Case Report

Presentation of occult Chiari I malformation following spinal anesthesia

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ABSTRACT

Chiari I malformation (CM-I) manifests with tonsillar herniation below foramen magnum. These patients are at high risk of respiratory depression and bulbar dysfunction in the perioperative period with underlying obstructive sleep apnea. However, the safe use of both general and regional anaesthesia has been documented in a known CM-I parturients. We describe the successful management of a patient who had hypercapnic respiratory failure in the post-anaesthetic care unit following an uneventful subarachnoid block for left knee replacement surgery. This patient was retrospectively diagnosed with occult CM-I and moderate to severe obstructive sleep apnea in the postoperative period.

Key words: Occult Chiari I malformation, respiratory depression, spinal anaesthesia

INTRODUCTION

Chiari malformations are developmental anomalies with cerebellar tonsillar herniation below the foramen magnum.[1] The incidence of Chiari I malformation (CM-I) by neuroimaging techniques range from 0.1 to 1% with the average age of presentation between 25 and 40 years.[2] These patients manifest with headache, neck pain, progressive scoliosis and cerebellar dysfunction due to cervico-medullary compression.[3] We describe the presentation of a patient with occult CM-I in an undiagnosed obstructive sleep apnea (OSA) patient who had hypercapnic respiratory failure in the post-anaesthetic care unit necessitating endotracheal intubation and unanticipated intensive care unit (ICU) admission after an uneventful elective knee surgery under subarachnoid block.

CASE REPORT

A 60 year old ASA III female patient (BMI: 40 kg/sq. m) was scheduled for left total knee replacement. Her past history was significant for morbid obesity, hypertension and on regular medications. She had past history of snoring but denied sleep studies. She had uneventful multiple General anaesthesia (GA) previously. She was evaluated for syncope while coughing 3 months back but her Holter monitoring and bilateral carotid Doppler studies were negative. Pan endoscopy done to evaluate her dysphagia revealed no abnormality. Her airway and physical examination were unremarkable. Her blood investigations were normal. Chest X-ray revealed cardiomegaly and electrocardiogram showed left ventricular enlargement. Moderate concentric left ventricular hypertrophy with inferior wall motion abnormality with ejection fraction of 56% was documented by transthoracic echocardiogram. Anaesthetic concerns were discussed and patient was consented for regional anaesthesia, subarachnoid block (SAB) along with left femoral nerve block.

In the operating room, baseline monitors (pulsoximetry, non-invasive blood pressure and electrocardiogram) were established. To avoid anxiety, 2 mg of i.v midazolam

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was administered. Under aseptic precautions, SAB was performed with 12.5 mg of bupivacaine and 100 mcg of morphine using 27G atraumatic spinal needle. She had an adequate surgical block with the sensory blockade to the level of T10 dermatome level. No further sedatives were given since she was drowsy with the initial dose of midazolam. However, she was responding to verbal commands with stable vital signs. The rest of the intraoperative period was unremarkable and transferred to post-anaesthetic care unit (PACU) subsequently.

In the PACU, she continued to be very drowsy, with worsening upper airway obstruction. There was an occasional drop in oxygen saturation below 90% despite oxygen supplementation. Arterial blood gas analysis done 2 hours later showed hypercapnic respiratory failure with pH of 7.28, $PaCO_2$ 65 mmHg and PaO_2 84 mmHg. Patient's trachea was intubated and lungs were ventilated in view of gradual deterioration of consciousness, airway protection and to facilitate ventilatory support. Oxygenation and ventilation were maintained easily after intubation (pH 7.34, $PaCO_2$ 38 mmHg, PaO_2 148 mmHg, inspired O_2 40% and PEEP 5 cm H_2O). She was transferred to ICU and gradually weaned over next 36 hours.

Following extubation, she developed difficulty in swallowing which persisted beyond 48 hours. She also complained of visual disturbance in left eye and syncope while coughing. However, she could ambulate on the floor. Ophthalmologist, neurologist and otolaryngologist consultations were done and magnetic resonance imaging (MRI) of the brain was done.

MRI brain showed CM-I with 17 mm cerebellar tonsillar herniation below the foramen magnum compressing lower medulla without hydrocephalus [Figure 1].

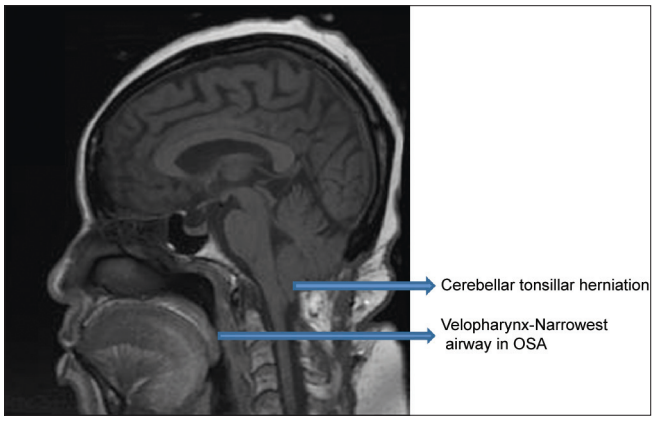

Figure 1: MRI scan showing cerebellar tonsillar herniation of 17 mm below foramen magnum

Neurosurgeons were consulted. They recommended conservative management followed by surgery at a later date. The symptoms improved with conservative management over a period of 2 weeks. The sleep study performed 2 months later was consistent with moderate-severe OSA and continuous positive airway pressure (CPAP) therapy was instituted. She had an uneventful posterior fossa decompression for CM-I after 3 months. She was symptomatically better and advised regular follow-up.

DISCUSSION

CM-I is a congenital anomaly seen in adult population and associated with craniocervical abnormalities. These patients with CM-I are more likely to manifest their symptoms when the cerebellar tonsillar herniation is greater than 5 mm and are invariably symptomatic when it is more than 12 mm.[2]

Dysphagia, blurred vision and syncope was described in 15.7%, 17% and 3.3% symptomatic CM-I patients, respectively.[1] However, our patient had preoperative syncope, mild dysphagia without major neurological or cardiorespiratory symptoms despite occult cerebellar tonsillar herniation of 17 mm below foramen magnum. Our patient developed delayed postoperative symptoms probably due to slow leak of CSF through puncture site with the compression of vagus nerve and subsequent changes in cranio-caudal CSF flow dynamics. In a retrospective review involving 109 OSA patients receiving 100-300 mcg of intrathecal morphine for total joint arthroplasty experienced more transient desaturations, defined as SaO2 <92% (11.2% vs 2.9%, P=0.0063) compared to non-OSA controls.[4]

Patients with CM-I have decreased ventilatory response to carbon dioxide and insensitivity to hypoxia. It is precipitated by the administration of benzodiazepines which further decrease the arousal response to hypoxia and hypercarbia and increase the duration of apnoea in OSA patients. The respiratory failure in our patient is probable to following reasons. Firstly, the local edema compressing medulla on the clivus or to the vasospasm causing brainstem ischemia due to slow seepage of CSF through puncture site. Secondly, the administration of i.v midazolam and intrathecal morphine with underlying OSA would have further precipitated her respiratory depression.

There are no consensus guidelines on the anesthetic management of patients with CM-I. Acute deterioration

of occult CM-I has been described in parturients.[5] On the contrary, the safe use of general anaesthesia (GA) and spinal anesthesia has been documented with known CM-I in parturients.[6,7] Uneventful spinal anaesthesia with narcotics added to local anaesthetic has been described in a known CM-I parturient for cesarean delivery.[8] However; the degree of tonsillar herniation was not described in the literature. There is limited data on the use of neuraxial techniques in patients with surgically corrected Chiari malformation. Though successful spinal anaesthesia for caesarean delivery in a woman with a surgically corrected CM-I has been described but the authors did advise a word of caution.[7]

In a recent systematic review on the effect of sedatives and anaesthetics in OSA patients, the authors emphasized temporary oxygen desaturation intraoperatively with the use of midazolam and fentanyl and those patients were retrospectively diagnosed with OSA.[9] Use of screening questionnaire for the diagnosis of OSA as well as a thorough neurological evaluation including MRI scan would have diagnosed OSA and CM-I, respectively.[10] If diagnosed early, anaesthetic techniques in the management of this patient could have included graded epidural anaesthesia or general anaesthesia with awake fibreoptic intubation and controlled ventilation. However, these patients require an extended duration of postoperative monitoring in these patients.[11]

CONCLUSION

Patients with occult Chiari malformation are at considerable risk of respiratory depression and bulbar dysfunction in the perioperative period with underlying OSA. A thorough literature search did not reveal any such other reported case of occult Chiari I malformation which was diagnosed postoperatively after uneventful knee surgery performed under spinal anaesthesia.

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