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Commentary

A Brief Note on the Angina Pectoris and its Pathophysiology

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DESCRIPTION

Angina (or angina pectoris) is a pressure-like substernal chest discomfort caused by physical or mental stress and cured by rest or nitroglycerin (also known as "typical angina"). It has long been regarded as the primary sign of ischemic heart disease. However, people without ischemia may experience very similar symptoms (e.g., typical angina) (esophageal diseases, gastric disease, bronchopulmonary disease, etc.). Furthermore, numerous diseases/disorders, such as hypersensitive heart syndrome, pulmonary hypertension, severe aortic or mitral stenosis, and others, cause similar symptoms and may or may not be related to cardiac ischemia. Angina can be acute (as in an acute coronary syndrome, for example, unstable angina) or chronic (recurring), as in chronic stable angina.

Myocardial oxygen delivery may be restricted owing to a restriction in coronary blood flow caused by obstructive atherosclerotic plaque, coronary artery spasm, coronary microvascular dysfunction, or a non-coronary problem such as acute anaemia or hypotension. It might also be caused by an excessive rise in myocardial oxygen demand. This might be related to an increase in heart rate (HR), which could be caused by supraventricular tachycardia (atrial fibrillation or flutter, for example), hyperthyroidism, or other factors. This increase in oxygen demand may occur with or without a rise in HR, as in aortic stenosis, systemic hypertension, or mental stress. Multiple variables frequently work together to decrease myocardial oxygen supply (e.g., obstructive plaque) and raise myocardial oxygen demand (e.g., physical effort) to create myocardial ischemia. Angina is quite common all around the world. Angina affects an estimated 8.2 million people in the United States alone. Importantly, it is a major cause of poor quality of life, disability, and excessive use of health-care resources.

Pathophysiology

Angina is often a sign of myocardial ischemia, which arises as a result of a mismatch between myocardial oxygen supply and demand. The myocardium receives oxygenated blood *via* epicardial coronary arteries that branch into arterioles. Arterioles branch out into a network of capillaries. Epicardial coronary arteries are typically a low resistance system. Changes in arteriole tone mostly provide autoregulation. Arterioles dilate in reaction to nitric oxide, prostaglandins, carbon dioxide, hydrogen ion, adenosine, and other nucleotides as myocardial oxygen demand increases. Blood flow to normal myocardium can be increased four to fivefold by autoregulation. This is known as myocardial perfusion reserve or coronary flow reserve. The resistance of the epicardial coronary arteries is increased by atherosclerotic plaques. Cross-sectional stenosis is an important factor influencing blood flow *via* coronary arteries. Arterioles dilate to sustain myocardial blood flow as coronary stenosis develops. Even at rest, there may be insufficient blood flow in the presence of an epicardial coronary stenosis greater than 70%, resulting in ischemia and angina.

Other factors that influence myocardial oxygen supply include collateral blood flow, left-ventricular end diastolic pressure (which can reduce perfusion pressure from epicardium to endocardium capillaries), and diastolic-perfusion time (related to HR and aortic diastolic stress), because myocardial perfusion occurs primarily during diastole. The essential cardiomyocyte substrate for energy synthesis in the form of adenosine triphosphate is oxygen (ATP). Myocardial oxygen demand is determined by myocardial wall stress (a pre-load marker), myocardial contractility, systolic blood pressure (an afterload marker), and HR. Furthermore, myocardial energy demand is affected by systolic wall tension and left-ventricular mass. Myocardial energy requirements can rise up to fourfold in aortic stenosis and thrice in essential hypertension.

Angina pectoris is characterised by visceral pain. The particular underlying processes responsible for the development of severe discomfort remain unknown. Ischemia of the heart causes acidosis and the lack of normal ATP sodium-potassium pump and membrane integrity. Adenosine, lactate, serotonin, bradykinin, histamine, and reactive oxygen species are released and trigger chemo-sensitive receptors. Stimulation of afferent sympathetic fibres in the upper thoracic spinothalamic tract causes chest and arm pain symptoms, whereas stimulation of vagal afferent fibres causes excitation of the cervical spinothalamic tracts causes neck and/or jaw pain symptoms.

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