Venous thromboembolism in the trauma patient

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Abstract

Deep vein thrombosis (DVT) is a common complication amongst patients who sustain major trauma. Whilst DVT may result in long-term morbidity, it is the potential for the fatal consequences of acute pulmonary embolism (PE) that remain a significant cause for concern in the severely injured patient. The incidence and risk factors for venous thromboembolism (VTE) are discussed. The aetiology of thrombus formation in trauma is reviewed in depth.

Multiple methods of thromboprophylaxis exist, both pharmacological and mechanical. Inferior venal caval filters, on the other hand, aim to prevent the emboli from DVTs that have already formed lodging within the pulmonary vasculature. All modalities have potential advantages and disadvantages.

The likelihood of DVT can potentially be predicted by scoring systems, whilst numerous methods of DVT detection can be employed. Once DVT has been diagnosed, treatment should be commenced.

Major trauma patients may sustain a vast array of injuries, and prevention and treatment of VTE in specific injury patterns are reviewed. However, further evidence in the form of multi-centre, randomized controlled trials must be obtained before a standardized protocol for thromboprophylaxis in major trauma can be produced.

Keywords deep vein thrombosis; prophylaxis; pulmonary embolism; trauma; venous thromboembolism

Introduction

It is well recognized that significant multisystem trauma is a risk factor for deep venous thrombosis (DVT) and pulmonary embolism (PE): an observation that was first made nearly 80 years ago.[1] Whilst DVT may cause long-term morbidity, it is the potentially fatal consequences of acute PE that pose particular risks to the trauma patient. Hence, commencement of appropriate thromboprophylaxis is an essential step in the management of the multiply injured patient. There are a number of methods of thromboprophylaxis available in the armamentarium of the clinician caring for trauma victims. However, the decision as to which of these to use, coupled with the timing of their commencement, has been long debated: the risk of thromboembolism must be balanced against the complications associated with the use of thromboprophylaxis.

Incidence

The reported frequency of DVT after trauma varies according to a number of factors, including patient demographics, the nature, site and severity of injury, and the method of detection. Without thromboprophylaxis, however, the incidence of DVT in trauma victims may be more than 50%.[2] DVT may be clinically silent and although DVT itself is not a life-threatening condition, subsequent PE has significant mortality.[3]

Complications of acute DVT

Pulmonary embolism

PE is the most important complication of DVT. Approximately 30% of untreated PEs will cause fatality.[3] It is the third most common cause of death in patients that survive the first 24 h after trauma.[2,4] Proximal lower limb DVTs are probably the source of the majority of PE's.[5]

Post-thrombotic syndrome

Also known as post-phlebitic syndrome, it is a late complication associated with DVT. Clinically it may manifest as pain, oedema, eczema, hyperpigmentation or ulceration, usually around the medial malleolus. Severe cases may result in venous claudication.

Acute recurrent DVT – any patient who has a proximal vein thrombosis must receive adequate treatment, otherwise they have a 47% chance of recurrent thrombus formation over 3 months. Conversely, in patients in whom adequate anticoagulation is achieved, the recurrence rate is 2–4% during the subsequent 3 months.[6]

Risk factors for VTE in trauma patients

A number of risk factors for VTE after major trauma have been put forward. These include increasing age, lower limb, pelvic, spinal or head injury, prolonged immobility, ventilatory support, haemodynamic instability and subsequent surgical intervention[7] (Table 1).

The Eastern Association for the Surgery of Trauma (EAST) has published proposed risk factors for DVT in trauma patients. The guidelines state the level of evidence associated with each specific risk factor according to the US Preventive Services Task Force ranking system. The evidence for patients with spinal cord injuries or spinal fractures is most compelling, with Level 2 evidence that the following risk factors are associated with the development of DVT: older age, higher injury severity score (ISS), blood transfusion rate, long bone fractures, pelvic fractures and head injuries, severe chest injuries and the requirement for mechanical ventilatory support (Table 2).[8]

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Risk factors associated with VTE in trauma patients

Age ≥40 years
Pelvic fracture
Lower extremity fracture
Spinal cord injury with paralysis
Head injury (abbreviated injury score ≥3)
Ventilator days >3
Venous injury
Shock on admission (BP <90 mmHg)
Major surgical procedure

Table 1

Pathogenesis of DVT in major trauma

In 1856, Rudolf Ludwig Karl Virchow proposed a principle for delineating the pathogenesis of venous thrombosis.[9] This came to be known as Virchow's Triad. The theory postulates that vascular endothelial injury, alterations in the constitution of the blood, and alterations in venous blood flow or stasis may precipitate thrombus formation. Whilst the pathogenesis of DVT in major trauma is a highly complex, multifactorial process, involving both acquired risk factors and genetic pre-disposition,[10] the principle of Virchow's triad remains a valid concept (Figure 1).

Venous stasis

Impairment of the usual laminar flow of blood through the venous system can occur in a number of ways. Venous obstruction, increased venous pressure, increased blood viscosity and venous dilation may all cause stasis.[6] Immobilization secondary to spinal protection, limb stabilization, anaesthesia, pain or functional impairment will diminish the 'calf pump' and contribute to stasis.[11] Inability to stand means that the pedal venous pump, triggered on weight bearing through the plantar arch, is abolished.[12] Venous stasis may also be encouraged by the inability of the immobile patient to weight bear. When venous stasis does occur, activated clotting factors can accumulate locally, leading to a prothrombogenic state.[13] Even if venous stasis can be avoided, local trauma may result in eddy currents and vortices within venous valve pockets that in turn may promote platelet deposition and thrombus formation.[14]

The EAST practice management guidelines for the prevention of venous thromboembolism in trauma patients

Level I:

- Patients with spinal cord injuries or spinal fractures are at high-risk of venous thromboembolism following trauma

Level II:

- Older age is an increased factor for venous thromboembolism, but it is not clear at which age the risk increases substantially.
- Increasing injury severity score and blood transfusion appear to be associated with a high-risk of venous thromboembolism in single institution studies, but on meta-analysis these factors this effect did not reach significance.
- Likewise traditional risk factors such as long bone fractures, pelvic fractures or head injuries in many studies may constitute a high-risk patient population in single institution studies but on meta-analysis it did not prove of major significance.

Table 2

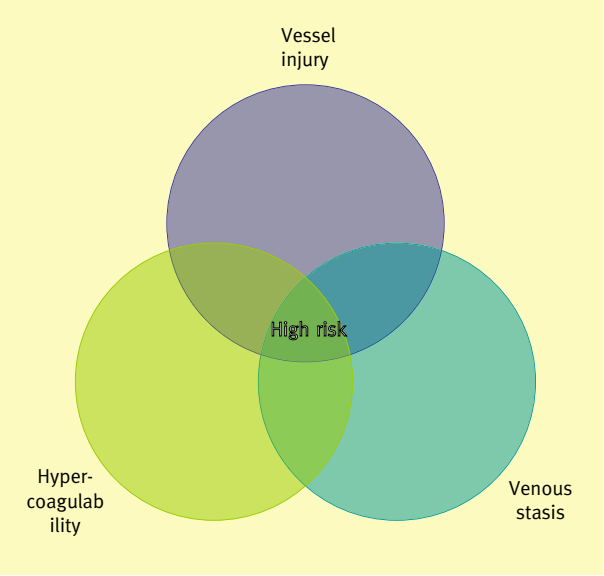

Figure 1 Virchow's triad.

Vascular injury

Vascular endothelial Injury may result from direct trauma, low oxygen tension, thrombin release or cytokines (IL-1/TNF).[6] If endothelial injury occurs, subendothelial cells are exposed to the circulating blood with expression of tissue factor (TF) that may also be produced by leukocytes attracted to the site of the vessel wall damage. Expression of Von Willebrand's Factor and fibronectin results in platelet aggregation and complementary activation of the clotting cascade.[10] Tissue damage may impede fibrinolysis, normally mediated by endothelial cell derived plasminogen activator inhibitor-1.[15]

Hypercoagulability

Although in the acute phase the significantly injured patient may develop a hypocoagulable state due to shock and iatrogenic fluid administration, once stabilized, trauma patients are prone to entering a state of hypercoagulability.[16] Imbalanced activation of the clotting cascade appears to be the most important factor in the development of acute deep vein thrombosis.[10] The clotting cascade is a complex pathway, which ultimately results in the formation of a cross-linked fibrin clot. Disruption of the endogenous coagulation or fibrinolytic systems may therefore contribute to a hypercoagulable state in the multiply injured patient. Tissue factor and markers of thrombin generation increase after trauma,[16,17] whilst endogenous anticoagulants are reduced.[18]

Tissue plasminogen activator (tPA) activity has been found to be suppressed in the multiply injured trauma victim, thus leading to a state of hypofibrinolysis, which may in turn result in venous thromboembolism.[19] Circulatory tPA is bound to its inhibitor