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Case Report

An Unusual Case of Thromboembolic Stroke - Silent Myocardial Infarction from Spontaneous Coronary Artery Dissection

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Abstract

An incidental finding on a patient who was followed up after presenting to the hospital with a cerebrovascular event. The angiogram performed showed Spontaneous Coronary Artery Dissection (SCAD) of Left Anterior Descending (LAD) artery. This has resulted in thrombus formation within the left ventricle which led to a thromboembolic stroke. The patient did not have any typical associated conditions for SCAD and it is theorized that heavy smoking and secondary polycythaemia might be contributing factors to the findings.

Introduction

Spontaneous Coronary Artery Dissection (SCAD) is a very rare cause of acute coronary syndromes [1]. The pathogenesis remains unclear. However, several diseases and conditions have been associated with SCAD, such as connective tissue disorders [2,3], increased sheer stress such as after exercise [3,4]. It affects predominantly females, particularly in the peri-partum and post-partum period [1,3,5-8]. We present an unusual case of SCAD in a male patient who presented with stroke.

Case Presentation

A 57-year-old Caucasian male was admitted with right sided weakness. Past medical history includes COPD with a history of 40 pack years. ECG showed inferior Q waves and lateral T-wave inversion (Figure 1). He denied any ischemic symptoms such as chest pain prior to presentation. Diabetes was ruled out with a normal fasting glucose. Admission bloods showed polycythaemia which was subsequently seen by the haematology team who concluded that it is secondary to smoking.

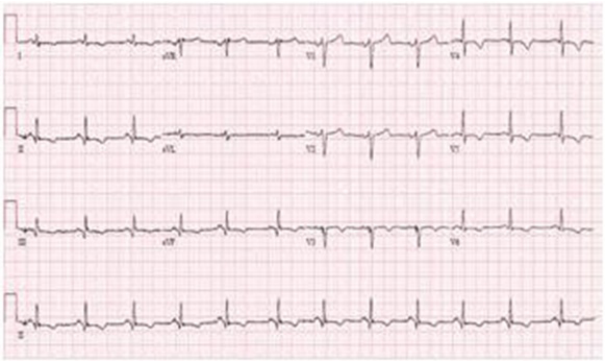

Figure 1: ECG showing inferior and lateral lead changes.

CT scan on admission showed an area of low attenuation in the right cerebellum and he was diagnosed clinically as left hemispheric stroke (Figure 2). He was initiated on antiplatelet therapy as per local stroke protocol. An echocardiogram was performed as to rule out embolic cause of stroke.

Echocardiogram showed an akinetic apex with thrombus within the apex and evidence of severe left ventricular systolic impairment with ejection fraction of <35% (Figure 3). He was started on apixaban by the stroke team (as an unlicensed indication as patient declined to be started on warfarin) and was referred to cardiology team.

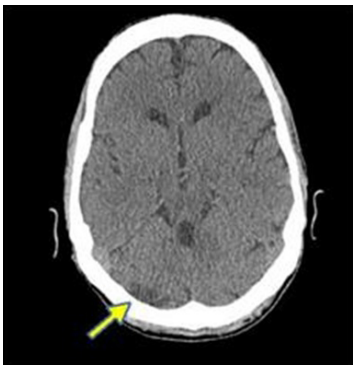

Figure 2: CT brain showing low attenuation in right cerebellar (Arrow).

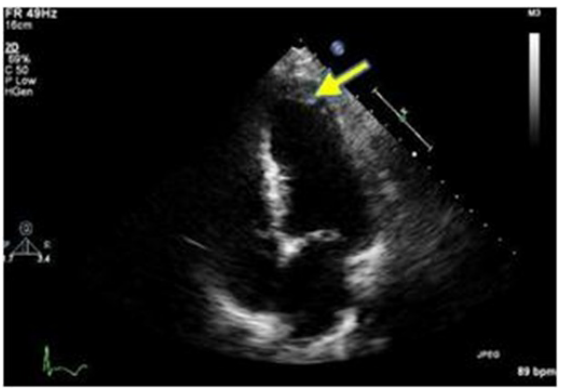

Figure 3: Echocardiogram showing thrombus in apex (Arrow).

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Given the evidence of akinesia and ECG changes, he was listed for a diagnostic coronary angiogram. Angiogram revealed Spontaneous Coronary Artery Dissection (SCAD) in middle segment of LAD and unremarkable findings in RCA (Figure 4).

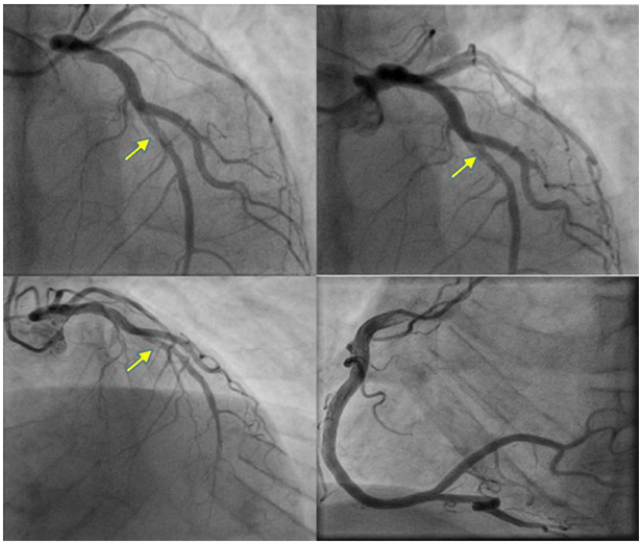

Figure 4: Angiogram showing SCAD in mid LAD (arrows) and normal RCA.

SCAD had resulted in myocardial infarction causing apical akinesia. This allowed propagation of a left ventricular thrombus, resulting in a left hemispheric stroke.

Given that the patient was stable, he was treated conservatively with anticoagulation and followed up in outpatient cardiology clinic.

Discussion

This is an interesting case of a male patient who initially presented with stroke as a result of SCAD without typical associated risk factors for SCAD. We theorized that the most likely contributing factors would be smoking and its related secondary polycythaemia.

Stroke is most commonly caused by carotid artery disease and in the younger population, clinicians should always be mindful towards embolic causes for stroke. This triggered a host of investigations in this gentleman's case which led to the findings of SCAD as the underlying cause for his symptoms.

It has been thought that SCAD starts out with the development of an intramural haematoma that usually runs in the outer media or between the media and adventitia of the coronary artery, resulting in a false lumen. This lumen expands through blood or clot propagation which then compresses the real lumen, causing ischemia [9].

It is thought that the increased blood volume and cardiac output during pregnancy and labour is one of the contributing factors which makes females in the peri-partum and post-partum period more prone in particular [9]. We understand through studies of patients with polycythaemia vera that blood rheological variables like haematocrit and blood viscosity can promote raised intima media thickness endothelial dysfunction and altered arterial compliance [10-12]. There are also a few case reports on SCAD and polycythaemia vera [13,14]. However, there are no reported cases SCAD with secondary polycythaemia.

Although smoking is one of the well-known and established cardiovascular risk factor by promoting vasomotor dysfunction, thermogenesis and thrombosis it is not typically associated with SCAD. Given the proposed pathogenesis of SCAD, the combination of heavy smoking and secondary polycythaemia could potentially be associated risks [15].

In this case, the differentials included an atherosclerotic plaque event resulting in a similar presentation or a Lotus-Root like lesion causing the angiographic findings. Motreff et al., identified 5 angiographic features which could assist clinicians towards the diagnosis of SCAD [16,17]. The patient had 3 out of the 5 criteria fulfilled i.e., absence of atheroma on other arteries, radiolucent flap generating two lumens and ending of angiographic ambiguity on a side branch. This makes the diagnosis of SCAD more probable. The diagnosis could be confirmed by optical coherence tomography which was not performed in this case.

Summary

Although carotid artery disease is a major culprit in ischemic strokes, it is important to exclude an embolic cause, especially in younger patients. Left ventricle thrombus is one of the potential sources. Our patient presented with stroke and an abnormal ECG. This prompted further investigations which revealed the underlying cause of stroke - SCAD. This highlights the importance of a comprehensive approach to diagnosis in all patients.

Competing Interests

The authors declare that there is no conflict of interest regarding the publication of this paper.

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