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Chest pain CT in the emergency department: Watch out for the myocardium

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ABSTRACT

Rationale and Objectives: To evaluate the frequency and relevance of hypodense myocardium (HM) encountered in patients undergoing chest-pain CT in the emergency department (ED).

Material and Methods: In this IRB-approved retrospective study, ECG-gated chest-pain CT examinations of 300 consecutive patients (mean age 60 ± 17 years) presenting with acute chest-pain to our ED were evaluated. Once ST-segment elevation infarction was excluded, chest-pain CT including the coronary arteries (rule-out acute coronary syndrome (ACS), pulmonary embolism (PE) and acute aortic syndrome (AAS): chest-pain $CT_{coronary}$, n = 121) or not including the coronary arteries was performed (rule-out PE and AAS: chest-pain $CT_{w/o\ coronary}$, n = 179). Each myocardial segment was assessed for the presence of HM; attenuation was measured and compared to normal myocardium.

Results: HM was identified in 27/300 patients (9%): 12/179 in chest-pain $CT_{w/o\ coronary}$ (7%) and 15/121 in chest-pain $CT_{coronary}$ (12%). Mean attenuation of HM (40 ± 17 HU) was significantly lower than that of healthy myocardium (103 ± 18 HU, $p < 0.001$), with a mean difference of 61 ± 19 HU. In 15/27 patients (55.6%) with HM, the final diagnosis was acute MI, and in the remaining 12/27 patients (44.4%) previous MI was found in the patients' history. Chest-pain $CT_{w/o\ coronary}$ identified HM in 10/15 patients (66.6%) with a final diagnosis of acute MI.

Conclusion: HM indicating acute MI are often encountered in chest pain CT in the ED, also in chest-pain $CT_{w/o\ coronary}$ when MI is not suspected. This indicates that the myocardium should always be analyzed for hypodense regions even when MI not suspected.

1. Introduction

Acute chest pain is a frequent reason for visiting the emergency department (ED) comprising 5–20% of all ED visits [1,2]. Various diseases including the esophagus, skeletal system, or more life threatening pathologies such as acute coronary syndrome (ACS) leading to myocardial infarction (MI), acute aortic syndrome (AAS) or pulmonary embolism (PE) can cause acute chest pain [1]. Thus, quick and reliable diagnosis or exclusion of these latter pathologies is highly desirable.

Computed tomography (CT) is the modality of choice when there is a clinical suspicion of PE and AAS [3,4]. In case of ACS, the patient's initial symptoms, cardiac enzymes and electrocardiograms (ECG) can be inconclusive requiring further work up and additional testing such as coronary CT angiography [5,6]. Coronary CT angiography is an established imaging test to rule out ACS in patients with low to intermediate risk presenting with acute chest pain. Hoffmann et al. [7] showed that an evaluation strategy consisting of an early coronary CT angiography improved the efficiency of clinical decision making for triage in the ED, with a shorter length of stay in the hospital and more direct discharges from the ED. Litt et al. [8] showed that a coronary CT angiography

Abbreviations: AAC/AHA, American College of Cardiology / American Heart Association; AAS, acute aortic syndrome; ACS, acute coronary syndrome; BPM, beats per minute; CAD, coronary artery disease; CI, confidence interval; CT, computed tomography; ECG, electrocardiography; ED, emergency department; HU, hounsfield unit; ICC, intraclass correlation coefficients; LAD, left anterior descending artery; MH, hypodense myocardium; MI, myocardial infarction; NPV, negative predictive value; NSTEMI, non-ST elevation myocardial infarction; PE, pulmonary embolism; PPV, positive predictive value; RCA, right coronary artery; CX, circumflex artery; ROI, region of interest

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based strategy for low- to intermediate-risk patients presenting with a possible ACS allows for a safe, expedited discharge from the ED.

If ACS, PE or ASS is suspected, the three separate CT examinations can be combined into one single CT examination, also known as chest pain CT [9,10]. Depending on the clinically suspicion, chest pain CT including the coronary arteries (rule out ACS, PE and AAS; rule-out ACS and PE; rule out ACS and AAS) or chest pain CT not including the coronary arteries (rule out PE and AAS) can be performed [11,12].

It is known that hypodense myocardium (HM) in CT indicates acute or chronic myocardial necrosis [13,14]. In the past years, we made the experience that HM indicating MI is encountered relatively often in chest pain CT examinations, even when the indication for chest pain CT did not include a specific request for an evaluation of the heart and coronary arteries. To our knowledge, no study so far assessed the relevance of HM in chest pain CT. Thus, the purpose of our study was first, to evaluate the prevalence of HM in chest pain CT examinations in ED patients presenting with acute chest pain, and second, to determine the significance of HM in these patients including an evaluation of potential culprit coronary lesions.

2. Material and methods

2.1. Study population

The study was approved by our institutional review board and local ethics committee. Written consent requirement was waived due to the retrospective nature of the study. Between June 2012 and November 2015, 300 consecutive chest pain CT examinations of patients (mean age 60 ± 17 years, range 18–101 years, 29% female, 71% male) presenting to our ED with acute chest pain were identified by using the radiology information system (Centricity RIS-I 5, Version 5.0.10.15, GE, Fairfield, Connecticut, USA) of our department. Chest pain CT examinations were subdivided into those including an evaluation of the coronary arteries, i.e. for ruling-out ACS, PE and AAS (n = 121), hereafter called *chest pain* $CT_{coronary}$ and those not including the coronary arteries, i.e. ruling-out PE and AAS (n = 179), hereafter called *chest pain* $CT_{w/o\ coronary}$. Patients with suspicion of ST-segment elevation MI do not undergo CT in our ED but are directly referred to catheter coronary angiography.

Each patient's electronic medical files were reviewed to assess type of chest pain, indications of CT, cardiovascular risk factors, known previous myocardial infarction (MI), results of catheter coronary angiography, suspected initial and final diagnosis causing acute chest pain. Final diagnosis of MI was based on results from all information available, including all imaging modalities performed within 30 days including catheter coronary angiography, cardiac magnetic resonance imaging and myocardial perfusion single-photon emission CT and was used as reference standard.

Type of chest pain was graded as typical (n = 18 [6%]), as atypical (n = 198 [66%]) and as non-anginal chest pain (n = 84 [28%]) (Table 1) [15]. 54 of the 300 patients (18%) had known coronary artery disease (CAD) (n = 34: previous MI, n = 38: previous stent implantation, and n = 13: previous coronary artery bypass grafting). In the 246 patients with suspicion of CAD, their risk was estimated using the Diamond and Forrester score [15]: A low risk for CAD was found in 14/246 (6%) patients, an intermediate risk in 219/246 (89%), and a high risk in 13/246 (5%) patients (Table 1).

2.2. CT data acquisition

All scans were performed on a second-generation dual-source CT scanner (SOMATOM Flash, Siemens Healthineers, Forchheim, Germany) located in the ED. No beta-blockers were administered prior to CT. For chest pain $CT_{coronary}$, sublingual nitroglycerin (Isosorbiddinitrat, Isoket Spray, 25 mg/ml, UCB-Pharma, Brussels, Belgium) was administered immediately prior to the scan, after checking for contraindications.

First, a non-enhanced single breath-hold prospectively ECG-gated scan in the high-pitch mode was performed with the following scan parameters: tube voltage 120 kVp, quality reference tube current-time product 150 mAs using automated tube current modulation, detector collimation 2 x 64 mm, slice acquisition 2 x 128 mm using the z-flying focal spot, gantry rotation time 280 ms and pitch 3.4. Images were reconstructed with sinogram-affirmed iterative reconstruction (SAFIRE) at a strength level of 3, with a medium smooth convolution kernel (I35f), slice thickness and increment of 3 mm, and a field-of-view of 200 x 200 mm.

Second, a contrast-enhanced retrospectively ECG-gated CT scan with ECG-pulsing was performed. For chest pain $CT_{w/o\ coronary}$, an ECG-pulsing window from 70-70% of the R-R interval was used to evaluate the aortic root. For chest pain $CT_{coronary}$, heart rate-dependent ECG pulsing was used to evaluate the aortic root and the patency of the coronary arteries: below 60 beats per minute (bpm) an R-R interval from 70-70%, heart rates from 60 to 70 bpm from 60 to 80%, heart rates from 70 to 80 bpm from 50 to 80%, and heart rates > 80 bpm from 30 to 80%. The following scan parameters were applied: reference tube voltage 100 kVp using automated attenuation-based tube potential selection (CAREkV), quality reference tube current-time product 300 mAs using automated tube current modulation, detector collimation 2 x 64 mm, slice acquisition 2 x 128 mm using the z-flying focal spot, gantry rotation time 280 ms, and pitch 0.2-0.5.

Depending on the body mass index, 80–100 ml of nonionic iodinated contrast media (370 mg/mL, Iopromide, Ultravist 370; Bayer Schering Pharma) was injected with a flow rate of 5–6 ml/sec into an antecubital vein, followed by the same volume with 20% contrast media and 80% saline solution and followed by a saline bolus chaser. The bolus tracking technique was used to trigger the scan with a circular region of interest (ROI) placed in the ascending aorta. The scan was started with a delay of 5 s after the attenuation threshold of 100 HU at 120 kVp was reached.

Images from contrast-enhanced CT were reconstructed using the following parameters: medium smooth convolution kernel (I30f), sinogram-affirmed iterative reconstruction (SAFIRE) at a strength level of 3, slice thickness of 0.75 mm, increment of 0.5 mm, and a field-of-view of 200 x 200 mm. In addition, 2-, 3-, and 4-chamber long axis and short axis reformations with a slice thickness of 5 mm and increment of 1 mm were used for evaluating possible myocardial segments with HM.

2.3. Data analysis

All images were reviewed by two independent and blinded radiologists (both with more than 5 years of experience in cardiovascular radiology). The myocardium was analyzed using the 17-segment model of the American College of Cardiology / American Heart Association (ACC/AHA). Readers reviewed all image reformations and were allowed to use their preferred individual window settings. Prior to the readout, the two readers performed a practice session together on two patients with HM, which were not included in the study.

Then, each myocardial segment was visually assessed for the presence of HM. If HM was present, attenuation of the HM was measured by placing a circular ROI in three distinct locations of the affected segment. The difference in attenuation between healthy myocardium and HM was calculated. Special care was taken to avoid possible false positives in the inferolateral wall from beam hardening artifacts by using multiple reformation and different heart phases if available [16]. The thickness of the affected segment with HM and the transmurality was semiquantitatively assessed by using a 3-point score (thinned myocardium = 0, normal thickness of the myocardium = 1, thickened myocardium = 2) and a 4-point score (transmurality 0–25% = 1; 26–50% = 2, 51–75% = 3; 76–100% = 4), respectively. Disagreement between readers in terms of affected myocardial segments, transmurality and thickness was solved in a consensus reading.

Table 1
Demographic data of all patients undergoing chest pain CT (n = 300).

	Total	Chest pain $CT_{coronary}$	Chest pain $CT_{w/o\ coronary}$
Number of patients	300 (100%)	121 (40.3%)	179 (59.7%)
Age (years) (mean ± SD)	60.0 ± 15.7	61.6 ± 16.6	58.9 ± 15.0
Sex			
Male	214 (71%)	78 (65%)	136 (76%)
Female	86 (29 %)	43 (35%)	43 (24%)
BMI (kg/m^2), (mean ± SD)	28.1 ± 12.3	27.9 ± 4.4	28.1 ± 15.6
Diabetes	39 (13%)	16 (13%)	23 (13%)
Hypertension	212 (71%)	90 (74%)	122 (68%)
Dyslipidemiass	138 (46%)	60 (50%)	78 (44%)
Current or former smoker	161 (54%)	62 (51%)	99 (55%)
Positive family history for CAD	103 (34%)	42 (35%)	61 (34%)
Chest pain (typical/atypical/non- anginal chest pain)	18 (6%)/ 198 (66%)/ 84 (28%)	4 (3%)/ 80 (66%)/ 37 (31 %)	14 (8%)/ 118 (66%)/ 47 (26%)
Diamond and Forrester score* (low/intermediate/high)	14 (6%)/ 219 (89%)/ 13 (5%)	9 (10%)/ 83 (88%)/ 2 (2%)	5 (3%)/ 136 (90%)/ 11 (7%)

SD: standard deviation; BMI: body mass index; CAD: coronary artery disease.
* in patients with suspicion of CAD (n = 246).

Attenuation of normal healthy myocardium was primarily measured in the septum. If hypodense myocardium was identified in the septum, measurements were performed either on the anterior or, in case of hypodense myocardium in the anterior wall and septum, on the posterior wall. Attenuation of the normal myocardium was measured similarly to the hypodense myocardium using a circular ROI in three distinct locations.

In chest pain $CT_{coronary}$, we searched for the presence of culprit coronary lesions potentially causing HM. If present, the degree of stenosis, plaque type (non-calcified, mixed and calcified) and presence of positive remodeling was noted, as previously shown [17].

2.4. Statistical analysis

Quantitative data are given as mean values and standard deviations, qualitative data as median and range. Intraclass correlation coefficients (ICC) were calculated to evaluate the interreader agreement between the readers regarding the measured attenuation values. Intraclass correlation coefficient less than 0.69 was defined as poor, ICC between 0.70 and 0.79 as fair, ICC between 0.80 and 0.89 as good, and ICC greater than 0.9 as high. Cohen κ was calculated to assess the interreader agreement regarding the qualitative parameters. κ between 0.0-0.20 was defined as poor, between 0.21-0.4 as fair, between 0.41-0.60 as moderate, between 0.61-0.80 as good and between 0.81–1.00 as excellent agreement. The paired student's *t*-test was used to compare the attenuation values of healthy and hypodense myocardium. The Wilcoxon signed-rank test was used to compare myocardial thickness of hypodense myocardium in patients with known acute and chronic MI. Sensitivity, specificity, positive (PPV) and negative predictive values (NPV) were calculated for detection of acute MI. A two-tailed P value below 0.05 was considered to indicate statistical significance. Statistical analyses were performed with commercially available software (SPSS, Version 22, Chicago/IL, USA).

3. Results

3.1. Interreader agreements

A total of 5'100 myocardial segments (17 segments in 300 patients) were evaluated. Agreement between readers was high for the attenuation values of hypodense (ICC = 0.947) and healthy myocardium (ICC = 0.948). Agreement between readers was good for the assessment of transmurality (κ = 0.64) and good for the assessment of myocardial thickness (κ = 0.70). Disagreement regarding the localization of HM occurred in 11 segments in 6 patients, regarding transmurality in 15 segments in 7 patients, and regarding myocardial thickness in 19 segments in 14 patients, respectively. All disagreements were solved in consensus reading.

3.2. Frequency of HM

HM was identified in 27/300 patients (9%) with 123 affected myocardial segments (Table 2). In 15/27 patients (56%) with 75 hypodense myocardial segments, the final diagnosis was acute MI. All these 15 patients had a non-ST segment elevation MI. In the remaining 12 patients (44%), medical records indicated previous, chronic MI. Among the 15 patients with acute MI and corresponding HM, chest pain $CT_{coronary}$ was performed in 9 patients (60%) and chest pain $CT_{w/o\ coronary}$ in 6 patients (40%) (Fig. 1).

3.3. Sensitivity, specificity, PPV and NPV for identification of acute MI based on HM

Among the total population of 300 patients, medical records revealed the final diagnosis of acute MI in 29 patients (10%). In all 29 patients with the final diagnosis of acute MI, catheter angiography revealed the corresponding culprit lesion. In 15 of 29 patients (52%) with final diagnosis acute MI, HM was observed (9 in chest pain $CT_{coronary}$ and 6 in chest pain $CT_{w/o\ coronary}$). In the remaining 14 patients (48%) with final diagnosis acute MI, no HM was observed (5 in chest pain

Table 2
Distribution of affected myocardial segments.

Myocardial Segments	Chest pain $CT_{coronary}$	Chest pain $CT_{w/o\ coronary}$	Total
1	4	3	7
2	4	4	8
3	6	3	9
4	3	4	7
5	5	3	8
6	5	2	7
7	4	3	7
8	4	7	11
9	4	5	9
10	2	3	5
11	3	5	8
12	4	4	8
13	4	3	7
14	5	4	9
15	2	0	2
16	4	2	6
17	3	2	5
Total	66	57	123

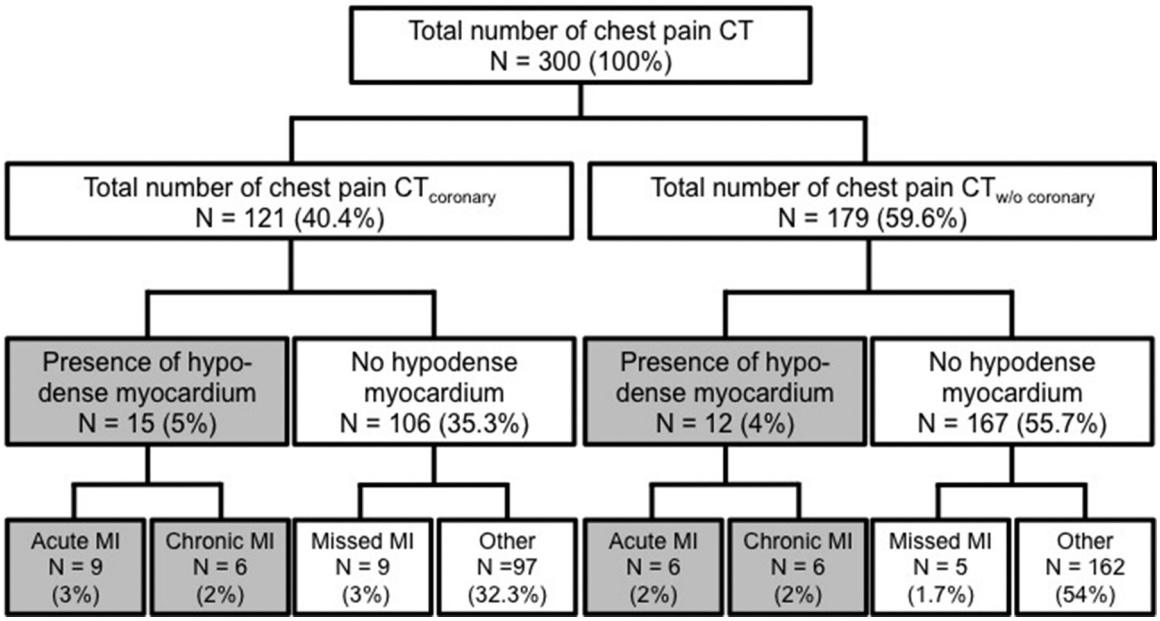

Fig. 1. Flowchart of the study. MI: myocardial infarction.

$CT_{coronary}$ and 9 in chest pain $CT_{w/o\ coronary}$). No false positive cases were observed. Sensitivity, specificity, PPV and NPV for the detection of acute MI by assessing HM was 52% (95% CI [32.5%–70.6%]), 100% (95% CI [98.7%–100%]), 100% (95% CI [100%]) and 95% (95% CI [93.0%–96.6%]), respectively.

3.4. Attenuation, thickness and transmurality of HM in acute MI

In patients with final diagnosis acute MI, attenuation of HM (40 ± 17 HU) was significantly lower than that of healthy myocardium (103 ± 18 HU, $p < 0.001$), with a mean difference in attenuation of 61 ± 19 HU (Figs. 2 and 3). Myocardial thickness of all hypodense myocardial segments was rated as follows: 8/75 (11%) thickened, 67/75 (89%) normal, 0/75 (0%) thinned. Transmurality was rated as follows: transmurality 0–25% in 1/75 (1%); transmurality 26–50% in 5/75 (7%); transmurality 51–75% in 20/75 (20%), and transmurality 76–100% in 54/75 (72%).

3.5. Attenuation values, thickness and transmurality of HM in chronic MI

In 12 of 27 patients with HM (44%) with 48 affected myocardial segments, previous chronic MI was found according to the medical records. In those patients, attenuation of HM (18 ± 37 HU) was significantly lower as compared to that of healthy myocardium (103 ± 18 HU, $p < 0.001$), with a mean attenuation difference of 89 ± 35 HU (Fig. 4 and 5). Myocardial thickness was as follows: 0/48 (0%) thickened, 19/48 (40%) normal, 29/48 (60%) thinned. Transmurality was as follows: transmurality 0–25% in 3/48 (6%), transmurality 26–50% in 0/48 (0%), transmurality 51–75% in 9/48 (19%), and transmurality 76–100% in 36/48 (75%).

3.6. Differentiation between acute and chronic MI

The calculated mean difference of attenuation between healthy and infarcted myocardium showed significant differences between patients with acute (61 ± 19 HU) and chronic (89 ± 35) infarction ($p < 0.001$). In patients with chronic infarction, HM was significantly thinner as compared to that in patients with acute infarction ($p < 0.05$).

3.7. Coronary arteries in chest pain $CT_{coronary}$

9 patients had MI and a chest pain $CT_{coronary}$ showing HM, culprit coronary lesions were identified in the following vessels: LAD 5/9 (56%), CX 2/9 (22%), RCA 2/9 (22%). For all patients correlating catheter angiography was available and confirmed the culprit lesions identified on CT. Grade of stenosis was rated as follows: 75–99% in 2/9 (22%) and occlusion in 7/9 (78%). Of the nine culprit plaques, positive remodeling was observed in 8/9 (89%) cases. Two of these 9 culprit plaques (22%) showed large calcifications, 6/9 (67%) were mixed with spotty calcifications, and 1/9 (11%) were non-calcified (see Fig. 2).

4. Discussion

Our study suggests that HM indicating acute MI can be observed relatively often in chest pain CT examinations of patients presenting to the ED with chest pain, even when the clinical indication does not explicitly includes an assessment of the coronary arteries. In patients with a final diagnosis of acute MI, HM was detected in 4% of chest pain $CT_{coronary}$ and in 6% of chest pain $CT_{w/o\ coronary}$. Sensitivity, specificity, PPV and NPV for the detection of MI by assessing HM was 52%, 100%, 100% and 95% respectively. In chest pain $CT_{coronary}$ showing HM and with proven final diagnosis MI, culprit coronary lesions were correctly identified in all patients. In chest pain $CT_{w/o\ coronary}$ with HM and proven final diagnosis MI, the corresponding culprit lesion was identified in 66%, even when it was not specifically asked for. Acute and chronic MI differs in regard to the attenuation of HM and myocardial thickness, with larger differences between affected and healthy myocardium and with thinner segments in chronic MI.

It is well known that HM correlates with MI [14], and that acute and chronic MI can be differentiated based on myocardial attenuation values and by assessing myocardial thickness [18]. Various previous studies in different patient populations showed moderate to good sensitivities and good specificity in the detection of MI by assessing hypodense myocardium ranging between 67–88% and 85–98% respectively [19–21]. Schepis et al. [21] showed in a patient population of 38 patients with ACS (NSTEMI or unstable angina) sensitivity, specificity, NPV and PPV of 88%, 86%, 80% and 91% respectively. Interestingly, the sensitivity in our study was lower as compared to the aforementioned study. However, Schepis et al. [21] used a dedicated protocol for

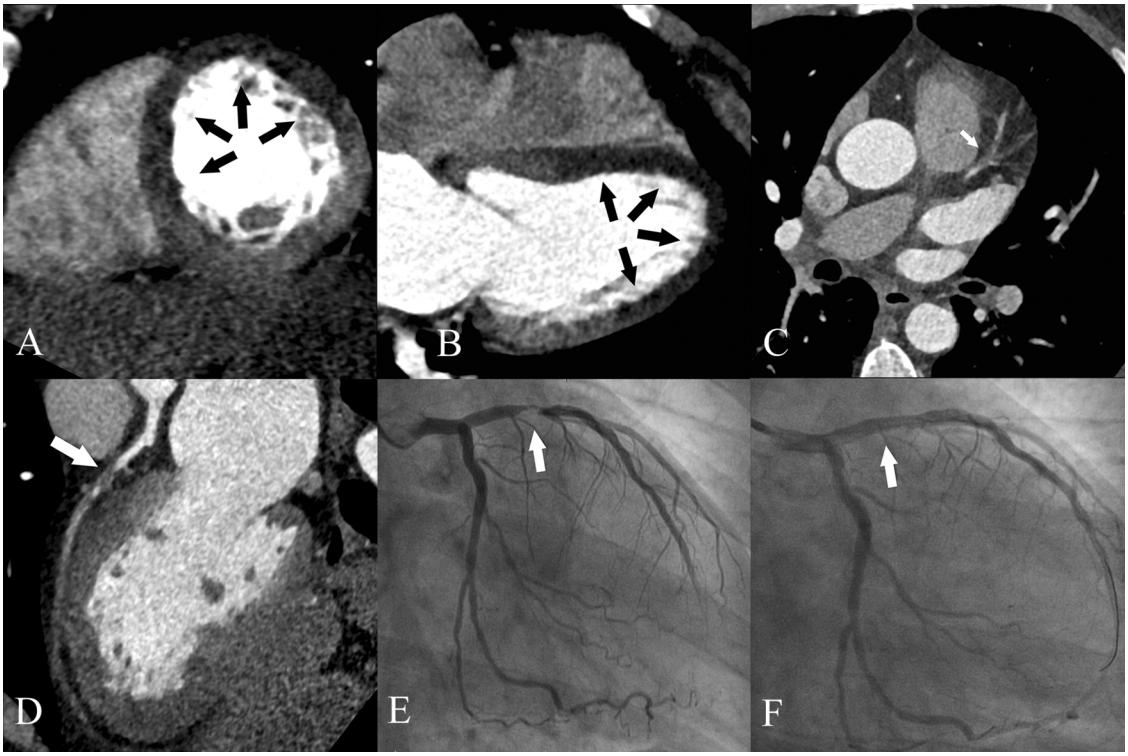

Fig. 2. Chest pain $CT_{coronary}$ in a 50-year-old male patient with acute atypical chest pain presenting to the emergency department. (A) Short axis and (B) 4-chamber reformations revealed transmural hypodense myocardium (black arrow) in the septal and anterior wall of the mid and apical left ventricle. (C) Axial and (D) oblique reformations revealed the corresponding culprit lesion with a soft plaque with slight positive remodeling (arrow) and thrombus occluding the left anterior descending artery. Subsequent catheter coronary angiography (E) verified the culprit lesion (arrow), which was treated with thrombus aspiration and stenting (F) (arrow).

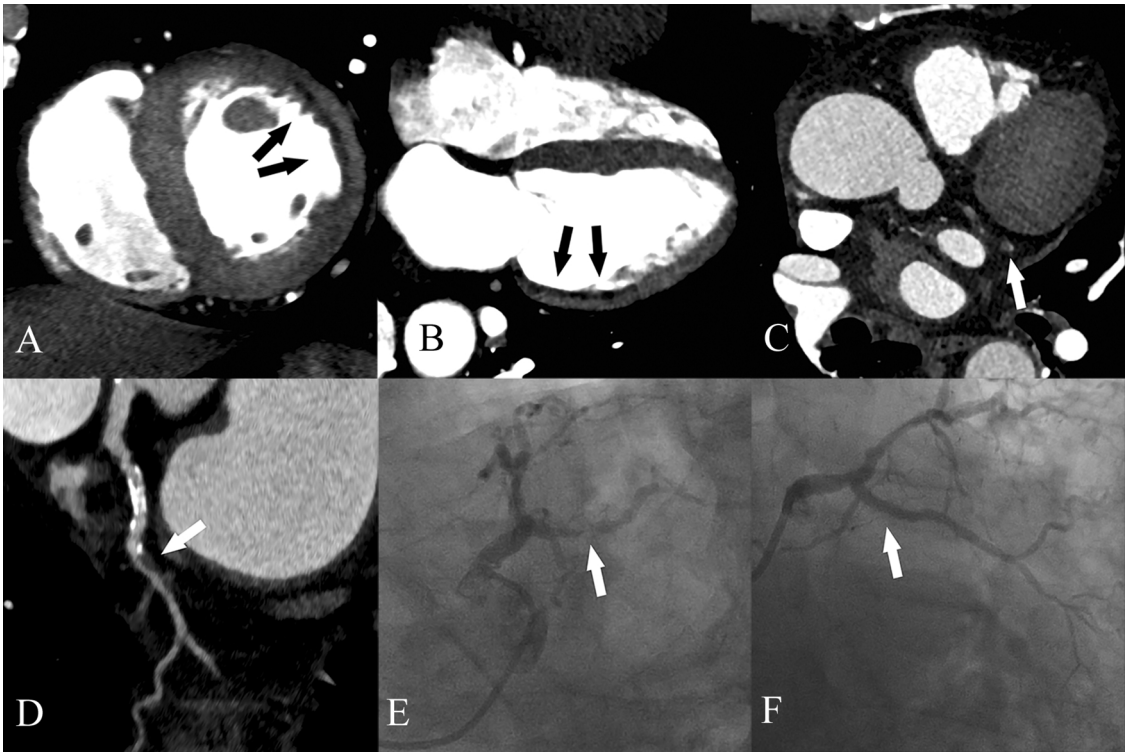

Fig. 3. Chest pain $CT_{w/o\ coronary}$ in an 84-year-old male patient with acute atypical chest pain presenting to the emergency department. (A) Short axis and (B) 4-chamber long axis reformations revealed subendocardial hypodense myocardium (black arrow) in the inferior part of the basal left ventricle. (C) Axial and (D) curved reformations of the circumflex artery revealed the corresponding culprit lesion (arrow) consisting of a plaque with spotty calcification. No positive remodeling was observed. The culprit lesion was verified with catheter coronary angiography (E) and was treated with stenting (F) (white arrow).

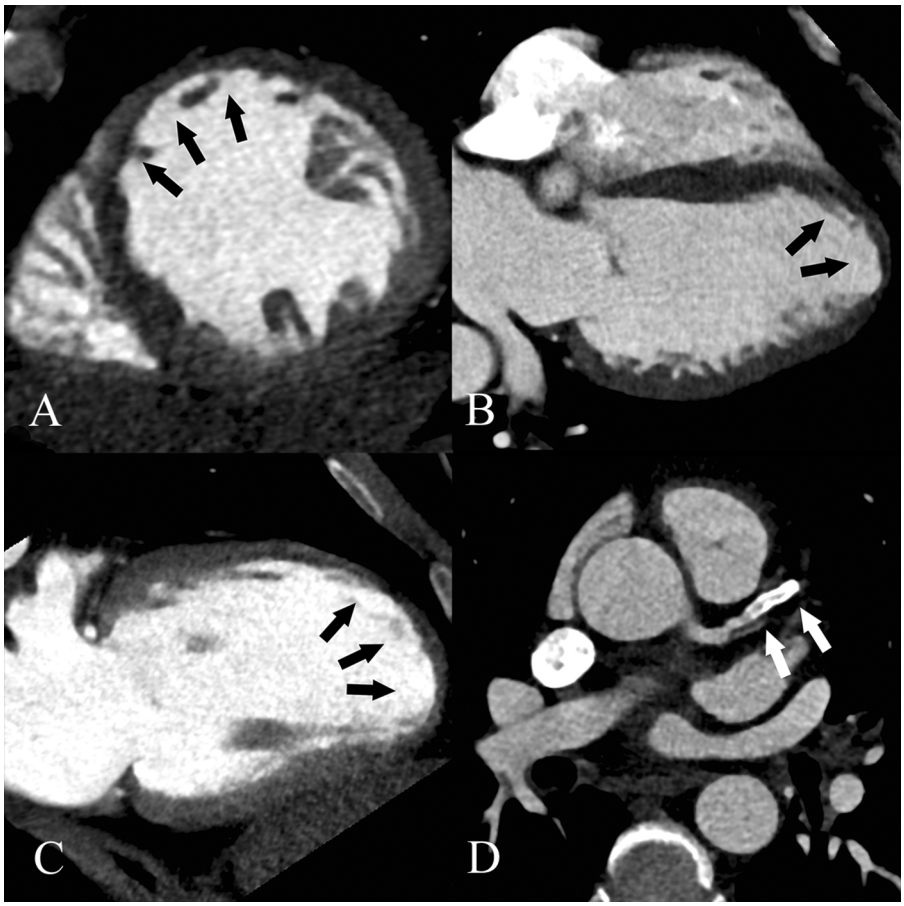

Fig. 4. Chest pain $CT_{coronary}$ of a 48-year-old male patient with acute chest pain presenting to the emergency department. (A) Short axis (B) 4-chamber and (C) 2-chamber long axis reformations revealed hypodense and thinned myocardium (black arrow) in the anterior wall of the mid and apical left ventricle. (D) Axial image shows a heavily calcified plaque and stent in the left anterior descending artery. Medical history revealed previous chronic MI.

determining myocardial perfusion defects during the arterial first-pass, while our protocol aimed at a simultaneous exclusion of AAS, PE and CAD. This could be a reason for the lower sensitivity in our study. In turn, we achieved high specificity and negative predictive value (both 100%) as compared to other studies.

To our knowledge, this is the first study investigating the prevalence and relevance of HM in patients referred to chest pain $CT_{coronary}$ and chest pain $CT_{w/o\ coronary}$, a patient population mainly consisting of patients with low to intermediate risk for acute MI. Even though the number of discovered acute MI based on the assessment of myocardium was small, we think it is still worth to assess the myocardium. In chest pain $CT_{w/o\ coronary}$, acute MI may be detected which would have

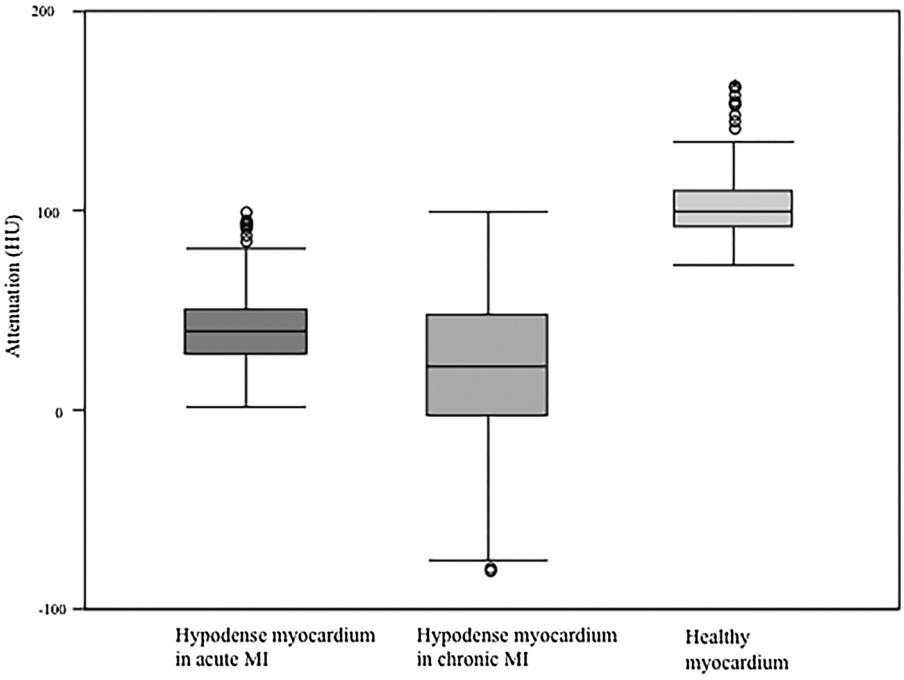

Fig. 5. Boxplots showing the attenuation values (HU) of HM of acute and chronic myocardial infarction (MI) and of healthy myocardium. The horizontal lines in the boxes correspond to the mean. The top and bottom lines of the boxes correspond to the first and third quartiles. The whiskers represent 1.5 the interquartile range (IQR). Circles represent outliers (1.5 IQR and 3 IQR from the near edge of the box).

otherwise been missed, and in chest pain $CT_{coronary}$ additional assessment of HM may increase the diagnostic confidence in ambiguous coronary findings. Of course we still have to keep in mind that given the low sensitivity of detecting acute MI using HM, the absence of HM is not accurate for excluding MI [19].

Some study limitations must be considered. First, the study was retrospective. Second, catheter coronary angiography was not performed in all patients to confirm the findings of coronary CT angiography. Finally, the culprit lesions were assessed in a relatively simple way, but not analyzed in more detail any further (e.g. presence of the napkin ring sign [22]).

In conclusion, our results suggest that there could be a relevant benefit for patients in the ED with chest pain who receive chest pain CT when the myocardium is also analyzed for the presence or absence of HM, even when the heart and coronary arteries were not specifically asked-for. Awareness for the potential presence of HM and its implications should be high for not missing this life-threatening finding and for triggering immediate further therapy, eventually improving outcome of these patients.

Conflict of interest

All authors involved in this work declare no conflict of interest

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