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PREVALENCE OF HEPATITIS B SURFACE ANTIGEN AMONG PREMARITAL PEOPLE IN SULAIMANI / IRAQ

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ABSTRACT

Background: The magnitude of chronic infection with hepatitis B virus (HBV) varies substantially between the countries. A better understanding of incidence and/ or prevalence of HBV infection and associated risk factors provides insight into the transmission of this infection in the community. Transmission of hepatitis B virus results from exposure to infectious blood or body fluids containing the virus. Possible forms of transmission include unprotected sexual contact, blood transfusions, re-use of contaminated needles, and vertical transmission from mother to child during childbirth. **Objective:** To know the prevalence of HBsAg among premarital people in Sulaimani province. And to identify the associated risk factors responsible for seropositivity. **Patients and methods:** This prospective study was conducted in the premarital consultation clinic in Sulaimani city from November 2008 to February 2009, including all premarital people from age 18 years and above of either sex. **Results:** The prevalence of HBsAg among premarital people was 0.67%. Equal number of both sexes was included in this study. In multivariate analysis, factors associated with an increased HBsAg risk were occupation, history of surgical operation, blood transfusion and tattooing. **Conclusion:** The prevalence of HBsAg among premarital people in Sulaimani province is lower than that previously reported, with male predominance. A positive relationship between HBsAg with occupation, history of surgical operation, blood transfusion and tattooing was reported.

INTRODUCTION

Hepatitis B viral infection_ is a disease caused by hepatitis B virus (HBV) which infects the liver of hominoidae, including humans, and causes an inflammation called hepatitis. Originally known as "serum hepatitis",[1] the disease has caused epidemics in parts of Asia

and Africa, and it is endemic in China.[2] About a third of the world's population, more than 2 billion people have been infected with the hepatitis B virus.[3] This includes 350 million chronic carriers of the virus.[4]

Viral structure

HBV is a double-shelled, enveloped DNA virus belonging to the family Hepadnaviridae (genus Orthohepadnavirus). The viral genome consists of partially double-stranded DNA, is 3.2 kb(kilobase) in length, and possesses four partially overlapping open reading frames that encode the genes for HBsAg (*S* gene), HBcAg (*C* gene), HBV polymerase (*P* gene), and a small protein that seems to have transactivating functions (*X* gene, HBxAg). The *S* gene has three start codons and is capable of producing three different sizes of HBsAg (small, medium, and large *S*). The *C* gene has two start codons and can produce two antigenically distinct products: HBcAg, which is retained in hepatocytes until assembled and incorporated into HBV virions, and hepatitis B e antigen (HBeAg), which is secreted into serum as a small soluble protein.[5]

Hepatitis B virus replication

The life cycle of Hepatitis B virus is complex. Hepatitis B is one of a few known non-retroviral viruses which use reverse transcription as a part of its replication process. The virus gains entry into the cell by binding to an unknown receptor on the surface of the cell and enters it by endocytosis. Because the virus multiplies via RNA made by a host enzyme, the viral genomic DNA has to be transferred to the cell nucleus by host proteins called chaperones. The partially double stranded viral DNA is then made fully double stranded and transformed into covalently closed circular DNA (cccDNA) that serves as a template for transcription of four viral mRNAs. The largest mRNA, (which is longer than the viral genome), is used to make the new copies of the genome and to make the capsid core protein and the viral DNA polymerase. These four viral transcripts undergo additional processing and go on to form progeny virions which are released from the cell or returned to the nucleus and re-cycled to produce even more copies.[6,7]

Pathogenesis

The hepatitis B virus primarily interferes with the functions of the liver by replicating in liver cells. HBV virions (DANE particle) bind to the host cell via the preS domain of the viral surface antigen and are subsequently internalized by endocytosis. HBV-preS specific receptors are primarily expressed on hepatocytes; however, viral DNA and proteins have also

been detected in extrahepatic sites. During HBV infection, the host immune response causes both hepatocellular damage and viral clearance. The adaptive immune response, particularly virus-specific cytotoxic T lymphocytes (CTLs), contributes to most of the liver injury associated with HBV infection. By killing infected cells and by producing antiviral cytokines capable of purging HBV from viable hepatocytes, CTLs eliminate the virus.[8]

Epidemiology

The prevalence of HBV carriers varies from 0.1 percent to 2 percent in low prevalence areas (United States and Canada, Western Europe, Australia and New Zealand), to 3 to 5 percent in intermediate prevalence areas (Mediterranean countries, Japan, Central Asia, Middle East, and Latin and South America), to 10 to 20 percent in high prevalence areas (southeast Asia, China, sub-Saharan Africa).[9,10]

The wide range in HBV carrier rate in different parts of the world is largely related to differences in the age at infection, which is inversely related to the risk of chronicity. The rate of progression from acute to chronic HBV infection is approximately 90 percent for perinatally acquired infection.[11,12]

The most common risk factors were sexual exposure (sexual contact with a person known to have hepatitis B, multiple sex partners, and men having sex with men) and injection drug use. These data indicate that vaccination programs targeted at high risk adults are needed to further reduce transmission of HBV in the United States.[13,14]

Transmission

Transmission of hepatitis B virus results from exposure to infectious blood or body fluids containing the virus. Possible forms of transmission include (but are not limited to) unprotected sexual contact, blood transfusions, re-use of contaminated needles & syringes, and vertical transmission from mother to child during childbirth. Without intervention, a mother who is positive for HBsAg confers a 20% risk of passing the infection to her offspring at the time of birth. This risk is as high as 90% if the mother is also positive for HBeAg. HBV can be transmitted between family members within households, possibly by contact of nonintact skin or mucous membrane with secretions or saliva containing HBV.[15] However, at least 30% of reported hepatitis B among adults cannot be associated with an identifiable risk factor.[16]

Source of Hepatitis B Infection And Risk of Chronic Infection.[17]

Route of transmission	Risk of chronic infection
Horizontal transmission	10%
Injection drug use	
Infected unscreened blood products	
Tattoos/acupuncture needles	
Sexual (homosexual and heterosexual)	
Vertical transmission	90%
HbsAg-positive mother	

Clinical manifestations and natural history of hepatitis B virus infection

The spectrum of clinical manifestations of hepatitis B virus (HBV) infection varies in both acute and chronic disease. During the acute Phase, manifestations range from subclinical or anicteric hepatitis to icteric hepatitis and, in some cases, fulminant hepatitis; during the chronic phase, manifestations range from an asymptomatic carrier state to chronic hepatitis, cirrhosis, and hepatocellular carcinoma. Extrahepatic manifestations also can occur with both acute and chronic infection.[18]

Acute hepatitis

The incubation period lasts one to four months. A serum sickness-like syndrome may develop during the prodromal period, followed by constitutional symptoms, anorexia, nausea, jaundice and right upper quadrant discomfort. The symptoms and jaundice generally disappear after one to three months, but some patients have prolonged fatigue even after normalization of serum aminotransferase concentrations.

Approximately 70 percent of patients with acute hepatitis B have subclinical or anicteric hepatitis, while 30 percent develops icteric hepatitis. The disease may be more severe in patients coinfected with other hepatitis viruses or with underlying liver disease.[18]

Fulminant hepatic failure is unusual, occurring in approximately 0.1 to 0.5 percent of patients. Fulminant hepatitis B is believed to be due to massive immune-mediated lysis of infected hepatocytes. This explains why many patients with fulminant hepatitis B have no evidence of HBV replication at presentation.[19]

Laboratory testing during the acute phase reveals elevations in the concentration of alanine and aspartate aminotransferase levels (ALT and AST); values up to 1000 to 2000 IU/L are typically seen during the acute phase with ALT being higher than AST. The serum bilirubin concentration may be normal in patients with anicteric hepatitis. The prothrombin time is the best indicator of prognosis. In patients who recover, normalization of serum

aminotransferases usually occurs within one to four months. Persistent elevation of serum ALT for more than six months indicates progression to chronic hepatitis.[20,21]

Chronic hepatitis

A history of acute hepatitis is elicited in only a small percentage of patients with chronic HBV infection. In low or intermediate prevalence areas, approximately 30 to 50 percent of patients with chronic HBV infection have a past history of acute hepatitis; such a history is lacking in the remaining patients in these areas and in the majority of patients in high prevalence areas (predominantly perinatal infection).[22]

Many patients with chronic hepatitis B are asymptomatic (unless they progress to decompensated cirrhosis or have extrahepatic manifestations), while others have nonspecific symptoms such as fatigue. Some patients experience exacerbations of the infection which may be asymptomatic, mimic acute hepatitis, or manifest as hepatic failure.[22]

Physical examination may be normal or there may be stigmata of chronic liver disease. Jaundice, splenomegaly, ascites, peripheral edema, and encephalopathy may be present in patients with decompensated cirrhosis. Laboratory tests may be normal, but most patients have mild to moderate elevation in serum AST and ALT. During exacerbations, the serum ALT concentration may be as high as 50 times the upper limit of normal and alfa-fetoprotein (AFP) concentrations as high as 1000 ng/mL may be seen.[22]

Progression to cirrhosis is suspected when there is evidence of hypersplenism (decreased white blood cell and platelet counts) or impaired hepatic synthetic function (hypoalbuminemia, prolonged prothrombin time, hyperbilirubinemia).

Chronic HBV infection can occur in the presence or absence of serum hepatitis B e antigen (HBeAg), and generally, for both HBeAg-reactive and HBeAg-negative chronic hepatitis B, the level of HBV DNA correlates with the level of liver injury and risk of progression.[23]

Diagnosis

Hepatitis B surface antigen and antibody — Hepatitis B surface antigen (HBsAg) is the serologic hallmark of HBV infection. It can be detected by radioimmunoassays (RIA) or enzyme immunoassays (EIA). HBsAg appears in serum 1 to 10 weeks after an acute exposure to HBV, prior to the onset of hepatitic symptoms or elevation of serum alanine aminotransferase (ALT).[24]

In patients who subsequently recover, HBsAg usually becomes undetectable after four to six months. Persistence of HBsAg for more than six months implies chronic infection.[25] The disappearance of HBsAg is followed by the appearance of anti-HBs. In most patients, anti-HBs persists for life, thereby conferring long-term immunity.

Coexistence of HBsAg and hepatitis B surface antibody (anti-HBs) has been reported in approximately 24 percent of HBsAg positive individuals[26] In most instances, the antibodies are unable to neutralize the circulating virions.[27] These individuals should therefore be regarded as carriers of the hepatitis B virus.

Hepatitis B core antigen and antibody — Hepatitis B core antigen (HBcAg) is an intracellular antigen that is expressed in infected hepatocytes. It is not detectable in serum. Anti-HBc can be detected throughout the course of HBV infection. During acute infection, anti-HBc is predominantly of IgM class. IgM anti-HBc is the sole marker of HBV infection during the window period between the disappearance of HBsAg and the appearance of anti-HBs.[28] The detection of IgM anti HBc is usually regarded as an indication of acute HBV infection.

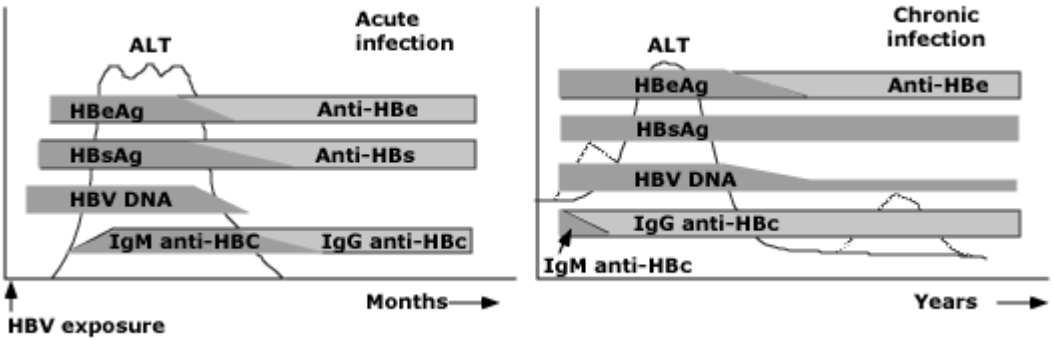
IgG anti-HBc persists along with anti-HBs in patients who recover from acute hepatitis B. It also persists in association with HBsAg in those who progress to chronic HBV infection.[28]

Isolated anti-HBc — Isolated detection of anti-HBc can occur in three settings: (1) during the window period of acute hepatitis B when the anti HBc is predominantly IgM class;(2) many years after recovery from acute hepatitis B when anti-HBs has fallen to undetectable levels;(3) and after many years of chronic HBV infection when the HBsAg titer has decreased below the cutoff level for detection.[29]

Hepatitis B e antigen and antibody — It is generally considered to be a marker of HBV replication and infectivity. The presence of HBeAg is usually associated with high levels of HBV DNA in serum and higher rates of transmission of HBV infection from carrier mothers to their Babies.[30,31]

HBeAg to anti-HBe seroconversion occurs early in patients with acute infection, prior to HBsAg to anti-HBs seroconversion.[24] Seroconversion from HBeAg to anti-HBe is usually associated with decrease in serum HBV DNA and remission of liver disease.[32,33]

More recently, PCR tests have been developed to detect and measure the amount of viral nucleic acid in clinical specimens. These tests are called viral loads and are used to assess a person's infection status and to monitor treatment.



Treatment

Treatment should be considered in patients with HBeAg positive or HBeAg negative chronic hepatitis. Patients with compensated cirrhosis and HBV DNA >2,000 IU/mL and those with decompensated cirrhosis and detectable HBV DNA by PCR assay should be considered for antiviral therapy, regardless of the serum ALT level.[34]

Treatment strategies for chronic HBV include interferon (standard and pegylated), lamivudine, adefovir dipivoxil, telbivudine and entecavir.[34]

Prevention

- Universal hepatitis B vaccination programs are ongoing in endemic areas, with encouraging results. The hepatitis B vaccine consists of recombinant HBsAg produced in yeast. A series of 3 injections may achieve HBsAb levels greater than 10 million IU/mL in approximately 95% of people vaccinated.
- Low response rates have been associated with obesity, smoking, immunosuppression, and advanced age.
- Vaccination with a single dose must be repeated every 5-10 years.
- All newborns must be vaccinated against hepatitis B. For infants born to mothers with active hepatitis B virus (HBV) infection, a passive-active (immunoglobulin and vaccination) approach is recommended.

- Healthcare workers or people who have had a needle-stick accident from a patient with active hepatitis B infection must receive the active-passive immunization approach (HBIg) and the first dose of the vaccine at the same time and at different site, and these individuals must be monitored with blood tests.[35]

Aim of the study

To know the prevalence of HBsAg among premarital people who underwent premarital screening investigations in premarital consultation clinic in Sulaimani city, and to identify the associated risk factors responsible for seropositivity.

Patients and methods

This is a prospective study conducted in premarital consultation clinic in Sulaimani from November 2008 to February 2009. One thousands five hundred apparently healthy subjects (750 males and 750 females) were evaluated; they attended central laboratory/ virology department in Sulaimani city.

All subjects were interviewed, history had been taken from each case by using special questionnaire form, including queries, namely; name, age, gender, occupation, history of dental procedure, surgical operation, endoscopy, blood transfusion, jaundice, tattooing, parentral drug using and history of trauma.

From each subject, 5 ml of blood was drawn, the sample was sent for premarital screening test which include blood group and Rh, complete blood count, HIV test and HBsAg test. Identifying of HBsAg was done by using ELISA technique.

Statistics

Statistical package for social science (SPSS) program version 15 was used for statistical analysis, the frequency distributions were obtained, after the grouping of data to different variables, and statistical significances of these ratios were analyzed by chi-square test to identify the effect of some factors, probability of p value equal or less than 0.05 was considered to be statistically significant.

RESULTS

In this study, 1500 healthy premarital persons were evaluated, 750 (50%) male and 750 (50%) females. Their ages ranged from 18 to 65 years and the mean of age was 25.84 years.

The prevalence of HBsAg positivity among the premarital people was (0.67%), positive HBsAg was found only in 10 subjects. Figure-1.

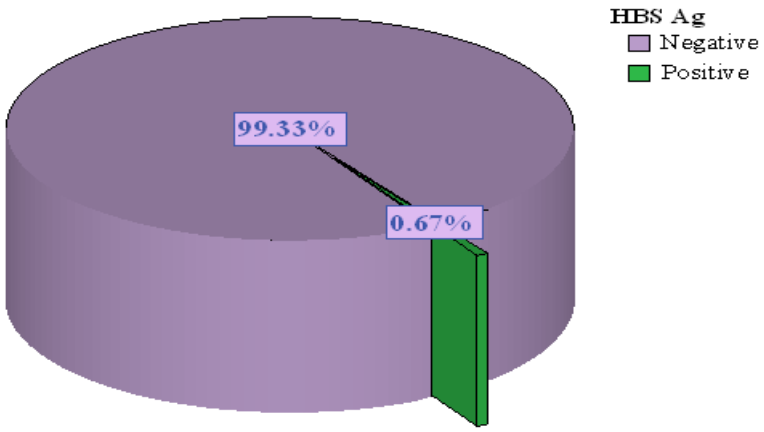


Figure 1: The prevalence of positive HBsAg among premarital people.

Approximately 813 (54.2%) had positive history of dental procedure while 687 (45.8%) had no history of dental procedure. Regarding the history of surgical operation, 354(23.6%) had positive history while 1146 (76.4%) had negative history. Table-1.

Table 1: Frequency and percentage of subjects according to the risk factors.

Variables	Option	Frequency	Percentage
History of dental procedure	No	687	45.8
	Yes	813	**54.2**
History of surgical operation	No	1146	76.4
	Yes	354	**23.6**
History of endoscopy	No	1443	96.2
	Yes	57	**3.8**
History of blood transfusion	No	1463	97.5
	Yes	37	**2.5**
History of jaundice	No	1433	95.5
	Yes	66	**4.4**
History of tattooing	No	1312	87.5
	Yes	188	**12.5**
History of parentral drug using	No	1496	99.7
	Yes	4	**0.3**
History of trauma	No	1392	92.8
	Yes	108	**7.2**

According to the occupation of the subjects, 33.13% were housewives, 31.8% of the subjects were free works, 18.87% were officers, 9.27% were students, and 6.93% were policemen. Figure-2

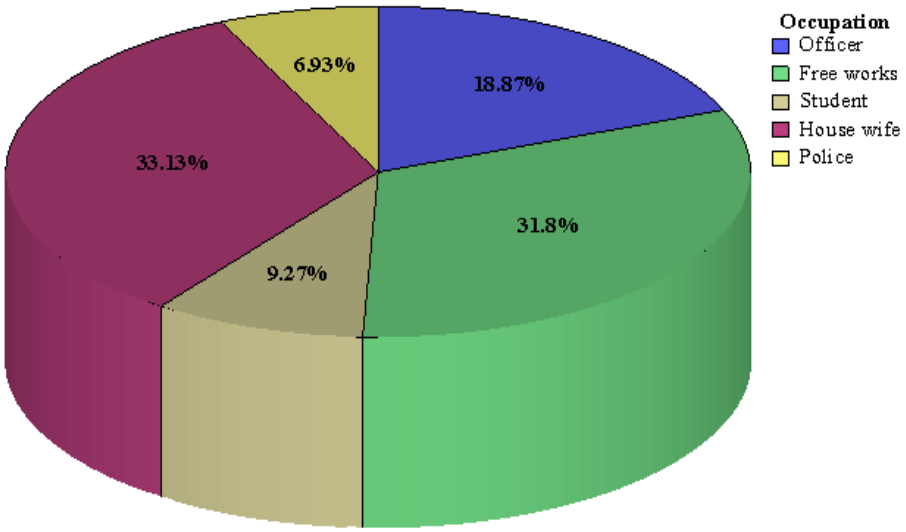


Figure 2: Percentage of the subjects according to their occupation.

Mean age among HBsAg positive people was 26.1years while of HBsAg negative was 25.8 with no statistically significant difference.

Table 2: The relation of HBsAg with mean age.

HBS Ag	Mean (years)± Std. Deviation	N	P value
Negative	25.8±6.02	1490	0.889
Positive	26.1±5.8	10	

The relationship of positive HBsAg with gender was not significant (p value=0.056). Regarding the occupation, there was a significant relation between the HBsAg and subjects who work as police (p value=0.016), as 104 of the subjects work as police, 3 (2.9%) of them had positive HBsAg. The relation of positive HBsAg with history of dental procedure was not significant (p value=0.484). The relation between positive HBsAg and history of surgical operation was significant (p value=0.015), as 354 of the subjects had history of surgical operation, 6 (1.7%) of them had positive HBsAg. There was no significant relation between HBsAg and history of endoscopy (p value=0.678). Also a significant relation is found with history of blood transfusion (p value=0.024), as 37 of the subjects had history of receiving blood, 2 (5.4%) of them had positive HBsAg. No significant relation was found between HBsAg and history of jaundice (p value=0.637). A significant relation was found between positive HBsAg and history of tattooing (p value=0.004), as 188 of the subjects had history of

tattooing, 5 (2.7%) of them had positive HBsAg. No significant relation was found between positive HBsAg and parentral drug using or history of trauma. Table-3.

Table 3: The association of positive HBsAg with risk factors.

Variables	HBS Ag		P value
	Negative N (%)	Positive N (%)	
Sex			
Female	748(99.7)	2(0.3)	0.056
Male	742(98.9)	8(1.1)	
Occupation			
Officer	283(100.0)	0(0.0)	**0.016**
Free works	472(99.0)	5(1.0)	
Student	139(100.0)	0 (0.0)	
House wife	495(99.6)	2(0.4)	
Police	101(97.1)	3(2.9)	
History of dental procedure			
No	683(99.4)	4(0.6)	0.484
Yes	807(99.3)	6(0.7)	
History of surgical operation			
No	1142(99.7)	4(0.3)	**0.015**
Yes	348(98.3)	6(1.7)	
History of endoscopy			
No	1433(99.3)	10(0.7)	0.678
Yes	57(100.0)	0(0.0)	
History of blood transfusion			
No	1455(99.5)	8(0.5)	**0.024**
Yes	35(94.6)	2(5.4)	
History of jaundice			
No	1424(99.3)	10(0.7)	0.637
Yes	66(100.0)	0(0.0)	
History of tattooing			
No	1307(99.6)	5(0.4)	**0.004**
Yes	183(97.3)	5(2.7)	
History of parentral drug using			
No	1486(99.3)	10(0.7)	0.947
Yes	4(100.0)	0(0.0)	
History of trauma			
No	1384(99.4)	8(0.6)	0.157
Yes	106(98.1)	2(1.9)	

DISCUSSION

Hepatitis B virus (HBV) is the most common cause of serious liver infection in the world. About a third of the world's population, more than 2 billion people have been infected with the hepatitis B virus.[3] This includes 350 million chronic carriers of the virus.[4]

We screened 1500 healthy premarital peoples, 750 males and 750 females; their ages range from 18-65 years with mean of 25.84 years, because of our oriental, Islamic society encourages early marriage.

Hepatitis B surface Ag was positive in 0.67% of the individuals entered the study; this result was lower than that of Mohammed O. et al[36], a study was done in Sulaimani, Kurdistan Region-Iraq in 2006, which showed that the prevalence of HBsAg positivity among 1514 blood donors was 2.3%, this reflect the improvement in people education and knowledge about the hepatitis B viral infection, effective blood screening, and increasing the vaccination program applied against HBV infection. This result was similar to that of Blakely T. et al[37], a study which estimates the hepatitis B surface antigen (HBsAg) carrier prevalence for adults in New Zealand, and showed that the prevalence of HBsAg was 0.5%. However our result was lower than that of Sebastiao Viana et al[38], a study which was done in the western Brazilian Amazon, and showed that of 2.656 samples, 3.3% were positive for HBsAg.

In our study there was no significant relation of HBsAg positivity neither with the age nor with the gender, p value= (0.889) and (0.056) respectively, a result which is similar to that of Blakely T. et al study.[37]

One hundred and four of the subjects were working as police; among them three (2.9%) were HBsAg positive (p value=0.016), this result is higher than that of Bandaranayake DR.et al[39] a study done in New Zealand in 1987, which showed that the prevalence ratio of HBsAg for police officers was 0.82%. this may be explained by the fact that of increase the rate of accidents and exposure among this group with subsequent risk of viral transmission. However this figure is slightly lower than that of Sonder GJ et al[40] a study done in the Amsterdam police force, 2000-2003, were 112 exposures with viral transmission risk were reported and finding 4% HBsAg positive.

Only 6 (0.7%) subjects out of 813 who had history of dental procedure were HBsAg positive which is statistically not significant (p value=0.484), this result is much lower than that of Odaibo GN. et al[41], a study done in Nigeria, and shows that the overall HBsAg infection rate

was 18.3%. this low result may be due to using a sterile or disposable instrument during dental extraction and filling, also may suggests that Dental Surgeons in this environment have been immunized at the beginning of their professional life with its effect in decreasing the rate of viral transmission form the dentist to their patients and vice versa.

The relation of HBsAg positivity with history of surgical operation was significant (p value=0.015), as 354 subjects had history of major surgery, among them, 6 (1.7%) were positive for HBsAg. This result is similar to that Mohammed O. et al[36], which showed that 177 blood donors who had been underwent surgical operation, among them 3 (1.7%) were HBsAg positive. this mean that surgery with contaminated instruments is another essential way of transmitting HBV infection especially in emergency department, where sometimes non-screened blood had been given and non sterilized instruments might have been used.

No significant relation was found between HBsAg and past history of endoscopy, as 57 subjects underwent endoscopy, among them no one showed positive HBsAg. This result is similar to that of Anna S. F. Lok et al[42], a study was done in Hong Kong, and showed that one hundred and eleven patients with no HBV markers underwent endoscopy; none of them showed seroconvertion to HBsAg. It was demonstrated that with a standard cleaning procedure there was no evidence of endoscopic transmission of HBV infection even in an endemic area.[42]

Thirty- seven subjects had the history of blood transfusion; among them two were HBsAg positive, (p value=0.024) which is considered a significant result. This result is higher than that of Mohammed O. et al[36], which showed that among 25 donors who had history of blood transfusion, no one had positive HBsAg, this may be explained by the fact that all donations issued for transfusion have been tested for hepatitis B surface antigen as a marker of transmissible hepatitis B virus. These measures have resulted in low rates of transmission by transfusion but have not eliminated all infectious donations from the blood supply. A donation is concluded as having been probably infectious if the donor was surface antigen negative but had evidence of acute infection or of carrying the virus (antibody to hepatitis B core antigen with no or low titers of antibody to surface antigen).[43] Mutant strains of hepatitis B virus not detected by routine surface antigen tests also pose a risk of infectious donations being transfused.[44] Another point which may be associated with increasing the risk of the transmission is the usage of non-screened blood for transfusion which sometimes occurs during emergency surgical operations.

No significant association was found between HBsAg and previous history of jaundice, among 66 subjects who mentioned to have history of jaundice, nobody showed positive HBsAg.

Among 188 subjects who had tattooing, 5 (2.7%) of them were positive for HBsAg, which is statically significant relation (p value=0.004), higher prevalence was seen in Shi MD. et al[45], a study which was done in Taiwan and showed among 476 young adult male, 11.3% of them were positive for HbsAg, this confirm the strong association between tattooing and the transmission of the virus.

In the developing countries the administration of unnecessary injections, because they think that this is the most effective mode of treatment, is mainly responsible for the transmission of blood pathogens, which also includes hepatitis B virus.[46] Though in this study there is no significant association with HBsAg, as among 4 subjects who had history of parentral drug using; no one of them had positive HBsAg. A similar result was found in Farid Ullah Shah. et al[47], a study which was done in Islamabad, and found that among 15 patients with chronic hepatitis B, 14.8% of them there was a history of repeated injection from the general practitioners, but with no body showed independent risk. This can be explained on the base of using disposable needles which is widely available.

Among 108 subjects who were exposed to trauma, only 2 (1.9%) were positive for HBsAg which is not significant (p=0.157). This result is much lower than of Villani C. et al[48], a study was done in Italia, and showed that the prevalence of HBV infection to be higher in the trauma patients (6.6%) who required orthopaedic surgery than in the elective orthopaedic patients (3.3%).

CONCLUSION

Premarital screening program is of paramount important project in detecting asymptomatic carriers of hepatitis B viral infection and in controlling the vertical transmission. This done by identifying HBsAg positive subjects before they get married and then applying a protective vaccination schedule against HBV in the affected partner.

The prevalence of HBsAg among premarital people in Sulaimani city is lower than that previously reported with male predominance. A positive relationship between HBsAg and occupation, surgical operation, blood transfusion and tattooing was reported.

Recommendations

1. Every couple should undergo premarital health screening to detect healthy carriers and plan for family protection by vaccination the healthy partner against HBV.
2. All blood and blood products should be screened before transfusion and apply more accurate diagnostic methods, like PCR for suspicious cases.
3. Intensify vaccination program for HBV among new born and high risk group including health workers.
4. Proper sterilization of reused surgical and dental instruments and proper disposal of infected materials.
5. Improving people's education through mass media about the protection against hepatitis B and C infection.
6. Expand the scope of premarital screening programs to cover other sexually transmitted diseases like HCV, HIV….etc. and common hereditary diseases like thalassaemia.

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REFERENCES

1. Barker LF, Shulman NR, Murray R, et al (1996). "Transmission of serum hepatitis. 1970". JAMA, 276(10): 841–4.
2. Williams R (2006)."Global challenges in liver disease". Hepatology, 44(3): 521–6.
3. WHO Hepatitis B.
4. (FAQ about Hepatitis B, Stanford University School of Medicine.).
5. William P. Arend, James O. Armitage, David R. Clemmons, Jeffrey M. Drazen, Robert C. Griggs, Nicholas Larrusso. Cecil medicine23rdedition 2007. Saunders.

6. Beck J, Nassal M (2007). "Hepatitis B virus replication". World J. Gastroenterol, 13(1): 48–64. BrussV(2007).
7. "Hepatitis B virus morphogenesis". World J. Gastroenterol, 13(1): 65–73.
8. Iannacone M, Sitia G, Ruggeri ZM, Guidotti LG (2007). "HBV pathogenesis in animal models: recent advances on the role of platelets". J. Hepatol, 46(4): 719–26.
9. Maynard, JE. Hepatitis B: Global importance and need for control. Vaccine, 1990; 8(Suppl): S18.
10. Alter, MJ, Hadler, SC, Margolis, HS, et al. The changing epidemiology of hepatitis B in the United States. Need for alternative vaccination strategies. JAMA, 1990; 263: 1218.
11. Stevens, CE, Beasley, RP, Tsui, J, et al. Vertical transmission of hepatitis B antigen in Taiwan., 20 to 50 percent for infections between the age of 1 and 5 years, and less than 5 percent for adult acquired infection.
12. Tassopoulos, NC, Papaevangelou, GJ, Sjogren, MH, et al. Natural history of acute hepatitis B surface antigen-positive hepatitis in Greek adults. Gastroenterology, 1987; 92: 1844.
13. Kim, WR, Ishitani, MB, Dickson, ER, et al. Rising burden of hepatitis B in the United States: Should the other virus be forgotten? (abstract). Hepatology, 2002; 36: 222A.
14. Wasley, A, Grydal, S, Gallagher, K. Surveillance for acute viral hepatitis--United States, 2006. MMWR Surveill Summ, 2008; 57: 1.
15. Petersen NJ, Barrett DH, Bond WW, Berquist KR, et al (1976). "Hepatitis B surface antigen in saliva, impetiginous lesions, and the environment in two remote Alaskan villages". Appl. Environ. Microbiol., 32(4): 572-574.
16. Hapiro CN (1993). "Epidemiology of hepatitis B". Pediatr. Infect. Dis. J., 12(5): 433–7.
17. R.W. Chapman, J.D. Collier, P.C. Hayes. Liver and biliary tract disease. In: Nicholas A. Boon, Nicki R. Colledge, Brain R. Wallker: Davidson’s principles and practice of medicine. 20th edition 2006. Churchil Livngstone Elsvier.
18. Liaw, YF, Tsai, SL, Sheen, IS, et al. Clinical and virological course of chronic hepatitis B virus infection with hepatitis C and D virus markers. Am J Gastroenterol, 1998; 93: 354.
19. Wright, TL, Mamish, D, Combs, C, et al. Hepatitis B virus and apparent fulminant non-A, non-Bhepatitis. Lancet, 1992; 339: 952.
20. Rehermann, B, Ferrari, C, Pasquinelli, C, Chisari, FV. The hepatitis B virus persists for decades after patients' recovery from acute viral hepatitis despite active maintenance of a cytotoxic T-lymphocyte response. Nat Med, 1996; 2: 1104.

21. Yotsuyanagi, H, Yasuda, K, Iino, S, et al. Persistent viremia after recovery from self-limited acute hepatitis B. Hepatology, 1998; 27: 1377.
22. Lok, AS, Lai, CL. Alpha-fetoprotein monitoring in Chinese patients with chronic hepatitis B virus infection: Role in the early detection of hepatocellular carcinoma. Hepatology, 1989; 9: 110.
23. Dennis L. Kasper, Eugene Brawnwald, Anthony S. Fauci, Stephen L. Hauser, Dan L. Longo, J. Larry Jameson: Harrison's principles of internal medicine. 17th edition 2008. Mc Graw-Hill. New York.
24. Krugman, S, Overby, LR, Mushahwar, IK, et al. Viral hepatitis, type B. Studies on natural history and prevention re- examined. N Engl J Med, 1979; 300: 101.
25. Chu, CM, Liaw, YF, Pao, CC, Huang, MJ. The etiology of acute hepatitis superimposed upon previously unrecognized asymptomatic HBsAg carriers. Hepatology, 1989; 9: 452.
26. Tsang, TK, Blei, AT, O'Reilly, DJ, Decker, R. Clinical significance of concurrent hepatitis B surface antigen and antibody positivity. Dig Dis Sci., 1986; 31: 620.
27. Tabor, E, Gerety, RJ, Smallwood, LA, Barker, LF. Coincident hepatitis B surface antigen and antibodies of different subtypes in human serum. J Immunol, 1977; 118: 369.
28. Perrillo, RP, Chau, KH, Overby, LR, Decker, RH. Anti-hepatitis B core immunoglobulin M in the serologic evaluation of hepatitis B virus infection and simultaneous infection with type B, delta agent, and non-A non-B viruses. Gastroenterology, 1983; 85: 163.
29. Lok, ASF, Lai, CL, Wu, PC. Prevalence of isolated antibody to hepatitis B core antigen in an area endemic for hepatitis B virus infection: Implication in hepatitis B vaccination programs. Hepatology, 1988; 8: 766.
30. Okada, K, Kamiyama, I, Inomata, M, et al. e antigen and anti-e in the serum of asymptomatic carrier mothers as indicators of positive and negative transmission of hepatitis B virus to their infants. N Engl J Med, 1976; 294: 746.
31. Beasley, RP, Trepo, C, Stevens, CE, Szmuness, W. The e antigen and vertical transmission of hepatitis B surface antigen. Am J Epidemiol, 1977; 105: 94.
32. Hoofnagle, JH, Dusheiko, GM, Seeff, LB, et al. Seroconversion from hepatitis B e antigen to antibody in chronic type B hepatitis. Ann Intern Med, 1981; 94: 744.
33. Realdi, G, Alberti, A, Rugge, M, et al. Seroconversion from hepatitis B e antigen to anti-HBe in chronic hepatitis B virus infection. Gastroenterology, 1980; 79: 195.
34. Liaw, YF, Leung, N, Guan, R, et al. Asian-Pacific consensus statement on the management of chronic hepatitis B: a 2005 update. Liver Int., 2005; 25: 472.

35. Centers for Disease Control and Prevention. Hepatitis B vaccination of adolescents--California, Louisiana, and Oregon, 1992-1994. MMWR Morb Mortal Wkly Rep., Aug 26 1994; 43(33): 605-9.
36. Mohammed O. Mohammed. Prevalence of Hepatitis-B and Hepatitis-C among Blood Donors Sulaimani City. (JZC) Journal of Zankoy Sulaimani, September 2006; 9(1): Part A 115-124.
37. Blakely T, Salmond C, Tobias M. Hepatitis B virus carrier prevalence in New Zealand: population estimates using the 1987 police and customs personnel survey., 1998 Apr 24; 111(1064): 142-4.
38. SEBASTIÃO VIANA, RAYMUNDO PARANÁ, et al. High prevalence of hepatitis B virus and hepatitis D virus in the Western Brazilian Amazon. Hyg., 2005; 73(4): 808-814.
39. Bandaranayake DR, Salmond CE, Tobias MI. Occupational risk of hepatitis B for police and customs personnel, 1991 Dec 15; 134(12): 1447-53.
40. Sonder GJ, Bovée LP, Coutinho RA, et al. Occupational exposure to bloodborne viruses in the Amsterdam police force, 200-2003. 2005 Feb; 28(2): 169-74.
41. Odaibo GN, Arotiba JT, Fasola AO, et al. Prevalence of hepatitis B virus surface antigen (HBsAg) in patients undergoing extraction at the University College Hospital, Ibadn., 2003 Sep; 32(3): 243-5.
42. ANNA S. F. LOK, CHING-LUNG LAI, WAI-MO HUI, et al. Absence of transmission of hepatitis B by fibreoptic upper gastrointestinal endoscopy. Journal of Gastroenterology and Hepatology, 10 Mar 2008; 2(2): 175 – 180.
43. Ilzuka H, Ohmura K, Ishijima A, Satoh K, Tanaka T, et al. Correlations between anti-HBc titres and HBV DNA in blood units without detectable HBsAg. Vox Sanguinis, 1992; 63: 107-11.
44. Jongerius JM, Wester M, Cuypers HTM, et al. New hepatitis B virus mutant form in a blood donor that is undetectable in several hepatitis B surface antigen screening assays. Transfusion, 1998; 38: 56-9.
45. Shi MD, Lee SY, Lee YB. Increased risk of viral hepatitis in Taiwanese maleconscriptees with tattoos., 2007 May; 172(5): 539-40.
46. Garfein RS, Vlahov D, Galai N, Donerty MC, Nelson KE. Viral infctions in short-term injection drug users: the prevalence of hepatitis C, hepatitis B, human immunodeficiency and human T-lymphotropicviruses. Am J Public Helth, 1996; 86: 665-61.
47. Farid Ullah Shah, Mohammad Salih, Iftikhar Ahmed Malik, et al. Federal Government Services Hospital, Islamabad. Pakistan J. Med. Res., 2002; 41: 2.

48. Villani C, Conte S, Chiozzi F, Iandolo C, Persiani P. Prevalence of hepatitis B and hepatitis C in an orthopaedics and traumatology ward., 2001 Apr-Jun; 86(2): 167-73.