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Case Report

Incidental findings of acute myocardial infarction detected during ECG-gated and nongated thoracic CTA: A report of four cases ☆

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ABSTRACT

Worldwide, myocardial infarction is a leading cause of mortality and disability. The phrase "myocardial infarction" refers to ischemia, which is the outcome of an imbalance in perfusion between supply and demand and results in the death of cardiac myocytes Myocardial ischemia is often diagnosed based on the patient's medical history and electrocardiogram (ECG) findings. Potential ischemic symptoms include a variety of chest, upper extremity, jaw, or epigastric pain or discomfort that typically lasts at least 20 minutes, is diffuse, not positional, not localized, not dependent on movement of the area, and may be accompanied by syncope, dyspnea, or nausea. These symptoms can occur at rest or after physical activity. These symptoms may be mistaken for other conditions since they are not specific to myocardial ischemia.

Radiologists play a crucial role in this scenario since imaging is increasingly being used to identify and categorize these individuals. We report 4 cases of myocardial infarction presenting without chest pain and discovered incidentally during imaging tests.

Introduction

Receiving the optimal care for all patients with acute coronary syndrome is significantly hampered by diagnostic delay. Chest symptoms are often missed in an unusual presentation. The use of imaging tests for evaluation of these patients, such as chest computed tomography (CT) and aortic CT angiography (CTA), has considerably increased.

Radiologists play a crucial role in this situation. The heart and coronary arteries must be taken into account while interpreting thoracic and abdominal CTA since these tests can

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detect the early signs of acute myocardial infarction and occlusive coronary artery lesions.

Case 1

A 77-year-old woman presented to the emergency department with epigastric pain that started 4 days prior to presentation and was constant at rest. The day before she was admitted, the pain got worse and spread to her back. There were no symptoms of deep vein thrombosis, dyspnea, or related chest pain, according to her report. Although this was the first time she had this kind of pain, she was at high risk for cardiovascular disease due to her history of smoking, hypertension and dyslipidemia. Moreover, an abdominal aortic aneurysm rupture that required aorto-iliac bypass and an intraventricular lipoma was a part of her medical history.

Her vital signs and the outcomes of her physical cardiac and pulmonary exams were also normal. During admission to the emergency room, aortic computed tomography angiography (CTA), normal laboratory tests, and electrocardiography (ECG) were requested in order to exclude myocardial infarction or aortic dissection. The ECG showed only mild, nonspecific alterations without Q waves or ST elevations. During the ECG, the patient's heartbeat was 83 beats per minute. The results of laboratory tests showed increased troponin I (4.2 g/L) and D-dimer (1548 g/L) levels.

Aortic CTA was performed with ECG gating using a multislice computed tomography (MSCT) scanner (Revolution CT GE Healthcare 256) after intravenous injection of 70 mL of iodinated contrast agent (Iomeprolo 400 mgI/mL) at flow rate of 5 mL/s using the bolus tracking technique triggered on the ascending aorta.

While CT angiography did not show any signs of dissection, it did, incidentally, show an abrupt interruption of opacification in the right coronary artery (RCA) about 1 cm from the ostium with endoluminal hypodensity that was suggestive of a total occlusion by a likely thrombus despite the presence of some artifacts from heart motion (high frequency rate). The endoluminal hypodensity, representative of a nonopacified occlusive lesion, extended from the proximal RCA to IVP and PLB. Moreover, gated CTA depicted myocardial hypoattenuation in the RCA territory, suggestive of severe ischemia or infarction (Fig. 1).

After that, she was transferred to the cardiac catheterization room where a traditional coronary angiography was done, which confirmed RCA occlusion. Three sequential stents were placed on the proximal and mid sections of the RCA.

Case 2

A 56-year-old woman presented at our Emergency Department transported by ambulance due to homely cardiopulmonary arrest. The patient was intubated and subjected to a continuous infusion of norepinephrine for cardiocirculatory shock and acute respiratory failure.

The husband reported the onset of his wife's symptoms about 20 days before when the woman began to complain of asthenia and chest pain, which progressively increased in the following days.

A chest CT was ordered to rule out cardiopulmonary disease. The baseline CT scan showed massive pericardial effu-

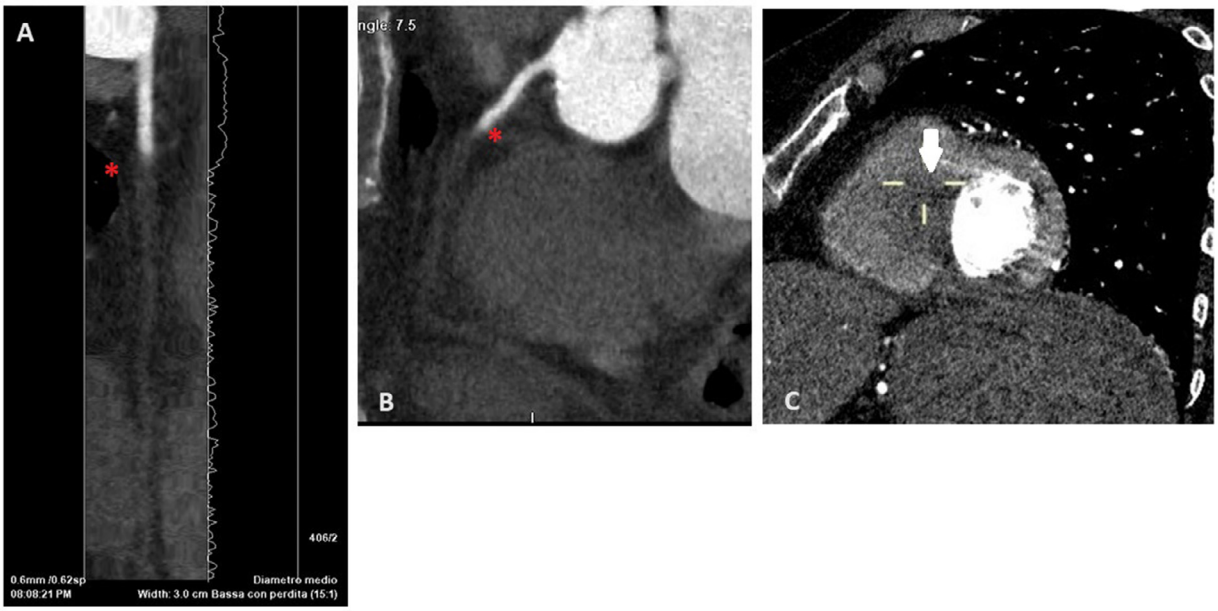

Fig. 1 – Case 1. (A and B) ECG gated thoracic CT, lumen and curved reconstruction show abrupt interruption of opacification in the right coronary artery (RCA) suggestive of total occlusion (red asterisk). (C) Cardiac CT, short axis view shows subendocardial hypodensity in the septal-inferior and inferior segments of the mid-apical myocardium, territory of RCA, suggestive of severe ischemia (white arrow).

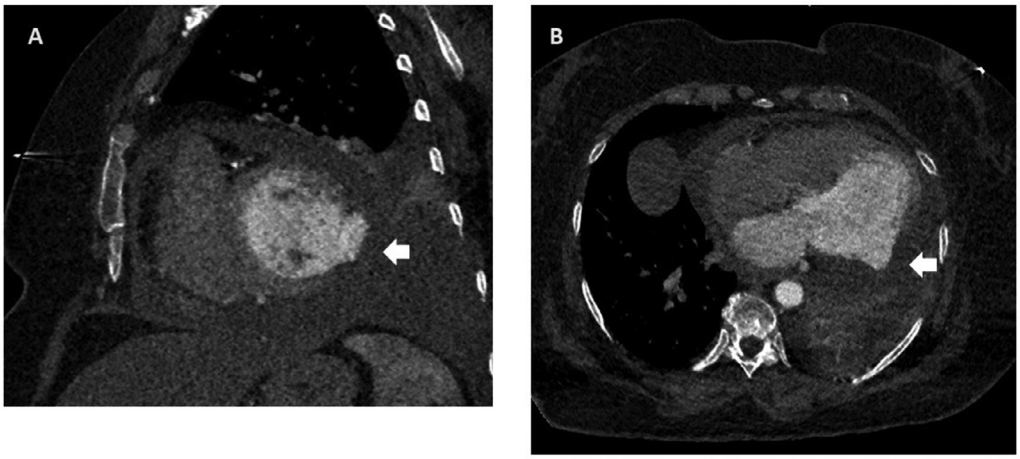

Fig. 2 – Case 2. (A and B) ECG gated thoracic CT, short axis and 4 chamber view show absent visualization of the lateral wall of the left ventricle with evidence of a pseudoaneurysmatic formation due to myocardial rupture contained locally by pericardial adhesions (white arrows).

sion, distributed over all the cardiac chambers, most conspicuously in correspondence with the mid-basal ventricular wall, and bilateral pleural effusion with atelectasis of the adjacent lung parenchyma.

Aortic ECG gating CTA was requested in order to exclude acute aortic syndromes. The scan was performed using a MSCT scanner (Revolution CT GE Healthcare 256) after intravenous injection of 70 mL of iodinated contrast agent (Iomeprolo 400 mgI/mL) at flow rate of 5 mL/s using the bolus tracking technique triggered on the ascending aorta.

CT angiography showed absent visualization of the lateral wall of the left ventricle with the exception of the apical tract with associated evidence of a pseudoaneurysmatic formation consistent with cardiac rupture contained locally by pericardial adhesions (Fig. 2). Evaluation of the coronary arteries documented the presence of subocclusive pathology at the ostium of the circumflex coronary artery (Fig. 3). The patient was immediately taken to the operating room but she deceased in the next hours.

Case 3

A 73-year-old smoker, dyslipidemic man with family history of myocardial infarction, presented to the emergency room referring dyspnea and nausea started 4 hours earlier. The patient denied chest pain or symptoms referable to deep vein thrombosis. Patient's physical examination revealed normal vital signs with no evidence of cardiovascular or pulmonary abnormalities.

Laboratory tests, cardiac enzymes, electrocardiogram (ECG) and baseline chest CT were requested. The laboratory analyzes proved to be within the norm, including the troponin with a value of 0.008 μg/L. The ECG also proved to be within normal limits with nonspecific ST segment alterations. Baseline chest CT showed no signs of pneumonia.

An echocardiogram was requested since the patient continued to complain of dyspnea. The echocardiogram showed hypokinesia of the apical anterior wall with evidence of a hyperechoic formation at the apex, suspected of thrombus. Therefore, it was decided to request a gated ECG cardiac CT with the aim of exclude a coronary occlusion.

Coro-TC was performed with ECG gating using a multislice computed tomography (MSCT) scanner (Revolution CT GE Healthcare 256) after intravenous injection of 50 mL of iodinated contrast agent (Iomeprolo 400 mgI/mL) at flow rate of 5 mL/s using the bolus tracking technique triggered on the ascending aorta.

The CT examination confirmed the presence of thrombotic formation in the apical site and demonstrated the presence of subocclusive lipid plaque at the middle tract of the DA with concurrent subendocardial perfusion defect at the anteroseptal segments in the basal site. Late acquisition for evaluation of delayed enhancement (DE), performed 10minutes after the injection of a second bolus of 80 mL of iodinated contrast agent (Iomeprolo 400 mgI/mL) at flow rate of 2.5 mL/s, demonstrated a subendocardial area of DE in the anterolateral subendocardial site, compatible with myocardial ischemia (Fig. 4).

The patient was transferred to the angiography room where a coronary angiography confirmed the subocclusion in the middle segment of the DA. It was opted to perform coronary angioplasty using a mono-stent.

Case 4

A 67-year-old man, presented to the emergency department for epigastrial pain occurred a week earlier. The patient with a history of type II diabetes, COPD and arterial hypertension referred that it was not the first time he perceived this pain but that the previous ones were milder and lasted for a shorter time.

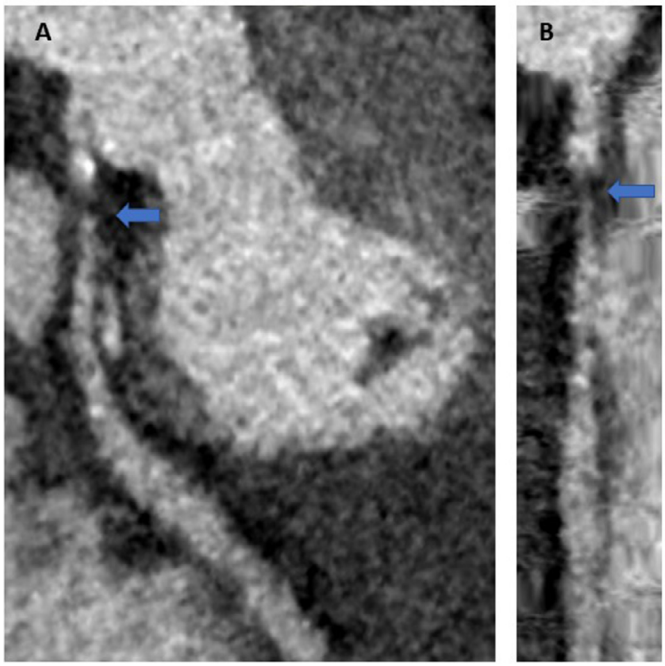

Fig. 3 – Case 2. ECG gated thoracic CT, lumen reconstruction shows abrupt interruption of opacification at the ostium of the circumflex coronary artery suggestive of total occlusion (blue arrows).

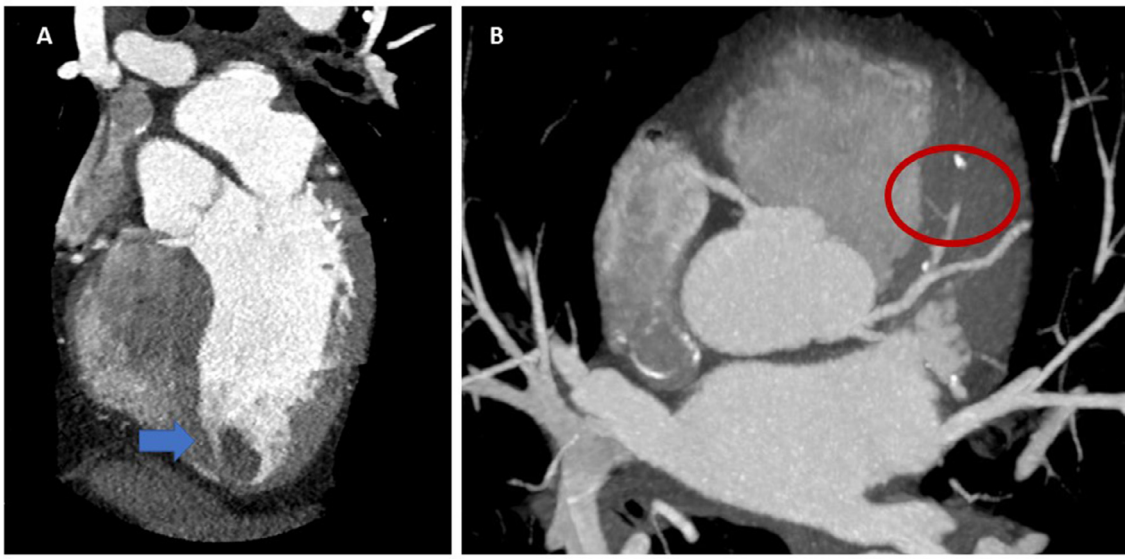

Fig. 4 – Case 3 (A) Cardiac CT, 3D MPR reconstruction showing the evidence of apical thrombus (blue arrow); (B) Cardiac CT, axial plane with maximum intensity projection (MIP); subocclusive type opacification defect of the middle segment of the DA (red circle).

Physical examination showed normal vital signs, with no evidence of cardiovascular or pulmonary abnormalities except for minimal tachycardia (103 bpm), stable for the next 3 measurements. Laboratory tests and cardiac enzymes showed a minimal, non-significant, rise in troponins levels with a value of 0.017 μg/L. ECG showed no alterations. In order to exclude gastrointestinal or lung disease a chest and abdomen CT scan were required.

TC was performed using a MSCT scanner (Revolution CT GE Healthcare 256) after intravenous injection of 100 mL of iodinated contrast agent (Iomeprolo 400 mgI/mL) at flow rate of 3.5 mL/s using the bolus tracking technique triggered on the descending aorta.

In the basal and portal phases, it was possible to note a mixed type plaque with a lipid core in the proximal portion of the DA. In the portal phase, when myocardium enhanced,

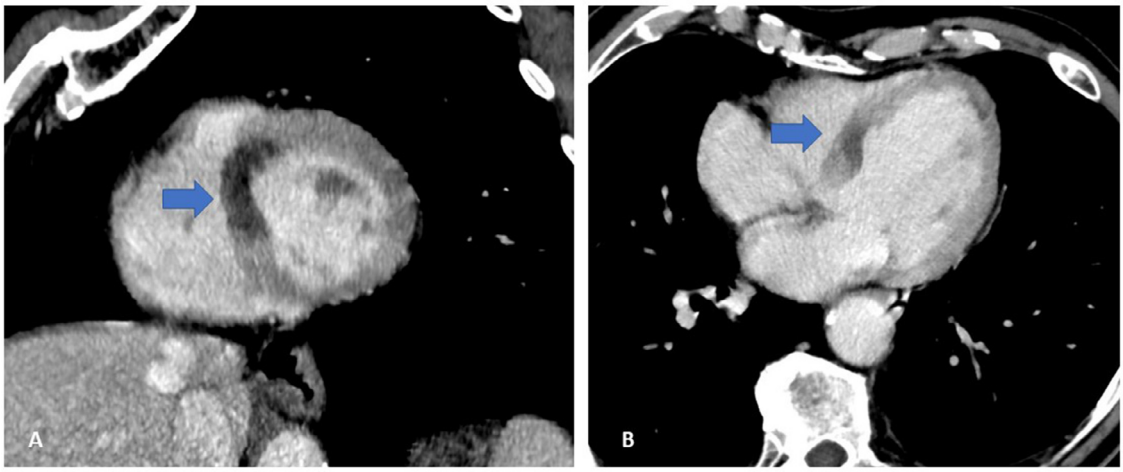

Fig. 5 – Case 4 (A and B) Not ECG-gated toraco-abdominal CT, portal phase, short axis and 4 chambers plane showing evidence of septal-anterior and basal portion of myocardium hypodensity (blue arrows).

it was possible to appreciate a subendocardial perfusion defect in the basal septal-anterior segments. Although not ECG-gated, the images were very clear (Fig. 5).

The patient was immediately taken to the angiography room where coronary angiography was performed which demonstrated NSTEMI in the area of vascular distribution of DA. An angioplasty using a balloon expander was performed and the patient was dismissed 3 days later.

Discussion

Diagnostic delay is a significant impediment to receiving the best care for all ACS patients [1,2].

Age, gender, race, and comorbidities have all been found to have an impact on how symptoms present themselves [3–5]. Although chest pain is the most typical ACS presenting symptom in patients of all ages, older patients report it less frequently than younger patients do [6–8].

Chest-related symptoms are frequently excluded in an unusual presentation [9]. A total of 33%-58% of all myocardial infarction (MI) presentations are made up of people who don't have chest pain [10–12]. Shortness of breath, sweating, back discomfort, exhaustion, and nausea are examples of unusual symptoms that make it challenging for persons experiencing them to identify their symptoms [10]. Practitioners also struggle to diagnose patients who present with unusual symptoms.

As imaging is increasingly used to diagnose and classify these patients, radiologists play a significant role in this scenario [13].

The use of chest CT and aortic CTA as imaging tests for the evaluation in the emergency room has significantly increased in recent decades. Because the investigation of cardiac diseases is often not the main purpose of these tests, radiologists tend to overlook the heart when interpreting nongated and gated thoracic CT [14,15].

Newer CT scanners should deliver more accurate and comprehensible heart and coronary pictures during thoracic CTA, even when employing nongated techniques, thanks to ongoing advancements in CT technology [16]. Since these evaluations can identify the initial signs of acute myocardial infarction and occlusive coronary artery lesions, as in the present case, it is important to consider the heart and coronary arteries even when interpreting nongated thoracic CTA [14,15,17,18].

Interpretation of gated thoracic CT, which is often required in suspected acute aortic syndromes, must include even more a careful evaluation of the coronary arteries. Although the examination is often partially marred by motion artefacts due to an elevated heart rate, the presence of occlusive or subocclusive stenosis of the coronary arteries must be excluded.

The spectrum of conventional ECG-gated and no gated CT findings that may be associated with acute MI includes coronary artery occlusion, perfusion abnormalities, left ventricular thrombi, focal wall thinning, ventricular aneurysms and pseudoaneurysms [14,19,20].

Coronary artery occlusion may be identified as an abrupt interruption of opacification in the coronary artery with endoluminal hypodensity that is suggestive of a total occlusion by a probable thrombus [15,21]. The obstruction of the native coronary artery with no luminal continuity and disruption of antegrade blood flow is referred to as total occlusion (CTO). Coronary artery subocclusion (SO), on the other hand, is classified as high-grade stenosis (99%) with antegrade flow. Clinically, it is crucial to distinguish noninvasively between CTO and SO since the latter suggests a better prognosis and fewer challenges with percutaneous coronary intervention [17,21]. The reverse attenuation gradient (RAG) sign is characterized as a gradient of reverse intraluminal opacification in arteries that are distant to the occlusive lesions [15,22]. It can be performed in both ECG-gated and no gated thoracic CT. The RAG exhibits more opacification at more distal places than the typical gradient pattern in normal coronary arteries, which has stronger attenuation in the proximal segment and gradually decreases attenuation along the vessel. The reverse attenuation gradient (RAG) sign is characterized as a gradient of reverse intraluminal opacification in arteries that are distant to

the occlusive lesions [22,23]. The RAG exhibits more opacification at more distal places than the typical gradient pattern in normal coronary arteries, which has stronger attenuation in the proximal segment and gradually decreases attenuation along the vessel. Combined with other parameters such as lesion length and bridging collateral vessels, CTO can be differentiated confidently from SO [22,23].

Perfusion abnormalities may be identified in some infarctions, especially in the acute phase as shown in our cases [24]. Perfusion abnormalities usually manifest first and most profoundly in the subendocardium. The subepicardium is usually less affected [3]. Myocardial perfusion defects may be very difficult to show on nongated studies, and conventional CT should not be performed to evaluate myocardial perfusion [17,19]. However, when an abnormality is clearly seen and a subendocardial-predominant perfusion abnormality is present, it should be noted and reported [14]. The presence of a subendocardial perfusion abnormality in an anatomic distribution suggestive of a coronary artery ought to be commented on, especially when other signs of MI are present [21].

Thrombus formation is one of the most important consequences of MI, present in around 10% of MIs, however the actual prevalence may be greater [25]. Most left ventricular thrombi develop within 2 weeks of the MI. Left ventricular thrombi are more commonly seen after anterior rather than inferior infarcts [25,26]. Thrombi are most commonly observed at the apex but may also be observed along the anterior wall and septum as showed in our case [13,26]. In CT scans, thrombi are typically seen as low-attenuation objects encircled by contrast enhancement [15,26]. Contrast enhancement frequently surrounds thrombi almost entirely, while some thrombi may layer against the subendocardial surface [27]. The majority of the time, thrombi do not enhance, while chronic organized thrombi may show some enhancement [27]. The presence of a thrombus is more likely to indicate MI because of the higher prevalence of infarcts as well as the stronger association between infarcts and thrombus formation [25,26,28].

One of the most typical signs of a previous MI is focal wall thinning. Myocardial necrosis follows coronary artery occlusion, which triggers inflammation and, finally, the elimination of the infarcted tissue [6]. The result is thinning of the myocardial wall in that location. It can be challenging to clearly identify minor areas of wall thinning in nongated examinations [17,19,21]. However, obvious large areas of severe wall thinning should raise the possibility of an MI.

After a transmural infarction, the resulting myocardial fibrosis and expansion result in a thin-walled projection from the ventricle, representing a chronic aneurysm [29,30]. Left ventricular aneurysm represents an outpouching containing endocardium, epicardium and thinned, abnormally contracting and scarred myocardium which show characteristic constant functional wall motion abnormality, usually dyskinesia [29,30]. They are more common in an apical, anterior, or anterolateral location and usually arise from an anterior descending vascular territory infarction [29,30]. Pseudoaneurysms are less common but are formed from transmural infarcts that rupture through the entire myocardial wall and are contained by the adjacent epicardial soft tissues and pericardium [29–31]. False aneurysms are more commonly inferior and lateral in location [29,30]. The presence of a small focal area of thinning at the apex of the left ventricle is a normal finding, and care should be taken to avoid overcalling this finding in healthy patients [29,30].

Conclusions

The increased use of CT in the evaluation of patients for a variety of indications gives the radiologist the unique opportunity to recognize findings consistent with MI in patients who may not have a prior diagnosis of ischemic heart disease. The heart and, in particular, the myocardium, should be carefully assessed in the analysis of all CT studies in which it is included in the FOV due to the prevalence of ischemic cardiomyopathy.

Patient consent

The authors declare that this report does not contain any personal information that could lead to the identification of the patients. Written informed consent for the publication of this case report was obtained from each of the 4 patients.

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