

شبكة المعلومات الجامعية التوثيق الإلكتروني والميكروفيلو

بسم الله الرحمن الرحيم





HANAA ALY



شبكة المعلومات الجامعية التوثيق الإلكتروني والميكرونيله



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جامعة عين شمس التوثيق الإلكتروني والميكروفيلم قسم

نقسم بالله العظيم أن المادة التي تم توثيقها وتسجيلها على هذه الأقراص المدمجة قد أعدت دون أية تغيرات



يجب أن

تحفظ هذه الأقراص المدمجة بعيدا عن الغبار



HANAA ALY

INTRODUCTION

Deep venous thrombosis (DVT) is a major cause of morbidity and mortality all-over the world. Complications include pulmonary embolism (PE), post-thrombotic syndrome (PTS), phlegmasia alba dolens, phlegmasia cerulea dolens and venous gangrene. The PTS is the most common cause of long-term morbidity and disability among the previous complications (Markel et al., 2003).

The pathology of DVT has been studied since the 19th century and based on the seminal work of Virchow. Stasis, hypercoagulability, and endothelial damage have long been considered the underlying etiologies. It is rational to relate this pathogenesis to increased risk of venous thromboembolism (VTE)is associated with immobility, major trauma, thrombophilia and various hematologic disorders which could be detected in up to 50% of patients with a spontaneous DVT (*Kyrle and Eichinger*, 2005).

Only 20-30% of patients with acute DVT manifested by symptoms and physical signs such as pain, swelling with pitting oedema, calf tenderness and Hoffman's sign. While 70% of patients manifested only by one of the classical signs or may be asymptomatic (*Dedden et al.*, 2002).

Doppler and duplex ultrasound studies are the initial investigation of choice in nearly all patients with suspected DVT (*Tovey and Wyatt, 2003*).

Introduction

Prevention and early detection of VTE before any clinical symptoms or signs are more cost effective than treatment of the complications when they occur (*Hauch et al.*, 2012).

The management of acute DVT and its complications remains controversial. The goal of therapy is to eliminate the thrombus, minimize the risk of pulmonary embolism, preserve valvular function, and prevent PTS (*Hyers*, 2011).

The current conventional treatment of acute DVT consists of anticoagulation and the use of compression stockings. Anticoagulation therapy consists of intravenous unfractionated heparin (UH) or subcutaneous low molecular weight heparin (LMWH) initially, followed by oral anticoagulants. UH or LMWH is continued for at least five days and warfarin therapy for at least three months, the total duration varying according to the underlying risk factors for recurrence and complications. In general, this approach to treatment is effective and safe in most patients. However, it neither promotes lysis to reduce the thrombus load, nor does it contribute to restoration of venous valve function. Anticoagulation alone, therefore, might not sufficiently protect the limb from PTS (Mismetti and Decousus, 2007).

The non-conventional thrombo-ablative therapies of acute DVT include systemic thrombolytic therapy; catheter-directed thrombolytic therapy (CDT) and surgical thrombectomy. Application of these techniques could be mandatory in acute extensive iliofemoral

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DVT with complications e.g. phlegmasia cerulea dolens and also could potentially result in a lowering of the PTS by preservation of the venous valve function (*Janssen et al.*, 2004).

Local-regional thrombolytic therapy has emerged in the past decade as a possible superior approach, allowing delivery of the pharmacological thrombolytic agent directly in the vicinity of the venous thrombus. This technique has evolved to address the main limitations of systemic thrombolysis (*Janssen et al.*, 2004).

Major advantages of catheter-directed thrombolytic therapy include:

- Insured exposure of pathologic thrombus to lytic agent
- More rapid clot dissolution
- Decreased infusion time
- Fewer complications(e.g. bleeding)

(Semba and Dake, 1994)

The thrombolytic agents are classified into three groups according to their mechanism of action:

- 1- Plasminogen activators: streptokinase (SK) and urokinase (UK).
- 2- Proteolytic enzymes: plasmin and brinase.
- 3- Fibrin-specific activators: tissue plasminogen activator (t-PA), single chain urokinase and anisoylated purified streptokinase activator complex (APSAC) (*Kayali and Stein, 2007*).

AIM OF THE WORK

The aim of this work is evaluation of the regional catheter-directed thrombolytic therapy versus standard systemic anticoagulant therapy in cases of acute ilio-femoral deep vein thrombosis as regard to efficacy, safety as well as complications.

Chapter 1

ANATOMY OF THE VENOUS SYSTEM OF THE **LOWER LIMB**

The venous system of the lower limb is divided into three groups:

- A- Superficial system, which lies outside the deep fascia.
- **B-** Deep system, which lies within the deep fascia.
- C- Perforating system, which passes through the deep fascia and connects the deep and superficial system.

(Michael, 2002)

A- The superficial venous system of the lower limb

1) The long or the great saphenous vein: (Internal or Saphena magna). The term saphenous is derived from the Greek word for "visible" (Williams, 2005).

The long saphenous vein is the longest vein in the body. It is formed by the union of veins from the inner part of the foot and the medial marginal vein and runs upwards for 1 to 1.5 inches in front of the medial malleolus of the tibia (Gray's Anatomy; 2005).

It extends upward along the anteromedial aspect of the leg and thigh to join the common femoral vein at the groin (Fig. 1) (Dodd and Cockett, 1996).

Terminal tributaries:

Just below the sapheno-femoral junction, It receives several additional tributaries, including the lateral and medial femoral cutaneous veins, the internal pudendal vein, superficial circumflex iliac vein and the superficial epigastric vein (Robert et. al., 2004).

2) The short saphenous: (external, small or lesser saphenous vein).

It begins along the lateral side of the dorsum of the foot as a continuation of the dorsal venous arch then it passes upward behind lateral malleolus, along lateral border of tendo-calcaneus (Henry, 2004).

In the foot and lower part of the calf it is accompanied by the sural nerve. At the popliteal fossa it typically passes between the two heads of the gasterocnemius and empties into the popliteal vein (Fig. 1) (Henry, 2004).

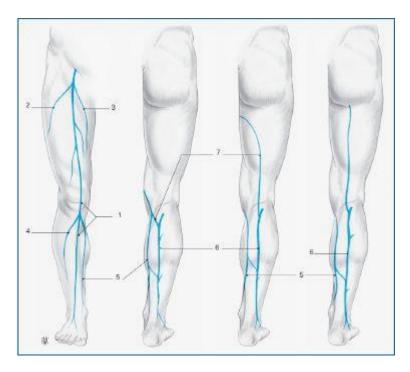


Figure (1): The superficial venous network of the lower limb: 1- great saphenous vein; 2-5 tributaries of the saphenous veins; 6- small saphenous vein; 7- vein anastomosing with the great and small saphenous veins (*Gray*, 2005).

B-The deep venous system of the lower limb

I. Deep veins of the leg:

- 1. The Posterior tibial vein: It receives blood from the medial and lateral plantar vein and drains the posterior compartment of the leg and plantar surface of the foot. This vein lies behind the tibia and joins the popliteal vein at the posterior knee (Lee et al., 2017).
- **2.** The Anterior tibial vein: It is the upward continuation of the dorsal pedal vein. It runs along the anterior compartment of

the leg just above the interosseous membrane between the tibia and the fibula, and joins the posterior tibial vein to form the tibioperoneal trunk and opliteal vein (Lee et al., 2017).

- **3. The peroneal vein:** It runs along the posteromedial aspect of the fibula and joins the posterior tibial vein (Lee et al., 2017).
- **4.** The popliteal vein: It is formed at the lower border of the popliteus muscle, ascends through the popliteal fossa to the hiatus of the adductor magnus, where it becomes the superficial femoral vein (Fig. 2) (Dodd and Cockett, 1996).

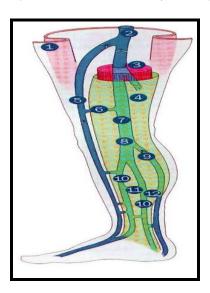


Figure (2): Anatomy of the popliteal vein. 1-skin. 2-common femur (thigh) vein. 3-muscles. 4-aponeurosis (fascia). 5-great saphenous (subcutaneous) vein. 6-perforating vein. 7-superficial femur vein. 8-popliteal vein. 9-small saphenous (subcutaneous) vein. 10-shin veins. 11-deep communicant veins. 12-perforating veins between small saphenous (subcutaneous) vein and deep shin veins (Geza and Perter, 2007).

In the lower part of its course, it is medial to the popliteal artery between the heads of gastrocnemius. It is superficial to it. And above the knee joint it is posterolateral to it (Dodd and Cockett, 1996).

Its tributaries are the small saphenous vein, veins corresponding to branches of the popliteal artery and muscular veins (Kobak and Lev, 1995).

II. Deep veins of the thigh:

The femoral vein is the continuation of the popliteal vein, accompanies it is artery at the adductor canal. In the distal adductor canal, it is posterolateral to the femoral artery; more proximally in the canal, and in the distal femoral triangle (i.e., its apex), it is posterior to it; at the triangle's base it is medial. The vein occupies the middle compartment of the femoral sheath, between the femoral artery and canal, fat in the latter allowing expansion of the vein (Gabella, 1995).

The profunda femoris vein lies in front of the artery and separates it from the adductor longus and the superficial femoral vein. The union of the superficial and profunda femoris is about 2 to 3cm below the bifurcation of the common femoral artery (Gabella, 1995).

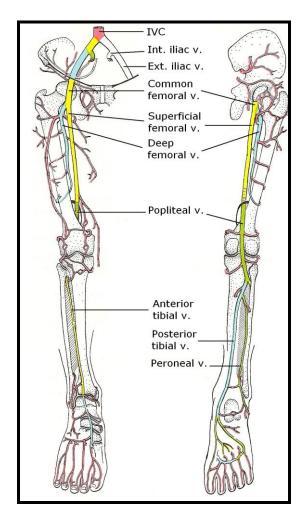


Figure (3): Deep veins of the lower limb (Havor and Galloway, 1997).

The common femoral vein lies medial and slightly behind the common femoral artery. A number of tributaries enter either the superficial femoral vein or the common femoral vein, (Fig. 3) usually the later. These are the long saphenous vein, medial and lateral circumflex femoral, deep external pudendal and many muscular veins (Gabella, 1995).

Anatomy of the iliac veins

I. The external iliac vein:

The proximal continuation of the femoral vein is the external iliac; it thus begins posterior to the inguinal ligament, ascends the pelvic brim and ends anterior to the sacro-iliac joint by joining the internal iliac to form the common iliac vein (Gabella, 1995).

II. The internal iliac vein:

This large vein is formed in the floor of the true pelvic cavity by the union of the gluteal, internal pudendal and obturator veins which originate in the buttocks, thigh and perineum. It ascends posteromedial to the internal iliac artery to join the external iliac vein, forming the common iliac at the pelvic brim, anterior to the lower part of the sacro-iliac joint. It is covered anteromedially by parietal peritoneum (Negus, 1995).

III. The common iliac vein:

The vein has a short and wide trunk, which passes upwards from the sacroiliac joint to end on the right side of the fifth lumber vertebra by uniting with its fellow to form the inferior vena cava. The right common iliac vein and the inferior vena cava run in a more or less straight line from the pelvis upwards, while the left common iliac vein joins it nearly at a right angle (Fig. 4) (Negus, 1995).

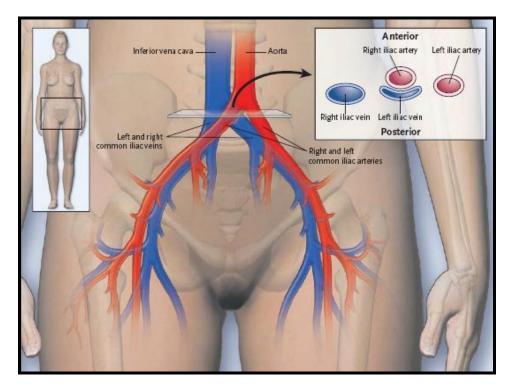


Figure (4): A pictorial representation of the anatomy of the iliac veins and arteries (Negus, 1995).

C-The perforating and communicating veins

The perforating veins connect the deep veins with the superficial veins and direct the flow from the superficial to the deep system. There are numerous perforators in the leg. Perforators are named after their locations. Major groups classify perforators according to their longitudinal location as ankle, leg, knee, and thigh perforators. Subgroups indicate side (i.e., anterior, posterior, medial, and lateral perforators). Thus, the complete name of the perforator is a combination of the level and side (i.e., the medial leg perforator or the anterior thigh perforator). More