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Does Deep Vein Thrombosis Usually Precede a Pulmonary Embolism

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Abstract

Background: Trauma patients are at considerable risk for developing a Deep Vein Thrombosis [DVT] and subsequently experiencing a Pulmonary Embolism [PE], a process known as Venous Thromboembolism [VTE], which is a life-threatening complication that continues to be a major medical challenge.

Objective: To determine if a trauma patient who sustains a serious chest trauma as defined by the Abbreviated Injury Scale of 3 or more should be considered a high risk candidate for PE.

Method: This is a twelve-year retrospective study done at St. Mary's Medical Center, a level II trauma center in West Palm Beach, FL, reviewing all trauma charts and autopsy reports of those patients who were diagnosed with PE, regardless of their mechanism of injury or initial diagnosis.

Results: A total of 18,451 patients were admitted to the trauma service during this time and 30 patients [0.16%] were diagnosed with a PE during their hospital stay or on autopsy. Among the 30 patients with a diagnosis of PE, 15 patients [50%] sustained chest trauma including diaphragmatic injuries, rib fractures, pneumothorax, and/or pulmonary contusion, and seven of those patients did not have a documented DVT. Among the 30 patients with PE, 18 [60%] did not have a detectable DVT.

Conclusion: This study supports the current literature that although DVTs are the most common cause of PE, severe chest trauma should be considered a significant contributing factor due to thrombi forming within the pulmonary vasculature.

Keywords: Deep vein thrombosis; Pulmonary embolus; IVC filters; Deep vein thrombosis prophylaxis

Background

Historically, traumatic brain injury, pelvic and lower extremity fractures, paralysis or any condition associated with prolonged immobilization were considered risk factors for Deep Vein Thrombosis [DVT] [1] which can predispose to a potentially life threatening pulmonary embolism [PE], a process known as Venous Thromboembolism [VTE] [2]. There is no disputing that the incidence of VTE depends on a patient's risk factors along with the modalities used to prevent and detect DVT and PE. However, there is some dispute as to whether a PE is usually preceded by a DVT, detected or undetected, and whether the prophylactic use of inferior vena cava filters is appropriate in these patients. Some recent studies suggest PEs are not always from a DVT but instead may originate from thrombi formed within the pulmonary vasculature following severe chest trauma [3]. In past years common knowledge presumed that a PE invariably resulted from a DVT within the deep veins of the lower extremities or within the pelvic iliac veins. Since not all DVTs are detected on ultrasound studies, the iliac veins were usually considered as the originating site when a PE was diagnosed without a positive ultrasound study for DVT. However, recent studies showed that in some patients who had experienced PE had a negative ultrasound and Computed Topography [CT] of the pelvis failed to reveal DVTs within the iliac veins as previously suspected [3-5]. These studies support the fact that PE may potentially develop de novo in the pulmonary circulation and not deep within the iliac veins as originally suspected [5]. In addition, the American College of Surgeons found the incidence of PE to be 42% higher in patients who sustained serious chest injury, defined as an Abbreviated Injury Scale score of 3 or higher, compared to those without serious chest injury [3]. In summary, these referenced studies provide data suggesting that patients with chest trauma are more at risk for PEs that originate from the pulmonary vasculature than originally thought. This twelve-year retrospective study was performed for the purpose of comparing the correlation of DVT and subsequent PE in the trauma population with and without severe chest trauma.

Pathophysiology

Three factors predispose to thrombus formation: endothelial injury, stasis or turbulence in blood flow, and hypercoagulability. These three factors are known as Virchow's triad. Endothelial injury occurs any time the collagen within the vasculature is damaged and the clotting cascade is initiated. Examples of injury to the endothelium can be damage to the vein from trauma or hypertension. With vessel injury, an alteration in blood flow occurs, causing turbulence and stasis within the vessel. Blood hypercoagulability is a result of a complex mechanism known as the clotting cascade. The clotting cascade involves several components, including platelets and numerous clotting factors used to maintain blood homeostasis. The clotting factors are composed of enzymes and proteins. In addition, there are three main pathway components: (1) the extrinsic or tissue factor pathway, (2) the intrinsic or contact activation pathway and (3) the common pathway. The extrinsic pathway is the most significant pathway in the clotting cascade. Once a pathway is triggered and chemical mediators have

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stimulated the common pathway, the clotting cascade is commenced and each stage of the process initiates the release of the next factor until fibrin is produced. The main factors within the clotting cascade include: fibrinogen, prothrombin, tissue factor, calcium ions, ace globulin, prothrombinase, antihemophilic Factor A, plasma thromboplastin component, Stuart factor, anti haemophilic Factor C, Hageman's factor, and fibrin stabilizing factor. The tissue factor pathway stimulates the release of thrombin normally, but when injury to the vasculature occurs the clotting cascade commences causing a hyper-coagulation state. In addition, coagulopathy is present in 25% of trauma patients and is linked with a 5-times increase in mortality [6].

PE is associated with respiratory and hemodynamic decline. From a respiratory stand point, increased alveolar dead space, hyperventilation and ultimately hypoxemia occur. The hypoxia is a result of ventilation-perfusion mismatch, which causes physiologic alveolar dead space, regional loss of surfactant and, although less frequently, pulmonary infarction. If the obstructed pulmonary vascular bed is substantial enough, it causes an increase in right ventricular afterload and, if persistent, results in a sequence of right ventricular enlargement, right-side heart failure, and hemodynamic decline. A trauma patient who sustains multiple injuries and displays nonspecific signs and symptoms proposes a considerable medical challenge in terms of diagnosing PE. Many times patients with blunt chest trauma may present complaining of chest pain, complaining of abdominal pain, or be unresponsive. Often, trauma patients are emergently rushed to the operating room to control bleeding and are then sedated and ventilated post-operatively. Unfortunately, this scenario can lead to a delay in diagnosis and subsequently a delay in treatment. The delay may consist of several days, especially when a patient is ventilated and sedated for an extended period of time. Patients who sustain a severe Traumatic Brain Injury [TBI] are also at risk for a delay in diagnosis of a PE due to difficulty of assessment when neuromuscular blockade, sedation, and mechanical ventilation are being instituted as part of the treatment of TBI patients. Tissue factors play an important role in coagulopathy, which in turn plays a significant role in patients with a traumatic brain injury due to the activation of the extrinsic pathway of blood coagulation. However, if no treatment is initiated for a PE; roughly one third of patients who survive an initial PE will die from an ensuing embolic episode [1].

Prophylactic Interventions at St. Mary's Medical Center

Inferior Vena Cava [IVC] filters are commonly used as prophylactic interventions for patients at risk for PEs due to DVTs at St. Mary's Medical Center. However, in patients with chest trauma who are potentially at risk for experiencing PEs due to an embolus arising primarily from the pulmonary vasculature, IVC filters would not be an effective prophylactic treatment [3]. However, several prophylactic interventions are used to reduce the risk of VTE. The interventions include pneumatic compression devices, routine venous Doppler screening, and anticoagulant administration, which are accepted interventions in most patients. Various prophylactic compression devices are available on the market, including pneumatic planter A-V foot pumps, sequential pneumatic compression, and graduated compression stockings. All of these devices function to [1] increase venous flow, [2] reduce venous stasis, and [3] ultimately reduce the risk of DVT. Routine venous Doppler screening aids in early detection of DVT and subsequent treatment. According to the American College of Chest Physicians [ACCP], a mechanical compression device is recommended until low-molecular-weight heparin is considered safe, and the use of an IVC filter for trauma and spinal cord injury is not recommended [7]. However, when anticoagulation is contraindicated in the trauma patient, an IVC filter may be indicated [8]. At St. Mary's Medical Center IVC filtering devices are used when DVT or PE are clinically suspected or diagnosed by Doppler ultrasound, CT scan, angiogram or other modalities. IVC filter are considered for exclusion criteria for DVT/PE prophylaxis with heparin or Lovenox, which include any patient with active bleeding, heparin induced thrombocytopenia, hypersensitivity to heparin or Lovenox, clotting disorder, traumatic brain injury or spinal cord injury within 7 days, recent chemotherapy, uncontrolled acute hypertensive emergency, recent spinal tap or indwelling catheter, recent intraocular surgery, and hemorrhagic stroke or gastrointestinal bleed within six months.

When not contraindicated, pneumatic compression devices are used for all trauma patients that are not ambulatory. Marketing claims are based on the premise that compression devices are used to decrease venous stasis by increase blood flow and velocity in the lower extremities, ultimately reduce the chance of DVT. There are several different devices available however; the protocol at St. Mary's Medical Center recommends the use of sequential calf compression. All non-ambulatory patients on the trauma service have ultrasound venous Doppler studies to the lower extremities every Tuesday and Friday while on the trauma service. Once a patient has an IVC filter device inserted then ultrasound studies are repeated every ten days or sooner if swelling and/or pain are present in the lower extremities.

Methods

Over 18,000 patients met trauma criteria using a trauma scorecard and were admitted to the trauma service at St. Mary's between January 1st, 2000 and December 31st, 2011. This twelve-year retrospective study, which was approved by International Review Board, was performed for the purpose of comparing the correlation of DVT and subsequent PE in the trauma population with and without severe chest trauma. Therefore, no statistical analysis was performed. All initial information was retrieved through the trauma-one registry, with a diagnosis of PE using the ICD-9 code. Once a list of medical records was obtained, the chart review process began. Many variables were reviewed including age, gender, mechanism of injury, diagnosis, pre-existing conditions, central line placement, IVC filter placement, injury severity score, length of stay, day of hospitalization that the PE/DVT was diagnosed, activity level, and when and if DVT prophylactic protocol was initiated. Although all these variables were recorded the variables used for this study included, [1] the diagnosis to determine severe chest trauma, as defined by the Abbreviated Injury Scale of 3 or more and [2] the diagnosis of DVT/PE. No trends were noted in the other variables that would add any value to this study and therefore were not included. All charts were reviewed in a methodical process to assure the correct ICD-9 codes were valid. Inclusion criteria consisted of only those charts with radiological confirmation of PE, or by autopsy reports. Exclusion criteria consisted of any charts that did not confirm a documented PE either by computerized tomography angiogram, lung scan, if a patient was allergic to the dye, or visualized on autopsy. A total of 30 charts met criteria and were included in the study. A positive DVT consisted of either complete or partial obstruction of a deep vein in the lower or upper extremities, detected by real time ultrasound compression and Doppler for augmentation.

Results

A total of 18,451 patients were admitted to the trauma service between January 1st, 2000 and December 31st, 2011. Throughout this time period 30 patients [0.16%] were diagnosed with a PE either during their hospital stay or on autopsy. Three patients were diagnosed with

a PE on admission and a total of 18 patients were diagnosed within 10 days of admission. Among the 30 patients with a diagnosis of PE, 15 patients [50%] sustained chest trauma including diaphragmatic injuries, rib fractures, pneumothorax, and/or pulmonary contusion, and seven of those patients [46.7%] did not have a documented DVT. A total of 15 patients did not sustain any chest trauma and 11 of those patients [73.3%] did not have a documented DVT. Among the 30 patients with PE, 18 [60%] did not have a detectable DVT 9 (Figure 1).

Discussion

Three patients were diagnosed with a PE on admission and a total of 18 patients were diagnosed within 10 days of admission suggesting perhaps a hypercoagulable state. This data parallels the National Trauma Data Bank study suggesting severely injured patients are already coagulopathic secondary to shock as their protein C becomes depleted and they become hypercoagulable [3]. The data obtained during this study at ST. Mary's Medical Center reflected that 60% of the patients who experienced a PE did not have a documented DVT which parallels the current literature that a PE may develop from severe chest trauma [5]. Although real time ultrasound venous Doppler studies are used to detect DVTs, they are limited to detecting thrombi that form within the deep veins of the lower extremity and are unable to detect thrombi that form within the iliac veins. Therefore, a negative DVT study does not always confirm the absence of a DVT. Additionally, patients who sustained injuries of the lower extremities, with stabilizing devices applied or massive tissue loss and bandages surrounding the extremity, are more difficult, if not impossible, to accurately assess via venous Doppler ultrasound making a diagnosis of DVT easy to miss. Because many patients on the trauma service sustain severe injuries that require the induction of a medically induced coma with sedation and ventilator support, it is conceivable that a patient may have experienced an undetected PE. These patients would be unable to display the typical signs and symptoms of PE, i.e., dyspnea, tachypnea, and/or chest pain.

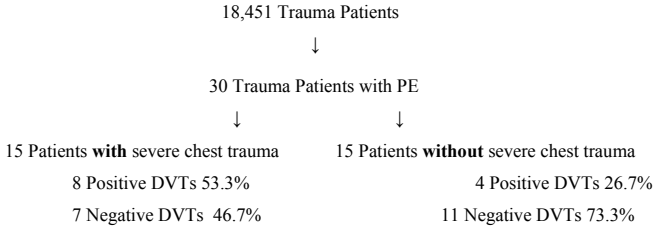

Figure 1: Study of Deep vein thrombosis.

All of these clinical limitations obscure the actual influence of a negative DVT study on the likelihood of PE in the context of trauma patients in general, and in particularly trauma patients who have sustained severe chest trauma. To more accurately assess risk or presence of PE in these patients, given a negative DVT study; it may be useful to employ a pelvic CT to further assess risk due to iliac thrombi and/or chest CT angiography to assess presence of embolism in the pulmonary vasculature.

Conclusion

Of the patients in this study, 50% sustained severe chest trauma prior to the PE and 46.7% of those patients did not have a documented lower extremity DVT. Therefore, it is valid to ask whether chest trauma should be considered as an important risk factor when evaluating patients for potential PE development. This study concurs with recent literature that some PEs do not stem from DVTs; in fact, 60% of the patients with a confirmed diagnosis of PE did not have a documented DVT. Additional beneficial research would seek to determine whether, in patients who have sustained chest trauma, the source of PEs are more likely from thromboses deep within the iliac veins or more likely from the pulmonary vasculature after sustaining chest trauma. Prospective studies using CT scans of the iliac veins and CT angiography of the chest would be beneficial in determining the origin of PEs in the presence of a negative DVT study. Such research could help form guidelines that would help avoid the installation of an unnecessary IVC filter and the risks associated with the procedure.

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