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Epinephrine Treatment of Anaphylaxis

An Extraordinary Case of Very Late Acute Stent Thrombosis

Colette E. Jackson, BSc (Hons), MRCP; Jonathan R. Dalzell, MRCP; Kerry J. Hogg, MD, FRCP

A 78-year-old man experienced marked angioedema of his face and tongue following ingestion of chocolate-coated peanuts. Paramedics administered 0.5 mg of intramuscular epinephrine within half an hour of symptom onset with rapid relief of symptoms and subsidence of the swelling. On route to the local Emergency Department the patient suddenly became pale, nauseous, and began sweating profusely. There was no chest pain. Blood pressure was 182/105 and heart rate 107 beats per minute. An ECG revealed sinus tachycardia and marked anterior ST elevation (Figure 1), and he was urgently transferred to the regional interventional cardiology center. Aspirin 300 mg and clopidogrel 600 mg were administered before transfer. He had a significant history of coronary artery disease and 4 years previously had undergone percutaneous coronary intervention to the proximal left anterior descending (LAD) and proximal circumflex arteries with bare-metal stents. Three months following this he developed in-stent restenosis in the LAD stent that was treated by further percutaneous coronary intervention with 2 overlapping drug-eluting stents. He experienced infrequent exertional angina over the next 4 years and at the time of this presentation was taking aspirin 75 mg as a sole antiplatelet. There was no history of diabetes, noncompliance with aspirin therapy, or any other medical history suggestive of a hypercoagulable state.

On arrival in the catheterization laboratory 5000 IU heparin was administered intravenously. Coronary angiography showed a large dominant right system supplying collaterals to the circumflex artery. The LAD was occluded midway through the drug-eluting stents (Figure 2 and Supplemental Figure A). The circumflex was also blocked within the bare metal stent but was an unlikely culprit lesion given the anterior ECG changes, and the collateralization provided by the right coronary artery (RCA) to the level of the circumflex stent. A guide wire

was passed to the distal LAD, the artery reopened and obvious focal clot visualized. Thrombus extraction, via an Export aspiration catheter, followed by balloon dilatation to high pressure restored TIMI 3 flow (Figure 3 and Supplemental Figure B) with complete resolution of the ECG changes (Figure 4). Surprisingly, there was no evidence of any significant in-stent restenosis and therefore no stent was deployed. Post-percutaneous coronary intervention medical care included glycoprotein IIb/IIIa inhibitor infusion and a recommendation for life-long dual antiplatelet therapy. The patient made an uncomplicated recovery and was provided with an epinephrine pen predischARGE.

In humans, exogenous epinephrine administration has been shown to promote platelet aggregation¹ by increasing platelet production of thromboxane B₂,² heightening the sensitivity of platelets to ADP² and promoting the thrombin induced binding of platelets to fibrinogen.³ Interestingly, platelets from angina patients are more sensitive to increased endogenous serum catecholamine levels, and thus more prone to aggregation compared with normal controls.⁴

Late and very late-stent thromboses are recognized complications of percutaneous coronary intervention occurring more than 30 days and 1 year, respectively, postprocedure. Discontinuation of antiplatelet therapy is the commonest factor associated with these rare complications. Factors known to be associated with stent thrombosis include, among others, left ventricular systolic dysfunction and index stenting in the setting of acute myocardial infarction, conditions that are both associated with increased circulating catecholamine levels.

We believe that this is the first reported case of acute drug-eluting stents thrombosis induced by exogenous epinephrine administration. The lack of in-stent restenosis in the culprit drug-eluting stents makes this case all the more

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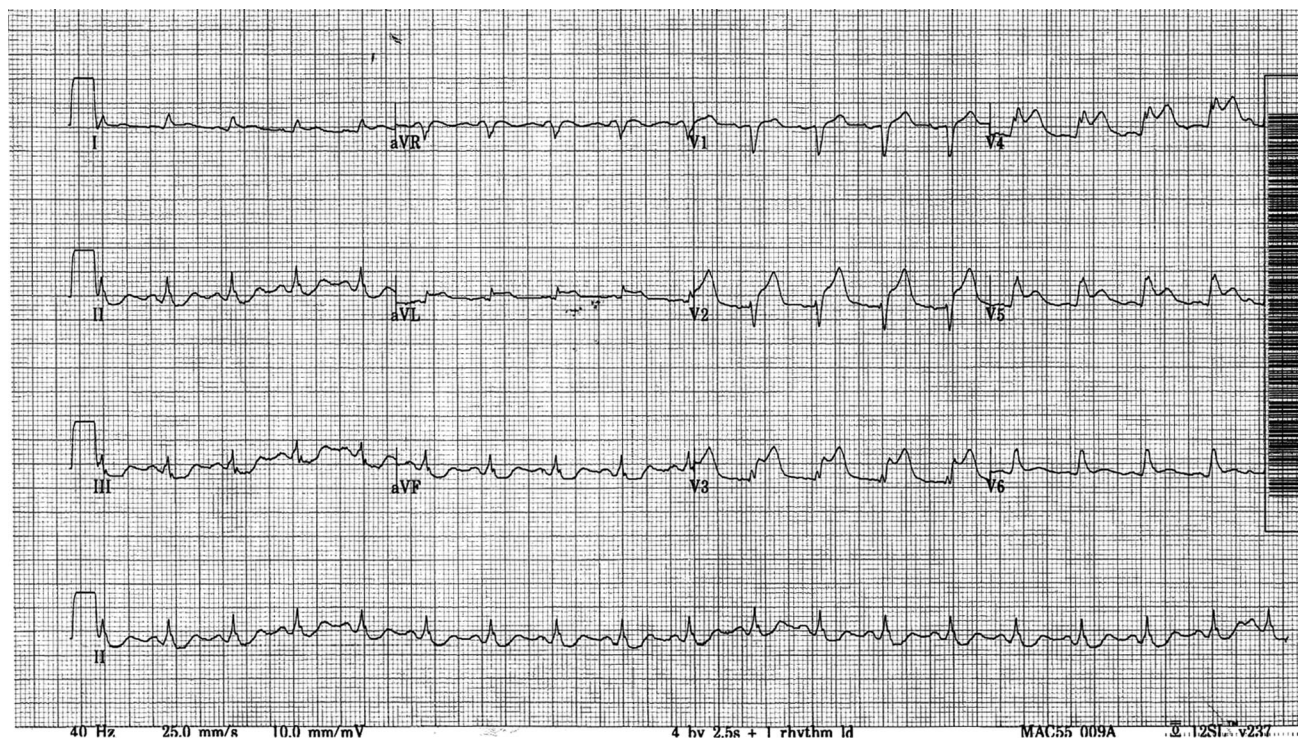


Figure 1. ECG on presentation showing acute anterior ST elevation myocardial infarction.

noteworthy as epinephrine induced occlusion of a significant in-stent restenosis would have been a more expected scenario. This case identifies the need for further work to ascertain any potential role of long-term dual antiplatelet therapy in patients with coronary stents in situ who are likely to require epinephrine therapy for allergic angioedema. Moreover, we are reminded that any stimulus

increasing catecholamine levels, be it exogenous or endogenous (eg, trauma or surgery), can predispose patients to thrombosis, which may be catastrophic for those with coronary stents in situ.

Statement of Responsibility

The authors had full access to and take full responsibility for the integrity of the data. All authors have read and agree to the manuscript as written.



Figure 2. Right anterior oblique view at emergency coronary angiography showing occlusion of the LAD and circumflex stents.

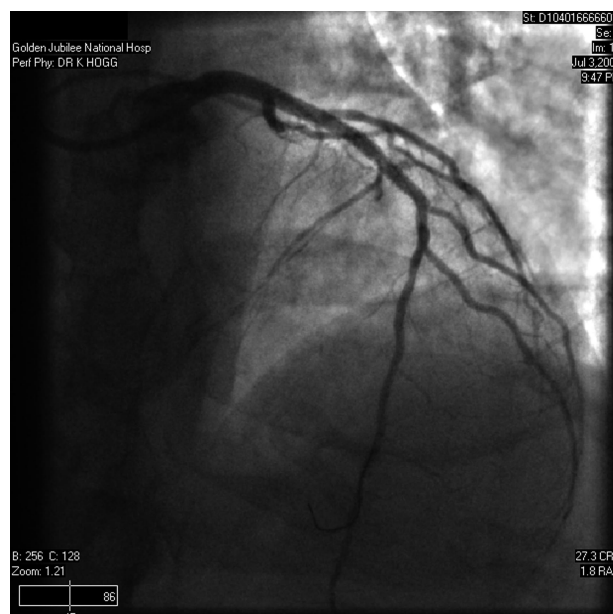


Figure 3. Restoration of flow within the LAD following thrombus extraction.

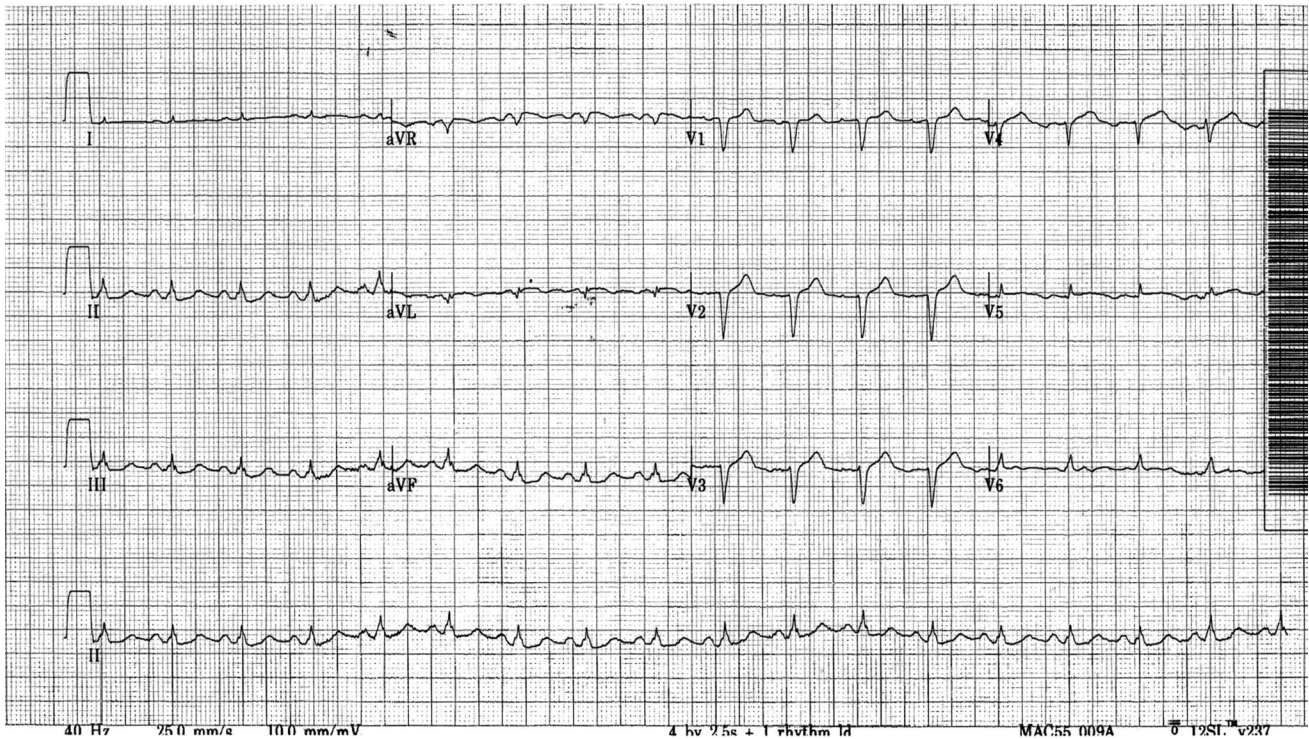


Figure 4. ECG post-LAD recanalization revealing resolution of the anterior ST elevation seen in Figure 1.

Disclosures

None.

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KEY WORDS: myocardial infarction ■ stents ■ thrombosis ■ anaphylaxis ■ epinephrine

Supplementary Material

Figure A (Supplemental file) Moving picture file of right anterior oblique view at emergency coronary angiography showing occlusion of the LAD and circumflex stents.

Figure B (Supplemental file) Moving picture file following thrombus extraction.

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Figure A (Supplemental file) Moving picture file of right anterior oblique view at emergency coronary angiography showing occlusion of the LAD and circumflex stents.

Figure B (Supplemental file) Moving picture file following thrombus extraction.