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A Case of Chronic Aortic Dissection with Medial Calcification

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Authors' contributions

This work was carried out in collaboration between all authors. Authors SK and SIO designed the study. Author SK wrote the first draft of the manuscript and author YM managed the literature searches. All authors read and approved the final manuscript.

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Case Report

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ABSTRACT

Aims: Cystic medial necrosis is the most common pathological change of dissection. Medial calcification is mainly recognized in peripheral arteries and arterioles, but it can occur in the aorta. We, herein, report a case with chronic aortic dissection associated with medial calcification.
Presentation of Case: A 67-year-old woman presented with chest discomfort. Chest computed tomography showed a dilated ascending aorta associated with chronic aortic dissection, and medial and intimal calcification in the descending aorta. The aortic root and ascending aorta were replaced with a valved conduit. The postoperative course was uneventful. Pathological examination of the resected aorta showed cystic necrosis and calcification in the media.
Discussion and Conclusion: We cannot define that medial calcification was a secondary change after dissection in our case, but we think that the calcification of the media could lead to weakening of medial function, causing arterial or aortic distension and/or dissection.

Keywords: Aortic dissection; medial calcification; atherosclerosis; Mönckeberg medial sclerosis.

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ABBREVIATION

CT : Computed Tomography.

1. INTRODUCTION

Causes of acute aortic dissection include degeneration of the media, trauma, and others. Cystic medial necrosis is the most common pathological change of dissection. Medial calcification is mainly recognized in peripheral arteries and arterioles. However, medial calcification can occur in the aorta. We experienced a chronic case of aortic dissection with aortic medial calcification.

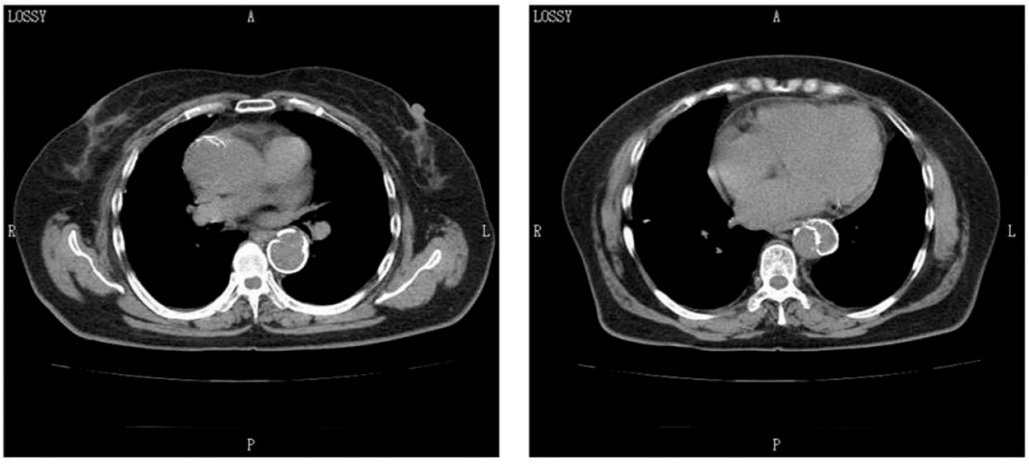
2. PRESENTATION OF CASE

A 67-year-old woman presented with sudden chest discomfort which was lasting for several hours. Her blood pressure was 164/92 with a regular rhythm. Electrocardiogram revealed a normal sinus rhythm without ischemic changes. A blood examination showed a total cholesterol level of 378 mg/dl and low-density lipoprotein cholesterol level of 274 mg/dl. The thyroid-stimulating hormone level was 109.3 μU/ml. Free thyroxine 3 and free thyroxine 4 levels were undetectable. The HbA1c level was 6.5% and renal function was normal. Chest computed tomography (CT) showed a dilated ascending aorta and sinotubular junction associated with chronic aortic dissection (Fig. 1-a). Medial and intimal calcification was clearly observed in the descending aorta (Fig. 1-b). An echocardiographic examination showed that left ventricular end-diastolic and end-systolic diameter was 47/27 mm and that the aortic valve was tricuspid associated with moderate regurgitation. A coronary angiogram showed a significant stenotic lesion at the orifice of the left main coronary artery.

The patient gave informed consent to undergo aortic root replacement and coronary artery bypass grafting. Right axillary artery and bicaval cannulation was chosen for cardiopulmonary bypass. After aortotomy, we confirmed that there were no entries at the ascending aorta and the aortic arch. The aortic root and ascending aorta were replaced with a valved conduit. This was followed by coronary artery bypass grafting to the left anterior descending and circumflex arteries under cardiac arrest. The postoperative course was uneventful. Pathological examination of the resected aorta showed cystic necrosis and calcification in the media (Figs. 2-a, 2-b).

3. DISCUSSION

Morphological changes of the aortic media are closely related to aortic dissection. Genetic disorders, such as Marfan syndrome, bicuspid aortic valve, and atherosclerotic diseases, could cause these changes. Aortic calcification is often recognized among an aged population and patients with diabetes mellitus and/or renal dysfunction. Most lesions of aortic calcification are associated with atherosclerotic changes of the intima.



1-a 1-b

Fig. 1. Preoperative chest CT findings
1-a) The ascending aorta is enlarged with calcification
1-b) The false lumen of the descending aorta is surrounded by a calcified wall, indicating calcification in the media and intima

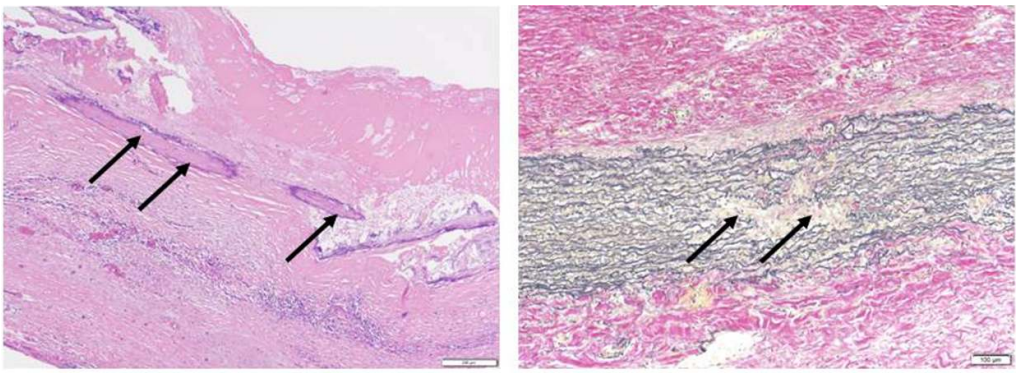


Fig. 2. Histologic findings
2-a) Hematoxylin-eosin stain. Calcification can be seen in the media (black arrows)
2-b) Elastica van Gieson stain. Cystic medial necrosis can be seen (black arrows)

Lanzer and colleagues stated that vascular calcification processes associated with atheroma formation may be triggered by specific biochemical cascades. These cascades are different from those initiated by primary damage to elastic fibers that occur in medial calcification [1]. Tsushima also described that medial calcification could be formed under molecular regulatory control, such as in bone by differentiated osteoblasts or chondroblasts from pericyte-like cells originating from smooth muscle cells [2]. Additionally, many substances, such as osteopontin, osteocalcin, and bone morphogenetic protein 2, were found in the calcified area. The author also observed degenerated elastin and macrophage-originated calcification in intimal calcification [2].

Medial calcification is mainly recognized in peripheral arteries and arterioles, as shown by Mönckeberg and colleagues, who referred to calcification of the media in the arteries of the extremities in 1903 [3]. They initially stated that only the media is involved. Patients with chronic kidney disease and diabetic patients tend to present with medial calcification. However, in 2008, Micheletti and colleagues indicated that Mönckeberg sclerosis involves the internal elastic lamina and media with calcification [4]. Fishbein and colleagues reviewed articles related to arteriosclerosis and vascular calcification, and they considered that calcified lesions actually begin in the internal elastic lamina, and then grow and extend into the media [5].

Hachiya and colleagues reported that calcification of the false lumen among 110 patients with chronic aortic dissection was observed in 12 patients by chest CT images [6]. They also described that an endothelialized false lumen might develop atheromatous changes much more rapidly than a true lumen. Secondary degenerative changes in the false lumen after dissection might have led to calcification in their cases. We cannot define that the medial calcification was a secondary change after dissection in our case. However, calcification was observed in the media of the ascending aorta, and medial calcification was observed in the whole thoracic aorta on chest CT. In our case, the medial calcification with cystic medial necrosis might have led to aortic dissection.

4. CONCLUSION

Calcification of the media reflects morphological changes, which could lead to weakening of medial function. As either primary or secondary changes, calcification in the media might lower its vascular structural strength, causing arterial or aortic distension and/or dissection.

ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Lanzer P, Boehm M, Sorribas V, Thiriet M, Janzen J, Zeller T, et al. Medial vascular calcification revisited: Review and perspectives. Eur Heart J. 2014;35:1515-25.

2. Tsushima M. Aortic calcification and calcium. Clin Calcium. 2010;20:1627-35.
3. Mönckeberg JG. Über die reine mediaverkalkung der extremitätenarterien und ihr Verhalten zur Arteriosklerose. Virchows Arch Pathol Anat. 1903;171: 141-67.
4. Micheletti RG, Fishbein GA, Currier JS, Fishbein MC. Mönckeberg sclerosis revisited: A clarification of the histologic definition of Mönckeberg sclerosis. Arch Pathol Lab Med 2008;132:43-7.
5. Fishbein MC, Fishbein GA. Arteriosclerosis: Facts and fancy. Cardiovasc Pathol. 2015; 5. DOI: 10.1016/j.carpath.2015.07.007
6. Hachiya J, Nitatori T, Okada M, Hanaoka H, Yoshino A, Furuya Y. Calcified false channel wall in aortic dissection. Nippon Acta Radiologica. 1994;54:258-63.

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