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CASE REPORT

Sudden cardiac death in a young adult man due to spontaneous coronary artery dissection

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Abstract

Spontaneous coronary artery dissection is a rare event and commonly associated with pregnancy and female gender. This condition can reduce or completely obstruct the blood flow to the heart, causing a myocardial ischaemia, abnormalities in heart rhythm or sudden death. We present a case of a 28-year-old Indian male with no previous medical illness who complained sudden onset of chest pain prior to his death. Autopsy revealed a left anterior descending coronary artery dissection associated with plaque rupture. The anterior wall of left ventricle showed contraction band necrosis. There was also atheroma present in the right coronary artery which was insignificant. Histologically, dissection was associated with atherosclerosis. There was no evidence of vasculitis. The cause of death was given as coronary artery dissection due to coronary artery atherosclerosis.

Keywords: Sudden death; coronary artery dissection; post-mortem

INTRODUCTION

Spontaneous coronary artery dissection (SCAD) is being recognised worldwide and are being investigated to understand further on the pathophysiology. The first case was reported in 1931 involving a 42-year-old woman.[1] It has been largely described in postpartum women. It can be missed unless there is a high level of suspicion and careful post-mortem examination of the coronary artery. SCAD can cause sudden cardiac death or result in myocardial infarction.

CASE REPORT

A 28-year-old Indian male complained of chest pain for almost 12 hours and worsened 2 hours prior to his death. He had no previous medical illness or surgical intervention. There was no cardiopulmonary resuscitation carried out. He was brought in dead by the police to the Forensic Medicine Department for post-mortem examination.

Post-mortem external examination showed a medium built adult male with congested face. Other external examination was unremarkable. On the internal examination, the heart weighed 280 grams and was not enlarged. All the coronary arteries originated normally from the ostium. The left anterior descending artery demonstrated coronary artery dissection at 1 cm from the ostium for about 2 cm in length (Fig. 1). There was no thrombus seen in the dissection and the lumen was not occluded grossly. No haemorrhage or fibrosis was seen on the myocardium. The right coronary artery showed non-significant luminal narrowing by atheroma (Fig. 2). The left circumflex artery was unremarkable. The aorta showed presence of fatty streak. The right lung weighed 320 grams and left lungs weighed 280 grams. Both lungs were not oedematous. Histologically, the left anterior descending coronary artery showed dissection associated with plaque rupture (Fig. 3). However, there was no evidence of vasculitis seen. Histology of the myocardium showed contraction band necrosis at the anterior wall of the left ventricle (Fig. 4). The level of troponin T taken at the time of autopsy was 324 ng/L. The toxicology was negative.

DISCUSSION

SCAD is defined as a non-traumatic and non-iatrogenic separation of the coronary arterial walls, creating a false lumen.[2] It is difficult to ascertain true prevalence of SCAD due to underdiagnosis and varying presentation. SCAD

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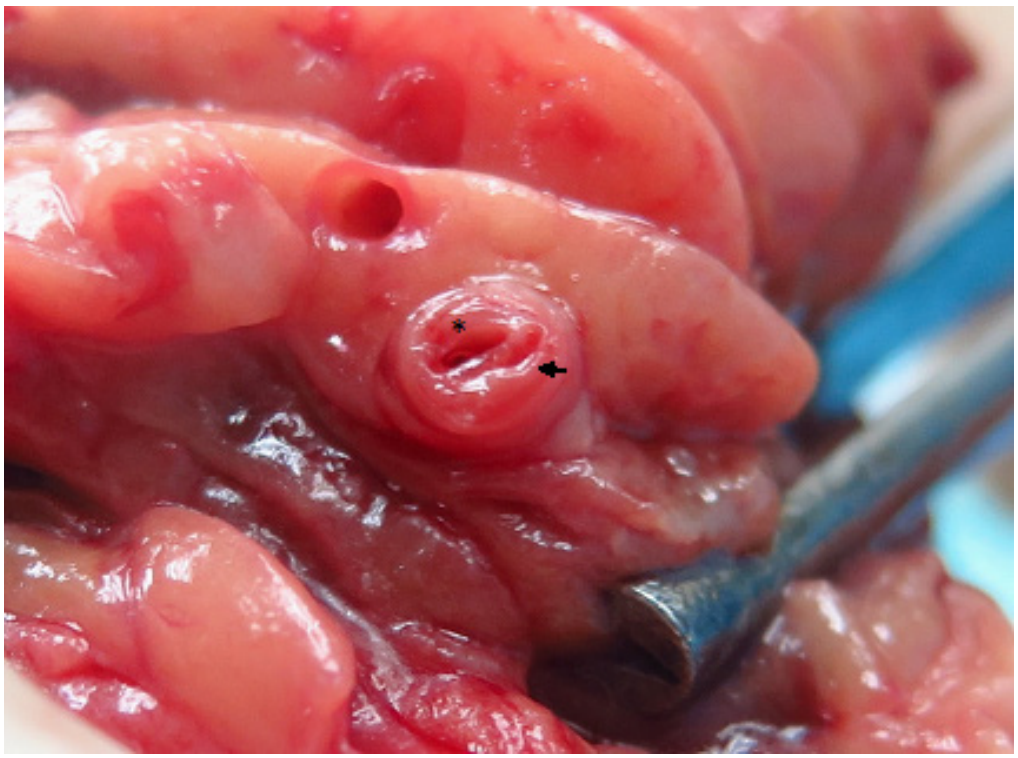

FIG. 1: Left anterior descending artery showed dissection. However, no thrombus was seen in the dissection plane (arrow) or in the true lumen (asterisk).

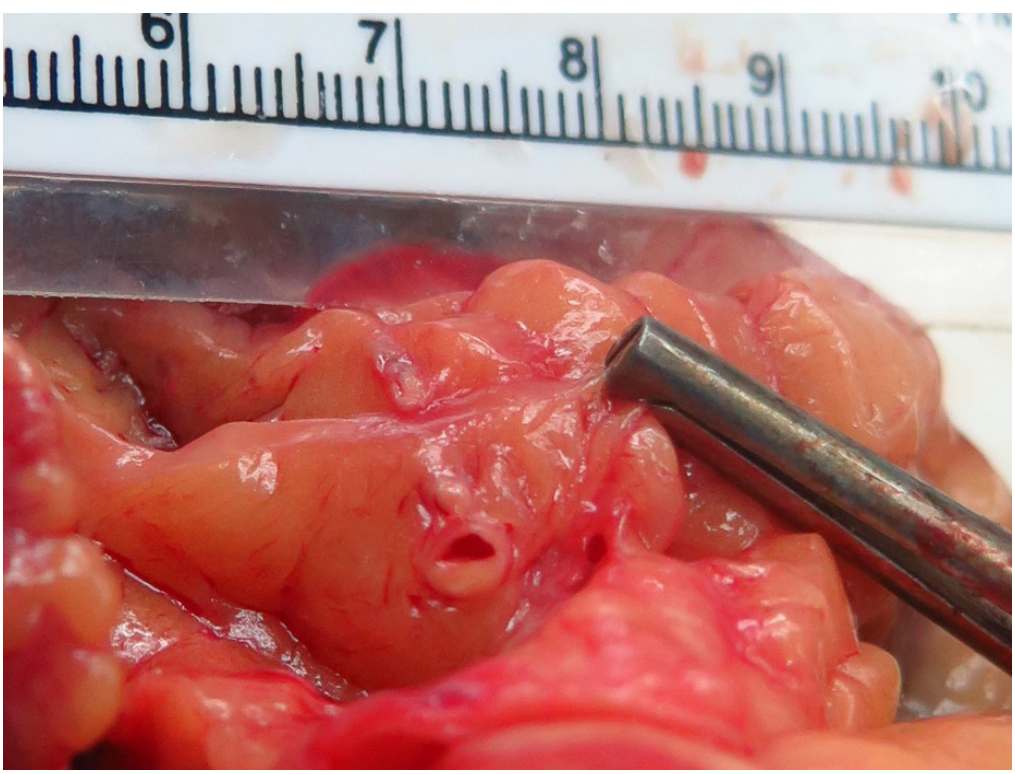

FIG. 2: Eccentric occlusion by atheroma of the right coronary artery. The occlusion was not significant to cause death.

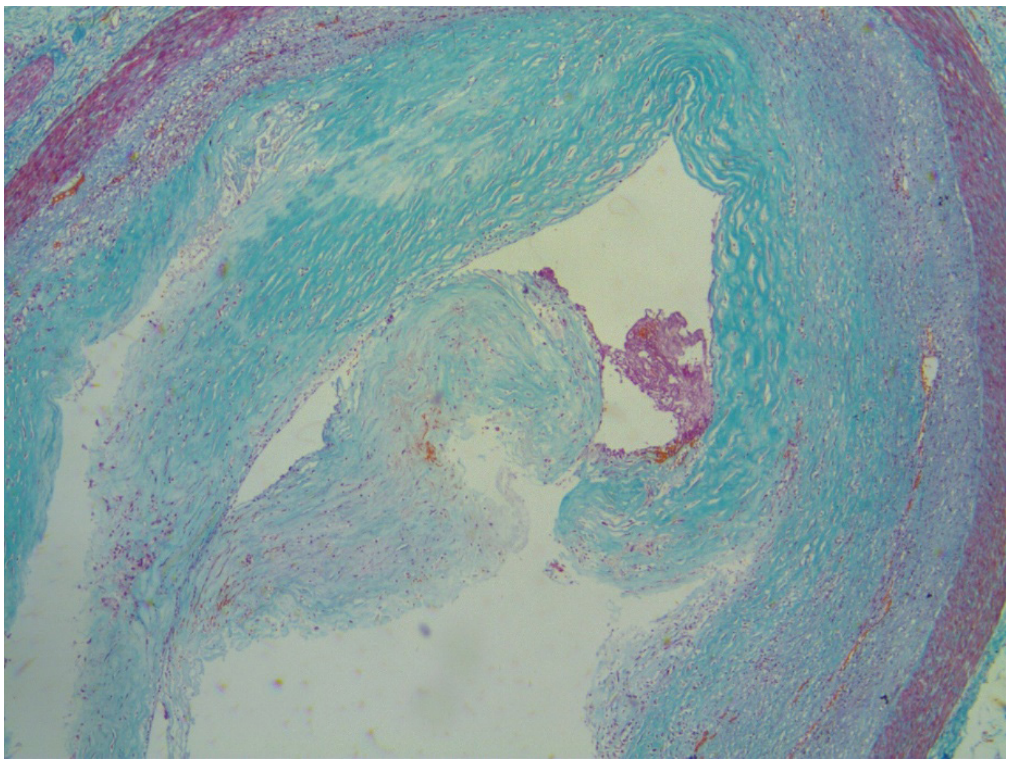

FIG. 3: Section of the left anterior descending artery showed plane of dissection associated with plaque rupture (Masson Trichrome, x4).

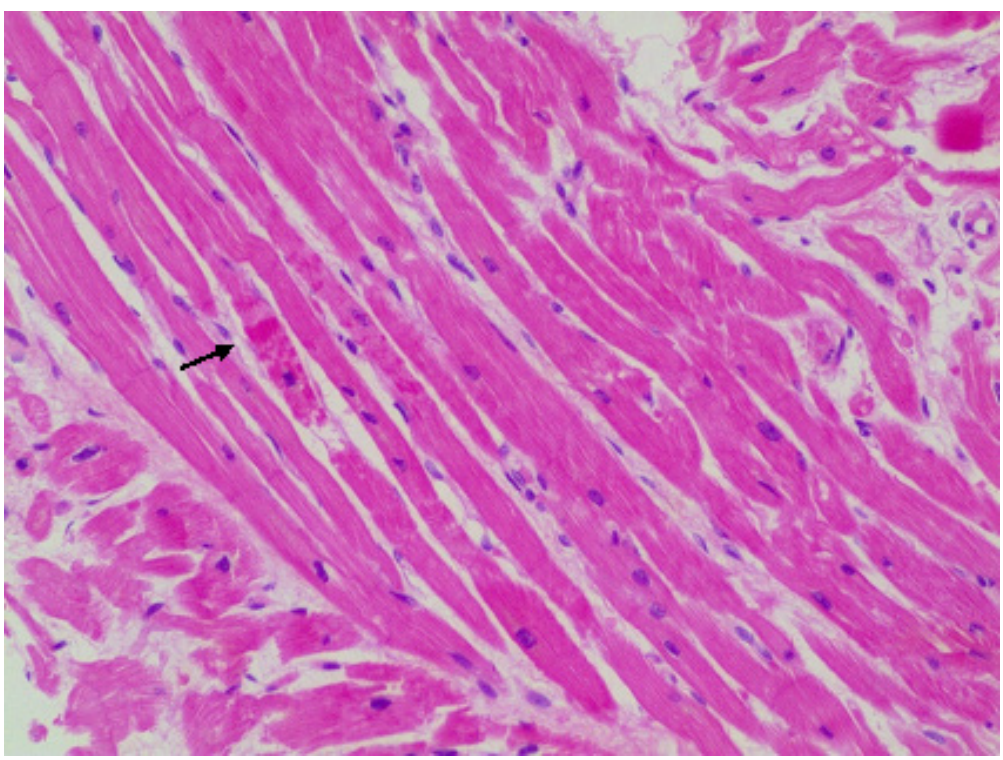

FIG. 4: Section of the anterior wall of left ventricle showed presence of contraction band necrosis (arrow) (H&E, x20).

should be suspected when there is myocardial infarction in the young, when there is little or no evidence of atherosclerosis in coronary artery, peripartum state, history of connective tissue disorder such as Marfan's syndrome, Ehlers Danlos syndrome, cystic medial necrosis, fibromuscular dysplasia and history of relevant connective tissue disorder such as polyarteritis nodosa, systemic lupus erythematosus, and Crohn's disease. Dissection due to atherosclerosis can occur even in minimal atherosclerosis. In atherosclerotic arteries, atherosclerotic plaque cause increase density of vaso vasorum which may cause bleeding and rupture of nidus.[3] Subsequently, it can cause dissection of adventitia from the media and rupture of intima. The other possible cause is an intimal tear progressing into media by the force of arterial pressure.[3]

Neri *et al*.[4] classified dissection into three main types of lesions based on proximal aortic dissection with coronary malperfusion. They are categorised as follows:

- Type A ostial dissection. This dissection is associated with a disruption of the inner layer limited to the area of the coronary ostium. Coronary insufficiency is caused by the local flap through trapdoor mechanism.
- Type B dissection with a coronary false channel. The blood flows in the false lumen during diastole and compresses the true lumen.
- Type C lesion is a circumferential detachment of coronary artery with an inner cylinder intussusception which causes luminal obstruction.

Although there was no thrombus seen in the dissection and the lumen was not occluded in our case, we believe that the possible mechanism that caused death is the type B (Neri *et al*)[4] dissection. The blood flow into the false lumen during diastole could have compressed the true lumen and caused myocardial insufficiency as what could have occurred in our case.

The common consequences of the dissection are sudden cardiac death or myocardial infarction. Ventricular arrhythmia had been reported in 8-14% of patients.[5,6] In this case, we believe the dissection itself caused obstruction of the left anterior descending coronary artery resulting in myocardial ischaemia as evidenced by contraction band necrosis in the anterior wall of left ventricle and raised troponin T. The level of troponin T was also raised in this case indicating myocardial damage and was 20-fold more than the required level to diagnose myocardial damage in living patients.

Although coronary artery dissection is a rare complication of atherosclerosis, it must be looked for especially when there is minimal atherosclerosis to cause death.

Conflict of interest: The authors declare that there is no conflict of interest.

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