

Lipid-Rich Plaque Masquerading as a Coronary Thrombus

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A 43-year-old woman presented with exertional chest pressure. Right coronary angiography showed a clear filling defect. Intravascular ultrasound revealed a plaque with 80% stenosis and a large lipid pool. Therefore, a stent was placed, and the patient became angina-free. Lipid-rich plaques are a cause of angiographic filling defects. Intravascular ultrasound is an integral part of coronary artery evaluation.

Keywords: Coronary calcification; Intravascular ultrasound; Lipid plaques and filling defects; Percutaneous coronary intervention

Coronary artery thrombosis was recognized as an important cause of myocardial infarction nearly a century ago.¹ With the introduction of coronary angiography, filling defects were identified and associated with coronary artery thrombosis.^{2,3} The presence of filling defects caused by coronary artery stenosis is associated with adverse cardiac events,⁴ may resolve with thrombolytic therapy⁵ and has important prognostic implications.⁶ Filling defects can be due to other causes, however. In this brief report, we describe a case of a lipid-rich coronary plaque presenting as an angiographic filling defect.

Case Report

A 43-year-old woman presented to the hospital with several days of exertional chest pressure. On the day of admission, her chest discomfort increased in frequency and severity. She experienced no discomfort at rest. Previous medical history included hypertension, diabetes mellitus, dyslipidemia and hypertriglyceridemia. The patient's medications on admission were 81 mg of aspirin once per day, 145 mg of Tricor once per day and 100 mg of metoprolol twice per day. Physical examination revealed a blood pressure of 176/82 mm Hg and a heart rate of 67 beats/min. Jugular venous pressure was normal, lungs were clear and cardiac examination was normal. A twelve-lead electrocardiogram was normal, and a computerized tomography scan was negative for pulmonary embolism. A dobutamine stress echocardiogram was normal, yet the patient's classic anginal symptoms were reproduced during the test. With failure of empiric medical therapy and continuation of typical anginal chest pains, coronary angiography was performed. The left coronary artery was essentially normal, and the right coronary angiogram showed a filling defect

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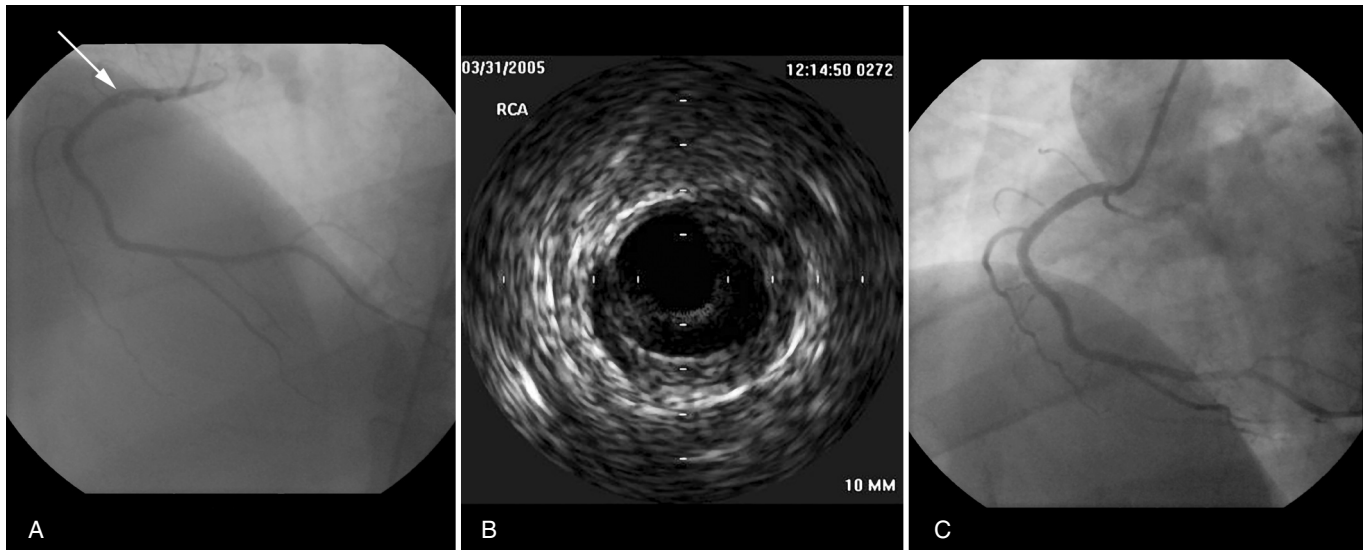


Figure 1. (A) Right coronary artery with a filling defect (arrow). (B) Intravascular ultrasound showing a stenosis with 80% occlusion and a large lipid pool. (C) Right coronary artery following placement of a coronary stent.

(figure 1A). Intravascular ultrasound revealed a plaque with 80% stenosis and a large lipid pool (figure 1B). A drug-coated stent was placed with good results, and the patient became angina-free (figure 1C). Clopidogrel was prescribed for 1 year.

Discussion

Acute coronary syndrome invariably results from an inflammatory process at the site of a vulnerable plaque. The plaque may rupture exposing the circulating blood to collagen, tissue factors and a variety of substances, and thrombus formation may result. During angiography, it is important to identify thrombus, which is defined as the presence of a filling defect within the coronary artery lumen with contrast around that defect and an absence of calcium within the defect.⁷ The presence of filling defects during angiography has been found to be a marker for restenosis⁷ and a surrogate for generalized atherosclerotic disease.⁸ It has also been found to be important for acute occlusion of a coronary artery following balloon angioplasty.⁹ Thus, the presence of filling defects on angiography may assist the clinician in making therapeutic and interventional decisions during the management of such patients.¹⁰

Not all filling defects identified during angiography are associated with thrombus. Two modalities, ultrasound and coronary angioscopy, have been utilized to investigate the accuracy of coronary angiography in detecting coronary thrombi identified as filling defects. Kotani et al¹¹ reviewed the angiographic findings of 4,083 coronary lesions. Filling defects were present in 6.8% of those reviewed. Intravascular ultrasound was performed prior to angiography in 78 of these patients, and it was found that of those with angiographically-identified filling defects only 61.5% showed evidence of thrombus on intravascular ultrasound. The others were due to a variety of causes, the most common being calcified plaques. Multiple studies have also been done utilizing angioscopy as the gold standard and have revealed

that angiography has a low sensitivity for detecting thrombus with a predictive value of about 70%.¹²⁻¹⁴

Other coronary conditions can mimic coronary thrombosis and appear as filling defects. The presence of significant calcium in a coronary plaque may present as a filling defect.¹¹ Whether the filling defect is a thrombus or a calcified plaque is crucial for planning intervention. While diffuse calcium can generally be seen during angiography, a localized calcified plaque may not necessarily be obvious. Coronary dissection¹⁵ or coronary embolism¹⁶ may also present as filling defects. Pseudo-thrombosis is a term that refers to filling defects resulting from collateral blood flow.^{17,18} In these cases, when the stenosis is relieved, the collateral flow will reverse, and the filling defects will be eliminated. Another condition that may present as a filling defect is woven coronary artery,¹⁹ a rare congenital anomaly in which the coronary artery branches into thin channels that then merge to form a normal vessel. Recognizing this condition is very important, performing an intervention at the site of a woven coronary artery will undoubtedly result in arterial rupture. Figure 2 summarizes the various conditions that may present as filling defects and mimic coronary thrombosis.

Since the presence or absence of a coronary artery thrombus is important for planning intervention, classic angiography identifying a filling defect may often be followed by intravascular ultrasound for a more accurate diagnosis.²⁰ Although angioscopy is a more definite tool for differentiation, it is not readily available in most catheterization laboratories.

This brief report presents yet another cause for filling defects, namely lipid-rich plaques. Intravascular ultrasound proved instrumental in its diagnosis and led to definitive management of this condition in our patient.

Coronary Filling Defects

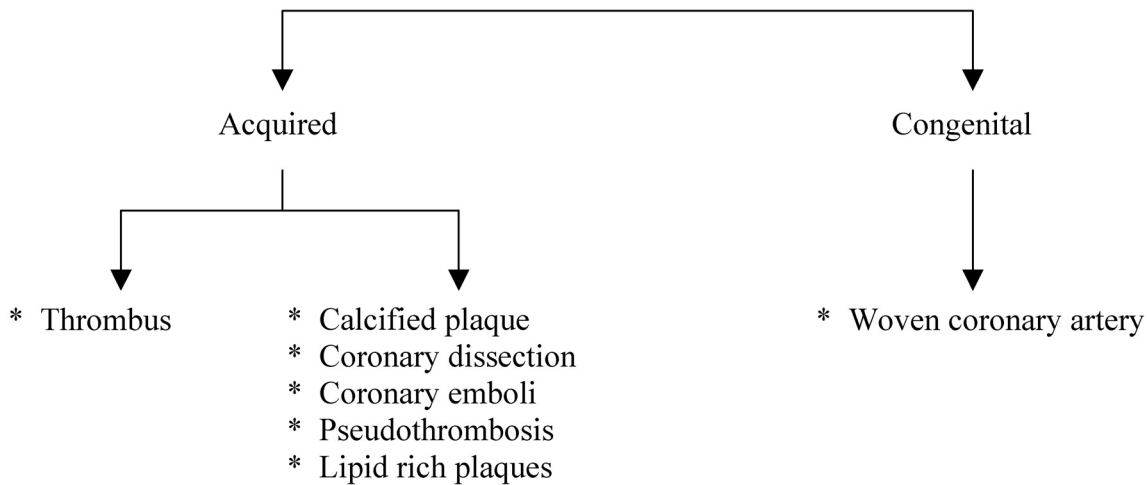


Figure 2. Coronary filling defects flow chart.

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References

1. Herrick JB. Clinical features of sudden obstruction of the coronary arteries. *JAMA* 1912;59:2015-2019.
2. Kranjec I, Delaye J, Didier B, Delahaye F, Grand A. Angiographic morphology and intraluminal coronary artery thrombus in patients with angina pectoris: clinical correlations. *Eur Heart J* 1987;8:106-115.
3. Rehr R, Disciascio G, Vetrovec G, Cowley M. Angiographic morphology of coronary artery stenoses in prolonged rest angina: evidence of intracoronary thrombosis. *J Am Coll Cardiol* 1989;14:1429-1437.
4. Davies SW, Marchant B, Lyons JP, Timmis AD, Rothman MT, Layton CA, Balcon R. Irregular coronary lesion morphology after thrombolysis predicts early clinical instability. *J Am Coll Cardiol* 1991;18:669-674.
5. Davies SW, Marchant B, Lyons JP, Timmis AD, Rothman MT, Layton CA, Balcon R. Coronary lesion morphology in acute myocardial infarction: demonstration of early remodeling after streptokinase treatment. *J Am Coll Cardiol* 1990;16:1079-1086.
6. Ambrose JA. Prognostic implications of lesion irregularity on coronary angiography. *J Am Coll Cardiol* 1991;18:675-676.
7. Violaris AG, Melkert R, Herrman JP, Serruys PW. Role of angiographically identifiable thrombus on long-term luminal renarrowing after coronary angioplasty: a quantitative angiographic analysis. *Circulation* 1996;93:889-897.
8. Triposkiadis F, Sitafidis G, Kostoulas J, Skoularigis J, Zintzaras E, Fezoulidis I. Carotid plaque composition in stable and unstable coronary artery disease. *Am Heart J* 2005;150:782-789.
9. Ellis SG, Roubin GS, King SB 3rd, Douglas JS Jr, Weintraub WS, Thomas RG, Cox WR. Angiographic and clinical predictors of acute closure after native vessel coronary angioplasty. *Circulation* 1988;77:372-379.
10. Williams DO, Topol EJ, Califf RM, Roberts R, Mancini GB, Joelson JM, Ellis SG, Kleiman NS. Intravenous recombinant tissue-type plasminogen activator in patients with unstable angina pectoris. Results of a placebo-controlled, randomized trial. *Circulation* 1990;82:376-383.
11. Kotani J, Mintz GS, Rai PB, Pappas CK, Gevorkian N, Bui AB, Pichard AD, Satler LF, Suddath WO, Waksman R, Laird JR Jr, Kent KM, Weissman NJ. Intravascular ultrasound assessment of angiographic filling defects in native coronary arteries: do they always contain thrombi? *J Am Coll Cardiol* 2004;44:2087-2089.
12. Teirstein PS, Schatz RA, DeNardo SJ, Jensen EE, Johnson AD. Angioscopic versus angiographic detection of thrombus during coronary interventional procedures. *Am J Cardiol* 1995;75:1083-1087.
13. Senneff MJ, Schatz RA, Teirstein PS. The clinical utility of angioscopy during intracoronary stent implantation. *J Interv Cardiol* 1994;7:181-186.
14. Uretsky BF, Denys BG, Counihan PC, Ragosta M. Angioscopic evaluation of incompletely obstructing coronary intraluminal filling defects: comparison to angiography. *Cathet Cardiovasc Diagn* 1994;33:323-329.
15. Mouhayar EN, Blankenship JC, Fenster BD, Iliadis EA, McConnell TR. Coronary artery "pseudothrombus" due to collateral flow artifact distal to left circumflex coronary stenosis. *J Interv Cardiol* 2002;15:425-429.
16. Sharma S, Sundaram U, Loya Y. Selective coronary angiography in intracardiac tumors. *J Interv Cardiol* 1993;6:125-129.
17. Fukuoka Y, Sonoda M, Terashi T, Kawabata K, Sannou K, Uenomachi H, Tanaka Y, Arima T. Collateral blood flow showing dissection-like filling defect on coronary arteriography: a case report. *J Cardiol* 2001;38:281-287.
18. Weinberg BA, Bourdillon PD. Coronary artery pseudothrombus: angiographic filling defects caused by competitive collateral flow. *Cathet Cardiovasc Diagn* 1990;20:196-199.
19. Martuscelli E, Romeo F, Giovannini M, Nigri A. Woven coronary artery: differential diagnosis with diffuse intracoronary thrombosis. *Ital Heart J* 2000;1:306-307.

20. Morocutti G, Spedicato L, Vendrametto F, Bernardi G. Intravascular echocardiography (ICUS) diagnosis of post-traumatic coronary dissection involving the common trunk. A case report and review of the literature. *G Ital Cardiol* 1999;29:1034-1037.

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