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Research paper

Non-gated high-pitch computed tomography aortic angiography: Myocardial perfusion defects in patients with suspected aortic dissection

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ABSTRACT

Objectives: To investigate the diagnostic value of first-pass myocardial perfusion defects visualised in non-gated high-pitch computed tomography angiography (CTA) in patients admitted to the emergency department (ED) for suspected aortic dissection.
Methods: We recruited 174 ED patients who underwent high-pitch CTA of the aorta because of suspected aortic dissection. We divided these patients into two groups (diseased and control groups) based on whether their clinical data fulfilled the third universal definition of acute myocardial infarction (AMI), specifically an increase in cardiac troponin (cTn) with at least one of the following: (a) symptoms of ischemia; (b) new ST-segment-T wave (ST-T) changes or new left bundle branch block (LBBB); (c) development of pathological Q wave; (d) new loss of viable myocardium or new regional wall motion abnormality; or (e) identification of an intracoronary thrombus by angiography or autopsy. Twenty-two patients with a clinical diagnosis of AMI were placed in the diseased group. Myocardial perfusion defects were evaluated qualitatively and quantitatively on the late arterial phase obtained 50 s post-threshold.
Results: Of the 22 patients with a final diagnosis of AMI, visually identifiable perfusion defects were observed in 12 patients. The sensitivity, specificity, negative predictive value, and positive predictive value of any perfusion defect for predicting AMI were 54.6%, 94.7%, 93.5%, and 60.0%, respectively. Quantitative analysis indicated that CT attenuation was significantly lower within perfusion defects than within the normal myocardium (67.3 ± 29.5 HU vs. 92.8 ± 19.7 HU; $p < 0.001$).
Conclusions: In patients with acute chest pain, the presence of myocardial perfusion defect observed on nongated high-pitch CTA of the aorta can be used to identify individuals with AMI with high specificity, but low sensitivity.

1. Introduction

Non-electrocardiogram (ECG)-gated contrast-enhanced computed tomography (CT) angiography (CTA) is available in most hospitals where patients with chest and/or back pain are admitted to the emergency department (ED).[1] Mano et al. investigated the role of perfusion defects in diagnosis of acute coronary syndrome in 154 consecutive patients admitted to the ED to differentiate acute aortic dissection from pulmonary thromboembolism (PE) by using non ECG-gated 64-slice CT. The results demonstrated a high sensitivity (93%) and negative predictive value (NPV, 98%) to identify acute myocardial infarction (AMI).[1] Coronary CT angiography (coronary CTA) can detect coronary artery disease in patients with chest pain. The addition of CT perfusion (CTP) to coronary CTA improves its specificity and positive predictive value (PPV) regarding the presence of myocardial ischemia.[2] The addition

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of CTP to coronary CTA increases the sensitivity to identify acute coronary syndromes (ACS) from 77% to 90%.[3] The demonstration of perfusion abnormalities in chest CT is difficult because the myocardium is less resolved in non-gated studies. With the second generation dual-source CT, the option to increase pitch above the traditional technical limit of 1.5, in single-source CT, has allowed to substantially reduce image acquisition time and to increase volume coverage.[4] High-pitch dual-source CTA of the entire aorta without ECG synchronisation is feasible in a very short time period and renders motion-free imaging of the aorta.[5] Although non-gated high-pitch aortic CTA has been established as an initial diagnostic imaging modality for acute aortic syndromes, its diagnostic value for detecting AMI in a population with suspected aortic dissection has not yet been investigated. This study determined the diagnostic value of first-pass myocardial perfusion defects visualized using non-gated high-pitch contrast-enhanced CTA in patients admitted to the ED for suspected acute aortic syndromes.

2. Methods

2.1. Patients

This study was approved by the institutional review board of the hospital. All patients provided written informed consent for CT imaging. However, the requirement of informed consent was waived for retrospective chart review. We retrospectively recruited patients with acute chest pain who underwent non-gated high-pitch CTA of the aorta to exclude acute aortic dissection in the ED from July 2010 to September 2014. We reviewed each medical chart to identify the final diagnosis for each admission. AMI diagnosis was confirmed by an attending physician or cardiologist according to the third universal definition of AMI released in 2012 by the ESC/ACCF/AHA/WHF,[6] which included a dynamic change of cardiac troponin (cTn) and at least one of the following: (a) symptoms of ischemia; (b) new ST-segment-T wave (ST-T) changes or new left bundle branch block (LBBB); (c) development of pathological Q wave; (d) new loss of viable myocardium or new regional wall motion abnormality; or (e) identification of an intracoronary thrombus by angiography or autopsy. Exclusion criteria were as follows: 1) history of coronary artery disease (CAD), 2) history of previous MI, 3) absence of ECG data at ED visit, 4) absence of two consecutive (within 24 h) cardiac troponin I (cTn-I) data at ED, and 5) poor imaging quality. We divided the recruited patients into two groups: the diseased group, comprising patients who had received a diagnosis of ST-elevation myocardial infarction (STEMI) or non-ST-elevation myocardial infarction (NSTEMI), and a control group, comprising patients who had not received a diagnosis of STEMI or NSTEMI.

2.2. CT imaging protocol

We used a high-pitch, 128-slice CT system (Definition Flash, Siemens, Forchheim, Germany) operated in single-source mode with a pitch of 2.0, collimation of 128 × 0.6 mm, rotation time of 0.28 s, tube potential of 120 kV, and 100–150 reference mAs. Data were acquired in the craniocaudal direction during a single breath hold. The imaging range extended from the upper thoracic aperture to the proximal femoral arteries. Patients were examined in the supine position. We injected 90 mL of an iodinated contrast material (iodine concentration of 400 mg/mL, Imeron 400, Bracco Imaging, Konstanz, Germany) at a flow rate of 3.5 cc/s by using an 18–20-G intravenous line followed by 50 mL of a saline chaser.

CTA was automatically started based on a bolus-tracking measurement at the level of the ascending thoracic aorta at a threshold of 100 Hounsfield units (HU).The post-threshold delay scan for early and late arterial phase was set to 13 s and 50 s, respectively. Transverse images were reconstructed at a 1.0-mm slice thickness with 0.7-mm increments by using a medium-soft convolution kernel (B30f) and a matrix size of 512 × 512. In addition, we reconstructed axial slices at a 5.0-mm thickness with 5.0-mm increment.

2.3. CT image analysis

An experienced cardiac radiologist (YST, 14 years of experience) and a senior radiology resident (LTH, 3 years of experience) blinded to clinical and angiographic results jointly reviewed CTA of the aorta and reached consensus in cases of disagreement. Axial images reconstructed perpendicular to the long axis of the body were evaluated. The window width and centre of all images were set at 700 HU and 100 HU, respectively. Enhancement of the left ventricular myocardium was visually assessed on late arterial phase. According to previous studies, a perfusion defect was visually defined as a region of lower attenuation and/or wall thinning within the normally enhanced myocardium.[7] Myocardial enhancement was measured in HU by circling the region of interest (ROIs) of approximately 10 mm^2 within the perfusion defects, normal interventricular septum (IVS), and the paraspinal muscle at the level of T10, in three consecutive axial images. The average attenuation values were used for statistical analyses. ΔHU myocardium was calculated and defined as the enhancement difference between the normal IVS and the paraspinal muscle in the control group, and between the myocardial perfusion defect and paraspinal muscle in the diseased group. Myocardium ratio was defined as the IVS myocardium divided by the paraspinal muscle in the control group and the myocardial perfusion defect in the diseased group. Of the 174 image data sets, 20 were randomly analysed by a second observer to ensure interobserver reproducibility.

2.4. Statistical analysis

Statistical analysis was performed using commercially available software (SPSS 17.0 for Windows; SPSS, Chicago, III). Continuous and categorical variables were expressed as medians ± standard deviations and percentages, respectively. The chi-square test was used for descriptive statistics. The independent *t*-test was used for evaluating the difference in average attenuation values and the adjusted attenuation value between the normal and infarcted regions. The receiver-operating characteristic (ROC) curves were constructed to assess the diagnostic accuracy of perfusion defects in AMI. Cut point estimates, 95% confidence interval (CI), and areas under the ROC curve (AUCs) were calculated. In addition, sensitivities and specificities at cut-off points were determined using the Youden index. A P value of <0.05 was considered statistically significant.

Interrater reliability (IRR) was assessed using Kappa statistics. IRR was poor, fair, good, and excellent for Cronbach's alpha values < 0.40, between 0.40 and 0.59, between 0.60 and 0.74, and between 0.75 and 1.0, respectively.[8] Intrarater reliability was evaluated using test–retest reliability. A Pearson correlation coefficient of >0.7 indicated good consistency.[9]

3. Results

3.1. Patients

From July 2010 to September 2014, 618 patients visited the ED and underwent high-pitch CTA of the aorta because they were suspected to have acute aortic dissection. Of them, 174 were