VENOUS THROMBOSIS

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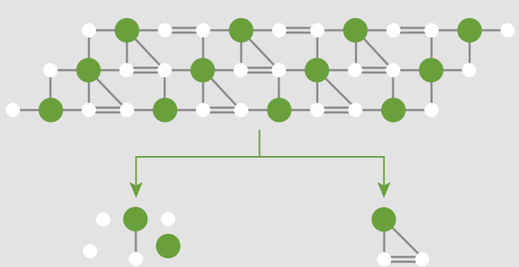
Activation of blood coagulation in veins leads to thrombosis. If the deep veins are affected, thrombosis can cause acute or chronic blood stasis that affects the local tissue. Furthermore, pulmonary embolism, which occurs when a thrombus dislodges and travels to the lung, can be life-threatening.

DIAGNOSIS

Deep-vein thrombosis is often asymptomatic and only becomes apparent when its consequences, such as pulmonary embolism and breathlessness, occur. In some cases, thrombosis in the leg veins above the knee can cause substantial blood stasis and swelling. However, leg swelling has many other possible causes; therefore, the likelihood of thrombosis — based on clinical presentation and risk factors such as immobility or hospitalization — must be assessed. Patients with a high likelihood should undergo diagnostic testing, which includes leg sonography for deep-vein thrombosis and lung CT or ventilation–perfusion scanning for embolism. Patients with a low likelihood are first screened using D-dimer measurement. As elevated D-dimer levels also occur in many other conditions, this screening primarily excludes thrombosis; a positive result still warrants diagnostic testing.

D-dimers are fibrin degradation products, which arise when a thrombus is degraded by fibrinolysis



PATHOPHYSIOLOGY

MANAGEMENT

The 'cornerstone' of treating venous thrombosis is anticoagulation. Several drugs are available to stop blood clotting by inhibiting the formation and function of coagulation factors. Traditional treatment regimens rely on parenteral anticoagulation with heparin or its derivatives, until orally administered warfarin sufficiently lowers the levels of vitamin K-dependent coagulation factors.

Newer drugs directly inhibit the enzymatic activity of coagulation factors and act much faster. Furthermore, they can be given in a fixed dose and do not need frequent monitoring, as is the case for warfarin.

QUALITY OF LIFE

Venous thrombosis primarily affects quality of life through its long-term complications. Residual thrombi can remain in the affected vessels, and inflammatory processes can lead to scarring and damage to venous valves. In the legs, these changes cause post-thrombotic syndrome, which is characterized by swelling and trophic changes that can lead to ulceration. The lungs can be similarly affected after embolism. Chronic thromboembolic pulmonary hypertension is a feared complication, as it puts the right ventricle under pressure and can cause heart failure.

OUTLOOK

Despite substantial advances in the management of venous thrombosis, more research to understand, predict and inhibit thrombogenesis is needed. Biomarkers that signal an increased thrombosis risk would be very helpful to select patients for prophylactic anticoagulation. Thromboprophylaxis requires a careful risk–benefit analysis, as all currently used anticoagulant drugs cause an increased bleeding risk.

An anticoagulant drug that effectively inhibits clotting and does not increase the risk of bleeding is the 'holy grail' of thrombosis research. This might be achievable by inhibiting specific coagulation factors.

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