

The value of coronary CT angiography in the evaluation of non-ST-elevation myocardial infarction

A 48-year-old man presented at the emergency department with a short episode of chest pain. His medical history was unremarkable. Besides smoking, there were no risk factors for coronary artery disease. The ECG and biomarker levels at presentation were normal.

As part of a quick rule out protocol, coronary CT angiography (CCTA) was performed. The scan showed multiple calcifications in the left anterior descending (LAD), left circumflex artery (LCX) and including a partially calcified plaque in the proximal LAD. The distal part of this plaque showed a very low attenuation profile with evidence of intraplaque dye penetration. This

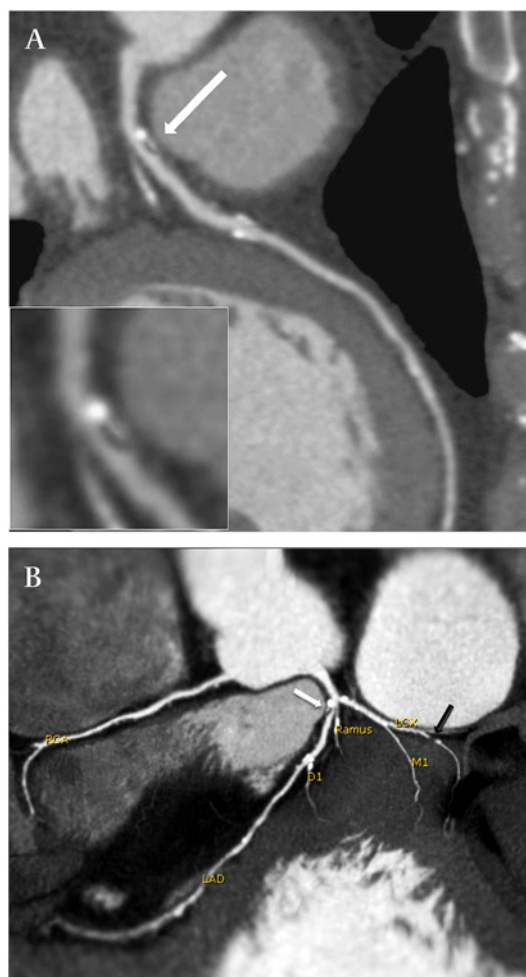


Figure 1 Coronary CT angiography at admittance. (A) Multiplanar reconstruction of the left anterior descending (LAD). In the proximal part of the vessel, a moderate partially calcified stenosis is present. The distal part of the lesion has a low attenuation profile and intradye penetration which suggests ulcerated plaque (white arrow). (B) 2D reconstruction. The black arrow shows a severe non-calcified lesion in the left circumflex artery (LCX) without any signs of instability.

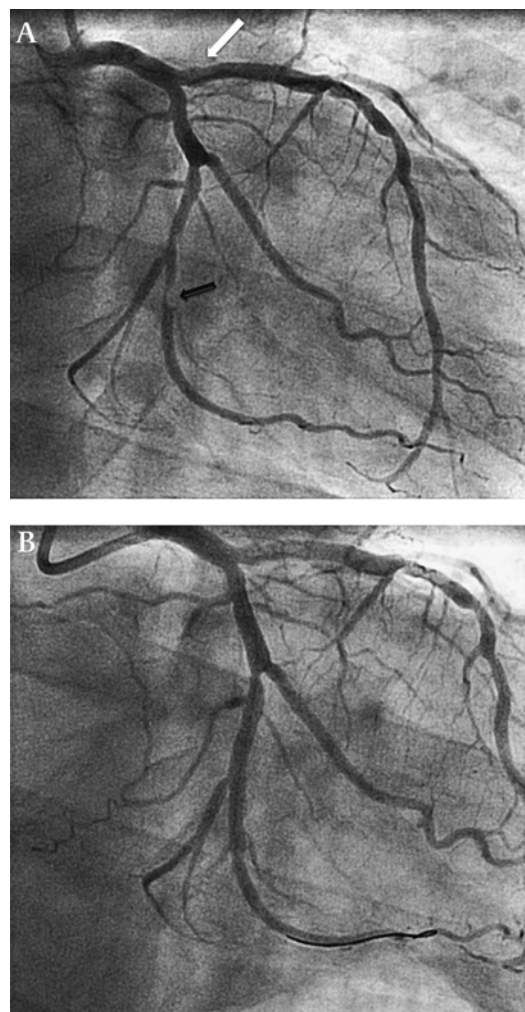


Figure 2 Coronary angiography performed 72 h after admittance. (A) Small indentation of the proximal LAD (white arrow). The lesion in the LCX is severe and treated by percutaneous coronary intervention (black arrow). (B) Result after percutaneous coronary intervention of the LCX.

pattern of a ring-like enhancement is considered to be suggestive of an ulcerated plaque (figure 1A). In addition, a severe non-calcified plaque was detected in the LCX (figure 1B). The patient was admitted to the hospital, and pharmacological treatment for non-ST-elevation myocardial infarction (NSTEMI) was commenced. This included anticoagulation and dual antiplatelet therapy. Repetitive troponin test results were positive. Coronary angiography was performed as part of an early invasive strategy after 72 h of pharmacological treatment.¹ Only a slight indentation in the LAD was observed this time (figure 2A). The lesion in the LCX was confirmed and treated by percutaneous coronary intervention (figure 2B). The clinical course was furthermore uneventful, and the patient was discharged the same day.

Although CCTA is the most accurate non-invasive test in ruling out coronary artery disease, its specificity has been questioned because CCTA has the tendency to overestimate the severity of disease.² This case illustrates the potential ability of CCTA to identify the culprit lesion by plaque characterisation during the initial presentation of a patient with suspected NSTEMI. Irregular or ulcerated plaques detected by CCTA are

more likely to represent ruptured and clinically unstable lesions.³ In contrast to conventional angiography, CCTA provides the ability to visualise the plaque itself with new possibilities for plaque characterisation.⁴ Lesions with low Hounsfield units, positive remodelling and spotty calcifications are thought to be prone to unstable ischaemic complications.⁵ However, it still remains difficult to differentiate between a soft plaque with a high lipid content, a thrombus or an (beam hardening) artifact.

This case demonstrates the supplementary ability of CCTA compared with conventional angiography in patients with acute chest pain. It was hypothesised that the ulcerated plaque in the LAD had stabilised after several days of anticoagulant therapy. Therefore, it was decided to leave the LAD untouched and perform a percutaneous coronary intervention of the severe stenotic lesion in the LCX. In conclusion, the early use of CCTA in patients with suspected NSTEMI may give important insight in the pathophysiology of an acute event by plaque characterisation and thereby identify the culprit lesion. Whether the specificity of lesion characterisation using CCTA in patients with NSTEMI is high enough to change clinical routines should be assessed by future research.

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REFERENCES

1. **Riezebos RK**, Ronner E, ter Bals E, *et al*; OPTIMA trial. Immediate versus deferred coronary angioplasty in non-ST-elevation acute coronary syndromes. *Heart* 2009;**95**:807–12.
2. **Schuetz GM**, Zacharopoulou NM, Schlattmann P, *et al*. Meta-analysis: noninvasive coronary angiography using computed tomography versus magnetic resonance imaging. *Ann Intern Med* 2010;**152**:167–77.
3. **Ambrose JA**, Winters SL, Arora RR, *et al*. Angiographic evolution of coronary artery morphology in unstable angina. *J Am Coll Cardiol* 1986;**7**:472–8.
4. **Madder RD**, Chinnaiyan KM, Marandici AM, *et al*. Features of disrupted plaques by coronary computed tomographic angiography: correlates with invasively proven complex lesions. *Circ Cardiovasc Imaging* 2011;**4**:105–13.
5. **Motoyama S**, Sarai M, Harigaya H, *et al*. Computed tomographic angiography characteristics of atherosclerotic plaques subsequently resulting in acute coronary syndrome. *J Am Coll Cardiol* 2009;**54**:49–57.