Primary liver cancer is now one of the top ten causes of cancer death in Australia. In 2019 the estimated mortality from liver cancer is around 3 times higher than in was 1982, early diagnosis remains the key to increased survival.¹

Chronic hepatitis B (CHB) is the most common cause of liver cancer worldwide.² Almost 800,000 Australians die from hepatitis B infection every year.³ Recent migration from countries with a high prevalence of hepatitis B has increased the local incidence of CHB and HCC,⁴ and screening people born in endemic countries is effective in identifying risk and linking to care.⁵ A large proportion of HCC is preventable through vaccination against hepatitis B.

Transmission of hepatitis B:
Hepatitis B can be transmitted:
- Vertically, from mother to child.
- Horizontally, through:
  - close household contact with an infected person
  - sexual contact with an infected person
  - skin piercing
  - medical procedures
  - open wounds from an infected individual.⁶

At a global level, mother to child transmission is responsible for the largest number of infections.⁷ 80-90 per cent of infections contracted before one year of age lead to chronic infection, compared with 30-50 per cent of childhood infections and less than 5 per cent of infections contracted in adulthood.⁸ In countries where CHB infection is uncommon in the general population, most infections are acquired in adulthood, are self-limiting and result in clearing the virus from the blood and liver and long-lasting immunity to re-infection.⁹ A small proportion of adults who become infected (< 5%) fail to clear the infection and may have ongoing viral replication.⁹

Working together to lessen the impact of cancer
Chronic hepatitis B prevalence in Australia, by risk group¹⁰

<table>
<thead>
<tr>
<th>Group</th>
<th>HBsAg prevalence in risk group (%)</th>
<th>Proportion of CHB in Australia (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>People born in high or intermediate-prevalence countries</td>
<td>2.4 (average)</td>
<td>56</td>
</tr>
<tr>
<td>People born in high or intermediate-prevalence countries</td>
<td>3.6 (Asia and Pacific regions)</td>
<td></td>
</tr>
<tr>
<td>People born in high or intermediate-prevalence countries</td>
<td>2.7 (Africa/Middle East)</td>
<td></td>
</tr>
<tr>
<td>People born in high or intermediate-prevalence countries</td>
<td>1.0 (Europe)</td>
<td></td>
</tr>
<tr>
<td>Aboriginal and Torres Strait Islander people</td>
<td>3.7</td>
<td>9</td>
</tr>
<tr>
<td>People who inject drugs</td>
<td>4</td>
<td>6</td>
</tr>
<tr>
<td>Men who have sex with men</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Non-indigenous Australian-born individuals*</td>
<td>0.3</td>
<td>19</td>
</tr>
<tr>
<td>Other or not stated</td>
<td>1.0</td>
<td>6</td>
</tr>
</tbody>
</table>

*excluding those belonging to the other priority populations listed above CHB, chronic hepatitis B; HBsAg, hepatitis B surface antigen

Additional reading
Further references to hepatitis B and liver cancer are available in B Positive. All you wanted to know about hepatitis B. A guide for primary care providers.¹¹
Symptoms of hepatitis B infection
The acute infection phase is commonly asymptomatic, but some people experience:
- jaundice
- dark urine
- extreme fatigue
- nausea
- vomiting and abdominal pain for a period of several weeks.
Acute liver failure occurs rarely, but can lead to death.6

Hepatitis B diagnosis
The hallmark of hepatitis B infection is the detection of the hepatitis B surface antigen – HBsAg in the serum.

Acute hepatitis B virus (HBV) infection is characterised by the presence of HBsAg and immunoglobulin M (IgM) antibody to the core antigen, HBcAg. During the initial phase of infection, patients are also seropositive for hepatitis B e antigen (HBeAg). HBeAg is usually a marker of high levels of replication of the virus. The presence of HBeAg indicates that the blood and body fluids of the infected individual are highly contagious.

Chronic hepatitis B infection is characterised by the persistence of HBsAg for at least six months (with or without concurrent HBeAg). Persistence of HBsAg is the principal marker of risk for developing chronic liver disease and liver cancer (hepatocellular carcinoma) later in life.6

See Figure 1.

Ascertaining the extent of liver fibrosis is critical for decision-making in patients with chronic liver disease. Transient elastography (TE, or FibroScan) has replaced liver biopsy as the clinical armamentarium for the diagnosis and assessment of liver fibrosis.

Figure 1: Natural history of chronic hepatitis B infection

Hepatitis B treatment
Acute hepatitis B generally requires supportive care only.
Chronic hepatitis B infection may require treatment with antiviral agents to slow the progression of cirrhosis, reduce the incidence of liver cancer and improve long-term survival.6 Current treatments are rarely curative, but suppress replication of the virus, so antiviral treatments are long-term (typically lifelong).

Prevention of hepatitis B and liver cancer
A comprehensive public health response to hepatitis B needs to integrate primary, secondary and tertiary prevention into a coordinated response.

Primary prevention of hepatitis B and liver cancer
Hepatitis B vaccination and reduced exposure to the virus (e.g. through screening blood donors and safe injection techniques) are effective approaches to primary prevention.

It is recommended that all newborns receive the first dose of hepatitis B vaccine within 24 hours of birth, followed by a further three doses in infancy, at 2, 4 and 6 months of age.

Adolescents not vaccinated as children and adults at risk of exposure, or significant exposure to HBV infection should also be targeted for vaccination. For further details, see the Australian Immunisation Handbook.

Secondary prevention: hepatitis B screening and management
As antiviral treatment can significantly reduce the incidence of HCC, screening and improved management of chronic viral hepatitis are integral components of HCC prevention.

The goals of therapy in HBV-infected patients include reducing the level of viraemia and a correction of liver dysfunction, with treatment indicated in people with chronic hepatitis B infection who have elevated ALT levels and elevated viral loads.6 Adequate control of chronic hepatitis B leads to a reduction in the risk of fibrosis and cirrhosis, HCC development and end stage liver disease. Markers of successful therapy include the clearance of HBeAg, seroconversion to anti-HBe antibodies, and a reduction in the circulating viral load.9
Tertiary prevention: screening for HCC

Liver ultrasound (US) is the test of choice for HCC screening, and combining this with serum alpha-fetoprotein (AFP) measurement increases HCC detection by a further 6-8 per cent.\textsuperscript{13} Six monthly follow-up is recommended, as a negative screening result cannot reliably exclude the presence of HCC.\textsuperscript{14}

Current management guidelines recommend that HCC surveillance should be offered to people with cirrhosis, or other HCC risk factors, including:

- Asian men over the age of 40
- Asian women over the age of 50
- African people older than 20 years of age
- Aboriginal and Torres Islander people over the age of 50\textsuperscript{15}\textsuperscript{16}
- People with a family history of primary liver cancer.\textsuperscript{15} 16

Population-level models of hepatitis B screening and treatment

Preventing HCC is contingent on educating and engaging the affected population to become active participants in their care.

In New South Wales, Western Sydney has the highest rate of liver cancer, above the national average.

Prevalence of chronic hepatitis B within this region is also above average.

The ‘Liver Wellness Program’ has been established in Western Sydney to support general practitioners and their patients living with chronic hepatitis B. More information about the program can be found at www.liverwellnessprogram.com

References

8. World Health Organization (WHO). Immunization surveillance, assessment and monitoring.: WHO.