# Liver Dysfunction
# In Intensive Care Patients

**An Essay**

***Submitted for partial fulfillment of Master Degree in intensive care***
***Presented* by**


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**Mohamed Abdel-Moneim Ibrahim Waly**

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# *LIST OF ABBREVIATIONS*

| | |
|---|---|
| **AAC** | Acute acalculous cholecystitis |
| **acetyl CoA** | Acetyl coenzyme A |
| **AFP** | α-Fetoprotein |
| **ALA** | Aminolevulinic acid |
| **ALF** | Acute liver failure |
| **ALP** | Alkaline phosphatase |
| **ALT** | Alanine aminotransferase |
| **APACHE** | Acute physiology and chronic health evaluation |
| **AST** | Aspartate aminotransferase |
| **ATP** | Adenosine triphosphate |
| **BCS** | Budd-chiari syndrome |
| **cAMP** | Cyclic adenosine monophosphate |
| **CHF** | Congestive heart failure |
| **COP** | Cardiac output |
| **CT** | Computed tomography |
| **CVP** | Central venous pressure |
| **DILI** | Drug-induced liver injury |
| **FFP** | Frozen plasma |
| **FXR** | Farnesoid X receptor |

| | |
|---|---|
| **GFR** | Glomerular filtration rate |
| **GGT** | Gamma-glutamyl transpeptidase |
| **HABR** | Hepatic arterial buffer response |
| **HAV** | Hepatitis A virus |
| **HBV** | Hepatitis B virus |
| **HE** | Hepatic encephalopathy |
| **HEV** | Hepatitis E virus |
| **HH** | Hypoxic hepatitis |
| **HIDA** | Hepatobiliary iminodiacetic acid |
| **HMB** | Hydroxyl-methylbilane |
| **HRS** | Hepatorenal syndrome |
| **ICG-PDR** | Indocyanine green plasma disappearance rate |
| **Ig** | Immunoglobulin |
| **IGF-I** | Insulin-like growth factor I |
| **IL** | Interleukin |
| **INR** | International normalized ratio |
| **IVC** | Inferior vena cava |
| **LDH** | Lactate dehydrogenase |
| **MELD** | Model for End-stage Liver Disease |
| **MODS** | Multiple organ dysfunction syndrome |
| **NADPH** | Nicotinamide adenine dinucleotide phosphate |
| **PBG** | Porphobilinogen |

| | |
|---|---|
| **PMN** | Polymorphonuclear cell |
| **PN** | Parenteral nutrition |
| **PO2** | Partial pressure of oxygen |
| **SAPS** | Simplified acute physiology score |
| **S-AT** | Serum aminotransferase |
| **SBP** | Spontaneous bacterial peritonitis |
| **SOFA** | Sequential organ failure assessment |
| **T3** | Triiodothyronine |
| **TIPS** | Transjugular Intrahepatic Portosystemic Shunt |
| **TNF** | Tumor necrosis factor |
| **TPN** | Total parenteral nutrition |
| **UDP** | Uridine diphosphate |
| **US** | Ultrasound |
| **VLDLs** | Very-low-density lipoproteins |
| **xULN** | Times the upper limit of normal |

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# *INTRODUCTION*

The liver is one of the most important organ in the body and serves a variety of important functions including metabolic, vascular, immunological, secretory and excretory functions. It plays a key role in the metabolism of carbohydrate, protein and fat, and metabolism of toxins and drugs, and in modulation of immunity in the human body ***(Mitra and Metcalf, 2012).***

Abnormal liver biochemical and function tests are found in the majority of critically ill patients and are associated with increased mortality. Frequent causes for elevated liver function tests in the intensive care unit are acute hepatic dysfunction due to acute hepatitis, acute liver failure, and drug-induced liver injury. Furthermore, exacerbations of pre-existing liver diseases (acute on chronic) and secondary liver injury during critical diseases such as sepsis, right heart failure, or cardiogenic shock, resulting in ischemic or hypoxic hepatitis ***(Koch et al., 2013)***.

In this review, we choose to define liver injury as an elevation in serum concentrations of routinely measured hepatic enzymes, including aminotransferases, alkaline

phosphatase, or γ-glutamyl transpeptidase. Hepatic dysfunction refers to derangement of pathways related to synthetic or clearance function, including international normalized ratio and bilirubin. Hepatotoxicity refers to hepatic injury and dysfunction caused by a drug or another noninfectious agent. Acute liver failure designates liver injury that results in life-threatening hepatic synthetic dysfunction and brain dysfunction (encephalopathy) ***(Lescot et al., 2012)***.

In critically ill patients, hypoxic, toxic, and inflammatory insults can affect hepatic excretory, synthetic, and/or purification functions, leading to systemic complications such as coagulopathy, increased risk of infection, hypoglycemia, and acute kidney injury. In severe cases, hepatic encephalopathy or brain dysfunction (acute liver failure) may occur. Because of the lack of specificity of standard laboratory investigations, identifying liver injury or dysfunction in critically ill patients remains a significant challenge ***(Lescot et al., 2012)***.

# Anatomy of the Liver

## Overview

The liver is the second largest (after the skin) organ in the human body and the largest gland (weighing an average of 1500 g). It is the only organ capable of regeneration. It lies under the diaphragm in the right upper abdomen and midabdomen and extends to the left upper abdomen (right hypochondriac and epigastric regions) and protected by the ribs. The liver has the general shape of a prism or wedge, with its base to the right and its apex to the left **(fig. 1).** It is pinkish brown in color, with a soft consistency, and is highly vascular and easily friable ***(Gray and Lewis, 2000).***

It extends along the midclavicular line from the right fifth intercostal space to just inferior to the costal margin. The anterior border of the liver then extends medially and crosses the midline just inferior to the xiphoid process. A small portion of the organ projects across the midline and lies in the upper left abdominal quadrant ***(Saxena et al., 2003).***

## Gross Anatomy

The liver is divided into 4 lobes: right, left, caudate, and quadrate. The right and left lobes are the largest, while the caudate and quadrate are smaller and located posteriorly. Two ligaments are visible anteriorly. Superiorly, the falciform ligament separates the right and left lobes. Inferior to the falciform ligament is the round ligament, which protrudes from the liver slightly ***(Eric and Allen, 2002).***

Also visible anteriorly on the most inferior portion of the right lobe is the gallbladder. Posteriorly, many more interesting structures are visible. The caudate lobe is located superiorly, approximately between the right and left lobes. Adjacent to the caudate lobe is the sulcus for the inferior vena cava. Just inferior to the caudate lobe is the porta hepatis, where the hepatic artery and hepatic portal vein enter the liver. The portal vein carries nutrient laden blood from the digestive system. Inferior to the porta hepatis is the bile duct which leads back to the gallbladder. Finally, the hepatic vein, where post-processed blood leaves the liver, is found inferior and adjacent to the sulcus for the inferior vena cava. The liver is held on place by a system of mesenteries posteriorly, and is also attached to

the diaphragm via the falciform ligament. Additionally, most of the liver is covered by visceral peritoneum ***(Eric and Allen, 2002).***

## Peritoneal connection

Peritoneal attachments of the liver are shown in (Fig. 2). The double layer of the parietal peritoneum continues to the falciform ligament and surrounds the liver except for the bare area, where the two layers separate to form the coronary ligament and the left triangular ligament. The left layer of the falciform ligament becomes the superior layer of the left coronary ligament. The right layer becomes the upper layer of the coronary ligament, which meets the lower layer to form the right triangular ligament. The lower layer of the coronary ligament continues on the posterior surface of the liver and can reflect on the upper part of the right kidney to form the hepatorenal ligament. Then it passes in front of the groove for the inferior vena cava (IVC), and, after a semicircular course in front of the caudate lobe, it meets the right leaf of the lesser omentum. The leaf of the lesser omentum continues in the posterior leaf of the left triangular ligament ***(John et al., 2004)***.