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Association between myocardial hypertrophy and apical diverticulum: more than a coincidence?

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PURPOSE
We aimed to investigate the possible association between the myocardial hypertrophy and the development of an apical diverticulum.

MATERIALS AND METHODS
We retrospectively reviewed 786 multidetector computed tomography (MDCT) coronary angiography examinations (520 males, 266 females; mean age, 57±15 years; age range, 18–78 years). The end-diastolic left ventricle wall thickness was measured in all patients, and a wall thickness of 11 mm was determined to be the cut-off value for myocardial hypertrophy. The ventricular apex and subvalvular area were evaluated for ventricular diverticula. The difference between the apical diverticula in patients with and without myocardial hypertrophy was determined.

RESULTS
There were 12 myocardial hypertrophy and nine apical diverticulum cases. Myocardial hypertrophy was observed in four (44%) of nine patients who had apical diverticula, and an apical diverticulum was observed in four (33%) of 12 patients who had myocardial hypertrophy. There was statistically significant difference for myocardial wall thickness between the apical diverticula in patients with myocardial hypertrophy and those without myocardial hypertrophy ($P = 0.011$).

CONCLUSION
Diagnosis of apical diverticula has become easier by using imaging modalities such as MDCT. There may be an association between myocardial hypertrophy and apical diverticulum.

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Apical diverticula are rare cardiac abnormalities consisting of a localized outpouching from the apex of the left ventricle. They are primarily congenital, occurring in isolation or in association with other abnormalities. Noncongenital cases are also seen in an isolated form in adults. A few cases have been described in which there is apparently an association between myocardial hypertrophy and apical diverticula (1–4).

Recently, multidetector computed tomography (MDCT) has become a major diagnostic tool in cardiac imaging, showing both the coronary arteries and other cardiac and extracardiac structures, such as the myocardium, cardiac chambers, major vessels, and mediastinum (5). In this study, we investigated the association between myocardial hypertrophy and the development of apical diverticula.

Materials and methods

Patient selection

We retrospectively reviewed 786 MDCT coronary angiography examinations performed in our department between October 2009 and May 2012, for the presence of apical diverticula.

The institutional ethics committee approved this retrospective study (No: 2012-77 GATA Haydarpaşa Teaching Hospital, İstanbul, Turkey). Written informed consent was obtained from all patients. Overall, there were 520 men and 266 women, with ages ranging from 18 to 78 years (mean, 57±15 years). The indications for MDCT coronary angiography were suspected coronary artery disease (n=681), coronary artery anomaly (n=36), coronary artery stent patency (n=27), and coronary artery bypass graft assessment (n=42).

Imaging

The MDCT coronary angiography examinations were performed using a 64-MDCT scanner (Brilliance-64, Phillips Medical Systems, Eindhoven, The Netherlands, or LightSpeed VCT, GE Healthcare, Milwaukee, USA). A volume data set was acquired from the level of the carina through the diaphragm using thin collimation and ECG gating. Image acquisition was performed after the intravenous injection of 50–100 mL of 350–400 mg I/mL of nonionic contrast material (iohexol, Omnipaque 350, GE Healthcare, Cork, Ireland; or iopromide, Iomeron 400, Bracco, Milano, Italy) administered at a rate of 6 mL/s via a power injector. The volumetric data set was reconstructed using retrospective ECG gating techniques at 5% phase intervals throughout the cardiac cycle, to include the systolic and diastolic phases. The images were reviewed on a three-dimensional workstation equipped with multiplanar and max-

imum intensity projection reformations to visualize the heart along the cardiac axes.

Image analysis

The diagnoses of apical diverticula were made in horizontal long axis views by two radiologists by consensus. After detection of the patients with apical diverticula, the patients' age, gender, septum and left ventricle wall thickness at the thickest segment, and diverticulum size in systole and diastole were recorded. Apical diverticula secondary to apical infarction were excluded. All MDCT examinations were reviewed for myocardial hypertrophy, with a cut-off thickness of 11 mm (6). Both focal and diffuse myocardial hypertrophy patients were included. Wall thickness measurements were performed manually in the end-diastolic phase to avoid misdiagnosis of hypertrophy in the systolic phase. Short axis images and the thickest wall were used for the myocardial thickness measurements.

To distinguish an apical diverticulum from an apical thin point, we used the following criteria: narrow neck, equal or greater maximal diameter when compared with the diverticulum neck, and outpouching of the diverticulum to the subepicardial fat, if present.

Statistical analysis

The defining statistics related to the characteristics of interest were expressed as mean, median, standard deviation, and minimum and maximum values. The Mann-Whitney U test, a nonparametric statistical analysis test, was used because of the small number of the patients with apical diverticulum and myocardial hypertrophy. Statistical significance was interpreted when *P* values were below 0.05. All statistical analyses were performed using a commercially available software (Statistical Package for Social Sciences, version 15.0, SPSS Inc., Chicago, Illinois, USA).

Results

Apical diverticula were observed in nine patients (1.14%) (Fig. 1). No left ventricular diverticulum near the subvalvular area was observed. Myocardial hypertrophy was recorded in 12 cases (1.52%). The median left ventricle wall thickness in patients without myocardial hypertrophy was 9 mm (range, 8–11 mm). Diffuse myocardial hypertrophy was observed in nine patients (9/12, 75%), and septal myocardial hypertrophy was observed in three patients (3/12, 25%). The prevalences of diffuse myocardial hypertrophy and septal myocardial hypertrophy were 1.14% and 0.38%, respectively, in all groups.

Apical diverticula were observed in four of 12 (33%) myocardial hypertrophy patients (Fig. 2). The median myocardial wall thickness at the thickest segment in the short axis was 18.5 mm (range, 16–34 mm). While the median myocardial wall thickness was 16 mm (range, 16–23 mm) in the myocardial hypertrophy patients with apical diverticula, it was 20.5 mm (range, 16–34 mm) in the myocardial hypertrophy patients without apical diverticula. There was not significant difference for myocardial wall thickness between patients who have both apical diverticulum and myocardial hypertrophy and patients who have only myocardial hypertrophy ($P = 0.165$).

Myocardial hypertrophy was observed in four of nine (44%) apical diverticula patients (Fig. 3). There was statistically significant difference for myocardial wall thickness between the apical diverticula patients with myocardial hypertrophy and those without myocardial hypertrophy ($P = 0.011$). While the median myocardial wall thickness in patients with both apical diverticulum and myocardial hypertrophy was 16 mm (range, 16–23 mm), the median wall thickness in the apical diverticula patients with-

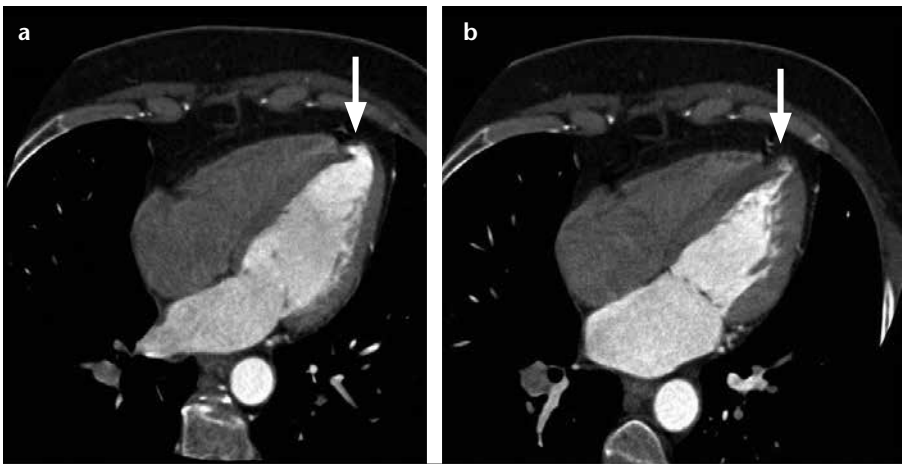

Figure 1. a, b. Four-chamber views of a MDCT coronary angiography in diastole **(a)** and systole **(b)** show an apical left ventricular diverticulum in diastole in a patient without myocardial hypertrophy (**a,** *arrow*). In systole, the apical diverticulum has almost completely contracted (**b,** *arrow*). Note the normal myocardial wall thickness.

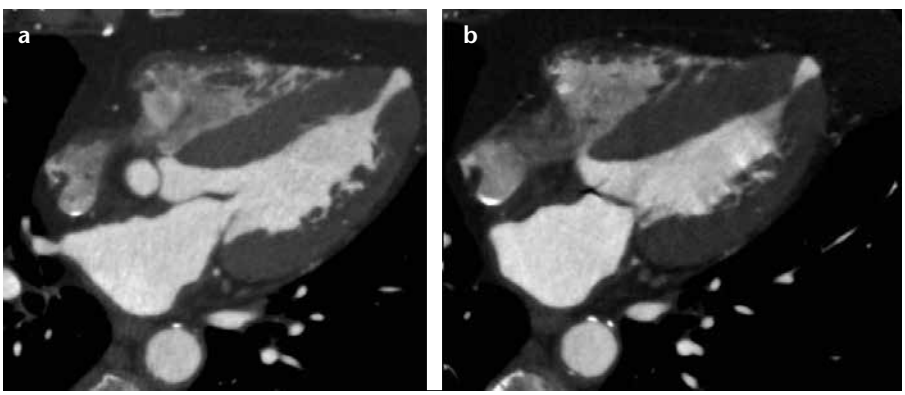

Figure 2. a, b. Reconstructed four-chamber views of a MDCT coronary angiography in diastole **(a)** and in systole **(b)** showing an apical left ventricular diverticulum with a narrow neck in diastole in a patient with myocardial hypertrophy. The neck is almost completely obliterated in systole due to muscular contraction. Myocardial hypertrophy is also evident.

out myocardial hypertrophy was 10 mm (range, 9–11 mm). The median myocardial wall thickness was 11 mm (range, 9–23 mm) in all apical diverticula patients. The apical thin point was almost always observed at the cardiac apex; there was no narrow neck or outpouching when both the systolic and diastolic images were evaluated.

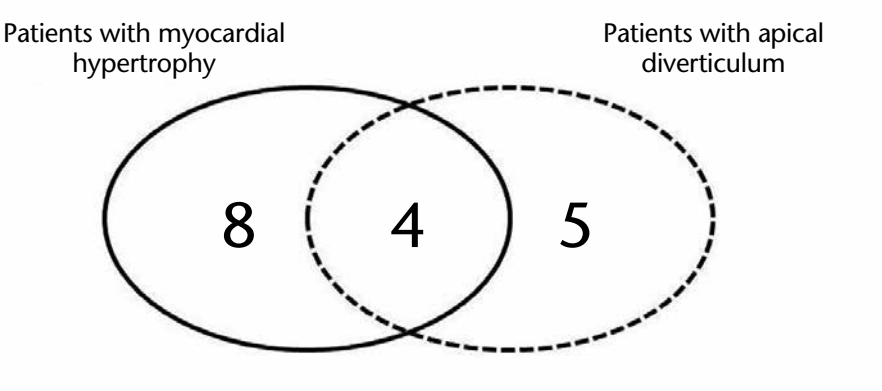

Figure 3. The numbers of patients with myocardial hypertrophy and apical diverticulum. Four patients had both pathologies concurrently.

Discussion

Left ventricular diverticulum is a rare cardiac malformation characterized by a localized outpouching from the cardiac chamber. A few pathologies mimic a ventricular diverticulum. Myocardial clefts or crypts and aneurysms can be differentiated from left ventricular diverticulum. Congenital myocardial clefts or fissures are commonly observed in the basal inferior wall of the left ventricle and the mid-to-apical segments of the interventricular septum. Both clefts and diverticula may contract with systole, but diverticula typically have a narrow neck with a wide outpouching, extending beyond the left ventricular cavity and myocardial margin. Clefts, in contrast, are fissure-like protrusions confined to compacted myocardium that are contractile and may obliterate during systole (7). Some myocardial clefts involve the full thickness of the interventricular septum and are detected only at diastole. Aneurysms of the ventricular wall secondary to myocardial infarction usually have a wide neck and thin wall and may exhibit no contraction with—and conversely characteristic dyskinesis during—systole (8). Another differential diagnosis is pseudoaneurysm, which is a form of myocardial rupture contained by the pericardium and typically the result of infarction, trauma, or tuberculosis. It does not contain all three layers of wall. Isolated ventricular noncompaction is another entity that needs to be differentiated. In cases of isolated ventricular noncompaction, endomyocardial morphogenesis is impaired and hypertrophy of the left ventricular myocardium with prominent trabeculation and deep intertrabecular recesses occurs. Although ventricular noncompaction can mimic a ventricular diverticulum, diagnosis of the latter usually requires the presence of more than three deep intertrabecular recesses (9).

According to the location, left ventricular diverticula can take apical and nonapical forms. The nonapical form is generally located in the subvalvular area. Two left ventricular diverticula phenotypes exist. Cantrell's syndrome is the most common type, accounting for 70% of cases. This syndrome is congenital, is usually detected in children with a pentalogy of midline thoracoabdominal defects, and presents as pericardial effusion, shock, or cardiac arrest, caused by acute rupture (10). The remaining cases are the isolated form, observed in adults who have no additional congenital malformation. The isolated form typically presents as a finding incidental to arrhythmia, systemic thromboembolism, congestive cardiac failure, acute chest pain, tamponade secondary to acute rupture, or sudden death (11).

In a cardiac catheterization study, the incidence of a left ventricular diverticulum was 0.26% (12). Another large retrospective echocardiographic study showed a prevalence of a left ventricular diverticulum of 0.04% (13). In our study, the prevalence of left ventricular apical diverticulum was 1.14%, higher than that reported previously. This higher prevalence may be associated with a higher number of heart screenings with MDCT coronary angiography than before.

We focused on apical diverticula of the left ventricle and did not encounter a nonapical diverticulum. Myocardial hypertrophy was observed in 44% of patients who had apical diverticula, and apical diverticulum was observed in 33% of patients who had myocardial hypertrophy. Thus, it seems that there may be an association between myocardial hypertrophy and apical diverticulum in adults. In a study by Bradfield et al. (14), the wall thickness of the left ventricle apex was measured at its thinnest site, and the authors concluded that there is always one point at which the myocardial thickness is 3 mm or less. This region was called the apical thin point (Fig. 4). Electrophysiological studies have shown that the apical region of the left ventricle is stimulated before the basal regions. Thus, the apical contraction would close off the thin point early in systole, protecting it from the higher pressures generated during ventricular systole (15, 16). We hypothesized that the apical thin point may be protected from the high intracavitary pressure during ventricular systole and is not able to protect patients with myocardial hypertrophy. Additionally, the high pressure at the ventricular apex may cause development of an apical diverticulum. Some case reports in the literature support this hypothesis (1–4).

In addition to classification by location, left ventricular diverticula are classified according to whether their content is muscular or fibrotic (17). Muscular ventricular diverticula show synchronous contraction with the ventricle during ventricular systole. Fibrous ventricular diverticula often show akinetic or dyskinetic contractile function during ventricular systole. Histologically, while muscular diverticula contain all layers of the ventricular myocardium, with preserved

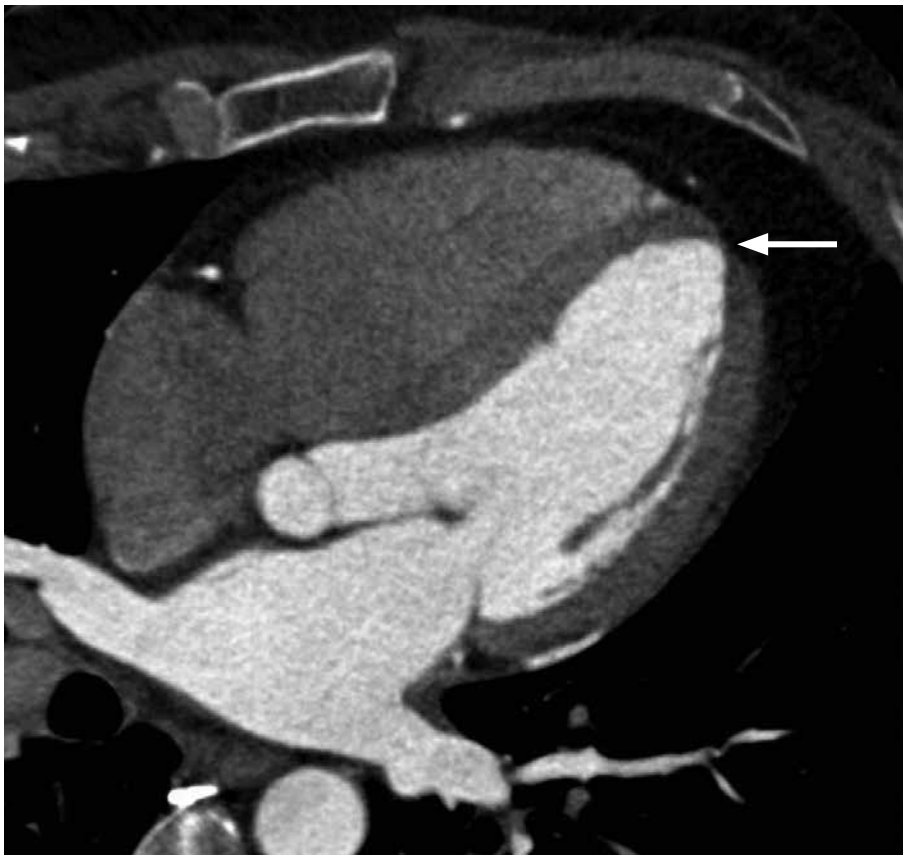

Figure 4. Four-chamber view of a MDCT coronary angiography shows how thin the left ventricular myocardium is at the apex *(arrow)*. Here is a known apical thin point.

myocardial architecture and minimal fibrous tissue, fibrous diverticula typically have connective tissue, composed of reticulin fibers; few or no muscle fibers are present (18). Fibrous diverticula with a narrow neck are commonly located in the subvalvular area and are often associated with valvular incompetence (19). A fibrous left ventricular diverticulum is associated with a higher probability of complications, such as systemic thromboembolism or rupture, than the muscular type (12).

On a plain chest radiogram, deviation of the heart border, abnormal cardiac contour, and cardiomegaly may be observed, depending on the size of the diverticulum. Apical diverticula may also be observed during angiographic studies. Transthoracic echocardiography and transesophageal echocardiography are commonly used to diagnose ventricular diverticula. MDCT and magnetic resonance imaging (MRI) better depict the location and morphology of the diverticulum. Although we did not compare methods of imaging ventricular diverticula in this study, the high spatial and temporal resolution offered by ECG-gated MDCT coronary angiography provides a unique opportunity to evaluate ventricular diverticula.

Our study has some limitations. First, the cases were identified by a retrospective review of images from MDCT coronary angiographic studies, and we do not know whether the asymptomatic population has diverticula. Thus, our prevalence does not reflect that of the general population. Second, no prospective imaging software is available. Thus, we used retrospective evaluation of MDCT coronary angiography images. This imaging method increases the radiation doses received by the patients. Third, we performed the study in an adult group and examined no pediatric patients. Thus, congenital ventricular diverticula were not evaluated. Fourth, MDCT coronary angiography was the only diagnostic tool, and other modalities, such as angiography, echography, and MRI were not performed. Therefore, no comparison of the modalities could be conducted. Fifth, no data for nonapical diverticula were collected because no case of this type was identified in our patient population. Finally, because no histological assessment was performed, the fibrous and muscular types of diverticulum were differentiated based only on MDCT coronary angiography findings.

In conclusion, our data suggest that the prevalence of left ventricular diverticula was higher than reported previously. Diagnosis of apical diverticula has become easier using imaging modalities such as MDCT. Additionally, our findings suggest an association between myocardial hypertrophy and apical diverticulum.

Conflict of interest disclosure

The authors declared no conflicts of interest.

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