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Perspective

Outcomes and Risk Factors for Stent Thrombosis

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DESCRIPTION

The thrombotic blockage of a coronary stent is known as Stent Thrombosis (ST). In contrast to restenosis, which is a progressive narrowing of the stent lumen due to neointimal proliferation, this is frequently an abrupt phenomenon. Acute coronary syndrome is frequently caused by stent thrombosis, while angina symptoms are frequently caused by restenosis. Stent thrombosis has been linked to patient/lesion, procedural, and stent variables in large randomized studies and registries. Non ST-Elevation Myocardial Infarction (NSTEMI) and ST Elevation Myocardial Infarction (STEMI) at presentation, angiographic thrombus load, and total stent length were found to be independent predictors of acute stent thrombosis in the champion phoenix trial. An acuity investigation indicated that diabetes, renal insufficiency, Duke Hazard score, final stent minimal luminal diameter, preprocedural thienopyridine medication, baseline hemoglobin, and the extent of coronary artery disease were all independent variables connected with early stent thrombosis. In an Intravascular Ultrasound (IVUS) sub study of the Horizon AMI (Acute Myocardial Infarction) trial, a small cross sectional area of less than 5 mm, malposition of stent struts, plaque prolapse or protrusion, edge dissection, and residual stenosis all played significant roles in predicting early stent thrombosis. Bifurcation stenting also resulted in stent thrombosis According to the Triton TIMI 38 trial, patients with STEMI have a higher risk of stent thrombosis regardless of stent type, and more effective antiplatelet therapy, such as clopidogrel, reduced that risk by 50%.

Stent Thrombosis (ST) continues to occur despite improved stent insertion methods and more effective antiplatelet regimens, with an estimated incidence ranging from 1 percentage point to 5 percentage points (1–6). This considerable variation in the estimated incidence clearly suggests that ST is a complex phenomenon. Stent thrombosis has been documented since the early days of stent deployment, with rates as high as 16 percentage points in some studies. Patients with stent thrombosis die in 15 to 30 percentage points of cases within 30 days of the occurrence. In the early days of stenting, Acetylsalicylic Acid (ASA), dipyridamole, Coumadin, and dextran were used to prevent the occurrence. The current practice of dual antiplatelet medication with high-pressure inflation has reduced the incidence of stroke to 0.7 percent in one year and 0.3 percent to 0.8 percent the following year. Elective Percutaneous Intervention (PCI) has a lower rate (0.3 percent to 0.5 percent), although acute coronary syndrome has a higher rate (3.4 percent). The rate of stent thrombosis between bare metal stents and eluting drug stents has not been found to be significantly different. Only the event's timing varies. The bare metal stent has an early event with a peak of roughly 30 days, but drug eluting stents have an event that can last anywhere from three months to even longer depending on the drug-coated.

Endothelialisation, vascular inflammation, and activation of tissue factor activity may all be affected differently by stents coated with novel cytotoxic medicines and polymers. Platelet adhesion and aggregation may be reduced by coating with NO donors. Stents coated with anti CD34 antibodies may be able to capture circulating endothelial progenitor cells and prevent thrombosis by enhancing and speeding up endothelial coverage. Additionally, the development of biodegradable stents could help to reduce the incidence of late and very late stent thrombosis. The discovery of new, more effective anticoagulants and antiplatelet medicines with a lower risk of bleeding problems is likely to improve anti thrombotic therapy. Patients and healthcare professionals should be aware of the dangers of stopping therapy too soon.

The development of new tests that can detect platelet inhibition could help doctors identify patients who aren't getting enough benefit from aspirin or clopidogrel, allowing them to tailor and optimize their antiplatelet therapy. Stent thrombosis has long been a known consequence of Percutaneous Coronary Intervention (PCI), and the risk of stent thrombosis may be enhanced with the use of Drug Eluting Stent (DES). As a result, the subject of in stent thrombus development has resurfaced as a hot topic. While the evidence is still ambiguous, certain studies

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suggest that DES implantation increases the risk of late and extremely late stent thrombosis. Importantly, it is unknown if very late stent thrombosis is a time limited phenomena, raising the possibility that the disease would worsen if incidents continue to accumulate.

CONCLUSION

As a result, large scale clinical trials with long term follow up, as well as mechanistic research are essential. It is currently unknown whether an extended course of dual anti-platelet medication can prevent extremely late stent thrombosis. Certainly, the problem of stent thrombosis highlights the significance of careful patient selection and individualized therapy, which may in the future be based in part on platelet inhibition intensity monitoring.