ANAESTHESIOLOGICAL MANAGEMENT OF PATIENTS WITH LIVER CELL FAILURE PREPARED TO UNDERGO ABDOMINAL SURGERY

Essay

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List of Abbreviations

Abb.	Full term
<i>ALF</i>	Acute liver failure
	Alanine aminotransferase
	Aspartate aminotransferase
BDZ	Benzodiazepine
BUN	Blood urea nitrogen
<i>CNS</i>	Central nervous system
<i>COPD</i>	Chronic obstructive pulmonary disease
<i>CPP</i>	Cerebral perfusion pressure
<i>CSF</i>	Cerebrospinal fluid
<i>CT</i>	Computed tomography
DDAVP	Diamino-8-D-arginine vasopressin
<i>DIC</i>	Disseminated intravascular coagulation
<i>ECG</i>	$Electrocardiogram$
<i>EDV</i>	End diastolic valume
<i>ESLD</i>	End-stage liver Disease
<i>FFP</i>	Fresh frozen plasma
<i>GABA</i>	Gamma-aminobutyric acid
GIT	Gastrointestinal tract
<i>GST</i>	Glutathione S-transferase
	High density lipoprotiens
	Hepatic encephalopathy
	Hemeoxygenase-1
	Hepatorenal syndrome
	Indocyanine green
	Intracranial pressure
	immunoglobulin A
	International normalized ratio
	Inferior vena cava
<i>LDH</i>	Lactate dehydrogenase

List of Abbreviations (cont...)

Abb.	Full term
<i>LDL</i>	Low density lipoprotiens
<i>MAP</i>	Mean arterial pressure
MEGX	Monoethyl gly-cinexylidide
<i>MELD</i>	Model of End-stage Liver Disease
<i>Mn</i>	Manganese
<i>MODS</i>	Multiple organ dysfunction syndrome
<i>MRI</i>	Magnetic resonance imaging
<i>OLT</i>	Orthotopic liver transplantation
<i>PEEP</i>	Positive endexpiratory pressure
PT	$prothrombin\ time$
<i>PTT</i>	Partial thrompoplastin time
<i>SIRS</i>	Systemic inflammatory response syndrome
TRALI	Transfusion-related acute lung injury
<i>UDP</i>	Uridine diphosphate
VLDL	Very low density lipoprotiens
<i>VTE</i>	\ldots Venous thromboembolism

Introduction

nesthesia and surgery in patients with liver diseases may cause concern because of the central role of the liver in many of the body's metabolic and synthetic functions. The liver is a vital organ responsible for protein synthesis, glucose homeostasis, bilirubin excretion, and toxin removal, among other critical functions. In general, the liver has significant functional reserve due to its dual blood supply: portal-venous (75%) and hepatic-arterial (25%). Hence, clinical manifestations of liver damage only occur after considerable injury (Mueller et al., 2004).

Liver disease includes a large spectrum of hepatic dysfunction. It includes asymptomatic transaminitis, cirrhosis, and end-stage liver disease. The most common causes of advanced liver disease are viral infection (hepatitis C and B), alcohol abuse, autoimmune disease, drug or toxin induced, metabolic disorders (e.g., alpha-1 antitrypsin, hemochromatosis, copper) and biliary tract diseases (*Ziser and Plevak, 2001*).

Many of the functions of the liver may be impaired in patients with liver disease. As a result of changes in binding to plasma proteins, detoxification, and excretion; the pharmacokinetic parameters of anesthetics, analgesics, sedatives and muscle relaxants, can be affected, bleeding risk can be increased because of coagulopathy; and susceptibility to infection can be increased because of altered functioning of



hepatic reticuloendothelial cells and other changes in the immune system as well as portal hypertension (Gholson et al., 1990).

The incidence of patients with advanced disease submitted for surgery is on the rise; despite advances in antiviral therapeutics, cirrhosis secondary to hepatitis C and chronic alcohol abuse continues to grow. Concurrently, the medications and treatments aimed at improving survival in these patients have been increasing. Therefore, it can be expected that a growing number of patients with liver disease will undergo surgery. It was estimated that up to 10% of patients with advanced liver disease will have a surgery in the last 2 years of their lives (Haranath and Brintha, 2006).

Several studies have demonstrated increased morbidity and mortality in patients with advanced liver disease undergoing anesthesia and surgery. The extent of surgery and co-morbid conditions also has a major impact. In the past few years, changes have been made in the diagnosis, preoperative anesthetic management preparation, surgical and perioperative care of patients with liver disease (Uddenfeldt and Danielsson, 2001).

Aim of the Study

Studying recent advances in management of patients with liver cell failure.

Anatomy of the Liver

The liver is the largest organ in the body it weighs 1200–1500 gram and comprises one-fifth of the total adult body weight. The liver has two surfaces a diaphragmatic surface in the anterior and superior directions and a visceral surface in the postero-inferior direction (Sherlock and Dooley, 2002).

Relations of the liver

The liver fills the right hypochondrium, epigastric region, and the left hypochondrium, just below the diaphragm. It is related by its upper surface to the diaphragm, which separates it from pleura, lungs, pericardium and heart. Its postero-inferior surface is related to the abdominal esophagus, stomach, duodenum, hepatic flexure of the colon and the right kidney and suprarenal, and the gall-bladder (Fig. 1 & Fig. 2) (*Ellis*, 2006).

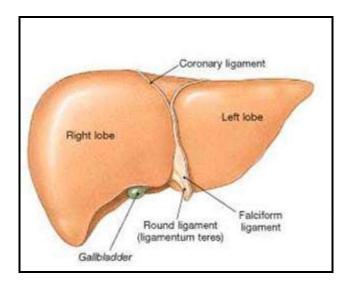


Fig. (1): Anterior surface of the liver (Ellis, 2006).

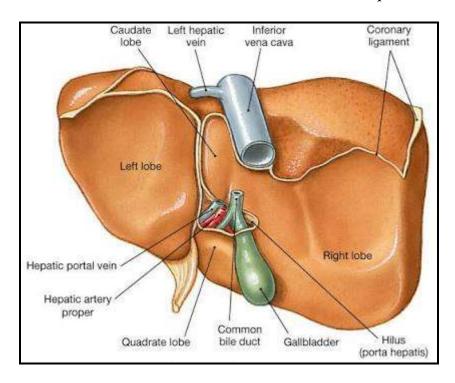


Fig. (2): Visceral surface of the liver (Ellis, 2006).

Ligaments related to the liver

- (1) The falciform ligament attachs the liver to the anterior abdominal wall and anterior portion of the diaphragm.
- (2) Ligamentum teres hepatis (The round ligament) lies in the free edge of the falciform ligament, extending from the umbilicus to the notch between the two lobes. It is the obliterated remnant of the left umbilical vein.
- (3) Ligamentum venosum which is a fibrous remnant of ductus venosus of the fetus (Skandalakis et al., 2004).

Anatomical divisions of the liver

The liver is formed of a large right and a small left lobe. The attachment of the falciform ligament marks this division. The right lobe of liver contains the quadrate and caudate lobes. The quadrate lobe is seen on the upper part of the visceral surface of the liver, while the caudate lobe is seen on the lower part of the visceral surface of the liver (*Blumgart and Hann, 2000*).

Blood supply

The liver has a double blood supply:

- (1) <u>Portal venous supply</u>: the portal vein is formed from the convergence of the superior mesenteric and splenic veins. It is about 8 cm long and lies anterior to the inferior vena cava and posterior to the neck of the pancreas.
- (2) Arterial supply: through, the common hepatic artery that originates from the celiac trunk, it divides into right and left hepatic arteries. Variations from the standard pattern above are common. The two most frequent variants are the right hepatic artery originating from the superior mesenteric artery (11–21%) and the left hepatic artery arising from the left gastric artery (10–30%). Such variant arteries represent the sole supply of a specific territory of the liver their ligation could produce hepatic ischemia of the area they supply (*Fasel et al.*, 2007).

Hepatic blood flow is about 1500 mL/min in adults. Twenty five to thirty per cent is derived from the hepatic artery and 70–75% from the portal vein. Time taken by the red blood

cells to traverse from the portal vein to the central vein is about 8–9 seconds, allowing enough time to contact the hepatocytes and Kupffer cells. The hepatic artery supplies 45–50% of the liver's oxygen requirements and the portal vein supplies the remaining 50–55%. The pressure within the portal vein is normally less than 10 mm Hg. Portal vein oxygen saturation is normally 85%. The total blood flow from this dual supply represents 25–30% of the total cardiac output. Hepatic arterial flow appears dependent on metabolic demand (autoregulation), whereas flow through the portal vein depends on blood flow to the gastrointestinal tract and the spleen (*Morgan et al.*, 2006).

Oxygenated blood to the liver is carried by the hepatic artery which is a branch of aorta. While the portal vein carries blood from the small intestine that is rich in nutrients. These vessels divide into smaller vessels ending in capillary network which is distributed to thousands of liver lobules. Each lobule is formed of hepatocytes. Their function is to add or remove substance from the blood reaching them. The blood then leaves the liver via the hepatic vein, returns to the heart, and is ready to be distributed to the rest of the body (*Tzanakakis*, 2000; *Gelman et al.*, 2001).

Venous drainage

The hepatic venous effluent drains into the inferior vena cava by means of three major hepatic veins, right, middle and left. These veins have an extrahepatic length of about 1 cm. Within the liver, the main trunks run between the Glissonian

territories (i.e. within the planes that lie between the areas supplied by a given portovenous, arterial and biliary branch) (Fasel et al., 2007).

Biliary drainage

The intrahepatic branching of the biliary ducts follows a segmental pattern; each region of the liver has its specific type of bile drainage as evidenced by investigations on the biliary system within the human liver. The intrahepatic bile ducts follow a similar type of branching to the corresponding branches of the portal vein and hepatic artery (*Fasel et al.*, 2007).

Innervation

Sympathetic fibers (T6–T11), parasympathetic fibers (right and left vagus), and fibers from the right phrenic nerve, all are forming nerve supply of the liver. Some autonomic fibers synapse first in the celiac plexus whereas other fibers reach the liver directly through splanchnic nerves and vagal branches before forming the hepatic plexus. Mostly sensory afferent fibers travel with sympathetic fibers. Nerve fibers enter at the porta and join vessels and ducts to the interlobular spaces, the non-medullated nerve fibers enter the lobules and branch between the cells and even within them, while the medullated nerve fibers are distributed almost exclusively to the coats of the blood vessels (*Gray*, 2000).