scarring and interstitial fibrosis in the subendocardial regions of the anterior and apical left ventricle, without significant cellular infiltrate. The pathologic findings were thought to be consistent with chronic ischemia in the distribution of the terminal circulation (left anterior descending artery territory). The authors of this earlier paper concluded that this portion of the heart had a combined ischemic blood supply coupled with an excessive demand on the limited circulation. Perhaps a similar pathophysiology contributed to the chest pain symptoms reported in their 59-year-old female patient.

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REFERENCES

Vertebral Artery Stenting
Not Quite Ready for Prime Time!

Atherosclerotic vertebral artery (VA) stenosis is a significant cause of vertebrobasilar ischemia. However, vertebral artery stenting (VAS) has not received the detailed scientific study that has been accorded to carotid artery stenting (CAS).

In their series, Jenkins et al. (1) show excellent results. These results add to the growing body of nonrandomized studies that demonstrate the feasibility and relative safety of VAS (2). Based on their outcomes, the authors recommend a more liberal use of VAS. However, several issues remain unresolved that beg for a more cautious approach.

No recent study of sufficient size has investigated the impact of optimal medical regimen on the natural history of VA disease or compared it with VAS (3). Further, there are several unresolved issues regarding optimal endovascular strategy. Bilateral VA stenosis presents a clinical challenge. Unlike anterior circulation ischemia, vertebrobasilar ischemia symptoms are difficult to lateralize to one side. It is not known whether unilateral VAS will resolve the symptoms or whether bilateral VAS is indicated. The authors report that 54.3% patients had bilateral VA disease, although only 6.3% of the subjects received bilateral stents. It is unclear how the stented side was chosen and whether symptoms resolved completely.

Subclavian artery stenosis without coexistent VA stenosis can cause vertebrobasilar ischemia (4). In the current study, 29.2% of the subjects had concurrent subclavian artery disease. It will be useful to know whether the subclavian artery was also stented concomitantly.

The present study did not use distal embolic protection, although this is the standard in CAS. This is an important issue in VAS with only limited data available (5). Another important issue is restenosis. Unlike CAS, which has a low risk of restenosis, VAS has a significantly higher restenosis rate (6,7). Little information is available regarding the use of drug-eluting stents, although initial reports indicate a lower restenosis rate (8).

Jenkins et al. (1) demonstrate that VAS is relatively safe and feasible. However, before more widespread use, VAS should undergo the same meticulous investigation as CAS has been accorded. This will involve a direct comparison with optimal medical therapy and use of current endovascular standards (distal embolic protection and perhaps drug-eluting stents).