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Knowledge and awareness of hepatitis B infection in a sample of Iraqi dental students

A Project

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CHAPTER ONE

REVIEW OF LITERATURE

Introduction

Hepatitis B is an acute systemic infection which mainly affects the liver and is transmitted usually by the parenteral route. It begins as an acute self-limiting infection, which may be either subclinical or symptomatic. Persistent hepatitis B virus infection may cause progressive liver disease including chronic active hepatitis and hepatocellular carcinoma (Dientardt , 1983).

Hepatitis B is endemic throughout the world, especially in tropical and developing countries. More than 2 billion people have evidence of past or current hepatitis B virus infection; 350 million are chronic carriers; 600000 deaths result annually from cirrhosis and hepatocellular carcinoma (WHO. Hepatitis B vaccines , 2009).

In India the prevalence of HBsAg among the general population ranges from 2 to 8%, which puts India in intermediate position of endemic zone Occupational exposure to hepatitis B is a well-recognized risk of health care workers who report 800000 cut and puncture injuries per year (Previsani et al., 2002).

Aims and objectives :

1. To assess the degree of awareness regarding hepatitis B infection among dental college students.

2. To compare gender differences on hepatitis B knowledge and attitudes.

Hepatitis 1.1

It is a systemic and viral infection, in which the inflammation of liver cells and necrosis occurn and these changes lead to biochemical and cellular clinical alterations. Literally, hepatitis means the "inflammation of liver (Smith and Watson , 2005)

The most important factors causing it are viruses, Alcoholic drinks, medicines, poisons and some hereditary diseases also bring about hepatitis (Smeltzer et al. , 2010).

In acute hepatitis, the diseases lasts for less than 6 months and finally leads to the complete removal of liver damage and the return of liver structure and function to normal level or leads to the immediate progression of acute damage toward extensive necrosis and the demise of patient. However, in chronic hepatitis, the process of disease lasts more than 6 months and patients does not have clinical symptoms (Carpenter and Lederman , 2001).

Most patients suffering from viral hepatitis do not have any symptoms and their disease is diagnosed by doing medical tests. Nonetheless, the acute symptoms of disease exist in some patients (Alavian , 2012).

Following the contact with the virus causing hepatitis and going through a period, which varies from a week to a few months and is called the latent period, the acute symptoms of suffering from viral hepatitis would emerge, which include: lack of appetite, excessive fatigue, exhaustion and vomit, abdominal pain, darkening of urine, paling of stool of skin into yellow (Kalantari , 2011).

The symptoms of the disease usually last for days or weeks and its symptoms would be eradicated automatically. It must be attended that symptoms resembling influenza could be seen as muscular pain, exhaustion, and slight fever days or weeks before the emergence of the disease acute symptoms (Smeltzer et al. , 2010).

1.2 Epidemiology

The Centers for Disease Control and Prevention (CDC) conducts national surveillance for acute and chronic hepatitis infection. Data on chronic infections is limited because not all states report this information: For the year 2014, the CDC received reports on chronic hepatitis B virus (HBV) infection from 40 states and on chronic hepatitis C infection from 37 states (Klevens et al. , 2014).

Beginning in 2011, the CDC incorporated a new method for estimating the number of cases of hepatitis infection to better account for underreporting . Between 2011 and 2013, however, the number of reported hepatitis A cases increased, including a large hepatitis A outbreak in 2013 due to imported pomegranate seeds (Klevens et al., 2014).

After adjusting for underreporting and under ascertainment, the CDC estimates that the actual number of new hepatitis A cases in 2014 was 2500. For HBV infection, rates of reported acute infections have been declining since 1990. There were 2791 cases of acute infection in 2014 (Klevens et al. , 2014).

With correction for asymptomatic cases and underreporting, the true number of cases of acute hepatitis B infection in 2014 was estimated at 18,100.The CDC estimates that approximately 850,000 to 2.2 million people in the U.S are chronically infected with HBV. Over 70% of these infections occurred in foreign-born individuals, and over half of the chronic infections occurred in individuals identifying as Asian/Pacific Islanders. The annual number of reported cases of acute hepatitis C increased steadily between 2010 and 2014 (Klevens et al. , 2014).

There were 2194 case of acute hepatitis C infection reported in 2014; after adjusting for underreporting, the CDC estimates that there were 30,500 new infections in 2014 (Edlin et al. , 2015).

Approximately 2.7-3.9 million people In the United States have chronic hepatitis C (Edlin et al. , 2015).

1.3 Etiology

Hepatitis viruses A, B, C, D (HAV, HBV, HCV, HDV [which requires coexisting HBV infection]), and E (HEV) cause the majority of clinical cases of viral hepatitis. Whether hepatitis G virus (HGV) is pathogenic in humans remains unclear. HAV, HBV, HCV, and HDV are the only hepatitis viruses endemic to the United States; HAV, HBV, and HCV are responsible for more than 90% of US cases of acute viral hepatitis. Whereas HAV and HBV are the most common causes of acute

hepatitis in the United States, HCV is the most common cause of chronic hepatitis (Adhami and Levinthal , 2002).

HAV infection occurs throughout the world, the risk is highest in developing countries, areas of low socioeconomic status, and regions without sufficient sanitation. Higher infection rates also exist in settings where fecal-oral spread is likely, such as daycare centers (Purcell et al ., 2002).

Other groups at high risk for HAV infection include international travelers, users of injection and noninjection drugs, and men who have sex with men (World Health Organization , 2016).

HBV is transmitted both parenterally and sexually, most often by mucous membrane exposure or percutaneous exposure to infectious body fluids. Saliva, serum, and semen all have been determined to be infectious (Adhami and Levinthal , 2002).

Percutaneous exposures leading to the transmission of HBV include transfusion of blood or blood products, injection drug use with shared needles, hemodialysis, and needlesticks (or other wounds caused by sharp implements) in healthcare workers
(Adhami and Levinthal , 2002).

Groups at high risk for HBV infection include intravenous (IV) drug users, persons born in endemic areas, and men who have sex with men (Adhami and Levinthal , 2002).

Others at risk include healthcare workers exposed to infected blood or bodily fluids, recipients of multiple blood transfusions, patients undergoing hemodialysis, heterosexual persons with multiple partners or a history of sexually transmitted disease, institutionalized persons (eg, prisoners), and household contacts or sexual partners of HBV carriers (Adhami and Levinthal , 2002).

HCV can be transmitted parenterally, perinatally, and sexually. Transmission occurs by percutaneous exposure to infected blood and plasma (Adhami and Levinthal , 2002).

The virus is transmitted most reliably through transfusion of infected blood or blood products, transplantation of organs from infected donors, and sharing of contaminated needles among IV drug users (Adhami and Levinthal , 2002).

Modes of transmission for HDV are similar to those for HBV. HDV is transmitted by exposure to infected blood and blood products. It can be transmitted percutaneously

and sexually (Adhami and Levinthal , 2002).

1.4 Pathophysiology

The incubation period of HBV infection is 40-150 days (average, approximately 12 weeks). As with acute HAV infection, the clinical illness associated with acute HBV infection may range from mild disease to a disease as severe as FHF (<1% of patients). After acute hepatitis resolves, 95% of adult patients and 5-10% of infected infants ultimately develop antibodies against hepatitis B surface antigen (HBsAg)—that is, anti-HBs—clear HBsAg (and HBV virions), and fully recover. About 5% of adult patients, 90% of infected infants, and 30-50% of children infected at age 1-5 years develop chronic infection (Klevens et al. , 2014).

Some patients, particularly individuals who are infected as neonates or as young children, have elevated serum levels of HBV DNA and a positive blood test for the presence of HBeAg but have normal alanine aminotransferase (ALT) levels and show minimal histologic evidence of liver damage. These individuals are in the so-called "immune-tolerant phase" of disease (Keeffe et al ., 2008 and Sorrell et al ., 2009).

Years later, some but not all of these individuals may enter the "immune-active phase" of disease, in which the HBV DNA may remain elevated as the liver experiences active inflammation and fibrosis. An elevated ALT level is also noted during this period. Typically, the immune-active phase ends with the loss of HBeAg and the development of antibodies to HBeAg (anti-HBe) (Keeffe et al ., 2008 and Sorrell et al ., 2009).

Patients with HBeAg-positive chronic hepatitis have signs of active viral replication, with an HBV DNA level greater than 2×10^4 IU/mL (McMahon , 2009 and Keeffe et al ., 2008).

Patients with HBeAg-negative chronic hepatitis were presumably infected with wild-type virus at some point. Over time, they acquired a mutation in either the precore or the core promoter region of the viral genome. In such patients with a precore mutant state, HBV continues to replicate, but HBeAg is not produced. Patients with a core mutant state appear to have downregulated HBeAg production (Keeffe et al ., 2004).

The vast majority of patients with HBeAg-negative chronic hepatitis B have a serum HBV DNA level greater than 2000 IU/mL. Typically, HBeAg-negative patients have lower HBV DNA levels than HBeAg-positive patients do. Commonly, the HBV DNA level is no higher than 2×10^4 IU/Ml (McMahon , 2009 and Keeffe et al ., 2008).

In HBV-induced cirrhosis, as in cirrhosis due to other causes, hepatic inflammation and regeneration appear to stimulate mutational events and carcinogenesis. However, in HBV infection, in contrast to other liver diseases, the presence of cirrhosis is not a prerequisite for the development of HCC. The integration of HBV into the hepatocyte genome may lead to the activation of oncogenes or the inhibition of tumor suppressor genes. As an example, mutations or deletions of the *p53* and *RB* tumor suppressor genes are seen in many cases of HCC (Tabor , 1997).

Simultaneous introduction of HBV and HDV into a patient results in the same clinical picture as acute infection with HBV alone. The resulting acute hepatitis may be mild or severe. Similarly, the risk of developing chronic HBV and HDV infection after acute exposure to both viruses is the same as the rate of developing chronic HBV infection after acute exposure to HBV (approximately 5% in adults (World Health Organization , 2016).

Chronic HBV and HDV disease tends to progress more rapidly to cirrhosis than chronic HBV infection alone does (World Health Organization , 2016).

Acute HEV infection is generally less severe than acute HBV infection and is characterized by fluctuating aminotransferase levels. However, pregnant women, especially when infected during the third trimester, have a greater than 25% risk of mortality associated with acute HEV infection (Kumar et al ., 2004).

1.5 Types of hepatitis

Five types of hepatitis have been found; A, B, C, D, and E. types A and E are akin with regard to the way of transmission (the stool-oral path). Types B, C and D are also similar to each other (Neighbors et al., 2007).

1.5.1Viral hepatitis, type A

This type is more common in developing countries or in crowded places with poor hygienic conditions. Its transmission way is through oral-stool path. Individuals are afflicted with it by having water and seafood tainted with sewage. This disease is not transmitted through blood but it is likely to be transmitted via sexual contact and having oral-anal contact (Black and Hawaks, 2009).

The common course of the disease is 15 to 150 days and it could lat from 4 to 8 weeks. This disease does not have chronic and carrier courses. In case of the emergence of jaundice, the disease is not probably infectious (Smeltzer et al., 2010).

The diagnosis method is based on symptoms, signs, physical examination and blood tests. Blood test usually remains as positive 5 days prior to the onset of symptoms up to 6 months after infection (Chord , 2002).

Individuals must be reminded that this type of hepatitis does not have any treatment, although most individuals might recuperate by supportive treatments. A person with hepatitis A virus requires in-hospital treatment for liver functioning. The patient must be trained to start walking slowly and progressively at once after feeling recuperated and to take rest after each time of walking (Smeltzer et al. , 2010).

1.5.2 Hepatitis E

This type is transferred via the oral-stool tract and mostly due to polluted water in areas that have weak and inappropriate sewage systems

It common period is variable and lasts between 15 to 65 days

It is akin to Hepatitis A with regard to clinical symptoms and transferring ways.

Jaundice almost always exists and avoidance from virus is made possible through rinsing hands as the main prevention method of this disease. The effectiveness of immune globins increating immunity against Hepatitis E is unknown. The clinical symptoms of hepatitis A and E are like influenza, appetite loss, indigestion, epigastria pain, vomiting, and irritation of stomach and acidity of abdomen alongside hatred of cigarette smoke (Smeltzer et al. , 2010).

Patients must have enough rest at the chronic phase of the disease. Patients can gradually resume their activities after feeling healthy and examining their laboratorial tests. However, after being active, they ought to rest and avoid taking part in drudgeries (Ignatavicius and Workman, 2006).

Rinsing and disinfecting fruits and vegetables, not using polluted water and foods (boiling water for one minute deactivates hepatitis virus) and not using personal things of others (Alavian et al. , 2008).

1.5.3 Hepatitis B

This type is transferred via blood (sub-phlegm and endodermal ways).

Its virus could be found in blood, saliva, semen and vaginal discharges and can be transferred via the phlegmatic membrane and

damaged skin. Also, pregnant mothers could be afflicted through sexual contact after or during delivery (Black and Hawaks, 2009).

Duration:

Its common course lasts from 1 to 6 months (Smeltzer et al., 2010).

Symptom:

The symptoms and signs of hepatitis B might be quite arcane, deceitful and variable. Respiratory and fever-related symptoms are rare. Some patients suffer from rash and joint pain. Patients might lose their appetite and suffer from indigestion, abdominal pain, general pain, ill health and weakness. Jaundice might occur or not. In case of its occurrence, it would be accompanied by light color stool and dark urine. Clinically, it has a lot of similarities with hepatitis A but its common course is longer (Osborn et al. , 2010).

More than 90 percent of infections can be prevented in women suffering from HBV and their infants can receive the hepatitis B vaccine and immune globins B in prophylaxis manner for preventing infection. Babies must undergo serological test within 9 to 15 months of age (Smith et al. , 2005).

It must be mentioned that at-risk individuals might be afflicted with the virus of hepatitis B via using polluted blood products, contact of polluted blood with eyes, mouth, bleeding or damaged skin of healthy people, transfer from mother to fetus, tattooing and cupping, sexual contact, non-sterile dental and medical devices, esp. shared and non-sterile syringe, razorblade or toothbrush. This virus is not transferred through shaking hand, kissing or going to pool (Mehrvarz , 2006).

Cautions:

These patients must be to have insight about their disease and to know the transmission ways of this disease. Knowing this disease affect the quality of life among individuals and their families (Samiei et al. , 2005).

Infant born from a mother infected with hepatitis virus B ought to receive immune globins within 12hours after birth (Black and Hawaks, 2009).

An individual who has been at exposure to hepatitis virus B and has not ever been afflicted with hepatitis and has not received vaccine must receive the immune globins injection at most 24 hours after contact with the virus (Neighbors et al. , 2007).

1.5.4 Hepatitis C

A significant percentage of hepatitis cases are neither A, B nor D.

Consequently, they are referred to as hepatitis type C. Hepatitis C is transmitted through blood, sexual contact, using septic syringe by the applicators of injective, intravenous medications, the abrupt penetration of needle head and other kinds of injuries among health care personnel (Lewis ShHeitkemper et al. , 2007).

Duration:

The latent course of this disease varies from 2 to 26 weeks. Most patients do not show any overt symptoms. In case the virus exists in the individual's body for more than 6 months and gradually damages the tissues of liver, the chronic hepatitis C emerges (Mehrvarz , 2006).

Symptoms:

Patient might complain about slight to severe lethargy and weakness, decrease in appetite, spew and vomit, having pain at the upper and right sides of abdomen (below ribs), fever and pain in joints (Hajrasooliha et al. , 2004).

Treatment:

One of the main treatments about hepatitis C is taking the medication group of interferon. Throughout undergoing treatment with interferon, patients must be examined closely by virtue of the possible side effects. Therefore, immediate monitoring while undergoing treatments is viable for reducing these side effects to some extent (Smeltzer et al. ,2010).

Cautions:

The hepatitis virus type C is transmitted through using septic blood products, the contact of septic blood with eyes, healthy individuals' mouth and injured skin, toothbrush, shaving machine and all septic personal things, tattooing and cupping, sexual contact, using septic dental and medical devices, esp. non-sterile and shared syringe. Hence, it is recommended to observe the personal hygienic points including of sterilizing the devices used in ear perforation and tattooing, using gloves and rinsing hands and not using others' personal devices. It is also recommended that individuals use condom when having sexual contact with the afflicted person (Hajrasooliha , 2004).

They are also advised to follow the hygiene of mouth and teeth. Cutting down on sugary drinks and foods, using florid supplements once a day, using floss, snit-microbe mouthwash, reducing cigarette and quitting it, regular examination of teeth, their whitening and taking care of false teeth are among the tips given to these patients (Black and Hawaks, 2009).

The individuals who are afflicted with Hepatitis C are recommended to inform the personnel at medical centers, dentist's, laboratories or nay other places which entails the possibility of virus transmission (Hajrasooliha, 2004).

1.5.5 Hepatitis D

In some cases, hepatitis B takes place due to the fact that its virus is at exposure with the superficial antigen and risk of hepatitis D. Anti-delta antibodies within the antigen in the blood sample ascertains the existence of hepatitis B. hepatitis D is prevalent among the users of injective, intravenous medications, hem dialysis patients and receivers of various blood donations.

Duration:

Its common course is between 21 to 40 days.

Symptom:

The symptoms of hepatitis D are akin to hepatitis B and its treatment is similar to all other forms of hepatitis (Smeltzer et al. , 2010).

1.6 Acute hepatitis symptom

Pain at the right and the upper abdominal area is due to the exposure of liver capsule to traction and it is removed by the amelioration of the disease. Getting jaundiced means the yellowness of the skin and eyes. It must be attended that most patients suffering from hepatitis A and C do not have any symptoms of acute hepatitis, esp. jaundice. Some patients complain of bodily irritation which could be rooted in liver damage. Joint-related pain is also a part of acute hepatitis syndromes (Gothomas et al. , 2005).

Symptoms of all types of viral hepatitis are similar and can include one or more of the following: • Fever • Fatigue • Loss of appetite • Nausea • Vomiting • Abdominal pain • Gray-colored bowel movements • Joint pain • Jaundice (Smeltzer et al ., 2010).

1.7 Clinical Presentation

The incubation period of hepatitis A virus (HAV) is 2-7 weeks (average, 28 days). Clinical symptoms then develop, often with a presentation similar to that of gastroenteritis or a viral respiratory infection. The most common signs and symptoms include fatigue, nausea, vomiting, fever, hepatomegaly, jaundice, dark urine, anorexia, and rash.

HAV infection usually occurs as a mild self-limited disease and confers lifelong immunity to the virus. Chronic HAV infection does not occur (Klevens et al. , 2014).

The incubation period for hepatitis B virus (HBV) is 30-180 days (average, approximately 75 days). Patients then enter the prodromal or preicteric phase, characterized by the gradual onset of anorexia, malaise, and fatigue. During this phase, as the liver becomes inflamed, the liver enzymes start to elevate, and the patient may experience right upper quadrant pain. About 15% of patients develop an illness resembling serum sickness. These patients may experience fever, arthritis, arthralgias, or an urticarial rash (Klevens et al., 2014).

The incubation period for hepatitis C virus (HCV) is 15-150 days, with symptoms developing anywhere from 5-12 weeks after exposure. During acute HCV infection, symptoms may appear similar to those of HBV infection. In up to 80% of cases, however, patients are asymptomatic and do not develop icterus (Wasley et al ., 2006).

The incubation period of hepatitis D virus (HDV) is approximately 35 days. Patients simultaneously infected with HBV and HDV often have an acute, self-limited infection (Adhami and Levinthal , 2002 and Previsani and Lavanchy , 2001).

Chronic HBV carriers who become superinfected with HDV tend to have a more severe acute hepatitis; 80% of these patients go on to develop chronic HDV infection. Chronic infection with HBV and HDV may lead to fulminant acute hepatitis and severe chronic active hepatitis with progression to cirrhosis (Adhami and Levinthal , 2002 and Previsani and Lavanchy , 2001).

The incubation period of hepatitis E virus (HEV) is 2-9 weeks (average, 45 days). HEV usually causes an acute self-limited disease

similar to HAV infection. Fulminant disease does occur in about 10% of cases. In women who are pregnant, HEV infection has a case-fatality rate of 15-20%. No reports exist of chronic infection with HEV (Adhami and Levinthal , 2002).

1.8 ORAL MANIFESTATIONS

The oral cavity can reflect liver dysfunction in the form of mucosal membrane jaundice, bleeding disorders, petechiae,increased vulnerability to bruising, gingivitis, gingival bleeding The main disorders associated with HCV infection are xerostomia, Sjögren's syndrome (SS), sialadenitis and particularly lichen planus (LP) (Grossmann et al ., 2009).

Epidemiological evidence suggests that lichen planus may be significantly associated to HCV infection, though the existing data are controversial (Ismail et al ., 2007).

In a study, patients with periodontal disease showed a higher detectability rate of Hepatitis B surface antigen HBsAg, Hepatitis B core antigen antiHBc, antiHCV in whole unstimulated saliva than in the controls and there by suggesting a possible association between hepatitis and oral lesions (Wedemeyer et al ., 1999).

Certain lesions in the oral cavity may be primarily related to dysfunction of the hepatocyte. There may be extraoral and/or intraoral petechiae and eccyhmoses, gingival hemorrhage due to the deficient clotting factors associated with malfunctioning hepatocytes and thrombocytopenia. Additional oral findings like pallor, angular cheilitis and glossitis can include manifestations of malnutrition such as vitamin

deficiencies and anemia. Additionally, the sweet ketone breath, indicative of liver gluconeogenesis, can raise the suspicion of hepatotoxicity (Omagari et al ., 2002).

Dryness of mouth results as an adverse effect of medications taken and may be related to virus associated salivary gland changes. It increases patient vulnerability to caries and oral soft tissue disorders which, in combination with deficient hygiene, in turn facilitate the development of candidiasis (Carrozzo , 2008).

A high prevalence of oral lichen planus in HBsAg positive patients was found.15 However, many studies and reports have suggested the role of HCV as a possible etiology. Virus replication may be associated with the oral epithelium and thus contributes directly to the development of lesions, or otherwise high mutation rate of the virus may result in repeated activation of immune cells, increasing the probability of cross-reactions and consequently the risk of autoimmune disease (Blonski et al ., 2010).

The role of HCV in pathogenesis of Sjogren Syndrome (SS) development and the characteristics distinguishing classic SS from HCV-related sialadenitis are still an issue (Grossmann et al ., 2009).

A study conducted in New Orleans reported that 21.2% of 99 patients with squamous cell carcinoma of the head and neck were co-infected with HCV (Lockhart et al ., 2003).

1.9 Complications

One of the major complications of hepatitis B is the development of chronic infection. An estimated 240 million people worldwide are chronically infected with HBV (Gilks et al ., 2006).

Patients with such infection are at risk for the subsequent development of chronic active hepatitis, cirrhosis of the liver, and eventual HCC. Each year, approximately 650,000 deaths occur worldwide as a result of chronic HBV infection (Gilks et al ., 2006).

Patients with chronic HBV infection are at significantly higher risk for HCC. In fact, HCC is the leading cause of cancer-related deaths in areas where HBV is endemic. Globally, HBV is responsible for 45% of the world's primary liver cancers (Gilks et al ., 2006).

Another major complication of HBV infection is development of FHF. In approximately 0.5-1% of HBV-infected patients, the disease progresses to FHF, with coagulopathy, encephalopathy, and cerebral edema. The case-fatality rate for these patients approaches 80% (Previsani and Lavanchy , 2002).

Cirrhosis related to chronic HCV infection is also strongly linked to the development of HCC, which usually develops after 30 years in patients who are chronically infected. Of patients with HCV-associated cirrhosis, 20-25% may progress to liver failure and death (Previsani and Lavanchy , 2002).

As noted earlier, in the United States, cirrhosis associated with chronic hepatitis C is a leading indication for liver transplantation (Edlin et al ., 2015).

No well-established antiviral therapy is available for acute hepatitis B virus (HBV) infection. Supportive treatment recommendations are the same for acute hepatitis B as for acute hepatitis A. Lamivudine, adefovir dipivoxil, and other antiviral therapies appear to have a positive impact on the natural history of severe cases of acute HBV infection. A study by Schmilovitz-Weiss described a rapid clinical and biochemical response in 13 of 15 patients with severe acute hepatitis B who received lamivudine (Schmilovitz-Weiss et al ., 2004).

Ideally, treatment of chronic hepatitis B would routinely achieve loss of hepatitis B surface antigen (HBsAg). Indeed, loss of HBsAg is associated with a decreased incidence of hepatocellular carcinoma (HCC) and a decreased incidence of liver-related death in patients with hepatitis B virus (HBV)-induced cirrhosis (Fattovich et al ., 1998).

Acute hepatitis C virus (HCV) infection is detected infrequently. When it is identified, early interferon (IFN) therapy should be considered. In one study, 44 patients with acute hepatitis C were treated with IFN alfa-2b at 5 million U/day subcuteaneously (SC) for 4 weeks and then three times per week for another 20 weeks (Jaeckel et al ., 2001).

About 98% of patients developed a sustained virologic response (SVR), defined as an undetectable level of serum HCV RNA 6 months after completion of antiviral treatment. Most experts now equate achievement of an SVR with viral eradication or cure of HCV infection (Jaeckel et al ., 2001 and Nelson et al ., 2009).

Treatment of patients coinfected with hepatitis B virus (HBV) and hepatitis delta virus (HDV) has not been well studied. The onl effective treatment for HBV/HDV coinfection is pegylated interferon (PEG-IFN) (Terrault et al ., 2016).

Lamivudine appears to be ineffective against HBV-HDV coinfection (Lau et al ., 1999 and Yurdaydin et al., 2008).

1.10 Prognosis

Hepatitis A virus (HAV) infection usually is mild and self-limited, and infection confers lifelong immunity against the virus. Overall mortality is approximately 0.02% (Klevens et al ., 2014).

Chronic infection develops in 50-60% of patients with hepatitis C. Chronically infected patients are at risk for chronic active hepatitis, cirrhosis, and HCC. In the United States, chronic HCV infection is the leading indication for liver transplantation (Edlin et al ., 2015).

Chronic coinfection with HBV/HDV often leads to rapidly progressive subacute or chronic hepatitis, with as many as 70-80% of these patients eventually developing cirrhosis (Klevens et al ., 2014).

CHAPTER TWO

Materials and Methods

Methods:

2.1 Study design and population:

This study was a cross sectional study among dental students of fifth class at college dentistry , university of Baghdad Study period from February till march 2018.

2.2 Study tool:

The study tool used was a pre-formed self-administered structured questionnaire. The questionnaire included questions on various aspects of hepatitis B infection such as causative organism, routes of transmission, at-risk groups, screening method, hepatitis B vaccine, study participants' vaccination status, other types of hepatitis infections and complications of hepatitis as well as questions on attitude towards hepatitis B infected patients (Previsani et al ., 2002).

2.3 Data collection method:

All the students who gave consent for the study were administered the preformed structured questionnaire. Students were asked to raise queries if they did not understand the questions in the questionnaire.

2.4 Data entry and analysis:

The collected data were entered in microsoft excel sheet and data analysis was done using SPSS - 20 software. Data were expressed in frequencies , percentages , mean , standared deviation .

CHAPTER THREE

RESULTS

3.1 Study sample

The total number of dental students were (200) from fifth class only 153 students accepted to participate with . in this study the number of female were (98)(64%) while male were (55)(35%) with mean age (21.97)(0.56) as show in the table (3-1).

Table (3-1) gender distribution among dental students

	number	percentage %
male	55	35%
female	98	64%

3.2 The answers of knowledge questions

In this study it was found that the number of dental students were answered with (yes) for question one were 78(50.9%) while the students that answered (no) were 27(17.6%) and the students that have no idea were 48(31.3%), the number of dental students were answered with (yes) for question two were 2(1.3%) while the students that answered (no) were 13(8.4%) and the students that have no idea were 138(90.1%), the number of dental students were answered with (yes) for question three were 22(14.3%) while the students that answered (no) were 110(71.8%) and the students that have no idea were 20(13%), the number of dental

students were answered with (yes) for question four were 150(98%) while the students that answered (no) were 2(1.3%) and the students that have no idea were 1(0.6%), the number of dental students were answered with (yes) for question five were 22(14.3%) while the students that answered (no) were 34(22.2%) and the students that have no idea were 97(63.3%), the number of dental students were answered with (yes) for question six were 15(9.8%) while the students that answered (no) were 29(18.9%) and the students that have no idea were 109(71.2%), the number of dental students were answered with (yes) for question seven were 102(66.6%) while the students that answered (no) were 14(9.1%) and the students that have no idea were 37(24.1%), the number of dental students were answered with (yes) for question eight were 125(81.6%) while the students that answered (no) were 11(7.1%) and the students that have no idea were 17(11.1%), the number of dental students were answered with (yes) for question nine were 67(43.7%) while the students that answered (no) were 14(9.1%) and the students that have no idea were 93(60.7%), the number of dental students were answered with (yes) for question ten were 64(41.8%) while the students that answered (no) were 4(2.6%) and the students that have no idea were 85(55.5%), the number of dental students were answered with (yes) for question eleven were 90(58.8%) while the students that answered (no) were 17(11.1%) and the students that have no idea were 47(30.7%), the number of dental students were answered with (yes) for question twelve were 10(6.5%) while the students that answered (no) were 12(7.8%) and the students that have no idea were 26(16.9%), the number of dental students were answered with (yes) for question thirteen were 142(92.8%) while the students that answered (no) were 3(1.9%) and the students that have no idea were 8(5.2%) as show in table (3-2).

Table (3-2) the answers of knowledge questions according to the dental students

Questions regarding knowledge	Yes	%	No	%	Idont know	%
1. Hepatitis B virus (HBV) is a member of the Hepa DNA virus family:	78	50.9 %	27	17.6%	48	31.3 %
2. HBV has a diameter of 42 nm:	2	1.3%	13	8.4%	138	90.1 %
3. There are four genotypes of HBV:	22	14.3%	110	71.8%	20	13%
4. Hepatitis B is an infectious illness caused by hepatitis B virus (HBV):	150	98%	2	1.3%	1	0.6%
5. Hepatitis B virus is a non-retroviral virus:	22	14.3%	34	22.2%	97	63.3 %
6. HBV is one of the smallest enveloped animal ruses:	15	9.8%	29	18.9%	109	71.2 %
7. Hepatitis B infection was originally known as serum hepatitis:	102	66.6%	14	9.1%	37	24.1 %
8. The acute illness causes liver inflammation:	125	81.6%	11	7.1%	17	11.1 %
9. The time between the removal of the HBS Ag and the appearance of anti-HBs is called the window period:	67	43.7%	14	9.1%	93	60.7 %
10. PCR tests have been developed to detect and measure the amount of HBV DNA, called the viral load, in clinical specimens:	64	41.8%	4	2.6%	85	55.5 %
11. The vaccine is administered in either two-,three, or four-dose schedules into infants and adults, which provides protection for 85-90% of individuals:	90	58.8%	17	11.1%	47	30.7 %
12. Individuals who remain HBs Ag positive for at least 6 months are considered to be hepatitis EB carriers:	115	6.5%	12	7.8%	26	16.9 %
13. The hepatitis B surface antigen (HBs Ag) is most frequently used to screen for the presence of this infection:	142	92.8%	3	1.9%	8	5.2%

3.3 The answers of awareness questions

In this study it was found that the number of dental students were answered with (yes) for question one were 150(98%) while the students that answered (no) were 3(1.9%) and the students that have no idea were 0(0%), the number of dental students were answered with (yes) for question two were 147(96%) while the students that answered (no) were 6(3.9%) and the students that have no idea were 0(0%), the number of dental students were answered with (yes) for question three were 129(84.3%) while the students that answered (no) were 18(11.7%) and the students that have no idea were 6(3.9%), the number of dental students were answered with (yes) for question four were 102(66.6%) while the students that answered (no) were 42(27.4%) and the students that have no idea were 9(5.8%), the number of dental students were answered with (yes) for question five were 148(96.7%) while the students that answered (no) were 3(1.9%) and the students that have no idea were 2(1.3%), the number of dental students were answered with (yes) for question six were 142(92.8%) while the students that answered (no) were 3(1.9%) and the students that have no idea were 8(5.2%), the number of dental students were answered with (yes) for question seven were 132(86.2%) while the students that answered (no) were 12(7.8%) and the students that have no idea were 9(5.8%), the number of dental students were answered with (yes) for question eight were 138(90.1%) while the students that answered (no) were 7(4.5%) and the students that have no idea were 8(5.2%), the number of dental students were answered with (yes) for question nine were 123(80.3%) while the students that answered (no) were 23(15%) and the students that have no idea were 7(4.5%) as show in table (3-3).

Table (3-3) the answers of awareness questions according to dental students

Questions regarding awareness	Yes	%	No	%	Idont know	%
1.Transmission of hepatitis B virus results from exposure to infectious blood or body fluids	**150**	**98%**	**3**	**1.9%**	**0**	**0%**
2. Transmission of hepatitis B virus results from needle stick injuries	**147**	**96%**	**6**	**3.9%**	**0**	**0%**
3. Transmission of hepatitis B virus results from unprotected sexual contact	**129**	**84.3%**	**18**	**11.7%**	**6**	**3.9%**
4. Hepatitis B cannot be transmitted through hand shaking	**102**	**66.6%**	**42**	**27.4%**	**9**	**5.8%**
5. Hepatitis B infection is preventable by vaccination	**148**	**96.7%**	**3**	**1.9%**	**2**	**1.3%**
6. Chronic hepatitis infection results in liver cirrhosis and heap to cellular carcinoma	**142**	**92.8%**	**3**	**1.9%**	**8**	**5.2%**
7. Patients undergoing surgical dental procedures to be investigated for HBV	**132**	**86.2%**	**12**	**7.8%**	**9**	**5.8%**
8. The hepatitis B surface antigen (HBs Ag is most frequently used to screen for the presence of this infection)	**138**	**90.1%**	**7**	**4.5%**	**8**	**5.2%**
9. Dental personnel infected with hepatitis B should avoid patients' treatment, especially surgeries	**123**	**80.3%**	**23**	**15%**	**7**	**4.5%**

CHAPTER FOUR

Discussion

In the present study, 98% students knew that Hepatitis B is an infectious illness caused by HBV and about 92.8% of the students believed that the Hepatitis B surface antigen (HBsAg) was most frequently used for the screening of the presence of this infection this agree with(Tirounilacandin et al., 2009). About 58.8% of the participants believed that the vaccine is administered for protection from infection for 85-90% of individuals this disagree with (Tirounilacandin et al., 2009). In general, analysis of students indicates that they have relatively a good level of knowledge about HBV. Regarding awareness of students about the transmission of HBV. About 98% of students knew that transmission of HBV results from exposure to infectious blood or body fluids. Majority (96%) of the participants believed that transmission of HBV results from needle stick injuries, and patients undergoing surgical dental procedures to be investigated for HBV. Majority (96.7%) of the students knew that Hepatitis B infection is preventable by vaccination. The overall awareness of students is high as per the knowledge regarding knowledge of HBV and awareness about its transmission. Our study showed that 96.7% of the students were aware of HBV vaccine, which is more when compared with studies conducted among medical and dental interns in Arupadai Veedu Medical College and Mahatma Gandhi Dental College at Pondicherry, India, which showed that 92.9% of the interns were aware of immunization against HBV (Tirounilacandin et al., 2009). But the percentage is less when compared with the Department of Dental Surgery and Periodontology at University of Dundee, Dundee, which showed that 99.2% medical and dental students were aware of HBV immunization (Ogden et al., 1998)This research was conducted among 153

undergraduate students only; therefore one could argue that the findings are not necessarily a generalization of all the undergraduate students' knowledge and awareness about the same. There is an essential need for further education among the students to improve and update their knowledge of Hepatitis B, by conducting well-designed seminars, programs and workshops.

CHAPTER FIVE

Conclusion

1. In the present study 98% students knew that Hepatitis B is an infectious illness caused by HBV, while 90.1% students have no idea about the diameter of HBV

2. In the present study 98% students aware that Transmission of hepatitis B virus results from exposure to infectious blood or body fluids , while 0% students have no idea about the Transmission of hepatitis B virus results from needle stick injuries

References

A

1. Alavian, S. M. (2012). On the Occasion of the World Hepatitis Day: World Hepatitis Day and Our Achievements and Responsibilities in Iran. International Journal of Preventive
Medicine.

2. Adhami T, Levinthal G. Hepatitis E and hepatitis G/GBV-C. *The Cleveland Clinic Disease Management Project*. May 29, 2002.

3. Alavian, S. M, & Hajarizadeh, B. Ahmadzadeh Asl M, Kabir A. (2008). Hepatitis B virus infection in Iran:a systematic review. Hept mon.

B

4. Black, J, & Hawaks, J. (2009). *Medical- Surgical Nursing*. International: Sanders Elsevier.

5. Blonski W, Kotlyar DS, Forde KA. Non-viral causes of hepatocellular carcinoma.World J Gastroenterol. 2010;16:3603-15.

C

6. Carpenter CJCFlanigan TP,Lederman MM. ((2001). HIV infection and the acquired immunodeficiency syndrome. In: Cecil Essential of medicine. USA: W.B Saunders company.

7. Chord, L. A. J. ((2002). Forgeneral readers a comprehensive discussion of the causes of hepatitis and of the treatment. University press of Mississipi.

8. Carrozzo M. Oral diseases associated with hepatitis C virus infection. Part 1. sialadenitis and salivary glands lymphoma. Oral Dis. 2008; 14:123-30.

D

9. Di Marco V, Marzano A, Lampertico P, et al, for the Italian Association for the Study of the Liver (AISF) Lamivudine Study Group, Italy. Clinical outcome of HBeAg-negative chronic hepatitis B in relation to virological response to lamivudine. *Hepatology*. 2004 Oct. 40 (4):883-91.

10. Dientardt F. Sero-epidemiological study of hepatitis B. WHO Chr. 1983;37(6):203-4.

E

11. Edlin BR, Eckhardt BJ, Shu MA, Holmberg SD, Swan T. Toward a more accurate estimate of the prevalence of hepatitis C in the United States. Hepatology. 2015:62(5):1353-63.

F

12. Fattovich G, Giustina G, Sanchez-Tapias J, et al. Delayed clearance of serum HBsAg in compensated cirrhosis B: relation to interferon alpha therapy and disease prognosis. European Concerted Action on Viral Hepatitis (EUROHEP). *Am J Gastroenterol*. 1998 Jun. 93 (6):896-900.

G

13. Grossmann Sde M, Teixeira R, de Aguiar MC, de Moura MD, do Carmo MA. Oral mucosal conditions in chronic hepatitis C Brazilian patients: a cross-sectional study. J Public Health Dent. 2009; 69:168-75.

14. Gupta S, Gupta R, Joshi YK, Singh S. Role of horizontal transmission in hepatitis B virus spread among household contacts in north India. Intervirology. 2008;51:7-13.

15. Gothomas, H, Lemon, S, & Zuckerman, A. (2005). Viral hepatitis. Blackwell Publishing L.td.

.

H

16. Hajrasooliha, H. (2004). The manual for patient about the special aspects of hepatitis C, Tehran; Rezvan Publications in cooperation with the artistic group of Chakameh.

I

17. Ismail SB, Kumar SK, Zain RB. Oral lichen planus and lichenoid reactions: etiopathogenesis, diagnosis, management and malignant transformation. J Oral Sci. 2007; 49: 89-106.

18. Ignatavicius, D, & Workman, L. (2006). Medical- Surgical Nursing Critical thinking for collaborative care. International W.B. Saunders Company.

J

19. Jaeckel E, Cornberg M, Wedemeyer H, et al, for the German Acute Hepatitis C Therapy Group. Treatment of acute hepatitis C with interferon alfa-2b. *N Engl J Med*. 2001 Nov 15. 345 (20):1452-7.

K

20. Kalantari, H. (2011). Viral hepatitis. Journal of Isfahan Medical school.29(128).

21. Klevens RM, Kruszon-Moran D, Wasley AM, Gallagher K, McQuillan GM, Kuhnert WL, et al. Seroprevalence of hepatitis A antibodies in the United States: results from the National Health and Nutrition Examination Survey. Public Health Rep. 2014;126(4):522-32.

22. Keeffe EB, Dieterich DT, Han SH, et al. A treatment algorithm for the management of chronic hepatitis B virus infection in the United States. *Clin Gastroenterol Hepatol*. 2009 Feb. 2 (2):87-106.

23. Keeffe EB, Dieterich DT, Han SH, et al. A treatment algorithm for the management of chronic hepatitis B virus infection in the United States: 2008 update. *Clin Gastroenterol Hepatol*. 2008 Dec. 6 (12):1315-41; quiz 1286.

24. Kamar N, Mansuy JM, Cointault O, et al. Hepatitis E virus-related cirrhosis in kidney- and kidney-pancreas-transplant recipients. *Am J Transplant*. 2008 Aug. 8 (8):1744-8.

25. Kumar A, Beniwal M, Kar P, Sharma JB, Murthy NS. Hepatitis E in pregnancy. *Int J Gynaecol Obstet*. 2004 Jun. 85 (3):240-4.

26. Keeffe EB, Dieterich DT, Han SH, et al. A treatment algorithm for the management of chronic hepatitis B virus infection in the United States. *Clin Gastroenterol Hepatol*. 2004 Feb. 2 (2):87-106.

L

27. Lewis ShHeitkemper M, Dirkseh Sh, O'Brien Medical- Surgical Nursing assessment and management of clinical problems. Mosby : Elsevire., 2007.

28. Lockhart PB, Gibson J, Pond SH, Leitch J. Dental management considerations for the patient with an acquired coagulopathy. Part 1: Coagulopathies from systemic disease. Br Dent J. 2003 Oct 25;195:439-45.

M

29. McMahon BJ. AASLD practice guideline update: chronic hepatitis B: update 2009. *Hepatology*. 2009 Sep. 50 (3):661-2.

30. Mehrvarz, A. (2006). liver and bilious tract diseases adapted from Green Books Collection; Tehran, Hayan Publication.

N

31. Neighbors, M, Sands, J, Monhan, F, Marek, J, & Green, C. (2007). Phipps Medical Surgical Nursing. Mosby, Elsevire.

O

32. Osborn, K. Wrach Ch, Watson A. ((2010). Medical-Surgical Nursing preparation for practice. International,Pearson.

33. Omagari K, Kadokawa Y, Masuda J, Egawa I, Sawa T, Hazama H, et al. Fatty liver in non-alcoholic non-overweight Japanese adults: incidence and clinical characteristics. J Gastroenterol Hepatol. 2002;17:1098-105.

P

34. Purcell RH. Relative infectivity of hepatitis A virus by the oral and intravenous routes in 2 species of nonhuman primates. The Journal of infectious diseases. 2002;185:1668-1771.1. Purcell RH. Relative infectivity of hepatitis A virus by the oral and intravenous routes in 2 species of nonhuman primates. The Journal of infectious diseases. 2002;185:1668-1771.

35. Previsani N, Lavanchy D, World Health Organization. Hepatitis E (WHO/CDS/CSR/EDC/2001.12.). 2001.

36. Previsani N, Lavanchy D, World Health Organization. Hepatitis B (WHO/CDS/CSR/LYO/2002.2). 2002.

S

37. Smith, G, & Watson, R. (2005). Gastrointestinal Nursing. Blackwell Sciences.

38. Smeltzer, F, Bare, B, Hinkel, L, & Cheever, H. (2010). *Brunner & suddarth's Text book of Medical- surgical Nursing*. International:.Lippincot, Williams & Wilkins

39. Sorrell MF, Belongia EA, Costa J, et al. National Institutes of Health consensus development conference statement: management of hepatitis B. *Hepatology*. 2009 May. 49 (5 suppl):S4-S12.

40. Samiei, S, Monjamed, Z, Mehran, A, & Taheri, Z. (2005). Investigation quality of self care in patients member of family which have simplex and multiplex hepatitis B.

41. Schmilovitz-Weiss H, Ben-Ari Z, Sikuler E, et al. Lamivudine treatment for acute severe hepatitis B: a pilot study. *Liver Int*. 2004 Dec. 24 (6):547-51.

T

42. Tabor E. The role of tumor suppressor genes in the development of hepatocellular carcinoma. In: Okuda K, Tabor E, eds. *Liver Cancer*. New York, NY: Churchill Livingstone; 1997. 89-95.

43. Terrault NA, Bzowej NH, Chang KM, Hwang JP, Jonas MM, Murad MH, et al. AASLD guidelines for treatment of chronic hepatitis B. *Hepatology*. 2016 Jan. 63 (1):261-83.

W

44. . World Health Organization. Hepatitis A. Fact sheet no 328. Updated: July 2016; Accessed: June 12, 2017.

45. World Health Organization. Guidelines for the prevention, care and treatment of persons with chronic hepatitis B infection. 2015 Mar.

46. Wasley A, Rydal S, Gallagher K, Centers for Disease Control and Prevention (CDC). Surveillance for acute viral hepatitis--United States, 2006. *MMWR Surveill Summ*. 2008 Mar 21. 57 (2):1-24.

47. Wedemeyer H, Yurdaydın C, Caruntu FA, Curescu MG, Yalc, ın K, Akarca US, et al. Pegylated-interferon-alfa-2a plus tenofovir or placebo for the treatment of hepatitis delta. 49th Annual Meeting of the European Association for the Study of the Liver (EASL 2014). London, April 24-28, 2014.

48. WHO. Hepatitis B vaccines: WHO position paper. Weekly Epidemiological Record. 2009;84(40):405-20.

Appendix

College of dentistry/Baghdad University
Department of medicine
The awareness and knowledge of Hepatitis infectious disease

Student name_____ age _____

gender_____ stage_____

Statements regarding knowledge

1. Hepatitis B virus (HBV) is a member of the Hepa DNA virus family:
   Yes No I don't know
2. HBV has a diameter of 42 nm:
   Yes No I don't know
3. There are four genotypes of HBV:
   Yes No I don't know
4. Hepatitis B is an infectious illness caused by hepatitis B virus (HBV):
   Yes No I don't know
5. Hepatitis B virus is a non-retroviral virus:
   Yes No I don't know
6. HBV is one of the smallest enveloped animal ruses:
   Yes No I don't know
7. Hepatitis B infection was originally known as serum hepatitis:
   Yes No I don't know
8. The acute illness causes liver inflammation:
   Yes No I don't know
9. The time between the removal of the HBS Ag and the appearance of anti-HBs is called the window period: Yes No I don't know
10. PCR tests have been developed to detect and measure the amount of HBV DNA, called the viral load, in clinical specimens: Yes No I don't know
11. The vaccine is administered in either two-,three, or four-dose schedules into infants and adults, which provides protection for 85-90% of individuals:
    Yes No I don't know
12. Individuals who remain HBs Ag positive for at least 6 months are considered to be hepatitis EB carriers:
    Yes No I don't know
13. The hepatitis B surface antigen (HBs Ag) is most frequently used to screen for the presence of this infection:
    Yes No I don't know

Statements regarding awareness

1. Transmission of hepatitis B virus results from exposure to infectious blood or body fluids

Yes No I don't know

2. Transmission of hepatitis B virus results from needle stick injuries

Yes No I don't know

3. Transmission of hepatitis B virus results from unprotected sexual contact

Yes No I don't know

4. Hepatitis B cannot be transmitted through hand shaking

Yes No I don't know

5. Hepatitis B infection is preventable by vaccination

Yes No I don't know

6. Chronic hepatitis infection results in liver cirrhosis and heap to cellular carcinoma

Yes No I don't know

7. Patients undergoing surgical dental procedures to be investigated for HBV

Yes No I don't know

8. The hepatitis B surface antigen (HBs Ag is most frequently used to screen for the presence of this infection)

Yes No I don't know

9. Dental personnel infected with hepatitis B should avoid patients' treatment, especially surgeries

Yes No I don't know