Acute Stent Thrombosis: Rare but Still There

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Since the first intracoronary stent implantation, the design and ease of use of coronary stents have continued to improve, and the indications for their use have continued to expand from bailout procedures to now-routine implantation in patients with myocardial infarction.

Coronary stenting reduces the risk of restenosis and the number of revascularizations compared to balloon angioplasty, but, because of the insertion in the vessel of a foreign material, the procedure can be complicated by stent thrombosis. Stent thrombosis can occur any time after the procedure and is accordingly classified as acute (occurring in the first 24 hours), subacute (occurring in the first 30 days after the procedure), and late (occurring more than 30 days after the procedure), with stent thromboses occurring in the first month sometimes called ‘early’ and those occurring after more that one year sometimes called ‘very late.’

In the early days of stenting, the incidence of early stent thrombosis was very high, as patients were predominantly treated with anticoagulants. The use of dual antiplatelet therapy and high pressure stent balloon inflation have markedly decreased the occurrence of stent thrombosis. The incidence of acute stent thrombosis ranges from 0.5–1.4% in bare metal stent studies and is similar in more recent drug-eluting stent studies. Although the incidence is low, the consequences are oftentimes dire with a majority of patients presenting with large myocardial infarctions, but also with sudden death. It is important to identify patients at risk and to have a high index of suspicion when evaluating patients who have undergone percutaneous coronary revascularization presenting with acute coronary syndromes.

Despite its low incidence, acute stent thrombosis is a serious concern given the overall high number of patients undergoing percutaneous revascularization. The patients with acute stent thrombosis often present with myocardial infarction with high thrombus burden, as well as electrical and hemodynamic instability. Despite restoration of flow, these patients will often have refractory angina, need repeat percutaneous or surgical revascularization, and up to 20% will die in the hospital. Several patient factors can lead to an increased predisposition for stent thrombosis. These include presentation with acute myocardial infarction, older age, diabetes mellitus, renal insufficiency, and low left ventricular systolic function. Bifurcation lesions, residual vessel dissection, residual thrombus, and inadequate stent apposition are procedural factors associated with acute/early stent thrombosis.

In the report of acute stent thrombosis published in this issue, the patient presented with clinical symptoms consistent with myocardial ischemia as well as conduction system abnormalities and hemodynamic instability. One of the possible predisposing factors for stent thrombosis in this case includes stent placement in the setting of acute myocardial infarction. Acute stent thrombosis has been described to occur more frequently in these patients, possibly related to the high thrombotic state, with local thrombosis at the site of plaque rupture and the increased systemic hypercoagulability attributed to an increased inflammatory state. As pointed out by the authors, the question of adequate absorption of antiplatelet agents and adequate distribution of anticoagulants in this patient who presented with hypotension, congestive symptoms, right and possibly left ventricular systolic dysfunction may have contributed to the described outcome. Despite the above-mentioned aspects, antiplatelet resistance is also a possibility in this patient. This has been documented in patients presenting with early stent thrombosis and laboratory evidence of inadequate platelet inhibition.

The pathophysiology of acute stent thrombosis has not yet been completely elucidated, but it is clear that its consequences can be very serious even though it is a relatively rare event. Because of this, clinicians in intensive care units and on telemetry floors need to be alert to the possibility of acute stent thrombosis. As shown in the case report by Tu et al, clinical judgment is the clinician’s best tool: even though the patient did not have new ECG changes, he underwent coronary angiogram and percutaneous intervention. Despite the fact that acute stent thrombosis is a rare event, it can still happen.

References


Please see “Acute In-Stent Coronary Thrombosis Without ST Change on Electrocardiography: A Case Report and Literature Review” on page 239 of this issue.