Thrombolysis for acute peripheral arterial thrombosis

Thesis

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By

Dr. Emad Ramadan Goda

M.B.B. CH Cairo university

Supervised by

Prof. Dr. Mohamed Khedr

Professor of General Surgery
Faculty of Medicine
Cairo University

Prof. Dr. Hussein Khairy

Professor of General Surgery
Faculty of Medicine
Cairo university

Assist. Prof. Dr. Alaa abdel Haleem

Assist. Professor of General Surgery
Faculty of Medicine
Cairo university

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Introduction

Thrombosis is one of the two main causes of peripheral arterial occlusion (PAO). Surgical treatment of acute limb ischemia because of related complications has a 30-day morality rate of 15% to 25%. Intra – arterial thrombolysis for lower extremity ischemia is a well accepted and frequently used technique. It may offer definitive treatment without the need for major surgery in a significant series of patients with acute occlusion of a native leg artery or a by-pass graft. Thrombolytic agents include streptokinase (SK), urokinase (UK), Pro-UK and recombinant tissue plasminogen activators (rt-PA- Alteplase and r-PA reteplase). All this agents induce a systemic fibrinolytic state **(Giannini & Balbarini 2004).**

Patients with acute limb ischemia secondary to in situ thrombosis usually require emergency revascularization either surgically or with the use of thrombolytic agents **(Ose, et al; 1994).**

Thrombolysis achieving dissolution of the occluding thrombus, reconstitution of blood flow and improvement in the status of the tissue bed supplied by the involved vascular segment. In addition thrombolysis can clear thrombus from small arteries that are inaccessible to a ballon catheter **(Ouriel 2002).**

Catheter – directed thrombolytic therapy has become an important part of the treatment of patients with acute arterial and graft occlusion. The underlying pharmacologic principle is the activation of plasminogen, bound to fibrin within the thrombus. Guide-wire passage predicts success of catheter – directed thrombolysis. The underlying disease process leading to thrombosis should be accurately identified and promptly

corrected to reduce the probability of recurrent occlusion **(Comerota & Cohen 1993).**

The best results occur when the thrombus is lysed within 30 days; however, successful thrombolysis has occurred up to four months after an arterial occlusion. Thrombolysis allows dissolution of thrombus in the small distal run off vessels, decreasing outflow resistance and enabling the native artery or bypass graft to remain open longer. When bypass grafts thrombose thrombolytic agents are usually successful in lysing the thrombus and identifying the cause for the thrombosis. With local intraarterial infusions, side effects and complications may be kept to a minimum **(Olin & Graor 1988).**

Aim of the work

Thromblysis is now well established, becoming employed increasingly in diseases of peripheral vasculature. This is a general review of its applications in acute peripheral arterial thrombosis **(in the lower extremity).**

Patients and methods

There will be a case presentation of 20 cases of acute thrombotic peripheral arterial occlusion with < 14 days duration in which thrombolysis is used in a retrospective study statistical analysis of these cases will be performed to find primary patency rate, secondary patency rate and complications.

بسم الله الرحمن الرحيم

"ويسألونك عن الروح قل الروح من أمر ربي وما أوتيتم من العلم إلا قليلاً"

صدق الله العظيم

الإسراء (85)

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List of abbreviations

CFA	Common femoral artery
SMCS	Smooth muscle cells
PAN	Polyartaritis nodosa
VWF	Von willibrand factor
AT III	Antithrombin III
TM	Thrombo modulin
APC	Activated protein C
t-PA	Tissue plasminogen activator
rt-PA	Recombinant tissue plasminogen activator
UK	Urokinase
SK	Streptokinase
Pro UK	Prourokinase
IATT	Intraarterial thrombolytic therapy
IIFT	Intraoperative intraarterial fibrinolytic therapy
EGF	Epidermal growth factor
TOPAS	Thrombolysis or peripheral arterial surgery
STILE	Surgery versus thrombolysis for ischemia of the lower extremity
APAO	Acute peripheral arterial occlusion
TIA	Transient ischemic attacks.
ABI	Ankle brachial index

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PATHOLOGICAL PREVIEW

Although blood vessels can be secondary affected by lesions in adjacent structures, primary vascular disease is the major concern of this chapter. In general, vascular abnormalities cause clinical disease by (1) progressively narrowing the lumina of vessels and producing ischemia of the tissue perfused by that vessel; (2) provoking intravascular thrombosis, causing acute obstruction or embolism (or both); (3) weakening the walls of vessels, thereby leading to dilatation or rupture ***(Pober & Cotran, 1990).***

Three basic structural constituents make up the walls of blood vessels: endothelium, smooth muscle, and connective tissue, including elastic elements. They are arranged in concentric layers (or tunicas) an intima, a media, and an adventitia (Fig. 1).

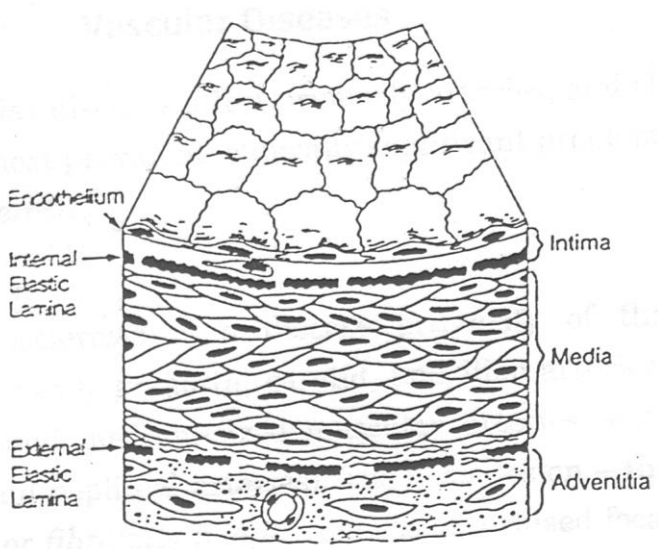

Fig- (1): Diagrammatic representation of the main components of the vascular wall, seen here in a muscular artery.

In normal arteries, the intima is composed of the lining endothelial cells with minimal underlying subendotheial connective tissue.

It is separated from the media by a dense elastic membrane (internal elastic lamina), which is interrupted by fenestrate, through which smooth muscle cells

may migrate from the media into the intima. The outer limit of the media of most arteries is marked by a well-defined external elastic lamina that is usually somewhat less well developed and delineated than the internal membrane.

The media varies among different vessels. In the larger elastic arteries (i.e., aorta and innominate, the adventitia is composed of fibrocellular connective tissue and contain a network of vasavasoram composed of small arteries, arterioles, capillaries, and venous channels as well as nerves that mediate smooth muscle tone and contraction. The adventitia varies in thickness and organization. In some arteries, such as the proximal renal and mesenteric trunks, the adventitia is a layered structure composed of both collagen and elastic fibers and may be thicker than the associated media.

Vascular Diseases

Vascular diseases affect primarily arteries, and of these, the most prevalent, clinically significant problem is atherosclerosis

Atherosclerosis is a disease primarily of the elastic arteries (e.g., aorta, carotid, and iliac arteries) and large and medium-sized muscular arteries (e.g., coronary and popliteal arteries). The basic lesion - the atheroma, or fibrofatty plaque consists of a raised focal plaque within the intima, having a core of lipid (mainly cholesterol and cholesterol esters) and a covering fibrous cap. Atheromas are sparsely distributed at first, but as the disease advances, they become more and more numerous, sometimes covering the: entire circumference of severely affected arteries. As the plaques increase in size, they progressively encroach on the lumen of the artery as well as on the subjacent media. Consequently, in small arteries, plaques are occlusive, compromising blood flow to distal organs and causing ischemic injury, but in large arteries they are destructive, weakening the affected vessel wall, causing aneurysms or rupture or favoring thrombosis***. (Strong, 1991).***