Delayed arterial healing and increased late stent thrombosis at culprit sites after drug-eluting stent placement for acute myocardial infarction patients: an autopsy study.

**Abstract:**
BACKGROUND: The long-term safety of drug-eluting stents (DES) for acute myocardial infarction (AMI) remains uncertain. Using autopsy data, we evaluated the pathological responses of the stented segment in patients treated with DES for AMI and compared with patients with stable angina. METHODS AND RESULTS: From the CVPath Registry of 138 DES autopsies, we identified 25 patients who presented with AMI and had an underlying necrotic core with a ruptured fibrous cap. Twenty-six patients who had stable angina with thick-cap fibroatheroma treated by DES were selected as controls. Histomorphometric analysis was performed in patients with >30-day stent duration. We compared the response to stenting at the culprit site in these 2 groups and to nonculprit sites within each stent. Late stent thrombosis was significantly less frequent in stable (11%) than in AMI (41%; \( P=0.04 \)) patients. Although neointimal thickness in the AMI culprit site was significantly less (median, 0.04 mm; interquartile range [IQR], 0.02 to 0.09 mm), the prevalence of uncovered struts (49%; IQR, 16% to 96%), fibrin deposition (63% to 28%), and inflammation (35%; IQR, 27% to 49%) were significantly greater compared with the culprit site in stable patients (neointimal thickness: 0.11 mm [IQR, 0.07 to 0.21 mm], \( P=0.008 \);
uncovered struts: 9% [IQR, 0% to 39%], P=0.01; fibrin: 36+/-27%, P=0.008; inflammation, 17% [IQR, 7% to 25%], P=0.003) and the nonculprit site within each stent. CONCLUSIONS: Vessel healing at the culprit site in AMI patients treated with DES is substantially delayed compared with the culprit site in patients receiving DES for stable angina, emphasizing the importance of underlying plaque morphology in the arterial response to DES. Our data suggest an increased risk of thrombotic complications in patients treated with DES for AMI.