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Acute Myocardial Infarction Due to Coronary Artery Dissection in a Premenopausal Woman, Possible Contribution of Aortic Coarctation and Review of Literature

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Authors' contributions

This work was carried out in collaboration between all authors. Author SC designed the study, wrote the protocol, and wrote the first draft of the manuscript. Authors PKC and TA managed the analyses of the study. Author FK managed the literature searches. All authors read and approved the final manuscript.

Short Communication

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ABSTRACT

It is well-known that coronary artery disease (CAD) is the most cause of death in all over the world and generally been considered as a disease of elderly people and occasionally middle-aged men. Emerging data displays an important sex-based difference in CAD. Therefore CAD in the female population especially in premenopausal women is still identified less often and is treated less aggressively compared to that in the male population. The incidence of CAD in women older than 65 years is similar to that in men. However It is generally acknowledged that the risk for CAD is very low in premenopausal women by the reason of protective effect of woman hormones. Herein we report acute myocardial infarction due to coronary artery dissection in a premenopausal women, and conceivable contribution of aortic coarctation.

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Keywords: Acute myocardial infarction; coronary artery dissection; premenopausal woman; aortic coarctation.

1. INTRODUCTION

According to updated statistics, cardiovascular disease is the first cause of death in all over the world and cardiovascular disease accounts for approximately 30% of all other deaths [1]. Spontaneous coronary artery dissection (SCAD) is an unusual cause of acute myocardial infarction and sudden death [2]. Firstly SCAD was described during the postmortem examination of a 42-year-old woman with chest pain and sudden cardiac death in 1931[3]. It is difficult to determine the incidence of SCAD and it is often detected only after the death of the patient. SCAD has been reported 0.1–1.1% as a cause of acute myocardial infarctions. Most of the patients are women, and half of the cases are observed in the postpartum period. About 20% of the survivors die within 3 years [4].

2. CASE PRESENTATION

43-year-old woman was admitted to the emergency department with chest pain longstanding approximately 2 hours. In her medical history, no coronary risk factors such as hypertension, diabetes, dyslipidemia and smoking was found. The patient did not have history of co-morbid disease (deep vein thrombosis, pulmonary embolism, stroke or transient ischemic attack, peripheral arterial disease, rheumatologic/oncologic disease and any prior coronary artery disease). Electrocardiography showed sinus tachycardia and ST-segment elevation in leads I, a VL (both 2 mm) and V4–V6 (all 3 mm), along with ST segment depression in leads III (1 mm) and V1 (3 mm), suggesting that an acute ST-segment elevation myocardial infarction (Fig. 1). On physical examination, a systolic murmur was auscultated on left subscapular site and echocardiography was performed. On echocardiography, aortic coarctation with the 45 mmHg gradient was detected. On laboratory, increased level of troponin-I and creatinin kinase-MB were found. After initial treatment with acetyl salicylic acid 300mg, Clopidogrel 300mg and unfractioned heparin in emergency room; coronary angiography and if appropriate, primary percutaneous coronary intervention was planned. On coronary angiography; 90% thromboses lesion at the level of ostial part of the left anterior descending artery (LAD), 99% thromboses lesion at the level of mid-part of left circumflex artery and Optus marginalis detected. Left anterior descending artery images were suggested that it was a coronary artery dissection (Fig. 2). Coarctation of the aorta with 45 mmHg gradient distal to the subclavian artery was detected on aortography (Fig. 3). At the same day, the patient underwent emergency coronary artery bypass grafting and repair of aortic coarctation. Under general anesthesia, after sternotomy, lesion of LAD was confirmed that it was a coronary artery dissection. Then left internal mammary artery (LIMA)-*obtuse marginal* branch (OM2) and right internal mammary artery (RIMA)- LAD autogenic bypass grafts were performed. Coarctation of the aorta with a 18mm Dacron graft has been fixed. Intra-aortic balloon pump was inserted in operation room. Intra-aortic balloon pump was removed at the second day and there were no complications during the postoperative follow-up. The patient who had no previous hypertension in pre operational period had stage 2 hypertension in early postoperative period and need to use beta blocker (metoprolol 50mg twice a day). There was no need to use antihypertensive drug in long term follow up (after 6 month).

Primary percutaneous coronary intervention was planned but after coronary angiography because of the risk of coronary vascular rupture, patient underwent emergency coronary

bypass surgery and coarctation operation. After the hospital postoperative period; when the patient was discharged, the patient was evaluated by the reumathologist. But there was no rheumatological disease.

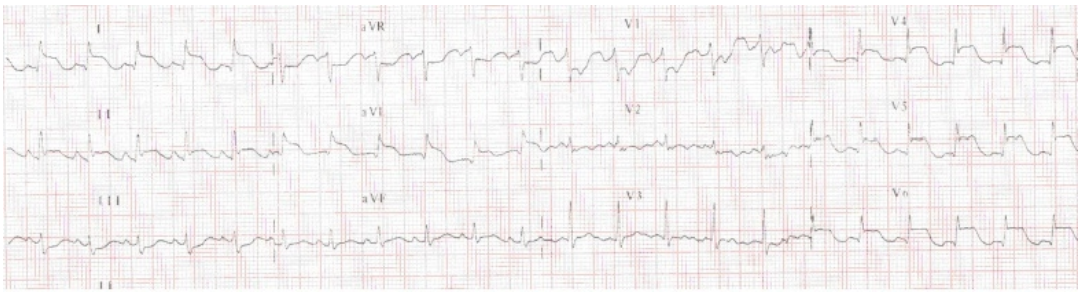

Fig. 1. Electrocardiography showed sinus tachycardia and ST-segment elevation in leads I, a VL (both 2 mm) and V4–V6 (all 3mm), along with ST segment depression in leads III (1mm) and V1 (3mm), suggesting that an acute ST-segment elevation myocardial infarction

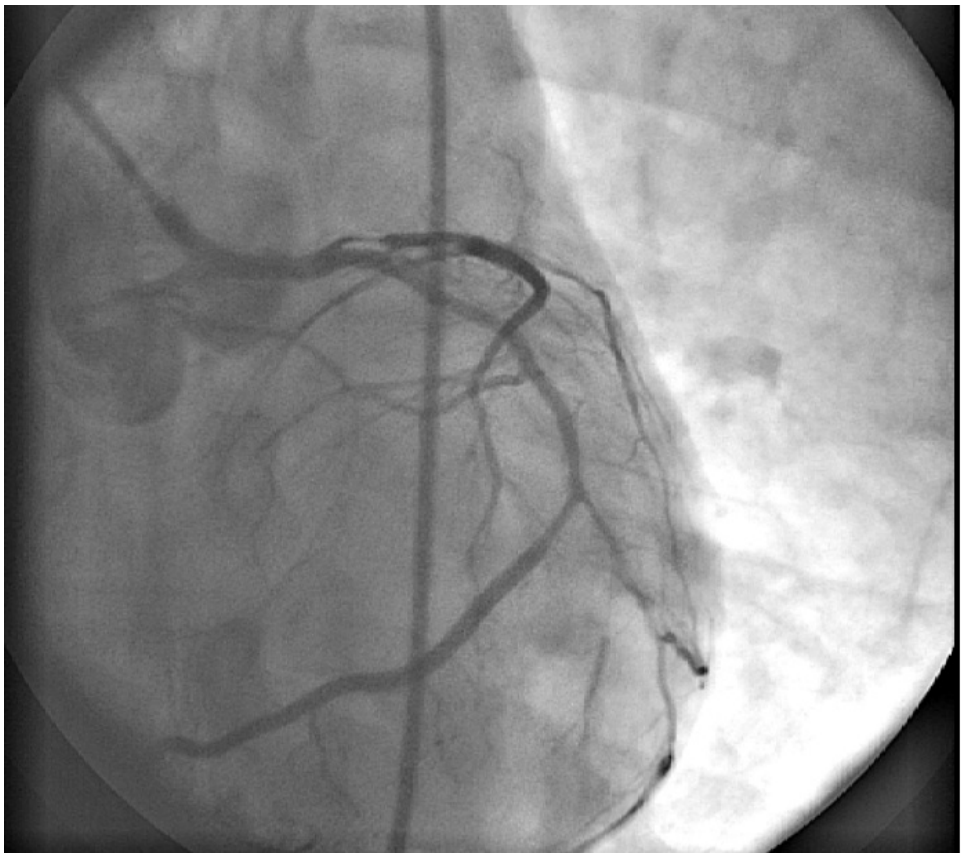

Fig. 2. Coronary angiography image of left anterior descending artery. Intraoperatively coronary dissection was confirmed

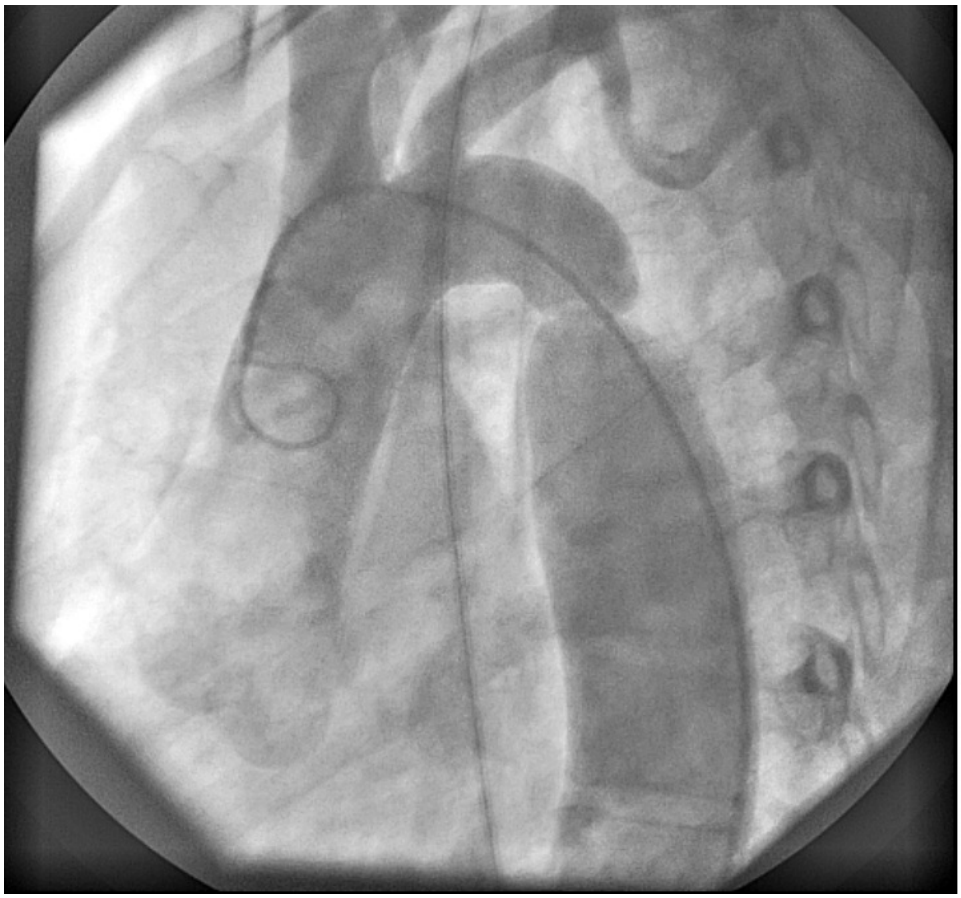

Fig. 3. Aortography of the patient illustrating coarctation of the aorta

3. DISCUSSION

The exact mechanism of SCAD has not been fully elucidated, but various pathogenetic mechanisms have been proposed. SCAD may occur primary (mostly and idiopathic) or secondary (rarely, traumatic) [4]. Possible mechanisms of SCAD include rupture of atherosclerotic plaque or vasa vasorum, hemorrhage between the outer media and external lamina with intramedial hematoma expansion, and compression of the vessel lumen [4]. In our patient, SCAD was diagnosed by angiography and then confirmed during coronary artery bypass surgery (there was a hematoma formation and swelling on the vessel wall in operation area). On the other hand, Robinowitz et al. [5] found eosinophilic infiltrates in the adventitia of 43% of the patients during postmortem examination. This situation also has to be evaluated in future studies.

Coronary angiography is gold standard for SCAD diagnosis. Management includes medical therapy and revascularization procedures using percutaneous intervention and coronary artery bypass grafting [4]. In our patient, due to two critical vessel disease and aortic coarctation, patient underwent emergency surgery.

In SCAD patients, the left coronary artery is involved in 66–75% of the cases in women, whereas the right coronary artery is predominantly involved in men. On the other hand in a recent review of coronary dissection, gender difference of LAD and right coronary is denied [4]. Our patient's SCAD lesion was in *LAD*.

SCAD is more common in women than in men [6]. Pregnancy and the early puerperium have been recognized as predisposing factors for SCAD. In pregnancy, physiological and hormonal changes may cause medial dissection of the coronary artery on account of impairment of collagen synthesis, proliferation of myocytes, and alteration in the mucopolysaccharide and protein content of the media. Furthermore; in pregnancy, the increased blood volume and cardiac output are thought to facilitate intimal rupture and medial dissection.

Yalonetsky S et al. [7] found that there was a evidence of systemic endothelial dysfunction in patient with cyanotic congenital heart disease and endothelial injury was a potential trigger for atherosclerosis.

Although aortic coarctation is a congenital heart defect, it is frequently diagnosed in adulthood because patients can remain symptom-free for many years. Coarctation is defined as a narrowing of the lumen of the aorta that obstructs flow. Typically, it is located at the insertion of the ductus or ligamentum arteriosum. The coarctation segment reveals an intimal and medial lesion consisting of ridges that protrude into the aortic lumen. The ductus or ligamentum arteriosus inserts at the same level anteromedially [8].

It is known that mostly patients with aortic coarctation develop persistent systemic hypertension and are at risk for premature coronary artery disease. Coarctation of the aorta causes some hemodynamic changes in proximal aorta, left ventricle [9]. In our patient; Coarctation of the aorta with 45mmHg gradient distal to the subclavian artery was detected on echocardiography and aortography. This gradient probably generated a turbulent flow. At this point; is it possible to say that hemodynamic changes had an effect on coronary artery dissection. Further studies in this field can explain this situation.

Coarctation can be explained by 2 theories; the hemodynamic theory and the ductus tissue theory. In the hemodynamic theory, coarctation results from reduced blood flow through the fetal aortic arch and isthmus. In the ductus tissue theory, coarctation develops as the result of migration of ductus smooth muscle cells into the periductal aorta, with constriction and narrowing of the aortic lumen. According to this theory, loose intimal and medial layers might contribute the formation of coarctation during coarctation development. From this aspect; in our patient, It may be speculated that loose vascular structure that cause aortic coarctation would have been the reason of coronary artery dissection. We did not prove a direct significant relationship between these two entities. It is possible to say some blood flow changes may have contributed to the development of coronary dissection.

Coronary artery dissection is rare and mostly diagnosed at necroscopy. Despite SCAD is a serious condition with high risk of death, there are no standardized management plans. The diagnosis of SCAD must be quick and CABG is a valid opportunity. Ferrari et al reported a case of spontaneous coronary artery dissection in a young woman with coronary artery bypass and heart transplantation [10]. In our patient, emergency coronary bypass surgery was done.

In a meta-analysis by Canto et al. [11] hypertension (52,3%), smoking (31,3%), dyslipidemia (28%), family history(28%) and diabetes (22,4%) were the the risk factors among patients with initial myocardial infarction patients. In our patient there is no risk factor including hypertension, smoking, dyslipidemia, family history and diabetes.

4. CONCLUSION

Spontaneous coronary artery dissection (SCAD) is a rare cause of acute coronary syndrome, cardiogenic shock, and sudden cardiac death in women of reproductive age who have no traditional risk factors for coronary artery disease; physicians must keep in mind acute coronary syndrome and coronary artery dissection. In these patients, there can be underlying vascular or hemodynamic problems.

CONSENT

All authors declare that 'written informed consent was obtained from the patient (or other approved parties) for publication of this case report and accompanying images.

ETHICAL APPROVAL

All authors hereby declare that "Principles of laboratory animal care" (NIH publication No. 85-23, revised 1985) were followed, as well as specific national laws where applicable. All experiments have been examined and approved by the appropriate ethics committee.

All authors hereby declare that all experiments have been examined and approved by the appropriate ethics committee and have therefore been performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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