



## Effect of Risk Factors on the Development of Arterial Thrombosis

Wen Yuman\*

Department of Medicine, University of Sichuan, Sichuan, China

### DESCRIPTION

Arterial thrombosis results from intravascular injury and to a lesser extent, changes in haemostasis balance. Although some hereditary and acquired haemostasis risk factors in the pathophysiology of venous thrombosis have been described, the extent and type of abnormalities that contribute to arterial thrombosis are not well understood. Damage to endothelial cells due to the production of pro-inflammatory mediators stimulates the process of arterial thrombosis. Most often this is the result of intravascular injury due to atherosclerosis, but similar reactions can occur in other medical conditions.

Similarly, after the onset of thrombosis, fluctuations in the activity of coagulation proteins and endogenous anticoagulants, as well as the dynamics of platelet aggregation, can alter the effectiveness of thrombus formation. Epidemiological studies identify some acquired or hereditary conditions that can lead to endothelial damage or altered haemostatic balance, thereby making patients more susceptible to arterial thrombosis. These include hyperhomocysteinemia, increased creatinine protein, antiphospholipid antibodies, increased fibrinogen, factor VII, plasminogen activator inhibitor 1 (PAI1), hereditary thrombophilia and platelet hypersensitivity.

This review reviews the current understanding of these risk factors in the development of arterial thrombotic events. Currently, the literature supports the role of hyperhomocysteinemia, elevated CRP and elevated fibrinogen as risk factors for arterial thrombosis. Similarly, the literature suggests that increased titers of lupus anticoagulants and to a lesser extent, cardiolipin IgG antibodies predispose to arterial vascular events. In certain subgroups of patients, including patients with cardiac risk factors <55 years old and women, hereditary thrombotic predisposition such as factor V Leiden carriers and prothrombin G20210A mutations causes arterial thrombosis. The risk can be high. However, data on Factor VII,

PAI1 and platelet receptor polymorphisms are inconsistent or non-existent.

Arterial thrombosis is a blood clot that develops in an artery. It's dangerous as it can obstruct or stop the flow of blood to major organs, such as the heart or brain. If a blood clot narrows one or more of the arteries leading to the heart, muscle pain known as angina can occur. If a blood clot blocks the arteries leading to part of the heart muscle, it will cause a heart attack. If it blocks an artery in the brain, it will cause a stroke. Arterial thrombosis is a major feature of APS. Arterial occlusion can affect the vascular tree, from the aorta to the small capillaries. Ischemic stroke or transient ischemic attack (TIA) is the most common form of arterial thrombosis. Other arterial sites, including the retina, coronary arteries, brachial arteries, mesentery and peripheral arteries, are also affected by thrombosis.

Arterial thrombotic events in young patients with no apparent cause pose significant challenges in diagnosis and management presents a structured diagnostic approach that considers not only common causes such as atherosclerosis and embolism, but also uncommon causes such as drugs and substances, vascular and anatomical abnormalities, and systemic disease, and thrombosis, focus on management areas that have evolved over the last five years, including the use of dual pathway inhibition in atherosclerosis, the choice of antithrombotic therapy for unexplained embolic stroke and left ventricular thrombosis and the role of foramen ovale obstruction. Guess Foramen ovale for secondary stroke prevention and the possibility of coronavirus disease 2019 infection and vaccination thrombosis.

The majority of arterial thrombosis is not treated by a haematologist. The causes of plaque rupture, atrial fibrillation and other atherosclerosis of atherosclerosis are the cause of most arterial events, and extensive consensus guidelines for assessing and treating organ-specific arterial thrombotic disease.

**Correspondence to:** Wen Yuman, Department of Medicine, University of Sichuan, Sichuan, China, E-mail: yumanwen@scu.cn

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