Cocaine-induced epicardial coronary artery thrombosis resulting in extensive myocardial injury assessed by cardiac magnetic resonance imaging

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Cocaine causes myocardial injury through multiple mechanisms including vasoconstriction, adrenergic hyper-stimulation, and de novo thrombus formation. This report presents two cases of cocaine-induced coronary artery thrombosis with no underlying stenosis, resulting in substantial myocardial injury.

A previously well 32-year-old man presented with chest pain which commenced 24 h after intranasal cocaine use. He denied use of any other eliciting substance, but had previously used cocaine. Twelve-lead electrocardiogram revealed inferior ST segment elevation. Urgent angiography demonstrated thrombotic occlusion of the right coronary artery (Panel A). Coronary aspiration restored TIMI III flow, but no underlying stenosis was identified (Panel B). Troponin peaked at 3409 ng/L (N < 14 ng/L). Cardiac magnetic resonance (CMR) imaging showed inferior akinesis, and extensive transmural late enhancement with microvascular obstruction (MVO: Panels C and D). No late enhancement was observed in other coronary territories.

A 43-year-old man presented with 4 h of chest pain 48 h after first time intranasal cocaine use. He denied other substance use. ST segment elevation in the anterior leads resulted in immediate thrombolysis and transfer. Troponin peaked at 6.5 μg/L (N < 0.03 μg/L). Angiography showed normal coronary arteries (Panel E), but ventriculography demonstrated akinesis of the anterior wall. Cardiac magnetic resonance confirmed this (Panel F) and showed increased T2-signal intensity (Panel G) and transmural late gadolinium enhancement with MVO (Panels H and I) supporting occlusion of his left anterior descending coronary artery as the cause of his presentation, not seen at angiography due to thrombolysis.

In these cases, CMR illustrates extensive myocardial injury resulting from cocaine-induced coronary artery thrombosis without underlying coronary artery disease.

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