Aortic Debris and Coronary Guiding Catheters

I read with interest the recent article by Keeley and Grines regarding scraping of aortic debris by coronary guiding catheters. This has been a problem for all interventional cardiologists, and it is rewarding to see this issue addressed in a quantitative fashion in this publication. We have had experiences similar to those of the authors in identifying atheromatous debris, particularly with the use of left coronary guiding catheters.

There are several issues not addressed in the article that I believe deserve mention. First, it has been our experience that the larger bore guide catheters cause release of more atheromatous debris. There seems to be a noticeable increase when switching from 8 fr to 9 fr guides.

Second, a method of minimizing or avoiding the scraping of aortic debris is the insertion of an obturator inside the guiding catheter. The one that we commonly use is a DVI introducing catheter, which is 110 cm long. For a 9-fr guide, we use a 7-fr obturator. An alternative would be the use of a 110 or 125 cm diagnostic multipurpose catheter to serve as an obturator. With the use of the obturator, there is a smooth transition from the wire to the guiding catheter and thus, the amount of aortic debris is significantly reduced.

In our experience, the most voluminous release of debris is during renal artery stenting procedures. Many of these patients have diffuse aortic atherosclerosis and the shape of the renal artery guide catheters is extremely conducive to scraping of aortic debris. Again, we have found that the use of obturators significantly minimizes this effect.

REFERENCE

Murakami are interesting and warrant the question of the role of the microvasculature in other syndromes associated with ST elevation, such as acute myocardial infarction.

In a study of the coronary slow flow phenomenon (angiographically normal coronary arteries with Thrombolysis in Myocardial Infarction trial [TIMI-2] flow) (2) we have observed similar findings to those of Murakami et al. During coronary hemodynamic studies to determine the pathophysiology of this disorder, we infused intracoronary acetycholine into eight patients in the absence of vasodilator therapy. In one patient, the infusion provoked chest pain with ST elevation in the absence of epicardial arterial spasm. We would therefore be interested to know if any of the three cases observed by Murakami et al. had evidence of the coronary slow flow phenomenon. The coronary Doppler flow velocity measurements would suggest that at least one patient may.

Surprisingly, our patient with ST elevation showed no prolongation in the TIMI-frame count; an observation consistent with Murakami’s findings from the coronary Doppler flow velocities. Furthermore, there was no evidence of net lactate production. These observations may reflect the limitations of the techniques utilized, or may alternatively demonstrate that ST segment changes may be independent of myocardial ischemia (3). Further studies of uncommon patients such as these may provide further insight into the mechanism of ST elevation.

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REFERENCES

REPLY

The phenomenon of ST-segment elevation with normal coronary angiography may be rarely observed in many institutions in the world where coronary angiography is routinely performed. To explain the mechanism for this strange phenomenon, microvascular constriction has been suspected, but no clear evidence has yet been provided. Our study (where we used a Doppler guide wire) provided new evidence, and we proposed the novel hypothesis of inappropriate microvascular constriction to explain this phenomenon (1). It is not clear whether microvascular spasm as described by some authors (2–5) certainly took place during this phenomenon. We still doubt that microvascular “spasm” is a pathogenetic cause of this phenomenon because there is no concrete evidence demonstrating its occurrence. It is still possible that microvascular constriction during the phenomenon is one of the physiologic responses in the local area, however, the pathologic manifestation can be seen in the whole heart.

To understand this phenomenon, the most important point would be whether it indicated cardiac ischemia, as Drs. Beltrame and Horowitz questioned. In our three patients, only two episodes in one patient (case 1) showed biphasic T wave after restoration of ST-segment. Both of these episodes persisted for over 15 min, but the other patients had the phenomenon within 5 min. The T wave inversion after ST-segment elevation was sometimes observed during PTCA. However, no obvious T wave inversion was observed when coronary occlusion time by angioplasty was substantially short. We currently have 10 episodes of ST-segment elevation in 9 patients (unpublished data). In these patients, only three episodes of biphasic T wave were observed in 2 patients, but no T wave inversion was observed. However, all of the 9 patients except one (patient 3 in our study) had severe chest pain during ST-segment elevation, and this chest pain was relieved after ST-segment had been restored, which may reflect cardiac ischemia.

Eight of 10 episodes had increased TIMI frame count, but two had a similar count compared with control angiography. The TIMI frame count during ST-segment elevation did not always show similar changes. The phenomenon of our patients was very similar to the study by Beltrame et al. (6). Although all of our three reported patients underwent intracoronary acetylcholine infusion test to provoke epicardial coronary spasm, one episode occurred in patient 1 before any drug infusion. The other three episodes took place after intracoronary acetylcholine infusion, not after nitroglycerin infusion, but the phenomenon was not observed immediately after acetylcholine infusion. There was significant time delay between ST-segment elevation and intracoronary acetylcholine infusion. Currently, half of 10 episodes were observed before either acetylcholine infusion or any other drug challenge.

Surprisingly, in our 10 episodes, two exhibited the phenomenon before initial angiography. The phenomenon was observed just after coronary catheter insertion. This fact suggested that iatrogenic microemboli through a catheter as a cause of the phenomenon may be discounted.

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REFERENCES