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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

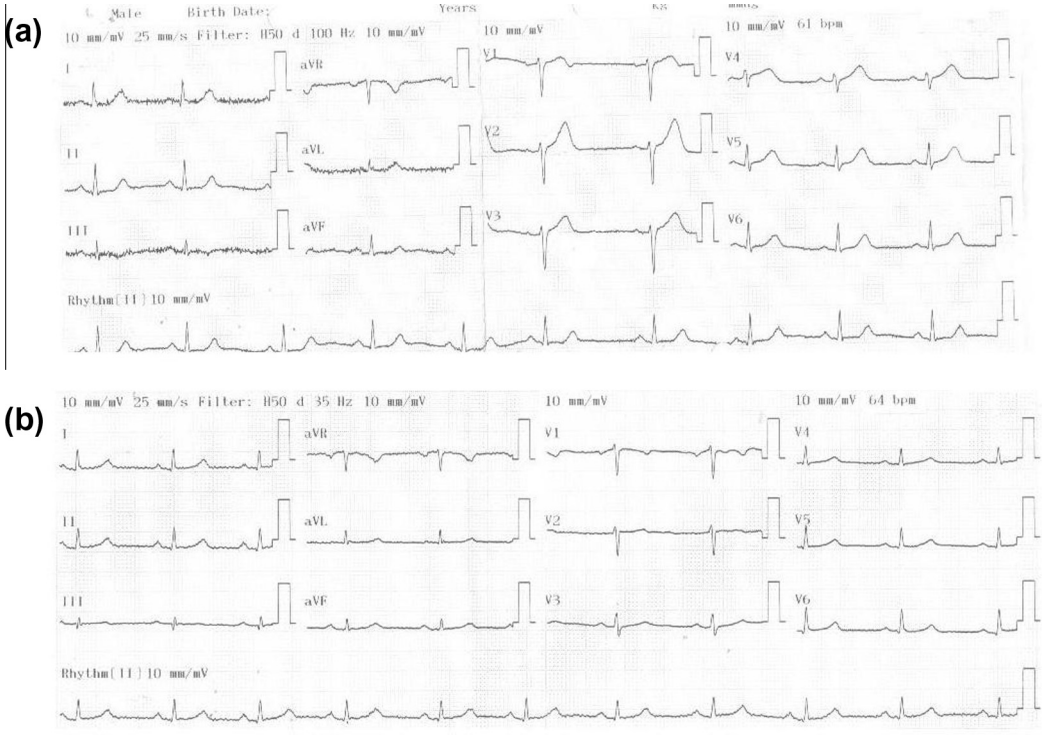

Figure 1 Electrocardiography showed minimal ST-segment elevation in leads V1–V4 (a), which improved spontaneously after transfer to the coronary care unit (b).

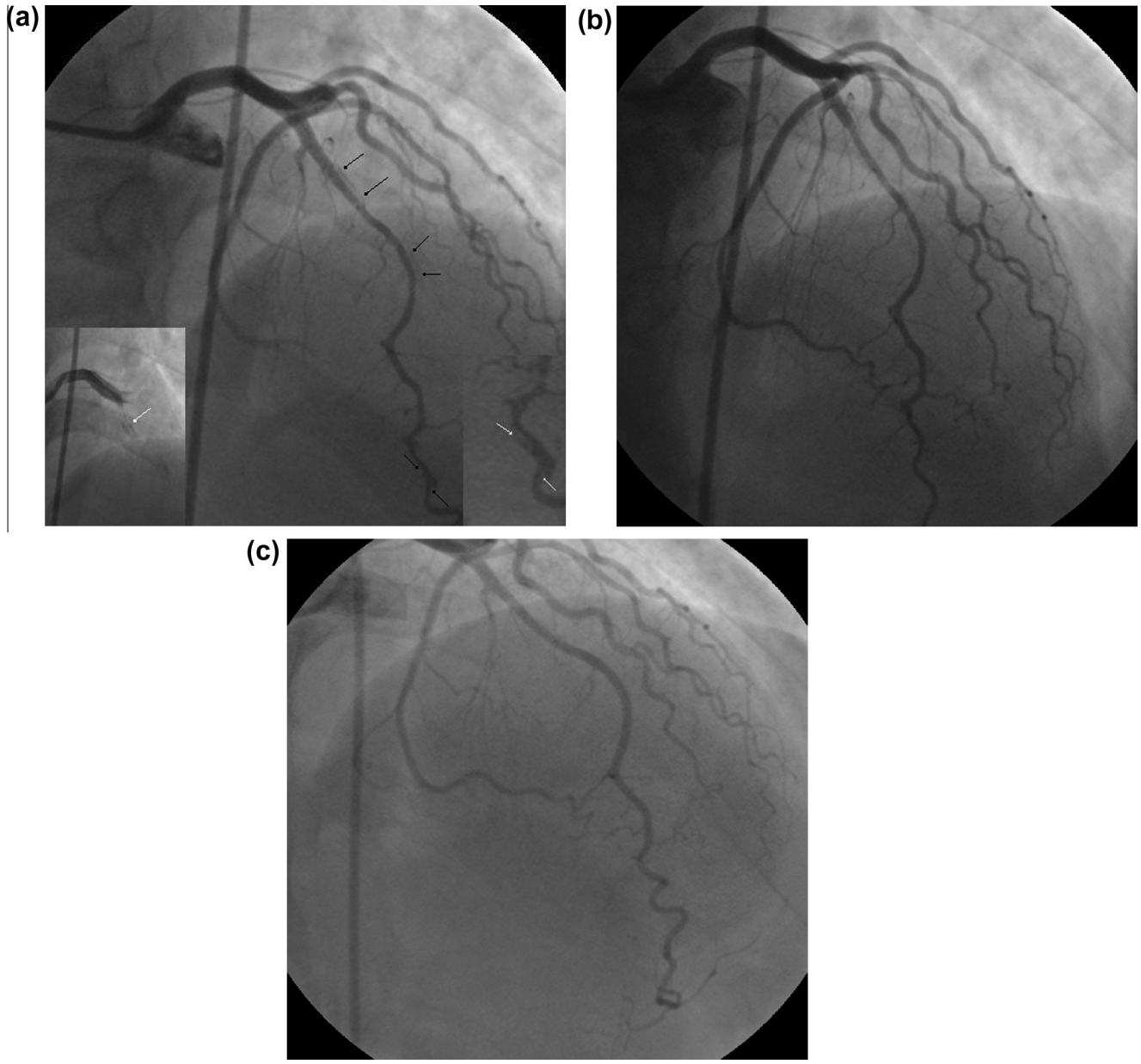

Figure 2 Left coronary angiogram; showing a long dissection plane (black arrows), with a classical dissection flap (Contrast staining on lower left corner small picture), originating from the middle segment of the left anterior descending artery (LAD) extending to the distal segment of the LAD (white arrows emphasize in magnification on the lower right corner) with no atherosclerotic coronary artery disease (a). Control angiography; displaying complete closure of the dissect segment with slightly decreased luminal caliber due to the intramural hematoma after one week (b). Final angiography; displaying complete healing of the dissect segment with good luminal caliber at sixth month (c).

is possibly due to either an intimal tear or the disruption of vasa vasorum subsequent to hemorrhage into the media[5], and the dissection plane usually cruises in the outer media or between the media and adventitia. The true lumen compression by the intramural thrombus in the false lumen may lead to blood flow obstruction and myocardial ischemia. A radiolucent line is seen and represents the separation of the true and false lumen on coronary angiography, which can confirm the SCAD diagnosis in most cases. An intimal flap can be visualized. Our case showed a long spiral dissection plane with well-visualized classical dissection flap as well as, true and false lumen that had developed in a normal, non-atherosclerotic coronary artery.

Optimal treatment strategy of SCAD, especially for long dissection, has not been well-defined. Management decisions are formed based on angiographic assessment of dissection site and extent, degree of flow compromise and clinical scenario, on a case-by-case basis. Treatment options include conventional medical therapy, thrombolytic therapy, percutaneous coronary intervention (PCI), and surgical revascularization. Medical therapy alone can be a choice in mid-or distal SCAD

with TIMI 2–3 flow[2], and stable patients with SCAD have a favorable outcome with medical management.[6] Thrombolysis that has been successfully used[6,7] in SCAD may be effective by lysing of the thrombus in the false lumen allowing the true lumen to re-expand. However, thrombolytic therapy has been associated with the extension of the dissection[8] and hemopericardium secondary to a ruptured coronary artery[9], so it is controversial. PCI with stenting has been increasingly used as a therapeutic option in angiographically eligible cases in which the dissection flap does not involve a long segment and true and false lumen are well-visualized.[2,4] Balloon angioplasty without stenting should be avoided as it can worsen the dissection.[10] CABGO has been successfully performed in patients who have multivessel or LMCA, proximal LAD dissection, and have undergone unsuccessful PCI.[2] The longitudinal extension of dissection may limit the use of treatment options including CABGO and stent-based therapy. Very long and multiple stents may also be necessary with subsequent risk of in-stent restenosis. Our patient was asymptomatic and hemodinamically stable. In addition, properties of angiographic lesion were not suitable to attempt grafting onto the long dissect segment and required multiple stents to cover dissection like a metal jacket. Therefore, medical follow-up is preferable to invasive strategy unless hemodynamic compromise and ishemic events emerge. Medical treatment with aspirin, clopidogrel and beta-blocker led to complete healing of dissect segment with good luminal caliber at the sixth-month control angiography.

4. Conclusion

In conclusion, SCAD should be considered in young patients who present ACS, especially in women with no cardiovascular disease and risk factors. Early coronary angiography should be performed in order to identify the preferred therapeutic option. In addition, medical management can be a therapeutic option not only for patients with a short dissection but also for those with a long dissection in any big vessel.

5. Disclosure

The authors report no financial relationships or conflicts of interest regarding the content herein.

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