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Cardiac Arrest During Hysteroscopic Myomectomy - A Combination of Venous Gas Embolism and Operative Hysteroscopy Intravascular Absorption Syndrome

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Abstract

Venous gas embolism is a potentially fatal complication during operative hysteroscopy. Excessive fluid uptake during operative hysteroscopy with Glycine 1.5% can cause hyponatraemia, hypo-osmolality, hyperglycinaemia and volume overload. We present a rare case of venous gas embolism with Operative Hysteroscopy Intravascular Absorption syndrome (OHIA) during hysteroscopic myomectomy.

Introduction

Operative hysteroscopy, a widely used procedure is associated with life threatening complications such as uterine perforation, haemorrhage, gas or air embolism, sepsis and fluid overload with hyponatraemia [1,2]. At times a combination of these complications occur which makes the diagnosis confusing and management complicated. We present a case of cardiac arrest probably due to venous gas embolism, complicated by the presence of operative hysteroscopy intravascular absorption (OHIA) syndrome.

Case report

A young lady had complaints of menorrhagia since one year. She was started on haemostatic agents and Tab. Regestrone and haematinics. Ultrasound showed a 5.8×4.9 cm fibroid in the uterine wall. Her investigations were within normal limits with serum sodium levels of 138 meq/l and potassium levels of 4.2 meq/l. Two units blood were given to correct anaemia. She was posted for hysteroscopic myomectomy.

Monitors included electrocardiogram, non-invasive blood pressure, pulse oximetry, end tidal CO2 and temperature. Patient was induced with glycopyrrolate 0.2 mg, midazolam 2 mg, ramoseteron 0.3

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mg, fentanyl 100 mcg, propofol 80 mg. Patient was intubated with 7.0 size endotracheal tube after 100 mg of succinyl choline. Anaesthesia was maintained with nitrous oxide (50%), oxygen (50%), sevoflurane (1%) and intermittent boluses of atracurium provided the desired relaxation. The patient was loaded with 500 ml 0.9% saline. Uterus was distended with glycine 1.5% and the resection of fibroid was started. Distension pressure was decided by the minimum pressure required for adequate surgical vision.

One hour after the start of surgery when approximately 2 litres of glycine 1.5% was used, patient developed severe bradycardia (86/min to 30/min), desaturation (100% to 70%), hypotension (130/80 mm Hg to 60 /40 mm Hg), hypocarbia (30 mm Hg to 14 mm Hg) and had a cardiac arrest.

Cardiopulmonary resuscitation was started immediately. She was resuscitated, ventilated with 100% oxygen and noradrenaline infusion was started to correct hypotension. Right internal jugular vein and right radial artery were cannulated. The central venous pressures were 10-12 cm of water. On auscultation, chest was clear. Arterial blood gas analysis showed pH of 7.1, PaO2 of 70 mm Hg, PCO2 65 mm Hg and HCO3 of 12 meq/l, sodium levels of 117 meq/l and potassium of 3.3 meq/l. Calcium and magnesium levels were normal. An intravenous bolus of sodium bicarbonate 7.5% (50 ml) was given. The surgery was abandoned after packing the vagina with wet gauze and the patient shifted to the ICU.

To find out the cause of cardiac arrest an electrocardiogram, echocardiogram, chest x-ray were asked for and these were essentially normal. Sodium correction was started as per protocol with 3% hypertonic saline at the rate of 42 ml/hr, which increased the sodium level by 0.5 mmol/l/hr till the sodium level was 120 meg/l after which correction was continued with 0.9 % saline. Patient was ventilated till fully awake and was extubated within a few hours. Sodium levels were corrected over 2 days and she was discharged home after an uneventful postoperative period.

A retrospective diagnosis of venous air embolism with Operative Hysteroscopy Intravascular Absorption (OHIA) syndrome was made. Two months later, she underwent an uneventful hysteroscopic myomectomy under general anaesthesia with strict monitoring of ETCO2, SPO2 and arterial blood pressure and regular sampling of serum electrolytes.

Discussion

VGE is a common complication during operative hysteroscopy [1]. This can be complicated by Operative Hysteroscopy Intravascular Absorption (OHIA) syndrome, caused by intravascular absorption of irrigation fluid during hysteroscopic surgery [2].

Embolism can occur due to either air (atmospheric air) or gas (ingress of insufflating gas such as carbon dioxide or smoke generated during electrothermal procedure) [1]. Embolism of smoke bubbles can be increased by absorption of irrigating fluid in excess of one litre [3].

Normally cardiac arrest during TURP/hysteroscopy is accompanied by volume overload and pulmonary oedema and arrhythmias such as ventricular tachycardia or ventricular fibrillation. In this case, we did not encounter any of these. Hyperkalemia following TURP syndrome could have caused the cardiac arrest but potassium levels was 3.3. Our initial diagnosis was a vasovagal attack, which was unlikely because there were no triggering factor and patient was under deep planes of anaesthesia. Venous embolism as a cause of asystole was retrospectively made by exclusion. The supporting evidences are a fall in BP, SPO2, ETCO2 and cardiac arrest.

Here the venous air embolism was complicated by the presence of OHIA syndrome. Severe hyponatremia and metabolic acidosis point to massive absorption of fluid used for uterine distension. An increased incidence of OHIA syndrome has been reported in myomectomies [2]. Menstruant women have a high risk of death and brain damage when they are exposed to modest hyponatremia

and hypo-osmolemia. This is due to the fact that sex hormones adversely affect the sodium pump in brain cells leading to cerebral oedema [2].

Metabolic acidosis could have been due to dilution, renal bicarbonate wasting secondary to autovolume expansion and probably related to post cardiac status [5].

The biochemical composition of the instilled fluids determines the physiologic alterations. Glycine 1.5% when absorbed in large amounts have been found to be cardiotoxic with echocardiogram changes, elevation of troponin, bradyarrhythmias and cardiovascular collapse [6-8]. The use of Glycine 1.5% might have aggravated the issues in our patients.

Anaesthestic management of these otherwise healthy women should emphasize the prevention of VGE and OHIA syndrome and its attendant morbidity and possible mortality.

To prevent VGE, avoidance of Trendelenberg position, nitrous oxide, ensuring an irrigation system free of air, maintaining uterine distension pressures to 50-100 mm Hg have been advocated [1].

To prevent fluid overload fluid deficit has to be calculated every 10 mins. Accurate calculation of fluid deficit can be made by the newer automated fluid management systems [2]. Mannitol 5% is safer than glycine. Although regional anaesthesia is preferred, GA can be given along with regular monitoring of serum electrolytes. The parotid area sign and the ethanol breath test can be used as early signs of fluid overload [9,10]. Bipolar resectoscopes employing normal saline as distending medium and vaporization techniques are safer alternatives.

Conclusion

Early detection and prompt management of VAE and massive absorption of fluid distention medium is important for patient recovery without neurologic complications. Although treatment regimens for VAE, hyponatremia, hypo-osmolemia and circulatory overload are well established, anesthetic management of these otherwise healthy women should emphasize the prevention of the OHIA syndrome and VAE and its attendant morbidity and possible mortality.

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