Complications of the hemodialysis synthetic arteriovenous graft (AVG) cause significant morbidity and mortality, and increase the cost of access-related care. Stenosis at the venous anastomosis or outflow vein leads to stasis, thrombosis, and ultimately, AVG abandonment. Consequently, survival of AVGs is less than 50% at three years [1]. The K/DOQI panel reasoned that delaying correction of stenosis until after thrombosis results in tight stenoses that are difficult to correct by percutaneous transluminal angioplasty (PTA). It was hoped that AVG survival would be improved by applying prospective surveillance followed by early preemptive PTA of hemodynamically significant stenosis. It is clear that surveillance can detect subcritical stenoses, but three recent randomized controlled trials have challenged the belief that surveillance improves AVG survival [2–4].

These disappointing results indicate that preemptive PTA may not have the beneficial effect that was originally hypothesized. This has led to renewed interest in application of endovascular stents to grafts that are resistant to PTA. Although previous studies have often been disappointing [5], the study by Sreenarasimhaiah et al [6] in this issue of Kidney International underscores the possibility that stents may prolong AVG survival.

The many types of available stents and the nonuniform study designs and documentation make it difficult to compare previous studies. Stents are currently available in stainless steel (Wallstent, Gianturco), nitinol (nickel and titanium alloy (e.g., SMART, Symphony, and Memotherm)), and as coated stents (ninitol coated with Dacron (e.g., Cragg), and cobalt/titanium coated with PTFE (e.g., Wallgraft)). Previous studies have employed Wallstent, Gianturco, and Cragg stents with inconsistent success in prolonging AVG survival. However, the nitinol self-expanding SMART stent may be better suited for use in AVGs because of important physical characteristics, such as superior flexibility and ability to be placed at areas of angulation [7].

Vogel et al [7] recently retrospectively studied the use of SMART stents to treat PTA-resistant stenosis in AVGs. Criteria for inclusion in the study included recurrent stenosis less than three months after PTA, venous rupture, and greater than 50% stenosis after PTA. Of 19 patients who had stents placed at sites of previous PTA, primary (unassisted) patency at six months increased from 14% for PTA without stenting to 72% after PTA with stenting; mean primary patency increased from 2.5 to 10.6 months.

Sreenarasimhaiah et al [6] obtained similar encouraging results in this issue of Kidney International. They studied the clinical course of 34 patients after placement of SMART stents as treatment of venous anastomotic lesions in thrombosed AVGs unresponsive to PTA. Criteria for inclusion in the study included PTA of the same lesion within the previous three months, lumen collapse immediately post-PTA because of elastic recoil, or significant residual stenosis post-PTA. Treatment with clopidogrel post-procedure was an additional feature of the study. The AVGs averaged 17.9 months of age, and 84% had one or more endovascular or surgical procedures before this thrombosis. The results of this study are remarkable: primary patency after stent placement was 63% and 36% at six months and one year, respectively, and secondary (assisted) patency was 88% and 86% at six months and one year, respectively.

In attempting to explain this study’s encouraging results, a potential role of clopidogrel should be considered. In AVGs, previous studies with bare-metal stents have not employed currently available antiplatelet agents. It should be noted that antiplatelet therapy has become the standard of care after intracoronary stent implantation. Regarding AVGs, Sreedhara et al demonstrated a significant reduction in the number of thromboses in newly created AVGs that were treated with dipyridamole compared with placebo, but there was no benefit in previously thrombosed AVGs [8]. However, more recent experience with antiplatelet agents has not been encouraging. A randomized controlled trial evaluated the role of clopidogrel plus aspirin in preventing AVG thrombosis [9]. The study was discontinued early because of a marked increase in bleeding episodes in the treatment group. The study showed no difference in thrombosis rates between the treatment and placebo groups. Thus, it is unclear whether clopidogrel played a significant role in the favorable outcome of Sreenarasimhaiah et al’s study [6]. K/DOQI does not currently recommend use of antiplatelet agents or anticoagulation to prevent AVG thrombosis.

The study by Sreenarasimhaiah et al indicates that despite improved patency, neointimal hyperplasia continues unabated [6]. For example, in the 21 of 34 patients

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that required repeat procedures, 81% (17/21) had lesions outside the stent. Moreover, in 43% of repeat procedures, the original stented lesion was not implicated as the cause of AVG dysfunction. Thus, stenting may delay abandonment of the AVG, but does not provide the more desirable (but not yet attainable) definitive prevention or treatment of neointimal hyperplasia. Nevertheless, this result, if confirmed, is noteworthy because any treatment that prolongs access function delays the day when the patient runs out of access sites.

In conclusion, we must find better ways to prevent and treat neointimal hyperplasia. Novel strategies to prevent AV graft stenosis by using pharmacologic and other therapies are underway (NIH DAC Study and others). Nevertheless, until truly effective therapies are found, our options are limited. In light of this new data with nitinol stents, we must push for randomized controlled trials that will test their efficacy as an adjunct to PTA. If future studies demonstrate that stent placement prolongs access survival and reduces intervention, the added costs of stents may be offset by decreased access morbidity and reduced total access-related costs. Drug-eluting stents are very expensive but may be even more effective. PTA combined with stent deployment may be the combination that opens a new era of improved therapy of recurrent stenosis.

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