Postoperative seizures: A manifestation of preoperative fasting

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A 29-year-old, American Society of Anesthesiologist (ASA) grade I male patient weighing 48 kg presented with post-traumatic low backache. There was no history of any respiratory, cardiovascular or neuro-psychiatric abnormality or any known drug allergy. Routine blood investigations, electrocardiogram (ECG) and chest X-ray were within normal limits. The patient was diagnosed to have L3-L4 and L4-L5 disc prolapse and was posted for L 3-4, L 4-5 discetomy and posterior fixation under general anaesthesia (GA). During the pre-anaesthetic check-up visit, the patient was advised preoperative fasting for 8 hours, as per the ASA task force recomendations[1] and was pre-medicated with intramuscular glycopyrrolate (0.2 mg) 30 minutes prior to surgery. The patient was scheduled to be the second case in the routine operation list.

In the operating room (OR), routing monitoring using pulse oximeter, ECG and non-invasive blood pressure was started. GA was induced with propofol 2 mg/kg and fentanyl 2 μg/kg followed by tracheal intubation with an 8.5-mm PVC endotracheal tube after administering rocuronium 1 mg/kg. The position of the tracheal tube was confirmed by auscultation and with a sustained capnogram. GA was maintained with sevoflurane (0.8-1.0 MAC) in a mixture of nitrous oxide and oxygen (2:1 ratio) along with intermittent boluses of vecuronium and fentanyl. Intraoperative fluid used was ringer lactate. The surgery lasted for 3 hours and the intraoperative course was uneventful. At the end of the procedure, neuromuscular blockade was reversed with neostigmine and glycopyrrolate and the trachea was extubated after the patient was awake and breathing adequately. The patient was then shifted to recovery room for observation. In the recovery room the patient was awake with stable haemodynamic parameters but was drowsy. After 15 minutes of recovery room admission the patient had generalised tonic clonic seizure (GTCS) with loss of consciousness. Immediately the patient's airway was secured and midazolam 2 mg was administered intravenously to control GTCS. The arterial blood gas analysis (ABG) of the patient did not reveal any blood gas or electrolyte abnormality. However, the patient's blood sugar was found to be 54 mg/dl. Immediately 50% dextrose (2 ml/kg) was administered to the patient

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followed by administration of 500 ml of 5% dextrose normal saline. The patient regained consciousness and remained seizure-free; thereafter, during his 5-hour stay in the recovery room. When enquired from the patient it was found that the patient had his last meal at 8 PM, the previous night and was continuously fasting till the time of surgery (at around 2 pm next day) for about 18 hours. The patient was observed overnight in the high dependency unit (HDU) and did not have any seizure throughout his hospital stay.

Various causes of postoperative seizure has been described in the literature such as electrolyte abnormality (hyponatraemia, hypocalcaemia), hypoxia, hypocarbia, hypoglycaemia, acidosis, pyrexia, drug related causes (local anaesthetics, inhalation anaesthetics, opioids, drug reaction) psychogenic seizures (pseudoseizures) and global or focal cerebral ischemia.[2] The cause of seizure in our patient was hypoglycaemia due to prolonged fasting. From the preoperative history, the nature of surgery and ABG, we ruled out rest of the above-mentioned causes of post-operative seizure. As we have not administered any dextrose containing solution to the patient intra-operatively, the patient was prone to become hypoglycaemic following such prolonged fasting.

Surgical stress increases blood glucose level[3] which potentially reduces the chances of developing hypoglycaemia during the perioperative period. This is due to the synergistic effects of stress hormones like cortisol, glucagon and epinephrine which promote gluconeogenesis.[4] However, opioids are known to obtund (if not completely abolish) the body's response to stress and cytokine production.[5] In our case, the adequate depth of anaesthesia and analgesia was maintained throughout the perioperative period which might have counteracted the stress response and occurrence of gluconeogenesis to certain extent. Moreover, the precise blood sugar level at which symptomatic hypoglycaemia occurs is variable among individuals.[6] In an awake patient the central nervous system manifestation of hypoglycaemia ranges from light headedness to coma and seizure.[6] However, under GA, autonomic responses are difficult to interpret as the responses are blunted by anaesthetics and patients are unable to report symptoms during general anaesthesia.

In our case, the patient remained fasting for 18 hours in spite of recommended (6-8 hours in adults) nil per oral advise.[1] Such situation may arise due to number of reasons like improper advise or lack of communication between the anaesthesiologist and the patient, language barrier, delay in the surgery due to any reason, etc. This case report remind us of the importance of proper communication and passing of preoperative orders to the patients and nursing staff involved in patient care and also confirming that the same has been properly followed before taking up a patient for any procedure under anaesthesia.[1] We would like to emphasise the importance of confirming the period of fasting (time since last meal) for every patient and glucose supplementation if required to those who remain nil per oral for long period.

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