Case report

Pulmonary embolism occurring early after major trauma

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SUMMARY

Pulmonary embolism (PE) secondary to trauma is the third most common cause of death in trauma patients who have survived 24 hours following injury. We describe a case of PE diagnosed within 3 hours of a major trauma in a previously well adolescent female. The early occurrence of PE in this case is at odds with what is generally reported (3–5 days) after major trauma. General consensus is that patients who suffer major trauma move from an initial hypocoagulable state, with increased risk of bleeding, to normocoagulable or hypercoagulable state, with a subsequent increased risk of venothromboembolism. However, Sumislawski *et al* recently demonstrated that a marginally greater proportion of trauma patients were in fact hypercoagulable rather than hypocoagulable on arrival to hospital and that trauma-induced coagulopathy tended to resolve within 24 hours; such data cause us to re-evaluate when to commence thromboprophylaxis for major trauma patients.

BACKGROUND

Pulmonary embolism (PE) is a common cause of death in patients who survive 24 hours after major trauma.¹ Older age and fracture(s) of long bones increase the risk of PE in such patients.² However, the factors that determine the balance between and timing of prothrombotic and antithrombotic effects after major trauma have not been fully elucidated. In particular, those factors that could predispose to early PE (within 3 hours of injury) are poorly understood. Much of the guideline-based therapeutic effort in this early phase focuses on management of the risk of bleeding and coagulopathy. It is worth considering the possibility that, in certain cases, a readily identifiable complex of clinical circumstances should alert doctors to the risk of early PE after major trauma.

CASE PRESENTATION

A 15-year-old girl with no significant medical or surgical history presented to the emergency department of our hospital as a victim of a road traffic accident with multiple injuries to her lower limbs. On her arrival to the hospital, her vital signs were respiratory rate (RR): 36 breaths/min, SpO₂: 98%, heart rate (HR): 140 bpm, blood pressure (BP): 72/42 mm Hg, Glasgow Coma Scale: 15. She did not complain of chest pain and no clinical signs of deep vein thrombosis were identified. The patient had no history of recent prolonged immobilisation

and no personal or family history of prothrombotic conditions (eg, antiphospholipid syndrome). She was a smoker, was on depot contraceptive therapy and had a body mass index of 23.7 kg/m². The patient was a back seat, unrestrained passenger who was ejected from the vehicle in a high-velocity collision of two cars. She had bilateral lower limb injuries: open fractures of her left tibia and left and right femurs. The accident occurred at approximately 13:00 and our patient was trapped at the scene for more than 30 min. At the scene, a left lower limb, above knee, tourniquet was applied, approximately 45 min after the injury. On her arrival to hospital at 14:30, approximately 90 min after injury, tranexamic acid (1 g) was administered intravenously and a major haemorrhage protocol was commenced. This was begun on the basis of the appearance of substantial blood loss on clothes and surroundings (although the volume of blood was not estimated) and haemodynamic instability (BP: 80/48 mm Hg, HR: 125 bpm).

INVESTIGATIONS

A contrast-enhanced CT scan (CECT) was performed at 15:45, approximately 2 hours and 45 min after the injury, which revealed bilateral pulmonary emboli with a significant burden of thrombus/embolus within the right pulmonary arterial system and extending into segmental branches of the right lower lobe, and likely further embolus within the left lower lobe (figure 1). CT pulmonary angiogram (CT-PA) was performed on third day postoperatively which revealed bilateral pulmonary emboli in lobar and segmental branches of both lower lobes. A subsequent CT-PA performed on the 12th postoperative day demonstrated complete resolution of the PE.

DIFFERENTIAL DIAGNOSIS

Following report CECT, which suggested acute PE, multidisciplinary discussion ensued and questioned this diagnosis because of timing so soon after injury. The possibility of pre-injury PE was not considered. The possibility of fat embolism syndrome (FES) was raised. However, filling defects in the pulmonary arterial system, as seen in this case, are not a feature of FES.⁴ Similarly, CT features of FES including parenchymal consolidation, ground glass opacification, nodules, septal thickening and pleural effusion were not seen on this patient's scan.

TREATMENT

On her arrival to the hospital, the following laboratory results were obtained: haemoglobin (Hb):

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Figure 1 Initial CT scan (contrast-enhanced CT scan) of thorax shows a filling defect (green arrow) in right lower lobe pulmonary artery, consistent with acute pulmonary embolism.

178 g/L, hematocrit (Hct): 0.596, platelets (Plt): 18×10^9 /L, prothrombin time (PT): 19.5 s, international normalized ratio (INR): 1.8, activated partial thromboplastin time (aPTT): 55 s. In total, our patient was transfused during the preoperative period with 10 units of packed red blood cells, 4 units of fresh frozen plasma, 1 unit of platelet concentrate and fibrinogen (2 g). After CT scan was performed, her left leg was judged to be unsalvageable due to the extent of tissue damage and she underwent below knee amputation under general anaesthesia. External fixation was performed on both femurs. No further tranexamic acid or any other blood product was administered intraoperatively. Management of bleeding risk intraoperatively was complicated by the fact that pulmonary emboli were known to be present at a time that INR, aPTT and PT indicated a hypocoagulable state. Transoesophageal echocardiography was performed throughout the procedure, revealing unaffected cardiac function and no evidence of right heart strain. The patient was admitted to the intensive care unit (ICU) postoperatively. Postoperatively, by her arrival to the ICU, the following results were obtained: Hb: 94 g/L, Hct: 0.261 Plt: 151×10^9 /L, PT: 12.1 s, INR: 1.1, aPTT: 27 s. A retrievable inferior vena cava (IVC) filter was inserted on the first postoperative day (subsequently removed on the 15th postoperative day). Anticoagulation with unfractionated heparin was commenced on the fourth day postoperatively and replaced by Tinzaparin 11 000 IU subcutaneously on the seventh day postoperatively. Thereafter, treatment with dabigatran 150 mg orally was prescribed two times per day for the ensuing 6 months.

OUTCOME AND FOLLOW-UP

On the second postoperative day, the patient underwent further orthopaedic surgery: intramedullary fixation of her left femur and open reduction and internal fixation of the right femur. The patient was weaned from mechanical ventilation and inotropic support over the subsequent 3 days and was discharged from ICU on the fourth day after her accident. Subsequently, she underwent several further surgical procedures including wound washouts, application of vacuum-assisted closure (VAC) dressings and re-closure of her stump wound. Shortly after her discharge from hospital, she moved to another country. She has not yet been evaluated at a haematology clinic, with other aspects of her recovery deemed to take priority.

Patient's perspective

'Thank you' ...is doing brilliantly, thanks to your wonderful team. Things could have been a lot worse.

Learning points

- Evidence suggests that there is a significant number of trauma patients in a hypercoagulable state early after trauma.
- Potential benefit of an earlier than usual thromboembolic prophylaxis in this group of patients might exist.
- Identification of patients at risk of early thromboembolism including pulmonary embolism after major trauma is a challenge, which requires further research.

DISCUSSION

Immediately after trauma, activation of the coagulation system occurs. Increased tissue factor production, thrombin generation and its activation take place. Major trauma leads to significantly increased and persistent thrombin generation with disruption of its regulation. Simultaneously, tissue plasminogen activator is released from the endothelium leading to fibrinolysis. Thus, prothrombotic and antithrombotic events occur as a result of major trauma and subsequent inflammation.⁵ The timing and balance between these two sets of events determine whether clotting or bleeding predominates and the time of greatest risk for venothromboembolism.

Trauma-induced coagulopathy refers to the complex of responses to tissue injury and hypoperfusion which results in clinical bleeding tendency. Interestingly, our patient developed PEs despite her abnormal coagulation demonstrated by her initial blood tests. In general, it appears that patients who have suffered major trauma move from a hypocoagulable to normocoagulable or hypercoagulable states, rendering them at risk of PE from 24 hours onwards. However, Sumislawski *et al*³ have demonstrated that 7% of trauma patients are already hypercoagulable on their admission to the hospital.

The dynamic nature of coagulation function under these circumstances may render clinical decisions difficult, as even recent laboratory studies may not reflect 'real time' function. We suggest that there is a role for point of care thromboelastography in this setting, although it was not used in this case. It may identify patients who are truly prothrombotic, who would otherwise be managed in the same way as those who have clinical or laboratory evidence of coagulopathy. We suggest that certain patients are at particular risk for early PE after major trauma and that it may be possible to identify factors (such as the application of lower limb arterial tourniquet) which predispose to this risk.

Contributors All the four authors participated in the care of the patient described in this case and also each contributed to the writing and editing of the case report submitted. PS led the manuscript preparation and participated in its conception. GS contributed to the conception of the idea and planning and design of the case report. MM contributed to the diagnosis, data collection and data interpretation. SPP contributed to the diagnosis, data collection and data interpretation.

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