Acute exacerbation of Chiari malformation: A rare cause for non-awakening from anaesthesia

Sir,

Arnold Chiari malformation (ACM) is described as underdevelopment of posterior fossa with varying degrees of cerebellar herniation.[1] It is commonly associated with hydrocephalus and meningomyelocele. We report non-awakening from anaesthesia in a patient with ACM with sacral meningomyelocele who underwent meningomyelocele correction.

A 2-year-old 12 kg male child, diagnosed with a case of sacral meningomyelocele with spina bifida was posted for surgical correction. Preoperatively, the child had weakness in lower limbs with no bladder and bowel control. Magnetic resonance imaging (MRI) showed spina bifida at S1 to S3 with meningomyelocele and low lying tethered cord. ACM with mild tonsillar herniation of 5 mm was present.

Anaesthesia was induced with fentanyl 25 mcg and propofol 30 mg. Atracurium 6 mg was used for orotracheal intubation. Intubation was uneventful, and the child was placed prone with head in neutral position. Anaesthesia was maintained with oxygen, air and sevoflurane with minimum alveolar concentration 1–1.1. The tethered cord was separated, large dural defect was repaired using dorsolumbar fascia, and a theco-peritoneal shunt was placed.

The total duration of surgery was 210 min. Blood loss was 30 ml, and the patient received 450 ml of crystalloids. There were no episodes of bradycardia or hypotension during surgery. At the end of surgery, neuromuscular blockade was reversed with neostigmine 600 mcg and glycopyrrolate 120 mcg. There were no signs of spontaneous respiratory effort. Only three doses of fentanyl (6 mcg each) were used for analgesia, and the nasopharyngeal temperature was 36.2°C.

Blood sugar, arterial blood gas (PH-7.38, PCO_2-38, PO_2-460, Na-135, K-4.2, Cl-108, HCO_3-22, BE--2 Lactate-0.9, Glucose-110) and serum electrolytes were done to rule out other causes for delayed awakening, all were within normal range.

After excluding anaesthesia related causes for delayed recovery and being a case of ACM, we suspected an intracranial complication such as cerebellar herniation, pneumocephalus or subdural haemorrhage. Furthermore, pupils were unusually dilated and reacted sluggishly to light. Urgent computed tomography (CT) brain was done which showed crowding at the foramen magnum. CT brain excluded pneumocephalus and intracranial bleed but was inconclusive for cerebellar herniation. An emergency MRI confirmed exaggeration of Chiari malformation with tonsillar herniation up to 28 mm [Figure 1]. The child underwent emergency foramen magnum decompression with C1–C2 laminectomy, but there was no improvement in neurological status.

Acute exacerbation of Chiari malformation may present as foramen magnum syndrome. There are previous reports of acute tonsillar herniation after lumbar puncture and lumboperitoneal shunt placement.[2] Acute cerebellar herniation presenting as non-awakening from anaesthesia has not been reported. In our case, preexisting ACM and change in craniospinal cerebrospinal fluid (CSF) pressure gradient after opening the dura could have caused the initial displacement. The arrested CSF flow at foramen magnum with a shunt at spinal level could have further worsened the herniation.[3]

Anaesthesiologist may minimise intra-operative CSF leak by maintaining head low position at the time of dural opening and repair. Further we should keep a close watch on the amount of CSF leak apart from haemodynamic monitoring. From this case report, we hypothesise tonsillar herniation probably had set

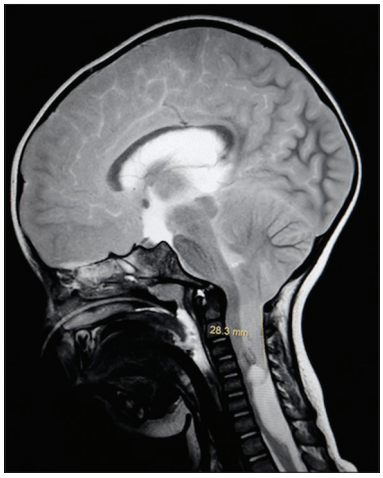

Figure 1: Magnetic resonance imaging showing tonsillar herniation with brain stem and spinal cord compression

in due to CSF leak during repair of the large dural defect, which further got aggravated by placement of theco-peritoneal shunt. This unforeseen neurological complication may present as non-awakening from general anaesthesia.

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Conflicts of interest

There are no conflicts of interest.

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