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EDITORIAL COMMENT

Long Stents as a Risk Factor for Late Stent Thrombosis

Size Does Matter*

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The issue of stent thrombosis (ST) and particularly late stent thrombosis (LST) (>30 days after deployment) (1) has recently haunted the use of stents.

Although it occurs in bare-metal stents, LST is slightly more frequent in drug-eluting stents (DES), with an approximate 0.5%/year increased rate over bare-metal stents with a persistent risk for several years (2). The manifestation of LST is myocardial infarction (MI) or death with a case fatality rate as high as 45% (3).

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There are numerous risk factors for LST but currently few answers. Among the many variables studied, patient characteristics for LST include the presence of diabetes (3), renal failure (3), and thiopyridine resistance or discontinuation (4). Lesion factors include small vessels (4), long stent lengths (5), increased total stent length (5), and bifurcation disease (6). Procedural factors include incomplete stent apposition or underexpansion of stents (7) and the use of "bifurcation stenting" techniques (8). Other proposed factors include delayed stent endothelization (9) and hypersensitivity to a DES component, such as the polymer (10).

The paper in this issue of *JACC: Cardiovascular Interventions* by Suh et al. (11) documents that, when stents \geq 31.5 mm in length are used, there is increased risk of LST, MI, and death. They studied 3,145 patients who had DES placed and followed them for a mean 29.6 months. It was found that, among the studied variables, stent lengths \geq 31.5 mm were strongly associated with rates of ST = 4.0%, death = 5.2%, and MI = 2.4%, as compared with stents <31.5 mm, which had rates of ST = 0.7%, death = 3.0%, and MI = 0.7%. Indeed, of the 68 patients who developed ST, 60 (88%) had stent lengths \geq 31.5 mm. Patients who had LST had received nearly 1 year of thiopyridine therapy. They noted that the event rates were lower in DES patients compared with patients who received similarly long bare-metal stents.

The risk of stent length has been previously documented by Moreno et al. (5), who noted in a meta-analysis of 10 studies that stent length was positively correlated with ST but—unlike Suh et al.—did not arrive at a threshold.

Unfortunately, there are no data on strategies to reduce the risk of LST, and none of the current studies propose such strategies. Many of the risk factors for LST are not modifiable (diabetes, renal insufficiency, small vessels); hence, we need to focus on strategies for the modifiable risk factors that might reduce LST. Certainly, there is a profile of high-risk patients that we are aware of, which now includes use of long stents. There are some strategies, although unproven but intuitively appropriate, that can be applied in practice that might contribute to reduction of LST, including:

- 1. If possible, avoid using super long stents (\geq 31.5mm);
- 2. Consider, when possible, to "spot stent" long lesions; the use of intravascular ultrasound might be helpful to locate minimally diseased areas;
- 3. The liberal use of intravascular ultrasound after long stent deployment might identify areas of incomplete stent apposition requiring further expansion;
- 4. In any high-risk patient (including long stents), consideration of prolonged thiopyridine treatment of at least 2 years and in many cases indefinite use of thiopyridine.

The solution to LST has not been obvious, but attention to modifiable risk factors such as super long stents as demonstrated by Suh et al. (11) might serve to identify those at risk and attempt to reduce the incidence of LST.

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^{*}Editorials published in *JACC: Cardiovascular Interventions* reflect the views of the authors and do not necessarily represent the views of *JACC: Cardiovascular Interventions* or the American College of Cardiology.

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