

# Prevention and Management of Thrombo-embolism in Pelvi Acetabular Fractures

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## Abstract

**Background:** Major orthopaedic trauma is a high risk factor for development of thrombo-embolism. The incidence of thrombo-embolism in these patients varies from 30 to 80% and 0.5 to 2% depending on the type of injury, patient profile and diagnosis. Pelvic and acetabular fractures are rare, complex injuries associated with significant morbidity. Fixation of these injuries requires major orthopaedic surgery which is associated with substantial blood loss owing to the extensile operative approach and prolonged operating time. The patient needs to be assessed clinically on the basis of various factors such as calf tenderness, swelling, fever and tachycardia. Proper assessment of the patient clinically is of utmost importance in order to rule out any existing DVT. Immediate stabilization of the fracture either externally or internally plays a vital role in reducing the risk of TE. Anticoagulants should be started pre-operatively and should be continued post-operatively under supervision as a prophylactic measure to prevent TE. Low molecular wt heparin is proven effective for patients undergoing elective surgery and reduces the chances of thrombo-embolism. Use of vena cava filter as a prophylactic treatment can decrease the incidence of pulmonary embolism.

**Keywords:** Pelvi-acetabular fractures, thrombo-embolism, prophylaxis, management.

## Introduction

Pelvic and acetabular fractures have been identified as risk factors for deep venous thrombosis and thromboembolic complications. Most thrombi in trauma patients are proximal in location.[1,2] In pelvis-acetabular injuries, high-velocity trauma itself is an inciting factor for TE, which is aggravated by surgical manipulation and prolonged postoperative immobilisation.[2,3] Proximal veins are involved in around 50% cases. Proximal thrombi have a high propensity to propagate and cause PE, which is the most common cause of death during postoperative period. The rates of PE in patients with pelvi-acetabular injuries range from 2 to 10%, compared to an overall rate

of 0.5 to 2% in trauma patients. [4] Injury to posterior structures is associated with more severe trauma than anterior injuries, and occurs when the limb is flexed and adducted at the time of impact, which is the same position that causes kinking of femoral vessels [5,6] An intrapelvic haematoma may compress the pelvic vasculature. Surgical treatment is often delayed and post-operative rehabilitation slow. Numerous studies suggest that deep-vein thrombosis in the pelvic venous plexus is more common after pelvic and acetabular fractures. [7,8,9] Thrombus formation in uninjured limb is due to procoagulant environment in post trauma patients, contributed by release of tissue factors, decreased Antithrombin III, release of acute phase reactants from liver and unregulated Thrombin formation.

high energy trauma, associated with injury to vascular structures and prolonged immobilization. The classical triad of Virchow can explain the pathogenesis of venous thrombosis in these patients. This triad labels the three factors that are thought to generate the thrombosis: hemodynamic changes, endothelial dysfunction and hyper-coagulability status. In these cases the deficiency of the laminar flow through the venous system can be caused by immobilization due to traumatic radicular lesions, limb fractures, stabilization, anaesthesia or pain. This situation will weaken the calf pump and contribute to vascular stasis. Vascular endothelial dysfunction may result from direct trauma or by the use of surgical techniques that may promote tissue necrosis or direct vessel damage. Pelvic fractures are profound stimuli for the activation of the coagulation cascade. Although initially, in the acute phase, the significantly injured patient may develop a hypocoagulable status, once stabilized, trauma patients are disposed to suffer a state of hypercoagulability, being the most important factor in the development of acute DVT the imbalanced

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## Pathophysiology And Risk Factors For Vte

Pelvic and acetabular fractures usually result from

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activation of the clotting cascade. Tissue factor and markers of thrombin generation increase after a major trauma, while the endogenous anticoagulants (i.e., antithrombin III) show a tendency to be decreased.

### Prevention

Prophylactic therapy after orthopedic surgery has been shown to prevent 49–71% of DVT. [10-12] Patients with pelvic trauma are at high risk of thromboembolic complications, but effective methods of prophylaxis have still to be accepted and adopted. TE after major trauma is the most common cause of morbidity and mortality in patients who survive the first 24 hours. The administration of LMWH (Enoxaparin; 40 mg by subcutaneous injection, once daily) within 24 hours of injury or on establishing haemo-dynamic stability is of prime importance. Colour Doppler is the mainstay in preventing TE by detecting blockage early in a pre-operative period. The patient should be assessed using colour-flow duplex ultrasonography of the external iliac, femoral and popliteal veins in both legs, if the transfer of the patient from the referring hospital had been delayed by seven days or more. The inferior vena caval filter should be used before operation if a proximal DVT was detected. The patient should be started on anticoagulants (abigatran, rivaroxaban, enoxaparin and apixaban) after 12 hours of surgery to prevent the development of fatal PE.

Temporary external fixation followed by internal fixation for open complex fractures of the lower limbs serves as damage control [13-15] and stabilisation. [16-19] Stabilization of fracture prevents abnormal mobility at the fracture site avoiding further soft tissue damage as well as neurovascular compromise. Mobility at the fracture site may lead to dislodgement of thrombus leading to TE.

Colour Doppler ultrasonography is largely insensitive in diagnosing pelvic vein thrombi, and a second diagnostic tool for evaluation of PE is needed. [20,21] The relatively low reported rates of TE could be attributed to this problem. [22,23] Ultrasonography is being used as screening tool to detect TE in pelvic and acetabular fracture patients. It is non-invasive and can be applied to patients bedside. Contrast enhanced CT can detect TE by scanning the chest, which differs from ultrasound. Contrast-enhanced CT can also be used to detect DVT [24], especially pelvic DVT, which is difficult to detect by ultrasound [25]. Magnetic resonance venography (MRV) is non-invasive and has 100% sensitivity and 97% specificity. [21] It can detect very small thrombi which may not be clinically significant. CT pulmonary angiography and indirect venography (CTVPA) are highly sensitive (97%) and specific (100%) for femoro-popliteal thrombus and can detect both PE and DVT using same contrast in the circulation. [26,27] It can also detect pelvic and vena caval thrombi, which are

not detected by ultrasonography.

### Management

Patient should be started on anticoagulants 12 hrs after the surgery is performed. It helps in improved limb circulation. Patient should be mobilized within 48 hrs from surgery. Use of DVT pumps has now-a-days increased and surgeons are taking due care to use them prior and after the surgery in cases of acetabular fractures where early mobilization is not possible.

The use of antifibrinolytics is still relatively new and their role is yet to be fully defined in the context of orthopaedic surgery. Major orthopaedic operations are associated with significant blood loss and provide a challenge in blood conservation. Prophylactic inferior vena cava filters (ICVFs) can be used to prevent pulmonary emboli as a common last resort. These endovascular devices do not prevent the development of a deep vein thrombus; they interrupt the flow in the inferior vena cava to prevent the most significant sequel of DVT, the life-threatening pulmonary embolus. Filters have been related as preventing PE in the existence of proven lower limb DVT in 98% of cases. [28]

### Conclusion

Pelvic-acetabular trauma is a significant risk factor for VTE, even in Indians. Patients who have posterior injuries or are operated on in the lateral position, or via the Kocher-Langenbeck approach have a significantly higher risk of VTE.

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