Idiopathic Great Saphenous Phlebosclerosis

Ahmadreza Jodati, Babak Kazemi, Naser Safaei, and Behrooz Shokoohi

Madani Cardiovascular Medical and Research Center, Tabriz University of Medical Sciences, Tabriz, Iran

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Abstract- Arterial sclerosis has been extensively described but reports on venous sclerosis are very sparse. Phlebosclerosis refers to the thickening and hardening of the venous wall. Despite its morphological similarities with arteriosclerosis and potential morbid consequences, phlebosclerosis has gained only little attention. We report a 72 year old male with paralysis and atrophy of the right leg due to childhood poliomyelitis who was referred for coronary artery bypass surgery. The great saphenous vein, harvested from the left leg, showed a hardened cord-like obliterated vein. Surprisingly, harvested veins from the atrophic limb were normal and successfully used for grafting.

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Introduction

Vascular grafts have been extensively used for many years for revascularization of ischemic cardiac muscle. The great saphenous vein (GSV) and the left internal thoracic artery are widely used as grafts to bypass stenotic or occluded coronary arteries. Despite the increased use of arterial grafts, due to their better long-term patency, the GSV remains the most commonly used conduit for coronary artery bypass grafting (CABG). The short and long term complications and morphological changes of these grafts in the post-operative period are well documented (1,2). Phlebosclerosis of GSVs appears to be exceptional thus warranting the presentation of this case with a review of the literature.

Case Report

A 72-year old diabetic and hypertensive man with recent unstable angina pectoris was referred to our center for CABG. He had quitted cigarette smoking for the last 20 years. General physical examination showed a paralytic and atrophic right lower limb due to childhood poliomyelitis (Figure 1).

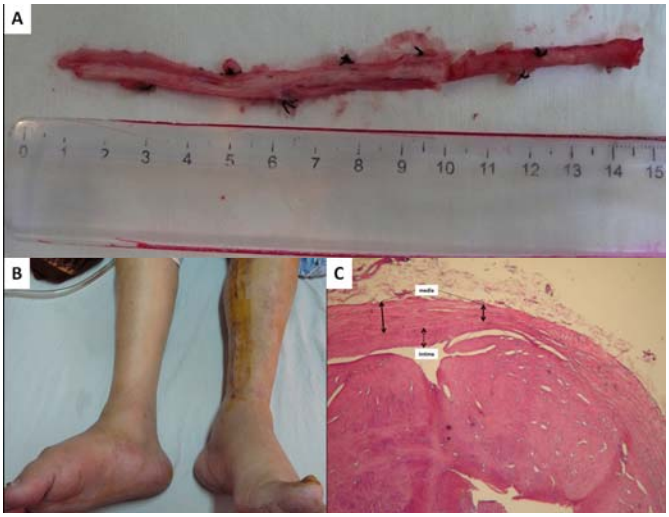

Figure 1. (A) Dissected cord-like and obliterated vein. (B) Atrophied right leg. (C) Photograph from light microscopy reveals near complete obliteration of venous lumina by dense fibrous tissue and presence of large areas of calcification, foci of neovascularization, and scattered mononuclear inflammatory cells. The wall shows extensive fibrosis with loss of muscle and marked thickening of the intima (Haematoxylin & Eosin stain; ×100).

Corresponding Author: Babak Kazemi
Department of Cardiology, Madani Heart Center, Tabriz University of Medical Sciences, Tabriz, Iran.
Tel/Fax: +98 411 3344021, E-mail: bkazemia1966@gmail.com

There was no evidence of varicose veins. Palpation of the peripheral arterial pulses was normal. Laboratory findings were unremarkable. Echocardiography showed mild left ventricular hypertrophy, mild to moderate mitral regurgitation, and a normal systolic function. All three major epicardial coronary arteries had significant proximal stenosis. Doppler ultrasonography of the carotid arteries showed non-obstructive plaques. The GSV, harvested from the left leg, showed a hardened cord-like obliterated vein (Figure 1). Surprisingly, harvested veins from the atrophic limb were normal and successfully used for grafting. Surgery was uneventful and the patient was discharged on the seventh postoperative day. At 1, 3, and 6-month follow-up visits, the patient was doing well.

Discussion

More than a century ago, phlebosclerosis was first defined and was also named as venofibrosis, peripheral phlebosclerosis, and hyperplastic endophlebitis (3). Later, hyalinization of the intima and hyperplasia of fibrous tissue in the media, leading to progressive, painless obliteration of the lumen was described (3). It was always bilateral, affecting both superficial and deep veins, and felt like hard mobile cords (3).

Phlebosclerosis constitutes a fibrous degeneration of the venous wall, predominantly the intima, with or without calcification. It is regularly found in non-varicose veins of aged persons (4). Although its incidence increases with age, the severity of the sclerosis is not correlated with aging. Both sexes and all the superficial leg veins (various levels of the GSV and minor venous branches) are affected (4). Although the morphological appearance is similar to that of arteriosclerosis, localization, progression and clinical consequences are different (4). Phlebosclerosis of superficial leg veins is a disorder of little direct clinical consequence. However, in the deep leg veins a distinct phlebosclerosis of the intimal layer may be responsible for the development of thrombosis. As a secondary alteration, phlebosclerosis may complicate varicose veins. Our patient did not have any history or clinical evidence of varicose veins or vein thrombosis.

Calcification of the leg veins is a rare finding. Kane-ToddHall *et al*. compared the histological specimens from 177 radial arteries with 168 of internal mammary arteries and GSVs from the same patients undergoing CABG (5). The degree of stenosis, active atheroma, intimal thickening, and medial hyaline sclerosis was assessed. Sclerosis and calcification was present in 36% and 1% of GSVs, respectively. Controversy about the definition, etiology, pathogenesis and histological aspects of phlebosclerosis exists. The exact pathophysiologic mechanisms are unknown. Despite the confusing terminology and limited scientific data, Tzogias *et al*. recently proposed that phlebosclerosis should be regarded as a distinct clinical entity and that its prevalence and clinical significance might be underestimated (6).

Our case comprised of a unilateral ossification of the GSV wall without any apparent etiology, which has been barely described.

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