Management of Post Thrombotic Limb

Essay
Submitted for partial fulfillment of master degree in ***general surgery***

Presented by:

Ramez Onsy Kamel
M.B.B.Ch Cairo University

Supervised by:

Prof. Dr. Hassan Sayed Tantawy
Professor of General Surgery
Faculty of Medicine- Ain shams university

Prof. Dr. Mohamed Ahmed Aamer
Professor of General Surgery
Faculty of Medicine- Ain shams university

Dr. Moheb Shoraby Eskandaros
Lecturer of General Surgery
Faculty of Medicine- Ain Shams University

Faculty of Medicine
Ain Shams University
2016

Acknowledgement

I would like to express my gratitude and appreciation to all those who gave me the possibility to complete this Essay.

A special thanks to ***Prof Dr. Hassan Sayed Tantawy,*** whose help, stimulating suggestions and encouragement, helped me to coordinate my Essay. I would also like to acknowledge with much appreciation the crucial role of ***Prof Dr Mohamed Ahmed Aamer, and Dr Moheb Shoraby Eskandaros*** who helped me to assemble the parts and gave suggestion for my essay.

Finally, I would like to thank my parents. Without their support I wouldn't have been able to be in this place.

My sincere gratitude to God, above All.

Dr Ramez Onsy

Contents

List of Abbreviations i

List of Tables ii

List of Figures iii

1. Introduction and Aim of the Work 1

2. Review of literatures

Anatomy of venous system of the lower limb 5

Pathophysiology of post thrombotic syndrome 29

Diagnosis of post thrombotic syndrome 44

Treatment of post thrombotic syndrome 57

3. Summary 95

4. References 97

5. Arabic summary ..... --

List of Abbreviations

ACCP	American college of chest physicians
AHA	American heart association
CDT	Catheter directed thrombolysis
CTO	Complete total occlusion
CTV	Computed tomography venography
DUS	Duplex ultra sound
DVT	Deep venous thrombosis
GCSs	Graduated compression stockings
GSV	Great saphenous vein
HIT	Heparin induced thrombocytopenia
IPC	Intermittent pneumatic compression
IVC	Inferior vena cava
IVUS	Intra vascular ultra sound
LMWH	Low molecular weight heparin
MRV	Magnetic resonance venography
PE	Pulmonary embolism
PTS	Post thrombotic syndrome
SSV	Small saphenous vein
SVC	Superior vena cava
TPA	Tissue plasminogen activator
UFH	Unfractionated heparin
VCT	Valve closure time
VKA	Vitamin K antagonist
VTE	Venous thrombo embolism

List of tables

Table	*Title*	*Page*
1	Changes in nomenclature of superficial and deep veins of the leg	26
2	Villalta PTS scale	46
3	Risk of venous thromboembolism in surgical patients without thromboprophylaxis	60
4	Caprini venous thromboembolism (VTE) risk assessment model	61
5	Comparison of the features of the new oral anticoagulants.	69
6	Dosing of currently available thrombolytic agents for venous thrombosis	82
7	Overview of technique of venous thrombectomy	85

List of Figures

Fig.	*Title*	*Page*
1	IVC anatomy	7
2	Emryological Development of the IVC	9
3	Anatomical variations of IVC, Iliac veins and its tributaries	12
4	Venous system of renal area	14
5	IVC collaterals	16
6	The SSV and lateral venous system of the calf	20
7	Schematic representation of the topography of the main groups of perforating veins (PVs)	23
8	L.L superficial and deep veins	28
9	Hypothetic basic mechanism is shown of early and later deep venous thrombosis-mediated vein wall injury of the whole vein.	31
10	The pathogenesis of chronic venous insufficiency	37
11	Ascending venogram opacifies only superficial network	48
12	Comparison of various modalities for detection of venous disease	54
13	Images obtained by venous intravascular ultrasound (IVUS)	56
14	Algorithm for management of ilio-femoral DVT	78
15	Sixty-five-year-old man presenting with right iliofemoral thrombosis**,** with massive swollen right lower limb in comparison to left	79

List of Figures

Fig.	*Title*	*Page*
16	Venograms of the IVC before and after thrombolysis and stenting	82
17	Venograms show left common and external iliac veins filled with thrombus (a), after catheter directed thrombolysis (b) and after intravenous stent insertion (c). An inferior vena cava filter (white arrow) was placed in this patient.	83
18	Contralateral iliocavagrams show nonocclusive thrombus in the vena cava	86
19	Surgical repair of venous thrombosis	87
20	Caval thrombectomy can be performed with a protective balloon	88
21	iliofemoral post-thrombotic obstruction before and after stenting	93

Abstract

Post-thrombotic syndrome (PTS) is a chronic disease following DVT , typical symptoms include pain, heaviness, swelling, and cramping of the leg, with significant impacts on patient quality of life and health-care burden, At each step of the treatment pathway in patients with acute DVT, options currently exist to reduce PTS, if applied. Treatment options include rapidly initiated and sustained therapeutic anticoagulation at early stage, leg compression, early ambulation and catheter directed thrombolysis.

K-Words

Post-thrombotic syndrome, deep venous thrombosis, reflux, valvular incompetence, venous insufficiency, duplex ultrasound, catheter-directed thrombolysis.

Introduction

Post-thrombotic syndrome (PTS) is a chronic disease following DVT, with significant impacts on patient quality of life and health-care burden. The precise etiology is unknown, though there are identified risk factors that increase the risk of developing PTS **(Wittens and Strijkers, 2014).**

Typical symptoms include pain, heaviness, swelling, and cramping of the leg, which are generally worsened by standing and exercising. Venous ulceration occurs in advanced cases, causing additional pain and disability and increasing the cost of treatment **(Henke et al., 2011)**.

Venous hypertension caused by a persistent venous obstruction, valve incompetence, or a combination of the two is considered to be the major cause of PTS. It is unclear whether an obstruction, reflux, or a combination of the two provides the main contribution to PTS **(Haenen et al., 1999).**

Biomarkers can be of use in further exploring the etiology as well as in developing risk stratification tools for PTS. The relationship between PTS and specific biomarkers may help guide prevention and therapy based on a patient's individual risk profile **(Bouman et al., 2014)**.

In a cohort study of patients with acute DVT, patients who developed PTS reported lower physical quality-of-life scores than the scores generally reported by patients of similar ages with arthritis and diabetes. Those who developed severe PTS had scores similar to those reported by patients with angina, cancer, and congestive heart failure. The costs associated with PTS are substantial, as demonstrated by studies conducted in Sweden and the United States. There are also important indirect costs associated with lost productivity; an estimated 2 million workdays are lost annually in the United States because of venous ulcers **(Cohen et al., 2012)**.

Post-thrombotic morbidity has been reported to occur in 25% to 46% of patients after anticoagulation alone for acute DVT. This was highlighted by a prospective study of a multicenter cohort of 387 patients with acute DVT. PTS developed in almost half of all patients' ≤2 years, with severe symptoms, including venous ulceration, developing in 3% **(Cohen et al., 2012)**.

PTS is more difficult to quantify. The best agreed-upon definition is the Villalta score, combining patient limb symptoms and signs in a graded scoring system. The higher

the score, the greater the severity of PTS **(Henke et al., 2011)**.

At each step of the treatment pathway in patients with acute DVT, options currently exist to reduce PTS, if applied. Treatment options include rapidly initiated and sustained therapeutic anticoagulation at early stage, leg compression, early ambulation and catheter directed thrombolysis **(Henke et al., 2011).**

Aim of the Work

The aim of the work is to highlight the cause, diagnosis and new modalities in management of post thrombotic syndrome

Anatomy

- **The Inferior vena cava normal anatomy**

The IVC, the largest vein in the body, has no valves except for a variable, non-functional one at its orifice in the right atrium of the heart. The IVC returns poorly oxygenated blood from the lower limbs, most of the back, the abdominal walls, and the abdomino-pelvic viscera. Blood from the abdominal viscera passes through the portal venous system and the liver before entering the IVC via the hepatic veins **(Moore et al., 2014).**

The IVC typically has an oval shape in cross- section but is easily deformed by adjacent abdominal or retroperitoneal masses. The average diameter of the infra renal IVC is approximately 23mm, although the infra renal segment is usually slightly larger. The IVC is an elastic structure that responds to decrease volume or increased intra abdominal pressure by collapsing. The dynamic nature of the IVC should be considered when interpreting imaging studies or interventions **(Kaufman and Lee, 2014).**

The inferior vena cava (IVC) begins at the confluence of the common iliac veins and ascends on the right side of the vertebral column, passes through the tendinous portion of the

diaphragm, and after a short course (approximately 2.5 cm) in the chest it terminates in the right atrium at the level of T9**(Moore et al., 2014).**

In the upper abdomen the IVC is located posterior to the duodenum, the head and neck of the pancreas, the lesser sac, and the liver. The intra hepatic portion of the IVC lies in a groove along the posterior aspect of the caudate **(Moore et al., 2014)**.

Tributaries of the IVC are the paired lumbar and renal veins and the hepatic veins, additionally on the right side the right gonadal, suprarenal, and inferior phrenic veins also drain into the IVC. The left gonadal and suprarenal veins join the left renal vein, the left inferior phrenic vein drains into the left suprarenal vein **(Moore et al., 2014).**

Paired parietal branches of the IVC include the inferior phrenic veins, the 3rd (L3) and 4th (L4) lumbar veins, and the common iliac veins. The ascending lumbar and azygos veins connect the IVC and SVC, either directly or indirectly providing collateral pathways **(Moore et al., 2014).**