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Original Research Paper

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VENOUS AIR EMBOLISM IN A CASE OF ORTHOTOPIC LIVER TRANSPLANTATION

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ABSTRACT Venous Air Embolism is a rare but fatal complication of many types of surgery in which a sudden entry of a large volume of air into a vessel leads to cardiovascular and neurological catastrophe like hypoxemia, hypercapnia, decreased end tidal CO2, arrhythmias, resistant hypotension, pulmonary hypertension , pulmonary oedema, myocardial and cerebral ischemia. VAE is associated mainly with neurosurgery for posterior fossa tumour, laparoscopic surgeries, vascular surgeries, orthopedic and pelvic surgeries.Vascular air embolism (VAE) during liver transplantation mainly in the dissection phase or during liver reperfusion. We present the successful management of case of a VAE in a case of orthotopic liver transplant in the dissection phase.

KEYWORDS : Venous Air Embolism, Liver Transplantation, Hypoxemia, Arrhythmias, End Tidal Co2, Coagulopathy.

BACKGROUND

Venous air embolism (VAE) is the entrapment of air from ruptured veins to the central venous system producing embolism at the right heart or pulmonary artery. The incidence of VAE is highest with posterior fossa tumour surgery (80%) followed by laparoscopic surgery (60%) , obstetric surgery (25-90%) and hip replacement (30%). The other procedures in which VAE might occur are Central venous catheter insertion, endoscopic procedures, trauma etc. The pathway of venous Air Embolism is shown in figure 1 and surgeries associated with risks of venous air embolism is shown in figure 2 respectively.

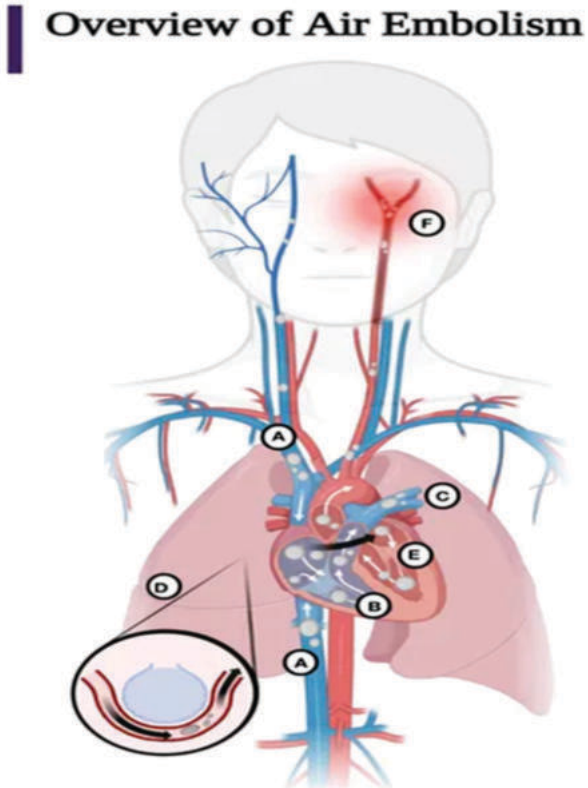

Figure 1: The Routes of Air Entry Resulting in Venous Air Embolism.

Point (A) indicates the entry of air into the superior and inferior vena cava from where most air follows the superior or inferior vena cava into the right ventricle.

The point (B) indicates air in the right ventricle may totally occlude the pulmonary outflow tract causing an "air lock" or "vapor lock" with resultant shock.

The point (C) shows that air may pass through the pulmonary artery, may diffuse into the alveoli, or be trapped in the pulmonary filter causing inflammation with impairment of gas exchange.

Point (D) Air may pass through the pulmonary circulation and enter the left heart and systemic circulation or traverse a patent foramen ovale or a right-to-left shunt (arrow).

(E) shows air may enter the left heart and systemic circulation directly by injection into the pulmonary vein or from the right heart via the lung, a right-to-left shunt, or a patent foramen ovale.

(F) Systemic air embolism, whether from the right heart or injected directly into the arterial circulation causes cardiac or cerebral damage.

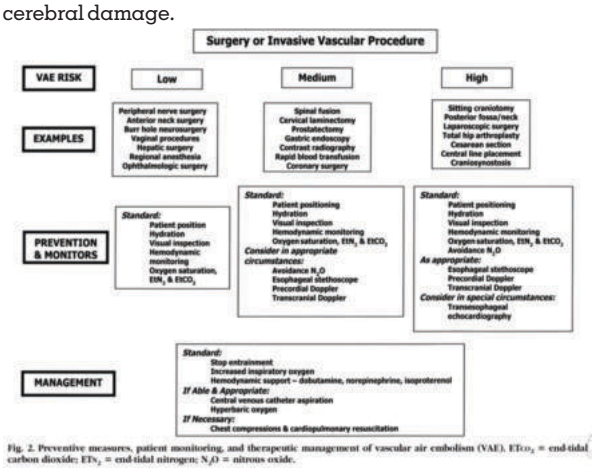

Fig. 2. Preventive measures, patient monitoring, and therapeutic management of vascular air embolism (VAE). $ETco_2$ = end-tidal carbon dioxide; ETN_2 = end-tidal nitrogen; N_2O = nitrous oxide.

Figure-2: The Surgeries Associated with Venous Air Embolism and their Management Algorithm.

A vascular access and a gradient between the injury site and the right heart are prerequisites for VAE to occur[1]. The severity of VAE is determined by the rate,volume, duration, site of air entry, type of gas, position of the patient, calibre of the venous entry and the general condition of the patient. The estimated fatal dose of air in adults ranges from 2-5 ml/kg.VAE leads to trapping of air bubbles in the pulmonary vessels producing gas exchange abnormalities like decreased PaO2, increased PaCO2, and an increase in the alveolar-arterial oxygen difference[2]. We present the case of a Venous Air Embolism(VAE) in a 40 years old male with decompensated chronic liver disease posted for Living Donor Liver Transplantation.

Case Presentation

A 40-year-old man weighing 74kgs, presented with jaundice, ascites, coagulopathy, history of hepatic encephalopathy and oliguria was diagnosed to have cirrhosis of liver and was

planned for orthotopic liver transplantation. The patient had a good effort tolerance with a Karnofsky performance status score of 70 and a MELD-Na score of 27 and CTP- C with Bilirubin- 3.38, INR- 1.81, Creatinine - 1.32, Albumin- 2.24 and Sodium- 126.

The 2D ECHO, ECG and Chest Xray were normal. The patient was evaluated and taken in for liver transplantation after 6hours of fasting. The operation was performed under general anaesthesia using Etomidate, fentanyl, and rocuronium for induction. Invasive lines in the form of a radial arterial line, a 4 lumen central venous catheter and a 8Fr, 11cm long sheath were inserted into the right internal jugular vein. The patient was maintained on 40% oxygen, air and isoflurane with intermittent positive pressure ventilation for maintenance. Infusions of cisatracurium and fentanyl were used for relaxation and sedation respectively. 6 litres of ascites was drained from the abdomen.

Liver Transplantation occurs in 3 phases of surgery namely, the dissection phase, the anhepatic phase and the neohepatic phase. Bleeding, coagulopathy and air embolism mainly occurs in the dissection phase or during reperfusion. After draining the ascites, the liver was freed from the surrounding ligaments, the porta and its structures were identified and encircled. After 3hrs and 42 mins of incision, there was a sudden drop in blood pressure, end tidal CO2 and a concurrent rise of heart rate and peak airway pressure. The blood pressure dropped from 117/67(87) to 37/26(30) mmHg, EtCO2 dropped from 28 to 17, peak airway pressure jumped up from 21 to 35cm H2O all in a minute or two. Sudden onset hypotension was accompanied by cardiac arrhythmias, desaturation and hypoxemia. Fio2 was increased to 100% and flow rate was increased to 4 litres per min.Durant position was used for venous air embolicm and almost 10-15ml of air was aspirated from the right IJV via sheath.

The surgeon confirmed that there was a tear in the inferior venacava and immediately clamped it both above and below the liver. This led to profound hypotension requiring triple inotropes and boluses of adrenaline. There was almost 2 litres of blood loss in 2-3 mins and the massive transfusion protocol was initiated.The hypotension was treated with inj ephedrine 6mg and adrenaline 800mcg along with infusion of Nor-Epinephrine , vasopressin and adrenaline. 3 units of LDPRBC , 2 units FFP and 1 litre of fluid were pushed to rise the ABP to 61/46(51) mmHg and then to 121/83(98) mmHg. Inj Sodium bicarbonate 150ml, 2gms Calcium were infused. Auscultation of the lung fields was normal. The arrhythmias resolved immediately but hypotension and hypoxemia persisted for around 6-8 mins. ABG revealed hypoxemia, metabolic acidosis and low Hb.

The first ABG was taken after 10 mins and showed metabolic acidosis which was corrected using 150ml of Bicarbonate over 20mins and infusion was started at 10ml/hr. Noradrenaline infusion at 20ml/hr (40mcg/ml), Vasopressin at 2.4 units/hr and adrenaline at 10ml/hr (100mcg/ml) were required till the next 40-60 mins and Adrenaline was gradually weaned off after achieving the required MAP with Noradrenaline 10ml/hr and vasopressin 2.4 units/hr.

TEG revealed significant coagulopathy with R- 8.8mins , K - 15.4 min, angle 15.3 and MA- 26.9

A total of 5 units of LDPRBC, 4 units FFP , 1 unit SDPC and 4 units of cryoprecipitate were used.

The patient became normotensive after about 10 mins , though on triple inotropes. The peak airway pressure decreased to 22, EtCO2 increased to 28mmHg, SPO2 was 98% on Fio2- 50%. Inj Furosemide 40mg was given as iv bolus and also 40 meq KCl was infused at 10meq/hr.

Table 1: ABGs at 1Hour Before VAE ,10 mins and 45 mins After VAE and at the End of the Surgery.

Time	1 hr before VAE	10 mins after VAE	45 mins after VAE	End of Surgery
PH	7.38	7.24	7.32	7.36
PaO2 /Fio2 (%)	182/40	88/100	118/50	196/45
PaCO2	34	26	28	32
HCO3	22.6	15.2	18.6	21.3
BE	-2.2	-10.4	-6.5	-4.8
Hb	8.2	5.3	7.8	8.6
Lactate	3.4	7.9	7.6	5.8
Glucose	156	96	102	172
Na/K+	128/4.2	131/3.9	132/3.6	133/3.9

The surgery was continued after the management of VAE and the graft was implanted.The patient tolerated reperfusion well and there was no significant hemodynamic instability.The surgery ended after 14 hours and the patient was shifted on ventilator to the liver transplant icu. A total blood loss was around 4.5 litres which required 14 units of LDPRBC, 8 units of FFPs, 8 units cryoprecipitate, 2 SDPCs and 1 Fibrinogen concentrate. Albumin was infused at 15ml/hr till the end of the surgery. Mannitol 150ml (30gms) was given before reperfusion and the total urine output was 3.6 litres. The patient was extubated on POD1 after the liver doppler and bedside ECHO revealed no abnormalities. The post operative period was smooth and he was discharged on POD 10 with no further complication.

DISCUSSION

Orthotopic liver transplantation is the definitive treatment of chronic liver disease and the surgery involves a number of complex steps associated with vascular identification, dissection, division, ligation and anastomosis. Liver transplantation is conducted in 3 phases namely the Dissection phase, the anhepatic phase and the neohepatic phase and all these steps are vulnerable for vascular air embolism. Most causes of air embolism occur during the surgical dissection phase and during the reperfusion phase [3]. The presence of ascitic fluid keeps the large vessels of the abdomen in a state of positive pressure and with it's drainage the abdomen gets decompressed and the large vessels mainly the IVC goes into a state of normal or negative pressure leading to a pressure gradient between the atmosphere and the IVC. The induced low CVP in recipient surgery further potentiates the gradient of pressure between the vessels and atmosphere.The severity of venous gas embolism depends on the rate of gas introduced into circulation, the volume of gas, and the patient's position. The lethal volume is between 200 and 300ml, or 3–5ml/kg in adults.

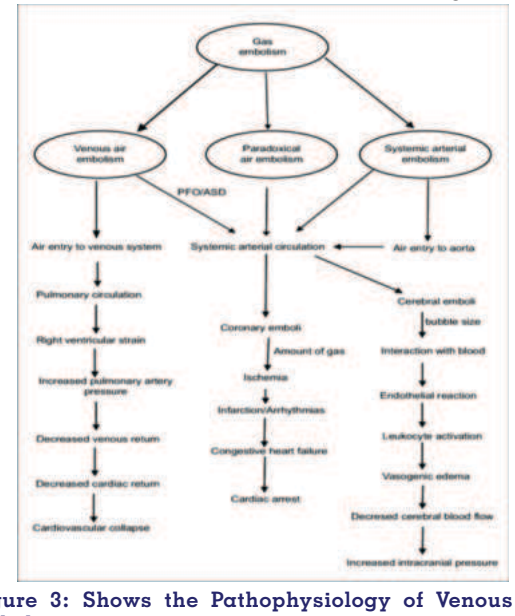

Figure 3: Shows the Pathophysiology of Venous Air Embolism.

The volume of air as well as rate at which the air is entrained usually determines the morbidity and mortality of air embolisms [4]. It is estimated that more than 5 ml/kg of air must be introduced into the venous system to produce symptoms but complications can occur with even 20 ml of air. Even an injection of 1 to 2 ml of air into the CNS can be fatal and as little as 0.5 ml of air introduced into the coronary arteries can initiate ventricular fibrillation [5]. The pathophysiology of venous air embolism is shown in figure - 3.

If a large amount of air enters the right side of the heart, an air-lock mechanism may occur, leading to RV outflow obstruction which causes decreased pulmonary venous return, decreased left ventricular preload and decreased cardiac output which may lead to cardiovascular collapse. There occurs a sudden onset ventilation/perfusion mismatch and alteration in the lung vessel resistance generates an intrapulmonary right-to-left shunt and increased alveolar dead space giving rise to arterial hypoxaemia, hypercapnia and decreased ETCO2 [6]. This patient had an iatrogenic tear in inferior venacava resulting in a VAE which presented as hypotension, arrhythmias, low cardiac output, hypoxemia and low EtCO2. Once it has been diagnosed as a case of air embolism, the position of the patient must be changed into left lateral with Trendelenburg (Durant position) for preventing the air from entering into the pulmonary circulation and also to facilitate aspiration of the air from the right side of the heart. The patient should be ventilated with 100% oxygen in order to wash out entrained air and to improve V/Q mismatch and hypoxemia. We put the patient in the Durant position and resuscitated him with 100% oxygen, inotropes, electrolytes, fluid, blood and products.

The essential steps in management of Vascular Air Embolism are shown in the figure 4 below.

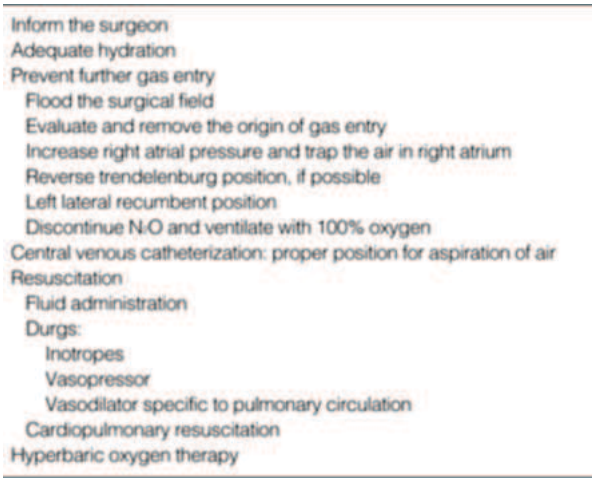

Figure 4 - The Management of Venous Air Embolism

CONCLUSION

High-risk procedures should be identified early and discussed prior to the surgery. Distinct planning, communication between the surgeons and anesthesiologist, strict vigilance during the surgery are the cornerstone in management of VAE. Close monitoring of the hemodynamics helps in early detection and prevention of further air entrainment. A quick attention to volume status is important, as maintaining preload will help to minimise the risk of air entrainment. Management includes resuscitation, prevention of further air entrainment and support of organ dysfunction.

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