Examining patients with dyslipidaemia may be useful in several ways. Some physical signs alert the examiner to the presence of a disturbance in lipoprotein metabolism and should prompt laboratory testing. In other patients dyslipidaemia may already have been documented in the laboratory and the physical examination is directed at finding physical signs that help clarify the nature and aetiology of the lipid disorder. A careful physical examination, especially of the cardiovascular system, may also reveal evidence of as yet unrecognised atherosclerotic complications and indicate the need for lipid-lowering treatment irrespective of the severity of dyslipidaemia.

This article describes physical signs that are directly attributable to dyslipidaemia. Most of these signs are easy to find and interpret, as they reflect the deposition of excess lipids in the skin, tendons or cornea. Plasma can be readily inspected after the blood sample has stood for a while (or has been centrifuged) and the ophthalmoscope allows the visualisation of the retinal vasculature.

Cutaneous signs
Triglyceride-rich lipoproteins have a particular predilection to deposit in the skin. Cutaneous stigmata of hyperlipidaemia are therefore most commonly seen in severe hypertriglyceridaemia but also occur in extreme hypercholesterolaemia.

Xanthelasma (Fig. 1) are the exception to the above rule. They do not always signify dyslipidaemia, but should prompt lipid screening. Xanthelasma do not aid classification of the dyslipidaemia.

Eruptive xanthomata (Fig. 2) occur with severe hypertriglyceridaemia of any cause. They are small yellow to flesh-coloured papules that may have an erythematous base. They are usually asymptomatic. They tend to occur in crops and then enlarge with time. Eruptive xanthomata may coalesce to form tuboeruptive...
More about...

xanthomata (Fig. 3). Eruptive xanthomata are most commonly found around the elbows, knees and buttocks, but can occur anywhere on the body. Because severe hypertriglyceridaemia can cause acute pancreatitis patients with eruptive xanthomata must be investigated and treated with urgency.

Planar xanthomata are flat yellow to orange plaques. If found in the palmar creases they are called palmar crease xanthomata (Fig. 4) and are diagnostic of dysbetalipoproteinaemia. Interdigital xanthomata (Fig. 5) and planar xanthomata in other skin creases are seen in homozygous familial hypercholesterolaemia and occasionally in dysbetalipoproteinaemia.

Tuberous xanthomata (Fig. 6) are firm painless yellow-red nodules that may coalesce to form multilobulated lesions. They are most commonly found on the extensor surfaces of the elbows and knees. Tuberous xanthomata are most commonly seen in homozygous familial hypercholesterolaemia, but are also found in severe heterozygous familial hypercholesterolaemia.

Tendon xanthomata

Tendon xanthomata are most commonly found in the Achilles tendon (Fig. 7) or the extensor tendons of the hand (Fig. 8). On examination one feels for a nodularity of the tendon surface or thickening of the tendon. The nodule moves with the tendon. Routinely palpating the Achilles tendons ensures that xanthomata are not missed and also gives one a good appreciation of the range of normality. Tendon xanthomata are very important physical signs; their presence always indicates a serious problem. They are most commonly found in familial hypercholesterolaemia, but also occur in dysbetalipoproteinaemia and some rare disorders of sterol metabolism.

Ocular signs

Arcus cornealis (Fig. 9) reflects the deposition of lipids in the cornea. Its prevalence increases with age and is also higher in smokers and blacks. If found...
in young (<40 years) patients it should prompt lipid screening. Arcus is associated with hypercholesterolaemia but its presence does not help to classify the lipid disorder. Lipaemia retinalis (Fig. 10) may be seen in severe hypertriglyceridaemia. Rare disorders of HDL metabolism may cause corneal clouding ('fish eye disease').

**Lipaemia**

White or milky serum or plasma (Fig. 11) indicates severe hypertriglyceridaemia and urgent action needs to be taken to avoid an episode of pancreatitis. Most commonly lipaemia will be observed in the laboratory and a comment of ‘lipaemic serum’ on the laboratory report should never be ignored.

**Rational use of lipid investigations**

Dyslipidaemia is a common and an important problem. It accounts for almost 50% of the population-attributable risk of myocardial infarction. Clinicians must therefore detect, treat and monitor lipid disorders, while keeping laboratory expenses reasonable. As with all laboratory investigations testing needs to be individualised.