

Management of Portal Vein Thrombosis in Living Donor Liver Transplantation

ESSAY For Partial Fulfillment of Master Degree of General Surgery

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علاج تجلط الوريد البابي الكبدي في مرضي زراعة الكبد

بحث توطئة للحصول على درجة الماجستير في الجراحة العامة مقدمة من

الطبيب/ إيهاب محمد الإمام علوان بكالوريوس الطب و الجراحة <u>تحت إشراف</u>

الأستاذ الدكتور: رفعت كامل أستاذ الجراحة العامة كلية الطب - جامعة عين شمس

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> كلية الطب جامعة عين شمس ٢٠١١

Conclusion

PVT is relatively uncommon in the general population, but is more frequent among cirrhotic patients and represents a "milestone" in the natural evolution of liver disease.

Local or systemic pro-thrombotic factors, alone or together, can play an important role in PVT pathogenesis, which is complex and different in each clinical context and in each patient.

The consequent changes in hepatic and splanchnic hemodynamic are responsible for a mild impairment in liver function, in absence of an overt liver disease, or can precipitate a preexistent metastable clinical status in cirrhotic patients.

Moreover, PVT might have indirect effects on other abdominal organs, causing intestinal ischemia and infarction, or predisposition to vascular neoformation and gastrointestinal bleeding.

الله المحالية

(سورة البقرة آية ٣٢)

Acknowledgment

First of all my deep grateful recognition and indebtedness is to **Allah**, the most merciful, the most gracious for helping us all to complete this work.

I would like to express my deepest gratitude of thanks and respect to **Prof. Dr Refaat Refaat kamel**, Professor of general surgery, ain shams university, for his fatherly encouragement, helpful supervision and continuous guidance during execution of this work.

I am also expressing my sincere appreciation and deepest gratitude to **Prof. Dr. Amr Ahmed Abd Elaal**, professor of general surgery, ain shams university, for his valuable support and advises, so to him I am deeply indebted.

My sincere gratitude to **Dr. Mahmoud Zakaria Elganzoury**, lecturer of general surgery, ain shams university, for his continuous supervision, valuable scientific observation and excellent guidance.

I will never forget the sincere encouragement and great help of my father, mother, sisters, my wife Eman and of course my little daughter **Salma**.

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List of Abbreviations

AASLD	American Association for Study of Liver Diseases
AT	Antithrombin
СРНТ	Cavoportal hemitransposition
СТ	Computerized tomography
ERCP	Endoscopic retrograde cholangiopancreaticography
EST	Endoscopic therapy
EUS	Endoscopic ultrasound
FISP	Fast imaging with steady state precession
HCC	Hepatocellular carcinoma
Нр	Horizontal part
INR	International normalization ratio
IVC	Inferior vena cava
LDLT	Living donor liver transplantation
LMWH	Low molecular weight heparin
LT	Liver transplantation
MOELD	Model for end stage liver disease
MPD	Myeloproliferative disease
MRI	Magnetic resonance imaging

MTHFR	Methylene tetrahydrofolate reductase
OLT	Orthotopic liver Transplantation
PC	Protein C
PS	Protein S
PNH	Paroxysmal nocturnal hemoglobinuria
PVT	Portal vein thrombosis
RAPV	Right anterior portal vein
RPPV	Right posterior portal vein
RPV	Right portal vein
SMV	Superior mesenteric vein
SPV	Splenic vein
TIPS	Transjugular intrahepatic portosystemic shunt
US	Ultrasonography

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INTRODUCTION



INTRODUCTION

Portal vein thrombosis (PVT) refers to complete or partial obstruction of blood flow in the portal vein due to the presence of a thrombus in the lumen of portal vein (*Bayraktar and Harmanci*, 2006).

Although in the general population PVT is considered a rare event, 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).

The first case of PVT was reported in 1868 by Balfour and Stewart describing a patient presenting splenomegaly, ascites and variceal dilation. Several etiological causes either of local or systemic origin might be responsible for PVT development. PVT clinical presentation is different in the context of acute or chronic onset and depends on the development and the extent of a collateral circulation (*Wang et al.*, 2005).

PVT can be classified into four categories (Yerdel's grading) depending on extension of PVT,

classification is useful to evaluate a patient's operability and clinical outcome.

1: PV minimally or partially thrombosed less than 50% of the vessel lumen.

2: More than 50% occlusion of the PV including total occlusion.

3: Complete thrombosis of both PV and proximal superior mesenteric vein.

4: Complete thrombosis of the PV as well as proximal or distal SMV. (*Jamieson*, 2000).

In the past, PVT was considered an absolute contraindication for liver transplantation because of the technical difficulties it entailed especially the inability to gain an adequate portal supply (*Gao et al.*, 2009).

The first successful liver transplant in a patient with a thrombosed portal vein was reported by Shaw in 1985 (Selvaggi et al., 2007).

Increased risk of PVT complications after liver Transplantation such as primary non function or dysfunction, thrombosis of the hepatic artery, relaparotomy, postoperative pancreatitis, sepsis, and renal failure, a poorer survival and a higher mortality (*Lendoire* et al., 2007).

PVT patients' rates of survival at one and 5 years after Orthotropic liver Transplantation (OLT) are equal as if once the peri-transplant period has been overcome, the future clinical destiny of recipients with or without a previous PVT could be overlapped. However among patients with PVT survival seems better in low grades of Yerdel classification. Transplantation at grade 1 PVT seems to carry results comparable to non-PVT patients (*Molmenti et al.*, 2007).

After liver transplantation PVT development is a rare but possible event especially in the early postoperative period, the incidence ranges between 1% and 2% (*Duffy et al.*, 2009).

Today thanks to great innovations in medical care, surgical techniques and radiological interventions, PVT has no longer to be considered a contraindication but only a disadvantage and in some cases might present a possible indication to liver transplantation (*Arcadipane et al.*, 2008).

The diagnosis of PVT can be quickly established by demonstrating the presence of solid material within the

portal vein lumen, ultrasonography and Doppler imaging usually the investigation of choice. Recently the endoscopic use of ultrasound (EUS) is more specific in PVT diagnosis (*Kocher and Himmelmann*, 2005).

Instead, CT scanning and magnetic resonance imaging (MRI) can easily obtain this information and in addition can estimate the impairment of the bowel and other adjacent organs. MR angiography has a high accuracy in the follow-up of the portal venous system before and after liver transplantation (*Ito and Siegel man*, 2000).

In PVT patients, liver function is typically conserved. Laboratory investigations will be normal or quite normal unless there is coexistence of a liver disease. However levels of prothrombin and other coagulation factors could be moderately decreased while D-dimer is usually increased (*Condat and Valla*, 2006).

A specific therapeutic management is mandatory to resolve portal vein obstruction and avoid serious complications, Anticoagulant therapy is the best way to obtain portal vein recanalization however there is no consensus on its application. Thrombolytic therapy is also effective to provide recanalization in acute PVT (*DeLeve et al.*, 2009).