

PULSE OXIMETRY AND PULMONARY OEDEMA

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A case is reported where reduced oxygen saturation was detected by a pulse oximeter before clinical signs of pulmonary oedema appeared. A 45 years old, 82 kilogram lady presented for emergency surgery with subacute intestinal obstruction. She had no previous history of any cardiovascular, respiratory or metabolic disorder. She underwent partial colectomy and colostomy. Two days later she was again brought to the operating room for revision of colostomy. On pre-operative examination she was conscious and orientated, afebrile, heart rate of 135 beats/minute and a respiratory rate of 45/minute. She was in a positive balance of + 1350 mls over the last 24 hours. On chest auscultation, she had only left basal crackles. The preoperative arterial blood gases showed a P_{O2} of 90.6 mm of Hg and oxygen saturation of 97.3% on 8 L of oxygen per minute via MC mask. The patient was preoxygenated through the Magill's circuit and a pulse oximeter probe (Ohmeda Biox 3700) was attached to her forefinger which showed an oxygen saturation of 98%. Anaesthesia was induced with a sleep dose of thiopentone, and 100mg of suxamethonium. She was intubated with cricoid pressure and anaesthesia maintained with fifty per cent nitrous oxide and oxygen, pancuronium and pethidine. Intraoperatively she remained stable and her oxygen saturation remained at 96%. The patient was reversed with atropine and neostigmine. After reversal, but before extubation she was noticed to have difficulty in breathing and the oxygen saturation dropped to 87% on 8L/minute of oxygen. The ventilation was thereafter assisted and the saturation showed very slight improvement to 89%. Chest auscultation at this stage did not reveal any new findings. Nerve stimulator showed a fully reversed block. Arterial blood gases showed a pH of 7.38, pCO₂ of 46.4 mm of Hg, P_{O2} of 55 mm of Hg, bicarbonate 24, base excess 2.2 and oxygen saturation 87.8%. She was transferred to the Intensive Care Unit with an endotracheal tube in situ and connected to a T piece circuit on 12 L/minute humidified oxygen. Her oxygen saturation was monitored continuously and was 90%. A chest x-ray was done. On endotracheal tube suctioning slightly pinkish tinged secretions were observed. Chest auscultation at that stage did not reveal any new findings. She was given furosemide 40 mgs I.V stat' and her oxygen saturation improved dramatically to 96-97% after diuresis. Her chest x-ray when received supported the findings of early pulmonary oedema. She was extubated one hour later.

DISCUSSION

The major causes of pulmonary oedema in the perioperative period are cardiac failure, fluid overload, airway obstruction, acid aspiration, gas embolism, allergy, higher oxides of nitrogen, reactions to blood and blood products¹. Usually pulmonary oedema does not present itself in the recovery because of peripheral vasodilatation. Only when peripheral vasoconstriction occurs due to pain or cold, fluid shifts from peripheral to central compartment and clinical pulmonary oedema occurs². Pulse oximeter is a non-invasive instrument that detects hypoxia and is accurate within 2%. Its use is now becoming a routine in the intraoperative period³. In our case the diagnosis of pulmonary oedema would have been delayed if the oxygen saturation monitoring was not used. Pulse oximetry as an early warning device could be an additional use of this useful non-invasive monitor.

REFERENCES

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