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OPERATIVE HYSTEROSCOPIC INTRAVASCULAR ABSORPTION (OHIA) SYNDROME- A COMPLICATION TO BE CONTEMPLATED

Anaesthesiology

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ABSTRACT

Operative Hysteroscopic Intravascular Absorption(OHIA) syndrome considered the gynaecological equivalent of Transurethral Resection of Prostate(TURP) syndrome occurs due to the absorption of fluid medium used during operative hysteroscopy. Use of non-electrolyte containing distending medium increases the risk of complications like hyponatremia, hypoosmolality, fluid overload and pulmonary edema. But when electrolyte containing solutions are used, patient presents with symptoms of fluid overload. We report a case of OHIA syndrome presenting during diagnostic hysterolaparoscopy for evaluation of primary infertility employing 0.9% normal saline as distending medium. This case report describes the successful management of this rare complication in a common procedure with emphasis on preventive measures.

KEYWORDS

INTRODUCTION

Hysteroscopy has been increasingly common since the 1990s.[1] Operative hysteroscopy is now routinely employed for endometrial abalation, septum resection, myomectomy and polypectomy. It has the potential for serious complications like uterine perforation, haemorrhage, air embolism, sepsis and fluid overload with hyponatremia.[2] Operative hysteroscopy intravascular absorption (OHIA) syndrome was first described in 1993 and is considered the gynaecological equivalent of transurethral resection of prostate (TURP) syndrome. It occurs due to absorption of fluid distension medium used during operative hysteroscopy in an exposed vascular bed.[1]

CASE REPORT

A 36 year old female with BMI of 22kg/m^2, presented with complaints of primary infertility, posted for diagnostic hysterolaparoscopy. She had no co-morbidities and her routine investigations were normal and ultrasonography (USG) abdomen revealed septate uterus. She was accepted for general anaesthesia (GA) under American Society of Anaesthesiologists (ASA) grade I.

In the operating room, standard monitors were applied and baseline heart rate 108beats/min, blood pressure 136/90mmHg recorded. A 20-gauge intravenous (IV) cannula was secured and ringers lactate (RL) started according to calculated requirements. Premedicated with inj midazolam 1mg IV, inj glycopyrolate 0.2mg IV, inj pentazocine 30mg IV. Anaesthesia was induced with inj propofol 100mg IV and inj atracurium 30mg IV and a 7.0mm cuffed endotracheal tube (ETT) secured. Anaesthesia was maintained with oxygen, air and isoflurane with intermittent doses of inj atracurium. Intraoperative monitoring consisted of electrocardiography (ECG), non-invasive blood pressure (NIBP), end-tidal carbon dioxide (EtCO2) and pulse oximetry (SpO2).

The patient was then placed in lithotomy position and hysteroscopy was done using 0.9% normal saline as irrigating fluid. Hysteroscopic septal resection was done and the procedure lasted for 90 minutes during which 4L of normal saline was used. This was followed by laparoscopic chromopertubation. Intraoperative hemodynamics was stable throughout the procedure with airway pressures ranging from 16-20cmH_2O. At the end of procedure, isoflurane was stopped and 100% oxygen was given and after adequate reversal of neuromuscular block with normal tone, power, reflexes and regular breathing, patient was extubated.

Within 2 minutes of extubation patient developed tachypnoea and started to desaturate and SpO2 dropped to 85%. . On auscultation diminished breath sounds with fine crackles were heard over bilateral lung fields. Suspecting pulmonary edema, patient was reintubated with 6.5mm ETT, after which pink frothy secretions noted within the ETT. Pulmonary edema was confirmed and the patient was shifted to the ICU.

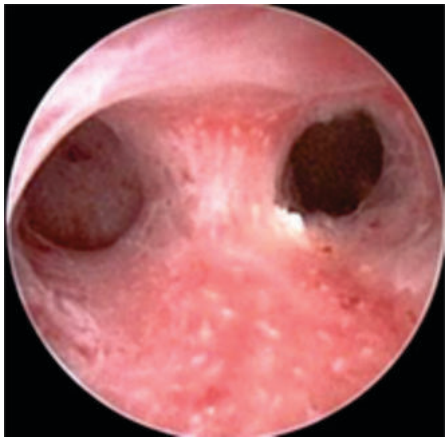

Figure 1- Hysteroscopic Septal Resection

In the ICU, the patient was connected to the ventilator on volume control mode (VCV), with FiO2 of 50%, tidal volume (TV) of 400ml and with a PEEP of 6cm of H2O. Inj Furosemide 40mg IV, inj. Morphine 3mg IV and inj Vecuronium 4mg IV bolus followed by 0.05mg/kg/hr infusion. A nasogastric tube and a foley's catheter was inserted. Blood samples were drawn and arterial blood gases (ABG) and serum electrolytes (SE) were done which was normal. Bedside portable chest radiograph was taken which showed bilateral interstitial infiltrates suggestive of pulmonary edema. After 6 hours of stabilisation and evidence of clinical recovery with good urine output the patient was put on SIMV mode and CPAP trial given for an hour and was extubated.

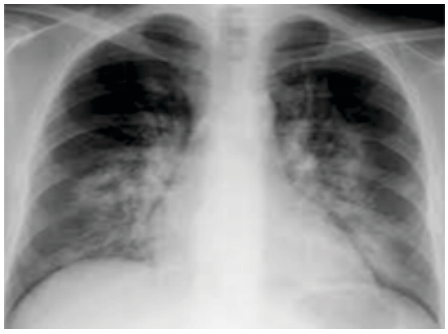

Figure 2- Chest X-ray

DISCUSSION

The incidence of complications of hysteroscopic surgery is as low as 0.24%, but may go up to 10% with more complicated surgeries.[2] During operative hysteroscopy, fluid medium is used to distend the uterine tissue to allow for optimal visualisation. Resectoscopes initially developed used monopolar current which necessitated the use of non-electrolyte containing medium like 1.5% glycine, 5% dextrose with water, 3% sorbitol. With the development of resectoscopes using bipolar current, isotonic electrolyte solutions like RL and 0.9% saline can be used as distension media.[1]

Intravascular absorption of fluid is driven by (1) increasing the hydrostatic pressure gradient between the distension fluid and the vasculature, and (2) increasing the surface area of vascular beds exposed to the distension fluid. Increased operative time increases the risk of absorbing larger amounts of fluid. Among hysteroscopic procedures, myomectomies and resections of uterine septa are at higher risk for increased fluid absorption.

Although OHIA syndrome has similar pathogenesis as TURP syndrome, the uterine wall is thicker and requires higher distending pressures. The pressure in the large venous sinuses in the myometrium is 10-15mmHg, while the intrauterine pressure for distension and flow is generally kept above 40-60mmHg. Intracavitary pressure in excess of mean arterial pressure (MAP) of 80mmHg or 40inches height of water can result in rapid intravasation. The absorbed volume depends of extent of transaction of vascular beds, intrauterine distension pressures, duration of procedure and surgical experience.[2]

OHIA can present with symptoms like perioral numbness, altered sensorium, restlessness and respiratory difficulty and signs like bradycardia, hypotension, oxygen desaturation, increase in peak airway pressures and decreased end tidal carbon dioxide (EtCO2) when patient is under GA.[3] When 1.5% glycine is used as the irrigating medium, they may present with signs and symptoms of glycine toxicity and hyponatraemia.[2] None of these signs were observed intraoperatively in our patient.

Treatment will vary based on symptoms and severity. If an electrolyte-containing isotonic solution such as 0.9% saline was used then the patient will most likely present with symptoms consistent with volume overload, and treatment will center on optimization of respiratory status with supplemental oxygen, non-invasive positive pressure ventilation or intubation as needed and correction of hypervolemia with loop diuretics[1] as seen with our patient. NS induced OHIA syndrome was not associated with dyselectrolytemia.[3]

Fluid absorption can be decreased by keeping the intrauterine or fluid pressure less than MAP, using appropriate distending media and delivery systems, fluid management strategies and keeping the operative time to a minimum. A protocol under GA with electrolyte measurements, echocardiographic or lung ultrasound monitoring for fluid status and observing for parotid area sign should be emphasised.[4] This was the limitation in our case. It may be advantageous to use regional anaesthesia because awake patients are likely to display precursor symptoms.

Figure 3- Parotid Area Sign

CONCLUSION

This report points to the potential of developing OHIA with the use of NS as distending medium. Emphasis is made on preventive measures, early detection and intervention. High vigilance is necessary in the anaesthetic management of these otherwise healthy women.

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