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Commentary

Pathogenesis, Prognosis and Management of Deep Vein Thrombosis

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DESCRIPTION

The development of a blood clot (partial or whole blockage) in venous or arterial blood arteries, which restricts the blood's normal flow and causes clinical sequelae, is referred to as thrombosis. Blood cells (including platelets), plasma proteins, coagulation factors, inflammatory factors and cytokines, and the endothelial lining of arteries and veins all work together to maintain a complex balance that permits blood to flow freely in vessels. A thrombosis rather than a coagulopathy may be more likely to develop when there is an imbalance with this physiological function (increased risk of bleeding). Patients may experience elevated thrombosis and concomitant bleeding risk under specific clinical conditions (e.g., disseminated intravascular Coagulopathy-DIC, or in patients with underlying malignancy who develop a coagulopathy). The diagnosis and treatment of thrombosis are therefore complicated. Any organ system might experience them, and depending on underlying comorbidities and the presence (or absence) of triggering stimuli, their clinical presentation can vary. Management choices can be influenced by a variety of circumstances, such as whether the condition is venous or arterial, acute or chronic, the first or subsequent episode, family history, risk factor assessment, and hemodynamic stability.

Pathogenesis

Venous stasis, vascular damage, and hypercoagulability are the three variables that Virchow's Triad, which was initially introduced in 1856, suggests as contributing to the development of thrombosis. The most important of the three components is venous stasis; however stasis by itself does not seem to be sufficient to result in thrombus development. However, there is a significant increase in the likelihood of clot formation when venous stasis, vascular damage, or hypercoagulability are present together. These include surgery or trauma, cancer, extended immobility, pregnancy, congestive heart failure, varicose veins, obesity, advanced age, and a history of Deep Vein Thrombosis (DVT). The clinical factors most closely associated with DVT are essentially linked to the components of Virchow's Triad.

The pockets next to valves in the deep veins of the leg are one example of a location where venous thrombosis frequently occurs because of reduced or mechanically changed blood flow. Although valves aid in promoting blood flow *via* the venous system, they can also be sites of venous stasis and hypoxia. Numerous postmortem studies have shown that the sinuses next to venous valves have a tendency to develop venous thrombi. Oxygen tension decreases as blood flow slows, and hematocrit rises concurrently. Certain antithrombotic proteins that are primarily expressed on venous valves, such as thrombomodulin and endothelial protein C receptor, may be downregulated in the resulting hypercoagulable microenvironment, Electronic Patient Care Record (EPCR). Hypoxia also inhibits key anticoagulant proteins while promoting the production of certain procoagulants. P-selectin is one of them, a tissue factor-containing adhesion protein that pulls immune cells to the endothelium. Whether tissue factor is expressed on the endothelium or by cells in the extravascular tissue is still up for debate, however it is generally accepted that tissue factor acts as the main nidus for thrombus development. It seems those P-selectin and tissue factors are both necessary for thrombus development.

Prognosis

The prognosis for thrombosis depends on the kind of thrombosis (venous or arterial), the location of the thrombosis, its severity and length, the persistence of risk factors and concomitant conditions, and whether it is the first or a subsequent or recurrent episode. Patients with venous thromboembolism are more likely than the general population to experience recurrent Venous Thromboembolism (VTE), particularly if they also have inciting risk factors at play. Within 10 years, recurrent venous thrombosis may occur in up to 25%of people who have had VTE. Patients who have several aggravating risk factors or underlying verified genetic or acquired thrombophilia are more likely to experience a recurrence. In the European Union, venous thromboembolism is thought to be the cause of 300000 fatalities annually. In the United States, cardiovascular disease continues to be the largest cause of death. In patients with cerebrovascular disease, mortality can reach 20% within the first 30 days of an ischemic Cerebrovascular Accident (CVA), and up to 30% of survivors will experience persistent disability. The prevention of venous and arterial

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thrombosis is essential, and patients should be made aware of lifestyle traits and modifiable risk factors.

Management

Acute Deep Vein Thrombosis (DVT) and Pulmonary Embolism (PE) are commonly treated in hospitalised patients by administering anticoagulation by IV Unfractionated Heparin (UFH) or low molecular weight heparin, followed by a switch to oral anticoagulation as described below. To stop the progression of thrombosis and lower associated mortality, rapid anticoagulation with heparin or Low-Molecular-Weight Heparins (LMWH) offers a quick therapeutic range. Once patients reach a therapeutic INR, vitamin K antagonists (such as warfarin) can be added for chronic and protracted therapy after starting these intravenous anticoagulants. The typical suggested period for an initial, induced thrombosis is three months, particularly for simple distal DVT. Consider at least 3-6 months of duration for patients who have numerous persistent provoking risk factors, major pulmonary embolism coupled with hemodynamic compromise, or the first incident of induced thrombosis with significant clot burden (or longer in select cases). Recurrent thrombosis risk can be increased by stopping medication before definite standards on duration have been established. To reduce the risk of recurrence, medication compliance and adherence are crucial. Determining strong inciting or temporary risk factors is crucial for directing treatment in individuals with recurrent DVT or PE since it complicates the length of therapy. Anticoagulation medication should normally be continued for 6 months or more in patients with active cancer as a provocation risk factor for thrombosis. In people with thrombosis and cancer, LMWH has traditionally been favoured. The use of specific direct oral anticoagulants (such as edoxaban) for therapy in patients with cancer is now included in current guidelines, but there are still some exclusions. Anticoagulation with thrombosis in pregnancy, perioperatively in patients with thrombosis (especially orthopaedic surgery), thrombosis in patients with hereditary or acquired thrombophilia, and management of patients with recurrent or "breakthrough" thrombosis (despite therapeutic anticoagulation) all become significantly more difficult. Under the direction of a haematologist consultant, these patients are best treated.