ORIGINAL RESEARCH PAPER | Medical Science | Volume : 6 | Issue : 7 | July 2016 | ISSN - 2249-555X | IF : 3.919 | IC Value : 74.50

A Case Report of Air Embolism During Diagnostic Hysteroscopy and Review of the Literature .

KEYWORDS: Diagnostic hysteroscopy,venous,air-embolism

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ABSTRACT *Though hysteroscopy is a safe and easily executed procedure, it may be associated with mild to severe morbidity and even mortality. The problems associated include innocuous perforation of the uterus , injury to the bladder, bowel and vascular,complications of the distending medium CO2 or air embolism,anesthetic complications and thermal injuries. Venous air embolism is a a rare but fatal complication of hysteroscopy.*

INTRODUCTION:

Here we present a case report of a young female who underwent diagnostic hysteroscopy and was suspected to have sustained air- embolism. The perioperative presentation, management , outcome and the review of literature is discussed.

CASE REPORTS

A 28 years female with bad obstetric history was posted for diagnostic hysteroscopy. The procedure was performed under sub-arachnoid block using heavy Bupivacaine (0.5%) injection 0.5% 2.6 ml at L3- L4 space The level attained was T10 and the patient was comfortable.Then diagnostic hysteroscopy was performed using 1.5% glycine as irrigation solution and manual pressure pump. After 20 minutes of the procedure the patient developed rapid fall in saturation,bradycardia and hypotension with loss of consciousness and sudden decrease in EtCO2 concentration. Immediate Cardio-pulmonary resuscitation was given and endotracheal intubation done.She was resuscitated successfully with a post- resuscitation ABG – pH 7.4,pCO2 52 cm H2O, pO2 50 cm H2O, Na 132 meq/l, K 2.9 meq/l.She was hemodynamically stable, on mechanical ventilation without any inotropic support.She was shifted to postoperative recovery ward on endotracheal tube in situ.She developed pulmonary edema which was treated with Frusenmide injection and she recovered slowly over the next two days.She was extubated on the third post-operative day and shifted to the ward.

DISCUSSION

Gas embolism is a rare complication of hysteroscopy and was first reported in 1985.(1)In a survey of 1000 outpatient diagnostic hysteroscopies no gas embolism was seen.(2) All reported cases of gas embolism have occurred during hysteroscopy associated with some operative procedures. Venous air embolism is a predominantly iatrogenic complication that occurs when atmospheric gas is introduced into the systemic venous system (1). It is mostly associated with neurosurgical procedures conducted in the sitting position.(2) More recently, venous air embolism has been associated with central venous catheterization(3), penetrating and blunt chest trauma(4),high-pressure mechanical ventilation(5), thoracocentesis, hemodialysis, and several other invasive vascular-procedures.) Rapid entry or large volumes of air entering the systemic venous circulation puts a substantial strain on the right ventricle, especially if this results in a significant rise in pulmonary artery (PA) pressures. This increase in PA pressure can lead to right ventricular outflow obstruction and further compromise pulmonary venous return to the left heart. The diminished pulmonary venous return will lead to decreased left ventricular preload with resultant decreased cardiac output and eventual systemic cardiovascular collapse. The rapid ingress of large volumes of air (>0.30 mL/kg/min) into the venous circulatory system can overwhelm the air-filtering capacity of the pulmonary vessels, resulting in a myriad of cellular changes. The air embolism effects on the pulmonary vasculature can lead to serious inflammatory changes in the pulmonary vessels; these include direct endothelial damage and accumulation of platelets, fibrin, neutrophils, and lipid droplets.(1) Secondary injury as a result of the activation of complement and the release of mediators and free radicals can lead to capillary leakage and eventual noncardiogenic pulmonary edema.(1,5) Alteration in the resistance of the lung vessels and ventilation-perfusion mismatching can lead to intra-pulmonary right-to-left shunting and increased alveolar dead space with subsequent arterial hypoxia and hypercapnea.(1,4)

Air embolism has also been described as a potential cause of the systemic inflammatory response syndrome (case report), triggered by the release of endothelium derived cytokines.(6)

In the present case the probable causes of air embolism during the present cases of hysteroscopy could be due to 1) excessive pressure of the irrigation solution exerted by manual pressure pumps, 2) traumatic cervical dilatation or uterine perforation leading to the opening of the venous channels or 3) the air can be introduced during the changing of the irrigation bottle. Open venous channels may be created during dilatation of the cervix for insertion of the hysteroscope; occult false passages may be created at the level of the internal os; or partial penetration into the myometrial wall may occur following forceful dilatation, leaving blood vessels open.(10,11) Ambient air, pressurized gas such as CO2 distension gas, or gaseous products of combustion, may then enter the circulation. Prevention of venous air- embolism during hysteroscopic procedure can be done by-

1. Avoiding Trendlenburg position , as it places the uter us above the level of the heart and creates a venous vaccum with each diastolic relaxation.
2. Minimal cervical trauma and if required the use of os motic dilators.
3. The os should always be kept occluded so as to pre vent the entry of room air.
4. The obstetrician should inform the anesthetist of any

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procedures as trans cervical resection of endometrium or myoma which can open venous sinuses and thus can open the potential portals of air entry.
5. Close monitoring of End –Tidal CO2 for early diagno sis of air embolism.
6. Avoid using manual pressure pumps for irrigation so lution.The use of endomats prevents air-embolism.

The earliest signs of air-embolism are sudden decrease in end tidal CO2 concentration,bradycardia,decrease in oxygen saturation or a mill-wheel murmur on precordial auscultation. However , the characteristic mill-wheel murmur may be a late manifestation of cardiovascular collapse.In our case reports the patients presented with bradycardia, decrease in oxygen saturation, hyotension, decrease in end tidal CO2 concentration.Laboratory tests are neither sensitive nor specific for the diagnosis of venous air embolism. The only indication for obtaining routine laboratory tests is to evaluate the associated end-organ injury resulting from air embolism.Arterial blood gas samples often show hypoxemia, hypercapnia, and metabolic acidosis secondary to right-to-left pulmonary shunting. Chest radiography may be normal or may show gas in the pulmonary arterial system, pulmonary arterial dilatation, focal oligemia (Westermark sign), and/or pulmonary edema.(13) Electrocardiographic (ECG) has low sensitivity for venous air embolism (VAE) detection. The findings closely resemble those seen with venous thromboembolism and include tachycardia, right ventricular strain pattern, and ST depression.End-tidal carbon dioxide (ETCO2) – VAE leads to V/Q mismatching and increases in physiologic dead space. This produces a fall in end-tidal CO2(normal value is < 5). A change in 2 mm Hg ETCO2 can be an indicator of VAE. However, this finding is nonspecific and may also occur with other disease states, such as pulmonary embolism (PE), massive blood loss, hypotension, circulatory arrest, upper airway obstruction, mouth breathing, and/or disconnection from monitor.(1.2.13) End-tidal nitrogen (ETN2) – Most sensitive gas-sensing VAE detection modality; measures increases in ETN2 as low as 0.04%. Response time is much faster than ETCO2 (30-90 s earlier). However, it does not detect subclinical VAE.(1) In our both cases there was a sudden decrease in the ETCO2 concentration. Transesophageal echocardiography (TEE) has the highest sensitivity for detecting the presence of air in the right ventricular outflow tract or major pulmonary veins. It can detect as little as 0.02 mL/kg of air administered by bolus injection.It also has the added advantage of identifying paradoxical air embolism (PAE).(12) CT scans can detect air emboli in the central venous system (especially the axillary and subclavian veins), right ventricle, and/or pulmonary artery. Small (< 1 mL) air defects, usually asymptomatic, occur during 10-25% of contrast-enhanced CT scans; thus, the specificity of this modality is best with large filling defects.(1)Pulmonary artery catheter – Can detect increases in pulmonary artery pressures, which may be secondary to mechanical obstruction/vasoconstriction from the hypoxemia induced by the VAE. However, it is a relatively insensitive/nonspecific monitor of air entrainment (0.25 mL/kg).[1] Central venous catheter – If in place, aspiration of air may help make the diagnosis. It is also helpful in monitoring central venous pressures, which may be increased in VAE(1)

The key to successful management of air- embolism lies in vigilant monitoring and early diagnosis and treatment and prevention the complications due to air- embolism.If air embolism is suspected the surgeon must be informed to terminate the procedure .The source of air entry must be identified and further air entry must be prevented.The patient must be stabilized hemodynamically. For severe refractory hypoxemia and respiratory distress 100 % oxygen and endotracheal intubation must be done. Institution of high flow (100%) O 2 will help reduce the bubble's nitrogen content and therefore size of the air bubbles.(1,2,13) The patient must be placed immediately in the left lateral decubitus (Durant maneuver) and Trendelenburg position. (1,13) This helps to prevent air from traveling through the right side of the heart into the pulmonary arteries, leading to right ventricular outflow obstruction (air lock). Direct removal of air from the venous circulation by aspiration from a central venous catheter in the right atrium may be attempted.(1,2,11) However, no current data support emergent catheter placement for air aspiration during an acute setting of VAE-induced hemodynamic instability. Other than maintaining cardiac output, Cardio-pulmonary resuscitation(CPR) may be initiated if necessary.CPR may also serve to break large air bubbles into smaller ones and force air out of the right ventricle into the pulmonary vessels, thus improving CO.(1,11) Indications for HBOT include neurological manifestations and cardiovascular instability.(15) Supportive therapy should include fluid resuscitation (to increase intravascular volume, increase venous pressure and venous return).(1,2) The administration of vasopressors and mechanical ventilation are two other supportive measures that may necessary.(16)

CONCLUSION

Air-embolism is a rare complication of hysteroscopy .So,the procedure must be performed withmonitoring of blood pressure,heart rate,oxygen saturation and end tidal CO2 concentration.

AKNOWLEDGEMENT

I would like to thank the Department of Anaesthesiology, Southern Railway Head Quarters Hospital, Perambur, Chennai for allowing me to perform the study. I also extent my gratitude to Dr.Kusuma Mathai, HOD Anaesthesiology, for her support.

REFERENCES

1. Mirski MA, Lele AV, Fitzsimmons L, Toung TJ. Diagnosis and treatment of vascular air embolism. *Anesthesiology*. Jan 2007;106(1):164-77.
2. Muth CM, Shank ES. Gas embolism. *N Engl J Med*. Feb 17 2000;342(7):476-82.
3. Pronovost PJ, Wu AW, Sexton JB. Acute decompensation after removing a central line: practical approaches to increasing safety in the intensive care unit. *Ann Intern Med*. Jun 15 2004;140(12):1025-33
4. Platz E. Tangential Gunshot Wound to the Chest Causing Venous Air Embolism: A Case Report and Review. *J Emerg Med*. Sep 15 2008
5. Van Hulst RA, Klein J, Lachmann B. Gas embolism: pathophysiology and treatment. *Clin Physiol Funct Imaging*. Sep 2003;23(5):237-46.
6. Kapoor T, Gutierrez G. Air embolism as a cause of the systemic inflammatory response syndrome: a case report. *Crit Care*. Oct 2003;7(5):R98-R100.
7. Spencer MP, Oyama Y. Pulmonary capacity for dissipation of venous gas emboli. Aerospace Med 1971; 42: 822–7.
8. Brooks PG. Venous air embolism during operative hysteroscopy. J Am Assoc Gynecol Laparosc 1997; 4: 399–402.
9. Corson SL, Brooks PG, Soderstrom RM. Gynecologic endoscopic gas embolism. Fertil Steril 1996; 65: 529–33.
10. Cooper JM, Brady RM. Intraoperative and early postoperative complications of operative steroscopy. Obstet Gynecol Clin North Am 2000; 27: 347–66.
11. Stoloff DR, Isenberg RA, Brill AI. Venous air and gas emboli in operative hysteroscopy. J Am Assoc Gynecol Laparosc 2001; 8: 181–92.
12. Maddukuri P, Downey BC, Blander JA, Pandian NG, Patel AR. Echocardiographic diagnosis of air embolism associated with central venous catheter placement: case report and review of the literature. *Echocardi*

Volume : 6 | Issue : 7 | July 2016 | ISSN - 2249-555X | IF : 3.919 | IC Value : 74.50

ography. Apr 2006;23(4):315-8.

13. Sheasgreen J, Terry T, Mackey JR. Large-volume air embolism as a com plication of augmented computed tomography: case report.*Can Assoc Radiol J*. Oct 2002;53(4): 199-201.
14. Benson J, Adkinson C, Collier R. Hyperbaric oxygen therapy of iatro genic cerebral arterial gas embolism. *Undersea Hyperb Med*. Summer 2003;30(2):117-26.
15. Archer DP, Pash MP, MacRae ME. Successful management of venous air embolism with inotropic support. *Can J Anaesth*. Feb 2001;48(2):204-8