CASE REPORT

Early intracardiac thrombus and pulmonary embolus after trauma

Neel Desai,¹ Julia Heid,² Andrew Leitch¹¹Department of Critical Care, Royal London Hospital, London, UK²Department of Cardiology, Angiology and Pneumology, University Hospital Heidelberg, Heidelberg, Germany**Correspondence to**
Dr Neel Desai,
neel_d83@hotmail.com

Accepted 7 July 2017

SUMMARY

Here, we present the case of a patient with the findings of an early intracardiac thrombus and a pulmonary embolus after major trauma. A large clot was identified extending from the inferior vena cava into the right atrium and ventricle in the setting of preserved right ventricular function. Post-traumatic intracardiac thrombus is extremely rare and no comparable cases have previously been described in the absence of a congenital heart defect and obvious myocardial injury. Best practice afterpost-traumatic intracardiac thrombus is not well established but we found that early inferior vena cava filter placement and treatment with therapeutic coagulation resulted in clinical improvement, resolution of the thrombus and no further emboli. The successful use of rivaroxaban, a direct-acting oral anticoagulant, to treat a right heart thrombus has, to our knowledge, not previously been reported. Early acute traumatic coagulopathy has received much attention but the hypercoagulable state that often follows is less well appreciated.

BACKGROUND

Major trauma can be complicated by venous thromboembolism (VTE) such as pulmonary embolism (PE) which can result in increased morbidity and mortality. It has previously been estimated that the incidence of clinically apparent PE after trauma is 0.93%.¹ However, the incidence of symptomatic PE after significant trauma varies and is dependent on how it is confirmed.² The median time of diagnosis of PE after acute trauma and elective orthopaedic procedures has been found to be 23 days after the date of injury or the surgical intervention.³ Post-traumatic intracardiac thrombus is extremely rare and, in the case of a right heart thrombus, is most often originated by embolisation from a peripheral venous source but can occur in the setting of a hypercoagulable state.⁴ Here, we present the case of a patient with the unusual findings of early intracardiac thrombus and PE after major trauma.

CASE PRESENTATION

A 24-year-old female was admitted to the emergency department of a tertiary trauma centre after a head-on car versus car road traffic accident associated with a combined speed of 60 mph. Her past medical history included well-controlled asthma and musculoskeletal disease. Of note, she had no personal or family history of thromboembolism and

was overweight with a body mass index (BMI) of 27.3 kg/m². She was not on the contraceptive pill/oral contraceptives and was not a smoker.

Multiple injuries were identified at the primary survey and on radiological imaging. She had a transverse colonic perforation with a moderate quantity of free intraperitoneal haemorrhage and had sustained bilateral femoral shaft fractures, a right tibial plateau fracture and a stable fracture of the third lumbar vertebra with a small paraspinal haematoma. Other than tachycardia, she was haemodynamically stable in the emergency department and her arterial point-of-care tests revealed a pH of 7.33, haemoglobin of 116 g/L, lactate of 2.7 mmol/L and base excess of −5.6 mmol/L.

An immediate exploratory laparotomy was undertaken where a transverse colectomy was performed with subsequent anastomosis of the ileum to the colon and both her femurs underwent retrograde intramedullary nailing. Operative time was 7 hours and she needed a low dose infusion of metaraminol for much of this time. Tranexamic acid was not administered. She was resuscitated intraoperatively with 3 L of crystalloid solution, 2 units of red blood cell and 2 units of fresh frozen plasma. By the end of the operation, all metabolic indices such as pH, base excess and lactate had normalised and oesophageal Doppler monitoring demonstrated a corrected flow time of 407 ms, a stroke volume of 67 mL and a cardiac index of 3.4 L/min/m².

On admission to the intensive care unit, she was initially stable but within 4 hours she had become breathless and there was a sudden increase in her oxygen requirements. From being on 4 L of oxygen via nasal cannulae, when she had desaturated to 82%, she was now on a fractional inspired oxygen concentration of 0.5 on OptiFlow (Fisher and Paykel Healthcare Limited, Auckland, New Zealand). Other observations included a respiratory rate of 20 breaths/min, oxygen saturations now of 92%, heart rate of 122 beats/min, blood pressure of 122/73 mm Hg and temperature of 37.3°C. On examination, auscultation of the chest was normal and no rash was found.

INVESTIGATIONS

Blood results were as follows: haemoglobin 99 g/L; white cell count 2.1×10^9 /L; platelets 142×10^9 /L; urea 4.2 mmol/L; creatinine 70 µmol/L; C reactive protein 91 mg/L; creatine kinase 1825 U/L; international normalised ratio 1.1 and activated partial thromboplastin time 26 s.

CrossMark

To cite: Desai N, Heid J, Leitch A. *BMJ Case Rep* Published Online First: [please include Day Month Year]. doi:10.1136/bcr-2017-220804

Figure 1 Four-chamber view from a transthoracic echocardiogram demonstrating an intracardiac thrombus (white arrows). LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.

Chest xray was unremarkable and the ECG demonstrated sinus tachycardia without any acute abnormalities. Bedside echocardiography found an echogenic structure consistent with a thrombus measuring at least 3.5×0.9 cm in the right atrium, mild tricuspid regurgitation, a dilated right atrium and ventricle, preserved right and left ventricular systolic function and an estimated systolic pulmonary arterial pressure of 35 mm Hg. Images were reviewed by cardiology and they identified a large clot extending from the inferior vena cava into the right atrium and ventricle and probably the pulmonary artery as well (figures 1 and 2). Subsequent ultrasound of the lower limbs showed normal bilateral flow and compression within the proximal femoral veins but a more distal deep venous thrombosis (DVT) could not be excluded as the distal deep venous system could not be visualised. CT pulmonary angiography concluded that there was evidence of a bilateral lower lobe PE with lower lobe consolidation which was likely in part to be infarcted in nature (figure 3).

TREATMENT

A decision was made to not administer thrombolysis as the patient was not haemodynamically unstable. Intravenous unfractionated heparin was commenced and unilateral left thromboembolic

Figure 2 Parasternal short-axis view from a transthoracic echocardiogram demonstrating an intracardiac thrombus (white arrows), measuring 3.5×0.9 cm. LV, left ventricle; RV, right ventricle.

Figure 3 CT pulmonary angiogram slice showing filling defects (white arrows) indicative of bilateral lower lobe pulmonary emboli.

compression stocking was already in place. An inferior vena cava filter (IVCF), the Celect (Cook Medical, Indiana, USA), was deployed within 2 hours of diagnosis to prevent further thromboembolism. Intermittent pneumatic compression was not used. Further treatment focused on conservative and supportive measures, as long as the patient did not clinically worsen, after multidisciplinary discussion with cardiologists, cardiothoracic surgeons, interventional radiologists and respiratory physicians.

Oxygenation and ventilation continued to be supported with OptiFlow and then subsequently with conventional continuous positive airway pressure. Intubation was not needed and within 96 hours, she was on 2 L of oxygen via nasal cannulae. Circulatory support with inotropic drugs was not required at any point. Heparin was converted to treatment dose tinzaparin <2 weeks after presentation. ECGs were repeated daily and regular sequential echocardiograms were examined for changes in the size of the thrombus and ventricular function as well as any other adverse features. IVCF was removed prior to discharge. Five weeks from the date of admission, she was discharged on rivaroxaban and her intracardiac thrombus resolved 4 weeks later.

DISCUSSION

Early acute traumatic coagulopathy has received much attention but the hypercoagulable state that often follows within 48 hours of injury is less well appreciated. It is driven by inflammation, disseminated intravascular coagulation, dysregulation of tissue factor and thrombin, and phospholipids.⁵ In the case of this particular patient, the thrombosis occurred within 18 hours of presentation and on the same day of admission. Consistent with this finding, a recent study found that PE was diagnosed in 57.1% of patients with femoral fracture within 24 hours of injury and in 89.1% of patients within 48 hours.⁶

Established risk factors for VTE in the context of trauma include older age, increasing injury severity score, spinal cord injuries and spinal fractures and blood transfusion. Traditional risk factors such as long bone fractures, pelvic fractures and head injuries, although significantly associated with risk of VTE in single-institution studies, were not found to be powerful risk factors on subsequent meta-analysis.⁷ In a previous study, tranexamic acid did not increase the risk of vascular occlusive events but the precision of the estimates were low and the possibility of some increase in risk could not be excluded. Non-fatal vascular occlusive events were recorded only when there was clear clinical evidence and thus the frequency of these events

may have been under-reported. Tranexamic acid does however reduce the risk of death from haemorrhage.⁸ Potential risk factors identified in this patient for VTE and PE were increased BMI, multiple fractures including those of the femurs and vertebra, prolonged operative time and the administration of blood. An inherited thrombophilia is a possible further risk factor though her family history was negative for VTE.

Chemical prophylaxis for VTE is often delayed after traumatic injuries because of concerns centred on the perceived risk of bleeding. In our opinion, the routine approach to the prescribing of chemical prophylaxis in trauma should include a careful and weighted evaluation of the risk and implications of major haemorrhagic complications against that of VTE. Such evaluation should take place at the earliest opportunity and be repeated if the clinical situation changes and at regular intervals. Relative contraindications to the use of thromboprophylaxis include severe head injuries, non-operatively managed liver or splenic injuries and spinal column fracture with epidural haematoma.⁹

Few case reports of post-traumatic intracardiac thrombus exist in the literature which underlines the rarity of this complication. It has previously been described in a patient who developed an atrial septal thrombus through a patent foramen ovale, associated with abnormal right ventricular movement secondary to blunt trauma and myocardial injury.¹⁰ In our case, there was an absence of a congenital heart defect and obvious myocardial injury, right ventricular function was preserved and the thrombus extended from the inferior vena cava into the right atrium and ventricle. De novo formation of an intracardiac thrombus and PE in a hypercoagulable environment is possible. Embolisation of a DVT could have caused both the intracardiac thrombus and the PE, though embolisation from the intracardiac thrombus alone could have very well produced the PE.

Best practice treatment of post-traumatic intracardiac thrombus is not well established. Usual practice in a non-traumatic intracardiac thrombus, which usually occurs in the left atrium and its appendage, is to consider the use of vitamin K antagonists or, increasingly, direct-acting oral anticoagulants. Rivaroxaban is a selective inhibitor of activated factor X and belongs to this group of drugs but there is a paucity of data regarding its use for right, rather than left, heart thrombus. Its use has been described in the setting of a non-traumatic right ventricular thrombus (RVT) related to dilated cardiomyopathy where it failed to diminish the RVT.¹¹ In this case, rivaroxaban was used successfully to treat a right heart thrombus which, to our knowledge, has not previously been reported.

A decision was made to insert an IVC filter for multiple reasons. First, though the thrombus was situated above the insertion site of the IVC filter, it was thought that it was most likely secondary to embolisation from the lower limbs. Second, in the context of trauma, multiple injuries and recent surgery, commencement of systemic anticoagulation was not without risk. Serious consideration would have been made to discontinue the anticoagulation if bleeding complications had occurred. Discontinuation of anticoagulation in the absence of an IVC filter would have thus left the patient exposed to the risk of potential further embolisation. Third, the patient had experienced an acute and significant deterioration in clinical status and, given the site and size of the thrombus, was at further risk of becoming haemodynamically unstable. Had she become haemodynamically unstable, the risks

associated with transport to interventional radiology for placement of an IVC filter would have been considerable. Early institution of therapeutic anticoagulation and IVC filter placement in our patient resulted in consistent clinical improvement, resolution of the thrombus and no further emboli.

Learning points

- ▶ Trauma can be associated with coagulopathy followed by the presence of a hypercoagulable state.
- ▶ Clinically apparent thrombosis can occur early in the setting of trauma.
- ▶ Chemical prophylaxis for venous thromboembolism (VTE) is often delayed after traumatic injuries because of concerns centred on the perceived risk of bleeding.
- ▶ Consideration of chemical prophylaxis in trauma should include a careful and weighted evaluation of the risk and implications of major haemorrhagic complications against that of VTE.
- ▶ Post-traumatic intracardiac thrombus is extremely rare, can arise de novo or secondary to embolisation from a deep venous thrombosis and has been shown in this case to be successfully anticoagulated with rivaroxaban.

Contributors ND: preparation of manuscript draft. All authors: revision of manuscript.

Competing interests None declared.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

© BMJ Publishing Group Ltd (unless otherwise stated in the text of the article) 2017. All rights reserved. No commercial use is permitted unless otherwise expressly granted.

REFERENCES

- 1 Paffrath T, Wafaisade A, Lefering R, *et al.* Venous thromboembolism after severe trauma: incidence, risk factors and outcome. *Injury* 2010;41:97–101.
- 2 Gudipati S, Fragkakis EM, Ciriello V, *et al.* A cohort study on the incidence and outcome of pulmonary embolism in trauma and orthopedic patients. *BMC Med* 2014;12:39.
- 3 Ho KM, Burrell M, Rao S, *et al.* Incidence and risk factors for fatal pulmonary embolism after major trauma: a nested cohort study. *Br J Anaesth* 2010;105:596–602.
- 4 Turhan S, Ozcan OU, Erol C. Imaging of intracardiac thrombus. *Cor Vasa* 2013;55:e176–83.
- 5 Holley AD, Reade MC. The 'procoagulopathy' of trauma: too much, too late?. *Curr Opin Crit Care* 2013;19:578–86.
- 6 Kim YJ, Choi DH, Ahn S, *et al.* Timing of pulmonary embolisms in femur fracture patients: incidence and outcomes. *J Trauma Acute Care Surg* 2016;80:952–6.
- 7 Rogers FB, Cipolle MD, Velmahos G, *et al.* Practice management guidelines for the prevention of venous thromboembolism in trauma patients: the EAST practice management guidelines work group. *J Trauma* 2002;53:142–64.
- 8 Shakur H, Roberts I, Bautista R, *et al.* Effects of tranexamic acid on death, vascular occlusive events, and blood transfusion in trauma patients with significant haemorrhage (CRASH-2): a randomised, placebo-controlled trial. *Lancet* 2010;376:23–32.
- 9 Gould MK, Garcia DA, Wren SM, *et al.* Prevention of VTE in nonorthopedic surgical patients: antithrombotic therapy and prevention of thrombosis. *Chest* 2012;141:e227s–77.
- 10 Finck C, Kozłowski L, Castle M, *et al.* Traumatic intracardiac thrombus. *J Am Coll Surg* 2000;190:95.
- 11 Elikowski W, Małek-Elikowska M, Słomczyński M, *et al.* [Rivaroxaban-resistant right ventricular thrombus, successfully treated with vitamin K antagonist in a patient with dilated cardiomyopathy]. *Pol Merkur Lekarski* 2016;41:238–42.

Copyright 2017 BMJ Publishing Group. All rights reserved. For permission to reuse any of this content visit <http://group.bmj.com/group/rights-licensing/permissions>.
BMJ Case Report Fellows may re-use this article for personal use and teaching without any further permission.

Become a Fellow of BMJ Case Reports today and you can:

- ▶ Submit as many cases as you like
- ▶ Enjoy fast sympathetic peer review and rapid publication of accepted articles
- ▶ Access all the published articles
- ▶ Re-use any of the published material for personal use and teaching without further permission

For information on Institutional Fellowships contact consortiasales@bmjgroup.com

Visit casereports.bmj.com for more articles like this and to become a Fellow