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DESCRIPTION

Factor V Leiden was identified in 1994 by Rogier M. Bertina, in Leiden, Netherlands. Factor V Leiden, a hereditary mutation of the factor V gene, causes a hypercoagulability disorder. The mutation of the factor V gene results in excessive blood clotting and resistance to activated protein C degradation, which, in combination with other risk factors, can cause significant morbidity (Andreoli, 2007). Factor V Leiden is the most common hereditary disorder that predisposes individuals to thrombosis.

NEUROPATHOLOGY/PATHOPHYSIOLOGY

In a healthy individual, the factor V protein functions as a cofactor with factor X, also known as Stuart–Prower factor. Factor X is an enzyme of the coagulation process, which, along with factor V, forms the prothrombinase complex (Andreoli, 2007). The prothrombinase complex catalyzes prothrombin, an inactive zymogen, into thrombin. Thrombin cleaves fibrinogen to fibrin, which polymerizes to form the majority of the meshwork of a thrombus, or blood clot. Protein C, a major physiological anticoagulant, is activated by thrombin into activated protein C (De Stefano & Leone, 1995). Once activated, protein C regulates blood clot formation by the cleaving and degrading of factor V.

Factor V Leiden is a genetic disorder resulting from a point mutation in the gene encoding of factor V. The mutation, a missense substitution, occurs when factor V protein's amino acid arginine is switched to glutamine. This mutation results in a factor V variant that is resistant to the regulatory work of activated protein C (Bertina et al., 1994). The resistivity of factor V Leiden to activated protein C results in the inability to cleave factor V and a deficiency in the anticoagulation system.

Individuals who are homozygous for the mutated allele are at a higher percentage for risks associated with deep vein thrombosis, than for those who are heterozygous (De Stefano & Leone, 1995).

NEUROPSYCHOLOGICAL/CLINICAL PRESENTATION

The excessive clotting associated with factor V Leiden is mostly restricted to veins, where the clotting may cause deep vein thrombosis. Deep vein thrombosis may occur without symptoms, but typically the affected extremity will be characterized by swelling, pain, redness, warmth of area, and engorging of the superficial veins. Long-term effects that can occur after deep vein thrombosis are edemas, discomfort, and skin complications.

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If the venous blood clots break off and travel to the lungs, pulmonary embolism occurs. Symptoms of pulmonary embolism may include difficulty breathing, chest pains, low blood oxygen saturation, cyanosis, rapid breathing, and heart palpitations. Severe cases of pulmonary embolism may involve collapsing, extremely low blood pressure, and sudden death.

It is extremely rare for factor V Leiden to result in blood clots forming in the arteries that may result in a stroke or myocardial infarction. Consequently, minimal research exists on the link between factor V Leiden and neurological complications. Two clinical cases of combined schizencephaly and thrombophilia have been reported in the literature, with both cases caused by mutations to methyltetrahydrofolate reductase and the factor V gene (Goez & Zelnik, 2009). Complications in these cases included both motor and cognitive deficits.

DIAGNOSIS

To diagnose a deep vein thrombosis, an intravenous venography may be performed. Intravenous venography involves injecting a peripheral vein of the affected limb with a contrasting agent and administering X-rays. Physical examinations, imaging of the extremity in question, and a blood test may also be performed in making a diagnosis.

For pulmonary embolisms, diagnosis is based primarily on observed clinical criteria combined with selective testing. Electrocardiogram and electrocardiography findings are used in conjunction with pulmonary angiography, CT scan, and blood test results to formulate a diagnosis.

Diagnosing factor V Leiden as the cause for a thrombotic event should be considered if a family history of venous thrombosis exists or if a Caucasian patient under 45 years of age presents. Methods for diagnosing factor V Leiden include screening with a snake-venom based test, a genetic test, or an Activated Partial Thromboplastin Time test.

TREATMENT

Diagnosing an individual with factor V Leiden occurs following a thrombotic event. Due to the genetic nature of the disorder, a cure does not currently exist. However, knowing your familial medical history may aid in prevention of either deep vein thrombosis or pulmonary embolisms.

Treatment for deep vein thrombosis may require hospitalization. Anticoagulation therapy using heparin is the usual treatment (Snow et al., 2007). Patients who suffer from reoccurring deep vein thrombosis may require life-long anticoagulation therapy. Elastic compression stockings are used in conjunction with anticoagulation therapy as well. If anticoagulation therapy is not successful, an inferior vena cava filter may be used. An inferior vena cava filter prevents new embolisms from entering the pulmonary artery. For extensive blood clots, thrombolysis may be performed.

The most popular treatment option for pulmonary embolisms involves anticoagulation therapy. Heparin or fondaparinux is administered initially, whereas warfarin, acenocoumarol, or phenprocoumon is administered later. If anticoagulation therapy is not successful, an inferior vena cava filter may be used. Both thrombolysis and surgical management are highly debated treatment options that exist as well (Turpie, 2007).

In treating thrombosis or blood clots, drugs such as heparin and warfarin are often used to inhibit the clot's formation and growth. These medications decrease blood coagulation through inhibition of vitamin K, an enzyme needed for forming mature clotting factors.

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