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A Report on Spontaneous Coronary Artery Dissection

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Brief Report

Atherosclerotic acute coronary syndromes (ACS) are caused by Spontaneous Coronary Artery Dissection (SCAD), which is a rare non-atherosclerotic cause. Because of increasing awareness and early use of invasive angiography in patients presenting with acute chest discomfort, more cases are now being discovered. It's crucial to recognise SCAD because the patient's features and treatment differ significantly from those seen in conventional ACS instances. Without the traditional cardiovascular risk factors, SCAD patients are often younger and more likely to be female. The results of Percutaneous Coronary Intervention (PCI) and Coronary Artery Bypass Grafting (CABG) revascularization are unsatisfactory. In the majority of uncomplicated instances, conservative therapy is chosen, with the dissection healing on its own.

SCAD is caused by an abrupt rupture of the coronary artery wall, causing the inner intimal lining to separate from the outer vessel wall. An intimal rip or bleeding from the vasa vasorum is thought to be the causes of intramural haematoma. The dissection plane propagates due to pressure-driven enlargement of the haematoma, resulting in the creation of a real lumen and a thrombus harbouring a false lumen. Dissection produced by plaque rupture in patients with atherosclerosis or catheter-induced iatrogenic dissections should be separated from SCAD. SCAD patients have thin artery walls with no atheroma or calcification to prevent dissection spread. As a result, dissections in SCAD cases are more common, while non-affected coronary artery segments appear smooth and disease-free on angiography.

Up to 50% of people with SCAD have ST-elevation Myocardial Infarction (MI), with the remainder having non-ST-elevation MI. Troponin levels will be increased in the majority of SCAD patients. Early consequences include life-threatening ventricular arrhythmias and abrupt cardiac death.

SCAD is diagnosed through a coronary angiography procedure.

There are three different types of findings:

Type 1: A longitudinal filling defect that represents the radiolucent intimal flap is the standard description. The artery wall is frequently stained with contrast, giving the illusion of a second lumen.

Type 2: Diffuse long smooth tubular lesions with no obvious dissecting plane that can culminate in full artery occlusion (due to intramural haematoma). Lesions are usually >30 mm long, with a sharp difference in vessel diameter between healthy and diseased segments. Intracoronary nitrates have no effect, and there are no atherosclerotic lesions in other coronary segments.

Type 3: Multiple localised tubular lesions mimicking atherosclerosis due to intramural haematoma. To make the diagnosis, intravascular imaging is essential.

SCAD is frequently misdiagnosed as atherosclerosis if the diagnosis is based solely on the imaging of the characteristic dissection flap. The most prevalent type of SCAD is Type 2, which causes lumen compression due to an intramural haematoma with no intimal flap (67 percent of cases). Type 1 SCAD with characteristic dissection flap is found in 29% of cases, while Type 3 SCAD imitating atherosclerosis is found in just 4% of instances. As a result, depending on coronary angiography to detect contrast wall staining (Type 1 SCAD) would result in a considerable number of instances being ignored. The diagnosis of Type 2 and Type 3 SCAD requires intravascular ultrasonography (IVUS) or Optical Coherence Tomography (OCT) imaging of the vessel wall. In a young female patient with generally smooth coronary arteries, diffuse lengthy coronary lesions should always raise the possibility of intramural haematoma in the differential diagnosis.

SCAD is a leading cause of ACS in young women who do not have typical cardiovascular risk factors. ACS induced by plaque rupture or erosion has a different aetiology and therapy. The majority of coronary dissections heal on their own, and in uncomplicated instances, conservative therapy is suggested. Although the long-term prognosis is favourable, there is a considerable chance of recurrent SCAD occurrences, which occurs at a rate of 5% per year on average.

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