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Perspective

Role of Anticoagulants in the Treatment of Pulmonary Embolism

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DESCRIPTION

The process that causes a blood vessel to stop bleeding is called hemostasis. It is a process with several interrelated steps. A "plug" is formed at the end of this chain of events, sealing off the bleeding-controlling blood vessel's injured area. The lining of the blood vessel is first damaged.

Blood clotting stages

There are four steps to the hemostasis mechanism. Blood vessel constriction is number one. 2) The temporary "platelet plug" that forms. 3) The activation of the coagulation cascade. 4) The last clot, or "fibrin plug," forms.

Injury: Injury or blood vessel damage triggers the beginning of the blood clotting process. This may take the shape of a tiny tear in the blood vessel wall, which could result in bleeding.

Vascular constriction: To stop blood loss, the body will tighten the blood vessel. The blood supply to the injured area will be constrained.

Platelet plug: The body stimulates platelets in reaction to damage. Small sacs in the platelets produce chemical signals at the same time to entice other cells to the region. They combine together to form a platelet plug. The von Willebrand factor (VWF), a protein, aids in the platelets' ability to adhere to one another.

Fibrin clot: When a blood vessel is injured, the blood's clotting or coagulation factors become active. The clotting factor proteins promote the synthesis of fibrin, a robust and strand-like material that helps to create a fibrin clot. This fibrin clot thickens for a few days or weeks before dissolving as the damaged blood artery walls shut and recover.

A vital mechanism called blood clotting can stop blood loss from injuries. Any irregularity in the process has the potential to cause harmful side effects, like significant blood loss. To avoid bleeding and injury, patients with coagulation issues are frequently under strict observation.

In order to restrict clotting and eliminate clots that are no longer necessary, the body employs regulatory mechanisms. Any aspect of the clotting or bleeding control system that is faulty might result in dangerously high levels of either clotting or bleeding. Even a little blood vessel lesion might result in significant blood loss when coagulation is inadequate. Small blood arteries in crucial locations may become blocked with clots when clotting is severe. Heart attacks and strokes can both be brought on by congested blood arteries in the heart and in the brain. Major arteries in the lungs can become blocked by fragments of blood clots from veins in the legs, pelvis, or abdomen (pulmonary embolism).

Platelets become active when a blood vessel wall breaks due to trauma. They transform from spherical to spiky, adhere to the wall of the damaged vessel and one another, and start to close the gap. In order to create fibrin, they also interact with other blood proteins. More platelets and blood cells are caught in a net of fibrin strands that forms a clot by plugging the break.

Drugs and blood clots

Drugs and the body's capacity to regulate bleeding (hemostasis) interact in a complex way. Hemostasis depends on the body's capacity to create blood clots, but excessive clotting raises the risk of a heart attack, stroke, or pulmonary embolism. Many medicines can interfere with the body's ability to produce blood clots, whether on purpose or accidentally.

Some patients have a high chance of developing blood clots, thus medicines are purposefully given to them to lower that risk. To prevent platelets from clumping together to obstruct a blood artery, medications that lessen platelet stickiness may be used. Aspirin, ticlopidine, clopidogrel, prasugrel, abciximab, and tirofiban are a few examples of medications that inhibit platelet activity.

An anticoagulant, a medication that blocks the activity of blood proteins known as clotting factors, may be administered to additional patients who are at risk of developing blood clots. Anticoagulants are frequently referred to as "blood thinners," yet

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they do not really thin the blood. Warfarin, taken orally and heparin, given intravenously, are two commonly used anticoagulants.

The potent proteins thrombin or activated factor X, which are required for clotting, are directly inhibited by Direct Oral Anticoagulants (DOACs). Dabigatran, apixaban, edoxaban, and rivaroxaban are a few examples of DOACs.Heparin or warfarin users must be closely monitored by a physician. Blood tests that assess the amount of time it takes for a clot to develop are used by doctors to track the effects of these medications, and the results are used to modify the dose. Too little of a dose might not stop clots, while too high of a dose might result in serious bleeding. Low-molecular-weight heparin is one class of anticoagulant medication that doesn't need as much care. DOAC users do not require routine coagulation tests in the lab.

Platelets become active when a blood vessel wall breaks due to trauma. They transform from round to spiky, adhering to the wall of the fractured vessel and to one another as they start to grow. A thrombolytic (fibrinolytic) medication can be administered to a patient who already has a blood clot to help dissolve the clot. Streptokinase and tissue plasminogen activators are examples of thrombolytic medications that are sometimes used to treat heart attacks and strokes brought on by blood clots. These medications may prevent death, but they may increase the likelihood of serious bleeding in the patient. Heparin, a medication used to lower the risk of clot formation, occasionally activates platelets paradoxically and unintentionally, increasing the risk of clotting (heparin-induced thrombocytopenia-thrombosis)

Estrogen, alone or in oral contraceptives, can have the unintended effect of causing excessive clot formation.