بسم الله الرحمن الرحيم

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تم رفع هذه الرسالة بواسطة / مني مغربي أحمد

بقسم التوثيق الإلكتروني بمركز الشبكات وتكنولوجيا المعلومات دون أدنى مسئولية عن محتوى هذه الرسالة.

ملاحظات: لا يوجد

Relationship and Inverse Relationship between HCV Treatment by DAA's and Portal Hypertension in Egyptian Patients with Combined Cirrhosis & Portal Hypertension

Thesis

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List of Contents

List of Tables

List of Tables (cont...)

List of Tables (cont...)

Table No.	Title	Page No.

List of Figures

List of Figures (Cont...)

List of Figures (Cont...)

List of Abbreviations

Abb.	Full term
2D-SWE	*2-D Shear-Wave Elastography*
ARFI	*Acoustic radiation force impulse*
CEUS	*Contrast Enhanced Ultrasound*
CIN	*Contrast induced nephropathy*
CPT	*Child–Pugh– Turcotte*
CSPH	*Clinically significant portal hypertension*
CT	*Computed Tomography*
DAA	*Direct-acting antivirals*
DLRE	*Deep learning Radionomics of elastography*
DM	*Diabetes mellitus*
ECM	*Extracellular matrix*
EGD	*Endoscopic signs using*
EGF	*Epidermal growth factor*
ELF	*Enhanced Liver Fibrosis*
EUS	*Endoscopic ultrasound*
EV	*Esophageal varices*
EV	*Esophageal varices*
FHVP	*Free hepatic vein pressure*
FSGS	*Focal seg-mental glomerulosclerosis*
GAVE	*Gastric antral vascular ectasia*
GOV	*Gastroesophageal varices*
GV	*Gastric varices*
HCC	*Hepatocellular carcinoma*
HCV	*Hepatitis C virus*
HSC	*Hepatic stellate cells*
HVPG	*Hepatic venous pressure gradient*
HVTT	*Hepatic vein transit times*
IGV	*Isolated gastric varices*
INR	*International normalization*

List of Abbreviations (Cont...)

Abb.	Full term
IVR	*Intrahepatic vascular resistance*
kPa	*Kilopascals*
LS	*Liver stiffness*
MELD	*Model of end- stage liver disease*
MMP-2	*Metalloproteinase 2*
MPGN	*Membranoproliferative glomerulonephritis*
MRI	*Magnetic resonance imaging*
NRH	*Nodular regenerative hyperplasia*
NS	*Nonstructural*
PC	*Phase contrast*
PDGF	*Platelet derived growth factor*
PEG-IFN	*Pegylated-interferon*
PHG	*Portal Hypertensive Gastropathy*
PI	*Pulsatility Index*
PPG	*Portal pressure gradient*
pSWE	*Point Shear Wave Elastography*
PVT	*Portal vein thrombosis*
RAP	*Right atrial pressure*
RBV	*Ribavirin*
RHP	*Regional hepatic perfusion*
RI	*Resistive Index*
ROI	*Region of interest*
SBP	*Spontaneous bacterial peritonitis*
SVR	*Sustained virologic response*
TE	*Transient elastography*
TE	*Transient Elastography*
TIMP-1	*Tissue inhibitor of metalloproteinase 1*
WHVP	*Wedged hepatic venous wedged hepatic venous pressure*

INTRODUCTION

Hepatitis C virus (HCV) is a major global health problem. The predestined prevalence of hepatitis C virus (HCV) in 2015 was (1.0 %) (71.1) million individuals of them are chronically infected. Viral hepatitis was rated to be the seventh leading cause of mortality worldwide, but it is estimated that only 20% of individuals with hepatitis C know their diagnosis, and only 15% of those with known hepatitis C have been treated. In many cases, it is attributed to (HCV), which is the main causes of liver fibrosis, cirrhosis and cancer worldwide, however, Egypt own the highest prevalence rate in the world ***(Mohd-Hanafiah et al., 2013; Spearman et al., 2019).***

It is universally accepted that the widespread of infection in Egypt was due to governmental implementation of mass population anti-schistosomal treatment with "tartar emetic injections" (took place from 1950s to 1980s) beside the usual modes of transmission, such as IV drug usage, shared or reused needles, poorly sterilized surgical or dental equipment, blood transfusions and vertical transmission ***(Elgharably et al., 2017)***.

A synergistic effect may complicate the course of HCV and schistosomiasis, vice versa. A long-term study showed that complications observed more frequently in those with coinfection with around 48% having cirrhosis ***(Elgharably et al., 2017)***.

It's estimated that 75–80% of HCV infected individuals will progress to chronic hepatitis C after exposure and about 15-30% of them will progress to cirrhosis and hepatic decompensation with or without hepatocellular carcinoma (HCC). Around 15–20% will die during the first year following decompensation ***(Spearman et al., 2019).***

Acute (HCV) infection is typically asymptomatic & anicteric. Only 25% of cases are clinically manifested. However, symptoms, if existed, become apparent 2–26 weeks after HCV exposure, and the acute hepatitis lasts 2–12 weeks. Hepatitis C antibodies started to emerge within 12 weeks of infection, as HCV RNA is detectable before antibodies, it was used in diagnosis of acute viral hepatitis C. Fulminant hepatitis is rare condition which resample (<1%), and associated chronic hepatitis B infection, HIV co-infection, and concomitant immunosuppression are risk factors for the development of this condition ***(Spearman et al., 2019).***

It is necessary to harbor the disease between 20–40 years and therefore has a chronic HCV infection. After that,

a noticeable symptoms or signs will occur. HCV is a significant precursor for fibrosis, cirrhosis, and ultimately, hepatocellular carcinoma in long-term manner especially in chronic cases.