



**RECENT STUDIES IN MANAGEMENT OF PORTAL VEIN
THROMBOSIS**

Essay submitted for partial fulfillment of Master
Degree in General surgery

By

Mohamed Megahed Megahed Ziehery

M.B.B.Ch Ain Shams University 2011

Under Supervision of

Prof. Dr. / Alaa Eldin Ismail Abd Almotelib

Professor of General surgery Faculty of medicine

Ain Shams University

Dr. / Ahmed Gamal Eldin Osman

Lecturer of General surgery Faculty of medicine

Ain Shams University

Dr. / Mohamed Abd Elsattar Abd Elhamid

Lecturer of General surgery Faculty of medicine

Ain Shams University

Faculty of Medicine

Ain Shams University

2016

ACKNOWLEDGEMENT

*I wish to express my sincere gratitude and profound thanks to **Prof Dr. Alaa Eldin Ismail Abd Almotelib**, Professor of general surgery, Faculty of Medicine, Ain Shams University, for his kind supervision, time, effort and indispensable remarks throughout this work.*

I would like to offer my appreciation and thanks Dr. / Ahmed Gamal Eldin Osman and Dr. / Mohamed Abd Elsattar Abd Elhamid for their guidance, help and encouragement throughout the progress of the work.

Lastly, but not the least, I would like to thank all my family members for their kind advice and support.

Mohamed Megahed Megahed Ziehery

Abstract

Portal vein thrombosis is a life threatening vascular disorder of the liver. I will review the recent advance regarding the epidemiology, etiology, management, and prognosis of portal vein thrombosis.

List of contents

ACKNOWLEDGEMENT	2
List of contents	3
List of figures	5
List of tables	9
Abbreviations	10
Keywords	12
Introduction	13
Aim of the work	17
Applied anatomy of portal vein	18
Pathology & risk factor of PVT	28
Classification of PVT	36

Clinical presentation of PVT	39
Recent methods for diagnosis	44
Methods of Management of PVT	64
Complication of PVT	103
Summary	113
Reference	119
Arabic Summary	131

List of figures

Figure 1 :Development of vitelline and umbilical veins(Bhargava, et al .2014).....	20
Figure 2: Lt&Rt vitelline V.(Keplinger, et al. 2014).	21
Figure 3: caudal to septum transversum (Keplinger, et al. 2014).	22
Figure 4:formation of portal vein (Bhargava, et al. 2014).....	23
Figure 5: The portal vein and its tributaries (Van Steenkiste, <i>et al</i> .2010)	24
Figure 6:Porto- systemic anastomosis all over the body (Moubarak, <i>et al.</i> 2012).	26
Figure 7:variation of portal vein anatomy (Varotti, et al. 2004). 27	
Figure 8: variation of portal vein anatomy(Gallego, et al. 2002).28	
Figure 9: classification of portal vein thrombosis (Rajesh, et al .2015).	38
Figure 10:yerdl classification of portal vein thrombosis(Groeschl,et al 2016).	38
Figure11:classification of portal vein thrombosis (Groeschl,etal.2016).	39
Figure 12:(a)and (b)Gray-scale US image showing thrombosed left portal vein (arrow in (a)). On application of color Doppler (b), hypertrophy of the accompanying branch of hepatic artery can be seen (black arrow in (b)) with opening up of peri portal collateral venous channels (white arrow) (Jayaprakasan, et al.2014).	47
Figure 13: contrast-enhanced US (a) and gray-scale image (b) demonstrating absence of enhancement of the portal vein thrombus in the arterial phase (arrow in (a)) signifying benign nature of the thrombus (M.Dˆanilˆa,et al.2011).....	48

Figure 14: Axial NCCT (a) and CECT (b) images demonstrating mildly hyper dense thrombus occluding the main portal vein (arrows).Corresponding images at a caudal level in the same patient showing hyper dense thrombus in the SMV with associated fat stranding in the adjoining mesentery (Martegani19,et al.2014).....	51
Figure 15: Axial CECT images obtained in the arterial (a) and venous (b) phases showing an abscess in the left lobe (asterisk) which had caused acute thrombosis of the left portal vein (pylephlebitis). Associated hepatic artery buffer response is seen in the form of increased enhancement of the left hepatic lobe in the arterial phase (arrows in (a)) which becomes essentially isodense on the portal venous phase (Martegani19, et al.2014)..	52
Figure 16:Coronal oblique CECT image of a patient with acute necrotizing pancreatitis demonstrates thrombosed splenic vein (thick white arrows) and a segmental branch of right portal vein (thin white arrow) with hepatic artery buffer response in the form of differential hyper enhancement of the affected liver segment (black arrows) (Martegani19,et al.2014).	52
Figure 17Axial T2-weighted MRI image demonstrating mildly hyper intense thrombus (arrow) in the right portal vein (Martegani19, et al.2014).....	53
Figure 18:Coronal oblique CECT image demonstrating thrombosed portal vein as well as the SMV (arrows) with rim enhancement of their walls(Martegani19,et al.2014).	53
Figure 19 Axial CEMRI images obtained in the arterial (a) and venous (b) phases showing a lobulated lesion showing arterial phase enhancement (asterisk in (a)) with washout of contrast on the venous phase. Associated enhancing right portal vein tumor thrombus (arrows) is present (Reddy, et al.2008).....	56

Figure 20: Gray-scale US (a) image showing replacement of the main portal vein by an ill-defined echogenic area containing multiple subtle anechoic tubular structures. On application of colour Doppler (b) turbulent flow can be seen within these anechoic structures consistent with portal cavernoma (Jayaprakasan, et al.2014).....	57
Figure 21 Coronal oblique CECT image (a) showing multiple paracholedochal collaterals (solid black arrows) causing extrinsic compression over the CBD (interrupted arrow). (b) 2D MRCP image of the same patient demonstrating undulating margins of CBD (arrow) due to the compression(Y.-H. Gai, et al. 2014).....	59
Figure 22: (a) Thick-slab 3D MRCP image of a patient with portal biliopathy demonstrating extrinsic vascular impression over CBD by the paracholedochal collaterals (solid arrows). The distal CBD is narrowed by these collaterals with resultant upstream biliary dilatation. Undulating margins of biliary system can also be seen (interrupted arrow) with a grossly distended gall bladder. (b) 3D MRCP image from another patient showing wavy contour of the mid- and distal CBD due to portal biliopathy with resultant narrowing and gross bilobar biliary dilatation(S. K. Sarin, et al.2006).....	61
Figure 23:Coronal oblique CECT image showing chronic, partially calcified, occlusive thrombus involving the main portal vein (black arrow) with multiple tortuous periportal collateral channels (solid white arrows). Splenic vein is also partially thrombosed (asterisk). Gall bladder calculi (interrupted arrow) and ascites can also be seen (R. de Franchis, et al.2010)	62

Figure 24 Axial CECT image of a patient with EHPVO showing multiple tiny paracholedochal collaterals appearing as continuous enhancement of one of the biliary radicals in right hepatic lobe (arrows) mimicking cholangiocarcinoma (pseudocholangiocarcinoma sign). Splenic infarct is also seen due to associated splenic vein thrombosis (interrupted arrow) along with ascites (asterisk) (Rajesh, et al. 2015).	62
Figure 25 Axial (a) and coronal (b) MIP images of a patient with liver cirrhosis and multifocal hepatocellular carcinoma demonstrating multiple thin streaks of arterial phase enhancement within the main portal vein (arrows in (b)) as well as its intrahepatic branches (arrows in (a)) consistent with tumor thrombus (threads-and-streaks sign) (Y.-H. Gai, et al. 2014.....	64
Figure 26: Angiography of the portal system after its punctures showing complete and extended thrombosis without cavernoma. (Fanelli, et al .2011).	90
Figure 27: Same patient as in figure after stent implantation (Viatorr), thrombus fragmentation, and thrombolytic therapy resulting in complete repermeation of the extrahepatic portal vein axis. The pigtail catheter remained in place for local treatment and control(Fanelli, et al .2011).	90
Figure 28: A - Venous anastomosis of the superior mesenteric vein; B - Venous anastomosis in the portal vein graft(Chen, et al. 2016)	102

List of tables

Table 1 Thrombotic risk factors with portal vein thrombosis (**Kumar**,et al 2001). 29

Table 2: Inherited and acquired risk factors for acute PVT (Plessier, et al.2012).

..... 34

Table 3: clinical presentation of PVT. 41

Table 4: Pharmacological properties of new oral anticoagulants: 76

Table 5 : Surgical shunting procedures, classified into selectivity 97

Abberviations

PVT: portal vein thrombosis

HCC: hepatocellular carcinoma

MTHFR: methylene tetrahydrofolate reductase

PAI:plasminogen activator inhibitor

SMVT: superior mesenteric vein thrombosis

EXPVO: extra hepatic portal vein obstruction

EUS: endoscopic ultra sonar

CUES: contrast enhanced ultra sonar

NCCT: non contrasted computed tomography

CE-MRI: contrast enhanced magnetic resonance imaging

ERCP: endoscopic retrograde cholangio-pancreatography

MRCP: magnetic resonance cholangio-pancreatography

APPT: activated thromboplastin time

HIT: heparin induced thrombocytopenia

UFH : unfraction heparin

LMWH: low molecular weight heparin

LBW: lean body weight

AJBW: adjusted body weight

NOACs : new oral anticoagulants

PPC: prothrombin complex concentrate

APCC: activated prothrombin complex

EST: endoscopic sclerotherapy

TIPS: trans jugular intrahepatic Porto systemic anastomosis

MELD: model for end stage liver disease

TPA: tissue plasminogen activator

TGF-B: transforming growth factor beta

PAR: protease activated receptors

SMA-SMV: superior mesenteric artery and vein

PH: portal hypertension

VB: variceal bleeding

VTE: venous thromboembolism

keywords

Portal vein thrombosis

Virchow's triad

Portosystemic anastomosis

Doppler and ultrasonar

Cavenoma

Variceal bleeding

Endoscopic ligation and sclerotherapy

Heparin and new oral anticoagulants

Transjugular intrahepatic portosystemic anastomosis

Surgical shunts

Liver transplantation

INTRODUCTION

The term portal vein thrombosis (PVT) refers to the complete or partial obstruction of blood flow in the trunk of portal vein, due to the presence of a thrombus in the vessel lumen (**Bayraktar and Harmanci**, 2006). This includes its right and left intrahepatic branches. It may even extend to the splenic or superior mesenteric veins or towards the liver involving intra hepatic portal branches (**Chawla**, et al. 2009).

PVT is considered a rare event. It can occur either in childhood or in adulthood, with the same incidence. The reported lifetime risk of developing PVT in the general population is approximately 1%. Its prevalence among cirrhotic patients ranges between 4.4%-15%, and is responsible for about 5%-10% of overall cases of portal hypertension (**Amitrano**, et al, 2004). Moreover, in patients with a hepatocellular carcinoma, the incidence of PVT rises to 10%-40%..the first case of PVT was reported in 1868 by Balfour and Stewart, describing a patient presenting splenomegaly, ascites, and variceal dilation (**Wang**, et al, 2005).

Several causes can be involved in the pathogenesis of PVT and, frequently, more than one coexist. A simple classification distinguishes between local (70%) and systemic (30%) risk factors, Inflammatory abdominal foci (such as appendicitis, diverticulitis,

inflammatory bowel disease pancreatitis, cholecystitis, hepatic abscesses, and cholangitis), liver cirrhosis or tumors, represent the most common local thrombotic risk factors. Malignancies, frequently of hepatic or pancreatic origin, are responsible for 21%-24% of overall cases of PVT. Direct vascular invasion, compression by tumor mass, or a hypercoagulable state are the mechanisms involved in neoplastic PVT development; hormonal factors might also play a role in this process, especially in men. PVT is common in patients affected by liver cirrhosis, with a risk related to the severity of the disease (Ponziani, et al, 2010). PVT can be classified also into four categories, depending on the extension: confined to the portal vein beyond the confluence of the splenic vein; extended to the superior mesenteric vein, but with patent mesenteric vessels; extended to the whole splanchnic venous system, but with large collaterals; or with only fine collaterals. This classification is useful to evaluate a patient's operability and clinical outcome. In fact, when thrombosis is extended to both portal and mesenteric veins, the risk of bowel ischemia is considerable and mortality high, despite a lower risk of variceal bleeding (Riva, et al, 2012).

Clinical presentation is different in the context of acute or chronic onset and depends on the development and the extent of a collateral circulation. Intestinal congestion and ischemia, with abdominal pain, diarrhea, rectal bleeding, abdominal distention,