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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 suffering with cancer, vascular disorders, or trauma. Situations in a healthy individuals life can also put them at risk including pregnancy, surgery, and medications (Bruni-Fitzgerald, 2015). In the United States 200,000-400,000 people develop DVT each year. The most serious complication of DVT is pulmonary embolism (PE), which occurs in up to 50% of cases and has a mortality rate of up to 30% (Anthony, 2013, p. 95). Medical costs for VTE in the US have been estimated to total $5–10 billion per year (Grosse, Nelson, Nyarko, Richardson, & Raskob, 2015, p. 4). In about 70% of patients with a PE, a DVT can be found in the lower extremity (Bruni-Fitzgerald, 2015, p. 95). The idea that DVT patients could be encountered in any practice makes it a concept that should be thoroughly understood by all medical professionals.

Risk Factors

The risk factors associated with DVT development are referred to as Virchow's triad. The triad includes the following:

- Venous stasis
- Injury to the vascular endothelium
- Hypercoagulability

Acquired hypercoagulability is associated the following situations:

- Pregnancy or postpartum
- Oral contraceptives or hormone therapy
- Cancer or cancer treatment
- Recent trauma

Inherited hypercoagulability is associated with the following:

- Sickle cell anemia
- May-Thurner syndrome
- Factor V Leiden

Pathophysiology

1. The pathophysiologic 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 allows platelet activation.
3. Platelets (thrombocytes) contain chemical mediators. Once activated by contact with collagen or von Willebrand factor the platelets change from a smooth disc to a spiny sphere. The activated platelets also release additional chemicals that will enhance smooth muscle contraction and activate neighboring platelets. 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.
   Initiation: 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.
   Amplification: Once thrombin is present the process is intensified. 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 XI 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 now consists of fibrin, platelets, and trapped blood. Factor XIIIa stabilizes the clot (Casey, 2011, p. 13).
4. Clot retraction occurs shortly after the clot is formed. The actin and myosin in the platelets that are trapped within the clot begin to contract. The fibrin strands of the clot are pulled toward the platelets. This action squeezes the serum from the clot causing it to shrink (D'Alesandro, 2016, p. 31)
5. Clot dissolution occurs when tissue plasminogen activator (tPA) is released from the healing endothelium and in response to the presence of thrombin. Tissue plasminogen activator converts plasminogen to plasmin, an enzyme that breaks down fibrin (Casey, 2011, p. 13).

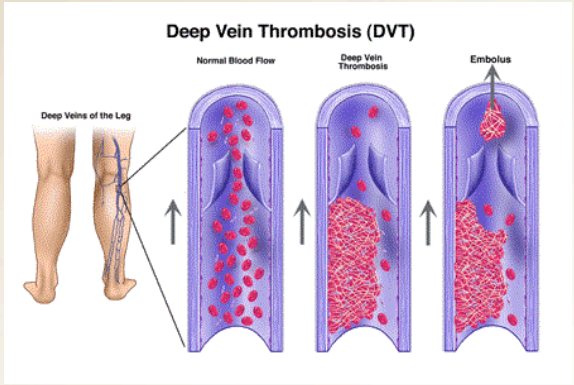

Figure 1. Deep vein thrombosis (WordPress 2014).

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 the clotting cascade by creating vasodilatation effects comparable to 10 to 14 days of inactivity. The ambulatory surgical patient is also subject to the traditional insults of "intraoperative immobility, tissue trauma, and surgical positioning" (Razzano, 2015, p. 568).
An organized DVT may become a pulmonary embolus (PE) if it breaks free from the vessel wall and travels to the pulmonary artery or its branches. Prevention of a DVT is an important step in preventing a life threatening PE (D'Alesandro, 2016, p. 31)
Cancer patients have an increased risk of DVT due to an increase in TF and cancer procoagulant. Additionally, the disease state and some treatments favor conditions for venous stasis. Deep vein thrombosis and pulmonary embolisms (PE) are the most common thrombotic events among cancer patients and most occur within the first three months of diagnosis (Kyriazi & Theodoulou, 2013).

Signs and Symptoms

A DVT may have no symptoms.
Classic symptoms include the following

- Edema
- A hardened cord-like area
- A feeling of fullness
- Warmth
- Tenderness or pain
- Redness

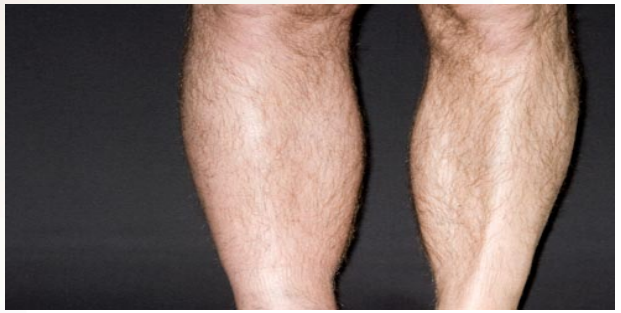

Figure 2. Lower Extremity DVT (WordPress 2014).

Diagnosis

Homan's sign has been shown to be nonspecific and nonsensitive in screening and thus of no clinical value in screening for DVT (Anthony, 2013, p. 98). An alternative to the use of Homan'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 enzyme linked immunosorbent fluorescent (ELISA) assay, which has 95% sensitivity for the screening of DVT and PE; however, the specificity is approximately 50%. Ultrasonography is a non-invasive imaging study used to diagnose DVT. The inability to compress the vein with the ultrasound probe is >95% sensitive and >95% specific for proximal vein thrombosis (D'Alesandro, 2016, p. 33).
Effective diagnosis of DVT is accomplished by combining the following tools:

- Predictive model score
- History and physical examination
- D-dimer
- Compression ultrasound

Treatment

Once a patient is diagnosed with a DVT, anticoagulation should be started immediately (D'Alesandro, 2016). It is strongly recommended that treatment continue for at least three months (Kearon et al., 2016). Anticoagulants do 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
- Warfarin
- Direct thrombin inhibitors
- Indirect factor Xa inhibitor

Nursing Considerations

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

The nurse can reduce the risk of PE by

- Assessing and reassessing each patient's risk factors
- Educating patients on the importance of ambulation or activity
- 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 pathologic process behind DVT in order to effectively prevent, diagnose, and treat. Patient education is a priority especially in high risk individuals. Anticoagulation therapy continues to advance, but still requires monitoring and follow up care.
Knowledge of the pathophysiology of a clot will aid in the understanding of treatment options.

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