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A commentary on Infarction

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Abstract

Infarction is tissue death necrosis due to inadequate blood supply to the affected area. It may be caused by artery blockages, rupture, mechanical compression, or vasoconstriction. The resulting lesion is referred to as an infarct from the Latin infarctus, "stuffed into". Infarction, death of tissue resulting from a failure of blood supply, commonly due to obstruction of a blood vessel by a blood clot or narrowing of the blood-vessel channel.

Introduction

Infarction Lung infarction, death of one or more sections of lung tissue due to deprivation of an adequate blood supply. The section of dead tissue is called an infarct. The cessation or lessening of blood flow results ordinarily from an obstruction in a blood vessel that serves the lung. The obstruction may be a blood clot that has formed in a diseased heart and has travelled in the bloodstream to the lungs, or air bubbles in the bloodstream (both of these are instances of embolism), or the blockage may be by a clot that has formed in the blood vessel itself and has remained at the point where it was formed (such a clot is called a thrombus). Ordinarily, when the lungs are healthy, such blockages fail to cause death of tissue because the blood finds its way by alternative routes. If the lung is congested, infected, or inadequately supplied with air, however, lung infarctions can follow blockage of a blood vessel.

Injury or death of tissue as of the heart or lungs resulting from inadequate blood supply especially as a result of obstruction of the local circulation by a thrombus or embolus the process of forming an infarct. Infarction may be caused by artery blockages, rupture, mechanical compression, or vasoconstriction. Infarction occurs as a result of prolonged ischemia, which is the insufficient supply of oxygen and nutrition to an area of tissue due to a disruption in blood supply. The blood vessel supplying the affected area of tissue may be blocked due to an obstruction in the vessel, compressed by something outside of the vessel causing it to narrow, ruptured by trauma causing a loss of blood pressure downstream of the rupture, or vasoconstricted, which is the narrowing of the blood vessel by contraction of the muscle wall rather than an external force (e.g., cocaine vasoconstriction leading to myocardial infarction).

Hypertension and atherosclerosis are risk factors for both atherosclerotic plaques and thromboembolism. In atherosclerotic formations, a plaque develops under a fibrous cap. When the fibrous cap is degraded by metalloproteinases released from macrophages or by intravascular shear force from blood flow, subendothelial thrombogenic material (extracellular matrix) is exposed to circulating platelets and thrombus formation occurs on the vessel wall occluding blood flow. Occasionally, the plaque may rupture and form an embolus which travels with the blood-flow downstream to where the vessel narrows and eventually clogs the vessel lumen. Nearly all cases of infarction are due to complete or near-complete occlusion of arterial blood flow due to embolism, most typically thromboembolism. Under rare circumstances arterial flow can also be completely disrupted anatomically due to twisting closed of the arterial supply as may occur during testicular torsion or bowel volvulus. Thrombotic or anatomic disruption of venous supply generally causes congestion rather than infarction as alternate routes of venous return usually exist.

Various organs are more or less prone to infarction than others. Generally, organs that have a single arterial supply are more at risk than those that have dual supplies. If the thromboembolic event occurs following a long-term, progressive stenosis of the artery then infarction is generally less severe as large numbers of alternative circulatory routes will likely have developed as a response to the chronically reduced blood flow. Additionally, some cells such as neurons and cardiomyocytes are much more sensitive to ischemia than others such as fibroblasts. Tissues which receive their blood solely from portal vessels, such as the posterior pituitary are much more prone to infarction than others as the blood they receive has a much lower oxygen tension.

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