Reversible cause of intra operative hypoxia in an aspirated patient

INTRODUCTION

We present a case of 19-year-old male with a history of road traffic accident with head injury and aspiration posted for decompressive craniotomy under general anaesthesia. Patient developed significant desaturation intraoperatively under general anaesthesia with isoflurane that reversed upon switching over to total intravenous anaesthesia (TIVA) with propofol. Apart from aspiration causing hypoxia, decrease in hypoxic pulmonary vasoconstriction (HPV) by inhalational agent could have also contributed to the desaturation.

CASE REPORT

A 19-year-old male with a history of road traffic accident underwent extradural and subdural haemorrhage evacuation and was on post-operative ventilation for 6 days. He became restless, irritable, tachycardic, tachypnoeic and hypoxic 6 h post-extubation. Auscultation revealed bilateral basal crepitations. Chest X-ray [Figure 1] showed bilateral basal infiltrates

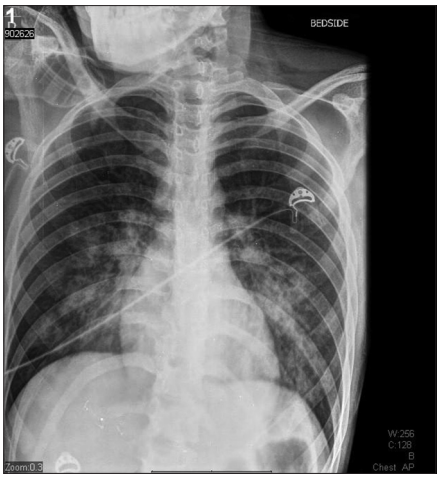

Figure 1: X-Ray Chest

consistent with aspiration. Arterial blood gas (ABG) analysis showed partial pressure of oxygen (PaO_2) of 82.8 mm Hg on FiO_2 of 0.4. Patient was reintubated and ventilated. Pupils were unequal and sluggishly reacting to light. Repeat computed tomography of the brain showed persisting cerebral oedema with a midline shift. A decompressive craniotomy was decided to reduce the increased intracranial pressure.

The patient was shifted to the operating room from the Intensive Care Unit (ICU) with a heart rate of 112 beats/min, blood pressure of 130/80 mm of Hg and oxygen saturation (SpO_2) of 100% on oxygen with Bain's circuit. His SpO_2 in the ICU post-intubation was between 98% to 100% with a FiO_2 of 0.4–0.5. As the patient was already intubated and brought to the operating room, general anaesthesia was administered with fentanyl 2 μg/kg, atracurium 0.5 mg/kg and maintained with isoflurane, oxygen and air with volume control ventilation, positive end-expiratory pressure (PEEP) of 5 cm of H_2O through a GE anaesthesia workstation (Aestiva™). Endotracheal tube position was confirmed after the patient was positioned for surgery by auscultation and by feeling the cuff in the suprasternal notch. Ten minutes post-induction of anaesthesia, with an end-tidal isoflurane concentration of 0.9, he started to desaturate gradually to a value of 88% from the baseline value of 98%. The surgeon had commenced the craniotomy by then. There was no kinking or change in position of the endotracheal tube. Air entry was equal bilaterally. An endotracheal suction did not yield any secretions. FiO_2 was gradually increased from 0.4 to 1, tidal volume from 8 ml to 10 ml/kg body weight, PEEP was increased from 5 to 10 cm of H_2O (all these changes were undertaken over a period of 20 min), which failed to improve the SpO_2, which remained around 88%-90%. Haemodynamic parameters were stable throughout the procedure with a blood pressure of 120–140/80–90 mm of Hg and a heart rate of 100–110 beats/min. Assuming loss of HPV due to inhalational anaesthetic (isoflurane) to be the cause of desaturation, the anaesthetic was switched from inhalational based maintenance to intravenous based maintenance by the use of propofol 100–150 μg/kg/min infusion. Saturation gradually improved to 96% in 5 min and at the end of procedure SpO_2 was 100% on a FiO_2 0.4 and a PEEP of 5 cm of H_2O. Peak airway pressures were within normal limits throughout the procedure.

DISCUSSION

Pulmonary aspiration could result in intrapulmonary shunt of as much as 75%.[1] HPV is a physiological response to alveolar hypoxia, which redistributes blood from hypoxic alveoli to better ventilated lung segments by vasoconstriction of the pulmonary artery. Pulmonary artery smooth muscle cells (PASMC) are located full length across the pulmonary artery tree. They respond to hypoxia with the influence of surrounding lung parenchyma or systemic transmitters. PASMC act as oxygen sensors to varying degree of hypoxia. Intracellular calcium increases in PASMC in response to hypoxia. Hypoxia mediated increase in calcium is dependent on voltage-gated calcium channels to a lesser degree and transient receptor potential channels to a greater degree. Increase in calcium in PASMC causes vasoconstriction.[2] HPV optimises ventilation-perfusion matching, reduces shunt fraction and increases systemic O_2 delivery in conditions such as atelectasis and pneumonia.[3]

Volatile halogenated inhalational agents, particularly isoflurane is known to inhibit protective HPV, thereby increasing venous admixture and decreasing PaO_2. Relative blood flow to non-dependant (hypoxic) lung is increased significantly by administration of 1.5% isoflurane during both one and two lung ventilation, suggesting the vasodilator effect of isoflurane on pulmonary vasculature, which counteracts the protective HPV.[4] Mechanism by which isoflurane attenuates HPV is unclear. Potassium channels might play a role in this inhibitory effect.[5] Intravenous anaesthetic agents like propofol do not inhibit HPV in humans and hence do not[6] increase shunt fraction or cause any change in arterial oxygen tension. The mechanism of preservation of HPV response by propofol is by attenuation in adenosine triphosphate

sensitive potassium channel mediated vasodilatation. Ketamine preserves while propofol potentiates HPV.[7] This likely explains the improvement in saturation after TIVA with propofol in our patient.

Of the various interventions like confirming the tube position, increase in FiO_2, increasing the tidal volume, increasing PEEP, the only change which improved the saturation in this report was switching over to TIVA. So the possible explanation of improvement in saturation is the restoration of HPV by propofol. ABG was not done at this point but an increase in saturation from 88% to 100% on pulse oximeter indicated an increase in arterial oxygenation based on oxygen dissociation curve. We could have reintroduced isoflurane to confirm our hypothesis. However, the surgery was almost completed and we would have not had enough time as it took almost 20 min in the previous attempt to improve the saturation.

CONCLUSION

There are several causes of desaturation intraoperatively. Loss of HPV is one such cause. HPV not only occurs in one lung ventilation, it can also occur in clinical conditions that could result in increase in dead space ventilation or intrapulmonary shunt resulting in V/Q mismatch such as aspiration. Understanding the causes of HPV and the factors influencing it helped us in effective management of our patient.

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