Case Report

A mysterious case of fat embolism

ABSTRACT

We report a patient who sustained catastrophic pulmonary fat embolism post-induction of general anesthesia during laparotomy for haemoperitoneum. The source being the fractured shaft of fracture femur which was missed during the primary survey in the chaos of a positive focused assessment with sonography for trauma and a transient responding patient. In this case report, we want to emphasize the importance of primary survey in a trauma patient, effective communication and documentation to prevent errors and for better management of patients.

Key words: Fat embolism syndrome; primary survey in trauma; point of care ultrasonography

Introduction

Acute trauma/fracture of the lower extremities has been associated with the risk of fat embolism. A subset of these patients lands up into fat embolism syndrome (FES) that is manifested by neurological, respiratory, and cutaneous manifestation. This case report shows the importance of the novel concept of point-of-care ultrasound (POCUS) in diagnosing patients on the OR table and improving management strategies. The case report also signifies the importance of the primary survey and use of clinical acumen.

Case Report

A 26-year-old male weighing 65 kg, with an alleged history of the roadside accident, was admitted in the emergency department (ED) as a transient responder associated with blunt trauma abdomen and open right tibia fracture. The patient was shifted to the operation theater (OT) for emergency laparotomy and external fixator application for a tibial fracture. In the OT patient was drowsy and tachypneic. Modified rapid sequence induction (RSI) was done with fentanyl, ketamine, and rocuronium, and the airway was secured with an 8 mm internal diameter endotracheal tube (ETT). As soon as ETT was connected with the circuit, it was seen that end-tidal CO_2 ($EtCO_2$) was low (8–10 mmHg). Blood pressure (BP) was in the normal range. Soon ventricular premature complexes (VPCs) started to appear on the electrocardiogram (ECG) monitor, which was treated with lignocaine but did not revert and the patient deteriorated into cardiac arrest. Cardiopulmonary resuscitation (CPR) was started as per latest advanced cardiac life support (ACLS) guidelines and the return of spontaneous circulation (ROSC) was achieved within one cycle of CPR. Noradrenaline infusion was started after securing central venous line in the right internal jugular vein (IJV) to maintain hemodynamics, but SpO_2 was 80 on FiO_2 of 100%, despite normalization of BP. Arterial blood gas (ABG) analysis was done which showed high levels of arterial CO_2 ($PaCO_2$),

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but $EtCO_2$ on monitors was on the lower side [Figure 1]. Suspicion of pulmonary embolism (PE) led us to do on table echocardiography (ECHO), which showed a dilated right ventricle (RV) and a shrunken left ventricle (LV) with McConnell's sign [Figure 2]. This confirmed our diagnosis of PE. Dobutamine infusion was started following which the saturation picked up to 100%. Laparotomy revealed a mesenteric artery tear and ileal perforation, which were repaired. Then, the orthopedic team was called for fixing the open tibial fracture. On examination, they found a shaft of femur (SOF) fracture on the left side. In view of the unstable vitals of the patient, the decision to apply external fixator on SOF was made. After confirmation of FES, a bolus of 200 mg hydrocortisone was given as a preventive measure. At the end of the surgery, the patient had landed into pulmonary edema, evidenced by pink frothy sputum seen in ETT. After completion of the surgery, the patient was shifted to the intensive care unit (ICU) for hemodynamic stabilization and mechanical ventilation. Unfortunately, the patient again had a cardiac arrest the following day and could not be revived after 30 min of CPR.

Discussion

FES is most commonly associated with closed long bone fracture of the lower limb, most commonly femur/pelvis. Several diagnostic criteria like Gurd and Wilson's, Lindeque's, Schonfeld's have been used to confirm findings. The pathophysiology of FES can be explained by mechanical and biochemical[1] theory which explains severe hypoxia, hemodynamic instability and pulmonary edema. Massive PE is associated with arterial hypotension, cardiogenic shock and separation phenomenon.[2] Separation is associated with a wide gap between $EtCO_2$ and $PaCO_2$, commonly observed with PE. The mortality associated with FES is 5–15%.[3]

The incidence reported of FES is up to 30%, but many mild cases may recover unnoticed.

Diagnosis of fat embolism is clinical with nonspecific, insensitive diagnostic test results.[4]

Since the diagnosis is clinical mainly and we could not get laboratory reports done in an emergency the diagnosis was based on clinical judgment and imaging (echocardiography). In an emergency bleeding patient with no prior history of long bone fracture, it becomes a diagnosis of exclusion. In our patient after induction, there was a sudden fall in $EtCO_2$ followed by arrhythmia which latter deteriorated into cardiac arrest. Such a picture could be seen in any patient suffering from hypovolaemia/having a prior cardiac history. In the retrospect, we can correlate that the patient was tachypneic and drowsy as well at induction, not an uncommon scenario in a patient having peritonitis; which fits the clinical picture of FES. The INR was also deranged which was again overlooked as it could have been due to some liver injury which the patient might have sustained. In the retrospect, we now know it was due to DIC caused by FES.

The first sign was suspicion was when the $EtCO_2$ levels were low post-intubation despite normal noninvasive blood pressure (NIBP) recordings. Even post-CPR the saturation initially improved and latter deteriorated with persistent low $EtCO_2$, hence we proceeded for on table echocardiography which confirmed our diagnosis. The RV was dilated and LV was shrunken with McConnell's sign (+). We could hemodynamically stabilize the patient only after starting Dobutamine infusion. It was only after the orthopedic surgeon came for fixing open fracture tibia, that we came to know about the SOF fracture. After the completion of the

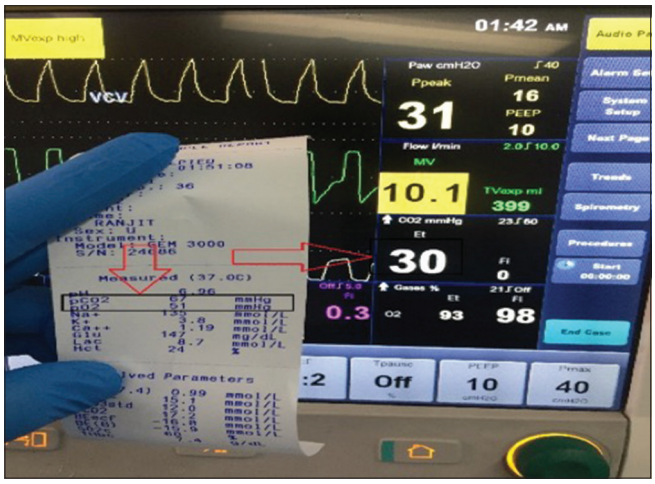

Figure 1: Arterial blood gas (ABG) analysis showing high levels of arterial CO2 ($PaCO_2$), but end-tidal carbon dioxide ($EtCO_2$) on monitors was on the lower side

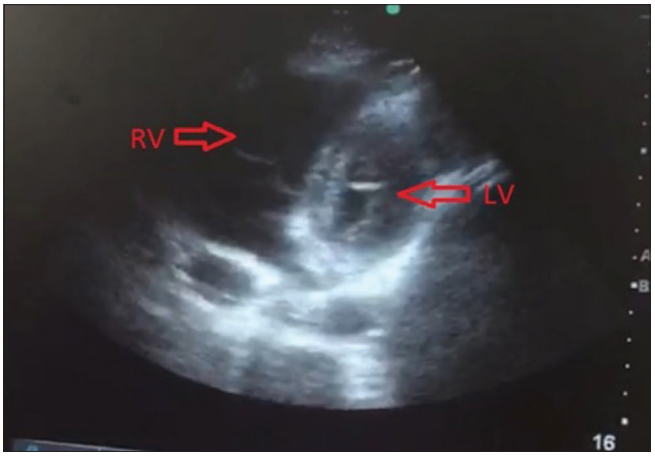

Figure 2: Positive "McConnellæs sign" described as hypo- or akinesis of the right ventricular (RV) free wall seen as dilated RV with preservation of the apex and, hence, a shrunken left ventricle (LV), is associated with acute pulmonary embolism (PE)

surgery, the patient had landed into pulmonary edema due to the inflammatory response to the fat embolus causing leaky pulmonary vasculature.

In the hindsight, had we known about the diagnosis we would have suspected FES earlier and would have been better equipped in handling the situation. The mainstay for the management of FES is preventive and supportive. Had the primary survey revealed a long bone fracture and the patient's limb would have been stabilized with a slab, the fat embolization would not have occurred. Supportive care in the form of oxygen therapy and parenteral steroid would have gone a long way in better management of this patient; by preventing inflammation, perivascular hemorrhage and edema. Heparin which has a stimulatory effect on lipase activity and helps in clearance of lipid from the circulation[1] could also have been used before the setting of coagulopathy had a primary survey revealed a long bone fracture and once FES confirmed in ED.

Conclusion

We faced mortality due to a lack of adequate time to complete the primary survey by the ED team. It is mandatory that the anesthesiologist should know about all possible injuries sustained by the patient in trauma and should be well versed in POCUS. At all times there should be communication between the ED team and anesthesiologist.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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