

Lethal Air Embolism during Javelin Removal from Skull of a Young Boy

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INTRODUCTION

Venous air embolism is fatal complication mostly occur in neuro-surgical operations particularly in sitting position. It can occur in any operation and in any position when a negative pressure gradient exists between the surgical site and the right atrium of the heart. (Operative site is higher than heart level and venous gradient in more than 5 cm). Its incidence ranges between 16% - 86% depending on the diagnostic technique and intervention used^{1,2}. The true incidence of venous air embolism much depending on the sensitivity of detection methods used during the procedure. VAE has also been reported during central venous catheterisation, endoscopic procedures, and inadvertent injection of air as contrast media and lung or abdominal trauma³. Veins are usually collapsible except veins of scalp and sinuses in the brain that's why venous air embolism is more common in neurosurgery. In addition many cases are at sub clinical level resulting no untoward outcome and go undetected. Here we are reporting a case of air embolism in intra operative period which had cardiac arrest and resuscitated with residual brain damage after successful resuscitation.

CASE REPORT

A 18 year old boy while playing cricket in the stadium where Javelin throw was also being practiced simultaneously. The boy was running to catch the ball, suddenly javelin strike on the head and penetrated the skull. He fell down on the ground and become unconscious. The handle of javelin was cut short and boy was shifted in Hospital for further management. On examination patient was un

conscious, restless, his GCS score was E1V3 M6, respiratory rate 20 / min, heart rate 110/min, blood pressure 128/66mmHg and Spo2- 100% in room air. His body weight was around 50 kg, right eye was black and swollen, not possible to open, left eye was normal reacting to light. The patient was investigated for other systemic or co morbid diseases. The patient was prepared for removal of Javelin under general anaesthesia. In operation room, ECG, NIBP, central venous pressure line, SpO2 and EtCo2 monitors were attached. patient was anaesthetised with midazolam 1 mg, fentanyl 50mg, propofol 120 mg and vecuronium bromide 6mg. patients oxygenation was maintained with oxygen, nitrous oxide and halothane with intermittent ventilation. He was intubated with 7.5 size endotracheal tube and shifted on ventilator for maintenance of anaesthesia. During Surgery Vitals were maintained to acceptable limit (heart rate 86/min, blood pressure 108/ 70mmHg, Spo2- 100%, EtCo2- 38mmHg, CVP 8mmHg). After removal of penetrating object from brain, sudden change in haemodynamic parameters were noticed. Heart rate increased to 150/min, blood pressure decreased to 76/50mmHg, spo2 reached below 70%, central venous pressure increased to 18 mmHg and Etco2 decreased to 12 mmHg). While turning head down and covering of wound site with saline soaked cotton and gauze piece, patient had cardiac arrest. Immediately chest compression was started, aspiration of air from central venous catheter was tried but no air was aspirated.

After about 5 minutes of continuous chest compression, 2 ampules of adrenaline IV bolus at 4minute interval, cardiac function returned with (blood pressure 76/45mmHg and heart rate 180/minutes). The blood pressure was maintained around 96/55mmHg, heart rate 160/min, spo 2 87% with 100% oxygen, etco2- 32 mmHg and CVP 16mmHg. with help of Noradrenaline, Dobutamine and fluid infusion. After haemostasis, wound was closed and patient was shifted in ICU for elective ventilation and further management. Next day patient was assessed and diagnosed as hypoxic brain damage therefore elective tracheostomy was done and

ventilated on support mode for 6 days. On 7th day patient was shifted on T piece with acceptable vitals and on 19th day T piece was removed because oxygen saturation was maintained around 91% to 94% on air and GCS was E1VtM3. The patient was discharged to home on request with tracheostomy tube and advised to review after 1 month. At home good nursing care was provided. After about 40 days of discharge, patient visited hospital for follow up in improved state to E3Vt M4, tracheostomy tube was removed and observed in hospital for 6 days and discharged to home with next follow up after 15 days.



Figure 1.

DISCUSSION

The venous air embolism has been reported during various therapeutic and diagnostic procedures with variable range of incidence between 1.3% to 86% depending on method of diagnosis and sensitivity of the equipment used. The volume of air embolism which prove to be lethal is 3-4ml/kg and the volume of air in venous system that trigger clinical manifestations is about 100 ml for adult². The venous air embolism is often a diagnosis of exclusion in absence of newer and highly sensitive monitoring equipment. Only Presence of air in the right atrium confirmed after aspiration through central venous catheter is 100% confirmatory but some time it is difficult to aspirate if central venous catheter tip is not at the proper place and patient is not in proper position. The sudden drop of end tidal Co₂ level and hypotension is highly suggestive of air embolism but it has moderate sensitivity and specificity² as our patient responded in intra operative period. Precordial Doppler and trans oesophageal echocardiography are highly sensitive to diagnose Venous air embolism. Small volume of air in the heart (0.01ml – 0.19ml/kg body weight) can be diagnosed using trans oesophageal echocardiography⁴.

The gravitational gradients from the venous site of air entrance to right side of heart as low as 5cm has been reported to produce embolization of venous pulmonary circulation. Trapping of air bubbles in pulmonary vessels may lead to cellular injury, lung oedema, release of vasoactive mediators and gas exchange abnormality such as decreased PaO₂, increased PCO₂ and increased A-a DO₂ in arterial blood gas sample^{3, 5}. Embolization of air in the brain may cause deterioration of conscious level and delayed recovery from anaesthesia. In Our patient inadequate recovery after reversal from anaesthesia may be due to air entrapment in the brain or may be because of global hypoxia due to cardiac arrest which persisted about up to 5 minutes.

Treatment of venous air embolism is directed to stopping further inflow of air in circulation and managing complications. If the venous air embolism is suspected, surgeon must be informed to start irrigation of surgical field and provide coverage of exposed blood vessel to prevent further aspiration of air in circulation. Stoppage of nitrous oxide use, 100% oxygen inhalation and other supportive measures, placing the patient in left lateral position so that air can accumulate in right atrium so that it can be easily aspirated are the main treatment. The venous air embolism and associated complications can be prevented using technical capabilities. The patient position, patients hydration, use of positive end expiratory pressure and avoidance of nitrous oxide use are some preventive measure that can reduce incidence and severity of venous air embolism⁶. Venous air embolism is a serious complication that can be prevented and treated by taking precautions during surgery and diagnosis in early stage using modern diagnostic methods.

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