The incidence of stent thrombosis (ST) following elective, urgent or emergent percutaneous coronary stent implantation is quite low (1-4%) [1-5]. However, the risk of recurrent stent thrombosis (RST) is much higher (11-36%) [2,6-9] and not much is known about the pathobiology, predictors and clinical outcomes of RST. Thus, the optimal management of RST in terms of prevention and treatment approach is also lacking.

Incidence and time of occurrence of RST: Depending on patient population and duration of follow-up, the reported incidence of RST varies widely among studies [2,6-9]. Lemesle et al. [6] revealed a high rate of RST (36%) over a follow-up period of 40 months. According to van Werkum et al. [7], the cumulative rates of definite or probable RST during hospitalization, at 30 days, 1, 2 and 3 years were 11.6%, 14.4%, 18.2%, 19.6%, and 20.1%, respectively. Alamalla et al [8] demonstrated the cumulative rates of definite RST during hospitalization, at 1 year, and at long-term follow-up (65 ± 30 months) were 7.5%, 9.9% and 10.9%, respectively. Furthermore, these studies [7,8] suggested that more than half of the RST (50-69%) occurred at the time of hospitalization for the initial stent thrombosis. The median time for the RST occurrence was 4-5 days after the index ST [6,7]. Lemesle et al. [6] found that all RST occurred within the first 11 days and van Werkum et al. [7] reported 72% of RST occurred within 30 days following the ST index event. After the first year, the risk of RST became relatively low (0-9%) [6-8].

The prognosis of RST is largely unknown. Following the index ST, the cumulative risk of RST and death at 30 days, 1, 2, and 3 years are 18%, 23.6%, 25.2%, and 27.9%, respectively [7] and the cumulative event rates of RST and death during hospitalization, at 1 year, and at long-term follow-up (65 ± 30 months) are 22.6%, 31.6%, and 44.6% per Alamalla et al [8].

The clinical presentation of RST has not been well described. If it is similar to that of the index ST, one would expect the majority to present as acute coronary syndromes (mostly STEMI), and the rest as cardiogenic shock or malignant arrhythmia [2,6,8-10].

The independent predictors for the combined endpoint of RST and death are diabetes mellitus, left ventricular ejection fraction <45%, complex coronary lesion, long total stent length, TIMI flow grade <3 after intervention, and implantation of additional stent(s) in the treatment of the ST [2,7]. Cardiogenic shock, TIMI grade flow after intervention and renal failure are predictors for RST and death according to Alamalla et al. [8]. History of neoplasia, residual stent diameter stenosis, residual dissection, use of Abciximab, and current cigarette smoking are found to be independent predictors for RST [2,6,8].

The risk of RST is similar in patients regardless of the timing of its occurrence (early vs late) or the type of stents implanted (DES vs BMS) [7]. There is no existing information regarding the duration and choice of dual antiplatelet therapy, nor the incidence of high on-treatment platelet reactivity in patients with RST.

The mechanism of RST is likely multifactorial and complete prevention of RST is improbable, but the occurrence of RST can be minimized using optimal pharmacomechanical treatment of the index ST and exhaustive follow-up care [11,12]. Since more than 70% of RST occur within the first 30 days of the index ST event, it is likely that the pathobiological mechanism is largely due to procedure-, stent-, or lesion-related factors and less likely resulting from pharmacologic-related or patient-related issues.

An evidence-based treatment strategy for RST will continue to evolve, guided by future clinical studies on ST and RST. Meanwhile, it is unclear why implanting a new stent is associated with a 5-fold increased risk for RST [2] and an increased mortality [13]. Is the implantation of a new stent a result of a dissection, untreated adjacent disease, significant residual stent luminal stenosis, TIMI flow <3 or some other unexplained reason? The nationwide CathPCI registry data [10] on “real-world” ST treatment in the modern era indicates that more than half of these patients received an additional stent irrespective of timing of ST (early, late or very late), and aspiration thrombectomy (an independent predictor of lesser MACE post-ST [8] and a 43% lower 1-year mortality though statistically non-significant [14]) is performed in only one-third of ST patients. Thus, prospective, randomized studies evaluating the benefits of thrombectomy, stenting (DES or BMS) or balloon angioplasty (POBA or drug-eluting) will provide invaluable information in the treatment of ST.

Since RST is a rare and challenging problem after coronary stent implantation, the fact remains that the scientific community
will continue to rely on retrospective observational studies derived from large national or international registries and meta-analyses as the most expedient means in providing the much needed knowledge to confront this rare but serious complication.

REFERENCES


