CASE REPORT

Virtual histology assessment of coronary atheroma influences treatment strategy in the young acute coronary syndrome patient

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SUMMARY

A 43-year-old woman having significant risk factors for ischaemic heart disease was admitted with an acute coronary syndrome (ACS). Coronary angiography revealed a non-flow limiting lesion in her right coronary artery with the rest of her arteries unremarkable. Risk stratification of the culprit lesion in the right coronary artery through intravascular ultrasound virtual histology demonstrated that the rupture plaque had less than 5% necrotic core with low vulnerability indices. This important finding suggested that the re-rupture risk was low so aggressive pharmacological treatment that can influence the plaque characteristics was instigated in preference to mechanical plaque sealing with a coronary stent. At a year of follow-up the patient was well and had no further events.

BACKGROUND

Non-flow limiting coronary lesions are not an uncommon finding on angiography in patients with acute coronary syndrome (ACS). Often this presents a dilemma with regard to implanting a stent particularly in the young otherwise fit patient. Our case demonstrates that risk stratification by assessing the plaque composition using virtual histology can be a useful tool to guide the modality of treatment.

CASE PRESENTATION

A 43-year-old woman was admitted via the emergency department with a 2 h history of retrosternal chest pain, on minimal exertion. On arrival she was haemodynamically stable. Her risk factors were hypercholesterolaemia, hypertension, previous smoking and a positive family history of coronary disease. Her pain resolved after the instigation of treatment for ACS with dual antiplatelet therapy (aspirin 300 mg and clopidogrel 600 mg) together with sublingual nitrates and opiate analgesia. Her medical history was otherwise unremarkable and she was not on any regular medications.

INVESTIGATIONS

- ▶ ACS
- ▶ Coronary or oesophageal spasm
- ▶ Myo/pericarditis
- ▶ Pulmonary embolus

DIFFERENTIAL DIAGNOSIS

- ▶ ECG revealed sinus rhythm at a rate of 75 bpm. Normal axis without any ischaemic changes.

- ▶ Chest X-ray was unremarkable with clear lung fields and no cardiomegaly.
- ▶ Cardiac enzymes (troponin T) were elevated at 136 ng/L (normal range <40 ng/L) rising to 462 ng/L at 6 h.
- ▶ Full blood count and electrolytes were normal.
- ▶ Cholesterol 7.59 mmol/L.
- ▶ Echocardiography revealed a structurally normal heart with good left ventricular systolic function and no wall motion abnormality. There was no evidence of pericardial disease or right heart strain consistent with pulmonary embolism.

TREATMENT

After stabilisation an urgent coronary angiogram was performed with the intention for percutaneous intervention on significant coronary disease if present. The procedure was performed using a 6 Fr sheath from the right radial artery, having given heparin 70 units/kg. This revealed that her left main stem was normal. The left anterior descending artery was a large-calibre vessel with no significant disease. The circumflex artery was non-dominant, smooth and unobstructed. The proximal right coronary artery had a 40–50% diameter stenosis (assessed visually and compared with the proximal reference diameter; figure 1). This was considered the likely source of her presentation.

Virtual Histology Intravascular Ultrasound (VH-IVUS) analysis (Eagle Eye, Boston Scientific, Natick, Massachusetts, USA) revealed the following characteristics: plaque burden 44%, necrotic core 5% (non-confluent), fibrous 11%, fibrofatty 27%, calcification 1%; figure 2).

As the lesion did not have the appearance of a vulnerable plaque and was non-flow limiting, a conservative medical approach was adopted instead of percutaneous coronary intervention with a stent. She was kept on dual antiplatelet therapy (aspirin 75 mg daily and clopidogrel 75 mg daily) for 1 year and was aggressively managed with high-potency statin (atorvastatin 80 mg once every night) and her blood pressure was controlled prior to discharge with an ACE inhibitor (ramipril 1.25 mg daily) and bisoprolol (1.25 mg daily) with a plan to uptitrate as tolerated.

OUTCOME AND FOLLOW-UP

At 1-year follow-up the patient remained asymptomatic and her clopidogrel was discontinued. Her other cardiovascular drugs were continued as planned.

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Figure 1 (A) Coronary angiography of the right coronary artery (LAO projection) showing a moderate stenosis of the mid-vessel (indicated by arrow). (B) RAO projection of the same lesion (arrow).

DISCUSSION

Premature coronary artery disease in patients under the age of 50 is often underdiagnosed. This is because such patients generally present atypically and also there may be a tendency to dismiss such symptoms as non-cardiac. Often, patients are put on a proton-pump inhibitor by their general practitioners at presentation as this is a more common differential diagnosis. A normal resting ECG does not exclude myocardial ischaemia as was apparent in this patient. Biomarkers (high-sensitivity troponin measurement) have revolutionised the triage of these patients for further investigation. However, biomarkers, although specific for cardiac muscle damage, may be raised in conditions such as pericarditis or pulmonary embolism. Echocardiography is very useful in the assessment of these differential diagnoses, with right ventricular dilation and strain in the latter. It is also useful in assessing wall motion abnormalities, which are suggestive of coronary ischaemia or previous myocardial infarction.

Coronary angiography remains the gold standard to evaluate obstructive coronary lesions that may lead to an ACS. However, lumenography has its limitations, since the nature of the atherosclerotic process lies within the vascular wall. The invasive and non-invasive imaging modalities have helped to gain an understanding of the pathogenesis when married with postmortem specimens. CT coronary angiography, although non-invasive, suffers from the inability to proceed directly to treatment of lesions that

are found. Intravascular ultrasound scanning can circumvent this limitation, with grey scale IVUS enabling lesions of diameter stenosis $>70\%$ to be managed directly with stenting. Lesions between 50% and 70% have often created a dilemma for the intervention cardiologist. This is because there is a risk of treating safe, stable lesions that would never cause a problem in the future, thereby subjecting the patient to a medical device (the stent) with the potential for restenosis in time, but more importantly the risk of sudden cardiac death due to thrombosis.

VH-IVUS analysis has been shown to be effective in demarcating plaques which are vulnerable to rupture or those that have previously ruptured that may potentially rupture again. Support for this comes from a prospective study of the natural history of lesions of different types (the PROSPECT study, wherein 697 patients underwent assessment of their full coronary tree including VH-IVUS and were then followed up over a median of 3.4 years.¹ Thin-capped fibroatheroma were particularly prone to cause late major adverse cardiac events. This result was confirmed by the VIVA study wherein 170 patients undergoing percutaneous coronary intervention for ACS or stable angina were assessed preprocedure and postprocedure. VH-IVUS was able to predict the type of plaque that was likely to rupture over a median follow-up of 625 days.² From these studies, vulnerability is suggested by a necrotic core $>10\%$ with an overall plaque burden (size of plaque compared with vessel lumen) $>40\%$.

Figure 2 (A) Virtual Histology Intravascular Ultrasound (VH-IVUS) image taken at the time of angiography. Different colours represent different material types within the lesion (green—fibrotic, yellow—fibrofatty, pink—necrotic, blue—calcific). (B) Line representation of the vessel and plaque showing necrotic areas (filled grey).

In our patient, although the plaque burden was 44%, the necrotic core was only 5% and was not adjacent to the vessel lumen. This suggested that it had a low probability of rupture and hence the risk of stenting outweighed the benefit. Studies have shown that these patients do benefit from high-dose statin

therapy which may result in modulating the plaque characteristics and lipid profile within the plaque itself.³ On the basis of this, we elected to aggressively manage our patient medically rather than implanting a stent.

Contributors JO and TJ wrote the paper under the supervision of SR who did the case.

Competing interests None.

Patient consent None.

Provenance and peer review Not commissioned; externally peer reviewed.

Learning points

- ▶ Premature coronary artery disease is underdiagnosed, particularly in women.
- ▶ Biomarkers can stratify the differential diagnosis.
- ▶ Coronary angiography does not demarcate plaque vulnerability but only gives a visual estimation of restriction of flow.
- ▶ Virtual Histology Intravascular Ultrasound analysis can help to determine plaque vulnerability and direct subsequent management.

REFERENCES

- 1 Stone GW, Maehara A, Lansky AJ, *et al.* PROSPECT investigators: a prospective natural-history study of coronary atherosclerosis. *N Engl J Med* 2011;364:226–35.
- 2 Calvert PA, Obaid DR, O'Sullivan M, *et al.* Association between IVUS findings and adverse outcomes in patients with coronary artery disease: the VIVA (VH-IVUS in vulnerable atherosclerosis) Study. *JACC Cardiovasc Imaging* 2011;4:894–901.
- 3 García-García HM, Klauss V, Gonzalo N, *et al.* Relationship between cardiovascular risk factors and biomarkers with necrotic core and atheroma size: a serial intravascular ultrasound radiofrequency data analysis. *Int J Cardiovasc Imaging* 2012;28:695–703.

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