Hypertriglyceridaemia: A practical approach for primary care

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Background
Hypertriglyceridaemia is commonly encountered in clinical practice and is associated with an increased risk of cardiovascular disease and acute pancreatitis. General practitioners play a central role in treating patients with hypertriglyceridaemia, ultimately with the aim of preventing associated complications.

Objective
The aim of this paper is to provide a contemporary approach to the management of patients with hypertriglyceridaemia in Australian primary care.

Discussion
Hypertriglyceridaemia is often attributable to secondary causes, which should be identified and addressed. Healthy lifestyle modifications targeting diet, exercise, alcohol consumption and weight are fundamental. Statins should be prescribed according to cardiovascular risk assessment and can reduce triglyceride levels. Icosapent ethyl is subsidised on the Pharmaceutical Benefits Scheme and is recommended to reduce cardiovascular risk in statin-treated patients with cardiovascular disease and mild-to-moderate fasting hypertriglyceridaemia (1.7–5.6 mmol/L). In patients with severe hypertriglyceridaemia (>5.6 mmol/L), intensive triglyceride-lowering with lifestyle modifications and pharmacotherapy is recommended to reduce pancreatitis risk. Specialist referral should be considered for severe cases of hypertriglyceridaemia or when primary genetic causes are suspected.

TRIGLYCERIDE-RICH LIPOPROTEINS (TRLs) are strongly associated with atherosclerotic cardiovascular disease (CVD; very low-density lipoproteins and their remnants, and chylomicron remnants) and acute pancreatitis (chylomicrons).[1] Plasma triglyceride levels are a surrogate biomarker of TRLs, with an elevated triglyceride level (or hypertriglyceridaemia) reflecting an increased level of TRLs.[1] The metabolism of TRLs is complex and hypertriglyceridaemia can be caused by primary genetic factors, secondary causes, or both.[1] Hypertriglyceridaemia is commonly encountered in primary care, with a prevalence of ~20–30% in adults.[2,3] The growing burden of metabolic diseases such as diabetes and obesity is likely to increase the prevalence of hypertriglyceridaemia.[4] General practitioners (GPs) play a central role in treating patients with hypertriglyceridaemia, ultimately with the aim of preventing associated complications.

In light of evolving evidence and guidelines, this article aims to provide a contemporary and practical approach to the management of patients with hypertriglyceridaemia in Australian primary care.

Classification

Plasma triglyceride levels can be measured in the fasting or non-fasting state, as both are correlated with the risk of CVD.[5] A non-fasting measurement might be more convenient for patients and non-fasting triglyceride levels might better predict the risk of CVD compared with fasting levels.[1] However, triglyceride levels can increase following consumption of a fatty meal and patients with non-fasting triglyceride levels ≥4.5 mmol/L should have a repeat measurement in the fasting state. Fasting measurements are also recommended for monitoring response to therapies.[6] Laboratories usually calculate low-density lipoprotein (LDL)-cholesterol using the Friedewald equation, which cannot be used when triglyceride levels are ≥4.5 mmol/L.[7] Direct measurement of LDL-cholesterol might need to be requested in such cases.[7] Hypertriglyceridaemia can be classified according to plasma triglyceride levels, although classifications differ among guidelines.[1,6,8–10]

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Table 1. Secondary causes of hypertriglyceridaemia

Category	Causes
Diseases	• Suboptimal glycaemic control in diabetes • Metabolic syndrome and insulin resistance • Overweight and obesity • Chronic kidney disease and nephrotic syndrome • Acute and chronic liver diseases • Hypothyroidism • Auto-immune diseases • Cushing syndrome • Familial partial lipodystrophy • Glycogen storage diseases • Multiple myeloma • HIV • Sepsis
Diet/lifestyle factors	• Alcohol use • Diets high in carbohydrates or saturated fats • Sedentary lifestyle • Smoking
Drugs	• Glucocorticoids • Anabolic steroids • Oral oestrogens • Beta-blockers • Thiazide and loop diuretics • Atypical antipsychotic agents • Isotretinoin • Tacrolimus/sirolimus • HIV protease inhibitors • Cyclophosphamide • Propofol • Bile acid sequestrants
Other	• Pregnancy

Adapted from Virani SS, Morris PB, Agarwala A, et al. 2021 ACC Expert consensus decision pathway on the management of ASCVD risk reduction in patients with persistent hypertriglyceridemia: A report of the American College of Cardiology Solution Set Oversight Committee. J Am Coll Cardiol 2021;78(9):960–93. doi: 10.1016/j.jacc.2021.06.011, with permission from Elsevier.[8]

Mild-to-moderate hypertriglyceridaemia has been defined as a fasting triglyceride level of 1.7–5.6 mmol/L (or a non-fasting level of 2.0–5.6 mmol/L).[6,8] Severe hypertriglyceridaemia has been defined as a fasting or non-fasting triglyceride level of ≥5.6 mmol/L.[6,8] Recent guidelines have also included the 'optimal' fasting triglyceride level as being <1.2 mmol/L.[1]

These classifications are helpful in practical terms, as the main therapeutic goal for patients with mild-to-moderate hypertriglyceridaemia is to address the increased risk of CVD, whereas in severe hypertriglyceridaemia cases, the priority shifts to mitigating the increased risk of acute pancreatitis, which is a potentially fatal complication.[11]

Identifying causes

The cause of hypertriglyceridaemia is often multifactorial. Secondary causes frequently contribute to, or cause, hypertriglyceridaemia (Table 1).[6,8] This makes a comprehensive history, examination and laboratory panel essential for their identification. Common secondary causes of hypertriglyceridaemia include suboptimal glycaemic control in diabetes, insulin resistance, central adiposity, a diet high in simple carbohydrates or saturated fats, excessive alcohol consumption and certain drugs. Addressing secondary causes of hypertriglyceridaemia, especially weight and glycaemic control, can beneficially modify plasma lipid levels and significantly reduce triglyceride levels.[6,8]

Genetic predisposition to hypertriglyceridaemia can increase the likelihood and severity of hypertriglyceridaemia. The majority of patients with hypertriglyceridaemia have a polygenic predisposition, resulting from the combined effect of multiple genetic variants, rather than a monogenic (single gene) cause.[12,13] Thus, routine genetic testing is not recommended. Patients with chylomicronaemia syndrome, for example, have very high levels of triglycerides along with clinical features such as lipaemia retinalis, eruptive xanthomas, acute pancreatitis and plasma that appears 'turbid' or 'milky' (ie lipaemic).[14] Even so, chylomicronaemia syndrome is 40- to 60-fold more likely to be caused by polygenic and secondary causes rather than monogenic causes such as autosomal recessive familial chylomicronaemia syndrome or familial partial lipodystrophy.[14] Monogenic causes of hypertriglyceridaemia are rare, but should be suspected when 'extremely' elevated triglyceride levels (eg >10 mmol/L) are detected with clinical features of hypertriglyceridaemia during childhood or adolescence.[13] A history of severe hypertriglyceridaemia in a sibling also suggests a strong genetic basis. Specialist referral should be considered in cases of severe hypertriglyceridaemia or when a primary genetic cause is suspected, particularly in the absence of identifiable secondary causes.

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Non-pharmacological management

Heart-healthy lifestyle modifications are the first-line therapy in the management of patients with hypertriglyceridaemia (Table 2), and includes smoking cessation.[8,15,16] Dietary modifications should focus on healthy eating patterns, with a reduction in calories, saturated fats, refined sugars and simple carbohydrates.[6,8] Increased omega-3 fatty acid consumption (eg fatty fish) is also recommended.[8] In patients with severe hypertriglyceridaemia, a low-fat diet with restriction of added sugars should be implemented.[8] A very-low fat diet is recommended if triglyceride levels are 'extremely' elevated (>10 mmol/L).[1,8] Referral to a dietitian can facilitate personalised education and nutrition recommendations that are likely to be more sustainable.[8] Physical activity can reduce triglyceride levels by up to ~30%, with varying response depending on the type, duration and intensity of exercise.[8,17] Guidelines recommend moderate-intensity exercise for ≥150 minutes/week or vigorous-intensity exercise for ≥75 minutes/week.[18] Implementing care plans that incorporate weight management is important, as weight loss is one of the most effective interventions for reducing triglyceride levels.[8,17] Weight loss through the combination of reducing calories, dietary modifications and exercise might reduce triglyceride levels by ~50%.[17] Patients with mild-to-moderate hypertriglyceridaemia should restrict alcohol consumption, whereas patients with severe hypertriglyceridaemia should avoid alcohol completely.[6] Shared decision making with patient-centred care plans and counselling can facilitate the implementation of, and adherence to, lifestyle modifications and therapies.[19]

Table 2. Lifestyle modifications to reduce triglyceride levels in patients with mild-to-moderate hypertriglyceridaemia[A]

Food group	Recommendation
Calories	Meet daily needs with minimally processed nutrient-dense foods; reduce caloric intake for weight loss if appropriate
Alcohol	Restrict; if choosing to drink, then consume ≤2 drinks/day for men and ≤1 drink/day for women
Sugar-sweetened beverages	Restrict
Fruits[B]	Mostly whole fruits; 2 servings/day
Vegetables[B]	2.5 cups/day
Grains[B]	Mostly fibre-rich wholegrains; 4–6 servings/day unless a lower-carbohydrate diet is indicated
Dairy[B]	Limit full-fat and sugar-sweetened dairy products; choose low-fat or fat-free unsweetened dairy or unsweetened dairy alternatives; 3 serves/day
Legumes, nuts and seeds	Mostly plant protein sources
Fish and seafood	Fatty fish; ≥2 servings/week
Poultry/meats	Choose lean cuts and avoid processed meats
Exercise	At least 150 mins/week of moderate-intensity exercise or 75 mins/week of vigorous-intensity exercise

[A]All recommendations for lifestyle modifications should be personalised. Patients with severe hypertriglyceridaemia ideally should be referred to a registered dietitian for a specialised nutrition plan.

[B]Serves/day based on 2000 calories/day.

Adapted from Virani SS, Morris PB, Agarwala A, et al. 2021 ACC Expert consensus decision pathway on the management of ASCVD risk reduction in patients with persistent hypertriglyceridemia: A report of the American College of Cardiology Solution Set Oversight Committee. J Am Coll Cardiol 2021;78(9):960–93. doi: 10.1016/j.jacc.2021.06.011, with permission from Elsevier.[8]

Pharmacological management

A comprehensive CVD risk assessment should be performed, as hypertriglyceridaemia often occurs with other risk factors such as diabetes, hypertension and obesity.[20] The Australian CVD risk calculator for primary prevention can facilitate risk assessment and guide the prescribing of statins and other preventive therapies.[21] Statins are the cornerstone pharmacotherapy to reduce the risk of CVD in patients at high risk or who have an elevated LDL cholesterol.[9,10] Statins can reduce triglyceride levels by 10–30% and are indicated as first-line management of hypertriglyceridaemia for the purpose of reducing the risk of CVD.[6,8] Notably, trials of statin therapy generally excluded patients with triglyceride levels >5 mmol/L.[22] Ezetimibe and therapies targeting proprotein convertase subtilisin/kexin type 9 (PCSK9), such as evolocumab or inclisiran, are also available in Australia to lower LDL cholesterol levels. Poor glycaemic control can increase triglyceride levels, underscoring the importance of the diabetes care plan and optimising diabetes management. In patients with obesity, pharmacotherapy such as incretin-based therapies (eg glucagon-like peptide-1 agonists) can significantly reduce triglyceride levels by addressing excess body weight and its metabolic consequences.[23] An Australian Obesity Management Algorithm for primary care has been published.[24]

Currently available non-statin pharmacotherapies that reduce triglyceride levels are summarised in Table 3. Fibrates can reduce triglyceride levels by 25–50%;[25] however, in statin-treated patients with mild-to-moderate hypertriglyceridaemia, fibrates have not been shown to reduce the risk of CVD events.[26,27] Fibrates are therefore not recommended in patients with mild-to-moderate hypertriglyceridaemia to reduce the risk of CVD.[6,8] Conversely, in patients with severe hypertriglyceridaemia, fibrates are recommended to reduce triglyceride levels and reduce the risk of acute pancreatitis.[6,8] Fibrates might also have microvascular benefits in patients with diabetes, with studies showing that fenofibrate can slow the progression of diabetes-related retinopathy irrespective of lipid levels.[28,29] If used with a statin, fenofibrate is preferred over gemfibrozil,

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Table 3. Non-statin agents currently available for reducing triglyceride levels[A]

Drug class or therapy	Triglyceride reduction (%)	Clinical use	Main adverse effects	Contraindications
Fibrates (eg fenofibrate or gemfibrozil)	25–50	Consider in patients with severe hypertriglyceridaemia to reduce triglyceride levels and the risk of acute pancreatitis	Gastrointestinal symptoms, myopathy, cholelithiasis, venous thromboembolism, increased creatinine, liver enzyme elevation	Hepatic impairment, gallbladder disease, severe renal impairment (CrCl <30 mL/minute), hypersensitivity
Combined EPA and DHA omega-3 fatty acid formulations	20–50	*Prescription* products might be considered in patients with severe hypertriglyceridaemia to reduce triglyceride levels and the risk of acute pancreatitis	Atrial fibrillation/flutter, gastrointestinal symptoms, increased risk of bleeding	Use with caution in patients allergic to seafood or who have a hypersensitivity
Icosapent ethyl (purified EPA ethyl ester)	20	Consider in high-risk, statin-treated patients with mild-to-moderate hypertriglyceridaemia (refer to Box 1)	As per row above	As per row above
Niacin (nicotinic acid)	20–50	Not routinely recommended by guidelines	Flushing, gastrointestinal symptoms, liver enzyme elevation, hyperglycaemia, hyperuricaemia	Symptomatic hypotension, hepatic impairment, recent myocardial infarction, hypersensitivity

[A]Most of these agents are not approved for use in paediatric populations and are contraindicated during pregnancy. The safety of high-dose omega-3 fatty acids during pregnancy has also not been established.

CrCl, creatinine clearance; DHA, docosahexaenoic acid; EPA, eicosapentaenoic acid.

as the latter can increase the risk of statin myotoxicity.[30] Omega-3 fatty acid supplementation with preparations containing various proportions of docosahexaenoic acid (DHA) and eicosapentaenoic acid (EPA) can reduce triglyceride levels by 20–50%, but have not been shown to reduce the risk of CVD events.[6,8,31–33]

Conversely, icosapent ethyl, a highly purified EPA, significantly reduced the risk of CVD events and CVD mortality in a trial when prescribed at 2 g twice a day.[34] Icosapent ethyl is recommended by several international guidelines and has received Therapeutic Goods Administration approval.[6,8,9] Icosapent ethyl is subsidised on the Pharmaceutical Benefits Scheme for patients with CVD on statin treatment (unless not tolerated or contraindicated), an LDL cholesterol level of 1.0–2.6 mmol/L and a fasting triglyceride level of 1.7–5.6 mmol/L (Box 1). The increased risk of atrial fibrillation and bleeding with icosapent ethyl requires consideration but is usually outweighed by its CVD prevention benefits.[34] It must be noted that the CVD benefits obtained with icosapent ethyl use were similar across baseline and on-treatment triglyceride levels, suggesting that mechanisms beyond triglyceride-lowering (eg anti-inflammatory, antithrombotic or other effects) are mediating its benefits.[34,35] Indeed, there is limited evidence from randomised trials of triglyceride-lowering agents that lowering triglyceride levels reduces the risk of CVD.[1]

Although niacin can reduce triglyceride levels by 20–50%, it is not recommended because of unacceptable adverse effects, in particular flushing.[6,8,36,37]

An example management algorithm is presented in Figure 1.[6] International guidelines recommend treatment goals for LDL cholesterol but do not specify treatment goals for triglyceride levels.[9] Apolipoprotein B and non-high-density lipoprotein cholesterol are alternate measures that incorporate TRLs and which have treatment goals;[9] however, apolipoprotein B testing is not rebated by the Medicare Benefits Scheme.

Emerging therapies

Novel therapies that target the triglyceride metabolism pathway, such as inhibitors of apolipoprotein C3 (APOC3) or angiopoietin-like protein 3 (ANGPTL3), are currently in advanced phases of clinical development.[38] Targeted delivery of ribonucleic acid (RNA)-based therapies against APOC3 and ANGPTL3 have demonstrated significant reduction in triglyceride levels.[38] These therapies can significantly reduce the risk of acute pancreatitis in patients with severe hypertriglyceridaemia, including patients with chylomicronaemia syndrome.[39] The long duration of action of RNA-based therapies, especially small interfering RNAs, has resulted in less frequent dosing that might improve adherence. Also, fibroblast growth factor-21 (FGF21) is a stress hormone that regulates lipid and glucose metabolism; FGF21 analogues are in clinical trials, with data showing promise in reducing triglyceride levels and improving hepatic steatosis.[38] Although a number of these therapies are progressing through rapid regulatory review in the US for recurrent pancreatitis, their ability to impact CVD outcomes in patients with mild-to-moderate hypertriglyceridaemia, and even in patients with severe hypertriglyceridaemia, remains to be determined.[38,40]

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Plain language summary

Hypertriglyceridaemia is a common lipid disorder that is associated with an increased risk of cardiovascular disease and acute pancreatitis. GPs play a central role in its management by identifying and addressing underlying causes such as obesity, suboptimal diabetes control, excessive alcohol intake and lifestyle factors. Lifestyle changes, including a healthy diet, regular exercise and weight loss, are the first steps in management. For patients with persistently high triglyceride levels or for those at high risk of complications, medications such as statins, icosapent ethyl and fibrates should be considered. Effective management requires a holistic person-centred approach, including improving glycaemic control in diabetes and attending to other cardiovascular risk factors such as excess body weight and high blood pressure. Regular follow-up and patient education are essential to achieving long-term benefits.

Conclusion

Hypertriglyceridaemia is a common issue that GPs encounter and often occurs with other risk factors for CVD. A structured approach to the assessment and management of patients with hypertriglyceridaemia, including addressing lifestyle factors and associated comorbidities, is essential to prevent CVD and acute pancreatitis. GPs are ideally placed to implement personalised approaches, holistic care plans and regular follow-up to improve outcomes for these patients.

Key points

- Hypertriglyceridaemia is associated with cardiovascular and acute pancreatitis risk.

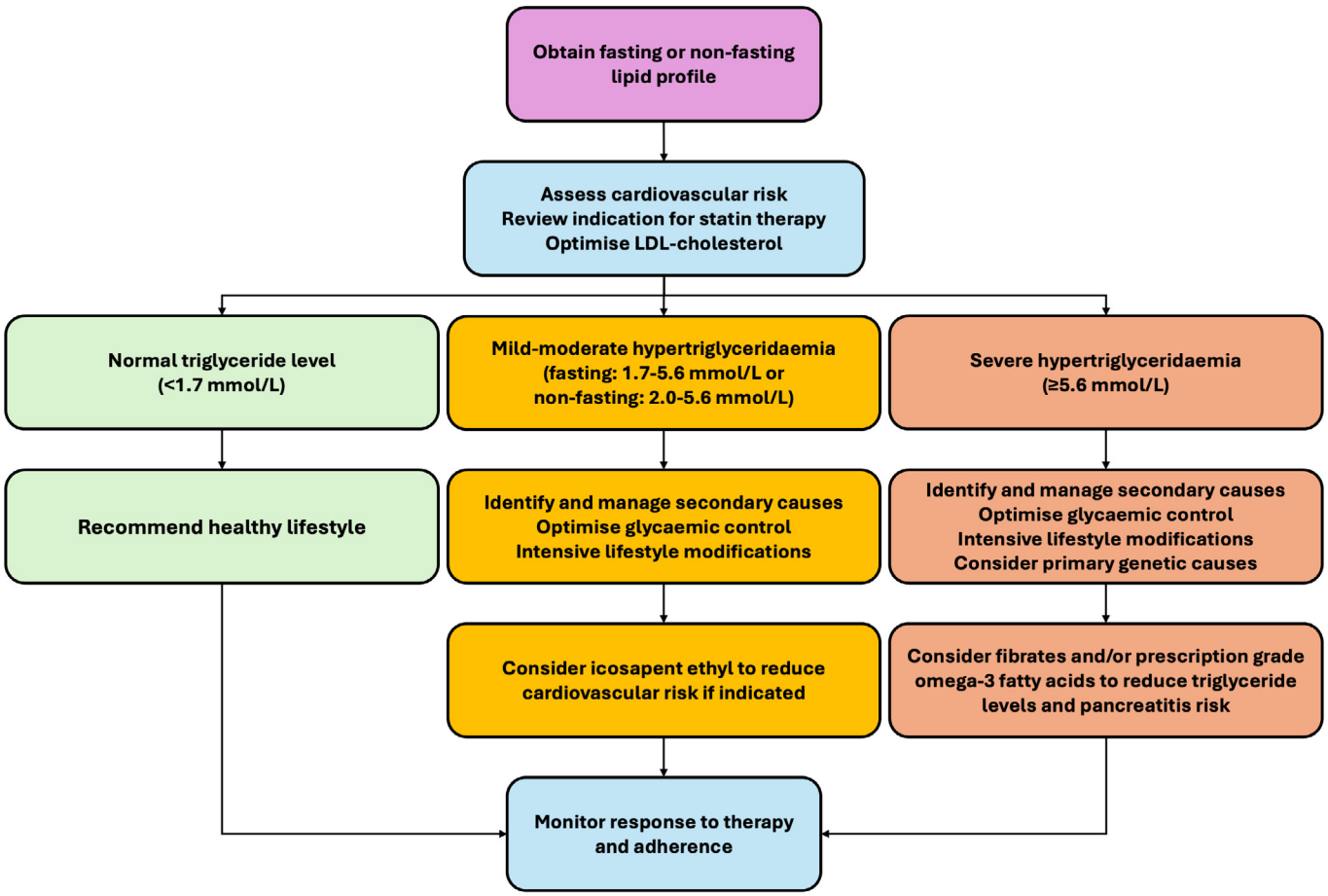

Figure 1. Example of a management algorithm.

Adapted from Oh RC, Trivette ET, Westerfield KL. Management of hypertriglyceridemia: Common questions and answers. Am Fam Physician 2020;102(6):347–54. Available at www.aafp.org/pubs/afp/issues/2020/0915/p347.html, with permission from the American Academy of Family Physicians.[41]

LDL, low-density lipoprotein.

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Box 1. Approved indication and current subsidy criteria for prescribing icosapent ethyl[A]

Indication approved by the TGA:

- To reduce the risk of CV events in adult statin-treated patients at high CV risk with elevated triglycerides (≥1.7 mmol/L) and established CV disease, or diabetes and at least one other CV risk factor

Criteria for PBS-subsidised prescription:

- The treatment must be in conjunction with dietary therapy and exercise

AND

- Patients must have at least one of: (i) coronary artery disease; (ii) cerebrovascular disease or carotid disease; and (iii) peripheral arterial disease

AND

- Patients must be treated with a stable dose of a 3-hydroxy-3-methylglutaryl coenzyme A (HMG CoA) reductase inhibitor (statin) to achieve target secondary prevention LDL-cholesterol levels for at least 12 consecutive weeks; *OR*
- Patients must have developed clinically important product-related adverse events necessitating withdrawal of statin treatment; *OR*
- Patients must be contraindicated to treatment with a HMG CoA reductase inhibitor (statin) as defined in the TGA-approved product information

AND

- Patients must have LDL-cholesterol level between 1.0 mmol/L and 2.6 mmol/L; *OR*
- Patient must have a non-HDL-cholesterol between 1.5 mmol/L and 3.5 mmol/L if LDL-cholesterol cannot be measured/detected

AND

- Patient must have a fasting triglyceride level between 1.7 mmol/L and 5.6 mmol/L

[A]Daily oral dose of four capsules taken as 2 × 998 mg capsules twice daily; refer to www.tga.gov.au and www.pbs.gov.au for updated information.

CV, cardiovascular; HDL, high-density lipoprotein; LDL, low-density lipoprotein; PBS, Pharmaceutical Benefits Scheme; TGA, Therapeutic Goods Administration.

- Addressing secondary causes and implementing lifestyle changes remain fundamental.
- Statins are the cornerstone drug therapy to reduce cardiovascular risk in the high-risk patient.
- In patients with hypertriglyceridaemia and high cardiovascular risk, icosapent ethyl can be prescribed to lower cardiovascular risk.
- Consider specialist referral in cases of severe hypertriglyceridaemia or for patients with a suspected primary genetic cause.

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