

**Comparative study between medical
treatment and intervention in acute ilio-
femoral DVT**

Master essay
Submitted for partial fulfillment of master degree in
general surgery

Submitted by

Mahmoud Fekry Mahmoud Ashour

M.B., B. Ch. ,2010

Faculty of Medicine – Cairo University

Under the supervision of

Prof. Dr. Rafik Ramsis Morcos

Professor of General Surgery,
Faculty of medicine Ain Shams University

Dr. Abd El Rahman Mohamed Ahmed

Lecturer of general surgery and vascular surgery,
Faculty of medicine Ain Shams University

AIN SHAMS UNIVERSITY

2015

Acknowledgment

I would like to show special gratitude to my thesis supervisors, who incited me to strive towards my goal, for your assistance and guidance with my Masters essay.

I would like to express my special appreciation to all my seniors; you have been tremendous mentors for me. I would like to thank you for encouraging me. Your advice on my career has been priceless.

Furthermore, special thanks to my family. Words cannot express how grateful I am to my mother and father for all of the sacrifices that you've made on my behalf. Your prayers for me were what sustained me that far.

At the end I would like to express my appreciation to my beloved wife for her love, kindness and support she has shown during the period it has taken me to finalize this thesis.

Many Thanks,

Mahmoud Fekry Ashour.

Index

• Introduction.	1
• Aim of the study.	6
• Anatomy of lower limb venous system.	7
• Deep and superficial venous systems of the foot.	8
• Deep and superficial venous system of the leg and thigh.	10
• Deep venous system of thigh.	22
• Veins of the pelvis.	25
• Inferior vena cava.	34
• Surface anatomy.	40
• Pathophysiology of acute iliofemoral deep venous thrombosis.	43
• Incidence.	44
• Venous muscle pump.	44
• Thrombogenesis.	46
• Risk Factors.	58
• Complications.	81
• Diagnosis of acute iliofemoral deep venous thrombosis.	88
• Clinical assessment.	89
• Diagnostic tests.	90
• Laboratory Investigations.	91
• Radiological Investigations.	93
• Methods Of Diagnosis.	102
• Diagnostic Strategies for Symptomatic DVT.	105
• Diagnosis of Acute Deep Venous Thrombosis.	107
• Management of acute iliofemoral deep venous thrombosis.	110
• Prevention of venous thromboembolism.	112
• General Measures.	115
• Mechanical Methods of Prophylaxis.	115
• Pharmacologic Methods of Prophylaxis.	121

Index

- Combination of Mechanical and Pharmacologic Methods. 131
- Recommendations for Prophylaxis of Venous Thromboembolism in Surgical Patients. 131
- Treatment of Deep Venous Thrombosis. 135
 - Initial Treatment of Deep Venous Thrombosis. 135
 - Long-Term Treatment of Deep Venous Thrombosis. 146
 - Acute Deep Venous Thrombosis: Surgical and Interventional Treatment. 147
 - Choosing the appropriate treatment option. 176
 - Published Guidelines. 179
- Summary. 181
- Recommendations. 187
- References. 189

List Of Abbreviation

- **ACCP:** American college of chest physicians.
- **aPTT:** activated partial thromboplastin time.
- **AVF:** arteriovenous fistula.
- **BMI:** body mass index.
- **CDT:** catheter-directed thrombolysis.
- **COPD:** chronic obstructive pulmonary disease.
- **COX:** cyclooxygenase enzyme.
- **CT:** Computed Tomography.
- **CTV:** Computed Tomographic venography.
- **CVD:** chronic venous disease.
- **DVT:** deep venous thrombosis.
- **ELISA:** enzyme linked immunosorbent assay.
- **FDA:** Food and Drug Administration.
- **GCSs:** Graduated compression stockings.
- **HIT:** Heparin-induced thrombocytopenia.
- **INR:** international normalized ratio.
- **IPC:** Intermittent pneumatic compression.
- **IVC:** inferior vena cava.
- **ISPMT:** isolated segmental pharmacomechanical thrombolysis.
- **LMWHs:** Low-Molecular-Weight Heparins.
- **MRI:** magnetic resonance imaging.
- **MRV:** Magnetic resonance venography.
- **MTHFR:** methyltetrahydrofolate reductase.
- **PAI-1:** plasminogen activator inhibitor-1.
- **PE:** pulmonary embolism.
- **PTS:** post thrombotic syndrome.
- **QoL:** quality of life.

List Of Abbreviation

- **Rt-PA:** recombinant tissue plasminogen activator.
- **THR:** total hip replacement.
- **UFH:** unfractionated heparin.
- **VKAs:** vitamin k antagonists.
- **VTE:** venous thromboembolic disease.

List Of Figures

Figure Num.	Description	page
Figure 1.1	Overview of veins of the lower limb.	3
Figure 1.2	Transverse section through the left leg, 6 cm proximal to the tip of the medial malleolus	6
Figure 1.3	The left saphenous opening, after removal of the cribriform fascia.	8
Figure 1.4	The long saphenous vein and its tributaries	12
Figure 1.5	Anatomy of the veins of the lower extremity. A, Superficial veins and perforators, anterior view. B, Superficial veins, posterior view. C, Deep veins, anterior view.	14
Figure 1.6	Popliteal Vein	16
Figure 1.7	Structures passing beneath the left inguinal ligament.	17
Figure 1.8	The left femoral vessels	18
Figure 1.9	External Iliac Vein	21
Figure 1.10	Veins of the female pelvis	24
Figure 1.11	Tributaries of the inferior vena cava and lumbar veins. Only the left lumbar venous system is shown, for clarity	27
Figure 1.12	The abdominal aorta, inferior vena cava and their branches in the male. The fascia, lymphatics and connective tissue have been removed for clarity	30
Figure 1.13	Inguinal region (bones and soft tissues) and femoral triangle (vessels and nerves)	35
Figure 2.1	Action of the calf muscle pump.	39
Figure 2.2	Thrombogenesis. Virchow's triad	42
Figure 2.3	Regulation of coagulation pathway. PAI-1, plasminogen activator inhibitor-1.	46
Figure 2.4	Natural history of venous thrombosis.	47
Figure 2.5	Origin of thrombus and its propagation.	51

List Of Figures

Figure 2.6	Mechanism of thrombosis in inherited thrombophilia.	57
Figure 2.7	Hemodynamic consequences of pulmonary embolism.	76
Figure 3.1	Venous thrombosis.	88
Figure 3.2	Venogram of a patient with intramedullary nail for fractured tibia, showing multiple filling defects in the calf veins consistent with deep venous thrombosis.	91
Figure 3.3	Computed tomography of a patient with deep vein thrombosis of the left femoral vein (arrow). The vein is not opacified or distended in comparison to the right femoral vein. Extensive subcutaneous edema appears on the left.	93
Figure 3.4	Magnetic resonance image of deep vein thrombosis.	95
Figure 3.5	An example of a diagnostic algorithm for evaluation of symptomatic patients with low probability of deep venous thrombosis (DVT).	102
Figure 3.6	An example of a diagnostic algorithm for evaluation of symptomatic patients with high probability of deep venous thrombosis (DVT).	103
Figure 4.1	Surgical repair of venous thrombosis.	161
Figure 4.2	After infrainguinal balloon catheter thrombectomy, flushing of the infrainguinal venous system with a heparin-saline solution is performed by placing a large red rubber catheter into the proximal posterior tibial vein and flushing vigorously with a bulb syringe.	163
Figure 4.3	Caval thrombectomy can be performed with a protective balloon catheter inflated above	165

List Of Figures

	the thrombus as an alternative to vena caval filtration if a clot is present in the vena cava.	
Figure 4.4	A, Placement of a piece of polytetrafluoroethylene or Silastic around the saphenous arteriovenous fistula. A large permanent monofilament suture is looped and clipped, with approximately 2 cm left in the sub-cutaneous tissue. B, Placement of a small infusion catheter (pediatric feeding tube) into the wound via a separate stab incision in the skin. It is inserted and fixed in the proximal posterior tibial vein.	168
Figure 4.5	Algorithm illustrating our current treatment protocol for patients with iliofemoral deep venous thrombosis (DVT). CD, Catheterdirected; PM, pharmacomechanical.	172

List Of Tables

Table Num.	Description	page
Table 2.1	<i>Causes of Hypercoagulapility</i>	52
Table 2.2	Incidence of inherited thrombophilia in normal subjects and patients who have venous thrombo-embolic disease (VTE)	53
Table 2.3	Incidence of venous thromboembolism in patients following surgery or trauma	60
Table 3.1	Modified wells criteria for DVT	98
Table 4.1	Caprini venous thromboembolism (VTE) risk assessment model. BMI, Body mass index; COPD, chronic obstructive pulmonary disease; DVT, deep venous thrombosis; PE, pulmonary embolism.	107
Table 4.2	Contraindication and precautions for the use of anticoagulant agents for venous thromboembolism prophylaxis	115
Table 4.3	Recommended Prophylaxis in Surgical Patients According to the Estimated Level of Risk in Patients Undergoing General Surgery	126
Table 4.4	Recommended Regimens and Doses for Pharmaco-logical Prophylaxis of Postoperative Venous Thrombo-embolism.	128
Table 4.5	Nomogram for Adjusting the Dose of Intravenous Heparin Based on the Results of the Activated Partial Thrombo-plastin Time.	131
Table 4.6	Dose of Low-Molecular-Weight Heparins (LMWHs) for Venous Thromboembolism	135
Table 4.7	Contraindications to pharmacologic thrombolytic agents.	146
Table 4.8	Dosing of Currently Available Thrombolytic Agents for Venous	151

List Of Tables

	Thrombosis.	
Table 4.9	OVERVIEW OF THE TECHNIQUE OF CONTEMPORARY VENOUS THROMBECTOMY	159

Introduction

The complications of acute venous thromboembolism (VTE) including deep venous thrombosis (DVT), pulmonary embolism (PE), and post-thrombotic syndrome, are important as they are most common preventable causes of hospital death. **(Hull RD, et al., 1986).**

Recurrent thrombotic events appear to compete with recanalization early after acute DVT. The importance of such events is well recognized clinically, although their frequency in patients studied prospectively has only recently been described. Most clinical studies have included both recurrent DVT and PE, with rates depending on treatment, location of the thrombus, and duration of follow-up. In patients with proximal DVT, recurrent thromboembolic events have been reported in 5.2% of those who were treated with standard anticoagulation measures for 3 months as compared with 47% in those who were inadequately treated with a 3-month course of low-dose subcutaneous heparin. Proximal propagation may also complicate isolated calf vein thrombosis in up to 23% of untreated patients. **(Philbrick JT, et al., 1988).**

PE, with its attendant mortality, is the most devastating complication of acute DVT. When associated with acute DVT, the majority of PE episodes may be clinically silent. In patients with symptomatic DVT 50% to 80% have evidence of asymptomatic PE. Conversely, in those with symptomatic PE, asymptomatic DVT can be demonstrated in about 80% of cases. **(Buller HR, et al., 2005).**

Introduction

Though less dramatic than PE, development of post thrombotic syndrome is the most important late complication of acute DVT, and it is responsible for a greater degree of chronic socioeconomic morbidity. As many as 29% to 79% of patients may have some degree of long-term manifestations such as pain, edema, heaviness, or hyperpigmentation, but severe manifestations occur in only 7% to 23%, while ulceration occurs in 4% to 6% of patients. **(Meissner MH, et al., 2007).**

The most common symptoms and signs of DVT have a wide range of reported sensitivities and specificities. For example, calf pain has a sensitivity of 75% to 91% and a specificity of 3% to 87%, and calf swelling has a sensitivity of 35% to 97% and a specificity of 8% to 88%. **(Anderson Jr FA, et al., 2007).**

Duplex ultrasonography has almost completely replaced Contrast Venography as the diagnostic test of choice for the detection of DVT. The benefits of duplex ultrasonography over Contrast Venography include a lack of radiation, portability, noninvasiveness, and relative cost effectiveness. In addition, ultrasound has the ability to distinguish among nonvascular pathologies, such as inguinal adenopathy, Baker's cyst, abscess, and hematoma. The main concern regarding ultrasound is whether its diagnostic ability is comparable to that of Contrast Venography. **(Goodacre S, et al., 2005).**

Introduction

The objectives in treatment of acute DVT are to prevent thrombus extension, early recurrence, and death from PE. In addition, treatment should attempt to prevent late recurrences and long-term consequences such as the development of Post Thrombotic Syndrome and chronic pulmonary hypertension. Since the landmark paper by Barrit and Jordan approximately 60 years ago, anticoagulation remains the mainstay of VTE treatment. In most cases, the initial treatment of DVT and PE is similar. Indeed, as many as 20% to 25% of patients with symptomatic DVT have asymptomatic PE when routine imaging techniques are performed. (**Eldor A., 2001**).

Patients with VTE should be treated with anticoagulants as soon as the diagnosis is confirmed by objective imaging techniques. However, if clinical suspicion is very high, treatment should be initiated until the diagnosis can be confirmed. There are two options for the initial anticoagulant treatment of VTE: (1) intravenous or subcutaneous UFH and (2) subcutaneous LMWHs. The current recommended approach is to start heparin or LMWH and VKAs together at the time of diagnosis and to overlap them for 5 to 10 days, with UFH or LMWH discontinued when the prothrombin time, expressed as the INR, is within the target (2.0 to 3.0) for 2 consecutive days. (**Pabinger I, et al., 1994**).

The basic mechanism of thrombolysis is the activation of fibrin-bound plasminogen and resultant production of plasmin. (**Sandoval JA, et al., 2008**).

Introduction

It is intuitive that delivery of the plasminogen activator within the thrombus would be more effective and potentially safer than systemic infusion of plasminogen activators. Additionally, intrathrombus delivery protects plasminogen activators from circulating plasminogen activator inhibitor and, more importantly, protects the active enzyme plasmin from neutralization by circulating antiplasmin. If lysis can be accelerated, the overall dose and duration of plasminogen activator infusion should be reduced, thereby decreasing the risk for treatment related complications. (**Sandoval JA, et al., 2008**).

Isolated Segmental Pharmacomechanical Thrombolysis. An interesting new technique is isolated segmental pharmacomechanical thrombolysis (ISPMT), which is achieved by using the Trellis catheter (Bacchus Vascular, Santa Clara, CA). This double-balloon catheter is inserted into the thrombosed venous segment with the proximal balloon positioned at the upper edge (cephalic end) of the thrombus. When the balloons are inflated, plasminogen activator is infused into the thrombosed segment isolated by the balloons. The intervening catheter assumes a spiral configuration and spins at 1500 rpm for 15 to 20 minutes. The liquefied and fragmented thrombus is aspirated and treatment successes evaluated by repeat segmental phlebography. (**Binkert CA, et al., 2006**).