-3 hours to avoid bladder retention. Failing may increase the risk of over distension, especially in the first pregnancy and has shown to trigger autonomic response leading to diverse symptoms including bradycardia, HT, arrhythmias or even asystole [2]. The characteristic symptoms of pain and lower abdominal discomfort may be masked by the regional anaesthesia, where bladder filling perception is abolished as in our case. Furthermore, the autonomic response is dependent on the presence of intact and uninhibited afferent pathways ascending in the spinotegmental tract, which run through the lateral funiculus of the spinal cord from the bladder to the brain. In contrast to that, hemodynamic disturbances are observed even under spinal and general anaesthesia [6-7], where spinal cord along with the sympathetic presynaptic neurons are blocked by the spinal anaesthetic, creating space for further discussion.

The alternative possible explanation is autonomic dysreflexia (AD). Classic autonomic dysreflexia is observed predominantly in patients with spinal cord lesions over T6 level and presents with bradycardia, arrhythmias and abrupt onset HT up to 260 mm Hg systolic and diastolic blood pressures >170 mm Hg[5].

However, there are several significant differences. It is classically elicited by visceral and not somatic stimulation at an anatomic level below the cord lesion. Blacker et al.[6] have, however, reported an unusual case of AD like symptoms in a patient with spinal cord lesion below at T12 level postulating that, low thoracic spinal cord lesion can still result in a physiologically distinct autonomic dysreflexia-like phenomenon in the operating room and the reflex can also be triggered by somatic stimulus. Similarly, Roche et al.[7] reported a series of 5 African American male patients with low thoracic or lumbar traumatic paraplegia exhibiting severe non-operative episodic hypertension and autonomic dysfunction. Using

a clonidine suppression test, these patients were shown to have a central origin of their autonomic dysfunction. Other case reports suggest that autonomic dysreflexia is not limited to complete spinal cord lesions or injuries at T6 or above, as it has been reported with incomplete cord lesions from multiple sclerosis.[8]

Furthermore, our patient had hypothyroidism with normal TSH-levels. As per report from Mahajan etal. [9], autonomic dysfunction with prominent sympathetic function abnormality may be seen without relationship between the autonomic function score and TSH or TPO levels in hypothyroid patients. Therefore there is room to speculate that, the inherent autonomic imbalance in our patient may have contributed to the exaggerated response to bladder distension. Another theoretical justification of AD in the absence of spinal cord injury is transient spinal cord ischemia. Sonia et al.[10] Postulated the association of Polycythemia Vera induced hypercoagulability and transient spinal cord ischemia, where the patient had paraplegia for 30 minutes with subsequent full recovery. In our case, we speculated that, the hypercoagulable state secondary to antiphospholipid syndrome may have triggered an embolic episode leading to transient ischemia of the spinal cord. The symptoms of this event which would otherwise present as paraplegia may have been masked by SA. The transient spinal cord ischemia may have created circumstances similar to spinal cord lesion conducive for AD. Although AD is predominantly seen in patients with spinal cord lesion, considering the reported diverse etiological factors and multiple possible triggering mechanisms, the occurrence of AD in our case may not be completely rejected. The question of transport of afferent signals of bladder distension in an anaesthetized spinal cord, however, remains unanswered.

4. Conclusion

The observations in our case present an important phenomenon, and in the event of severe hypertension after spinal anaesthesia, presence of bladder distension should be considered and to avoid the adverse hemodynamic events, it is advisable to reconfirm if the urinary bladder is evacuated in the preoperative period.

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