In the next few months there will be changes in the Washington State investigation guidelines for chronic hepatitis B and chronic hepatitis C infections. The changes will be reviewed in a later issue of epiTRENDS.

Viral Hepatitis

Hepatitis is a general term for inflammation of the liver which has many causes, among them various infectious and non-infectious agents. Hepatotoxic chemicals include alcohol, chaparral and carbon tetrachloride. Early recorded outbreaks of jaundice may have been due to bacteria such as *Leptospira*. Multiple viral infections including mononucleosis may involve liver inflammation.

The term “viral hepatitis” is usually used to describe illnesses due to three common and two rarer viruses. Although genetically unrelated, with different replication mechanisms and risk factors, these viruses are confusingly named hepatitis A through hepatitis E. In this country the predominant viral hepatitis types are hepatitis A, hepatitis B, and hepatitis C, which are restricted to humans with the exception of rare cases induced in other primates.

Clarification of viral hepatitis etiologies began when it was recognized that jaundice could be epidemic. The first described outbreaks were associated with unsanitary crowded situations, often during wartime, and the condition was termed ‘infectious hepatitis.’ A second type of outbreak was called “serum hepatitis” was associated with smallpox variolation using human-derived material in the 19th century and with serum-derived vaccines and the reuse of needles or syringes during mass vaccination campaigns in the 20th century. It was subsequently realized that there were at least two different types of serum hepatitis based on several discrete infections in those receiving multiple blood transfusions such as occurred for persons with hemophilia. Lack of filterability in the laboratory suggested the agents were viral.
Hepatitis B virus was characterized in 1963 based on the ‘Australian antigen’ which was later identified as hepatitis B surface antigen (HBsAg). By 1973 hepatitis A virus had also been described. Even when blood donors were screened for hepatitis B there still occurred many cases of post-transfusion infections that were referred to at the time as non-A non-B hepatitis. Various studies, now considered unethical, were conducted involving prisoners and institutionalized children to elucidate the situation. In 1988 hepatitis C was identified and screening eliminated almost all transfusion-related hepatitis.

**Hepatitis Symptoms**

The viruses causing hepatitis B and hepatitis C can be transmitted readily through body fluids such as blood, blood products, and sexual fluids, and less commonly through other secretions. Neither is transmitted fecally.

Acute illness with these viruses is indistinguishable from other causes of acute hepatitis, whether infectious or chemical. In acute viral hepatitis the symptoms include fatigue, abdominal pain, loss of appetite, nausea, and vomiting. In more severe infections there may be jaundice, which includes yellowing of the eyes and skin, dark urine, and pale stools; liver enzymes (liver function tests) are elevated in such patients. Rarely, acute infections result in fulminant liver necrosis and can be fatal. Hippocrates described jaundice and John Graunt’s 1662 Bills of Mortality for London listed “jaundies” as a cause of death, although in both situations the etiology may have been another agent such as leptospirosis or chronic alcohol use rather than a virus.

Only 30-50% of all acute hepatitis B virus infections and 20-30% of all acute hepatitis C virus infections are symptomatic. Hepatitis B and hepatitis C infections can become chronic with lifelong persistence of the virus. Chronic infections therefore represent the greatest ongoing source of exposure in a population. Chronic infections can result even if there are not acute symptoms. The likelihood that acute infection progresses to a chronic state differs by condition and is in part influenced by age. Without prophylaxis, approximately 90% of infants infected perinatally with hepatitis B virus will develop chronic infection. Overall, initial hepatitis B infections will progress to chronic in approximately 5-10% of unimmunized older children and adults. For hepatitis C virus there is an overall 75-85% chance of an acute infection resulting in a chronic infection. Currently there is a vaccine for hepatitis B prevention but no vaccine to prevent hepatitis C, while anti-viral therapeutics are better for hepatitis C than hepatitis B.
Although most cases are initially asymptomatic, chronic viral hepatitis over decades may result in overt illness such as chronic liver disease, cirrhosis, or hepatocellular cancer. There is also a risk of fulminant liver failure if acute infection occurs with another hepatitis virus, such as a person with chronic hepatitis C developing acute hepatitis A virus infection.

**Hepatitis Surveillance**

Key risk factors for transmission of hepatitis B and hepatitis C are shared with other blood-borne pathogens, although their primacy differs by condition. Transmission risks for hepatitis B include birth to an infected mother, sexual contact with an infected person, and sharing injection drug equipment. For hepatitis C transmission risk is greatest from sharing injection drug equipment, while perinatal transmission is much less common. While the nature of these risks is observed more generally, data currently remain insufficient to accurately measure their influence in Washington State.

Acute infections with hepatitis B and hepatitis C viruses both have large proportions of asymptomatic cases which are never recognized. In addition, chronic hepatitis infections are often asymptomatic until long-term complications develop. As a result, both these conditions are under-recognized so surveillance activities identify only part of the disease burden.

Acute hepatitis surveillance provides incidence data. To be included, a person must have symptoms and also receive correct diagnostic laboratory testing. In Washington State acute hepatitis B has been reportable since 1965 and acute hepatitis C has been reportable since 1981, reflecting when serologic tests became available. Rates for both acute conditions peaked at the end of the 1980s, with hepatitis B reaching a rate of 22.3 per 100,000 population (Figure 1). For annual report data on acute viral hepatitis in Washington, see: [http://www.doh.wa.gov/Portals/1/Documents/Pubs/150-028-ChronicHepatitisBandCSurveillanceReport.pdf](http://www.doh.wa.gov/Portals/1/Documents/Pubs/150-028-ChronicHepatitisBandCSurveillanceReport.pdf)

Surveillance for chronic hepatitis C uses a period prevalence measure because even the year of exposure or of onset is typically unknown so reporting is by date of diagnosis. Deaths involving chronic hepatitis are tracked and over recent decades demonstrate a marked increase in deaths related to hepatitis C, almost entirely due to sequelae of chronic infections (Figure 2); since 1998 deaths involving hepatitis C have exceeded deaths due to HIV infection (not shown). For a report on chronic viral hepatitis in Washington see: [http://www.doh.wa.gov/DataandStatisticalReports/DiseasesandChronicConditions/CommunicableDiseaseSurveillanceData/AnnualCDSSurveillanceReports.aspx](http://www.doh.wa.gov/DataandStatisticalReports/DiseasesandChronicConditions/CommunicableDiseaseSurveillanceData/AnnualCDSSurveillanceReports.aspx)
Chronic hepatitis B is seen most frequently in persons born in high-risk countries where perinatal screening and prophylaxis are less widespread (Figure 3), as well as in those with sexual or injection drug use risks. Vaccine can reduce the spread of hepatitis B in a population and prevent acute and therefore chronic hepatitis B cases. Hepatitis C, which lacks a vaccine for prevention, is now the most common bloodborne disease in this country. Although transmission of hepatitis C, and subsequent chronic infection, is often correlated with injection drug use, the greatest overall burden of chronic hepatitis C (approximately 75%) currently exists among persons born from 1945 through 1965, though they account for only an estimated 27% of the U.S. population. Such “baby boomers” are thought to have contracted hepatitis C at a time when incidence rates were highest, and widespread screening of the nation’s blood supply had not yet begun. Anybody in this age group who does not know their status should be tested once for hepatitis C.

Reporting and investigations are one aspect for controlling hepatitis B and hepatitis C, and for reducing the burden of choric infections. Public health agencies face many challenges in understanding and controlling these conditions.

http://www.cdc.gov/media/dpk/2013/dpk-vs-hepatitisC_testing.html