Graft and patient survival in portal vein thrombosis in living donor liver transplantation

Thesis

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List of Abbreviations

AIT ..... Alanine aminotransferase

AST ..... Aspartate aminotransferase

CHD ..... Common hepatic duct

CT..... Computed tomography

DDLT ..... Decreased donor liver transplantation

DVT..... Deep venous thrombosis

EUS ..... Endoscopic ultrasound

GIT..... Gastrointestinal tract

GSV ..... Great saphenous vein

HCC ..... Hepatocellular carcinoma

ICU..... Intensive care unit

LDLT ..... Living donor liver transplantation

LHD..... Left hepatic duct

LPV ..... Left portal vein

LRV..... Left renal vein

MRI..... Magnetic resonance imaging

PHA..... Proper hepatic artery

PV..... Portal vein

PVT ..... Portal vein thrombosis

RHA ..... Right hepatic artery

SMV ..... Superior mesenteric vein

US..... Ultrasound

INTRODUCTION

Portal vein thrombosis (PVT) is a common complication of end-stage liver disease with an incidence of 0.6–16% in patients with well-compensated disease, increasing up to 35% in cirrhotic patients with hepatocellular carcinoma **(Kim et al., 2011).**

Portal vein thrombosis (PVT) is an obstruction of the portal vein trunk and/or its branches by a blood clot, which includes the splenic, superior mesenteric, and inferior mesenteric veins. It can present in a variety of conditions, including cancer, infections, myeloproliferative diseases, inflammatory conditions, following ablative therapy for hepatocellular carcinoma (HCC), and cirrhosis **(Kinjo et al., 2014).**

From a clinical point of view, there are two types of PVT;Acute: sudden formation of a thrombus within the portal vein, which was not detected during the previous biannual ultrasound. Occlusion may be complete or partial. Chronic (portal cavernoma): replacement of the normal portal vein by a network of

hepatopetal collateral veins. It functions as a portoportal shunt **(Kumar et al., 2015).**

All patients with confirmed PVT were retrospectively classified into four grades according to the extent of thromboses: Grade I: minimally or partially thrombosed PV, in which the thrombus is mild or, at the most, confined to < 50% of the vessel lumen with or without minimal extension into the superior mesenteric vein (SMV). Grade II showed > 50%occlusion of the PV, including total occlusion with or without minimal extension into the SMV. Grade III were complete thromboses of both PV and proximal SMV with an open distal SMV. Grade IV was complete thrombosis of the portal vein as well as the proximal and distal SMV **(Yerdel et al., 2000).**

Complications of PVT include variceal bleeding, failure of endoscopic control of bleeding, intestinal ischaemia (in patients with extension of the thrombus into the superior mesenteric vein), and portal biliopathy (causing partial or complete bile duct obstruction) **(Dhiman et al., 2007).**

Most patients with cirrhosis are diagnosed with asymptomatic PVT during routine ultrasound. The sensitivity and specificity of Doppler ultrasound are 89% and 92%, respectively, so it is the primary method of choice in this context. If Doppler ultrasound shows portal vein patency, no further studies are indicated **(Plessier et al., 2012).**

Enhanced computed tomography and magnetic resonance imaging are the best methods to assess the extent of the PVT. In addition, they provide information about the development of collateral circulation, the status of adjacent organs, and are indicated if intestinal ischaemia or HCC are suspected **(Kumar et al., 2015).**

The importance of treating a cirrhotic patient who is being considered for liver transplantation and has PVT is to achieve recanalization and thus achieve a physiological portal vein anastomoses and ensure portal flow to the graft. Transplanting in the presence of extensive portal vein thrombosis makes surgery more complex and is associated with higher morbidity and mortality **(Englesbe et al., 2010).**

To date, only few studies have evaluated the benefits of anticoagulation in individuals with cirrhosis. An obvious goal of anticoagulation is PV recanalization: when cirrhotic individuals with PVT are treated with anticoagulation, complete recanalization has been described in 33–45% while partial PV recanalization is observed in 15–35% of cases **(Tripodi et al., 2011).**

LDLT has emerged as the alternative life-saving treatment to DDLT. Over the past 2 decades, the number of LDLTs has steadily increased in many transplant centers, especially in Asia **(Lee et al., 2009).**

LDLT has the following advantages over DDLT: a shorter wait time, a shorter cold ischemic time, and a better organization of the surgery time **(Maluf et al., 2005).**

As adequate portal vein (PV) flow is essential for proper liver graft function following reports have introduced surgical procedures transplantation, many such as thrombectomy, jump graft implantation, renoportal anastomosis, porto caval hemitransposition, and PV arterialization to ensure sufficient portal blood flow to liver grafts **(D'Amico et al., 2013).**

The separation between occlusive and non-occlusive thrombosis is very important; in patients with partial PVT, post-transplant mortality outcomes are no different from non-PVT patients but it is significantly increased in patients with complete PVT **(Rodriguez-Castro et al., 2012).**

Aim of the Work

The aim of this study is to assess impact of preoperative PVT on the patient and graft survival post LDLT.

Surgical Anatomy of the Liver

The human liver is the largest solid organ of the body. The principal landmarks defining liver anatomy include the falciform ligament, umbilical fissure, gallbladder fossa, and transverse hilar fissure. These landmarks delineate four lobes (Figure 1): left (medial to falciform), right (lateral to falciform), quadrate, and caudate (spigelian) **(Standring, 2015)**.

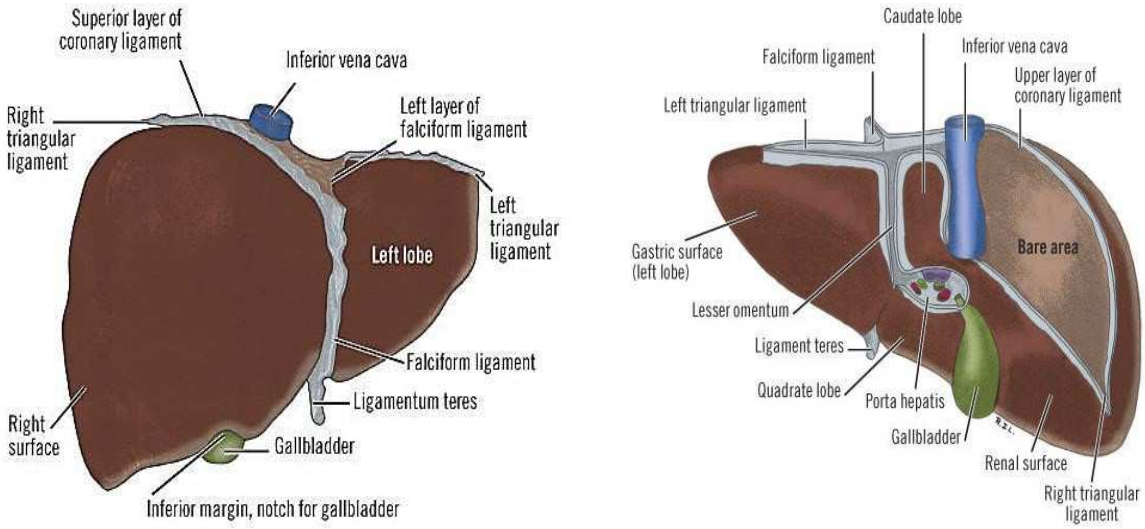

Figure (1): Surgical anatomy of the liver **(Macchi et al., 2013)**.