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SHORT COMMUNICATION

Successful treatment of long spontaneous coronary dissection with medical management: Not to intervene

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Abstract Spontaneous coronary artery dissection (SCAD) is an uncommon cause of acute coronary syndrome (ACS) and optimal therapy has not been well-defined. We present a case of long SCAD with complete healing due to medical management. A 47-year-old woman presented to emergency department because of sudden onset of typical chest pain. Electrocardiogram (ECG) showed minimal ST-segment elevation in leads V1–V4. Coronary angiography showed a long spiral dissection extending from the middle segment to the distal segment of the left anterior descending artery and TIMI flow grade three. We decided to follow-up with medical management and have control angiography unless hemodynamic instability and chest pain emerged. Control angiography displayed complete healing of dissect segment after six months. SCAD should be considered, especially in women who present with an ACS without a history of cardiovascular disease and risk factor. This report offers the idea that medical management can be a choice even if in the long segment SCAD setting.

1. Introduction

Spontaneous coronary artery dissection (SCAD) is an uncommon cause of acute coronary syndrome (ACS) and sudden death. Its incidence has increased progressively since the first angiographic report by Ciraulo in 1978,[1] consequent of common use of coronary angiography. In a recent angiographic study, prevalence of SCAD was higher in women than in men and increased as age decreased and reached 7.6% and 10.8% below the age of 40 years and below the age of 50 years for women presenting with an ACS with ST segment elevation, respectively.[2] Although there is growing knowledge, there is no consensus concerning SCAD treatment. Here, we present a

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case of long SCAD in the left anterior descending artery (LAD) with complete healing from medical management.

2. Case presentation

A 47-year-old woman without a history of cardiovascular disease and risk factor for cardiovascular disease presented to the emergency department because of sudden onset of typical chest pain continuing one hour. Cardiovascular and other system examinations were normal. Initial electrocardiogram (ECG) demonstrated minimal ST-segment elevation in leads V1-4 (Fig. 1A). Cardiac troponin (TrI) was 0.02 ng/mL (0.00–0.02 ng/mL), and other laboratories, including cholesterol levels were normal. After transfer to the coronary care unit with acute anterior myocardial infarction diagnosis, chest pain relieved, and ECG showed ST resolution (Fig. 1B). We decided not to give fibrinolytic therapy and initiated medical management with aspirin, loading doses of 300 mg clopidogrel, bisoprolol, atorvastatin and enoxaparin. Echocardiography showed antero-apical wall hypokinesia. After 6 h, repeat ECG demonstrated deep negative *T* waves in the anterior leads and TrI reached 0.5 ng/mL. The next day, coronary angiography was performed and revealed a long dissection plane with a classical dissection flap originating from the middle segment of left anterior descending artery (LAD), extending to the distal segment of LAD with no atherosclerotic coronary artery disease (Fig. 2A). Thrombolysis in Myocardial Infarction (TIMI) flow grade was three with moderate luminal compromise, and other coronary arteries were angiographically normal. Thus, we decided medically to follow-up unless hemodynamic compromise and ischemic events emerged. She remained stable during her hospital stay, and control angiography displayed complete closure of the dissect segment with slightly decreased luminal caliber after one week (Fig. 2B). She was discharged home on medical management with aspirin, clopidogrel, and bisoprolol. At the sixth month of follow-up, the echocardiography and exercise stress testing were normal. Control angiography displayed complete healing of the dissect segment with good luminal caliber (Fig. 2C).

3. Discussion

Recognition of SCAD has increased because of widespread use of coronary angiography. In one series, prior to frequent use of coronary angiography, 62 of 83 cases that have been described were diagnosed at autopsy.[3] Clinical presentation of SCAD ranges from asymptomatic to acute coronary syndrome and sudden cardiac death, depending on the involved arteries, extension of the dissection, and luminal compromise. SCAD can involve right and left coronary systems, but the left coronary artery is more frequently involved (78% vs 32%).[4] Left main and multivessel involvement are more frequent in women than in men (29% vs 5%, 33% vs 9%, respectively) and 41% of women are in the peripartum period.[4] Although the pathogenesis and etiology of SCAD have not been explained fully, connective tissue disease, atherosclerosis, coronary vasospasm, blunt chest trauma, medications (oral contraceptives and immunosuppressive therapy), cocaine abuse, and intense physical activity have been implicated as possible causes of SCAD.[4] However, in a substantial percentage of SCAD, as in our case, none of these causes were present and also antinuclear antibody and Anti ds-DNA levels were negative. The primary dissection

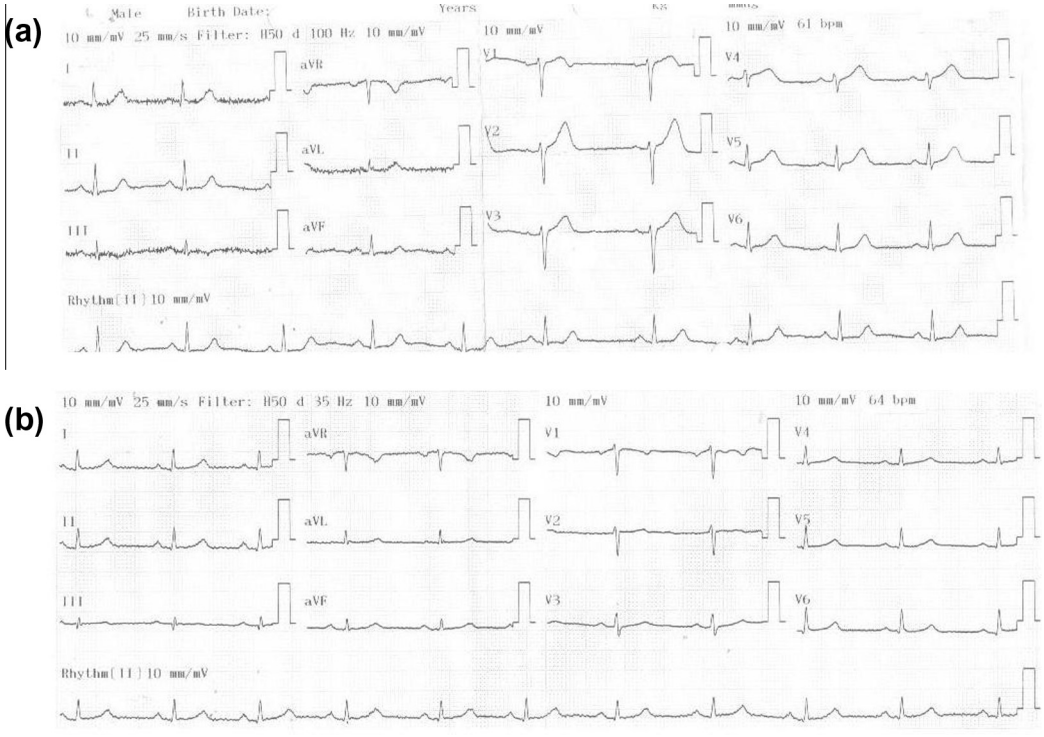

Figure 1 Electrocardiography showed minimal ST-segment elevation in leads V1–V4 (a), which improved spontaneously after transfer to the coronary care unit (b).