ANAESTHETIC MANAGEMENT FOR PATIENTS WITH LIVER DISEASE IN NON HEPATIC SURGERY

Submitted for Partial Fulfillment of
Master Degree in Anaesthesia

By

MAHMOUD EL SAYED MAHMOUD AHMED
M. B .B .Ch.
Al Azhar Faculty of Medicine, Cairo

Under Supervision of

Prof. Bahaa El-din Ewees Hassan
Professor of Anaesthesia & Intensive Care
Faculty of Medicine – Ain Shams University

Dr. Mahmoud Hassan Mohamed
Lecturer of Anaesthesia & Intensive Care
Faculty of Medicine – Ain Shams University

Faculty of Medicine
Ain Shams University
(2012)

ACKNOWLEDGMENT

Before and above all, Praise and Thanks to **ALLAH** *for all countless gifts I have been offered, one of these gifts is accomplishing this research work.*

I would like to express my profound gratitude to ***Professor dr / BAHAA EL-DIN EWES HASSAN,*** *Professor of Anesthesia and Intensive Care, Faculty of Medicine, Ain Shams University for his most valuable advises and support all through the whole work and for dedicating much of her precious time to accomplish this work.*

My special thanks and deep obligation to ***Dr/ MAHMOUD HASSAN MOHAMED****, Lecturer of Anesthesia and Intensive Care, Faculty of Medicine, Ain Shams University for his continuous encouragement and supervision and kind care .*

MAHMOUD ELSAYED

LIST OF CONTENTS

LIST OF ABBREVIATIONS

ALT	Alanine aminotransferase.
AST	Aspartate aminotransferase.
ASA	American society of anesthesia.
CO	Cardiac output.
COPD	Chronic obstructive pulmonary disease.
CT	Computed tomography.
CTP score	Child-Turcott- Pugh score.
DIC	Disseminated intravascular coagulopathy.
ECG	Electrocardiogram.
FRC	Functional residual capacity.
FHF	Fulminant hepatic failure.
GABA	Gamma amino butyric acid.
GFR	Glomerular filteration rate.
G6PD	Glucose-6-phosphate dehydrogenase.
HO-1 enzyme	Haemoxygenase-1 enzyme.
HPS	Hepatopulmonary syndrome.
HRS	Hepatorenal syndrome.
ICU	Intensive care unit.
ICP	Intracranial pressure.
INR	International normalized ratio.
LFTs	Liver function tests
MELD	Model for end stage liver disease.
MMCC	Moemen modified Child classification.
MPAP	Mean pulmonary artery pressure.
NAFLD	Non-alcohlic fatty liver disease.
OLT	Orthotopic liver transplantation.
P(a/A)O_2	arterial/alveolar oxygen tension ratio.

LIST OF ABBREVIATIONS (Cont...)

PCWP	Pulmonary capillary wedge pressure.
PEEP	Positive end expiratory pressure
PPHTN	Portopulmonary hypertension.
PT	Prothrombin time.
PTT	partial thromboplastin time.
PVR	Pulmonary vascular resistance.
TAFI	Thrombin activatable fibrinolysis inhibitor.
TEG	Thromboelastograph.
TIPS	Transjugular intrahepatic portosystemic shunting.
TRALI	Transfusion – related acute lung injury
UNOS	United network for organ transplantation.
VATS	Video-assisted thoracoscopic.
Vd	Volume of distribution.
V/Q	Ventilation – perfusion ratio.
VTE	Venous thromboembolism.
WBC	White blood count.

LIST OF TABLES

LIST OF FIGURES

INTRODUCTION

The prevalence of chronic liver disease is increasing all over the world because of viral hepatitis, alcohol and non-alcoholic fatty liver disease (NAFLD) ***(Noble et al.,1993).***

It has been estimated that about 10% of patients with cirrhosis will undergo surgery in the last 2 years of their life ***(Farnsworth et al., 2004).***

General anesthesia and surgery may lead to complications in a significant proportion of patients with well-compensated or occult cirrhosis, and these complications may result in considerable morbidity and mortality. The reported mortality rates in patients with cirrhosis undergoing various surgical procedures range from 8.3% to 25% in comparison to 1.1% in non-cirrhotic patients ***(Del Olmo et al.,2003).***

This wide range of mortality rates is related to severity of liver disease, type of surgery, demographics of patient population, expertise of the surgical, anesthesia and intensive care unit (ICU) team and finally, reporting bias. It is therefore, important to assess the risk in relation to the type of surgery that is performed; arbitrarily, the type of surgery could be categorized into high, moderate or low risk surgery ***(Ziser et al., 1999).***

End-stage liver disease is usually manifested as cirrhosis. Cirrhosis is a combination of fibronodular hyperplasia and bridging fibrosis. Functionally, there are hepatocellular synthetic dysfunction, cholestasis and portal hypertension ***(Vaja et al., 2010)*.**

Preoperative Preparation

In patients with compromised liver function, preservation of the remaining function is essential, otherwise, peri-operative complications are high. Cirrhosis is a final common pathway for chronic liver diseases of different etiologies. In Egypt, cirrhosis is mostly due to bilharzialperi-portal hepatic fibrosis or repeated viral attacks leading to chronic hepatitis. Thedeath rate due to liver cirrhosis in Egypt is not exactly known due to deficient statistical evaluation. In United States of America, cirrhosis is the 9th leading cause ofdeath, known as Laennec's cirrhosis due to alcohol consumption ***(MerritandGelman,1998).***

It has been estimated that 10% of all patients with liver disease undergo operative procedures during the final two years of their lives**(Farnsworth et al., 2004).**

Liver diseases:

Liver diseases include hepatitis, liver cirrhosis and hepatobiliary disease. **Hepatitis may be acute or chronic**.

Acute hepatitis:results from viral infection, a drug reaction or exposure to a hepatotoxin.Hepatocellular injury with cell necrosis may present either as a mild inflammatory reaction or acute hepatic failure.Viral hepatitis is mostly due to

hepatitis A, B or C viruses. Hepatitis B and hepatitis C (previously known as non A, non B) viruses are transmittedpercutaneously and / or by contact with body fluids. Patients have mild prodromal illness that may be followed by jaundice lasting for 2-12 weeks with complete recovery within 16-20 weeks. The disease may turn chronic and infectious carriers pose a major health hazard to operating room personnel. Drug induced hepatitis can result from direct toxicity of drugs as acetaminophen, salicylates and tetracyclins; from idiosyncrasy to volatile anaesthetics, phenytoins and sulfonamides or from primary cholestasis by chlorpromazine, cyclosporins and oral contraceptives ***(Morganet al.,2006).***

Fulminant hepatic failure (FHF) is so acute that the physical findings of chronic liver disease, except for jaundice, are often absent. Unfortunately, cerebral oedema occurs and is a striking finding***(Ware et al.,1971).***

Signs of increased intracranial pressure (ICP) may be present in as many as 80% of patients with FHF***(Gimson et al.,1982).***

Increased ICP is considered a specific neurological complication that is quite distinct from the hepatic

encephalopathy seen in chronic liver disease***(Berkand Popper,1978).***

An increase in the plasma concentration of serum alanine aminotransferase (ALT) and serum aspartate aminotransferase (AST) is often accepted as an index of liver cell damage. However, these enzymes may not reflect the extent of hepatic damageaccurately, being not very specific to the liver ***(Cerwenka et al., 1998).***

Some investigators tried to link the ratio of AST/ALT to diagnose the etiology of liver disease, but this approach has been plagued by inconsistency (***Toblas,1998).***

Chronic hepatitis: represents persistent hepatic inflammation for longer than six months evidenced by elevated aminotransferases. Chronic persistent hepatitis and chronic lobular hepatitis do not usually progress to cirrhosis, but chronic active hepatitis due to hepatitis B or C viruses progress to cirrhosis and are usually managed with long-term steroids ***(Zuckerman, 1990).***

Liver cirrhosis: when described as post-necrotic is due to chronic active hepatitis; when described as biliary is due to chronic inflammation or obstruction;and when described as cardiac is due to congestive heart failure. In all these types,however, hepatocyte necrosis takes place with different

severities,followed by fibrosis and nodular regeneration. Bilharziasis produces periportal hepatic fibrosis without hepatocellular necrosis or nodular regeneration and the hepatocellular function is often preserved regardless the etiology of cirrhosis. Distortion of the normal vascular architecture obstructs the portal venous flow and leads to portal hypertension ***(Morganet al.,2006).***

Systemic manifestations ofliver disease:

* **Cardiovascular system:**

Most patients with advanced liver disease have a normal or even supernormal ejection fraction judged by echocardiography. Thus, physicians previously assumed that cardiac function was normal in most patients with liver disease. However, further investigation had uncovered multiple problems in cardiac performance that place patients at risk of heart failure ***(Mandell and Tsou, 2008).***

Patients with liver cirrhosis have a hyperdynamicsyndrome. The components of this syndrome include an increased cardiac output, increased heart rate, vascular volume, reduced arterial pressure, reduced systemic vascular resistance and impaired renal perfusion. The hyperdynamic circulatory consequences of this syndrome can lead to morphologic alterations in the heart

to include right atrial and right ventricular dilatation ***(Karasu et al., 2004).***

Patients may appear to have adequate cardiac reserve at rest, but they decompensateunder physiological or pharmacological stress. Cirrhotic cardiomyopathy is defined by the presence of one or more of the following:

- Increased cardiac output, but blunted inotropic response to stress.
- Diastolic dysfunction.
- No overt left ventricular failure at rest
- Electrophysiological abnormalities (chronotropic incompetence andprolongedQT interval)***(Biancofiore et al., 2010).***

Rhythm disturbances in patients with cirrhosis have also been described. These include atrial flutter/fibrillation, as well as ectopic beats***(Zambruni et al., 2006).***

Early histological changes in cirrhotic hearts include myocardial hypertrophy, interstitial and cellular edema, and signs of cellular injury ***(Milani et al., 2007).***

This causes global thickening of the left ventricle with the septum affected more than the free wall ***(Ma and Lee,1996).***

The fluid retention that occurs in cirrhotic patients can lead to the development of ascites, hydrothorax, anasarca and pericardial effusion ***(Karasu et al., 2004).***

* **Respiratory:**

Several unique respiratory pathologies may beassociated with cirrhosis and portal hypertension.These include the hepatopulmonary syndrome,portopulmonary hypertension and hepatichydrothorax. In addition, other factors contributingto respiratory compromise may include pre-existing chronic obstructive pulmonary disease (COPD), tense ascites and muscle wasting***(Karasu et al., 2004).***

Diaphragmatic splinting from ascites or the presence of pleural effusions restricts alveolar ventilation, reduces functional residual capacity(FRC), and predisposes to atelectasis and hypoxia. Associated gastro-esophageal reflux disease, acute alcohol ingestion, and massive ascites may increase the risk of aspiration of gastric contents. Intrapulmonary arterio-venous shunting may also occur. Patients may experience dyspnea and hypoxemia when sitting upright, which improves on lying flat (orthodeoxia) ***(Wiklund, 2004).***

The presence of pulmonary vascularshunting is best detected on echocardiography (this incorporates the ‘saline