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CASE REPORT
Cardiac imaging

Case report of non-ST-segment elevation myocardial infarction diagnosed in spectral detector-based computed tomography performed for the diagnosis of acute pulmonary embolism

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Background Contrast-enhanced spectral detector-based computed tomography (SDCT) allows for the comprehensive and retrospective analysis. We report a case of pulmonary thromboembolism (PE) accompanied by non-ST-segment elevation myocardial infarction (NSTEMI) diagnosed by SDCT.

Case summary A 72-year-old man with diabetes mellitus, hypertension, and prostate cancer suddenly developed chest and back pain and had difficulty in breathing at rest. Electrocardiography showed a right bundle branch block without significant ST-segment change. The initial serum troponin I level was 0.05 ng/mL, and the d-dimer level was 14.7 μg/mL. Spectral detector-based computed tomography showed bilateral scattered PE. After admission, his chest pain persisted, and the serum troponin I level 3 h after admission was elevated to 0.90 ng/mL. Reconstruction of SDCT images showed a perfusion defect of the posterolateral left ventricle myocardium. A coronary angiogram showed total occlusion of the obtuse marginal branch (OM); percutaneous coronary intervention was performed. Furthermore, we administered him with oral anticoagulants (OACs) for PE. Spectral detector-based computed tomography tests performed 6 months after the treatment was initiated, until when the dual antiplatelet therapy and OAC therapy were continued, showed improvement in perfusion defects of both pulmonary fields and the myocardium. His treatment was deescalated to single antiplatelet therapy and OAC, and the patient has had a good course.

Discussion Non-ST-segment elevation myocardial infarction is sometimes difficult to diagnose accurately, especially in the hyper-acute phase or in the OM branch. The reconstruction of spectral images from enhanced SDCT was helpful to diagnose this unique combination of PE and NSTEMI and may be useful for evaluating therapeutic effects in such patients.

Keywords Case report • Contrast medium-enhanced spectral detector-based computed tomography • Acute coronary syndrome • Pulmonary thromboembolism

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Learning points

- Non-ST-segment elevation acute coronary syndrome (ACS) can be difficult to diagnose accurately, especially in the hyper-acute phase or in the obtuse marginal branch where changes in physiological tests are less likely to be reflected.
- The reconstruction of spectral images from contrast medium-enhanced spectral detector-based computed tomography even without electrocardiogram synchronization may be helpful to improve the diagnostic accuracy of ACS and may be useful for evaluating the therapeutic effects.

Introduction

Acute chest pain is one of the most important clinical symptoms and one of the main reasons for presentation to the emergency department. Patients with acute coronary syndrome (ACS), aortic dissection, or pulmonary thromboembolism (PE) may present with similar symptoms, and the diagnosis of the latter two diseases is usually made with contrast-enhanced computed tomography (CT) without electrocardiogram (ECG) synchronization. Although coronary CT angiography (CCTA) provides accurate diagnosis for coronary artery lesions, the diagnostic value of non-ECG-synchronized CT, which is often performed in the emergency department, is not as high when compared to CCTA.[1–5] Besides, ECG synchronization is needed to obtain CCTA images and the heart rate must be within the optimal range. Reperfusion time is especially important in ACS, and therefore it is often difficult to obtain CCTA images. The evaluation of myocardial perfusion in non-ECG-synchronized CT may sometimes detect myocardial infarction; however, if the infarcted region is small, this may be overlooked.[6,7] Thus multiple imaging techniques are performed to make a differential diagnosis; however, we sometimes meet cases that are difficult to diagnose.

Contrast medium-enhanced spectral detector-based computed tomography (SDCT) has the potential for comprehensive analysis of the coronary artery morphology as well as changes in myocardial perfusion.[6,8–10] Tissues in the human body and iodine-based contrast media have unique absorption characteristics when penetrated with different X-ray energy levels, which enables mapping of the iodine and blood distribution. SDCT can retrospectively reconstruct images to clarify the iodine and blood distribution.

We experienced a case of PE accompanied by ACS in the obtuse marginal branch (OM) which is sometimes difficult to diagnose. If the patient is initially scanned with SDCT, adding the retrospective valuation of the images made a clear diagnose possible.

Timeline

Time	Events
2 years previously	Diagnosed with diabetes mellitus, hypertension, and prostate cancer accompanied by multiple bone metastasis. Medication therapy was started

Continued

Continued

Time	Events
Day 1	
13:00	The patient had sudden-onset chest pain and dyspnoea
15:00	He was transferred to our emergency department and diagnosed with pulmonary thromboembolism by enhanced computed tomography
18:00	His chest pain persisted and his serum troponin I level, 3 h after the admission was elevated to 0.90 ng/mL. Reconstruction of the spectral detector-based computed tomography (SDCT) images showed a perfusion defect of the posterolateral left ventricle myocardium
18:30	A coronary angiogram was performed, which showed total occlusion of the obtuse marginal branch. Consequently, percutaneous coronary intervention (PCI) was performed at the occlusion site. After thrombectomy, two drug-eluting stents were deployed
	Peak post-PCI creatine kinase was 2034 IU/L. Dual antiplatelet therapy (aspirin 100 mg and clopidogrel 75 mg/day) and oral anticoagulant (OAC) therapy (rivaroxaban 30 mg/day for 3 weeks and 15 mg/day after 3 weeks) were started
Day 14	He had a good clinical course and was discharged
6 months after the treatment started	The SDCT showed an improvement in the perfusion defects of both pulmonary fields and the myocardium
	Single antiplatelet therapy (clopidogrel 75 mg/day) and OAC therapy (rivaroxaban 15 mg/day) were continued, and the patient felt well without any recurrence

Case presentation

A 72-year-old man with diabetes mellitus, hypertension, and prostate cancer accompanied by multiple bone metastasis had sudden chest and back pain and difficulty in breathing at rest. His chest pain was accompanied by heaviness, squeezing, and discomfort. His prostate cancer had been treated with bicalutamide (80 mg/day) for ~2 years,

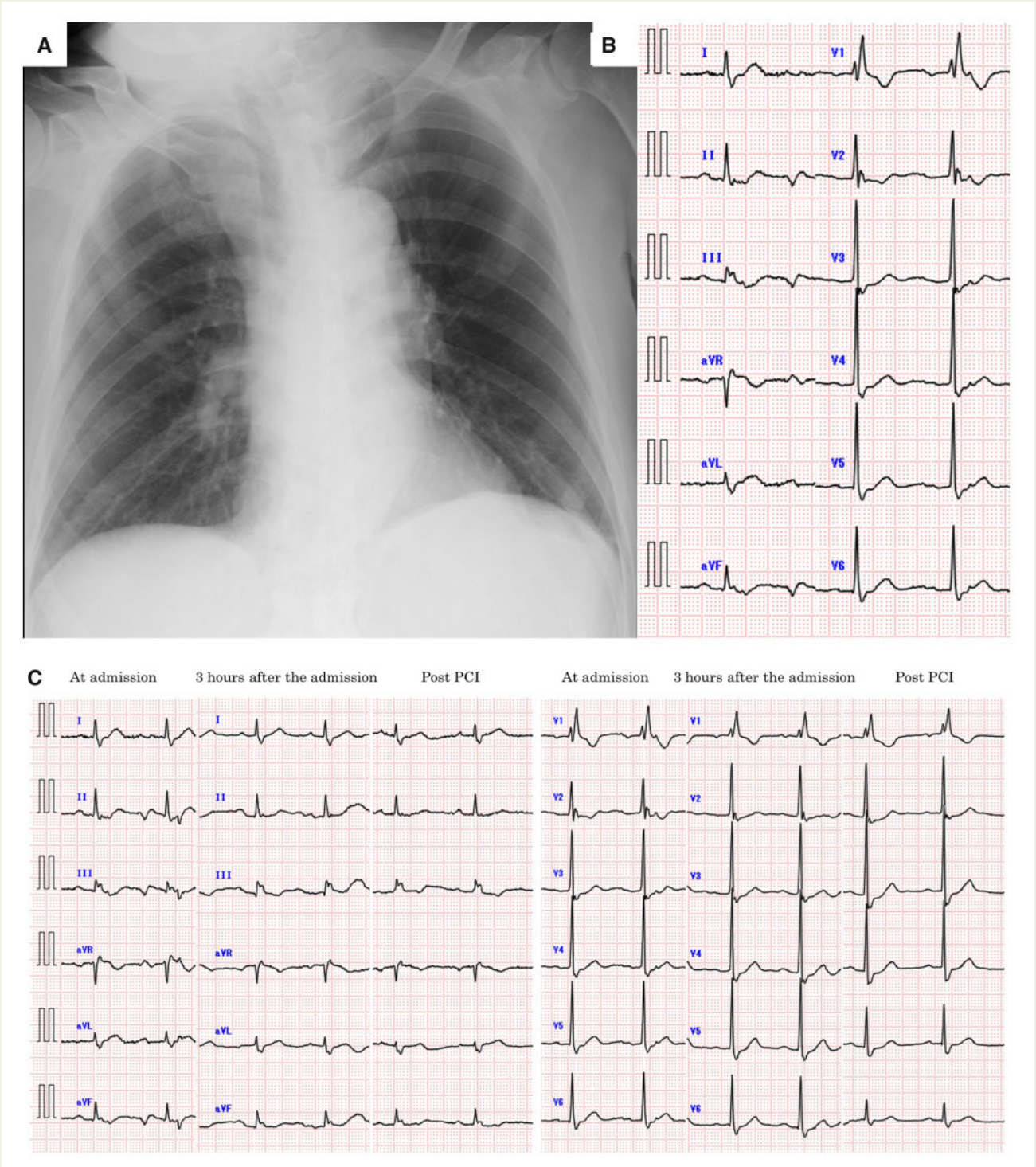


Figure 1 (*A*) Chest X-ray images in the anteroposterior view demonstrating no congestion and pleural effusion. (*B*) Electrocardiogram demonstrating sinus rhythm with complete right bundle branch block and no significant ST-segment change. (*C*) Serial electrocardiogram tests demonstrating no significant ST-segment change.

and his cancer stage was stable. He was brought to our hospital by ambulance and his vital signs were as follows: heart rate 72/min, respiratory rate 24/min, an axillary temperature 36.2°C, saturation of percutaneous oxygen under room air 90%, and peak blood pressure of 190/108 mmHg. There was no pleural effusion or congestion in the chest radiograph (*Figure 1A*). Electrocardiography showed sinus rhythm with a complete right bundle branch block (CRBBB) and no significant ST-segment changes (*Figure 1B*). His initial serum troponin

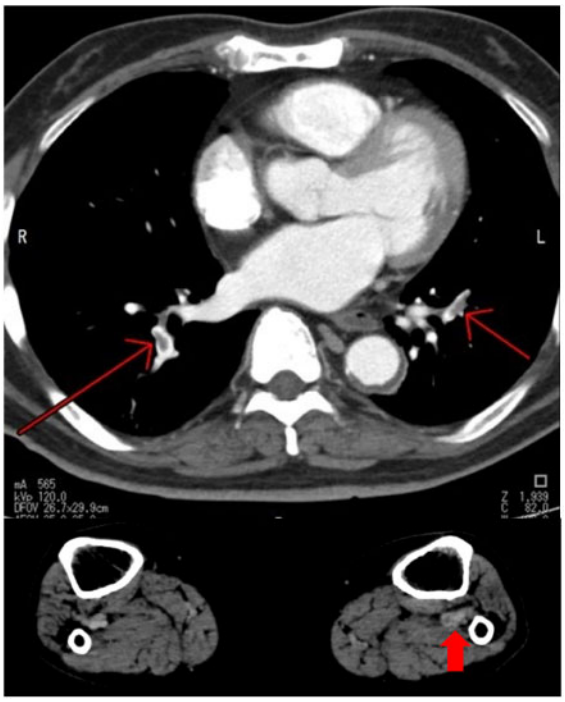


Figure 2 Enhanced computed tomography scan in the axial plane and in the soft tissue window demonstrating bilateral scattered pulmonary thromboembolism and left deep vein thrombus in the soleus vein (red arrows).

l level was 0.05 ng/mL (normal value: <0.04 ng/mL) and the d-dimer level was 14.7 μg/mL (normal value: <1.0 μg/mL). White blood cell count was 12 900/μL (normal value: 4000–7500/μL) and brain natriuretic peptide was 110 pg/mL (normal value: <18.4 pg/mL); other laboratory values were within normal range. The echocardiogram did not show any wall motion abnormality, significant valve disease, right ventricle dilation, or obvious congenital heart disease such as a patent foramen ovale. His chest CT with contrast showed bilateral scattered PE and left deep vein thrombus (from popliteal vein to soleus vein) (*Figure 2*). After admission, his chest pain persisted, and the serum troponin I level and ECG were checked repeatedly. Although his serial ECG did not show no significant changes (*Figure 1C*), his serum troponin I level 3 h after the admission was elevated to 0.90 ng/mL. Furthermore, reconstruction of the SDCT (IQon Spectral CT; Philips healthcare) images clearly showed a perfusion defect of the posterolateral left ventricle myocardium (*Figure 4C*), suggesting a combination of PE and non-ST-segment elevation myocardial infarction (NSTEMI).

Following these findings, coronary angiogram was then performed which showed total occlusion of the OM branch. Although atherosclerotic plaques were seen in multiple coronary arteries, significant stenosis was not observed except for the OM branch (*Figure 3A and Video 1*). Consequently, percutaneous coronary intervention (PCI) was performed to the occlusion site. After thrombectomy, intracoronary imaging using intravascular ultrasound was performed, and atherosclerotic plaque lesions were observed (*Figure 3B*). Two drug-eluting stents were deployed (Synergy 2.75/28 mm and Synergy 2.25/20 mm), and Thrombolysis in Myocardial Infarction 3 flow was achieved (*Figure 3C and Video 2*). The creatine kinase (CK) and CK-MB peaked at 2034 and 278 IU/L (normal values: CK 60–287 IU/L and CK-MB <25 IU/L) 8 h after the admission.

Following treatment of ACS, we began treatment for PE with an oral anticoagulant (OAC, rivaroxaban 30 mg/day).[11] He was relieved of his chest symptoms and was rescued from a hypoxic state. He was discharged on the 14th day after admission. Enhanced SDCT and associated reconstructed images of effective atomic number (Z-effective) 6 months after the treatment started, until when dual antiplatelet therapy (aspirin 100 mg and clopidogrel 75 mg/day) and OAC therapy (rivaroxaban 30 mg/day for 3 weeks and 15 mg/day after 3 weeks) were continued, showed improvement in perfusion defects in both pulmonary fields (*Figure 4A and B*) and the myocardium (*Figure 4C and D*). His treatment was deescalated to single antiplatelet therapy (clopidogrel 75 mg/day) and OAC,[12] and he has had a good course without recurrence.

Discussion

Generally, the risk of PE as venous thrombosis and the risk of ischaemic heart disease, which is mainly due to plaque rupture or atherosclerosis, do not necessarily match. In this case, he had a cancer-bearing status as a risk factor of PE, with multiple coronary risk factors. Furthermore, his prostate cancer had been treated with bicalutamide, a non-steroidal anti-androgen. Although the risk of thrombosis with this drug is lower than that with conventional steroid-based drugs, we cannot deny the thrombotic effects of the drug. The presence of multiple coronary risk factors and arteriosclerotic lesions might be related to the coronary plaque rupture. We believe that the presence of multiple risk factors and the anti-androgen therapy led to the onset of the rare combination in our patient.

Acute chest pain is one of the main reasons for presentation to the emergency department. The diagnostic usefulness of CCTA for patients presenting with acute chest pain to the emergent department has been established.[13] This case showed sinus rhythm within normal heart rate range which might enable to take CCTA and it might be possible to take less time for reperfusion. Since D-dimer level was high and ECG and the serum troponin I level were within normal range, we did not strongly doubt ACS. If SDCT was taken in ECG synchronization, it might be possible to reduce reperfusion time.

Clinical applications of SDCT have been widely reported, and the usefulness of its application in cardiovascular disease has also been documented.[14–16] Reconstruction yields a colour map of iodine content within tissues. The iodine distribution is determined based on the unique X-ray absorption characteristics at different kilovoltage levels. The reconstruction of the image was helpful for the recognition of the perfusion defect and may be beneficial for diagnosis of myocardial infarction.

We could not diagnose ACS immediately due to the lack of a troponin level elevation in the initial test or ECG changes in the serial tests, and CRBBB made it more difficult to detect ST-segment changes. Moreover, PEs show similar symptoms to ACS. Although biomarkers such as CK, CK-MB, and troponin I are measured in patients with chest pain, troponin I becomes positive 3–4 h after the

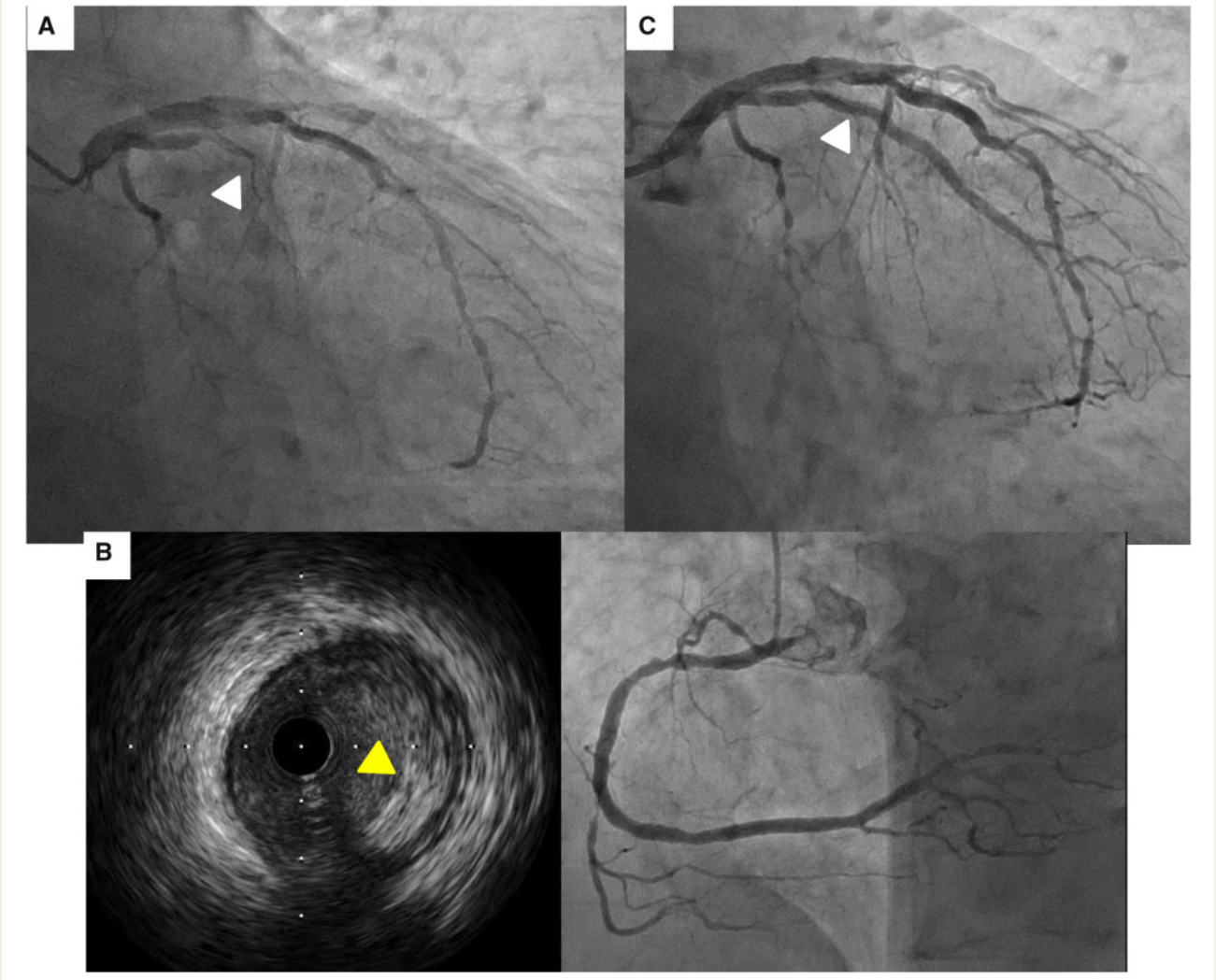


Figure 3 (*A*) Coronary angiography demonstrating atherosclerotic plaques in multiple coronary arteries and total occlusion of the obtuse marginal branch (white triangles). (*B*) Intravascular ultrasound after thrombectomy demonstrating atherosclerotic plaque lesions in the middle of the obtuse marginal branch. (*C*) Percutaneous coronary intervention was performed to the occlusion site and two drug-eluting stents were deployed, and Thrombolysis in Myocardial Infarction 3 flow was achieved.

onset and CK becomes positive 4–6 h after the onset. Therefore, it is not necessarily useful for diagnosis of the hyper-acute phase of ACS. The diagnosis of ACS is sometimes challenging when ECG does not show any significant ST-segment changes, especially when the culprit lesion is in the left circumflex artery and/or in the side branch of a main coronary artery.[17,18] We thought that this case was an ACS of the OM branch, which did not have a comparatively large perfusion area. We also thought that this was not a case of Type 2 myocardial infarction due to acute PE, because (i) there was further increase in myocardial deviant enzymes, (ii) thrombectomy in PCI improved the coronary flow of the OM branch, and (iii) the perfusion defect corresponding to the region of the OM branch improved in the chronic phase. These facts support that the complete blockage of coronary flow from the thrombotic formation by a plaque rupture of OM branch, rather than myocardial injury due to hypoxia; furthermore, we think that it occurred at approximately the same time as the onset of PE.

Assessment of myocardial perfusion may enhance the ability of CT to detect ACS in patients with acute chest pain.[19] In this case, *Figure 2* shows a mild perfusion defect in the posterolateral region of the myocardium; but we cannot deny the possibility of contrast non-uniformity and cannot definitely diagnose a myocardial infarction. By incorporating the reconstruction of effective atomic number images of the thoracic region, it is possible to arrive at a diagnosis.

Furthermore, the PE lesions were significantly improved by the administration of OAC and we could confirm the remarkable improvement of the perfusion defects in the SDCT images. Spectral detector-based CT was also able to identify pulmonary perfusion defects. The diagnostic accuracy of SDCT in PE has also been reported.[20,21]

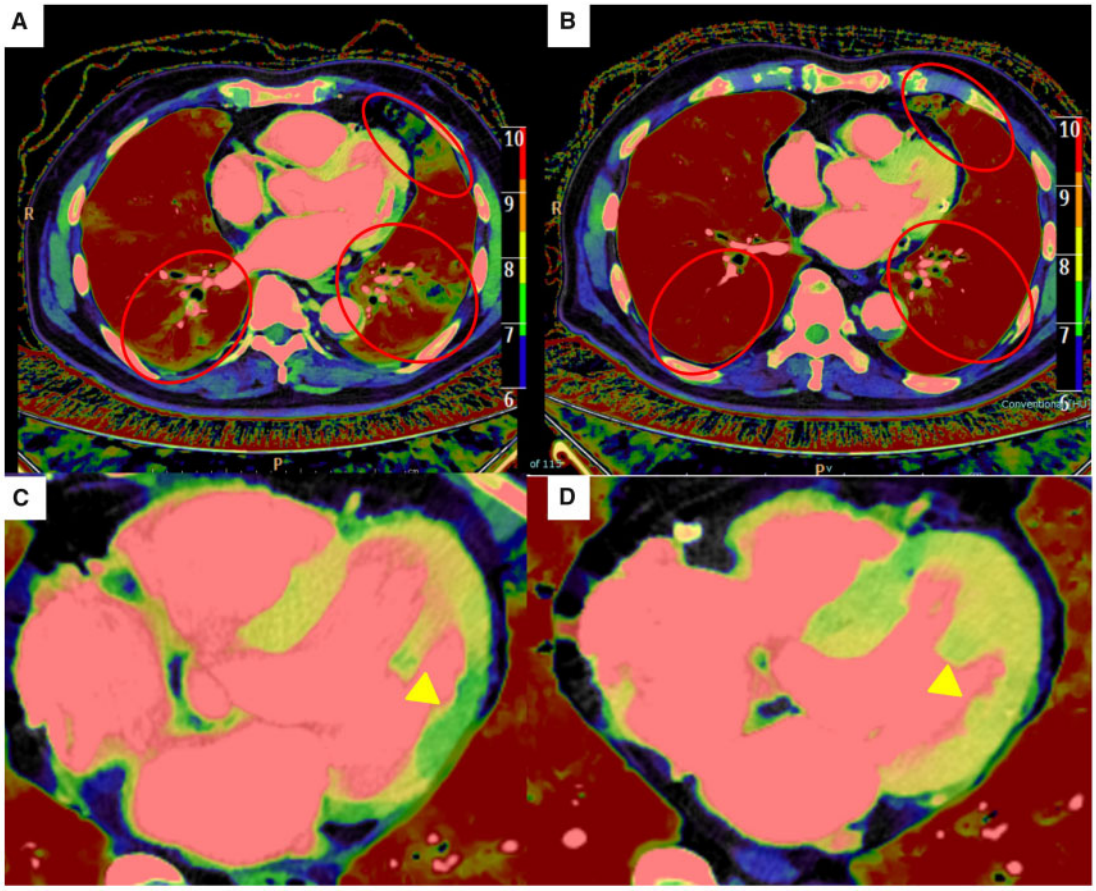
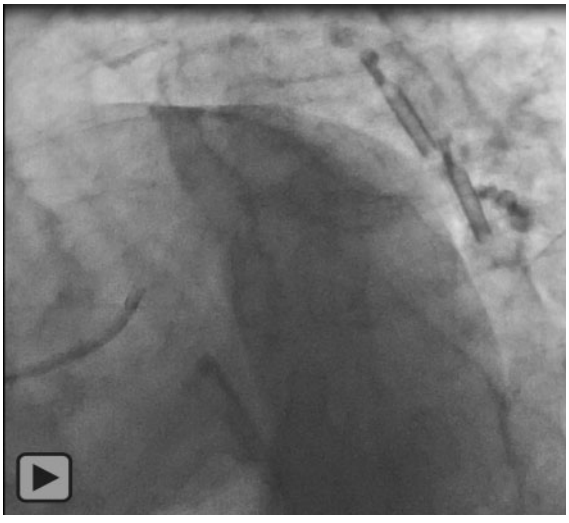
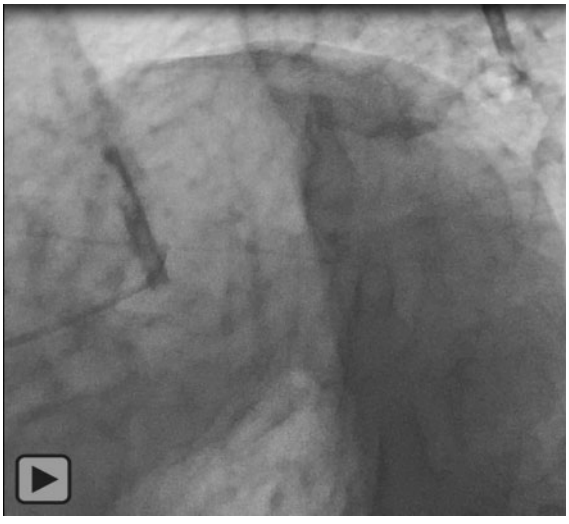


Figure 4 The reconstructed effective atomic number (Z-effective) images of enhanced spectral detector-based computed tomography demonstrating the improvement of the perfusion defects both pulmonary fields (red circles) and posterolateral left ventricle myocardium (yellow triangles). (*A*) Pulmonary fields at the admission. (*B*) Pulmonary fields 6 months after the treatment started. (*C*) Myocardium at the admission. (*D*) Myocardium 6 months after the treatment started.



Video 1 CAG before PCI. Left coronary angiography in the left anterior oblique caudal view demonstrating total occlusion of the obtuse marginal branch.



Video 2 CAG after PCI. Percutaneous coronary intervention was performed to the occlusion site and two drug eluting stents were deployed, and TIMI 3 flow was achieved.

Non-ST-segment elevation myocardial infarction ACS can sometimes be difficult to diagnose accurately, especially in the hyper-acute phase or in the OM branch where changes in physiological tests are less likely to be reflected. The reconstruction of spectral images from contrast medium-enhanced SDCT even without ECG synchronization was helpful to diagnose this unique combination of PE and NSTEMI and may be useful for evaluating the therapeutic effects.

Lead author biography

Rie Aoyama is a head physician in the Cardiology Department of Tokyo Metropolitan Geriatric Hospital and an interventional cardiologist with over 15 years of experience in the field. Her clinical interests include complex PCI, coronary imaging, TAVR, and interventional approach for hypertrophic cardiomyopathy. She was graduated from Nippon Medical graduate school in 2016, PhD in Coronary imaging and interventional approach for hypertrophic cardiomyopathy. Her research interests include intra-left ventricular blood flow and myocardium metabolism in hypertrophic cardiomyopathy and valvular heart disease and exploring sex-based differences in valvular heart disease.

Supplementary material

Supplementary material is available at *European Heart Journal - Case Reports* online.

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

Consent: The author/s confirm that written consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: none declared.

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