Deep Vein Thrombosis Prophylaxis in the Neurosurgical Patient

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KEYWORDS

• DVT • PE • DVT prophylaxis

KEY POINTS

- The optimal approach for deep vein thrombosis (DVT) prophylaxis in neurosurgical patients continues to be a challenge of balancing the reduction in DVT and pulmonary embolus (PE) without risking an increase in catastrophic hemorrhages.
- All patients should have mechanical prophylaxis before surgery and continuing after surgery.
- Use of pharmacologic prophylaxis seems to result in an increased rate of hemorrhage in neurosurgery, but weight of the evidence suggests that the addition of pharmacologic prophylaxis reduces the rate of DVT and PE without incurring a high risk of intracerebral hemorrhage once hemostasis is secured and confirmed within 24 to 48 hours after surgery.

CAUSE

Deep vein thrombosis (DVT) is the result of a complex interplay of multiple inherited or acquired factors. The interplay of these factors is summarized using the "Virchow's triad," which includes disturbances in blood flow patterns, blood clotting factors promoting coagulation, and vessel wall endothelial injury. These elements combine to disrupt the balance of coagulation and fibrinolysis mechanisms leading to venous clot formation and propagation.[1] Specifically, the intact endothelium of blood vessel plays an important role in maintaining hemostasis and preventing the formation of clots. Injury to the endothelium of blood vessels, for example, during surgery or with insertion of central venous catheters, exposes subendothelial tissue factor and reduces venous flow, which activates the clotting cascade resulting in thrombus formation.[2]

DVT can form without evidence of injury to the endothelium[3] when stasis and flow turbulence occur in the valve pockets of deep veins.[3–5] These changes lead to local hypoxia that promotes thrombosis by inhibiting expression of anticoagulants and stimulating expression of prothrombotic substances.[6] Acquired factors that promote stasis, such as prolonged immobility, venous valve dysfunction with aging, or increased venous pressure due to heart failure, increase the risk of DVT formation. Similarly, inherited or acquired factors that increase the level/activity of procoagulant or decrease the level/activity of anticoagulants also increase the risk of DVT formation.[2,4–6]

EPIDEMIOLOGY

General Population

Overall estimates for incidence of DVT in the general population range from 45 to 117 per 100,000 persons. DVT formation rates are higher for men (130 per 100,000 persons) than women (110 per 100,000) in all age groups except during the

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childbearing years (when women are at higher risk). Incidence per 100,000 is highest among African Americans (141) compared with Caucasians (104), Hispanics (55), whereas Asian/Pacific Islanders (21) have the lowest incidence.[7] Age is associated with increased risk of DVT (related to venous valve dysfunction that occurs with aging) development in both men and women.[8] The reported incidence of DVTs has increased with the rising use of routine diagnostic imaging.[8,9]

Neurosurgical Patients

The rate of DVT formation in neurosurgical patients without prophylaxis varies between 0% and 34%.[10–15] Neurosurgical patients receiving at least one form of DVT prophylaxis who underwent screening (asymptomatic) with Doppler ultrasound had a 3% to 16% incidence of DVTs.[16–18] Reported rates of symptomatic DVTs from pooled studies and national databases range between 1% and 4%.[19–22] Differences in reported DVT formation in neurosurgical patients are primarily due to the use and method of screening to detect DVT. Higher-intensity screening has led to higher estimates of DVTs (eg, routine screening of asymptomatic patients). Studies reporting higher rates of DVT used routine screening with high sensitivity tests, such as radioactive fibrinogen uptake, compared with the use of Doppler ultrasound screening of symptomatic patients.[14,23,24]

The risk of DVT formation is associated with neurosurgical procedure location, surgical features, and patient characteristics. Rates are higher for patients undergoing craniotomy compared with spine surgery. Analysis of the National Surgical Quality Improvement Program database (2006–2011) revealed DVT formation was 3.4% after cranial and 1.1% after spinal surgeries.[19,25] Estimates of the incidence of symptomatic DVT from regional hospital discharges reveal a rate of 3.9% for craniotomy patients[26] and a rate of 0.5% to 2% over the 90 days after discharge for the spinal surgery patient.[27] Specifically, among patients undergoing cranial procedures for tumor, the rate of DVT formation was 2% to 10%.[26,28,29] DVT formation in subarachnoid hemorrhage patients ranges between 3.5% and 18%.[20,30,31] The rate of DVT after deep brain stimulation surgery has been reported at 1%.[32] Risk of DVT after surgery for spine trauma, deformity, and degenerative spine surgery is between 0% and 19%, 2% and 14%, and 0% and 9%.[33,34] Patients undergoing a cranial procedure are considered a high- to very high-risk population, whereas patients undergoing spine procedure are considerate a low- to moderate-risk population by the American College of Chest Physicians (ACCP).[35]

Risk factors associated with DVT formation in the neurosurgical patients also include the presence of malignancy, prior episode of DVT and pulmonary embolus (PE), type of surgery (cranial, spinal, or cerebrovascular), duration of surgery, oral contraceptive use, stroke, sepsis, heart failure, radiation therapy, paraparesis, altered mental status, heart failure, smoking, obesity, presence of deep venous catheters, age, and inherited hypercoagulable disorders.[16,19,25,35,36] Risk factors specific for patients undergoing spine surgery include combined anterior-posterior approaches, multilevel surgeries, surgery for trauma, and surgery for deformity correction.[33,35]

PULMONARY EMBOLUS IN THE NEUROSURGICAL PATIENT

PE can result after a DVT travels to the lung vasculature resulting in incomplete or complete blockage of the pulmonary artery (**Fig. 1**). PE is associated with a high rate of morbidity and mortality in the neurosurgical population. Signs and symptoms of PE include tachycardia, pleuritic chest pain, shortness of breath, hemoptysis, and tachypnea. The most severe cases of PE present with sudden cardiopulmonary arrest. Approximately 25% of patients who develop a PE die suddenly. Patients (75% of all PE patients) who survive the acute impact of PE have a 7-day survival rate of 70%.[16] A comprehensive review of the literature estimated the overall rate of PE in neurosurgical patients (patient receiving or not

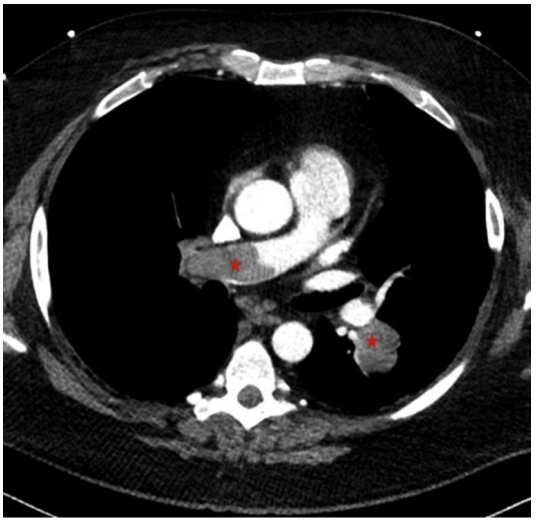

Fig. 1. Computer tomography angiography shows bilateral acute PE (*red star*) in a patient who underwent craniotomy for cranial metastasis removal a few weeks earlier. This patient was also found to have bilateral DVTs in Doppler ultrasound.