

بسم الله الرحمن الرحيم



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شبكة المعلومات الجامعية التوثيق الالكتروني والميكرونيلم





جامعة عين شمس

التوثيق الإلكتروني والميكروفيلم

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بالرسالة صفحات لم ترد بالأصل





Incidence of Reactivation of Hepatitis B in Co-infected HB V/HCV patients treated with Regiments of Sofosbuvir and Daclatasvir with or without Ribavirin

Thesis

For Partial Fulfilment of Master Degree in Gastroenterology and Hepatology Medicine

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List of Abbreviations

Abb.	Full term
AASLD	American Association for the Study of Liver
	Diseases
<i>ALT</i>	Alanine transaminase
$anti ext{-}HBc$	Hepatitis B core antigen
<i>DAA</i>	Direct-acting antiviral
<i>EASL</i>	European Association for the Study of the Liver
<i>GLE</i>	Glecaprevir
HBsAg	Hepatitis B surface antigen
<i>HBV</i>	Hepatitis B virus
HCV	Hepatitis C virus
<i>LFTs</i>	Liver function tests
<i>NCCVH</i>	National Committee for Control of Viral
	He patitis
<i>PIB</i>	Pibrentasvir
<i>RAS</i>	$Resistance - associated \ substitutions$
<i>RBV</i>	Ribavirin
SOF	So for buvir
<i>VEL</i>	Velpatas vir
<i>VOX</i>	Voxilaprevir

Introduction

pidemiological studies suggest that approximately 240 million people worldwide are infected with the hepatitis B virus (HBV) (*WHO*, 2015).

The clinical course of HBV infection varies and may include asymptomatic carriage, acute hepatitis or chronic active hepatitis, which can progress to cirrhosis and hepatocellular carcinoma (*Ott et al.*, 2012).

HBV reactivation is characterized by an increase of HBV DNA in the serum and is well documented in patients with previously undetected HBV DNA, due to inactive or resolved HBV infection (*Bessone et al.*, 2016).

Reactivation is usually followed by the reappearance of HBV activity (HBV DNA recurrence or increase > 1 log) or a flare of hepatitis in previously minimal or inactive disease (*Hoofnagle et al.*, 2009).

HBV reactivation mainly occurs in patients who receive immunosuppressive or cytotoxic chemotherapy for various malignancies (*Huang et al.*, 2012) and autoimmune diseases (*Kato et al.*, 2011) or in patients who undergo solid organ transplantation (*Aggeletopoulou et al.*, 2017).

The reappearance of serum HBV DNA has also occasionally been observed in patients with the hepatitis B

surface antigen (HBsAg) seroclearance, suggesting that HBV reactivation occurs in patients who have recovered from hepatitis B and have antibodies for the hepatitis B core antigen (anti-HBc) but have no detectable serum HBsAg (*Terrault et al.*, 2016).

Hepatitis with alanine transaminase (ALT) elevation due to reactivation of HBV has been reported in HCV/HBV coinfected patients who were treated with direct-acting antiviral (DAA) regimens for chronic hepatitis C virus (HCV) infection (*Takayama et al.*, 2016).

Patients with HCV/HBV coinfection have a significantly higher risk of developing hepatic cirrhosis and hepatocellular carcinoma (*Kruse et al.*, 2014).

In HCV/HBV coinfection, HCV has been shown to suppress HBV replication, resulting in decreased HBV antigen levels (*Collins et al.*, 2015).

The administration of HCV treatment may lead to an increase in HBV replication and in lower rates of HBV DNA clearance. Pegylated interferon-based treatments could suppress both HCV and HBV replication (*Konstantinou et al.*, 2015).

However, the development of interferon-free treatments with DAA agents has changed the course of treatment for chronic HCV infection (*Alexopoulou et al.*, 2015).