Hepatitis in refugees who settle in Australia

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BACKGROUND

The World Health Organisation estimates that 2 billion people have been infected with hepatitis B and about 180 million people infected with hepatitis C worldwide. More than 350 million have chronic hepatitis B and 130 million have chronic hepatitis C infection. Most infections of hepatitis B and C are from unsafe injection practices, both medical and nonmedical; from household contacts; or, in the case of hepatitis B, from 'vertical' transmission from mother to child.

OBJECTIVE

This article discusses screening and management of hepatitis B and C in refugees who settle in Australia.

DISCUSSION

Most people carrying hepatitis will be asymptomatic with infection detected by screening. Refugees need counselling, education and support to come to terms with the implications of hepatitis B and C for both themselves and their families. In Australia both viruses can be treated in those with active infection and general practitioners can be involved in diagnosis, follow up and shared care management.

Case study

Narges aged 42 years, arrived in Australia with her four daughters after being detained in a detention centre for several months. Her husband Qadir, a journalist, left Afghanistan 5 years earlier and ended up in the USA. He has applied to be reunited with his family in Australia. Narges had escaped with the help of relatives after being badly beaten by the Taliban for showing her arm while shopping. She was afraid for the safety of herself and her daughters in Afghanistan and fled by sea with the help of people smugglers.

After appropriate pretest counselling you arrange blood tests for Narges and her family for hepatitis B virus (HBV) and hepatitis C virus (HCV). Blood tests show that Narges is HBV surface antibody (anti-HBs) positive, HBV core antibody (anti-HBc) positive and HBV 'envelope' antibody (anti-HBe) positive. Her two eldest daughters have no HBV antigen or antibody detected. Her two younger daughters, aged 10 and 12 years, are both HBV surface antigen (HBsAg) positive, anti-HBc positive and HBV 'envelope' antigen (HBeAg) positive. They both have normal alanine aminotransferase (ALT). Qadir has been found to be HCV antibody positive. Narges is concerned for the health of herself and her children and for the possibility of transmission of HBV and HCV within the family.

Narges probably contracted HBV between her second and third child. It was common in Afghanistan to have a tetanus injection with each pregnancy and this may have been the source of the infection. She has now cleared the virus and is immune but has passed it on perinatally to her two youngest children at a time when she was still HBsAg positive. Her two older girls do not have the infection and have no immunity. Because they are at risk of contracting the virus from their two younger sisters they will each need a course of hepatitis B vaccination. If Narges had been in Australia for the last two pregnancies the babies would have been given hepatitis B immune globulin (HBIG) and a course of hepatitis B vaccine neonatally.

The two younger girls will need to be followed up every 6 months to watch the course of the disease. There is a possibility that they may clear the virus, but may eventually need treatment to stall the development of cirrhosis or hepatocellular carcinoma. Other family members will not need following up for HBV.

When Qadir arrives from the USA he will need to have a HCV qualitative polymerase chain reaction (PCR) to ascertain whether he has cleared the virus or whether it is still active and he is infectious. If the PCR is positive, a genotype should be done and he will need to be assessed by a specialist in viral hepatitis as to whether he qualifies for treatment with pegylated interferon and ribavirin. The possibility of Qadir passing HCV on to his family by razors, toothbrushes and any circumstance involving even small amounts of blood should be discussed. There is only a limited risk of contracting HCV during sexual intercourse and Narges and Qadir should be counselled in this regard.

Health authorities will need to be notified about the children with active HBV and Qadir's HCV.

The World Health Organisation (WHO) estimates that 2 billion people have been infected with hepatitis B virus (HBV) worldwide with more than 350 million of these having chronic infection. Over 1 million people die each year from cirrhosis of the liver or hepatocellular carcinoma (HCC) caused by chronic HBV.[1] Fifty-two percent of all cases of HCC are caused by HBV and 92% of the world's HBV related HCC is estimated to occur in developing countries.[2] Most infections throughout the world are 'vertical' (ie. from mother to child), from household contacts, by sexual transmission, or from contact with unsterilised needles used by health professionals for treatment, immunisation or acupuncture.[1]

Australia, like most industrialised countries, has a predominantly 'horizontal' pattern of HBV transmission where the majority of infection is sexually acquired or through the sharing of injecting drug equipment. The hepatitis B vaccine has been available since 1982 and in 1991 the WHO called for all children to be vaccinated against HBV. In 2000, universal hepatitis B vaccination was introduced into Australia with catch up programs implemented for teenagers in schools.[1]

The notification rate of HBV fell in Australia to 1.5 per 100 000 in 1997 but has since increased to 2.2 per 100 000 in 2001. With this has come a rising prevalence of HCC.[3] This increase in notifications is thought to be due to the increase in migrants and refugees from high prevalence countries. A recent study in central Sydney (New South Wales) found that almost 70% of those with chronic HBV were born overseas.[3] Screening of refugee populations is therefore important (*Table 1*).

Table 1. Reasons for screening for HBV[6]

- Counselling about ways to reduce HBV transmission
- Vaccination of close contacts, sexual partners and household
- Infants born to HBsAg positive women need vaccine and immune globulin
- Risk for progression to cirrhosis and hepatocellular carcinoma
- Eligibility for treatment
- Educating patients on medical and social consequences of infection including public health notifications

Table 2. Prevalence of hepatitis B[1]

	Percentage of population positive		Mode of infection
Area	HBV surface antigen (infectious) (HBsAg)	HBV core antibody (past exposure) (anti-HBc)	Neonatal/childhood (vertical) or in adulthood by sexual or percutaneous transmission (horizontal)
Northern, western central Europe North America Australia	0.2–0.5	4–6	Mostly horizontal
Eastern Europe Mediterranean Russia and the Russian Federation Southwest Asia Central and South America	2–7	20–55	Frequently vertical
Parts of China Southeast Asia Tropical and sub-Saharan Africa Parts of Middle East	8–20	70–95	Mostly vertical

HBV and refugees to Australia

Refugees to Australia come from countries such as sub-Saharan Africa and Asia where 10% or more of the population have chronic HBV, the majority contracted in childhood. Children from the poorest countries have not usually been vaccinated as governments cannot afford it or have chaotic infrastructure due to war or civil unrest. Ninety percent of those infected in the first year of life and 30–50% of those infected from 1–4 years of age will go on to develop chronic infection. These children have a 25% risk of dying of the sequelae of their HBV.[1]

The greatest risk of HBV infection in refugee populations is in those children of infected mothers (*Table 2*). Other high risk groups include: those who have had medical procedures such as injections, as part of an immunisation program, with childbirth or as a preferred treatment for common illnesses;[4] or circumcision, which is becoming more common in parts of Africa as it is thought to decrease the transmission of HIV.[5] Hepatitis B increases with age and is also more common in men. The risk of HBV, as with all infectious diseases, is increased in the country of origin of refugees because of poor nutrition, crowded conditions and lack of health facilities.[6]

Immunological stages and transmissibility

The first stage of HBV infection is usually asymptomatic with:

- no changes in liver function tests, but
- active viral replication within the hepatocytes with secretion of HbsAg and HBeAg.

Table 3. Definitions for hepatitis B[1,24]

Hepatitis B virus (HBV): is a hepadnavirus, 42 nm partially double stranded DNA composed of a 27 nm nucleocapsid core (HBcAg) surrounded by an outer lipoprotein coat (also called envelope) containing the surface antigen (HBsAg). HBV is present in blood, saliva, semen, vaginal secretions and menstrual blood of infected individuals

Hepatitis B surface antigen (HBsAg): a serologic marker on the surface of HBV. It can be detected in high levels in serum during acute or chronic hepatitis. The presence of HBsAg indicates that the person is infectious. The body usually produces antibodies to HBsAg as part of the normal immune response to infection

Hepatitis B surface antibody (anti-HBs): also abbreviated as HBsAb. The presence of anti-HBs is generally interpreted as indicating recovery and immunity from HBV infection. Anti-HBs also develops in a person who has been successfully vaccinated against HBV

Total hepatitis B core antibody (anti-HBc): also abbreviated as HBcAb. Appears at the onset of symptoms in acute hepatitis B and persists for life. The presence of anti-HBc indicates previous or ongoing infection with HBV in an undefined time frame

Hepatitis B 'envelope' antigen (HBeAg): a secreted product of the nucleocapsid gene of HBV found in serum during acute and chronic HBV. Its presence indicates the virus is replicating and the infected individual has high levels of HBV

Hepatitis B 'envelope' antibody (anti-HBe): also abbreviated as HBeAb. Produced by the immune system temporarily during acute HBV infection or consistently during or after a burst in viral replication. Spontaneous conversion from e antigen to e antibody (a change known as seroconversion) is a predictor of long term clearance of HBV in patients undergoing antiviral therapy and indicates lower levels of HBV

Hepatitis B immune globulin (HBIG): a product available for prophylaxis against HBV infection. HBIG is prepared from plasma containing high titres of HBsAb and provides short term protection (3–6 months)

Hepatitis B core IgM will develop in recognition of the presence of HBV, but this immune response is not sufficient to eliminate the virus. This stage of immune tolerance can last several decades in those who contracted HBV 'vertically' but only 2–4 weeks in those who contracted HBV 'horizontally' as adults.[7]

In the second stage there is:

- an immunologic response with the development of HBV antibodies
- hepatitis B viral levels decline, but
- often 'flares' of hepatitis with increases in alanine aminotransferase (ALT) levels as the body attempts to rid itself of infected hepatocytes.

There is an increased likelihood of progressive disease in those who have more frequent flares or in those whom this phase persists for a prolonged period (weeks to years).[7] People who are immunosuppressed may have high viral loads but no illness as it is the immune response that causes the destruction of liver cells and 'flares'. If the immunosuppression improves, patients who are chronic carriers may develop severe hepatitis.

In the third phase, the body has cleared most of the virus and the ALT may return to normal. About 50% of patients who contract HBV horizontally will clear HBeAg and develop anti-HBe within 5 years of diagnosis, 70% within 10 years. A minority of patients will go on to become HBsAg negative and anti-HBs positive.[7] The persistence of HBsAg for more than 6 months usually means the patient will remain chronically infected with HBV. This has been described as an 'inactive carrier state' but the patient remains infectious and HBV can be reactivated either spontaneously or as a result of immunosuppression.[8]

The presence of maternal HBeAg increases the risk of perinatal transmission of HBV to the infant (10–40% risk to infants of HBeAg negative mothers vs. 79–90% risk to infants of HBeAg positive mothers).[7] Ninety percent of those with vertically transmitted HBV remain HbsAg positive[9] and rarely convert to HBeAg negative and anti-HBe positive.

Blood tests and what they mean

The interpretation of blood tests for HBV is confusing, but for general practitioners screening refugees for HBV it is important to have some understanding of their meaning so as to decide on prevention, follow up and treatment options. *Table 3* and *4* describe the various tests and their interpretation.

Screening, diagnosis and monitoring

Screening initially involves testing for:

- anti-HBs to check immunity, and
- HBsAg to check for infectivity (*Figure 1*).

Some clinicians and laboratories will also do routine anti-HBc to screen for past infection.

If HBsAg is positive then:

- ALT
- HBeAg and anti-HBe

should be done to guide further management. The presence of HBeAg indicates a high viral load and often there will be active hepatitis. It is not found in people who are HBsAg negative. Those who are HBeAg positive therefore require more regular monitoring of their liver function. A person who is HBsAg positive and negative for both HBeAg and anti-HBe may have a precore mutant and the viral load should be measured. Commonly those with a precore mutant will be anti-HBe positive and the extent of disease can only be clarified with a viral load assessment.[8] Because of the current cost of this test it needs to be done in the hospital environment. However, in the near future hepatitis B DNA assays will be publicly funded for pretreatment and monitoring of patients with chronic HBV.

Those who are HBsAg positive have a 5 year cumulative incidence of progression to cirrhosis of 8–20%. Without treatment, cirrhosis can cause ascites, jaundice, bleeding and hepatic encephalopathy with only a 71% survival rate at 5 years (*Table 5*).[9]

Screening for HCC by ultrasound and

α-fetoprotein should be done in patients who are chronically infected, in particular those at high risk (eg. men aged >45 years, those with cirrhosis, and those with a family history). Hepatocellular carcinoma arises most commonly in patients with cirrhosis but can also occur with only minimal signs of other liver damage.[9] A male child infected at birth has a 25–40% lifelong risk of developing cirrhosis or HCC.[7] Therefore GPs need to continue to follow up those who are HBsAg positive looking for both evidence of cirrhosis and of HCC. An online algorithm to help with screening can be accessed at http://clinicaloptions.com/hepatitis/management%20series/chronic%20care.aspx.

Co-infection with hepatitis D

The hepatitis D virus (HDV) is a defective virus whose outer coat is derived from HBV surface antigen. Hence it needs the HBV to exist and is transmitted by the same routes. Hepatitis D virus can be acquired either as a co-infection (occurs simultaneously) with HBV or as a superinfection in those with existing chronic HBV infection. Hepatitis D antigen and antibody should be requested where liver function tests are persistently elevated in a person who is HBsAg positive but has a low viral load and no other comorbid risk factors. HBV-HDV co-infection may have more severe acute disease and a higher risk (2–20%) of developing acute liver failure compared with those infected with HBV alone. Chronic HBV carriers who acquire HDV superinfection usually develop chronic HDV infection and have an increased risk of progression to cirrhosis.[10]

Treatment

Treatment for HBV will take place after referral to a specialised unit, and liver biopsy for those with abnormal ALT levels and a high viral load. Success is defined as:

- loss of HBeAg
- elimination of detectable HBV from the blood, and
- normalisation of liver function tests.[9]

Lamivudine, entecavir and adefovir are nucleoside analogues that have conventionally been used in the treatment of HBV. They are well tolerated and have few side effects with a seroconversion rate from HBeAg positive to HBeAg negative of 73% at 4 years.[9] Problems include the development of drug resistance and the low rates of sustained seroconversion.[11] Weekly subcutaneous pegylated interferon has recently been approved for use in those with liver biopsy signs of cirrhosis and/or fibrosis with a success rate of 40% after a 6 month course.[9] Early diagnosis of HCC allows for

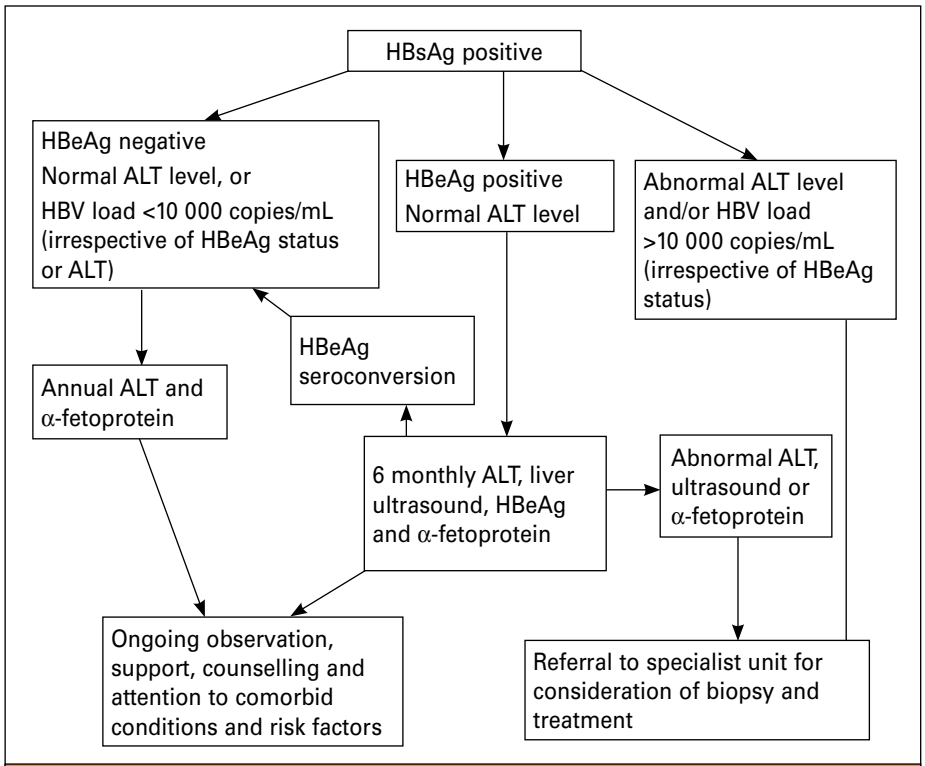

Figure 1. Screening and monitoring algorithm for hepatitis B[8]

Table 4. Interpretation of HBV test results[24]

HBsAg anti-HBc anti-HBs	Negative Negative Negative	Susceptible to infection
HBsAg anti-HBc anti-HBs	Negative Positive Positive	Immune due to natural infection
HBsAg anti-HBc anti-HBs	Negative Negative Positive	Immune due to hepatitis B vaccination
HBsAg anti-HBc anti-HBc IgM anti-HBs	Positive Positive Positive Negative	Acutely infected
HBsAg anti-HBc anti-HBc IgM anti-HBs	Positive Positive Negative Negative	Chronically infected
HBsAg anti-HBc anti-HBs	Negative Positive Negative	Four interpretations possible*

* Might be: recovering from acute HBV infection, distantly immune and test not sensitive enough to detect very low level of anti-HBs in serum, susceptible with a false positive anti-HBc, or undetectable level of HBsAg present in the serum and the person is actually chronically infected

treatment by transplantation, ablation, resection, hemihepatectomy, intra-arterial chemotherapy, embolisation or percutaneous intralesional ethanol injection.[9]

Hepatitis C

The WHO estimates that about 180 million people have been infected with hepatitis C (HCV) and about 130 million still have chronic infection. It has estimated that 3–4 million new infections occur each year.[12]

In Egypt, where many Sudanese refugees stay before they come to Australia, the prevalence of HCV antibodies is 15–20%.[13] Prevalence of HCV antibodies is 5% in young sexually active adults in the Central African Republic[14] and 3% and 8% respectively in migrants to Australia from Laos and Cambodia.[15] The prevalence in Australia is <1%.[12] The prevalence in Afghanistan (where our case study family is from), is not accurately known but it is probably around 1–2%.[16]

Transmission

Hepatitis C virus is blood borne and only usually infects humans. Processes that move blood from one person to another transmit it. The major risk factor for transmission is exposure to contaminated needles and syringes. This may occur due to medical, surgical or dental treatment particularly in resource poor settings or in the context of injecting drug use.[17] The risk of mother to child transmission is around 5%. The factors that put someone at increased risk of HCV may also increase their risk of contracting HIV.

Sexual transmission is possible although rare, and can occur if blood is present during sex, particularly in the presence of genital ulcer disease (eg. herpes). Transmission has been reported in HIV positive men having unprotected anal intercourse.[18]

Course of HCV

Hepatitis C virus is cleared by the immune system in about 30% of people infected.[19] If HCV is cleared, the antibody remains in most people but the virus is not detected by polymerase chain reaction (PCR). There is no ongoing liver damage and the person cannot infect others. Viral material may be identified within some cells but the person is not infective. The antibody does not protect against further infection.

When someone is identified as HCV antibody positive, qualitative HCV PCR must be performed to determine whether the virus is present. Acute HCV can occur up to 6 months following infection.[20] It is usually a relatively mild disease and most people do not recognise they have been infected.

Chronic HCV is usually indolent.[19] Hepatocytes are the primary focus for the virus but the virus is not directly cytotoxic. Damage is done to the liver and other body organs through immune complex deposition and autoimmune stimulation but occurs slowly over many years.

Liver injury is rated on the degree of fibrosis and continuing activity.[20] Cirrhosis, decompensation and HCC are possible liver related disease outcomes after many years of infection (*Table 5*). Ongoing liver damage is more common in people with abnormal liver function tests (LFTs) but also occurs in about 20% of people with normal LFTs.[20]

Table 5. Clinical evidence of cirrhosis[25]

- Subtle features include:
  - lethargy
  - right upper quadrant discomfort
  - hard liver edge
  - splenomegaly
  - blood tests
  - slightly low serum albumin
  - slightly prolonged INR
  - aspartate aminotransferase (AST) greater than ALT
  - thrombocytopaenia
  - cryoglobulinaemia
- May be asymptomatic
- Skin rash, palpable purpura on lower limbs, proteinuria
- More significant features
  - ascites
  - muscle wasting
  - hepatic encephalopathy
  - upper gastrointestinal bleeding from oesophageal or gastric varices
  - hypersplenism
- Thrombocytopaenia, leukopaenia

Treatment

Treatment is available and should be discussed with all patients who have a positive PCR (*Figure 2*). Treatment is with pegylated interferon and ribavirin.[21] Treatment outcome depends on genotype. In Australia, the most common genotypes are 1 and 3.[22] Genotypes 3 and 2 require 6 months treatment with ~80% chance of clearing the virus (sustained viral response [SVR]). Genotypes 1 and 4 require 12 months treatment and have SVR of about 50%. Response rates are decreased if cirrhosis is present.

Some people progress through treatment without difficulty. However, most will experience side effects including flu-like symptoms, irritability, insomnia, anorexia, nausea, vomiting, rash, myalgia, arthralgia, depression and psychosis.[23] Bone marrow depression can lead to anaemia, thrombocytopaenia and leukopaenia. Treatment can also precipitate thyroid dysfunction.

Treatment may aggravate existing diabetes, epilepsy, psoriasis, depression and psychosis. Patients must be adequately advised, and if there is a history of depression or psychosis, assessed by a psychiatrist experienced in managing people being treated with interferon before commencing treatment.

Treatment can only be undertaken once within the Australian Pharmaceutical Benefits Scheme (PBS). If a person withdraws from treatment or their virus is resistant, pegylated interferon is no longer available to them under the PBS. Further information about HCV can be found at www.racgp.org.au/guidelines/hepatitisc.

Counselling

A pretest discussion to gain informed consent should be done with an accredited interpreter. It might include the following: 'there are some illnesses that might have only minimal or no symptoms but which can still be important and are often successfully treated in Australia. This includes syphilis, schistosomiasis, hepatitis B, hepatitis C and HIV. We test for these illnesses so we can try to protect people from their

effects if left untreated and help protect the rest of the family from catching them. Some of the illnesses are contagious and we need to tell the appropriate health worker at the department of health if you have them as they like to be sure that we are looking after you and your family. If you're uncomfortable about having any of these tests now and would like to think about it or discuss it further with the doctor or your family, we can talk about it again later'.

For many refugee patients, the only blood borne virus they have known is HIV and they may be confused about the implications and significance of HBV or HCV. The diagnosis of a potentially serious illness with difficult treatment will be added to other stressors such as a new country, a strange health system, limited support and the fear of transmission. A good understanding about HBV and HCV, thoroughly explained with the aid of an accredited interpreter, is of the utmost importance at all stages through screening, vaccination, notification to health authorities, follow up and treatment. Many states have support services such as PEACE or the Hepatitis Council (*Table 6*).

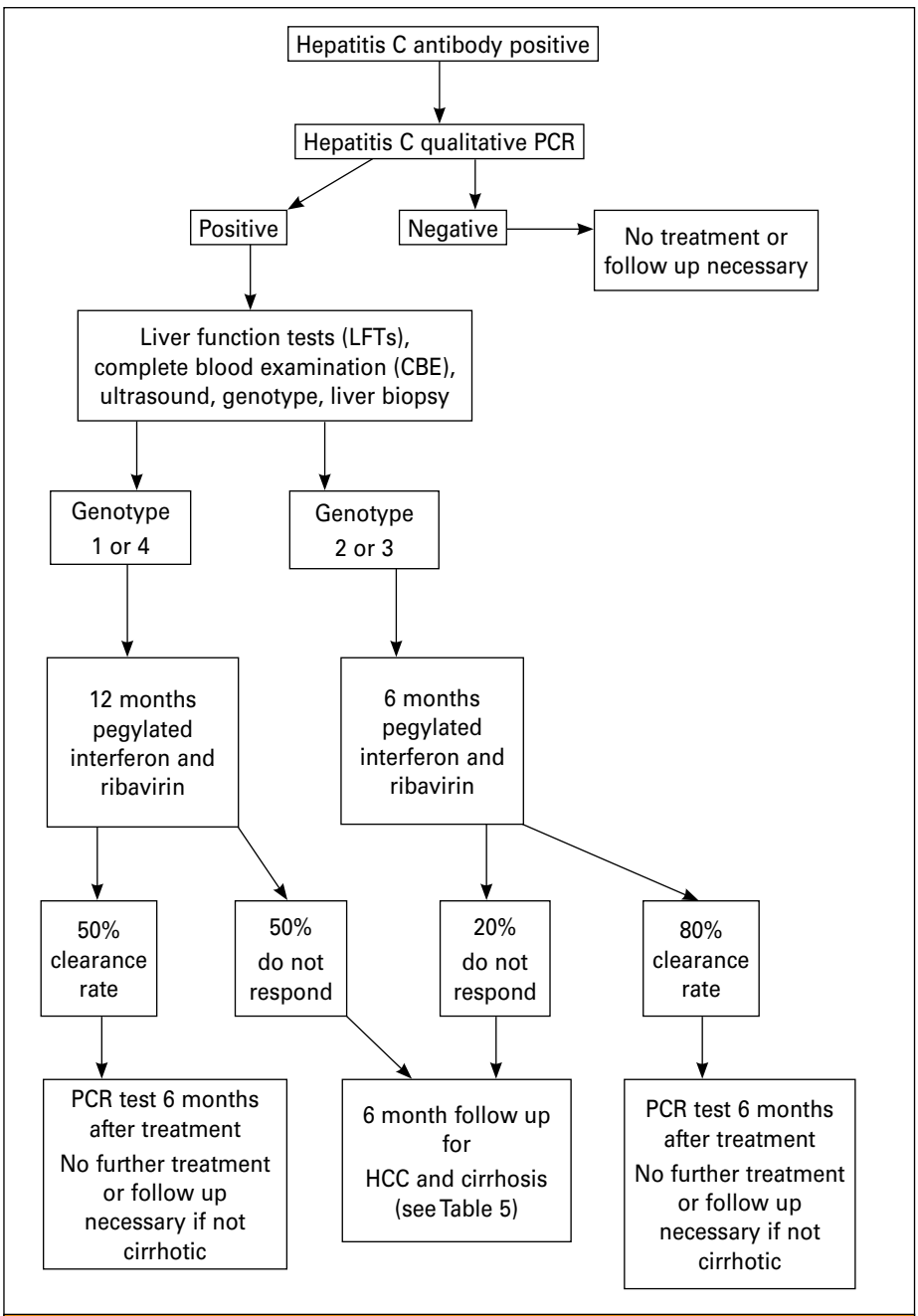

Figure 2. Hepatitis C treatment algorithm

Conclusion

As more people settle in Australia from countries with a higher prevalence of HBV and HCV, all GPs will need to be aware of the latest screening and treatment protocols. Refugees often come to Australia from countries with limited health care facilities and may be asymptomatic and unaware that they are carrying these blood borne viruses. The diagnosis, treatment and prognosis of hepatitis depends on appropriate screening, immunisation, follow up and referral. For people such as Qadir, Narges and their children, education and support are important and needs to be ongoing.

Conflict of interest: none declared.

Acknowledgment

The authors would like to thank Professor Justin La Brooy for his support in the preparation of this article.

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Table 6. Support services

Australian Hepatitis Council
Tel 02 6232 4257
Fax 02 6232 4318
Email info@hepatitisaustralia.com
www.hepatitisaustralia.com

Australian Capital Territory
Tel 02 6257 2911
Fax 02 6257 1611
Email info@acthepc.org
www.acthepc.org

New South Wales
Tel 02 9332 1599
1800 803 990 (country)
Fax 02 9332 1730
Email hccnsw@hepatitisc.org.au
www.hepatitisc.org.au

Northern Territory
NT AIDS and Hepatitis Council
Tel 08 8941 1711
1800 880 899 (free call line)
Fax 08 8941 2590
Email info@ntahc.org.au
www.ntahc.org.au

Queensland
Tel 07 3236 0610
1800 648 491 (country)
Fax 07 3236 0614
Email admin@hepatitisc.asn.au
www.hepatitisc.asn.au

South Australia
Tel 08 8362 8443
1800 021 133 (country)
Fax 08 8362 8559
Email admin@hepccouncilsa.asn.au
www.hepccouncilsa.asn.au

Tasmanian Council on AIDS, Hepatitis and Related Diseases
Tel 03 6234 1242
1800 005 900 (country)
Fax 03 6234 1630
Email mail@tascahrd.org.au
www.tascahrd.org.au

Victoria
Tel 03 9380 4644
1800 703 003 (country)
Fax 03 9380 4688
Email info@hepvic.org.au
www.hepcvic.org.au

Western Australia
Information line 08 9328 8538
Tel 08 9227 9800
1800 800 070 (country)
www.hepatitiswa.com.au

South Australia
Email nceta@flinders.edu.au
www.nceta.flinders.edu.au

PEACE Multicultural HIV and Hepatitis Services
Tel 08 8245 8100
Mobile 0418 824739
Email e.oudih@rasa.org.au

Multicultural HIV/AIDS & Hepatitis C Service
Tel 02 9515 5030
Fax 02 9550 6815
www. multiculturalhivhepc.net.au

Multicultural Health & Support Service
Tel 03 9342 9721
Fax 03 9342 9799
Mobile 0400 075983
Email naomin@nrchc.com.au

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