Deep Vein Thrombosis (DVT)

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Introduction

Deep vein thrombosis prevention and treatment touches a vast array of patient populations. Development of a DVT can occur when an individual is at home or in the hospital. Patients at risk for DVT include those undergoing surgery, vascular disorders, or trauma. Situations in which individuals can also put them at risk including pregnancy, surgery, and medications (Bruni-Fitzgerald, 2015). In the United States, 200,000-600,000 people develop DVT each year. The most serious complications from a deep vein thrombosis (DVT) include pulmonary embolism (PE), which occurs in up to 5% of patients and has a mortality rate of up to 30% (Anthony, 2013, p. 95). Medical costs for DVT in the US have been estimated to total $5-10 billion (Nelson, 2011, p. 1). Deep vein thrombosis prevention in the nursing setting is a priority especially in high risk individuals. Anticoagulation therapy remains the primary treatment, but still requires monitoring and follow-up. Knowledge of the pathophysiology of a clot will aid in the understanding of treatment options.

Pathophysiology

1. The pathophysiological process of developing a DVT is a complex series of events. Hemostasis is the process of clot formation at the site of vessel injury and is divided into five steps. 2. Contraction of the smooth muscle in the vessel wall is initiated by an injury to the endothelium. The contraction slows the blood flow and platelet activation. 3. Platelets (thrombocytes) contain chemical mediators. Once activated by contact with collagen or von Willebrand factor the platelets change from a disc shape to a spiny sphere. The activated platelets also release additional chemicals that will enhance smooth muscle constriction, which will begin to stick to one another and form a plug. Additionally, receptors on the platelets for thrombin, fibrinogen and clotting factors will begin the clotting cascade (Casey, 2011, p. 12). Coagulation can be subdivided into three series of events. Antithrombotic: Tissue factor (TF) binds to activated factor VII (VIIa) in plasma. The TF and VIIa together activate factors IX and X. Activated factor X (Xa) converts a small amount of prothrombin to thrombin. Antithrombin: Thrombin in present in the process is neutralized. More factor IX is activated along with factor VIII. Together IX and VIII trigger factor X. The thrombin also activates the platelets. Propagation: Activated platelets express factor X and factor V on their membranes. The combination of factors Xa and Va with calcium and platelet phospholipid, produces a burst of thrombin. Thrombin converts fibrinogen to fibrin on the platelet surface and the clot. The clot consists of fibrin, platelets, and trapped blood. Factor XIII stabilizes the clot (Casey, 2011, p. 13).

Significance of Pathophysiology

Patients having procedures in ambulatory surgical centers should not be overlooked regarding assessment and education. General anesthesia has been shown to contribute to venous stasis and therefore increase the risk of developing a DVT. General anesthesia affects factor the platelets will begin to stick to one another and form a plug. Additionally, receptors on the platelets for thrombin, fibrinogen and clotting factors will begin the clotting cascade (Casey, 2011, p. 12). Coagulation can be subdivided into three series of events. Antithrombotic: Tissue factor (TF) binds to activated factor VII (VIIa) in plasma. The TF and VIIa together activate factors IX and X. Activated factor X (Xa) converts a small amount of prothrombin to thrombin. Antithrombin: Thrombin in present in the process is neutralized. More factor IX is activated along with factor VIII. Together IX and VIII trigger factor X. The thrombin also activates the platelets. Propagation: Activated platelets express factor X and factor V on their membranes. The combination of factors Xa and Va with calcium and platelet phospholipid, produces a burst of thrombin. Thrombin converts fibrinogen to fibrin on the platelet surface and the clot. The clot consists of fibrin, platelets, and trapped blood. Factor XIII stabilizes the clot (Casey, 2011, p. 13).

Signs and Symptoms

A DVT may have no symptoms. Common symptoms include the following:

- Edema
- A hard cord-like area
- A feeling of fullness
- Warmth
- Tender or pain

References

Nurses must understand the increasing options for anticoagulation therapy. Personal understanding of therapy will allow the nurse to educate patients about

- The importance of follow up care
- Bleeding precautions and warning signs
- Possible dietary considerations
- Necessary blood tests
- Therapy complications of the use of PE
- Assessing and reassessing each patient’s risk factors
- Anticoagulation therapy varied by healthcare setting
- Managing the use of compression stockings or mechanical devices

Conclusion

Deep vein thrombosis is a common diagnosis in many patient populations and has serious medical consequences if not treated. Medical professionals must be aware of the pathophysiological process behind DVT in order to effectively prevent, diagnose, and treat. Patient education is a priority especially in high risk individuals. Anticoagulation therapy remains the primary treatment, but still requires monitoring and follow-up. Knowledge of the pathophysiology of a clot will aid in the understanding of treatment options.

Diagnosis

Human’s signs has been shown to be both specific and nonspecific in screening and thus no clinical value in screening for DVT (Anthony, 2013, p. 98). An alternative to the use of Human’s sign, a predictive model for DVT development should be included in the nursing assessment and taught to nursing students. The Wells model is a predictive model that includes nine elements based on the patient’s health history and examination. Points are assigned to each element and a total score is then assigned that indicates risk of the individual for developing a DVT (Anthony, 2013). D-dimer is a serum test that tests for plasmin and thrombin production. D-dimer can be tested by an enzyme linked immunosorbent assay (ELISA) assay; which has 95% sensitivity for the screening of DVT and PE; however, the specificity is approximately 50%. Ultrasonography as a non-invasive imaging study to diagnose DVT. The inability to compress the vein with the ultrasound probe is a highly sensitive and 95% specific for proximal vein thrombosis (D’Alexander, 2016, p. 33). Effective diagnostic of DVT is accomplished by combining the following tools:

- Predictive model
- D-dimer
- Compression ultrasound

Treatment

Once a patient is diagnosed with a DVT, anticoagulation should be started immediately (D’Alexander, 2014). It is strongly recommended that treatment continues until the DVT is no longer present. Anticoagulation can not break up the existing clot; however, the medications prevent the growth of the clot. Different types of anticoagulants work at different levels of the coagulation cascade. Options for anticoagulation include:

- Heparins
- Warfarins
- Direct thrombin inhibitory factors

Figure 2. Lower Extremity DVT (Wordpress, 2014).

Figure 2. Deep vein thrombosis (Wordpress.com 2014).