Relief of Angina Pectoris When Carrying Heavy Loads with the Left Hand in a Patient with Previous Coronary Artery Bypass Graft Operation Who Has Severe Exercise Angina: A Case Report

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ABSTRACT

In patients with known coronary artery disease and/or a history of revascularization, angina pectoris or unstable coronary syndromes are usually attributed to the progression of atherosclerotic lesions rather than an unrecognized great vessel disease. However, for patients with a previous coronary artery bypass graft operation (CABG), during which a left internal mammary artery (LIMA) conduit has been used, great vessel disease, especially subclavian artery stenosis should also be suspected. We present a case of a patient with a LIMA conduit who has angina pectoris on exertion, but interestingly the pain is relieved when he carries heavy loads with his left hand, which can be due to increased blood flow to the LIMA conduit during heavy lifting because of increased peripheral resistance.

Key words: angina pectoris, subclavian stenosis, LIMA conduit, heavy lifting, peripheral resistance

Introduction

We present a patient who had undergone coronary artery bypass graft operation with a LIMA conduit 11 years ago. When carrying heavy loads with the left hand, the peripheral resistance in the left arm is increased which causes an increase in blood flow to the LIMA conduit. The local regulation of blood flow during muscle contractions and the vasoconstrictor and vasodilator mechanisms of the autonomic nervous system during heavy lifting are briefly reviewed.

Case Report

A 79-year-old man was admitted to our hospital with severe angina on exertion. He had retrosternal squeezing chest pain radiating to his neck, when he walked fast or climbed 1 flight of stairs, which was relieved when he rested. Interestingly, he was able to climb the stairs in his house to the third floor without chest pain if he carried a few kilograms of weight with his left arm. He had undergone coronary artery bypass graft surgery in 1996 (left anterior descending artery—LIMA; circumflex artery—saphenous vein; right coronary artery—saphenous vein).

Physical examination revealed a left subclavian bruit and a significant blood pressure difference between his right and left arms (150/90 mm Hg versus 110/70 mm Hg). Hemogram and blood chemistry were in the normal range and there were no significant changes in the 12 lead ECG.

Coronary angiography demonstrated that the saphenous vein grafts to the right coronary artery (RCA) and the circumflex artery (Cx) were occluded, and the native second obtuse marginal (OM2) and the third obtuse marginal (OM3) were completely stenotic. The left anterior descending coronary artery (LAD) and LIMA conduit were patent. A near total stenosis was present at the ostium of the left subclavian artery (Figure 1). Digital subtraction angiography was performed to observe the ascending aorta, aortic arch vessels, and left subclavian artery which were normal. The 95% stenosis at the prevertebral part of the subclavian artery was first predilated with a balloon catheter then a stent was implanted. The symptoms relieved after the procedure and the blood pressure difference was diminished.

Discussion

In patients with known coronary artery disease and a history of revascularization, unstable coronary syndromes are likely to be attributed to the progression of the coronary artery disease rather than an unrecognized great vessel disease. However, a subclavian artery lesion should be suspected in patients with recurrent ischemic symptoms following a coronary artery bypass graft operation during which a LIMA conduit has been used since the subclavian artery is the vessel which gives origin to the LIMA conduit.^{1,2} Proximal subclavian artery stenosis is usually caused by atherosclerosis, but several other pathologic processes can lead to this condition including Takayasu's arteritis,

Figure 1. Coronary angiography, showing the near total stenosis at the ostium of the left subclavian artery.

radiation arteritis or giant cell arteritis.³ In the present case, during the coronary angiography the subclavian stenosis is clearly demonstrated and it is definitely the cause of the angina pectoris. The interesting part is the relief of pain during heavy lifting with the left hand.

We relate the relief of pain during heavy lifting to the increase of blood flow to the LIMA conduit due to an increase in peripheral resistance in the left arm. During heavy lifting there is an increase in sympathetic stimulation, a rise in plasma catecholamines and a decrease in parasympathetic stimulation. Increase in sympathetic drive is partially due to the sustained static component of lifting.^{4,5} In order to overcome the extraluminal pressures of the exercising muscles, systemic arterial blood pressure must be high enough intraluminally. So higher blood pressure is needed to offset the increase in extraluminal pressure, which means sympathetic activation and vasoconstriction.⁶ There is considerable α 1 and α 2 adrenergic receptor mediated vasoconstriction in active skeletal muscles even at heavy exercise intensities.⁷

The high intramuscular pressures seen in lifting also promotes occlusion of the arterial vessels. The systemic resistance is elevated by the occlusion and the mean arterial pressures are increased thereafter. Also the occlusion of the arteries decreases blood flow to active tissue, so metabolic by-products such as lactic acid, hydrogen ion, and adenosine diphosphate (ADP) are increased. This activates nerve endings, which then elevates mean arterial pressure via the pressor reflex.^{8,9,10,11,12}

Local regulation of blood flow involves a balance between vasodilator and vasoconstrictor mechanisms. Different researchers have found that total peripheral resistance

decreases with heavy lifting, while others found that it increases.^{6,13,14,15,16} The discrepancy might be a result of differences in active muscle mass. Nevertheless, our case is an example of increased peripheral resistance since blood flow to the LIMA conduit increases as systemic circulation through the brachial artery is impeded by increased peripheral resistance and less blood is diverted to the upper extremity.

The vasoconstrictor and vasodilator influences may be altered in elderly individuals which is probably the condition in this 79-year-old patient.¹⁷ Endothelium dependant vasodilation is one potential local control mechanism that may undergo age-related impairment. In a number of studies, intra-arterial infusion of vasoactive drugs into a resting limb, allowed assessment of the intrinsic vasoreactivity of the resistant vessels, namely the small arteries and arterioles. In these studies forearm vasodilation is blunted in sedentary older men compared to younger control subjects.^{18,19,20,21} It can be suggested that endothelium dependant vasodilation in peripheral circulation declines with advancing age in humans. Possible mechanisms are diminished eNOS activity,²² reduced arginine substrate availability,²³ decreased expression of antioxidants,^{24,25} and increased cyclooxygenase dependant vasoconstriction.²⁶

In another recent study, older men exhibited larger reductions in exercising leg vascular conductance following acute sympathetic stimulation (cold pressor test) than younger men suggestive of augmented sympathetically mediated vasoconstrictor responsiveness in leg vessels of older humans.²⁷

Two subsequent studies involving women²⁸ and men²⁹ described augmented sympathetic vasoconstriction with age in a dynamically contracting forearm. Evidence suggest that augmented sympathetic vasoconstrictor responsiveness in exercising forearm muscle of older adults could be due to impairments in functional (exercise) sympatholysis.^{28,29}

In another study comparing young men with healthy older men, greater forearm vasoconstriction during brief submaximal dynamic leg exercise that appears to be mediated by augmented constriction in the skeletal muscle circulation is demonstrated.³⁰ It is claimed that, this does not represent a nonspecific hyperreactivity to all forms of acute stress because augmented responses were not observed during a nonexercise sympathoexcitatory stimulus like the cold pressor test.

In the present case, we concluded that the vasoconstrictor response predominated in his arm vessels during heavy lifting, considering the endothelial dysfunction and other previously mentioned mechanisms that were described in the elderly; all of which contributed to the increase in peripheral resistance.

The exertional angina relieved when the subclavian stenosis was corrected by percutaneous interventional techniques and the amount of blood flow through the

subclavian artery to the LIMA conduit was normalized. The mechanisms described were still valid, but no longer strong enough to cause symptoms.

In differential diagnosis we considered a rare condition called coronary-subclavian steal syndrome, which is characterized by angina pectoris with upper arm exercise in the presence of subclavian stenosis.^{1,2,3,31} During normal circumstances, blood flow to the exercising muscle needs to be increased during exercise. In this syndrome, angina pectoris is due to the reversal of blood flow in the LIMA conduit during upper extremity exercise.

Our patient has angina when walking fast, climbing upstairs, but not with upper extremity exercise. In addition to this, he has no angina when climbing upstairs with a few kilograms of weight in his left hand. So a diagnosis of coronary-subclavian steal syndrome is unlikely.

In conclusion, the reason for pain relief in this 79-year-old patient with a LIMA conduit and subclavian stenosis when carrying heavy loads with his left hand, can be increased blood flow to the LIMA conduit because of increased peripheral resistance, due to both vasoconstriction during heavy lifting since endothelium dependant vasodilation is impaired in the elderly and increase in the hydrostatic pressure as the blood flow to the extremity is impeded by arterial occlusion caused by muscular contractions

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