

Case Report

Oxygen embolism: an unusual consequence of hydrogen peroxide irrigation

Pravin Tukaram Survashé^{1*}, Sachin Guthe¹, Vernon Velho², Laxmikant Bhopale¹

¹Department of Neurosurgery, M. Ch. Neurosurgery Institute, Grant Government Medical College and Sir J.J. Group of Hospitals, Mumbai, Maharashtra, India

²Department of Neurosurgery, M. Ch. Neurosurgery, DNB Neurosurgery Institute, Grant Government Medical College and Sir J.J. Group of Hospitals, Mumbai, Maharashtra, India

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*Correspondence:

Dr. Pravin Tukaram Survashé,

E-mail: pravinsurvashé97@gmail.com

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ABSTRACT

Three-percent solution of hydrogen peroxide is commonly used as a wound antiseptic agent to irrigate the operative field in Neurosurgical cases. We report a case of 45 year male who had undergone lumbar laminectomy under general anaesthesia in prone position and irrigation of surgical wound with hydrogen peroxide led to massive gas embolism during the surgery.

Keywords: Hydrogen peroxide, Embolism

INTRODUCTION

Prone position is regularly used in neurosurgical practice while operating posterior fossa lesions or spinal pathologies. Three-percent solution of Hydrogen peroxide is commonly used as a wound antiseptic agent in all surgical fields especially in Neurosurgery. It demonstrates broad-spectrum efficacy against viruses, bacteria, yeasts, and bacterial spores.¹ It is a common practice to irrigate the surgical wound with Hydrogen peroxide before closure. The release of effervescent oxygen bubbles help in evacuating debris in surgical site as well as it occludes micro vessels and act as a hemostatic agent. A rare, but known complication of hydrogen peroxide irrigation is systemic gas embolism. It is caused due to sudden release of large amount of Oxygen which liberates in to the venous circulation from vascular bed. Negative intrabdominal pressure generated in prone position aids in rapid absorption of oxygen bubbles in to venous circulation increasing the risk of embolism substantially.

We report a case of 45 year male who had undergone lumbar laminectomy under general anaesthesia in prone position and irrigation of surgical wound with hydrogen peroxide led to massive gas embolism intraoperative. After irrigation, the patient suddenly developed tachycardia, hypotension, and rapid oxygen desaturation. Immediate resuscitation was instituted and patient did survive. Hydrogen peroxide induced gas embolism in prone position is never been reported in Neurosurgical literature.

CASE REPORT

45 year old right handed male patient presented in our OPD with low backache and claudication pain in both lower limbs while walking. MRI spine was suggestive of severe lumbar canal stenosis at L3-4-5 level due to ligamentum flavum hypertrophy. He was investigated further and planned for lumbar laminectomy. He had no other comorbid condition with ASA Gr-1 status. All routine preoperative investigations were within normal limits and 2D Echocardiography was suggestive of 60 % ejection fraction.

In the operation theatre he was premedicated with Midazolam and Fentanyl and induced with Propofol and Scolin. One central venous line and 2 peripheral lines were inserted. After tracheal intubation prone position was given. General anaesthesia was maintained with Sevoflurane, a mixture of N₂O with O₂ and Vecuronium. Arterial oxygen saturation (SPO₂), End-tidal carbon dioxide concentration ETCO₂, noninvasive blood pressure (NIBP) and Electrocardiograph (mode V5) monitoring was started. Patient's vitals were maintained throughout the surgery. His blood pressure (BP) stayed at 110–120/70–80 mmHg, heart rate (HR) remained 70-80 beats per minute (BPM), saturation of pulse oxygen (SPO₂) was 97%-99% and ETCO₂ was 29-31mmHg. L3/4/5 laminectomy was done. Care was taken to occlude the bony bleeding sites with bone wax. Before heading to closure of the surgical wound it was irrigated with around 10 ml of 3% Hydrogen peroxide followed by 0.9% Normal saline as a routine practice. Within 2 minutes of irrigation the SPO₂ decreased rapidly to 84% and the ETCO₂ to 16 mm Hg. BP came down to 90/60 mmHg with heart rate showing tachycardia.

The anaesthetic and breathing systems were immediately checked to exclude any malfunction. As patient's chest and endotracheal tube was not accessible the wound was closed rapidly and patient's position changed to supine. Suddenly ECG started showing ST segment elevation suggestive of acute myocardial ischemia and blood pressure went down to 60 mm Hg Systolic and ventricular tachycardia developed. 'Mill-wheel' sound could be heard on auscultation of the heart. All these parameters were pointing towards gas embolism so Trendelenburg position was given. Anaesthetic gases were discontinued and 100% oxygen was administered. Aspiration through Central venous line revealed lot of air bubbles mixed in blood. Chest compression and Presser support was started. Defibrillation was done with two DC shocks each of 200 J instituted within 30 sec. Gradually all cardiac parameters returned to near normal in next half an hour. After correcting metabolic and respiratory acidosis patient was shifted to neurosurgical ICU. He was kept paralysed and electively ventilated for next 24 hours. HRCT chest and 2D echocardiography didn't show any significant abnormality. Gradual reversal done and patient was extubated uneventfully. Post extubation he was conscious and oriented without any neurological deficit.

DISCUSSION

H₂O₂ rapidly dissociates to water and oxygen and is therefore often considered to be a harmless antiseptic and cleansing agent.¹ When hydrogen peroxide interacts with catalase in human tissue oxygen is produced. During this reaction 1 ml of 3% H₂O₂ produces 9.8 ml oxygen and gas embolism can occur if this is liberated in the vascular bed.

Once produced, the venous oxygen bubbles are carried to the right heart where they can impede cardiac output, or are trapped in the lungs, with a spatial distribution depending on their buoyancy and the flow dynamics within the pulmonary circulation.² If the lungs are severely overloaded with gas, bubbles may also pass through the pulmonary shunt vessels and increase the likelihood of coronary or cerebral vascular obstruction.^{3,6}

Early diagnosis of venous gas emboli can be made by precordial Doppler, transthoracic and transoesophageal echocardiography, the latter detecting as little as 0.02 ml kg⁻¹ of air.⁴ Less sensitive are the presence of a decreased ETCO₂ an increased mean pulmonary arterial pressure, a 'mill-wheel' murmur, hypoxaemia, hypercapnia and hypotension.⁵

In our case, Oxygen bubbles must have entered through raw muscle surface or bony bleeding sites not adequately plugged with bone wax. The diagnosis was made by the development of sudden hypoxia associated with a decrease in ETCO₂ and a 'mill-wheel' sound on cardiac auscultation soon after in irrigation of the solution. ST segment elevation and arrhythmia was suggestive of cardiac ischemia secondary to coronary embolization.

The patient was placed in the Trendelenburg position to entrap bubbles in the right ventricle and reduce the rate of embolization. This allowed us to aspirate the gas bubbles from central venous line. Nitrous oxide is known to increase the size of any gas bubble dramatically so N₂O was stopped and patient was ventilated with 100% oxygen. Active and prompt CPR helped to prevent cardiac standstill and patient survived.

CONCLUSION

Gas embolism associated with the use of hydrogen peroxide, is a rare but potentially fatal complication. We recommend avoiding hydrogen peroxide in procedures where a particular position like prone or sitting increases the risk of venous embolism.

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