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Clinical approach to dyslipidaemia

High cholesterol is a common general practice problem. This article guides the GP on its management.

Patients with 'high cholesterol' are a common occurrence in general practice. They often present with a high cholesterol level determined at a pharmacy, at work or for insurance purposes. Irrespective of where and how the test was done, the GP is usually left with the responsibility of interpreting the result and deciding on further management. Getting the initial assessment and management right can profoundly influence the patient's future health, both physically and financially! Depending on the clinical context, a lipid value that may be of little concern in one patient will clearly indicate therapy with lipid-lowering drugs in another patient. Getting it wrong means that some patients are denied access to therapy that may significantly reduce mortality, while others take expensive and unnecessary therapy for many years. Failure to recognise the genetic nature of a dyslipidaemia results in a missed opportunity for family screening and prevention of premature ischaemic heart disease (IHD) in individuals at very high risk.

Failure to realise the genetic nature of a dyslipidaemia results in a missed opportunity for family screening and prevention of premature ischaemic heart disease (IHD) in individuals at very high risk.

Cardiovascular disease remains the leading cause of mortality. Given the long-term implications of any decision regarding risk management, it is worthwhile investing time in a thorough consultation and discussion with the patient. A clear understanding of the disease and the benefits of long-term therapy will often result in better compliance with lifestyle changes and medication.

EVALUATING THE DYSLIPIDAEMIC PATIENT

Dyslipidaemia is associated with two major complications. Atherosclerosis is most commonly associated with dyslipidaemia, but pancreatitis secondary to severe hypertriglyceridaemia is the most dangerous in the short term. A hypertriglyceridaemia of more than 10 - 15 mmol/l (or patients with lipaemic (white) serum) is a medical emergency. Such hypertriglyceridaemic patients must be evaluated immediately and should urgently be referred to a centre with lipid expertise. Delaying management or choosing the incorrect drug could result in pancreatitis and even death.

As atherosclerosis is the commonest complication of dyslipidaemia, this article will focus mainly on the evaluation of patients with pure hypercholesterolaemia or mixed hyperlipidaemias that are not associated with pancreatitis.

History

It is useful to define the purpose of a consultation. Does the patient want advice for an incidentally discovered hyperlipidaemia or was the dyslipidaemia found after an

ischaemic event? Does the patient have other cardiovascular risk factors or a family history of premature heart disease? Were lipids specifically screened? Did the patient present with pancreatitis or one of the physical signs of lipid disorders?

Taking a history proceeds along standard lines, but particular attention should be paid to the following points.

The dyslipidaemia

When was the dyslipidaemia first discovered and what was the range of lipid tests? If the patient is on lipid-lowering drugs, can a baseline untreated lipid profile be traced? Was the sample taken in the fasting state (important for triglyceride values, which fluctuate much more than cholesterol values)? Was the sample taken during an acute-phase response (> 24 h after a myocardial infarction (MI) or any severe stressor (e.g. surgery, severe influenza)) when cholesterol values will be falsely low? Are there other diseases or drugs that could be causing the hyperlipidaemia? (See article on secondary dyslipidaemia on p. 365 of this issue of *CME*.)

Complications of the dyslipidaemia

Is there a history of IHD, cerebrovascular disease, peripheral vascular disease or aortic aneurysm? Has the patient had pancreatitis or severe unexplained abdominal pain?

Additional risk factors for atherosclerosis

Is the patient diabetic or hypertensive? Specifically ask about smoking, both current and previous. Try to estimate the number of pack years ((cigarettes per day/20 × years)) smoked and ask whether the patient has considered or previously tried quitting. How did the patient try to quit and what was

the reason for failure?

Family history

Taking a good family history is particularly important in dyslipidaemic patients, especially in South Africa where there is a particularly high prevalence of severe monogenic disorders of lipid metabolism. The family history is best taken while sketching a family tree, to which as much information as possible is added. Ask about lipid values in family members or whether anybody is taking medication for ‘cholesterol problems’. In South Africa founder effects are seen for familial hypercholesterolaemia (FH). This means that in some population groups the prevalence of FH is extremely high, up to 1:70 compared with the average of 1:500 worldwide. In South Africa Afrikaners, Lithuanian Jews, Indians and Christian Lebanese are at particular risk of FH, so asking about the ancestry of your patient is important. As cardiovascular disease is so common it is important to ascertain whether it has occurred prematurely in the family. Generally an MI in men younger than 50 years or women under 55 years is considered to be premature cardiovascular disease. A family history of premature IHD may indicate a genetic disorder with greatly increased risk, while late-onset cardiovascular disease in family members does not have the same implication. The metabolic syndrome (and ultimately type II diabetes in some patients) also has a heritable component, and a family history of diabetes means a higher risk of diabetes for the patient, especially if he or she is overweight or has a sedentary lifestyle.

Box 1, together with Fig. 1, are examples of a family tree indicating autosomal-dominant inheritance and premature IHD in a family with familial hypercholesterolaemia. The family tree also illus-

trates the inheritance of the problem to the patients and helps to decide which relatives are at risk and should be screened.

Box 1. Family tree illustrating the inheritance of familial hypercholesterolaemia

The family tree (Fig. 1) was drawn from information provided by the patient (arrow) at his first visit. Squares represent males and circles represent females. A line through the symbol indicates that the person is deceased. Half-shading of symbols indicates that the individual has presumed FH (based on lipid values or premature heart disease). In this family the patient inherited FH from his father who had a fatal myocardial infarction (MI) at 45 years of age. His father’s sister also died of a premature MI. The mutated gene can be traced back even further to his paternal grandmother, as she also had premature IHD. The grandmother was of Afrikaner origin. Although one of the patient’s maternal uncles also died of an MI, his death was not premature. The patient did not know the lipid values of his two living older brothers and although they are well, they may still have inherited FH. The patient and his siblings have a total of 19 children. Two of these children have FH based on their lipid values, while FH has been excluded in 5 children by lipid testing. Each of the 5 children of the oldest brother has a 50% chance of being affected and needs to be screened. The 3 children of the youngest brother are unlikely to be affected, as their father’s cholesterol level is not raised. The risk for the remaining 4 children cannot be quantified and their father should be screened initially.

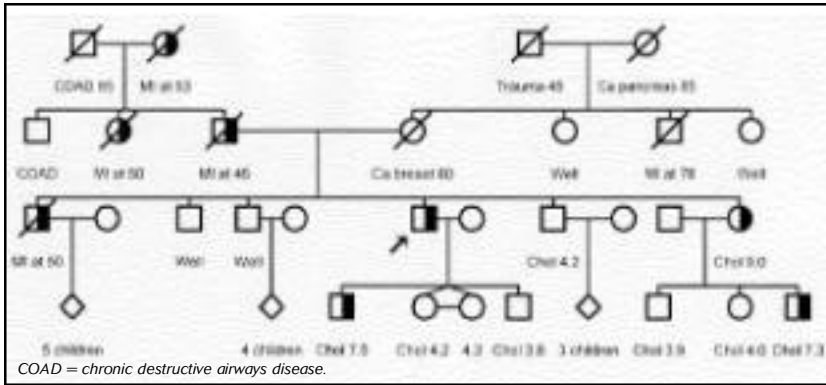


Fig. 1. Family tree illustrating the inheritance of familial hypercholesterolaemia.

Dietary history

The patients' diet can rapidly be assessed by simply asking them to describe what they eat on a typical day and how their food is prepared. Try to assess how much fat is being consumed overall and what the spread is between saturated, monounsaturated and polyunsaturated fat. Cholesterol intake is determined by the amount and type of animal products consumed. All muscle tissue (meat) contains about 75 mg cholesterol per 100 g. Egg yolks contain about 250 mg cholesterol and organ meat is higher in cholesterol. A brief dietary history is worth while even if the patient will be seeing a dietitian, as one can quickly judge whether a patient claiming to be on a 'low-cholesterol diet' has really changed the diet adequately. Knowing how 'bad' the diet was previously gives one a feel for the expected improvement in the lipid profile with dietary modification.

Exercise

Patients should be asked specifically about their exercise habits and level of physical activity. This is usually a good opportunity to explain the benefits of regular exercise on cardiovascular health.

Examination

Special attention needs to be given to the following points when examining the dyslipidaemic patient.

Clinical evidence of vascular disease

All peripheral pulses should be palpated and major vessels auscultated. Measure the blood pressure and examine the heart and fundi. Patients with clinically manifest vascular disease have a very high risk of further complications and most will require lipid-lowering therapy as part of secondary prevention.

Evidence for secondary dyslipidaemia

Look for evidence of liver, renal or thyroid disease. Abdominal obesity, acanthosis nigricans and excessive skin tags may be a marker of insulin resistance and the metabolic syndrome.

Physical signs of dyslipidaemia

The physical signs of dyslipidaemia are easily found if looked for. As a general rule cholesterol deposits in tendons (thickening, irregularity, xanthomata), while hypertriglyceridaemia causes skin infiltration (eruptive or tubo-eruptive xanthomata, palmar crease infiltration). In extreme hypercholesterolaemia (usually in patients with homozygous familial hypercholesterolaemia) both skin and tendon infiltration may be found. Severe genetic mixed hyperlipidaemias, such as dysbetalipoproteinaemia, may also affect both skin and tendons.

Finding the signs of hyperlipidaemia

Look for the following:

- **Xanthelasma** are not specific for any lipid disorder and many patients with xanthelasma will not be dyslipidaemic. However, they should be screened for dyslipidaemia if this has not already been done.
- The prevalence of **arcus cornealis** increases with age and is therefore of most diagnostic value if found in patients under the age of 40 years. Arcus cornealis in young patients generally indicates severe genetic hypercholesterolaemia or abnormal high-density lipoprotein (HDL) metabolism. The upper eyelid may often hide a superior arcus. Lift the eyelid and ask the patient to look down.
- **Eruptive xanthomata** (Fig. 2) are associated with severe hypertriglyceridaemia and urgent measures are required as the risk of pancreatitis is very high.



Fig. 2. Eruptive xanthomata. The patient initially presented to a dermatologist with a rash. He had newly diagnosed diabetes and his triglyceride level was 85 mmol/l. There is probably some (minor) genetic predisposition aggravated by the diabetes.

- **Palmar crease xanthomata** — yellow infiltrations of the palmar crease — are rare. They are diagnostic of dysbetalipoproteinaemia.
- **Tuberous xanthomata** are generally found in severe hypercholesterolaemia, while tuberuptive xanthomata are associated with



Fig. 3. Achilles xanthomata. This patient has the unusual combination of a tuberous xanthoma at the ankle and a xanthoma in the Achilles tendon.

mixed hyperlipidaemia. Fig. 3 shows a tuberous xanthoma at the ankle. Tuberous xanthomata are most commonly found at the elbows.

- **Tendon xanthomata** can be seen or felt as nodules that move with the tendon. They are firm and non-compressible, which helps to distinguish them from ganglia. The best places to look are the extensor tendons of the hands and the Achilles tendons. In Fig. 3 the nodule in the Achilles tendon is a xanthoma. The Achilles tendons are palpated to assess their dimensions (one's own tendons can be used for comparison if no genetic hyperlipidaemia is present) and to feel for any irregularities, roughness or lumps.
- In patients with severe hypertriglyceridaemia there may be two additional clues to the problem. **Lipaemic** (white) serum can be observed if the blood is left undisturbed for a while after venesection, but lipaemia may be observed directly on fundoscopy (**lipaemia retinalis**). Fig. 4 shows lipaemic serum after the blood has been centrifuged.

Laboratory tests

Initial screening for dyslipidaemia is often done with a random cholesterol measurement. If the cholesterol is elevated (> 5 mmol/l) it should be followed up by a fasting

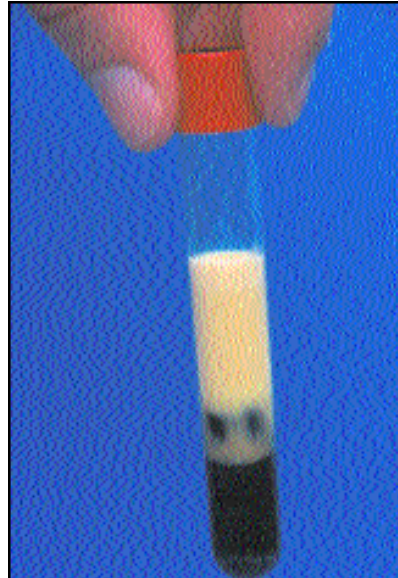


Fig. 4. Lipaemic serum is associated with severe hypertriglyceridaemia and carries a high risk of pancreatitis.

lipid profile. In the lipid profile total cholesterol, triglyceride and HDL cholesterol levels are measured, while the low-density lipoprotein (LDL) cholesterol level is usually calculated from the above values. Remember that if triglyceride levels are elevated the cholesterol level will also be high, because the lipoproteins carrying triglycerides also contain cholesterol. The commonest problem in patients with extreme hypercholesterolaemia (> 15 mmol/l) is severe hypertriglyceridaemia. Lipoprotein (a) measurement adds extra cost, and is most useful in patients with borderline indications for lipid-lowering drugs. A high Lp (a) would influence one to treat a patient with borderline risk. As lipoprotein (a) is not much influenced by lipid-lowering therapy (except niacin) it should not be measured repeatedly on follow-up.

Further useful laboratory investigations at the initial consultation include:

- **Urine dipstick** to screen for renal disease.
- **Renal function tests**. Abnormalities alert to renal dis-

ease as a cause of secondary dyslipidaemia, and with significant renal impairment dosages of lipid-modifying drugs, especially fibrates, may require adjustment.

- **Thyroid-stimulating hormone (TSH)**. Hypothyroidism is a common cause of secondary dyslipidaemia and is often clinically occult.
- **Fasting glucose**. The metabolic syndrome is a common cause of secondary dyslipidaemia and is characterised by insulin resistance, which may result in impaired glucose tolerance or even frank diabetes. Diabetes is associated with markedly increased cardiovascular risk.
- **Baseline liver function** tests. These tests screen for underlying hepatic disease, such as cholestasis, which may result in secondary dyslipidaemia. Liver function may also be a pointer to alcohol abuse or non-alcoholic steatohepatitis (NASH). Transaminase levels may increase on statin therapy, and it is important to have baseline values.
- **Creatinine kinase (CK)**. Measuring CK at baseline provides a useful reference point if patients should develop muscle problems when using statins. If the CK level is raised at baseline the patient should be evaluated for an underlying myopathy or metabolic disturbance, e.g. hypothyroidism or hypercalcaemia.

Deciding on the diagnosis

It is worthwhile to try making a more precise diagnosis than simply saying 'high cholesterol or high triglycerides'. If a specific diagnosis is made, deciding on therapy is easier and the risk for other family members can be better quantified. Initially consider the lipid phenotype. Is there predominant hypercholesterolaemia, hypertriglyceridaemia or is a mixed hyperlipidaemia present?

MAIN TOPIC

In patients with predominant hypercholesterolaemia consider the following categories:

- Most patients will have **polygenic hypercholesterolaemia**. Their total cholesterol is usually between 5 and 7.5 mmol/l and they do not have tendon xanthomata or a family history of severe hyperlipidaemia or premature heart disease.
- Patients with **monogenic disorders** of lipid metabolism (FH, familial binding defective apoB100 (FBD) and familial combined hyperlipidaemia (FCH)) usually have a total cholesterol level of more than 7.5 mmol/l. There is often a family history of hyperlipidaemia or premature heart disease. Patients with FH or FBD may have tendon xanthomata, which do not occur in FCH.

Severe hypertriglyceridaemia can be classified into:

- **Primary hypertriglyceridaemia.** This occurs in patients with genetic defects of lipoprotein metabolism. Primary hypertriglyceridaemia may be recessively inherited (the parents have normal lipid values). The risk is increased in consanguinous marriages and each child of the couple has a 25% chance of being affected. Hypertriglyceridaemia is present from birth and pancreatitis may occur in infancy. Other primary hypertriglyceridaemias may present in adulthood.
- **Secondary hypertriglyceridaemia.** This is much more common than primary hypertriglyceridaemia. Patients with secondary hypertriglyceridaemia have a limited capacity to metabolise triglyceride-rich lipoproteins, but can maintain normal lipid values in the absence of additional 'stresses' on the system. If triglyceride production is increased or metabolism further impaired, severe

hypertriglyceridaemia results. Examples of such 'second hits' are obesity, diabetes, alcohol abuse or drug therapy.

In patients with mixed hyperlipidaemia consider the following categories:

- **Genetic causes.** These include FCH and dysbetalipoproteinaemia. The inheritance of FCH is autosomal dominant and can cause varied lipoprotein phenotypes in affected family members. Dysbetalipoproteinaemia causes a severe mixed hyperlipidaemia and in most cases the inheritance is recessive.
- **Polygenic mixed hyperlipidaemia.** This results from the interaction of multiple ill-defined genetic and environmental factors such as diet and exercise.

Deciding on management

With all the necessary information at hand, deciding on future management is often not difficult. All patients should be counselled to stop smoking, exercise regularly and follow a diet low in saturated fat and cholesterol, emphasising unsaturated and omega-3 polyunsaturated fat. Medical conditions affecting cardiovascular risk, such as diabetes or hypertension, should be optimally managed. Secondary dyslipidaemia should be excluded and causative factors reversed (if possible) before considering lipid-lowering drugs.

Severe hypertriglyceridaemia (>10 mmol/l) requires urgent attention. Patients should be referred to or discussed with staff

Some common cholesterol myths busted

- *'I can't have a cholesterol problem because I am not fat.'*
Adipose tissue contains triglyceride. Although obesity is associated with an increased prevalence of dyslipidaemia it certainly is not a prerequisite! Patients with monogenic lipid disorders may be extremely thin and fit and still have markedly raised lipid levels.
- *'I feel well, therefore my cholesterol must be normal.'*
Hypercholesterolaemia is a "silent killer". There are generally no symptoms until vascular complications set in.
- *'Diet does not work and it is easier to take pills.'*
The lipid profile after dietary changes improves variably, depending on the previous diet and the individual's metabolic set-up. Even if the lipid levels do not decrease dramatically, changing the nature of fatty acids consumed has a beneficial effect on, for instance, platelet adhesiveness and cardiac arrhythmogenicity. Diet and lifestyle changes have proven effects on mortality and morbidity (with many dietary studies reporting better reductions in mortality than statin studies) and medication alone is therefore not 'the full deal'. Having an inherited hyperlipidaemia is also no reason to disregard the diet!
- *'All hypercholesterolaemia is genetic and therefore fate.'*
Only a small minority of hypercholesterolaemic patients have an identifiable genetic disorder. The majority are hypercholesterolaemic because they have a susceptible genetic background and are exposed to negative environmental influences. If the environment is corrected fate can be reversed!
- *'Avocados contain cholesterol.'*
For practical purposes no plant contains any cholesterol at all. Plants do contain sterol molecules, but these are different from cholesterol and not absorbed by humans. Advertising plant products (e.g. cooking oil) as cholesterol-free is therefore correct, but another product on the shelf will be just as cholesterol-free if it is of pure plant origin.

Table 1.

Section A: Men

Age (yrs)	30 - 34	35 - 39	40 - 44	45 - 49	50 - 54	55 - 59	60 - 64	65 - 69	70 - 74
Points	-1	0	1	2	3	4	5	6	7
TC (mmol/l)	< 4.1		4.2 - 5.2		5.3 - 6.2		6.3 - 7.2		>7.2
Points	-3		0		1		2		3
HDLC (mmol/l)	< 0.91		0.91 - 1.16		1.17 - 1.55				> 1.55
Points	2		1		0				-2
BP (mmHg)	< 130/< 85		130 - 139/85 - 89		140 - 159/90 - 99				160/ 100
Points	0		1		2				3
Other	Non-smoker		Smoker		Not diabetic				Diabetic
Points	0		2		0				2

Section B: Women

Age (yrs)	30 - 34	35 - 39	40 - 44	45 - 49	50 - 54	55 - 59	60 - 74
Points	-9	-4	0	3	6	7	8
TC (mmol/l)	< 4.1		4.2 - 5.2		5.3 - 7.2		>7.2
Points	-2		0		1		3
HDLC (mmol/l)	< 0.91		0.91 - 1.16		1.17 - 1.29		> 1.55
Points	5		2		1		-3
BP (mmHg)	< 120/< 80		120 - 139/80 - 89		140 - 159/90 - 99		160/ 100
Points	-3		0		2		3
Other	Non-smoker		Smoker		Not diabetic		Diabetic
Points	0		2		0		4

Section C: Risk (% of cohort who will have MI in next 10 years)

Points	-2	-1	0	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
Men	2	3	3	4	5	7	8	10	13	16	20	25	31	37	45	>53				
Women	1	2	2	2	3	3	4	4	5	6	7	8	10	11	13	15	18	20	24	>27

Section D: Risk for population (% who will have MI in 10 years)

Age (yrs)	30 - 34	35 - 39	40 - 44	45 - 49	50 - 54	55 - 59	60 - 64	65 - 69	70 - 74
Men	3	5	7	11	14	16	21	25	30
Women	< 1	< 1	2	5	8	12	12	13	14

TC = total cholesterol; HDLC = high-density lipoprotein cholesterol; BP = blood pressure; MI = myocardial infarction.

at a lipid clinic, or referred to a specialist with experience in dyslipidaemia. Patients with hypercholesterolaemia or mixed hyperlipidaemia (without severe hypertriglyceridaemia) are not at immediate risk and can be managed stepwise with initial lifestyle modification, followed by titration of lipid-lowering medication if indicated.

Patients with clinically overt vascular disease are at very high risk of further events and require 'secondary prevention'. If they do not achieve their LDL cholesterol target of less than 3 mmol/l on lifestyle modification alone, drug therapy is necessary. Following the results of the recent Heart

Protection Study many would argue that in the setting of secondary prevention statins should be used even in patients who do not have elevated lipid values by conventional definition.

When assessing patients without clinically overt atherosclerosis it is important to distinguish between patients with monogenic lipid disorders and those with polygenic/moderate hyperlipidaemia.

Monogenic lipid disorders increase cardiovascular risk dramatically and almost all patients will need therapy with lipid-lowering drugs. Patients with FH should have their partner's cholesterol checked before starting a family and be counselled about the risk of having

a child with homozygous FH. Once a patient has been diagnosed with a monogenic lipid disorder attempts should be made to screen other potentially affected family members. During family screening children with hyperlipidaemia are often identified. The decision at which age to start lipid-lowering therapy is often difficult and a consultation at a lipid clinic can be very helpful.

The majority of patients seen in a general practice will have polygenic hypercholesterolaemia. The indication for lipid-lowering therapy in these patients is based on their absolute risk of an MI during the next 10 years. For young patients the risk can be projected to 60

years of age. Absolute risk is calculated using risk calculation tables derived from data gathered during the Framingham study. Table I illustrates the currently recommended risk calculation in South Africa. This calculation can be done in less than a minute. The calculation incorporates age and sex as non-modifiable variables. The clinical variables are blood pressure, smoking status and diabetes. The laboratory variables are total cholesterol (used as a surrogate for LDL-cholesterol) and HDL-cholesterol levels. Lipid-lowering therapy is recommended if the absolute risk for MI is more than 20%, despite lifestyle modification.

With regard to Table I, to derive the absolute risk as a percentage of subjects who will develop MI over 10 years, add the points for each risk category. For men consult

section A and for women, section B. For the BP score, use the highest score of either diastolic or systolic pressure. The risk associated with the total points is derived from section C for men and women. The average population risk from which the data were derived is given in section D over various age intervals. The following risk factors are not included: obesity, family history, definite diagnosis of monogenic lipid disorder (to be considered in cases where cholesterol concentration is > 7.5 mmol/l) and sedentary lifestyle. These factors should be borne in mind when assessing global risk. Note that the score is gender dependent: men with 9 risk points have the same 20% risk as women with 15 risk points. To extrapolate the risk to age 60 years, add the difference between the age points for age 60 - 64 years and

those for the current age to the total score.

FURTHER READING

Expert Panel on Detection, Evaluation and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). Executive summary of the Third Report of the National Cholesterol Education Program (NCEP). *JAMA* 2001; **285**: 2486-2496.

Heart Protection Study Collaborative Group. MRC/BHF heart protection study of cholesterol lowering with simvastatin in 20 536 high-risk individuals: a randomised placebo-controlled trial. *Lancet* 2002; **360**: 7-22.

South African Medical Association and Lipid and Atherosclerosis society of Southern Africa Working Group. Diagnosis, management and prevention of the common dyslipidaemias in South Africa — Clinical Guideline. *S Afr Med J* 2000; **90** (2): 161-168.

IN A NUTSHELL

Dyslipidaemia is common in general practice and a major risk factor for atherosclerosis.

Severe hypertriglyceridaemia (> 10 mmol/l) can cause pancreatitis and may be associated with eruptive xanthomata and lipaemic serum.

The assessment of dyslipidaemic patients should focus on the exclusion of secondary disorders, global evaluation of cardiovascular risk, lifestyle evaluation and identification

of monogenic lipid disorders.

Tendon xanthomata are always pathological and are diagnostic of a monogenic lipid disorder.

Taking a family history is very important to identify monogenic disorders.

Screening the family of patients with monogenic lipid disorders will reduce the incidence of premature IHD by identifying for treatment those who are at high risk.

Patients with monogenic lipid disorders are at a very high risk for developing cardiovascular disease and

almost all require lipid-lowering drugs.

Patients with clinically evident vascular disease have an LDL-cholesterol target of 3 mmol/l.

Patients with polygenic dyslipidaemia and no vascular disease should be given lipid-lowering drugs if their calculated risk of MI is more than 20% during the next 10 years after lifestyle modification.

Lifestyle interventions are very effective and cheap and should be applied to all patients.

SINGLE SUTURE

Fasting reduces cholesterol

During the month of Ramadan, observant Muslims typically change their eating habits from three meals a day to two. A small study compared 28 men with high lipid concentrations who 'fasted' during Ramadan and 10 men with normal lipid concentrations who did not fast, and found that a low-fat, low-calorie diet made no difference to the non-fasters. It produced a significant reduction in cholesterol concentrations and energy intake, however, for those who fasted.

(*Saudi Medical Journal* 2003; 24: 184-188.)