ISSN: 2581 – 3633
PubMed - National Library of Medicine - ID: 101745081

International Journal of Medical Science and Advanced Clinical Research (IJMACR)

Available Online at:www.ijmacr.com

Volume – 6, Issue – 4, July - 2023, Page No. : 55 - 59

Acute Subdural Hematoma after Spinal anesthesia for Post-partum Sterilization: Case report and review of literature.

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How to citation this article: Dr Fozia Jeelani Wani, Dr Meka Krishna Kumari, "Acute Subdural Hematoma after Spinal anesthesia for Post-partum Sterilization: Case report and review of literature", IJMACR- July - 2023, Volume – 6, Issue - 4, P. No. 55 – 59.

Type of Publication: Case Report

Conflicts of Interest: Nil

Abstract

Acute subdural haemorrhage (SDH) is a serious life-threatening condition which needs urgent intervention. We report a case of acute SDH in a patient who had normal vaginal delivery and underwent postpartum sterilisation on postnatal day 3. Post-partum sterilisation was performed under spinal anesthesia (SA) using 25 G Quincke spinal needle in second attempt. She developed acute severe headache along with loss of consciousness and was diagnosed with acute SDH on CT scan. Urgent surgical decompressive craniectomy was performed by our neurosurgical team. She recovered completely without any neurological deficit. This report emphasises the need and importance of appropriate and immediate diagnosis along with prompt surgical intervention. This report also suggests that pregnancy, post-partum period and early ambulation of post-natal mothers are risk factors responsible for development of SDH even though there are no other obvious causes.

Keywords: Acute Subdural Hematoma, Spinal Anesthesia, Post Dural Puncture Headache

Introduction

SA is a safe procedure and used on day-to-day basis for majority of the obstetric procedures. However, the procedure is not without complications like severe headache, hypotension, haematoma or local infection. Among these, the most frequent complication after SA is severe headache known as post-dural puncture headache (PDPH).[1] PDPH usually occurs within 5 days after SA and is due to cerebrospinal fluid leakage through the dural puncture.[2] Diagnostic feature of PDPH is headache which aggravates on sitting or standing and is relieved on lying supine. A rare but significant complication of SA is subdural hematoma (SDH) with reported prevalence between 1/5,00,000 and 1/1,000,000.[3] SDH after SA may be misdiagnosed as PDPH and treatment can be delayed with significant maternal morbidity and mortality. SDH may present as

acute, sub- acute, or chronic. Acute SDH occurs within 7 days of SA for obstetrical procedures. The need of accurate and prompt diagnosis, presence of multidisciplinary team and appropriate intervention is important for managing such cases.

Case Report:

A 26-year-old female, G4P3L3 with 40.3 weeks of gestation and previous all normal vaginal deliveries was admitted for induction of labor. Her antenatal period was uneventful with regular antenatal visit in our hospital. Her last childbirth was 2 years back. Her medical and surgical history was not significant. There was no history of headaches or migraine in past or during pregnancy. She never used oral contraceptive pills or any other hormones. She belonged to lower-middle class family and was living in a nuclear family. She was an average built female with weight of 65 kgs with pre-pregnancy BMI of 24 kg/m^2 and gained 8 kgs of weight through her pregnancy. She was well hydrated with no sign of anemia or pre-eclampsia. General physical and systemic examination was with-in-normal limits. Abdomen was distended with full term pregnancy. Routine blood test and coagulation profile was normal. Induction of labour was done by mechanical method using intra-cervical foley's catheter. After 6 hours, labour pains started and labour progressed spontaneously. Progress of labour and vital signs were monitored regularly and were normal. She delivered a healthy male baby by normal vaginal route without an episiotomy after 4 hours. Her immediate postpartum period was uneventful. Patient was ambulatory and taking normal diet. On post-natal Day 3, postpartum sterilization was planned after taking informed and written consent. SA was given using 25 G Quincke Spinal needles between L3 and L4 in sitting position and was successful in second attempt. For SA 10 mg Hyperbaric Bupivacaine and 25 mcg Fentanyl was used. Modified Pomeroy's technique for tubectomy was performed via mini-laparotomy and procedure was completed within 20 minutes without any obvious complication. Patient was stable and shifted to post-natal ward after 4 hours. In post-natal ward patient was ambulatory, having normal diet, breast feeding and taking care of the baby. However, after 24 hours of surgery she developed severe headache along with the loss of consciousness. Immediate evaluation by multidisciplinary team of doctors involving ICU physician, neuro-physicians and obstetrician was done. On examination, patient was unresponsive to painful stimulus. Her recorded pulse rate was 94 beats per minute, SPO2 was 98% on room air and blood pressure was 140/90 mmHg. Neurologic examination revealed Glasgow coma scale of E1V1M4, with right sided pupil 4 mm reactive but sluggish and left sided pupil 2 mm non-reactive. Immediate resuscitative measures with intubation and mechanical ventilation were started. Computed tomography brain revealed a well-defined heterogenous hyper-density along the right fronto-parieto-temporal lobes extending into right tentorium cerebelli with maximum thickness measuring 5.9 mm in the right frontal region, 9.2 mm in the right parietal region and 7.2 mm in the right temporal region causing mass effect with compression of adjacent brain parenchyma, ipsilateral lateral ventricle and midline shift of 9.4 mm to the left suggestive of right sided acute SDH (Figure 1). She underwent emergency right decompressive craniectomy and evacuation of thick hematoma. Bone grafting was deferred at the time of primary surgery in order to monitor patient for rebleed (Figure 2). She was discharged after 5 days of primary

surgery and was followed up regularly. Right fronto-parieto-temporal autologous cranioplasty was done after 2 months and 20 days of initial decompressive surgery. She is being regularly followed in our outpatient services and is without any neurological deficit at eight months follow up.

Discussion:

Acute SDH is a rare but life-threatening complication after SA in obstetric patients. During SA leakage of CSF through the dura mater puncture site may occur and lead to a reduction of intracranial pressure. This causes a sudden caudal displacement of the brain that stretches the pain-sensitive structures and bridging veins. Stretch of the pain sensitive structures results in PDPH, while rupture of the bridging veins results in SDH. Presence of thin walls, circumferential arrangement of collagen fibers and lack of outer reinforcement by arachnoid trabecules make bridging veins susceptible to rupture on traction.[4] It can occur as early as 4 hours and as late as 29 weeks after SA. This variation of onset may be due to the age of patient, size & rate of the clot formation, topography, pressure effect on the adjacent structures and co-existing morbidities.[5] Risk factors responsible for SDH after SA are repeated attempts at puncture, coagulation disorder, use of anticoagulants, recent history of brain trauma and presence of aneurysmal vascular malformations.[6] During pregnancy congestion of bridging veins make them more susceptible to rupture and therefore increase the risk of SDH.[5] Dehydration and early ambulation can also contribute to the occurrence of SDH.[7] Cuypers et al reviewed 20 cases with SDH following SA for obstetric procedures and found no risk factor in majority of patients with anticoagulant use in one case and multiple spinal punctures in 4 cases.[8]

Calibre, design and direction of bevel of needle has also been related to development of SDH.[9] However, Zeidan et al found that size of the needle does not paly significant role because in their study SDH also developed in patient with fine needle use.[10]

Acute SDH presents with presence of the persistent non-postural headache and varied other neurological symptoms like vertigo, diplopia, motor deficit, altered consciousness, convulsions, cranial nerve palsy etc.[11] It may be asymptomatic or present with subtle symptom like neck stiffness as reported by Domoto et al.[12]

Differential diagnosis of SDH include postpartum pre-eclampsia, migraine, cerebral vascular accidents, venous sinus thrombosis and posterior reversible cerebral vasoconstrictive syndrome.[13] Non-contrast Computed tomography of the brain is the imaging of choice due to speed, widespread availability and specificity to detect SDH. CT scan reveals hyper-density along the involved areas of brain. [14] MRI is useful in detecting chronic small SDH as well as to rule out other conditions like reversible cerebral vasoconstrictive syndrome.[15]

Management of SDH can be surgical or medical depending on the severity of symptoms and size of SDH. Small hematomas resolve gradually with bed rest, analgesic and hydration and are followed up by repeated scans. Surgery is needed for larger hematomas with midline shift and with deteriorating neurological status. In majority of cases surgical management involves craniotomy or burr holes and drainage of hematoma. However, in our case CT scan revealed acute SDH involving entire right fronto-parieto-temporal region with midline shift of 9.4mm. Decompressive craniectomy involving right fronto-parieto-temporal bone was performed along with evacuation of

hematoma. Almost three months later autologous transplantation was performed.

Amorim et al reported 2 cases and reviewed 33 cases of intracranial SDH following SA. In their review, 27 patients (77%) required surgical intervention, 4 (11%) developed neurologic deficits, and 4 (11%) died.[5] Gago et al reported two cases of SDH following SA for caesarean section. [16] First patient was managed conservatively and second one needed surgical decompression, thereby depicting varied presentations and management approaches to SDH.

Studies show that complete recovery occurs in 70% of case, 25% can have varied degrees of neurological deficit and in 5% cases it may be fatal.[8] Gioia et al reported a case of fatal acute SDH where patient succumbed to the condition.[17]

Our case had no other risk factors apart from being pregnant and two attempts were done for SA. Early ambulation due to lack of family support might have precipitated the rupture of veins and subsequent development of acute SDH. High clinical suspicion with immediate CT scan of brain aided in prompt diagnosis and subsequent surgical decompression craniectomy. So apart from technical aspect for the development of SDH, social support and care is also important for overall well-being of post-partum mothers. However, to conclude early ambulation as a causative factor in the development of SDH needs further studies.

Conclusion

Clinicians involved in the care of obstetric patient should have high suspicion for diagnosis of SDH in cases with persistent non-postural headache or rapid neurological deterioration after SA. Neuroimaging should be done as early as possible to diagnose this condition. Immediate and appropriate intervention can significantly reduce the morbidity and mortality associated with SDH.

Acknowledgement: We would like to thank Neurosurgical team headed by Dr. Kaushal Ippili Apollo Hospitals Hyderabad, India.

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Legend Tables

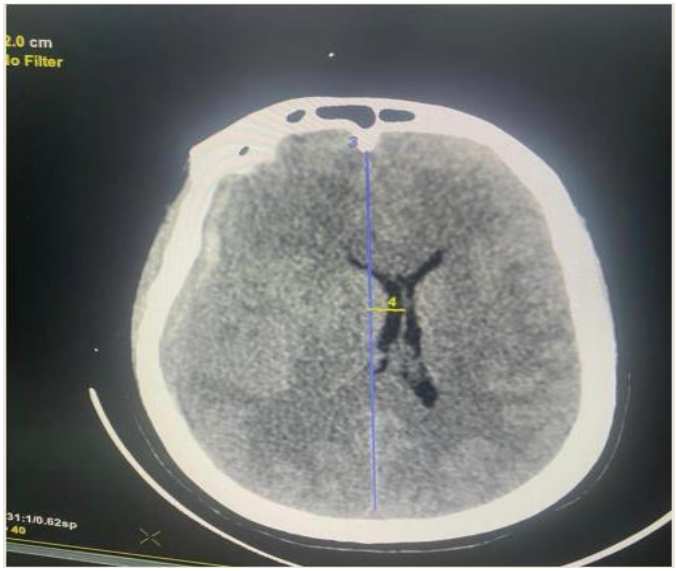

Figure 1

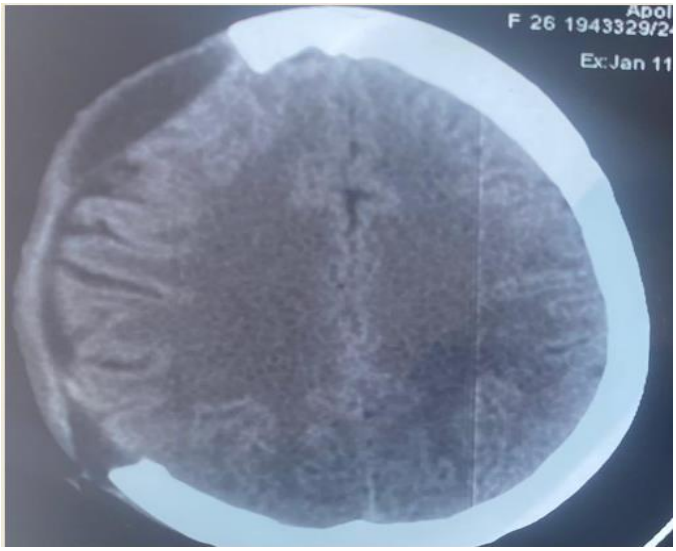

Figure 2