

Otterbein University

Digital Commons @ Otterbein

Nursing Student Class Projects (Formerly MSN)

Student Research & Creative Work

Summer 2015

Deep Venous Thrombosis in Surgical Orthopedic Patients

Kelly Marsh

Otterbein University, kelly.marsh@otterbein.edu

Follow this and additional works at: https://digitalcommons.otterbein.edu/stu_msn



Part of the [Cardiovascular Diseases Commons](#), [Medical Pathology Commons](#), and the [Nursing Commons](#)

Recommended Citation

Marsh, Kelly, "Deep Venous Thrombosis in Surgical Orthopedic Patients" (2015). *Nursing Student Class Projects (Formerly MSN)*. 89.

https://digitalcommons.otterbein.edu/stu_msn/89

This Project is brought to you for free and open access by the Student Research & Creative Work at Digital Commons @ Otterbein. It has been accepted for inclusion in Nursing Student Class Projects (Formerly MSN) by an authorized administrator of Digital Commons @ Otterbein. For more information, please contact digitalcommons07@otterbein.edu.

Deep Venous Thrombosis in Surgical Orthopedic Patients

Kelly Marsh, RN

Otterbein University, Westerville, Ohio

Introduction to DVT

- The term venous thromboembolism (VTE) is defined as a syndrome that collectively comprises both deep vein thrombosis (DVT) and pulmonary embolism (PE). Each year approximately 350,000-900,000 people in the United States develop VTE and approximately 100,000 people die according to this cause (Streiff et al., 2014). Unfortunately, the reoccurrence rate for VTE remains very high, with an estimated risk of 10%-30% of VTE patients will develop another VTE within five years of the first incident (Streiff et al., 2014).
- Deep vein thrombosis (DVT) is blood clot that forms in a large vein, usually in areas of slow blood flow such as the leg or pelvis. Clots can form in the deep veins (DVT) or superficial veins (called superficial thrombophlebitis or phlebitis). Superficial blood clots rarely cause serious problems.
- A blood clot can detach or embolize, becoming mobile in the bloodstream. This embolism can travel to other places most lethally the heart (resulting in a myocardial infarction), the brain (resulting in a stroke) or the lungs (causing a pulmonary embolism).
- A DVT however, does NOT cause heart attack or stroke. Due to the anatomy of blood flow throughout the body, a DVT can only cause a PE. A blood clot in the artery, termed an arterial thrombosis, can embolize to cause a heart attack or stroke (Centers for Disease Control and Prevention [CDC], 2014).
- DVT causes significant morbidity and mortality. The most common complication associated with DVT is post-thrombotic syndrome (PTS) which occurs in approximately one-third of patients that experience DVT (Bonner & Johnson, 2013). PTS can cause symptoms of chronic pain, heaviness, and swelling to the affected extremity that can result in development of venous leg ulcers in severe cases (Bonner & Johnson, 2014). PTS can significantly affect a person quality of life and lead to serious disability.
- While other risk factors exist, the incidence rate of DVT is known to increase in association with surgical procedures particularly orthopedic surgery. Without prophylaxis, the incidence rate of documented DVT in the orthopedic surgical patients is reported to range from 50%-60% (Snyder, 2008). As part of ongoing efforts to increase the efficiency and overall cost-effectiveness of hospital operations, the Centers for Medicare and Medicaid Services (CMS) and the Joint Commission adopted standardized performance measures for hospitals. Among other performance measures, VTE related total hip replacement and total knee replacement surgeries were added to the "never event" list defined by CMS as "preventable medical errors that result in serious consequences for the patient" (Baser, Supina, Sengupta, Wang, & Kwong, 2010). CMS no longer reimburses hospitals for VTE associated with total hip and knee replacement surgeries" (Baser, Supina, Sengupta, Wang, & Kwong, 2010).
- Although guidelines exist for the prevention and management of DVT, their implementation remains problematic and VTE incident rates within the United States remains high. Several prospective analyses have shown that VTE guidelines are not being routinely followed and this gap between guidelines and actual medical practice can partly be explained by physician knowledge, attitudes or beliefs (Baser, Supina, Sengupta, Wang, & Kwong, 2010).
- Research within this topic was performed in anticipation to gain a deeper understanding of DVT along with the implications for nursing care. Inquiry and discussion within this area serves to bring to light the importance of researching the most current and relevant evidenced based practice guidelines regarding DVT risk assessment, signs and symptoms, pathophysiology and the significance of DVT pathophysiology in orthopedic patients.

Presentation of Case

The patient is a 60 year old female in an inpatient rehab facility for intensive physical therapy following a right total knee replacement that was performed one week ago. She complains of right lower extremity pain that is worse upon ambulation. The patient denies any chest pain or shortness of breath. Vitals are stable, she is afebrile. The patient has a past medical history of hypertension, congestive heart failure (CHF), obesity and smoking. Upon physical examination the patient's right calf is noted to be erythematous and swollen twice the size of the unaffected extremity. 2+ pitting edema is noted to the right lower extremity (RLE) extending into the patients thigh. The patient does not report increased pain upon dorsiflexion of the right foot indicating a negative Homans sign. A right pedal pulse is palpable but slightly weak (2+). Localized tenderness is noted to the right calf experienced upon light palpation. The total knee replacement incision is healing, well approximated, staples intact without any drainage, redness or other signs or symptoms of infection noted. Given this patient's recent surgical procedure, history of CHF and with the presented signs and symptoms, a DVT to the RLE is highly suspected. A RLE venous ultrasonography is ordered.

Signs and Symptoms

The classic DVT signs and symptoms include calf or thigh pain, unilateral extremity edema or enlargement, and calf tenderness with foot dorsiflexion (Homans sign), although this is neither sensitive nor specific (Meguid, 2011). The pain may be described as varying from a dull ache to sharp pain (Meguid, 2011). The pain is most likely attributable to pressure on adjacent nerves and due to the inflammatory process initiated by the coagulation cascade (Meguid, 2011). Increased venous pressure can cause symptoms of tachycardia, low grade fever, increased skin turgor, and distention of the superficial venous collaterals (Meguid, 2011). Approximately one-third of DVT cases occur in the proximal leg beginning at the femoral vein and progressing proximally to the iliac vein (Meguid, 2011). With a DVT in this location, causing occlusion of the femoral and iliac veins; the skin will become cyanotic or pale as a result of severe venous obstruction, cool to the touch with massive swelling and restriction of arterial blood flow (Meguid, 2011).

Figure 1. (Remedica Journal, 2013)

While it is highly important to assess for DVT in susceptible patients, up to 50% of patients will have no associated clinical findings in the extremities of venographically proven DVT (Meguid, 2011).

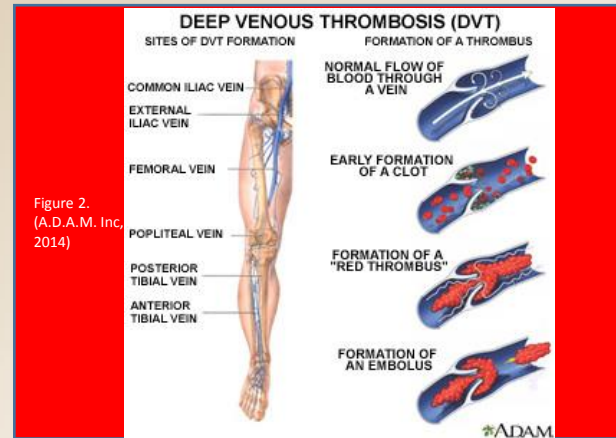


Figure 2. (A.D.A.M. Inc, 2014)

Underlying Pathophysiology

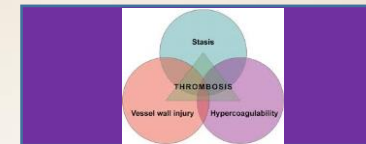
In 1856, Rudolf Virchow postulated that hypercoagulability of blood, stasis of blood, and intimal vessel wall injury are casually related to thrombus formation (Meeto, 2010). This postulation still stands today and is now referred to as Virchow's triad. The hemostatic system is faced with the complex task of not only maintaining adequate blood circulation to sustain life but also must simultaneously be able to convert blood to an insoluble gel to assist in repair at site of vascular injury (Meeto, 2010). Thus, direct damage from a multitude of causes is one of the extrinsic factors that causes a trigger of the clotting cascade leading to the possible development of a DVT.

Hypercoagulability, or thrombophilia, is condition which directly places an individual at risk for a blood clot and is associated with a number of primary (hereditary) and secondary (acquired) causes (McCance, Huether, Brashers & Rote, 2014, p. 1048). Primary causes are attributable to defects in proteins involved in hemostasis and secondary causes include a variety of clinical disorders or conditions (McCance, Huether, Brashers & Rote, 2014, p. 1048). Some disorders associated with a high risk for venous thrombosis or thromboembolism include orthopedic surgery, malignancy, congestive heart failure, varicose veins, estrogens and sepsis (McCance, Huether, Brashers & Rote, 2014, p. 1048). On the contrary, some disorders associated with a high risk for arterial thrombosis or thromboembolism include diabetes mellitus, atherosclerosis, smoking, hypertension and polycythemia (McCance, Huether, Brashers & Rote, 2014, p. 1048).

Abnormal clots that adhere to the vessel wall are known as thrombi and are composed of blood cells, platelets and fibrin. Arterial thrombi are mostly composed of platelet aggregates and fibrin, while venous thrombi are mainly composed of red blood cells (Snyder, 2008). The difference in the clot composition is attributed to the difference in blood flow conditions in which the clot is formed. Arterial thrombi form under conditions of high blood flow and are mainly composed of platelet aggregates and fibrin (Snyder, 2008). In sites of turbulent blood flow in the arteries the multitude of platelets and endothelial cells may be elevated leading to thrombosis (McCance, Huether, Brashers & Rote, 2014, p. 1048). In venous blood flow, condition of low flow cause venous stasis which allows platelets to stay in contact with the endothelium for prolonged times and clotting factors that are normally diluted with an increase in blood flow are not diluted which as a result contribute to clot formation (McCance, Huether, Brashers & Rote, 2014, p. 1048).

If a thrombus detaches from the endothelial wall it becomes an embolus. This mobile clot travels through the circulation until it lodges in a blood vessel, blocking blood flow and depriving associated tissues or organs of oxygen and nutrition. A major sequelae occurring in 20% of DVT patients is the development of a pulmonary embolism (PE) caused by the dislodgment of the thrombus (Meguid, 2011). PE's have a high potential to become a fatal event, with ten percent of fatalities occurring within only the first hour from the onset of symptoms (Meguid, 2011).

Figure 3. Virchow's Triad. (Kyrle & Eichinger, 2010).



Significance of Pathophysiology

DVT is associated with significant morbidity and mortality and therefore imposes a considerable economic burden within the United States (LaMori, Shoheiber, Mody, & Bookhart, 2014). The pathogenesis associated with DVT plays a critical role in the increased risk of development following total hip or knee replacement surgeries. During orthopedic surgeries, substantial trauma to the soft tissues or bone is inevitable and it is this injury that plays a key role in the activation of the coagulation cascade (Fisher, 2011). Mechanical destruction of the bone marrow during surgery can also release marrow cells and bone fragments into circulation (Fisher, 2011). Cessation or a decrease of blood flow not only occurs with manipulation of the limb during surgery, but there is also evidence of decreased blood flow following surgery (Fisher, 2011). Venous blood flow is temporarily reduced for up to 6 days following a total knee replacement and for up to 6 weeks following a total hip replacement (Fisher, 2011). In addition, postoperative immobilization and swelling after surgery further contribute to reduced venous return and thus an increased risk of DVT.

Implications for Nursing Care

It remains critical that all patient care staff understand that patients undergoing orthopedic surgeries are at a high risk for DVT or PE and be vigilant in assessing for DVT. Assuring that these patients are placed on DVT prophylactic medication as prescribed by the physician is crucial in DVT prevention. "Health care systems have the potential to reduce the clinical and economic burden of VTE by ensuring that evidenced-based, guideline recommended anticoagulation therapy is adhered to by patients with an initial VTE" (LaMori, Shoheiber, Mody, & Bookhart, 2014). There are numerous measures that nursing staff can utilize to prevent DVT in this extremely high risk group. Prevention of venous stasis can include interventions that encourage early ambulation or the application of compression hose or intermittent pneumatic compression boots. Hydration maintenance by encouraging oral liquids or assessment for the need of I.V. fluid therapy will assist in decreasing states that may lead to hypercoagulability. Patient education is of utmost importance for the prevention and early recognition of DVT.

Conclusion

It is now becoming increasingly recognized that VTE can have a far-reaching impact on patients, physicians, and other health care providers including hospitals and nursing facilities. VTE is a common complication following orthopedic surgery. An understanding of the signs and symptoms of DVT along with the underlying pathophysiology provide health care employees the necessary knowledge needed to recognize this preventable health care problem. Prevention of DVT in this patient population requires a multimodal approach with emphasis upon DVT prophylaxis as a standard of care for all patient undergoing total hip or knee replacements. Although guidelines for the prevention and treatment of DVT exist, their implementation remains problematic in hospitals across the United States (Baser, Supina, Sengupta, Wang, & Kwong, 2010). Currently, hospital stays for DVT and PE represent a substantial cost burden to the U.S. health care system (LaMori, Shoheiber, Mody, & Bookhart, 2014). Direct patient care staff including nurses and physicians alike play a key role in the primary prevention of DVT.

References

- Anderson, D. R., Dunbar, M. J., Bohm, E. R., Belzile, E., Kahn, S. R., Zukor, D., & ... Vendittoli, P. (2013). Aspirin versus low-molecular-weight heparin for extended venous thromboembolism prophylaxis after total hip arthroplasty: A randomized trial. *Annals Of Internal Medicine*, 158(11), 800-806. doi:10.7326/0003-4819-158-11-201306040-00004.
- Baser, O., Supina, D., Sengupta, N., Wang, L., & Kwong, L. (2010). Clinical reports: venous thromboembolism. Impact of postoperative venous thromboembolism on medicare recipients undergoing total hip replacement or total knee replacement surgery. *American Journal Of Health-System Pharmacy*, 67(17), 1438-1445. doi:10.2146/ajhp090572.

References Continued

- Bonner, L., & Johnson, J. (2014). Deep vein thrombosis: diagnosis and treatment. *Nursing Standard*, 28(21), 51-58. doi:10.7748/ns2014.01.28.21.51.e8222.
- Centers for Disease Control and Prevention. (2014, December 10th). Deep vein thrombosis (DVT)/pulmonary embolism (PE)-blood clot forming in a vein. Retrieved from: <http://www.cdc.gov/ncbddd/dvt/facts.html>.
- Fisher, W. (2011). Impact of venous thromboembolism on clinical management and therapy after hip and knee arthroplasty. *Canadian Journal Of Surgery*, 54(5), 344-351. doi:10.1503/cjs.007310.
- Kachroo, S., Boyd, D., Bookhart, B. K., Lamori, J., Schein, J. R., Rosenberg, D. J., & Reynolds, M. W. (2012). Quality of life and economic costs associated with postthrombotic syndrome. *American Journal Of Health-System Pharmacy*, 69(7), 567-572. doi:10.2146/ajhp110
- Kyrle, P., & Eichinger, S. (2010). Virchow's Triad. Retrieved from: www.bloodjournal.org
- LaMori, J. C., Shoheiber, O., Mody, S. H., & Bookhart, B. K. (2015). Inpatient resource use and cost burden of deep vein thrombosis and pulmonary embolism in the united states. *Clinical Therapeutics*, 37(1), 62-70. doi:10.1016/j.clinthera.2014.10.024
- McCance, K.L., Huether, S.E., Brashers, V.L., & Rote, N.S. (2014). *Pathophysiology the biologic basis for disease in adults and children (7th ed.)*. St. Louis, MI: Elsevier Mosby.
- Meeto, D. (2010). In too deep: understanding, detecting and managing DVT. *British Journal Of Nursing*, 19(16), 1021.
- Meeto, D. (2013). Understanding and managing deep vein thrombosis. *Nurse Prescribing*, 11(8), 390-395.
- Meguid, C. (2011). Best practice for deep vein thrombosis prophylaxis. *Journal For Nurse Practitioners*, 7(7), 582-587. doi:10.1016/j.nurpra.2011.04.002
- Snyder, B. (2008). Venous thromboembolic prophylaxis: the use of aspirin. *Orthopaedic Nursing*, 27(4), 225-232.
- Streiff, M. B., Brady, J. P., Grant, A. M., Grosse, S. D., Wong, B., & Popovic, T. (2014). CDC grand rounds: Preventing hospital-associated venous thromboembolism. *MMWR: Morbidity & Mortality Weekly Report*, 63(9), 190-193.



OTTERBEIN UNIVERSITY

Table 2. Risk of objectively confirmed asymptomatic and symptomatic DVT and PE in major orthopedic surgery [1].

Surgery	DVT risk (%)	PE risk (%)
Hip arthroplasty	42–57	0.9–28
Knee arthroplasty	41–85	1.5–10
Hip fracture surgery	46–60	3–11

DVT: deep venous thrombosis; PE: pulmonary embolism.

Image courtesy of Remedica Journals
<http://www.remedicajournals.com/Advances-in-Venous-Arterial-Thrombolism/BrowseIssue.asp?Volume=11/Issue=2/Article=Venous-Thromboembolic-Risk-and-the-Current-Strategies>