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Transmyocardial laser revascularisation and other treatment modalities for angina pectoris

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Abstract Ischaemic heart disease is one of the leading causes of morbidity and mortality in the western world. This paper provides an overview of the different treatments for one of the most common manifestations of ischaemic heart disease: angina pectoris. Besides the currently available conventional methods, several alternative treatments are described, with a special focus on transmyocardial laser revascularisation.

Keywords Angina pectoris · Angiogenesis · Denervation · Perfusion · Refractory · Treatment

Introduction

Ischaemic heart disease is one of the leading causes of morbidity and mortality in the western world. Atherosclerosis of the coronary arteries leads to a mismatch between the demand for and supply of oxygen in the heart muscle. In the healthy heart with normal coronary arteries an increased oxygen demand, for example during exercise, is met by increased coronary blood flow, coronary vasodilation and increased oxygen extraction. However, atherosclerosis in coronary arteries prevents an effective increase in supply during increased demand. When the obstruction is severe, this results in myocardial ischaemia, which can be either reversible or irreversible. The latter is usually caused by total occlusion of a coronary artery, during which the myocardium distal from the occlusion is deprived of oxygen. If blood flow through the artery is not restored, the myocardium can either die, resulting in infarction and permanent damage to the tissue, or, thanks to collateral blood supply, remain ischaemic or hibernating. In the situation of reversible myocardial ischaemia, the coronary artery is usually not completely occluded and when the oxygen demand is decreased the myocardium has the ability to recover from the episode of oxygen shortage, resulting in relatively little or no permanent damage. One of the most common clinical manifestations of these periods of reversible myocardial ischaemia is angina pectoris. This paper aims to provide an overview of the different conventional and alternative (i.e. for patients with refractory angina) treatment regimens for angina, with special emphasis on the clinical efficacy and working mechanism of transmyocardial laser revascularisation.

The clinical problem of angina pectoris

The typical clinical presentation of angina pectoris was first described by Dr William Heberden, a famous physician of the 18th century (Fig. 1). In 1772 he published the article ‘Some account of a disorder of the breast’ [1], in which he wrote: ‘There is a disorder of the breast, marked with strong and peculiar symptoms, considerable for the danger belonging to it...Those who are afflicted with it are seized, while they are walking, and more particularly when they walk soon after eating, with a painful and most disagreeable sensation in the breast...the moment they stand still all this uneasiness vanishes....After it has continued some months, it will not cease so instantaneous upon standing still...most whom I have seen, who are at least twenty, were men, and almost all above 50 years old, and most of them with a short neck, and inclining to be fat....But the natural tendency of this illness be to kill the patients suddenly. ...The os sterni is usually pointed to as the seat of this malady...and sometimes there is with it a pain
about the middle of the left arm.' [2] At the time, Heberden did not consider the heart to be a possible source of this typical pain. Indicating the location of the pain, he referred to it as ‘pectoris dolor’, which was later changed by his son to ‘angina pectoris’.

Over two centuries later, Heberden’s description of angina pectoris is still remarkably accurate. According to the current guidelines of the American Heart Association (AHA) and the American College of Cardiology (ACC), angina pectoris is defined as ‘...a clinical syndrome characterised by discomfort in the chest, jaw, shoulder, back, or arm. It is typically aggravated by exertion or emotional stress and relieved by nitroglycerin. Angina usually occurs in patients with coronary artery disease involving ≥1 large epicardial artery. However, angina can also be present in patients with normal coronaries and myocardial ischaemia related to spasm or endothelial dysfunction [3]. The neural aetiology of anginal pain is poorly understood. Activation of cardiac nociceptors is thought to be caused by ischaemia-induced release of specific substances (such as adenosine [4]). The pain stimulus is transported through cardiac afferent fibres to the spinal cord. After converging with other afferent fibres the stimulus is transported to the somato-sensible cortex of the cerebrum, where it is registered as typical angina pectoris [5]. The convergence of different afferent fibres from different dermatomes in the spinal cord is probably the reason why activation of cardiac nociceptors is experienced not only as pain in the cardiac region (chest) but also in other dermatomes, such as the jaw, arm or shoulder (‘referred pain’) [6].

Angina can be classified as either stable or unstable. The severity of stable angina can furthermore be classified according to the classification of the Canadian Cardiovascular Society (CCS) [7] or according to the classification of the New York Heart Association (NYHA) [8] (for a comparison see http://www.cochranfoundation.com/docs/nyha-class.htm). In stable angina, the severity of the disease can increase but will do so in a fairly constant manner. Angina-triggering events are usually predictable (e.g. walking up stairs). In unstable angina (which has a separate classification [9]), unpredictable episodes of severe myocardial ischaemia can lead to myocardial infarctions or sudden death, and hospital admittance is indicated.

Treatment of angina pectoris

The treatment of angina pectoris is generally based on three different approaches: medication, aimed at (a) decreasing myocardial oxygen demand, (b) increasing oxygen supply (e.g. by vasodilation), and (c) reducing the risk of further thrombotic coronary occlusions; revascularisation techniques, i.e. location-specific restoration of blood flow to the ischaemic myocardium; and symptomatic treatment, used for angina refractory to medication and revascularisation.

Medication

The first approach, reducing myocardial oxygen demand through cardiac medication, is the most widely used. It is also the first option that should be considered when a patient presents with angina pectoris. Three main types of cardiac medication are used for angina pectoris: β-blockers [10], calcium antagonists [11] and long-acting nitrates [12]. In short, β-blockers inhibit β receptors of the sympathetic nervous system, decreasing both heart frequency (negative chronotrope) and the strength of the cardiac contraction (negative inotrope), and thereby decreasing the oxygen demand. Calcium antagonists act mainly by reducing calcium concentrations in vascular smooth muscle cells, which induces (a) coronary vasodilation, leading to improved myocardial perfusion, and (b) peripheral arterial vasodilation, leading to an afterload reduction and subsequent decreased myocardial oxygen demand. Furthermore, some calcium antagonists also have a negative chronotrope effect. Long-acting nitrates are vasodilators that work in two ways. They reduce the cardiac preload and therefore the myocardial oxygen demand through dilation of the peripheral venous system, and they increase the myocardial blood supply through their (nitric oxide-mediated) vasodilatory effect on the coronary arteries. These three types of cardiac medication are often used in combination, forming the well known ‘triple therapy’ regimen. A fourth type of medication which is widely used in patients with angina pectoris is antiplatelet therapy (e.g. aspirin), which reduces platelet aggregation [13]. Unlike β-blockers, calcium antagonists and long-acting nitrates, this therapy is not aimed at direct treatment of anginal symptoms but more at the prevention of new thrombotic coronary occlusions.