ORIGINAL RESEARCH PAPER

Anaesthesiology

NONCARDIOGENIC PULMONARY EDEMA AS SEQUEL OF VENOUS AIR EMBOLISM IN A CASE OF OPERATIVE HYSTEROSCOPY – A CASE REPORT.

KEY WORDS: Hysteroscopy, embolism, pulmonary edema, mill wheel murmur

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ABSTRACT

Hysteroscopy is minimally invasive procedure performed widely for diagnosis and treatment of intrauterine and endocervical pathologies. Venous air embolism, though rare complication, is catastrophic if encountered. We report one such case of venous air embolism leading to non-cardiogenic pulmonary edema encountered by us during hysteroscopic uterine septum resection done for infertility under general anaesthesia. Quick identification, preventing further gas entry and timely intervention saved our patient.

INTRODUCTION

Operative hysteroscopy has been utilized for many years both to diagnose and treat a number of intrauterine pathologies. Although considered to be a very safe procedure, hysteroscopy has several complications. It can be due to cervical trauma, uterine perforation, infection or can be related to distention medium like fluid overload, air embolism. We are reporting a case of noncardiogenic pulmonary edema as a sequel of venous air embolism in a patient undergoing hysteroscopic uterine septum resection. Non cardiogenic pulmonary edema following air embolism is rare event noted primarily in neurosurgery. Pulmonary edema can occur quickly or may take some time and usually occur after multiple air traps. (1) Air bubbles are toxic to pulmonary cells causing increase in vascular permeability and disruption of capillary integrity leading to protein rich exudate leak.

Case Report

A 30 years old, 70 kg woman, with history of two abortions was scheduled for hysteroscopic uterine septum resection in our ambulatory surgical unit. She had no significant past medical or surgical history. Her physical examination and preoperative investigations were also normal. She was taken for surgery under ASA 1 risk status.

After confirming nil per oral status and informed consent for the procedure, she received Inj.Cefotaxime 1 gram iv, Inj Pantaprazole 40 mg iv and Inj. Ondansetron 4mg iv preoperatively. She was taken on operation table. ASA standard monitors like electrocardiogram, pulse oximeter, non-invasive blood pressure monitoring, end tidal carbon dioxide monitoring were established. She was preoxgenated with 100% Oxygen for 3 minutes and premedicated with Inj.Fentanyl 100 mcg iv. Anaesthesia was induced with Inj. Propofol 100mg iv. After check ventilation Inj. Atracurium 30mg iv was given and after ventilating for 3 minutes Proseal LMA no.3 was introduced. Anaesthesia was maintained with Oxygen, Air, Sevoflurane on closed circuit controlled mechanical ventilation. Patient was placed in lithotomy position with 15 degree head low prior to the start of surgery. Patient was vitally stable with Sp02 of 99% and EtCO2 36 mmHg. The distension fluid Glycine 1.5% was placed at height of 60 cm and was infused through Y connectors to allow change over without risk of air bubbles entering the system.

After 30 minutes of procedure when surgeon has just removed the hysteroscope, bradycardia with heart rate of 38/min was noticed on monitor and EtCO2 came down to 8mmHg. Simultaneously blood pressure dropped to 70/40 mm Hg, patient appeared cyanosed but Spo2 was 98%. At the same time, on auscultation mill wheel murmur was present over precordium and bilateral air entry was present. Venous embolism was suspected so immediately, surgeon was informed. Inj. Atropine 0.6 mg iv and Inj. Mephenteramine 6 mg iv were administered .Durant position was given and patient was ventilated manually with 100% oxygen. Intravenous fluids were rushed and blood sample was sent for ABG analysis which showed pH 7.2, pO2 60 mm Hg, pCO2 55 mm Hg.

After 2 minutes of beginning of episode, patient Spo2 dropped to 78%, LMA was removed and patient was intubated with cuffed endotracheal tube no 7. Central venous catheter was inserted and 20ml blood was aspirated but no visible air was retrieved from it. This was followed by improvement in hemodynamics, heart rate 90/min, saturation 95 %, blood pressure 100/60mmHg , EtCO2 28 mm Hg. Patient's color improved and on auscultation murmur disappeared .After spontaneous emergence from anaesthesia patient was extubated with no residual neuromuscular blockade.

In post anaesthesia care unit, patient was awake, conscious, oriented with heart rate of 110/min, blood pressure of 110/ 70 mm hg, oxygen saturation 94% on O2 @ 6 l/min with Hudson's mask. 12 lead ECG and bedside 2D Echo screening was done which showed no abnormality except slightly raised pulmonary artery pressure.

Meanwhile patient became tachypneic (RR= 24/min), bilateral basal crepts were present and immediately Chest X-ray was done which showed bilateral homogenous opacity, suggestive of pulmonary edema. Inj.Furosemide 40 mg iv was given and Oxygen supplementation increased to 10 liters per minute using Hudson's mask with reservoir bag.

After half an hour in PACU, patient developed hypotension so low dose Inj. Noradrenaline infusion was started which was gradually tapered off in next 12 hours. Following day, Chest Xray and detailed 2D Echo was done in which no abnormality was detected .After observation for 2 days patient was discharged.

DISCUSSION

George A Vilos et al in a study on hysteroscopic endometrial ablation found that though the incidence of venous air embolism in hysteroscopy is high, serious consequences are rare (1/1140 cases).(2)

The causes of venous air embolism during hysteroscopy can be multifactorial. It can be gaseous due to CO2 used as distention medium and vapors generated electrosurgically or it can be due to air arising from irrigation solution lines and traumatic insertion of hysteroscopic instruments.(3)

During hysteroscopic procedures, head low position is to be avoided as negative pressure in pelvic veins is created which facilitates air entry.(4) Our patient had a 15 degree head-

down tilt that resulted in a gradient to cause ingress of air into the venous system.

Measures to prevent venous embolism during hysteroscopy such as avoiding head low position, minimizing intrauterine pressure to <100 mm Hg, purging of irrigation fluid tubings, keeping account of irrigation fluid volume, limiting height of fluid bottle to <1 meter, atraumatic hysteroscopic instrument insertion should be considered.

Our patient developed noncardiogenic pulmonary edema 30 min after an episode of venous air embolism. Arora et al reported similar case report of noncardiogenic pulmonary edema following venous air embolism in a patient undergoing transphenoidal pituitary surgery. They believed acute pulmonary edema was due to increased microvascular permeability or rupture of capillary integrity occurring within several hours of the air embolus.(5)

Similar case of non cardiogenic pulmonary edema following air embolism has been reported by Samuel et al during posterior fossa craniotomy.(6)We have not found any similar case during operative hysteroscopy in our literature research.

Another cause of noncardiogenic pulmonary edema during hysteroscopic procedures can be fluid overload due to distension medium used. Jerome J. Grove et al reported a case of non cardiogenic pulmonary edema in a case of operative hysteroscopy and myomectomy where 18 litres of Ringer Lactate was used as distension media with only 12 litres returned and patient developed intraoperatively pulmonary edema.(7) Hysteroscopic procedure for prevention of fluid overload needs meticulous calculation of fluid and electrolyte status, limiting of intrauterine pressure to <100 mm Hg and short duration of surgery. Our patient received 1 litre of Glycine 1.5% as distension medium and surgery lasted for half an hour so there was no fluid overload and it seems to be less likely cause of pulmonary edema. The mechanism of pulmonary edema following air embolism is proposed to be microvascular injury, increase in capillary permeability from mechanical obstruction, toxic oxygen metabolites and leukocyte production leading to protein rich exudates in lung extravascular space.

CONCLUSION

We encountered a rare complication of a relatively rare event. Measures to prevent venous embolism during hysteroscopy should be considered and awareness about them to be increased. In our case venous air embolism lead to noncardiogenic pulmonary edema leading to hemodynamic instability, need for inotropic support postoperatively and intensive care unit stay of patient. Thorough monitoring and vigilance should be used during hysteroscopy for early detection and timely intervention which in our case saved the patient.

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