Hepatitis B virus: epidemiology and transmission risks

Hepatitis B virus (HBV) infection is a serious and common infectious disease of the liver, affecting millions of people throughout the world. The incubation period for HBV is 45-180 days, most commonly 60-90 days.[1]

Clinical information

Acute infection is clinically recognised in only a small proportion of cases; less that 10% of children and 30-50% of adults show icteric disease. In those with clinical illness, the onset is usually insidious, with anorexia, vague abdominal discomfort, nausea and vomiting, sometimes arthralgia and rash, often progressing to jaundice. Following acute HBV infection, the risk of developing chronic infection varies inversely with age. Chronic HBV infection occurs among about 90% of infants infected at birth, 25-50% of children infected at 1-5 years of age and about 1-10% of persons infected as older children and adults. An estimated 15-25% of persons with chronic HBV infection will die prematurely of either cirrhosis or hepatocellular carcinoma.[1]

Vaccination

HBV can be effectively prevented by vaccination. A safe and effective vaccine has been available since the 1980s. The complete vaccine series induces protective antibody levels in more than 95% of infants, children and young adults. After age 40, protection following the primary vaccination series drops below 90%. Protection lasts at least 20 years and should be lifelong.[2] Since 2008, hepatitis B vaccine has been included in the childhood immunisation programme in Ireland, alongside the targeted immunisation programme for those individuals who are at increased risk of HBV because of their occupation, lifestyle or other factors. These include healthcare workers (HCW), prison and security personnel, contacts of cases, people who inject drugs, people with certain medical conditions, clients in learning disability centres, people with multiple sexual partners, men who have sex with men, prisoners, and travellers to and immigrants from HBV endemic areas.[3]

Transmission

HBV has been found in virtually all body secretions and excretions. However, only blood, body fluids containing visible blood, semen and vaginal secretions represent a risk of transmission.[4] HBV is transmitted by percutaneous and mucosal exposure to infective blood or body fluids. Major modes of HBV transmission include sexual or close household contact with an infected person, perinatal mother to infant transmission, injecting drug use and nosocomial exposure.[1]

Percutaneous exposures that have resulted in HBV transmission include transfusion of unscreened blood or blood products, sharing unsterilised injection needles for IV drug use, haemodialysis, acupuncture, tattooing and injuries from contaminated sharp instruments sustained by hospital personnel.[5]

HBV is stable on environmental surfaces for at least 7 days and is 100 times more infectious than HIV.

Serological markers for HBV

HBsAg: Hepatitis B surface antigen is a marker of infectivity. Its presence indicates either acute or chronic infection.

HBeAg: Hepatitis B e antigen is a marker of a high degree of infectivity and correlates with a high level of HBV replication.

Anti-HBs: Antibody to hepatitis B surface antigen is a marker of immunity, either an immune response to HBV infection or to vaccination.

Anti-HBc: Antibody to hepatitis B core antigen is a marker of HBV infection.

Prevalence of HBV infection in Ireland, Europe and the world

Ireland

The prevalence of HBV in the general population in Ireland is low. However, HBV is more prevalent in certain sub-groups of the population.[6] The prevalence of HBV is higher in injecting drug users, people born in countries of intermediate (2-7%) or high (>8%) hepatitis B endemicity, MSM, people with multiple sexual partners, household or sexual contracts of known cases.[7]

The prevalence of HBV infection is generally lowest in the blood donor population, followed by the general population, then pregnant women, then high-risk groups. To determine the risk of HBV in migrant populations, it is necessary to look at data on their country of origin.

The World Health Organization has classified Ireland as a country of low prevalence for HBV, i.e. prevalence of HBsAg <2%.[6] The European Centre for Disease Prevention and Control (ECDC) carried out a literature review in 2010 of publications dated 2000-2009 on the prevalence of viral hepatitis in Europe.[8] It reported that the HBsAg prevalence in the general population in Ireland is estimated to be 0.1%. Ireland and the Netherlands have the lowest prevalence of HBV infection in Europe. It also reported that the HBsAg prevalence rates in blood donors and pregnant women in Ireland are among the lowest rates in Europe.

Low risk populations in Ireland

General population

A European HBV seroprevalence study using residual sera showed a low prevalence in Irish samples collected in 2003 (anti-HBc 1.7%, HBsAg 0.1%).[9] A national study of oral fluid samples collected by postal survey in 1998-1999 estimated anti-HBc prevalence in Ireland to be 0.51%.[10]

Blood donors

Of 313,019 first time blood donors tested by the IBTS between 1997 and 2015, 34 (0.011%) were found to be HBsAg positive. (Personal communication, Dr Joan O'Riordan, IBTS, July 2016).

Pregnant women

Routine antenatal testing for HBsAg was introduced in the Rotunda Hospital in 1998. Uptake was almost 100% and >16,000 pregnancies were screened between January 1998 and June 2000. This showed a HBsAg prevalence of 4.2% in non-EU women and 0.03% in Irish women tested.[11] Screening of >24,000 pregnant women in the West of Ireland in 2004-2009 demonstrated a prevalence of HBsAg of 0.21%, and all positive women were thought to be of non-Irish origin.[12]

High risk populations in Ireland

Prisoners

A national cross sectional survey of Irish prisoners in 1998 showed a prevalence of anti-HBc of 8.7% total, and of 18.5% in prisoners who were people who inject drugs.[13]

People who inject drugs

A cross-sectional study of 316 opiate users attending 21 addiction treatment centres in the HSE East was carried out between Dec 2001 and Jan 2002. The prevalence of HBsAg was 2% and of anti-HBc was 17%.[14]

Homeless people

Homeless people also have evidence of increased exposure to HBV, with a prevalence of anti-HBc of 9% in a study performed in Dublin in 1999-2000.[3]

Asylum seekers

Screening of asylum seekers in the HSE eastern region 1999-2003 found a prevalence of HBsAg of 5% .[15]

Trends in hepatitis B infection in Ireland

Hepatitis B is a notifiable disease in Ireland. There was a dramatic increase in annual HBV notifications between 1997 (31 cases) and 2008 (919 cases), mostly attributable to large numbers of people immigrating to Ireland from HBV endemic countries. Between 2000 and 2010, 95% of asylum applicants, and 73% of new work permit recipients, were from countries with intermediate or high HBV endemicity. The number of hepatitis B cases reported in Ireland increased by 5% in 2014 with 445 cases (9.7/100,000) compared with 425 cases in 2013. However, there has been a general downward trend in the number of reported cases since peak levels in 2008 (n=901).[7] The trend (a decline since the peak in 2008 with an increase in 2014) correspond to immigration trends in Ireland during the same period.[16]

Between 2010 and 2014, 8% of reported cases of hepatitis B were acute and 92% were chronic. The majority of acute cases of hepatitis B were sexually acquired. Where reported, the risk factors for chronic infection included, born in an endemic country (69%), sexually acquired (13%) and vertical transmission (5%).[16]

Hepatitis B infection in Europe

Although there is a decreasing trend in HBV, each year there are between 7,000 and 8,000 newly diagnosed cases of HBV in the EU/EEA region.[17] There has been a steady downward trend in the reported rates of acute cases in Europe that is likely related to the impact of vaccination campaigns.[18] 99% of countries have integrated HBV into routine immunisation (2014).[19] The total percentage of people infected with HBV varies between different countries, with higher rates in the southern part of Europe. The country with the highest prevalence (>4%) is Romania followed by medium prevalence countries (>1-2%), Spain, (parts of) Italy, and Greece. Countries with a low prevalence (<1%) include Belgium, the Czech Republic, Finland, Germany, Ireland, Netherlands, Slovakia and Sweden.[17]

The most severely affected population groups are people who inject drugs, sex workers, men who have sex with men, people living with HIV, inmates, and immigrants from high-endemic regions. In some countries, sexual transmission is more common than transmission through household contacts or injecting drug use.[17]

In 2013, 19,101 cases of hepatitis B infections were reported in 28 EU/EEA member states, a crude rate of 4.4 / 100,000 population.[18]

Maps of HBV prevalence in different population groups by country in Europe are available in the ECDC Technical Report .[8]

Global distribution

The global prevalence of chronic HBV infection (based on % of population HBsAg positive) is as follows[20]:

High prevalence (≥8%): sub-Saharan Africa, South-East Asia, the Eastern Mediterranean countries, south and western Pacific islands, the interior of the Amazon basin and certain parts of the Caribbean.

Moderate prevalence (2–7%): in south-central and south-west Asia, eastern and southern Europe, the Russian Federation and most of central and South America.

Low prevalence (<2%): Australia, New Zealand, northern and western Europe, and North America.

Transmission risks

The hepatitis B virus can survive outside the body for at least 7 days.[21] Several factors influence the risk of transmission of HBV infection, including the viral load of the source.

In a healthcare occupational context, the level that is regarded as "high" for a viral load differs in various regions. In America and Ireland, HCWs who are infected with HBV but have a circulating viral burden <10^4 genome equivalents/ml are allowed to continue working unrestricted.[22,23] Transmission of HBV via a percutaneous route is considered unlikely at HBV DNA levels below 10^7 genome equivalents/ml.[24]

Needlestick injuries

Those who are e antigen positive generally have higher viral loads, and the transmission rate of HBV following a needlestick injury from a source who is e antigen positive is estimated to be between 30% and 62%.[4, 22] The same injury with exposure to blood from a source who is e antigen negative is associated with 6-37% risk of serological evidence of HBV infection in the recipient.[4, 22] Some patients are infected with pre-core mutant viruses. This is associated with a high viral load in the absence of the e antigen, and thus is also associated with a high risk of HBV transmission.[22]

The risk from needlestick injuries in the community is more difficult to estimate and the exact incidence of needlestick injuries and the transmission rate is unknown. The limited published case reports[25, 26] would indicate that there is a very low risk of HBV transmission associated with community acquired needlestick injuries.

Other healthcare setting exposures

Spring loaded lancets have been implicated in the transmission of HBV to patients[27,] as have reusable sub-dermal EEG electrodes.[28] There is a report of transmission of HBV to a patient during an endoscopic procedure, although no biopsies were taken, but bleeding gastric ulceration was identified. The presumed source was HBeAg positive .[29]

Cleveland et al report that HBV infection prevalence in dentists increases with longer duration in practice.[30]Although rates in a reference control population were not included in this report, increasing prevalence with longer duration of practice indicates that there is potential for transmission to dentists during their work.

Other percutaneous exposures

There are case reports documenting the transmission of HBV among butchers.[31, 32] These are attributed to small hand cuts, and sharing knives, which can carry the virus on the handle. It is also thought that HBV can be transmitted via small cuts acquired in barber shops.[33]

Body fluid exposures

HBV DNA has been detected in body fluids apart from blood, including saliva, urine, nasopharyngeal fluid, semen, cervicovaginal fluids and tears.[34-37]. HBV transmission can occur following exposure to non-intact skin and mucous membranes. A case report describes transmission of HBV via broken skin, following contact with saliva and nasopharyngeal fluids from the source.[38]

Human bites

Case reports have documented HBV virus transmission via a human bite, when associated with the skin being broken.[39, 40]

Sexual exposures

HBsAg has been found in seminal fluid and vaginal secretions, although concentrations in these fluids are lower than in blood.[41] The risk of transmission of HBV following sexual exposure depends on the type of exposure, the viral load of the source, and the presence of sexually transmitted infections.[42] The prevalence of HBV in heterosexuals is increased in those with multiple sexual partners[42-44], and those who have markers for HIV or syphilis.[45] An infection rate of 18-44.2% is seen in regular heterosexual partners of HBV infected patients [46-48] In addition, female commercial sex workers with a history of having anal intercourse had an increased risk of HBV infection.[45]

The risk of developing HBV infection is particularly high among men who have sex with men.[41, 49] For men who have sex with men, the prevalence of HBV infection is increased in those who have a history of an ulcerative sexually transmitted infection, chlamydia, gonorrhoea, commercial sex work, or multiple partners.[50] There is also a significant risk associated with unprotected insertive anal intercourse.[51]

Hepatitis B transmission risk by exposure type

Exposure		Risk per exposure (unless otherwise stated)
Needlestick	Healthcare setting, patient known	HBeAg (+) = 37-62% risk of serologic evidence of infection in recipient HBeAg (-) = 23-37% risk of serologic evidence of infection, 1-6% clinical infection[4]
	Healthcare setting, patient unknown, or patient known but serology unknown	Requires risk assessment
	Community setting	2 case reports only.[25, 26] Risk very low. Requires risk assessment. For example, if the local PWID population has a seroprevalence of 50%, the risk from a community acquired needlestick is 12-31%.[52] (Note: seroprevalence in PWID in Ireland is lower than 50% - see epidemiology section).
Other percutaneous injuries with blood exposure	Healthcare sharp (e.g. lancet)	Risk per exposure unknown. 36.8%[53] -42%[27] developed HBV after repeat exposures.
	Exposure prone procedure by infected healthcare worker	Transmission rates vary between 6 and 15%[54] - most were before standard precautions introduced
Transfusion		52-69% transmission if transfused with HBsAg (+) blood[55]
Human bites		Risk negligible in the absence of visible blood. Case reports only. Requires risk assessment.
Percutaneous exposure to other body fluids (e.g. saliva)		Very low risk. Case reports - HBeAg (+) source.[38] Requires risk assessment.
Sexual exposures	Heterosexual exposures in general	18%[46] – 40%[47] - 44.2%[48] infection rate seen in regular partners of HBV infected people Increased risk if: multiple partners[42, 44], syphilis[44, 45, 56], gonorrhoea[56], receptive anal intercourse[45]
	Men who have sex with men	Increased risk of HBV transmission associated with ulcerative STI, gonorrhoea/chlamydia, sexual partner with HIV/AIDS, multiple sexual partners, commercial sex work[50], history of insertive anal intercourse[51]
	Receptive oral sex (fellatio)	Possible means of transmission[57]

Risk assessment

- Type/details of injury – as above
- Source status – increased risk with HBeAg, high viral load
- Recipient status – increased risk if immunocompromised
- For unknown source, consider where injury occurred – community setting versus hospital setting
    - If in hospital – consider high-risk ward/patients
    - If in community – consider prevalence of HBV and of PWID locally

References

1. American Public Health Association. Control of communicable diseases manual. 19th ed. Washington 2008.
2. World Health Organization. Hepatitis B fact sheet No 204. Revised August 2008. http://www.who.int/mediacentre/factsheets/fs204/en/
3. Royal College of Physicians of Ireland, National Immunisation Advisory Committee. Immunisation Guidelines for Ireland 2008 [cited 2012]. Available from: http://www.immunisation.ie/en/HealthcareProfessionals/ImmunisationGuidelines2008/.
4. Centers for Disease Control and Prevention. Updated U.S. Public Health Service guidelines for the management of occupational exposures to HBV, HCV, and HIV and recommendations for postexposure prophylaxis. MMWR 2001;50(RR-11):1-52.
5. World Health Organization Hepatitis B 2002. Available from: http://www.who.int/csr/disease/hepatitis/HepatitisB_whocdscsrlyo2002_2.pdf
6. World Health Organization. Introduction of hepatitis B vaccine into childhood immunization services. Management guidelines, including information for health workers and parents 2001. Available from: http://www.who.int/vaccines-documents/DocsPDF01/www613.pdf.
7. ealth Protection Surveillance Centre. Hepatitis B. Annual Report [Internet]. 2014. Available from: http://www.hpsc.ie/A-Z/Hepatitis/HepatitisB/HepatitisBreports/HepatitisAnnualReports/File,15351,en.pdf
8. European Centre for Disease Prevention and Control. Hepatitis B and C in the EU neighbourhood: prevalence, burden of disease and screening policies 2010. Available from: http://ecdc.europa.eu/en/publications/Publications/TER_100914_Hep_B_C%20_EU_neighbourhood.pdf
9. Nardone A, Anastassopoulou CG, Theeten H, Kriz B, Davidkin I, Thierfelder W, et al. A comparison of hepatitis B seroepidemiology in ten European countries. Epidemiol Infect 2009;137(7):961-9.
10. O'Connell T, Thornton L, O'Flanagan D, Staines A, Connell J, Dooley S, et al. Prevalence of hepatitis B anti-core antibody in the Republic of Ireland. Epidemiol Infect 2000;125(3):701-4.
11. Healy CM, Cafferkey MT, Butler KM, Cahill I, McMorrow J, Philbin M, et al. Antenatal hepatitis B screening - is there a need for a national policy? Ir Med J 2001;94(4):111-2,4.
12. O'Connell K, Cormican M, Hanahoe B, Smyth B. Prevalence of antenatal hepatitis B virus carriage in the west of Ireland. Ir Med J 2010;103(3):91-2.
13. Allwright S, Bradley F, Long J, Barry J, Thornton L, Parry JV. Prevalence of antibodies to hepatitis B, hepatitis C, and HIV and risk factors in Irish prisoners: results of a national cross sectional survey. BMJ 2000;321(7253):78-82.
14. Grogan L, Tiernan M, Geogeghan N, Smyth B, Keenan E. Bloodborne virus infections among drug users in Ireland: a retrospective cross-sectional survey of screening, prevalence, incidence and hepatitis B immunisation uptake. Ir J Med Sci 2005;174(2):14-20.
15. Doyle S. An evaluation and audit of the asylum seeker communicable disease screening service in the Eastern Region. Thesis submitted for Membership of the Faculty of Public Health Medicine: RCPI; 2006.
16. Hennessy S, Thornton LFlanagan P. World Hepatitis Day, 28th July 2015: Trends in hepatitis B and C in Ireland. Epi-Insight [Internet]. 2015; 16(7). Available from: http://ndsc.newsweaver.ie/epiinsight/1iqjpgft8ni?a=1&p=48942722&t=17517774
17. European Centre for Disease Prevention and Control. Info Sheet. Hepatitis B and C. Current situation in EU/EEA 2010. Available from: http://ecdc.europa.eu/en/press/news/Documents/1010_HepatitisAandB_info_sheet.pdf.
18. European Centre for Disease Prevention and Control. Surveillance Report. Hepatitis B surveillance in Europe 2013. Available from: http://ecdc.europa.eu/en/publications/Publications/hepatitis-b-surveillance-in-europe-2013.pdf
19. World Health Organisation. WHO/Unicef Coverage Estimates 2014 revision. July 2015 Immunization, Vaccines and Biologicals, (IVB), World health Organisation. Available from: http://www.who.int/immunization/monitoring_surveillance/burden/vpd/surveillance_type/passive/HepB_coverage.jpg?ua=1
20. World Health Organization. Hepatitis B vaccines. WHO position paper. Weekly Epidemiological Record [Internet]. 2004; 79(28):[253-64 pp.]. Available from: http://www.who.int/immunization_delivery/adc/hepb_wer.pdf.
21. World Health Organisation. Fact Sheet. Hepatitis B. Available from: http://www.who.int/mediacentre/factsheets/fs204/en/#
22. Department of Health and Children. The prevention of transmission of blood-borne diseases in the health-care setting 2005. Available from: http://www.dohc.ie/publications/transmission_of_blood_borne_diseases_2006.html.
23. Henderson DK, Dembry L, Fishman NO, Grady C, Lundstrom T, Palmore TN, et al. SHEA guideline for management of healthcare workers who are infected with hepatitis B virus, hepatitis C virus, and/or human immunodeficiency virus. Infect Control Hosp Epidemiol 2010;31(3):203-32.
24. Buster EH, van der Eijk AA, Schalm SW. Doctor to patient transmission of hepatitis B virus: implications of HBV DNA levels and potential new solutions. Antiviral Research 2003;60(2):79-85.
25. Res S, Bowden FJ. Acute hepatitis B infection following a community-acquired needlestick injury. J Infect 2011;62(6):487-9.
26. Garcia-Algar O, Vall O. Hepatitis B virus infection from a needle stick. Pediatr infect Dis J 1997;16(11):1099.
27. Polish LB, Shapiro CN, Bauer F, Klotz P, Ginier P, Roberto RR, et al. Nosocomial transmission of hepatitis B virus associated with the use of a spring-loaded finger-stick device. New Engl J Med 1992;326(11):721-5.
28. An outbreak of hepatitis B associated with reusable subdermal electroencephalogram electrodes. Hepatitis B Outbreak Investigation Team. CMAJ. 2000;162(8):1127-31.
29. Birnie GG, Quigley EM, Clements GB, Follet EA, Watkinson G. Endoscopic transmission of hepatitis B virus. Gut 1983;24(2):171-4.
30. Cleveland JL, Siew C, Lockwood SA, Gruninger SE, Gooch BF, Shapiro CN. Hepatitis B vaccination and infection among U.S. dentists, 1983-1992. J Am Dent Assoc 1996;127(9):1385-90.
31. Mevorach D, Eliakim R, Brezis M. Hepatitis B--an occupational risk for butchers? Ann Intern Med 1992;116(5):428.
32. Mevorach D, Brezis M, Ben Yishai F, Sadeh T, Shouval D, Eliakim R. Increased risk of exposure to hepatitis B infection among butchers sharing knives. Am J Med 1999;106(4):479-80.
33. Mariano A, Mele A, Tosti ME, Parlato A, Gallo G, Ragni P, et al. Role of beauty treatment in the spread of parenterally transmitted hepatitis viruses in Italy. J Med Virol 2004;74(2):216-20.
34. Kidd-Ljunggren K, Holmberg A, Blackberg J, Lindqvist B. High levels of hepatitis B virus DNA in body fluids from chronic carriers. J Hosp Infect 2006;64(4):352-7.
35. van der Eijk AA, Niesters HG, Hansen BE, Pas SD, Richardus JH, Mostert M, et al. Paired, quantitative measurements of hepatitis B virus DNA in saliva, urine and serum of chronic hepatitis B patients. Eur J Gastroenterol Hepatol 2005;17(11):1173-9.
36. Heiberg IL, Hoegh M, Ladelund S, Niesters HG, Hogh B. Hepatitis B virus DNA in saliva from children with chronic hepatitis B infection: implications for saliva as a potential mode of horizontal transmission. Pediatr Infect Dis J 2010;29(5):465-7.
37. Ayoola EA, Ladipo OA, Odelola HA. Antibody to hepatitis B core antigen, e-antigen and its antibody in menstrual blood and semen. International Journal of Gynaecology and Obstetrics 1981;19(3):221-3.

38. Williams I, Smith MG, Sinha D, Kernan D, Minor-Babin G, Garcia E, et al. Hepatitis B virus transmission in an elementary school setting. JAMA 1997;278(24):2167-9.
39. Hui AY, Hung LC, Tse PC, Leung WK, Chan PK, Chan HL. Transmission of hepatitis B by human bite--confirmation by detection of virus in saliva and full genome sequencing. J Clin Virol 2005;33(3):254-6.
40. Stornello C. Transmission of hepatitis B via human bite. Lancet 1991;338(8773):1024-5.
41. Catterall RD. Some observations on the epidemiology and transmission of hepatitis B. British Journal of Venereal Diseases 1978;54(5):335-40.
42. Alter MJ, Ahtone J, Weisfuse I, Starko K, Vacalis TD, Maynard JE. Hepatitis B virus transmission between heterosexuals. JAMA 1986;256(10):1307-10.
43. Alter MJ, Coleman PJ, Alexander WJ, Kramer E, Miller JK, Mandel E, et al. Importance of heterosexual activity in the transmission of hepatitis B and non-A, non-B hepatitis. JAMA 1989;262(9):1201-5.
44. Corona R, Caprilli F, Giglio A, Stroffolini T, Tosti ME, Gentili G, et al. Risk factors for hepatitis B virus infection among heterosexuals attending a sexually transmitted diseases clinic in Italy: role of genital ulcerative diseases. J Med Virol 1996;48(3):262-6.
45. Rosenblum L, Darrow W, Witte J, Cohen J, French J, Gill PS, et al. Sexual practices in the transmission of hepatitis B virus and prevalence of hepatitis delta virus infection in female prostitutes in the United States. JAMA 992;267(18):2477-81.
46. British Association of Sexual Health and HIV, Clinical Effectiveness Group. United Kingdom National Guideline on the Management of the Viral Hepatitides A, B & C 2008. Available from: http://www.bashh.org/guidelines.
47. Brook MG. Sexual transmission and prevention of the hepatitis viruses A-E and G. Sex Transm Infect. 1998;74(6):395-8.
48. Inaba N, Ohkawa R, Matsuura A, Kudoh J, Takamizawa H. Sexual transmission of hepatitis B surface antigen. Infection of husbands by HBsAg carrier-state wives. British Journal of Venereal Diseases 1979;55(5):366-8.
49. Hahne SJ, Veldhuijzen IK, Smits LJ, Nagelkerke N, van de Laar MJ. Hepatitis B virus transmission in The Netherlands: a population-based, hierarchical case-control study in a very low-incidence country. Epidemiol Infect 2008;136(2):184-95.
50. Remis RS, Dufour A, Alary M, Vincelette J, Otis J, Masse B, et al. Association of hepatitis B virus infection with other sexually transmitted infections in homosexual men. Omega Study Group. Am J Public Health 2000;90(10):1570-4.
51. Kingsley LA, Rinaldo CR, Jr., Lyter DW, Valdiserri RO, Belle SH, Ho M. Sexual transmission efficiency of hepatitis B virus and human immunodeficiency virus among homosexual men. JAMA 1990;264(2):230-4.
52. O'Leary FM, Green TC. Community acquired needlestick injuries in non-health care workers presenting to an urban emergency department. Emerg Med (Fremantle) 2003;15(5-6):434-40.
53. Centers for Disease Control and Prevention. Transmission of hepatitis B virus among persons undergoing blood glucose monitoring in long-term-care facilities--Mississippi, North Carolina, and Los Angeles County, California, 2003-2004. MMWR 2005;54(9):220-3.
54. Gunson RN, Shouval D, Roggendorf M, Zaaijer H, Nicholas H, Holzmann H, et al. Hepatitis B virus (HBV) and hepatitis C virus (HCV) infections in health care workers (HCWs): guidelines for prevention of transmission of HBV and HCV from HCW to patients. J Clin Virol 2003;27(3):213-30.
55. Centers for Disease Control and Prevention. Public Health Service inter-agency guidelines for screening donors of blood, plasma, organs, tissues, and semen for evidence of hepatitis B and hepatitis C. MMWR 1991;40(RR-4):1-17.
56. Lim KS, Wong VT, Fulford KW, Catterall RD, Briggs M, Dane DS. Role of sexual and non-sexual practices in the transmission of hepatitis B. British Journal of Venereal Diseases 1977;53(3):190-2.
57. Edwards S, Carne C. Oral sex and the transmission of viral STIs. Sex Transm Infect 1998;74(1):6-10.