# Delayed awakening after sevoflurane anesthesia for MRI brain in a child with undiagnosed mitochondrial disorder

Sir,

Anesthesia in the magnetic resonance imaging (MRI) suite can pose multiple challenges because of the uniqueness of the MRI environment. For most pediatric MRIs, we use sevoflurane-based anesthesia, which has been shown to be safe and efficacious.[1,2] We describe a 2-year old male child with an undiagnosed mitochondrial disorder who had delayed awakening after sevoflurane anesthesia.

The child presented with complaints of gait abnormality since 1 year and one episode of seizures 6 months back. On examination, the child was neurodevelopmentally normal, with normal blood investigations, blood gas, and echocardiography. An MRI brain was requested for the gait abnormality and seizures.

On the day of MRI, with the child adequately fasted, and pre-medicated with oral midazolam 0.5mg/kg. Anesthesia induction commenced with incremental sevoflurane upto 8% in an oxygen and nitrous mixture. After achieving an adequate depth, a 24G cannula was inserted in the right hand. At this point, the child became apneic, necessitating bag mask ventilation. Sevoflurane was decreased to 1%, with oxygen/nitrous kept at 1 liter/min and the mask secured via harness. As the child remained apneic, bag mask ventilation continued. Vital parameters were normal with a pulse rate of 120/min, $SpO_2$ of 99%, and end tidal carbondioxide of 34mmHg. A 10mL/kg bolus of Ringer's Lactate was given. On completion of the scan, lasting for 50 minutes, all agents were turned off and 100% oxygen was administered. Despite washing out all agents, the child showed no respiratory efforts and was unresponsive to a deep painful stimulus. Other causes were ruled out, such as drug error, hypothermia, hypoglycemia, hypovolemia, and acute brain changes (as reported by the radiologist). A blood gas sent revealed a pH of 7.19, a $pCO_2$ of 35mmHg, $HCO_3$ of 13mmol/L, and lactate of 4.6mmol/L. After another fluid bolus and continued mask ventilation for 30 minutes, the child began to have spontaneous respiratory efforts with respiration gradually becoming regular as the child became more responsive.

The child was shifted to the recovery area fully awake, with normal vitals and a repeat ABG showed a pH of 7.33, $pCO_2$ 32mmHg, $HCO_3$ 16.3 mmol/L, and lactate 3.8mmol/L.

On follow-up, the child was suspected to have Leigh's disease based on MRI changes, including bilateral symmetrical hyper-intensities in the mid-brain. MR spectroscopy showed a lactate peak, diagnostic of mitochondrial disorder. On subsequent evaluation, a SURF 1 gene mutation pathognomic of Leigh's disease was detected.[3]

Leigh disease is an extremely rare mitochondrial disorder presenting mainly with neurological signs. Anesthetic risks include hypoventilation, irregular respiration, apnea, lactic acidosis, and delayed awakening. There is no clear consensus as to what is the safest anesthetic approach, and both inhalational agents and propofol have been used.[4,5] Our patient demonstrated an increased sensitivity to sevoflurane, receiving a low dose of sevoflurane for the most part and a high dose momentarily. This case shows the need for an anesthesiologist to be aware of the possibility of an undiagnosed mitochondrial disorder in a child with subtle neurological signs and the anesthesia management should include the avoidance of prolonged fasting, hypothermia, and succinylcholine and the careful use of inhalational agents/propofol.

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## Conflicts of interest
There are no conflicts of interest.

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