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Cholesterol emboli

What are cholesterol emboli?

Cholesterol emboli are small deposits of cholesterol that become lodged inside the blood vessels of the skin or other internal organs. This leads to a blockage of the flow of blood through small calibre arteries and causes malfunction or death of the tissue supplied by the affected blood vessels. The process is also called atheroembolism.

What is cholesterol?

Cholesterol is a sterol (a combination of steroid and alcohol). It is a fatty, waxy substance found in the membrane that surrounds every cell of the body. It is an essential component of tissues in animals, plants and fungi.

Cholesterol in humans is mainly manufactured by the liver, but some is of dietary origin. It is transported in the blood stream in small low-density and high-density lipoprotein particles (LDL and HDL cholesterol). High levels of cholesterol, particularly LDL, promotes the development of atherosclerotic plaques in arteries. These cause narrowing of the arteries in coronary artery disease and central or peripheral vascular disease

What causes cholesterol emboli?

The cholesterol emboli originate from ulcerated arteriosclerotic plaques, which can release cholesterol fragments (emboli) into the bloodstream.

This may occur spontaneously, but procedures that disrupt the plaque surface such as angiography, angioplasty, vascular surgery, intra-aortic balloon pumps, and even cardiopulmonary resuscitation are known triggers. Studies suggest it may occur following up to 1% of vascular procedures.

Trauma to the abdomen can rarely precipitate this syndrome by disrupting arteriosclerotic plaques within the abdominal aorta.

Drugs that are used for thrombolysis (to reopen a clotted vessel following a heart attack or stroke) or anticoagulation (to thin the blood) may also predispose to embolisation of cholesterol from unstable plaques secondary to dissolution of the overlying blood clot, or haemorrhage of the arteriosclerotic plaque. The risk however is thought to be very low, and no casual relationship has been demonstrated.

The symptoms are usually noticed within hours to days following a procedure or thrombolysis but usually after 2 months of anticoagulant therapy.

Who gets cholesterol emboli?

Those who have atherosclerosis are at risk of cholesterol emboli. This includes patients with ischaemic heart disease or peripheral vascular disease, as well as those with risk factors such as diabetes, high blood pressure, smoking, obesity, older age, and high levels of cholesterol in the blood.

What are the symptoms and signs?

The two most classic constellations of symptoms are:

- livedo reticularis, leg/foot pain, and good foot pulses
- livedo reticularis, kidney impairment, and eosinophilia (high numbers of circulating eosinophils in the blood).

Over one-third of patients experience skin symptoms including [livedo reticularis](#), gangrene, cyanosis ('blue toe syndrome' secondary to lack of oxygenated blood supply), ulceration, painful red nodules, and [purpura](#) (purple patches).

Purpura due to cholesterol emboli



Cholesterol fragments blocking blood vessels to other organs may result in specific symptoms and signs:

- Acute renal failure (kidney), in 25–50% of cases
- Retinal ischaemia (eye)
- Pancreatitis
- Intestinal infarction

Non-specific symptoms such as fever, myalgia (muscle ache), headache, and weight loss may be present. Interestingly, neither myocardial infarction (heart attack) nor stroke are typical manifestations of this syndrome.

What investigations are helpful to make the diagnosis?

A high index of suspicion is required especially in a patient with known atherosclerosis who has developed the typical skin changes, kidney failure, abdominal pain or diarrhoea, following a vascular procedure.

Definitive diagnosis is made from a [skin biopsy](#) or a biopsy of other involved tissue. The biopsy should show diagnostic cholesterol crystals or clefts within the blood vessel wall, along with thrombi (blood clots). The clefts are spaces left by the crystals that have been washed out by the tissue fixation.

Blood tests show an eosinophilia in up to 80% of affected patients. Other changes that may be seen include:

- Raised white cell count and/or platelet count
- Microscopic blood in the urine or stool
- Elevated erythrocyte sedimentation rate (ESR)
- Deranged kidney function tests
- Raised amylase levels
- Decreased serum complement.

What is the treatment?

The best strategy is prevention of arteriosclerosis with control of vascular risk factors.

Once cholesterol emboli have occurred, procedures to prevent further embolisation such as removal or stenting of unstable atheromatous plaques may be considered. Drugs such as statins, iloprost, pentoxifylline, and steroids have been reported to have limited success to minimise organ damage.

What other substances can lead to embolism?

Cholesterol is most common cause of cutaneous embolic syndrome. However other causes include:

- Oxalate crystals
- Atrial myxomas
- Bacterial endocarditis
- Fat embolism (following major trauma).

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Related information

References:

- Textbook of Dermatology. Ed Rook A, Wilkinson DS, Ebling FJB, Champion RH, Burton JL. Seventh edition. Blackwell Scientific Publications 2004.
- <http://www.uptodate.com>

On DermNet NZ:

- [Vasculitis](#)

Other websites:

Emedicine:

- [Cutaneous Cholesterol Emboli](#)
- [Cutaneous Manifestations of Cholesterol Embolism](#)
- [Cholesterol Embolism](#)

Books about skin diseases:

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