Spontaneous occlusion of the left anterior descending artery immediately following coronary angiography

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During coronary angiography of the patients with acute coronary syndrome, exposure of highly thrombogenic subendothelial collagen and local thrombin generation may rarely counteract with the contrast media used resulting promotion of platelet recruitment and degranulation. Final state may result in potentially fatal complications, such as abrupt occlusion of the coronary artery. In this article, a 60-year-old case with unstable angina who had developed spontaneous occlusion of the left anterior descending artery immediately following coronary angiography.

Key words: Angiography; coronary artery; spontaneous occlusion.

Coronary angiography is the imaging method of choice for establishing the presence of coronary artery disease. However, it is an invasive procedure in which a non-physiologic solution, the contrast media, comes in direct contact with the endothelium, and a foreign body, the polyurethane and polyethylene material of the cardiac catheter, directly impacts the endothelium. During coronary angiography and vascular intervention, for example percutaneous transluminal coronary angioplasty (PTCA), many factors may cause platelet activation and degranulation. Exposure of highly thrombogenic subendothelial collagen and local thrombin generation occur upon rupture of the atherosclerotic plaque during PTCA, and these promote platelet recruitment and cause platelet degranulation. Radio-opaque angiographic contrast media are widely used in coronary and peripheral angiography, balloon angioplasty, and stent implantation. Different elements of blood and blood vessels are affected by the contrast media during coronary angiography. As these agents cause activation of thrombotic pathways in vitro, this may have a potentially significant clinical impact. In vitro studies have demonstrated that contrast media have noticeable effects on platelet function and thrombosis and that these effects differ between various contrast agents.

CASE REPORT

A 60-year-old male was referred to our department for coronary angiography due to unstable angina. His history revealed hypercholesteremia, smoking, and hypertension. On admission, the patient was asymptomatic. An electrocardiogram (ECG) showed...
sinus rhythm, pathologic Q waves in leads II-III-aVF, and T-wave inversion in leads II-III-aVF and V4-V6. Echocardiography revealed the presence of hypokinesia of the inferior and posterior walls. The patient was then referred to the coronary care unit. Blood tests, including troponin I which had been measured on admission and six hours afterwards, were normal. The coronary angiography which was performed on the same day of his admission using the non-ionic commercially available agent iopromide (Ultravist 370), showed total occlusion of the proximal right coronary artery (RCA), critical stenosis (75%) of the proximal left anterior descending artery (LAD) and thrombolysis in myocardial infarction (TIMI) grade 3 flow. Subcritical stenosis (50%) of the small first and second diagonal branches and subcritical stenosis of the distal obtuse marginal branch were also present.

Immediately after the coronary angiography, the patient complained of sudden-onset chest pain. His systolic arterial blood pressure instantly dropped to 60 mmHg, and the ECG showed sinus rhythm, a heart rate of 95 bpm, and a 3 mm ST-segment elevation in leads V1-V6. The coronary angiography was repeated and showed complete occlusion of the proximal LAD at the site of the previously documented stenosis. The patient was given intravenous (i.v.) unfractionated heparin, i.v. glyceryl trinitrate, and fluids, and his arterial pressure progressively rose to 90 mmHg. Percutaneous coronary intervention (PCI) with direct stent implantation of the LAD was immediately performed, resulting in TIMI grade 3 flow restoration. There was an immediate resolution of his chest pain and the elevation of ST-waves disappeared. Blood tests after the procedure were normal, including troponin I at six and 12 hours. The patient was discharged 72 hours later.

DISCUSSION

Contrast media is used to define coronary artery anatomy during angiography and to help in treating disease by percutaneous coronary interventions (stents, rotablation, atherectomy, etc.). Any procedural factor that would increase the susceptibility of the circulating platelets to these agonists and cause increased platelet degranulation would promote the thrombotic tendency and the local release of growth factors, such as platelet-derived growth factor (PDGF), within the damaged coronary artery lumen.

As previously stated, radio-opaque angiographic contrast media are widely used in coronary and peripheral angiography, balloon angioplasty, and stent implantation. Any effect of contrast media on platelet function may be important to the clinical outcome, but it may also confound studies of platelet function in the angioplasty. Different contrast media have been found to have different effects on in vitro platelet function. Previous studies have suggested that the use of different contrast media is associated with different rates of thrombus formation during angioplasty, suggesting that different effects of contrast agents on platelet function and thrombosis demonstrated in vitro may have important clinical implications.

During angiography, blood is transiently replaced by the contrast media, which also rapidly reaches the vascular smooth muscles and affects different elements of the blood and blood vessels. The extracellular matrix is rich in tissue factor, which is thought to play a key role in atherosclerotic plaque disruption with superimposed thrombosis. Many in vitro studies have investigated the chemical and mechanical effects of stimuli, which are similar to those involved in coronary angiography, on different elements of the microcirculation, such as the endothelium, platelets, leukocytes, smooth muscles, and inflammatory mediators. Although the activation caused by the contrast media appears to be independent of thrombin, high levels of local intracoronary thrombin generation in response to vessel wall injury and clot-bound thrombin could independently cause platelets to degranulate. Hutcheson et al. also found that radiographic contrast media exert direct pharmacological effects on the endothelium that depress production of nitric oxide in response to the mechanical stimulus of shear stress and the endothelium-dependent agonist acetylcholine.

Our case is especially important since it shows that a widely used contrast media which was previously considered to be safe, iopromide, can cause sudden platelet degranulation which would result in potentially fatal coronary artery occlusion. Therefore, invasive cardiologists should be aware of this rare but clinically very important complication.

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