Post Operative Infections after Liver Transplantation

Essay

Submitted in Partial Fulfillment of Master Degree in Intensive Care

By Ibrahim Mohamed Eltokhy (M.B.B.ch.)

Supervisors

Prof.Dr. Seif Eleslam Abdelaziz Shaheen

Professor of Anaesthesia and ICU Faculty of Medicine, Ain Shams University

Prof.Dr. Sherif Farouk Ibrahim

Professor of Anaesthesia and ICU Faculty of Medicine, Ain Shams University

Dr.Amr Hanafy Mahmoud

Lecturer of ICU
Theodor Bilharz Research Institute

Anaesthesia and ICU Department Faculty of Medicine Ain Shams University 2013

بِسْمِ اللَّهِ الرّحَمَٰنِ الرّحيمِ

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صدق الله العظيم

النمل. اية رقم ١٩



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List of Abbreviations

ADH : Alcohol dehydrogenase

AIDS : Acquired immunodeficiency syndrome

ALF : Acute liver failure

ALT : Alanine aminotransferase

ARDS : Acute Respiratory Distress Syndrome

ARF : Acute renal failure

ASTS : American Society for Transplant Surgeons

AVT : Anti-viral therapy

BCAA : Branched chain aminoacids

BCM : Body cell mass

BG: b-glucan

CBD : Common bile ductCLD : Chronic liver diseaseCMV : Cytomegalovirus

CNS : Central nervous system
CRF : Chronic renal failure

CRI : Catheter related infections

CSF : Cerebrospinal fluidCTP : Child-Turcotte-Pugh

CYP : Cytochrome p

DAAs : Direct antiviral agents

EBV : Epstein bar virus

ELISA : Enzyme linked immunosorbent assayESBL : Extended spectrum beta lactamase

ESLD : End stage liver disease

ETR : End of treatment responseGM : Aspergillus galactomannanHAT : Hepatic artery thrombosis

List of Abbreviations (Cont...)

HBF : Hepatic blood flow

HCC: Hepatocellular carcinoma

HCV : Hepatitis c virusICG : Indocyanine greenICU : Intensive care unit

IFI : Invasive fungal infections

IL : Interleukin

ILTS : International Liver Transplantation Society

IVC : Inferior vena cava

LDL : Low density lipoproteinLT : Liver transplantation

LTBI : Latent tuberculosis infection

MASP2 : MBL-associated serine protease 2

MBL : Mannan-binding lectinMDR : Multidrug resistant

MEGX : Monoethylglycinxylidide metaboliteMELD : Model for end stage liver disease

MEOS : Microsomal ethanol oxidizing system

MMF : Mycophenolate mofetil

MRSA : Methicilline resistant staph aureusNAS : Non-anastomotic biliary strictures

P.Is : Protease inhibitors

PAMPs: Pathogen-associated molecular patterns

PBC : Primary biliary cirrhosisPCR : Polymerase chain reaction

Peg-IFN : Pegylated interferonPH : Partial hepatectomyPOL I : Polymerase inhibitor

List of Abbreviations (Cont...)

PRRs : Recognition receptors

PSC: Primary sclerosing cholangitis

PVT : Portal vein thrombosis

RBV : Ribavirin

SDD : Selective digestive decontaminationSNPs : Single nucleotide polymorphisms

SOT : Solid organ transplantationSVR : Sustained virologic response

TB : Tuberulosis

TEG : ThrombelastographyTLR : Toll-like receptor

TRALI : Transfusion related acute lung injuryVAP : Ventilator associated pneumoniaVLDL : Very low density lipoprotein

VRSA : Vancomycin resistant staph aureus

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Introduction

rthotopic Liver Transplantation (OLT) has become a widely accepted treatment for a variety of liver diseases such as viral and alcoholic cirrhosis, liver malignancy, acute liver failure, and many metabolic abnormalities (*Feltracco et al.*, 2011).

As a result of improvement in anesthesiological and surgical skill, organ support device adoption, advanced understanding of transplant immunology, and better critical care management of complications, liver transplanted patients survive longer. Patient survival in one- and five-year is around 90% and 80% respectively (*van Hoek et al.*, 2012).

According to many reports, infections in the early period after OLT remain important causes of morbidity and mortality, despite advanced medical care. The infection-related mortality rate in nearly 50% when accompanied by septic shock. It is estimated that up to 80% of liver recipients will develop at least one infection during the first year after transplantation, and, while most are successfully treated, some will result in death (*Xu et al.*, 2012).

Bacterial infections are most frequent (70%), followed by viral (20%) and fungal infections (8%). The diagnosis is difficult among this group of patients because of the usually mild or often absent signs and symptoms of infection. Nevertheless even mild infections in immunocompromised patients may produce catastrophic effects (*Romero and Razonable*, 2011).

Both donor and recipient factors as well as aspects related to the transplant operation contribute to the risk of infection after OLT. Recently genetic polymorphisms in the innate immune system, from both donor and recipient, have been identified as important risk factors for infection after OLT (*Vera et al.*, 2011).

Infection control strategies should be an integral component of liver transplant programs in order to reduce its incidence and transmission. With surveillance, cohorting, contact isolation, and nasal decolonization (*Russell et al.*, 2008).

Treatment of bloodstream infections should be directed towards the elimination of the predisposing factor, combined with pathogen-directed antimicrobial therapy that is guided by antimicrobial susceptibility testing. For persistent bloodstream infections like endocarditis should be evaluated by means of a transesophageal echocardiogram. Indwelling vascular and urinary catheters should be removed, intra-abdominal abscesses should be drained, and other potential nidus of infection should be surgically corrected, if feasible (*Mathers et al.*, 2009).

Aim of the Study

The aim of the work is to review infections as a postoperative complication after liver transplantation in the intensive care unit and the strategies to deal with to achieve the most favourable outcome.

Anatomy and Physiology of the Liver

Anatomy of the liver

The liver is the largest gland in the body and, after the skin, the largest single organ. It weighs approximately 1500g and accounts for approximately 2.5% of the adult body weight (*Moore and Dalley, 2006*).

The liver is a solid gastrointestinal organ that largely occupies the upper quadrant of the abdomen. The costal margin coincides with the lower margin and the superior surface is draped over by the diaphragm. Most of the right liver and most of the left liver is covered by the thoracic cage. The liver extends superiorly to the height of the fifth rib on the right and the sixth rib on the left (*Townsend et al.*, 2004).

The posterior surface straddles the inferior vena cava (IVC). A wedge of liver extends to the left half of the abdomen across the epigastrium to lie above the anterior surface of the stomach and under the central and left diaphragm. The superior surface of the liver is convex and is molded to the diaphragm, whereas the inferior surface is mildly concave and extends to a sharp anterior border (*Townsend et al.*, 2004).

Surfaces of the liver

The diaphragmatic surface is smooth and dome shaped where it is related to the concavity of the inferior surface of the diaphragm. Subphrenic recesses exist between diaphragm and anterior and superior aspects of diaphragmatic surface of the liver. The hepatorenal recess is a posteriosuperior extension of the subhepatic space that is a gravity-dependant part of the peritoneal cavity in the supine position (*Moore and Dalley, 2006*).

The visceral surface is covered with perioteneum except at the fossa for gallbladder and the portahepatis. The visceral surface bears multiple fissures and impressions from contact with other organs (*Moore and Dalley*, 2006).

Ligaments of the liver

The liver is attached to the anterior abdominal wall by the falciform ligament and, except for a small area of the liver against the diaphragm (the bare area), the liver is almost completely surrounded by visceral peritoneum. Additional folds of peritoneum connect the liver to the stomach (hypogastric ligament), the duodenum (hepatodudenal ligament), and the diaphragm (right and left triangular ligaments and anterior and posterior coronary ligaments) (*Darke et al.*, 2005).

Important relations

Anteriorly, the liver is related to the diaphragm, right and left costal margins, right and left pleura, and lower margins of both lungs, xiphoid process and anterior abdominal wall in the subcostal angle.

Posteriorly, the liver is related to diaphragm, right kidney, hepatic flexure of the colon, duodenum, gallbladder, IVC, esophagus, and fundus of the stomach (*Snell*, 2004).

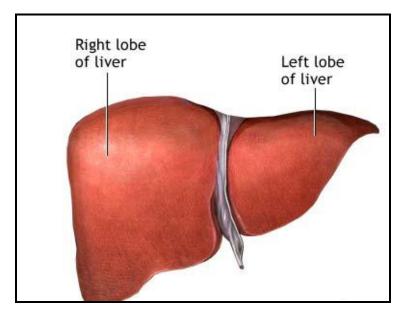


Figure (1): Anterior surface of the liver. Sherlock and Dooley (2002).

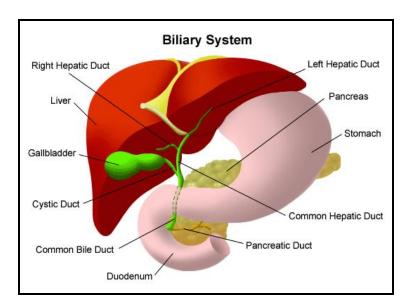


Figure (2): Visceral surface of the liver. Sherlock and Dooley (2002).