Drug-eluting stents (DES) minimized the occurrence of in-stent restenosis but with a penalty of increased risk of late stent thrombosis. In patients with late stent thrombosis, adverse vessel wall reactions such as uncovered and malapposed stent struts, coronary aneurysms, and accelerated neointimal proliferation have been identified. The stent polymer is suspected to induce an inflammatory response, resulting in vessel wall abnormalities.

Identification of patients with an inflammatory response to DES might be essential in preventing late stent thrombosis. Inflammatory processes increase vessel wall permeability, leading to edema, and both native and peri-stent vessel wall inflammation may be associated with tissue edema. Cardiovascular magnetic resonance (CMR) performed with a T2-weighted short-τ inversion recovery sequence (T2-STIR) has recently been shown to identify localized coronary edema in the culprit artery of patients with acute myocardial infarction. Parameters for the ECG-triggered, navigator-gated, dark-blood, T2-STIR fast-spin-echo sequence were as follows: repetition time, 2 RR intervals; echo time, 100 milliseconds; echo train length, twenty 0.68×0.68×8-mm3 voxels; and 2 signal averages. Here we present intervals; echo time, 100 milliseconds; echo train length, twenty

**Patient 1**

A 41-year-old woman experienced very late stent thrombosis 15 months after treatment with DES in the proximal left anterior descending artery (LAD). Coronary angiography showed peri-stent staining in the proximal LAD (Figure 1, left), suggestive of adverse vessel wall reaction. Frequency-domain optical coherence tomography (C7, St. Jude Medical, St. Paul, MN) showed coronary aneurysms in the stented area of the LAD with several thrombi (Figure 1C, middle; Movie I in the online-only Data Supplement). T2-STIR CMR demonstrated localized edema in the stented LAD area and in the native atherosclerotic coronary vessel wall at the proximal LAD and left main coronary artery, supporting an inflammatory cause (Figure 1A–1C, right). The patient was treated with dual antiplatelet therapy indefinitely. Follow-up was performed at 20 months to evaluate whether cessation of dual antiplatelet therapy before necessary, unrelated major surgery was safe. Progression of the vessel wall changes was detected (optical coherence tomography; Movie I in the online-only Data Supplement), and the patient was treated with bypass surgery. Histology of the peri-stent vessel wall verified infiltration by degranulating eosinophils (Figure 1G).

**Patient 2**

A 42-year-old man was treated by DES in the LAD and first diagonal artery. Baseline study optical coherence tomography showed well-apposed stent struts in the LAD (Figure 2C). At the planned 8-month follow-up, the coronary angiography showed extensive peri-stent staining (Figure 2A). Multiple aneurysms in a flower-like pattern were identified by optical coherence tomography (Figure 2D and 2E; Movie II in the online-only Data Supplement). Stent struts were apposed despite the severe vessel wall abnormalities. A high proportion of uncovered stents were identified. T2-STIR CMR at 8 months demonstrated peri-stent edema (Figure 2B).

**Patient 3**

A 61-year-old woman was treated by 3 DES in the right coronary artery and in the LAD. Elective coronary angiography after 4 weeks revealed early peri-stent staining, indicative of aneurysm formation around the implanted stents. Planned coronary angiography after 1 year showed increased peri-stent vessel wall abnormalities.
Figure 1. Coronary angiogram from patient 1 with stent thrombosis after 15 months showing peri-stent staining in the proximal left anterior descending artery (LAD, top). Optical coherence tomography (OCT) (A–C) showed an eccentric fibrotic plaque formation (yellow arrows) in the left main coronary artery (A) and, in the proximal LAD circumflex artery bifurcation, a tangential dropout artifact (yellow star in B). The stented area of the LAD revealed several thrombi (dotted yellow arrows in C) and aneurysm formation (An). The stent struts are highlighted by the yellow arrowheads. $T_2$-weighted short-$\tau$ inversion recovery ($T_2$-STIR) cardiac magnetic resonance (CMR) immediately after the medically treated stent thrombosis demonstrated hyperintense signal intensity indicative of coronary edema (D–F, white arrows) corresponding to the atherosclerotic plaques in the vessel wall of the proximal LAD (E) extending to the left main coronary artery (D). The stented area also demonstrated the presence of peri-stent edema (F). Histology of excised peri-stent vessel (obtained at bypass surgery at 21 months) showed infiltration by eosinophils (G). DES indicates drug-eluting stents.

Figure 2. Coronary angiogram from patient 2 at the 8-month follow-up after treatment by drug-eluting stents (DES) showed substantial peri-stent staining in the proximal left anterior descending artery (LAD; A). $T_2$-weighted short-\(\tau\) inversion recovery ($T_2$-STIR) cardiac magnetic resonance (CMR) showed hyperintense signal intensity (white arrow in B) in the peri-stent area of the proximal LAD, indicating the presence of peri-stent edema (B). Optical coherence tomography (OCT) after percutaneous coronary intervention showed well-apposed stent struts with some stent distortion caused by final kissing balloon dilatation (C). At the 8-month follow-up, OCT showed aneurysm formation between struts, resulting in a flower-like pattern with large extra stent space (D). The extent of sacculate evaginations following the strut pattern was readily visualized by 3-dimensional OCT reconstruction of the stented vessel (E) (QAngioOCT RE, Medis Specials, the Netherlands).
staining (Figure 3, top). CMR at 1 year showed peri-stent tissue edema at the proximal LAD extending to the left main coronary artery, where an eccentric plaque was located (Figure 3A and 3B). The patient underwent subacute coronary bypass grafting. Histological examination of a biopsy from the stented area of the LAD showed substantial peri-strut inflammation with degranulating eosinophils.

These 3 cases represent the first in vivo demonstration of peri-stent edema after treatment by DES by noninvasive, non–contrast-enhanced imaging. Coronary edema detected by T$_2$-STIR CMR might emerge as a surrogate marker for inflammation, thus possibly identifying patients with an increased risk of late coronary stent thrombosis.

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**References**


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Coronary Edema Demonstrated by Cardiovascular Magnetic Resonance in Patients With Peri-Stent Inflammation and Aneurysm Formation After Treatment by Drug-Eluting Stents

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