Expert Q&A´s

Spontaneous coronary artery dissection (SCAD)

Professor Bob Graham, executive director of the Victor Chang Cardiac Research Institute is leading Australia's first research into SCAD.

If you are an Australian SCAD survivor and would like to be involved in the Australian first research program at the Victor Chang Institute, please email: scad@victorchang.edu.au

1. What causes the coronary arteries to tear their inner layer and create blockages?

The cause is unknown. It is likely due to a genetic and/or hormonal predisposition coupled with an increase in emotional or physical stress.

The tear itself rarely causes the blockage of blood flow. Rather, it lets blood track into the underlying artery wall. As the blood accumulates in the artery wall it causes the wall to bulge into the artery lumen (cavity), which is what obstructs the flow of blood leading to a heart attack and/or death.

Only 30% of SCAD cases are associated with a tear in the lining of an artery. The other 70% are due to a haemorrhage into the wall of a coronary artery, due probably to rupture of the small blood vessels (vasa vasorum) providing blood to the artery itself. As a result of the haemorrhage into the wall, it bulges into the lumen of the affected vessel and blocks the flow of blood to the heart.

2. Can these tears be repaired?

Fortunately, the tear heals spontaneously, generally within 35 days.

3. Statistics show 80-90% of SCAD patients are women with no other risk factors for heart disease and it's most likely to affect those in their 40s-50s. What can they do to prevent it?

At present there are no known ways of preventing it, but there is observational data to suggest that controlling blood pressure and the use of a beta-blocking drug can reduce the chance of recurrences, which occur in up to 30% of cases.

4. Research shows between 12-29% of those diagnosed experience it again. Do we know why?

Presumably the same mechanisms that caused the first episode also causes subsequent attacks. Recurrences always involve different branches of the coronary artery tree, not the one involved in the initial episode.

5. Do hormones play a part in SCAD? Eg, the physical changes women experience after major life events such as childbirth and menopause.

This is very likely but the mechanisms involved are not understood.

6. SCAD is considered a completely different type of heart attack. How does it differ to what we consider a 'regular' heart attack?

A regular heart attack is due to the accumulation of plaque (fatty material and inflammatory cells) in a coronary artery, which can then break away from the artery wall. This is a very powerful stimulus for a clot to form that then obstructs blood flow.

These heart attacks are common in people with risk factors (such as high cholesterol, smoking, high blood pressure, diabetes, a family history of premature heart disease, etc.) for the development of atherosclerosis, a condition characterised by the accumulation of plaque in an artery.

In contrast, plaque and clot formation play little role in a SCAD heart attack, which is due rather to a haemorrhage (bleed) into the wall of a coronary artery, which causes the wall to bulge into the lumen of the artery and obstruct blood flow.

These people often have few if any of the traditional risk factors mentioned. So, the pathophysiology of a regular and SCAD heart attack are diametrically opposite; the former resulting from a clot and the latter from a bleed, but in both case the blood flow in the affected artery is reduced, which causes the heart attack.