INTRODUCTION

The first human liver transplantation was performed in 1963 by a surgical team led by Dr. Thomas Starzi in United States. Dr. Starzi performed several additional transplantation operations over next few years before the first short-term success was achieved in 1967. In 1980 liver transplantation was considered as a standard clinical treatment for patient with end stage liver disease (ESLD). Now liver transplantation is performed in many centers with improvement of the outcome of the operation ***(Munoz et al., 2000).***

Patients should be considered for liver transplantation if they have evidence of fulminant hepatic failure; a life threatening systemic complication of liver disease, a liver based metabolic defect or more commonly cirrhosis with complications such as hepatic encephalopathy, ascites, hepatocellular carcinoma, hepatorenal syndrome or bleeding caused by portal hypertension ***(Wiesnor et al., 2003).***

The early post-operative period is a crucial time when strict monitoring and sustainment of cardiorespiratory function, frequent assessment of allograft performance, timely recognition of unexpected complications and prompt treatment of extra hepatic organ system dysfunction are mandatory. Intensive care management of liver transplanted

patients mainly centers on rapid hemodynamic stabilization, correction of coagulopathy, early weaning from mechanical ventilation, proper fluid administration, kidney function preservation, graft rejection prevention, and infection prophylaxis ***(Feltracco et al., 2011).***

Early post-operative complications are often defined as complications occurring within the first 3 months after transplantation because most of deaths that occur in the first post transplantation year happen in this period ***(Gilbert et al., 2002).***

Complications in this period can be broadly divided into surgical and non surgical complications. Surgical complications are mostly operation- related and can be further subdivided into post-operative hemorrhage, portal vein obstruction, hepatic vein thrombosis and biliary complications. Non-surgical complications include pulmonary, renal, cardiovascular, coagulopathy, neurological and infection in the form of wound infection, pneumonia, opportunistic infections & recurrence of hepatitis B virus ***(Bucaloiu et al., 2010).***

AIM OF THE WORK

The aim of the study is to overview postoperative management of liver transplantation patients in ICU, to come up with recommendations for post-operative management with the goal of rapid recovery, short hospital stay, and to decrease morbidity and mortality.

Chapter 1

ANATOMICAL ASPECTS OF THE LIVER

Segmental anatomy of the liver:

The liver is one of the largest organs in the body, representing 2% of the total body weight. In classic descriptions, the liver was characterized as having four lobes: right, left, caudate, and quadrate; however, this is an overly simplistic view that fails to consider the much more complex segmental anatomy ***(Gerard, 2010).***

The liver is divided into eight segments based on the branching of the portal triads and hepatic veins. The structures of the portal triad (hepatic artery, portal vein, and biliary duct) are separate extrahepatically but enter the hepatic hilus ensheathed within a thickened layer of the Glisson capsule ***(Sleisenger, 2010).***

The three main hepatic veins divide the liver into four sectors, each of which is supplied by a portal pedicle. The caudate lobe is an exception because its venous drainage is directly into the vena cava and therefore independent of the major hepatic veins. The four sectors delimited by the hepatic veins are called the portal sectors and these portions of the

parenchyma are supplied by independent portal pedicles arising from the right or left main pedicles ***(Gerard, 2010).***

The divisions separating the sectors are called portal scissurae, within each of which runs a hepatic vein. Further branching of the pedicles subdivides the sectors into segments. The liver is thus subdivided into eight segments, with the caudate lobe designated as segment I. Segments I-IV comprise the left liver, and segments V-VIII, the right. Each segment is supplied by an independent portal pedicle, which forms the basis of sub-lobar segmental resections Figure (1) ***(Sleisenger, 2010).***

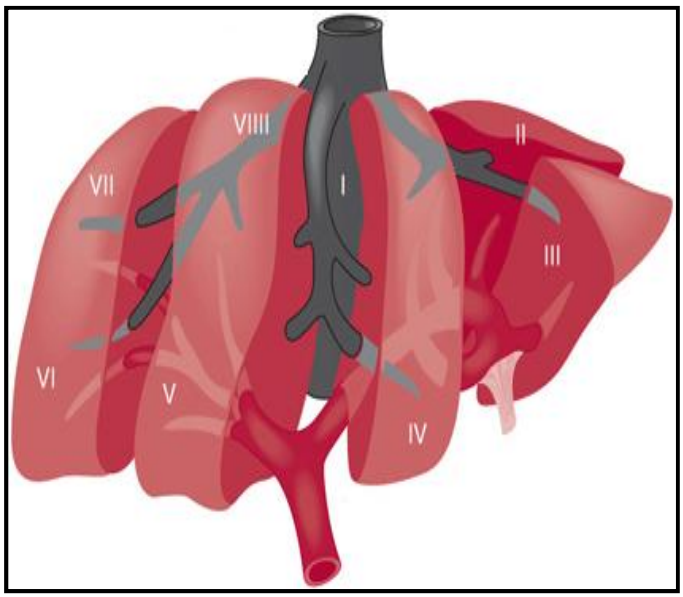

Fig. (1): Segmental anatomy of the liver ***(Sleisenger, 2010).***

The anatomical right and left hemi-livers are separated by an imaginary line running from the medial aspect of the

gallbladder fossa to the inferior vena cava, running parallel with the fissure of the round ligament. This division is known as the Cantlie line or the principal plane and marks the course of the middle hepatic vein ***(Gerard, 2010).***

The right hepatic vein further subdivides the right liver into anterior (segments V and VIII) and posterior (segments VI and VII) sectors, while the umbilical fissure subdivides the left liver into the medial sector (segment IV) and left lateral segment (segments II and III) ***(Sleisenger, 2010).***

Vascular Anatomy of the Liver

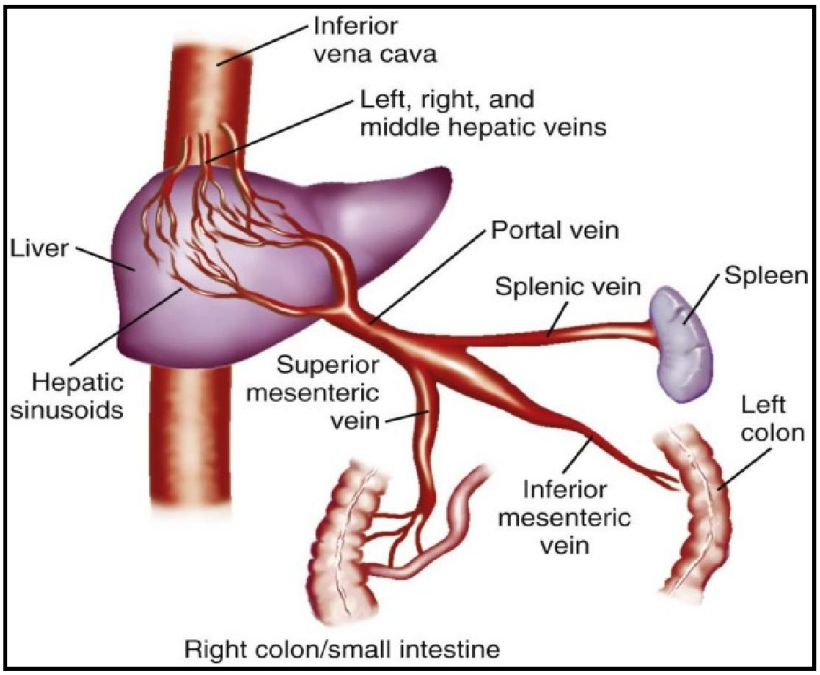

Fig. (2): Vascular anatomy of the liver
(Gerard, 2010).

The liver receives approximately 70% of its blood supply and 40% of its oxygen from the portal vein and 30% of its blood supply and 60% of its oxygen from the hepatic artery Figure (2) ***(Sleisenger, 2010).***

I. Hepatic Artery

The liver has a dual blood supply consisting of the hepatic artery and the portal vein. The hepatic artery delivers approximately 30% of the blood supply, and the portal vein approximately 70%. The common hepatic artery arises from the celiac axis (trunk), as well as the left gastric and splenic artery ***(Charles, 2010).***

The common hepatic artery then divides into the gastro-duodenal artery and the hepatic artery proper. The right gastric artery typically originates of the hepatic artery proper, but this is variable. The hepatic artery proper divides into the right and left hepatic arteries ***(Charles, 2010).***

This "classic" or standard arterial anatomy is present in approximately 75% of cases, with the remaining 25% having variable anatomy. It is critical to understand the arterial (and biliary) anatomic variants to avoid surgical complications when operating on the liver, gallbladder, pancreas, or adjacent organs ***(Charles, 2010).***

Portal Vein

The portal vein is formed by the confluence of the splenic vein and the superior mesenteric vein. The inferior mesenteric vein usually drains into the splenic vein upstream from the confluence. The main portal vein traverses the porta hepatis before dividing into the left and right portal vein branches. Closer to the liver, the main portal vein typically gives off a short branch (posterior lateral) to the caudate process on the right side. Figure (2) (***Sleisenger, 2010).***

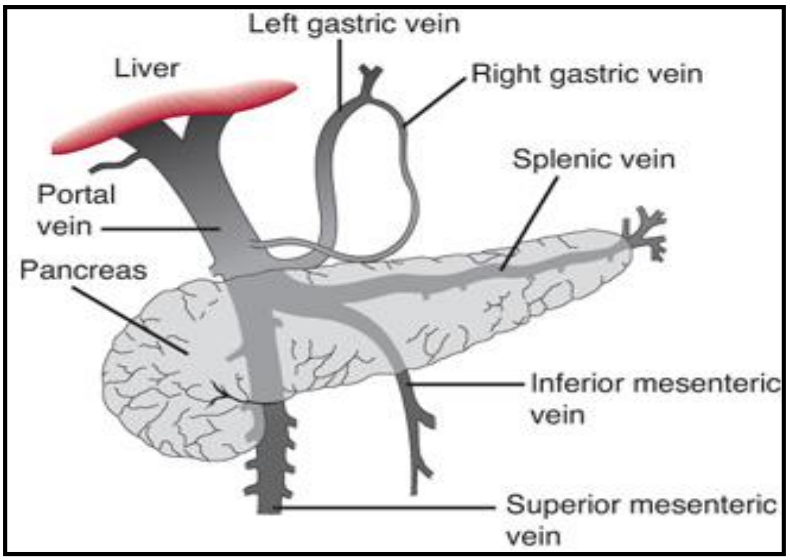

Fig. (3): The portal veins ***(Gerard, 2010).***

The left portal vein typically branches from the main portal vein outside of the liver with a sharp bend to the left and consists of the transverse portion followed by a 90-degree turn at

the base of the umbilical fissure to become the umbilical portion before entering the liver parenchyma ***(Charles, 2010).***

The left portal vein then divides to give off branches to the segment III and II (the left lateral segment), as well as the segment IV feedback branches that supply the left medial segment. The left portal vein also provides the dominant inflow branch to the caudate lobe (although branches can arise from the main and right portal veins also); usually close to the bend between the transverse and umbilical portions. The division of the right portal vein is usually higher in the hilum and may be close to (or inside) the liver parenchyma at the hilar plate ***(Sleisenger, 2010).***

The portal vein drains the splanchnic blood from the stomach, pancreas, spleen, small intestine, and majority of the colon to the liver before returning to the systemic circulation. The portal vein pressure in an individual with normal physiology is low at 3 to 5mmHg ***(Charles, 2010).***

The portal vein is valveless, however, and in the setting of portal hypertension, the pressure can be quite high (20 to 30mmHg), this results in porto-caval anastomoses. Most commonly anastomoses occures via the coronary (left gastric) vein, which produces esophageal and gastric varices with the

propensity for major hemorrhage. Another branch of the main portal vein is the superior pancreatico-duodenal vein ***(Sleisenger, 2010).***

II. Hepatic Veins

There are three hepatic veins (right, middle, and left) that pass obliquely through the liver to drain the blood to the suprahepatic IVC and eventually the right atrium. The right hepatic vein drains segments V to VIII; the middle hepatic vein drains segment IV as well as segments V and VIII; and the left hepatic vein drains segments II and III ***(Charles, 2010).***

The caudate lobe is unique because its venous drainage is directly into the IVC. In addition, the liver usually has a few small, variable short hepatic veins that directly enter the IVC from the undersurface of the liver. The left and middle hepatic veins form a common trunk approximately 95% of the time before entering the IVC, whereas the right hepatic vein inserts separately (in an oblique orientation) into the IVC Figure (4) ***(Charles, 2010).***

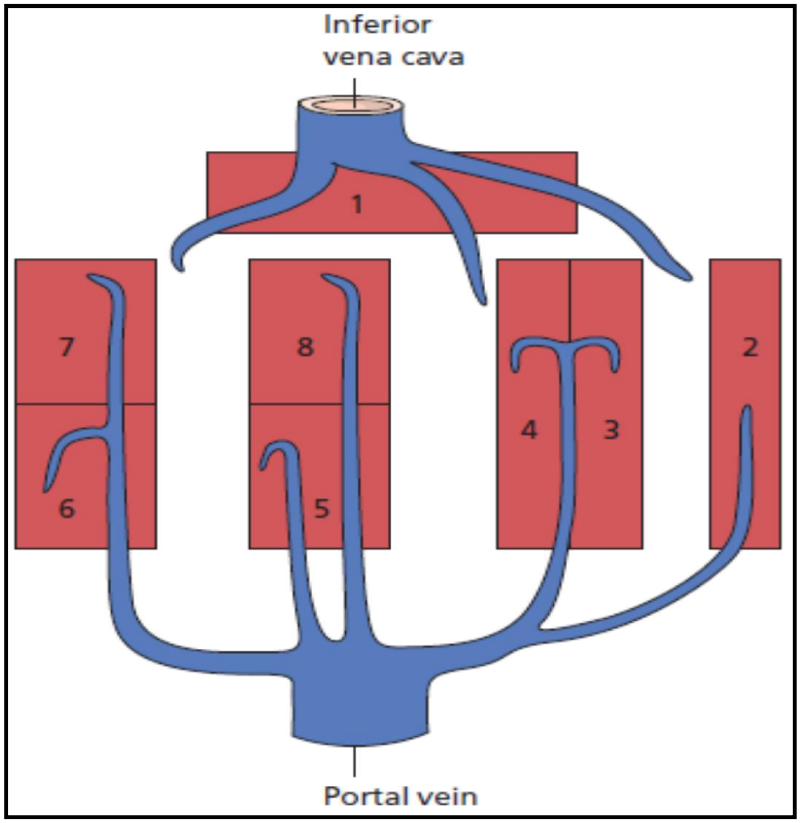

Fig. (4): Diagram of the hepatic venous drainage to the IVC ***(Dooley et al., 2011).***

There is a large inferior accessory right hepatic vein in 15 to 20% of cases that runs in the hepato-caval ligament. The hepatic vein branches bisect the portal branches inside the liver parenchyma (i.e., the right hepatic vein runs between the right anterior and posterior portal veins; the middle hepatic vein passes between the right anterior and left portal vein; and the left hepatic vein crosses between the segment III and II branches of the left portal vein ***(Charles, 2010).***

III. Inferior Vena Cava

The IVC and its tributaries are derived in the 6th to 10th week of life from the fusion and obliteration of several paired embryonic veins. The IVC ascends in the abdomen and ends at the right atrium. It lies to the right of the midline, lateral to the aorta, and receives veins of the lower extremity in addition to a number of lumbar veins that connect with the vertebral and paravertebral venous plexuses Figure (5) ***(Gerard, 2010).***

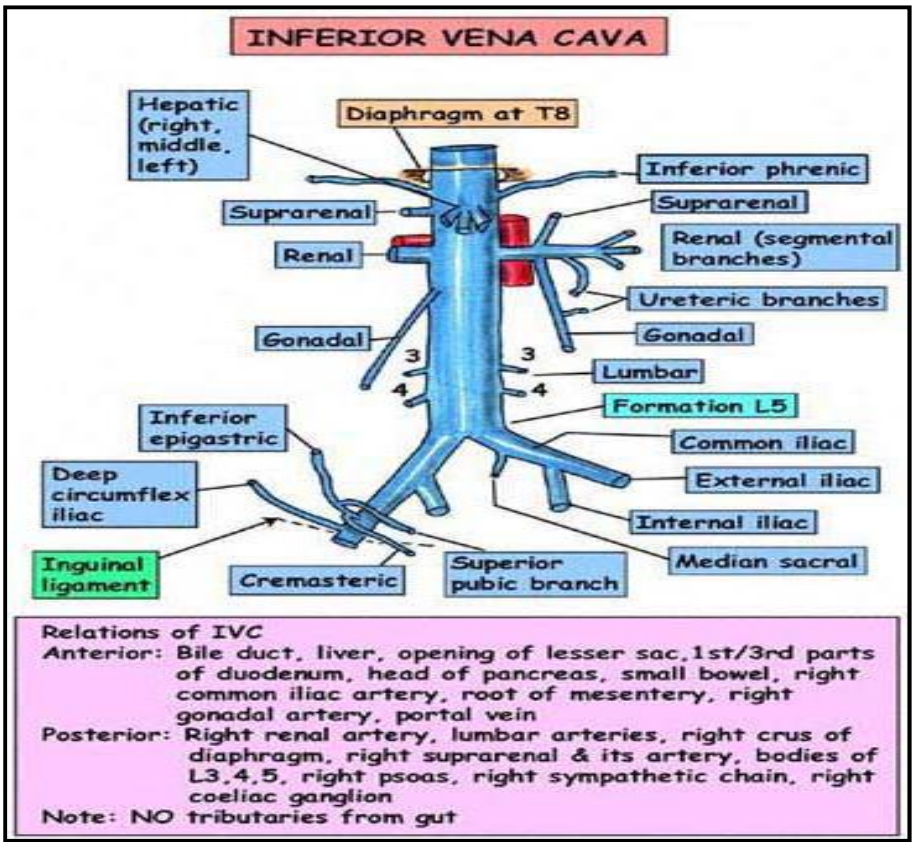

Fig. (5): Inferior vena cava ***(Charles, 2010).***

Chapter 2

INDICATIONS OF LIVER TRANSPLANTATION

The natural history of the patient's disease must be carefully compared to the anticipated survival following transplantation. The clinical tools most widely used to do this include the prognostic Model for End Stage Liver Disease (MELD), the Child-Turcotte-Pugh classification, plus the impact of specific complications of cirrhosis on patient survival. Because none of these tools is perfect, they should be considered complimentary measures, which, when used in combination, give the most accurate estimate of the prognosis of most patients with chronic liver disease ***(Gines et al., 2003).***

The Model of End-stage Liver Disease (MELD) was originally derived from a study of patients undergoing transjugular intrahepatic Porto systemic shunt (TIPS) procedures. Serum bilirubin, international normalized ratio of pro-thrombin time (INR), serum creatinine, and the underlying diagnosis of liver disease were found to be the best predictors of post-TIPS survival. It is calculated according to the formula MELD = 3.78 X log (bilirubin mg/dl) + 9.6 X log (serum creatinine mg/dl Mg-ldl) + 11.2 X log (INR) + 6.43 ***(Kamath et al., 2007).***

In interpretating the MELD score in hospitalized patient the three months mortality is:

- 40 or more → 71.3% mortality
- 30 – 39 → 52.6% mortality
- 20 – 29 → 19.6% mortality
- 10 – 19 → 6.0% mortality
- < 9 – 1 → 1.9% mortality

(Wiesner et al., 2003)

Subsequent studies of this model demonstrated its utility as an effective tool for determining the prognosis in patients with chronic liver disease. The MELD score also has been found useful in predicting short-term survival in patients on the waiting list for liver transplantation as well as estimating the risk of post-operative mortality ***(Thio et al., 2004).***

A simplified MELD model is now used to prioritize patients for donor allocation in the United States. In this model, patients are assigned scores from 6 to 40, which equate to estimate three-month survival rates without transplantation ranging from 90% to 7%, respectively ***(Goyal et al., 2004).***

The Child-Turcotte-Pugh (CTP) classification, originally designed to determine the risk of portacaval shunt surgery, also

has been used to determine the prognosis of patients with chronic liver disease (Table 1) ***(Hwang et al., 2006).***

The CTP score can be quite effective in determining short-term prognosis among groups of patients awaiting liver transplantation (Table 1) (***Suzuki et al., 2004).*** More than one-third of those with CTP scores > 10 can be expected to die within a year ***(Hennig et al., 2002).***

In contrast, patients with CTP scores of 7-9 have an 80% chance of surviving five years, and those with CTP scores of 5-6 have a 90% chance of surviving more than five years without transplantation (Table 2) ***(Romero-Gomez et al., 2004).***

Table (1): Child – Turcotte - Pugh (CTP) scoring system.

Points	1	2	3
Encephalopathy (grade)	None	1 and 2	3 and 4
Ascites	Absent	Slight	Moderate
Bilirubin (mg/dL)	1-2	2-3	>3
Albumin (g/dL)	3.5	2.8-3.5	<2.8
Prothrombin time (seconds prolonged)	1-4	4-6	>6
Or (INR)	M1.7	1.7-2.3	>2.3
For primary biliary cirrhosis: bilirubin (mg/dL)	1-4	4-10	>10

(Hwang et al., 2006)