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Perspective

The Outcomes of Initial PCI in Non-Acute Stent Thrombosis ST-Segment Elevation Myocardial Infarction (STEMI)

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DESCRIPTION

Due to the high mortality rate of acute stent thrombosis, there have long been worries over stent thrombosis following stent placement. The frequency of acute stent thrombosis has dramatically dropped as a result of advancements in stent coatings and materials as well as the advent of more effective antiplatelet medications. Even yet, patients may still experience non acute stent thrombosis (subacute, late, and late stent thrombosis) as a result of poor stent intima adhesion or new plaque rupture with longer recovery times. Patients with non-acute stent thrombosis may exhibit clinical manifestations such as unstable angina pectoris, acute myocardial infarction, etc. With an incidence of 59%-80%, or 3.2% of all STEMI patients, ST-Segment Elevation Myocardial Infarction (STEMI) is the most prevalent manifestation. Patients with non-acute stent thrombosis respond well to Percutaneous Coronary Intervention (PCI), yet the clinical outcome is still unclear. This study aimed to identify the risk factors for Major Adverse Cardiovascular Events (MACEs) during follow-up by analyzing and comparing the clinical and prognostic differences between patients with STEMI due to de novo lesions and patients with non-acute stent thrombosis. Based on when thrombosis occurs following stent implantation, there are four types of stent thrombosis: acute stent thrombosis (0–24 hours), non-acute (subacute) stent thrombosis (24–30 days), late stent thrombosis (30 days–1 year), and very late stent thrombosis (>1 year). Acute stent thrombosis was found to be frequently associated with the following risk factors: preoperative cardiac dysfunction; postoperative Thrombolysis In Myocardial Infarction (TIMI) blood flow < level 3; stent mal apposition; high platelet reactivity during treatment; vascular characteristics such as bifurcation, small vessels, and Type C lesions; and stopping dual antiplatelet medication. However, insufficient stent endothelialization, stent restenosis, poor late acquired stent adhesion during vascular remodeling, and rupture of new atherosclerotic plaques are frequently linked to late and very late stent thrombosis. Previous research indicates a significant fatality rate and a poor prognosis for acute stent thrombosis. According to our research, patients with non-acute stent thrombosis had worse clinical outcomes than those with de novo lesion, including an overall greater frequency of MACEs and a substantially higher probability of myocardial infarction recurrence. But there was no discernible difference between the two groups in terms of all-cause mortality. Age, sex, cardiac function, and non-acute stent thrombosis appeared to be independent predictors of combined MACEs, according to multivariate regression analysis. In addition, a greater percentage of patients with non-acute stent thrombosis had previously undergone cardiovascular care, including CABG treatment. Additionally, the non-acute stent thrombosis group at admission had a poorer left ventricular EF and a higher rate of heart failure, according to our study. Furthermore, we discovered that a low EF independently predicted 2-year mortality. The non-acute stent thrombosis group's increased event rate prior to matching could be attributed to the larger concentration of risk variables in this patient population. At the 1 and 2-year follow-ups, the non-acute stent thrombosis group had a greater rate of recurrent MI than the *de novo* lesion group. That being said, there was no statistically significant difference. According to earlier research, the majority of recurrent MI cases were associated with recurrent stent thrombosis, and the incidence of recurrent MI was greater in the stent thrombosis group at the 6-month follow-up as well as in the hospital. According to earlier research, stent thrombosis recurrence rates varied from 5.9% to 14.3%. This could be associated with things like patients' inadequate stent dilatation in cases of stent thrombosis, persistent thrombotic burden, and nonresponse to antiplatelet medications.

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