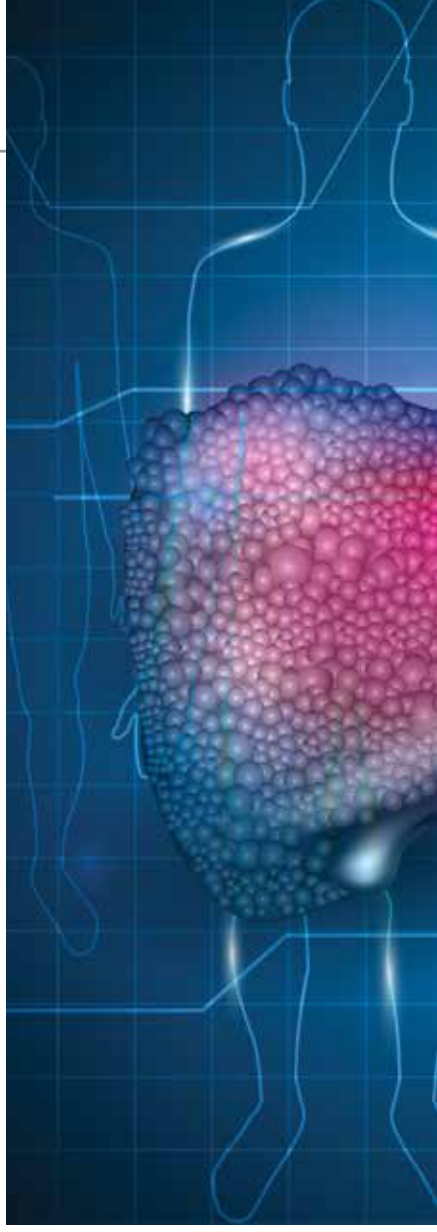

Managing the rising burden of chronic liver disease

Nonalcoholic fatty liver disease and alcoholic liver disease

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The rising prevalence of chronic liver disease is contributing to higher rates of cirrhosis, liver failure and liver cancer. GPs can share the increasing burden with specialist services by recognising chronic liver disease, assessing its severity, managing risk factors and comorbidities and referring appropriately. Two of the four most prevalent chronic liver diseases are discussed in this article, namely nonalcoholic fatty liver disease and alcoholic liver disease.

KEY POINTS

- Nonalcoholic fatty liver disease (NAFLD) is by far the most common chronic liver disease in Australia, accounting for about 90% of cases. Chronic hepatitis B, chronic hepatitis C and alcoholic liver disease are the next most common.
- Alcoholic liver disease is the most common cause of hospitalisation and mortality from advanced liver disease.
- The rising prevalence of chronic liver disease contributes to increasing rates of cirrhosis, liver failure and liver cancer.
- In patients with NAFLD, the leading cause of mortality is cardiovascular disease.
- It is essential to recognise risk factors for liver disease in order to diagnose patients and then assess and manage them to prevent disease progression.
- Patients with advanced fibrosis and cirrhosis of any cause should be referred to a liver specialist.

Chronic liver disease affects more than 6 million people in Australia and the prevalence is expected to rise to over 8 million by 2030.[1] Nonalcoholic fatty liver disease (NAFLD) is by far the most prevalent of the chronic liver diseases, representing about 90% of all cases. Chronic hepatitis B and C together represent about 5% of all cases. Alcoholic liver disease is also prevalent and is the most common cause of hospitalisation and mortality from advanced liver disease.

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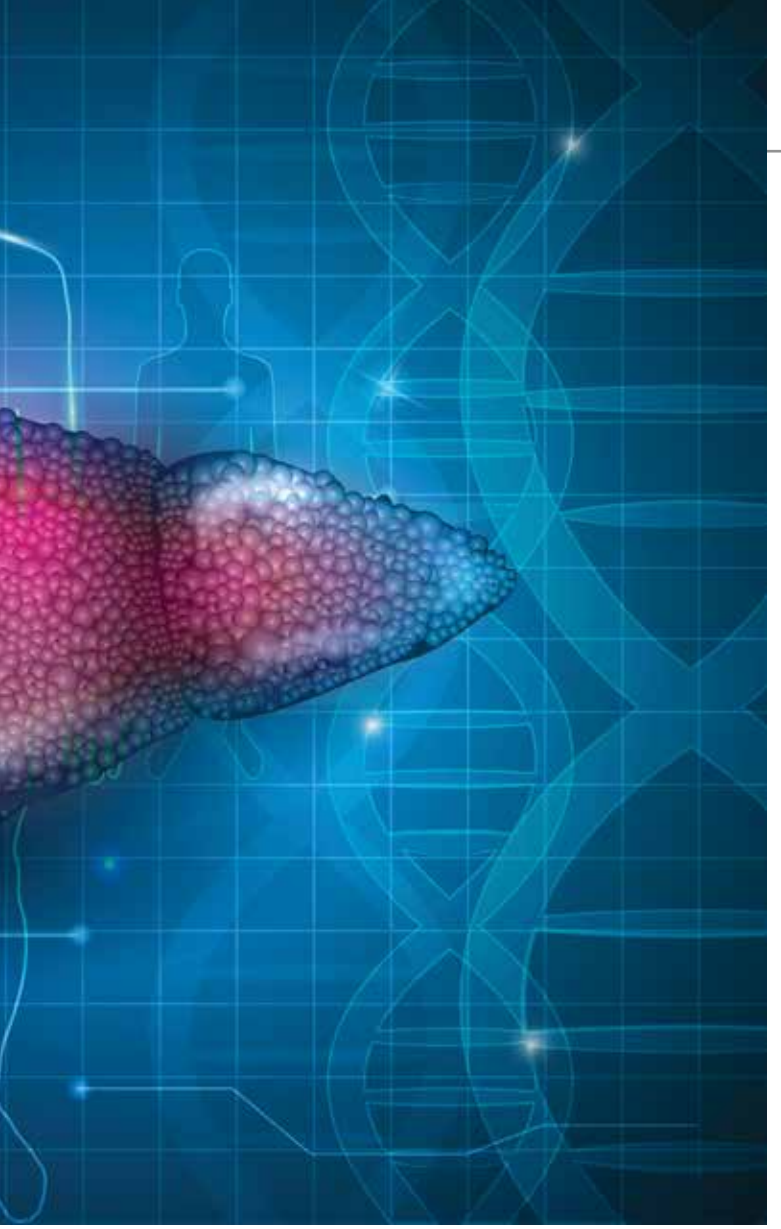


This article aims to help GPs recognise and diagnose patients with the chronic liver diseases NAFLD and alcoholic liver disease in primary care, assess the severity of liver disease using clinical and noninvasive markers, recognise the need to manage comorbidities in patients with and at risk for chronic liver disease and appropriately refer patients who require specialist assessment and management. Chronic hepatitis B and C will be discussed in a separate article, to be published in the February 2017 issue of *Medicine Today*.

Nonalcoholic fatty liver disease

Case study 1

Mrs FL, aged 66 years, presents to her GP with vague abdominal discomfort. She has had abnormal liver function tests for many years. She has longstanding type 2 diabetes and hypertension, and is obese (body mass index, 44 kg/m^2). She drinks 10 to 20 g of alcohol about four times a week. On examination, she has palmar erythema and three spider naevi on her chest; mild ankle oedema is observed.

Investigations reveal an albumin level of 34 g/L (normal range [NR] >35 g/L), and elevated liver enzyme levels (alanine aminotransferase [ALT], 54 U/L [NR <35 U/L]; aspartate aminotransferase [AST], 63 [NR <35 U/L]; gamma glutamyl transferase [GGT], 86 U/L [NR <60 U/L]). The glycated haemoglobin (HbA_{1c}) level is 55 mmol/mol (7.2%). Serum lipids measurement shows total cholesterol level of 6.0 mmol/L (NR <4.0 mmol/L), HDL cholesterol 0.5 mmol/L (NR >1.0 mmol/L), LDL cholesterol 2.9 mmol/L (NR <2.5 mmol/L) and triglycerides 3.5 mmol/L (NR <1.5 mmol/L). The platelet count is 130 x 10^9/L (NR 150 to 400 x 10^9/L). A liver ultrasound demonstrates an enlarged liver with diffuse fatty infiltration and mild splenomegaly. A 4 cm mass is seen in the left lobe of the liver.

What is the most likely diagnosis?

Mrs FL has probable NAFLD complicated by cirrhosis and hepatocellular carcinoma (HCC). She has features of the metabolic syndrome (obesity, type 2 diabetes, hypertension, dyslipidaemia), which are known risk factors for NAFLD and nonalcoholic steatohepatitis (NASH). She has physical signs of cirrhosis (spider naevi, palmar erythema), a low serum albumin level (indicating reduced hepatic synthetic function) and thrombocytopenia and mild splenomegaly (indicating early portal hypertension), due to the NAFLD-associated cirrhosis.

Of greatest concern is that Mrs FL's abdominal pain and a mass lesion in the liver seen on ultrasound in this setting indicate probable liver cancer. She is therefore referred to a liver specialist service. A 4-phase CT scan of the liver demonstrates typical radiological features of HCC. She is currently undergoing transarterial chemoembolisation (TACE) to treat the tumour.

NAFLD is a spectrum of liver disease characterised by accumulation of liver fat (steatosis) that, by definition, occurs in the absence of significant alcohol intake (≥30 g/day for men and ≥20 g/day for women; one standard drink in Australia contains 10 g alcohol). NAFLD ranges from bland hepatic steatosis without significant liver inflammation (nonalcoholic fatty liver [NAFL], 70 to 75% of patients) to NASH (25 to 30% of patients), an inflammatory process causing hepatocellular injury that has the potential to progress to hepatic fibrosis, cirrhosis and liver cancer.

NAFLD has emerged as the leading cause of liver disease in Australia, in line with the rising prevalence of overweight and obesity in the Australian population. In 2012, NAFLD was the second highest cause of death from liver failure or liver cancer in Australia (behind chronic hepatitis C), although this mortality associated with NAFLD is likely to surpass that associated with hepatitis C in the long term due to both the rising prevalence of obesity and the highly efficacious treatments now available for the eradication of chronic hepatitis C.[1,2] The leading cause of mortality in patients with NAFLD, however, is cardiovascular disease.

TABLE 1. PHYSICAL SIGNS SUGGESTIVE OF CIRRHOSIS

Physical sign*	Mechanism in cirrhosis
Palmar erythema, spider naevi; tender gynaecomastia in males	Elevated endogenous oestrogen due to reduced hepatic metabolism
Leuconychia, leg oedema	Low albumin due to reduced hepatic synthesis of albumin
Jaundice, scleral icterus	Impaired hepatic excretion of bilirubin
Bruises, easy bleeding	Reduced hepatic synthesis of clotting factors; low platelets due to hypersplenism (if associated portal hypertension is present)
Dilated veins on abdominal wall and/or around umbilicus (caput medusa)	Portal hypertension
Liver – hepatomegaly	Fatty infiltration, alcoholic hepatitis, other infiltrative liver disease; hepatocellular carcinoma
Liver – small, nodular, hard	Shrunken nodular liver due to chronic hepatic fibrosis; hepatocellular carcinoma
Splenomegaly	Portal hypertension
Ascites	Portal hypertension, fluid retention
Asterixis (hepatic flap), hepatic fetor, spectrum of neurological depression from increased somnolence, impaired memory, disorientation to coma	Hepatic encephalopathy due to liver failure and/or portosystemic shunting; accumulation of nitrogenous and other compounds and consequent diffuse disturbances of brain function
General: muscle wasting, weight loss	Overall catabolic state in advanced liver disease/cirrhosis; under-nutrition

* Multiple differential diagnoses; finding should be considered in context with other clinical signs and investigation results.

Risk factors for NAFLD

In the primary care setting, the presence of NAFLD should prompt the evaluation and management of the associated risk factors, which also contribute to morbidity and mortality.

Insulin resistance is the key antecedent to the development of NAFLD. NAFLD could be considered the hepatic manifestation of the metabolic syndrome due to its strong association with insulin resistance, type 2 diabetes, central obesity and dyslipidaemia. Although NAFLD is present in 20 to 30% of the general population, the prevalence is as high as 70 to 90% among people with obesity and type 2 diabetes.

Other conditions associated with NAFLD include polycystic ovary syndrome, hypothyroidism and obstructive sleep apnoea.

Consequences of NAFL and NASH

Cirrhosis will develop in approximately 4% of patients with NAFL and 20% of patients with NASH.[3] Although the liver-related mortality (liver failure and HCC) associated with NAFLD is of concern due to its rising prevalence, liver disease is the third leading cause of death in these patients behind cardiovascular disease and cancers other than HCC. It is prudent to emphasise that in patients who have progressed to NASH, half of the deaths are due to cardiovascular disease or malignancy.[4] This highlights the importance of identifying and managing risk factors to reduce cardiovascular disease and cancer in patients with NAFLD.

A common concern is that prescribing statins (HMG-CoA reductase inhibitors) in patients with NAFLD and abnormal liver function tests (LFTs) may cause hepatic toxicity. There is a lack of evidence that patients with NAFLD are at increased risk for serious drug-induced liver injury from statins, hence statins can be prescribed to treat dyslipidaemia in those with NAFLD. Statin use reduces the risk for cardiovascular events by 68% in patients with abnormal LFTs and may reduce the incidence of HCC.[5]

As NAFLD becomes more common, it is becoming a significant co-factor in individuals with concomitant chronic liver disease (chronic hepatitis B, chronic hepatitis C and/or alcoholic liver disease), hastening the progression of the liver disease, again highlighting the importance of identifying and managing these patients. Ongoing management of NAFLD and the associated cardiovascular risk is essential in these individuals even when viral hepatitis is effectively treated.

Noninvasive assessment of liver fibrosis in NAFLD

The greatest predictor of liver-related mortality in patients with NAFL or NASH is the presence of hepatic fibrosis.[6] Noninvasive tools are available to estimate the risk and degree of hepatic fibrosis in NAFLD and it is essential that every patient with NAFLD undergoes an assessment of hepatic fibrosis in primary care. Most patients with NAFLD do not have significant fibrosis and this assessment facilitates referral for specialist review of only those with the highest likelihood of significant hepatic fibrosis, with referral being avoided in many patients.

The NAFLD Fibrosis Score may be used in the primary care setting to estimate the degree of hepatic fibrosis and guide management. It is calculated using age, body mass index (BMI), presence of impaired fasting glucose or type 2 diabetes, platelet count and AST, ALT and albumin levels. An online calculator is available at

TABLE 2. INVESTIGATION RESULTS SUGGESTIVE OF CIRRHOSIS

Test	Result	Mechanism in cirrhosis
FBC	Low platelets	Portal hypertension; low 'normal range' platelets should also raise suspicion for cirrhosis and portal hypertension
EUC	Low sodium*	Impaired excretion of sodium with net fluid retention; diuretic effects
	Elevated creatinine*	Diuretic effects; hepatorenal syndrome; chronic kidney disease
LFTs	Elevated bilirubin*	Impaired hepatic excretion
	Low albumin*	Reduced hepatic synthesis of albumin; under-nutrition
	Elevated liver enzymes	Indicative of presence of liver disease; pattern may suggest underlying aetiology
Coagulation	Elevated INR; prolonged PT	Reduced hepatic synthesis of coagulation factors
Liver ultrasound	Small nodular liver ± liver lesions	Shrunken nodular liver due to chronic hepatic fibrosis ± hepatocellular carcinoma
	Splenomegaly, varices, recanalisation of umbilical vein, ascites	Portal hypertension

Abbreviations: EUC = electrolytes, urea and creatinine; FBC = full blood count; INR = international normalised ratio; LFT = liver function test; PT = prothrombin time.
* Multiple differential diagnoses; finding should be considered in context with other clinical signs and investigation results.

www.nafldscore.com; a low NAFLD score (below –1.455) excludes advanced fibrosis with high accuracy (negative predictive value of 88 to 93%), and a high score (above 0.675) indicates advanced fibrosis with high accuracy (positive predictive value, 82 to 90%).[7]

Transient elastography (FibroScan) is a noninvasive ultrasound-based modality that can be useful in the assessment of fibrosis in NAFLD. Testing can be accessed through specialist centres and is becoming increasingly available. Transient elastography measures liver stiffness (in kPa), which correlates well with the degree of hepatic fibrosis (fibrosis stage F0 to F4 [cirrhosis]) on liver biopsy. Other available (but less well validated) noninvasive measures of hepatic fibrosis include shear wave elastography and acoustic radiation force impulse (ARFI).

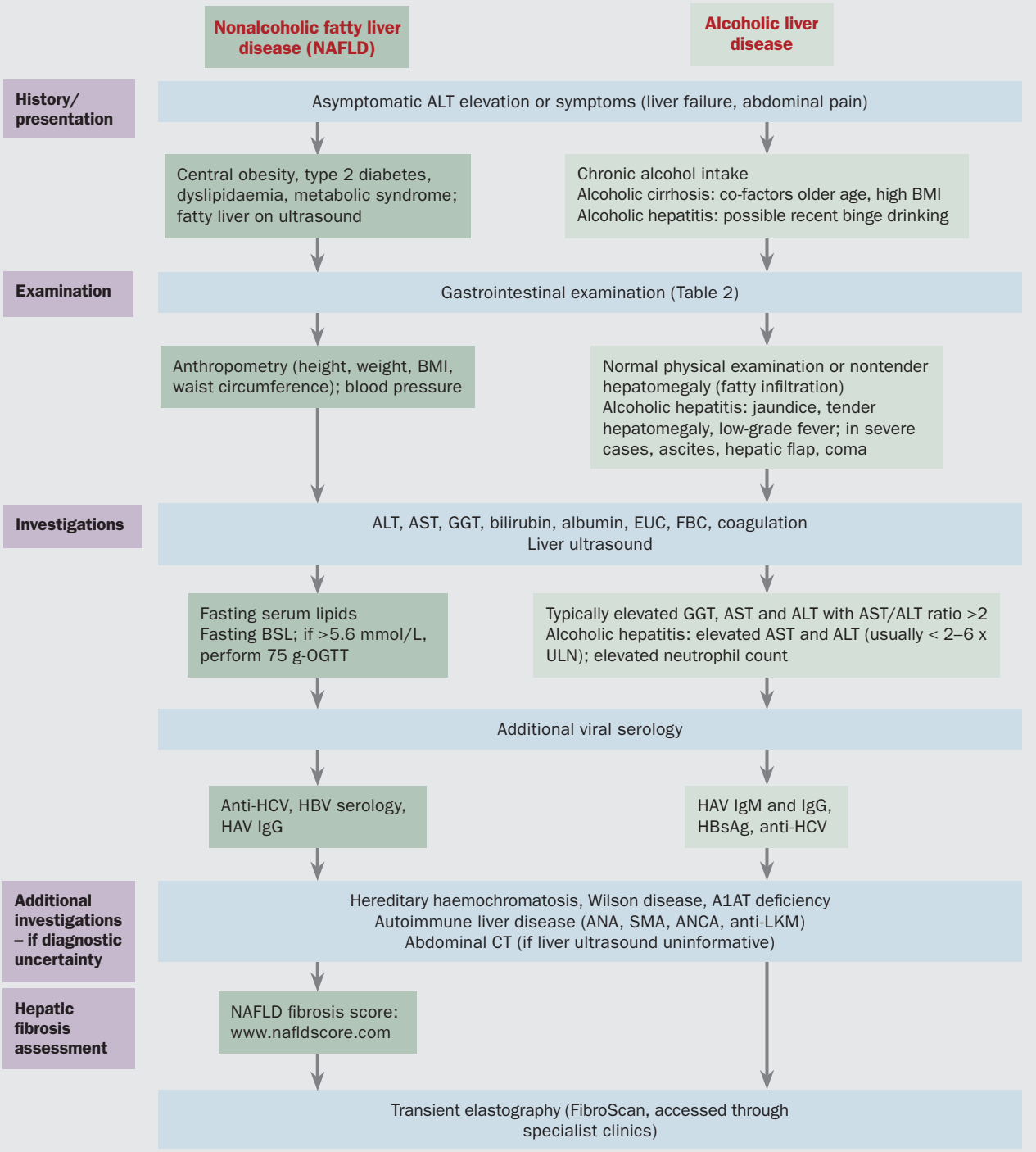
Management of NAFLD in the primary care setting

Patients with NAFLD with low likelihood of significant fibrosis can be managed in primary care with particular attention to management of cardiovascular disease and lifestyle modification. Clinical features and investigation results suggestive of cirrhosis are given in Tables 1 and 2. The key steps in evaluating a patient with suspected NAFLD are outlined in Flowchart 1.

Lifestyle modifications are the mainstay of NAFLD management (Flowchart 2). Efforts to achieve and maintain a healthy body weight through calorie-restricted diet and exercise are recommended. Loss of at least 3 to 5% of body weight may improve steatosis, but a greater weight loss (up to 10%) may be needed to improve hepatic necroinflammation.[8,9] Referral to a dietitian to provide specific nutritional recommendations may benefit patients with NAFLD and also type 2 diabetes and/or dyslipidaemia but there is only weak evidence for dietary macronutrient recommendations for those with NAFLD alone (low carbohydrate *vs* high carbohydrate diets, low fat *vs* high fat diets, unsaturated fat *vs* saturated fat diets). Avoidance of foods containing fructose (to reduce insulin resistance) and trans fats (to reduce triglycerides) is recommended.[8] Although there is currently limited evidence that the Mediterranean diet (which is high in monounsaturated fatty acids) influences NAFLD itself, there is strong evidence that this diet is beneficial for NAFLD-related disease states such as metabolic syndrome and cardiovascular disease and their risk factors, and it should therefore be considered in patients with NAFLD.[9,10]

Exercise (independent of its effects on weight loss) has been shown to reduce the degree of hepatic steatosis.[8] Exercise could therefore be recommended, such as a brisk walk of 30 to 60 minutes duration two or three times a week. Patients with NAFLD should also be counselled not to consume 'heavy' amounts of alcohol.[8] National Health and Medical Research Council (NHMRC) guidelines recommend consumption of no more than two standard drinks per day in healthy individuals.[11] There is currently no clear evidence on how much is a safe alcohol level to consume in the setting of NAFLD. In clinical practice, we recommend alcohol abstinence for patients with significant or advanced hepatic fibrosis and all patients with cirrhosis. Patients with NAFLD with bland steatosis (normal LFTs, no steatohepatitis) could conservatively be recommended a limit of one standard drink per day.

1. NAFLD AND ALCOHOLIC LIVER DISEASE: AN APPROACH TO PATIENT EVALUATION



Abbreviations: A1AT = alpha-1 antitrypsin; ALT = alanine aminotransferase; ANA = antinuclear antibody; ANCA = antineutrophil cytoplasmic antibodies; anti-HCV = hepatitis C virus antibody; anti-LKM = anti-liver kidney microsomal antibody; AST = aspartate aminotransferase; BMI = body mass index; BSL = blood sugar level; CT = computed tomography; EUC = electrolytes, urea and creatinine; FBC = full blood count; GGT = gamma glutamyl transferase; HAV = hepatitis A virus; HBsAg = hepatitis B surface antigen; HBV = hepatitis B virus; HCV = hepatitis C virus; IgG = immunoglobulin G; IgM = immunoglobulin M; NAFLD = nonalcoholic fatty liver disease; OGTT = oral glucose tolerance test; SMA = smooth muscle antibody; ULN = upper limit of normal.

Management of NAFLD in specialist centres

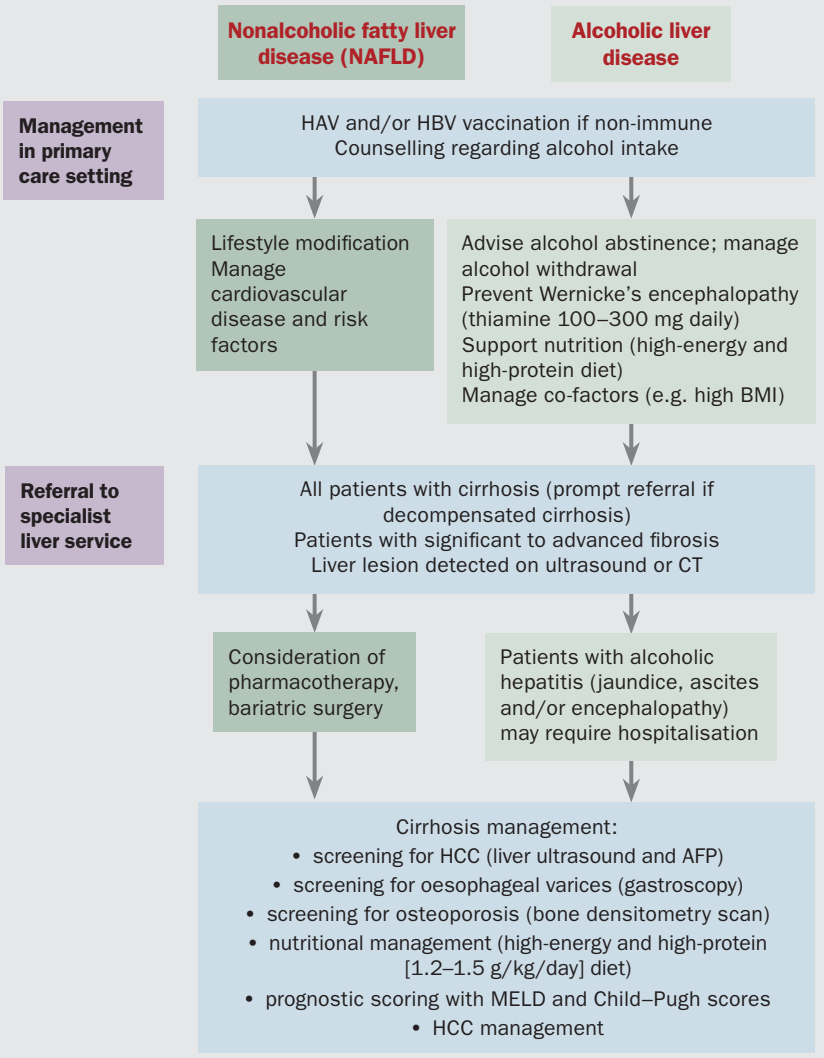
Referral to a specialist service is recommended in patients with positive noninvasive indicators of significant or advanced fibrosis and in those who have already developed clinical features of cirrhosis. Liver biopsy is not always indicated in NAFLD when clinical and noninvasive assessments provide sufficient information to guide management. Liver biopsy may still be useful when specific pharmacotherapy is being considered (see below) or when cirrhosis or concomitant liver disease requiring alternative management may be present that cannot be excluded with other methods.

A major role of specialists in the care of patients with advanced hepatic fibrosis or cirrhosis is to assess and monitor for liver-related complications such as HCC and oesophageal varices. Pre-emptive management and early diagnosis of complications has a significant impact on survival of these patients.

Specific pharmacotherapy for NAFLD is limited. Pioglitazone has been shown to improve steatohepatitis in patients with biopsy-proven NASH, but long-term safety and efficacy of pioglitazone in nondiabetic patients with NASH is not established.[8] Moreover, pioglitazone is associated with weight gain and is therefore not suitable for patients with NASH who are already overweight or obese. Metformin has no significant effect on liver histology and is not recommended as a specific treatment for NASH;[8] it may, however, be safely continued if indicated for type 2 diabetes, even if NASH-related cirrhosis is present. Continuing metformin use in cirrhosis patients with diabetes is associated with a significant survival benefit over those who cease metformin.[12] Furthermore, metformin has been linked to reduced incidence of HCC in patients with diabetes and NAFLD. Vitamin E (α-tocopherol) administered at a daily dose of 800 IU/day improves liver histology in adults with biopsy-proven NASH who do not have diabetes, and therefore should be considered a first-line pharmacotherapy for this patient population. However, vitamin E is not recommended for the treatment of NASH in patients with diabetes, NAFLD without liver biopsy, NASH cirrhosis or cryptogenic cirrhosis.[8]

Bariatric surgery is not contraindicated in patients with NAFLD who require the surgery for other indications, unless cirrhosis is already established. Even in the presence of cirrhosis, bariatric surgery can be considered on a case-by-case basis. There is currently insufficient evidence to recommend bariatric surgery as a specific treatment for NASH alone.[8]

2. NAFLD AND ALCOHOLIC LIVER DISEASE: AN APPROACH TO PATIENT MANAGEMENT



Abbreviations: AFP = alpha-fetoprotein; BMI = body mass index; CT = computed tomography; HAV = hepatitis A virus; HBV = hepatitis B virus; HCC = hepatocellular carcinoma; MELD = Model for End Stage Liver Disease; NAFLD = nonalcoholic fatty liver disease.

Management of cirrhosis

In patients who have already developed cirrhosis (of any cause), management includes both managing the specific underlying disease and screening for and preventing the complications of cirrhosis (see Flowchart 2). Nutritional advice in patients

with cirrhosis is focused on maintaining muscle mass through having a high-protein diet (1.2 to 1.5 g/kg/day), including a late evening snack, which may stave off muscle loss and improve quality of life.[13]

In patients with decompensated cirrhosis, specialist management may occur in either the outpatient or inpatient setting depending on the severity of the liver disease. Management of ascites (salt and/or fluid restriction, diuretics, therapeutic paracentesis, transjugular intrahepatic portosystemic shunting [TIPSS]), prevention and treatment of spontaneous bacterial peritonitis (antibiotics), hepatic encephalopathy (lactulose, rifaximin), variceal bleeding (octreotide, terlipressin, endoscopic management) and appropriate referral for liver transplantation are all important considerations.

Prognostic scoring with the Model for End Stage Liver Disease (MELD) and Child–Pugh scores can be used to monitor decline or improvement in overall liver function in patients with cirrhosis.

The MELD score (requiring creatinine, INR and bilirubin) is a prognostic score for patients with cirrhosis; higher scores predict higher 90-day mortality and help guide management. The MELD score can be calculated at www.mdcalc.com/meld-score-original-pre-2016-model-end-stage-liver-disease/.

The Child–Pugh score (requiring bilirubin, albumin, prothrombin time [PT], ascites status and encephalopathy status) is another useful prognostic scoring system, with patients being stratified from Child–Pugh class A (compensated cirrhosis) to class C (more severe, decompensated cirrhosis). The Child–Pugh score can be calculated at www.hsls.pitt.edu/medcalc/ChildTurPuScore_SI.htm.

Management of hepatocellular carcinoma

HCC that is diagnosed in earlier (asymptomatic) stages is potentially curable. Hence the importance of identifying patients with chronic liver disease in the primary care setting and the performing of liver ultrasound to screen for HCC as part of their evaluation.

The detailed management of HCC accessed through specialist services is beyond the scope of this article. Management ranges from potentially curative therapies (liver transplant, liver resection, percutaneous ablation) to treatments that improve survival (transarterial chemoembolisation, selective internal radioembolisation, oral sorafenib). Patients with advanced HCC in the setting of liver failure should receive symptomatic/supportive therapies only, in conjunction with palliative care services.[14,15]

Alcoholic liver disease

Case study 2

Mr JW is a 54-year old businessman who presents to his GP feeling nauseous and with a swollen abdomen. He has a history of hypertension for which he takes amlodipine. He admits that he used to drink at least one bottle of wine a day for many years, but says he has quit now and not had an alcoholic drink for over a week.

Physical examination revealed scleral icterus. He has reduced muscle mass in his upper arms and legs. There is no hepatic flap (asterixis). The abdominal examination reveals a tense, nontender distended abdomen with shifting dullness. The liver and spleen are impalpable. There is no pedal oedema.

Blood tests show bilirubin 88 µmol/L (NR <20 µmol/L), GGT 460 U/L (NR <60 U/L), AST 85 U/L (NR <35 U/L), ALT 40 U/L (NR <35 U/L) and albumin 34 g/L (NR >35 g/L). The platelet count is reduced at 124 x 10^9/L (NR 150 to 400 x 10^9/L) and the INR is 1.1. The sodium level is 134 mmol/L (NR >135 mmol/L) and the creatinine level is 80 µmol/L (NR <110 µmol/L). Liver ultrasound confirms a nodular liver with associated mild splenomegaly; the bile ducts are not dilated. There is a large volume of ascites seen on the ultrasound.

Mr JW is prescribed thiamine 100 mg three times daily and spironolactone 50 mg daily, and referred to the liver clinic for prompt assessment.

Alcoholic liver disease is a common cause of chronic liver disease in Australia and the most common cause for hospitalisation and mortality from advanced liver disease.[1] Alcohol is a common cause of abnormal liver function in the community but reliable prevalence figures for alcoholic disease in the community are not available. The true prevalence of alcohol-related chronic liver disease is likely underestimated because the disease is largely asymptomatic in the early stages, before complications of cirrhosis have occurred. Furthermore, alcohol is a co-factor recognised to accelerate the progression of other chronic liver diseases to cirrhosis and HCC (e.g. viral hepatitis B and C) and to steatohepatitis related to metabolic syndrome. About 20% of patients with a history of alcohol abuse have a secondary or coexisting aetiology for liver disease.[16]

The NHMRC recommends in their guidelines for reducing health risks from drinking alcohol that, for healthy men and women, drinking no more than two standard drinks on any day reduces the lifetime risk of harm from alcohol-related disease or injury.[11] Consuming higher levels of alcohol may lead to a spectrum of alcohol-related liver disease.[17]

Presentation of alcoholic liver disease

The several ways patients with alcoholic liver disease may present are summarised in Flowchart 1. Alcoholic liver disease is a spectrum from asymptomatic alcoholic steatosis (fatty liver with normal LFTs) through alcoholic steatohepatitis (fatty liver with elevated LFTs and/or hepatic fibrosis) and alcoholic cirrhosis to acute alcoholic hepatitis.

Alcoholic steatosis, alcoholic steatohepatitis and alcoholic cirrhosis

Alcoholic steatosis is usually asymptomatic and develops in about 90% of individuals who drink more than 60 g/day of alcohol

but may also occur in individuals who drink less. This steatosis may be reversible within weeks of cessation of alcohol intake. Continued alcohol intake of more than 40 g a day is associated with a risk of progression of alcoholic steatosis to hepatic fibrosis (steatohepatitis) or cirrhosis of 37%.[18]

Acute alcoholic hepatitis

Acute alcoholic hepatitis has a poor short-term prognosis. Typically symptomatic, patients present with advanced liver disease (cirrhosis in over 50%), and superimposed acute hepatic decompensation with jaundice, coagulopathy and renal impairment. Severe alcoholic hepatitis is associated with a high mortality – up to 40% within six months of presentation. Clinical factors that may help distinguish acute alcoholic hepatitis from decompensated alcoholic cirrhosis (suggestive but not entirely diagnostic) include a younger age, a recent history of binge drinking, tender hepatomegaly and low-grade fever and/or neutrophilia without evidence of a septic source.[19]

Management of alcoholic liver disease in the primary care setting

The management of alcoholic liver disease in the primary care setting includes counselling of alcohol abstinence and management of any alcohol withdrawal. Alcohol withdrawal may occur with mild symptoms of tremor and agitation, but in about 5% of cases may be severe and lead to delirium and seizures.[20] Alcohol withdrawal symptoms are most likely to occur between 24 and 72 hours after the last drink and usually abate by five to seven days after the last drink. Management of alcohol withdrawal is recommended according to current guidelines and, where appropriate, in conjunction with local specialist drug and alcohol services.[20]

All patients being managed for alcoholic liver disease are at risk of Wernicke's encephalopathy (with potential sequelae of seizures and coma) and should therefore receive thiamine 100 mg intramuscularly (if not contraindicated by coagulopathy) and 100 to 300 mg daily orally thereafter.[20] Supporting nutrition with a diet high in both protein and energy and management of any co-factors (e.g. high BMI) is also recommended.

Noninvasive tests used in evaluation of alcoholic liver disease

As for patients with NAFLD, noninvasive tests can help identify patients who have more advanced alcoholic liver disease or poorer prognosis and who may require referral to specialist liver services (see Flowcharts 1 and 2).

Liver stiffness measurement by FibroScan may be useful in patients with suspected alcoholic liver disease, particularly to exclude the presence of significant hepatic fibrosis. Liver stiffness measurements are generally not indicated when liver failure or overt signs of cirrhosis are already present.

The MELD score and the Child–Pugh score may also be used for prognostic purposes in alcoholic cirrhosis, as used in NAFLD.

When to refer to liver specialist services

Patients with alcoholic steatohepatitis should be referred to a liver specialist for assessment, further investigation and management. This management may ultimately be maintained in the primary care setting, focusing on alcohol abstinence, nutritional advice and management of any co-factors, as previously described for NAFLD.

All patients with suspected alcoholic cirrhosis should be referred to specialist services, and prompt specialist evaluation is recommended for patients with decompensated cirrhosis.

Patients with suspected acute alcoholic hepatitis with signs of liver failure (jaundice, ascites, hepatic encephalopathy) should be referred promptly to a liver specialist centre and may require hospitalisation.

Conclusion

Chronic liver diseases such as NAFLD and alcoholic liver disease are a challenge seen both in primary practice and in specialist settings. As the burden of these liver diseases and also of chronic hepatitis B and C increases in Australia, identification of those at risk needs to occur to prevent the morbidity and mortality associated with chronic liver disease. Collaboration between community GPs and specialist services is essential in managing these patients in the long term – that is, we 'unite and conquer'. MT

References

A list of references is included in the website version of this article (www.medicinetoday.com.au).

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