Time Relationships Between Plaque Rupture and Infarction

To the Editor:

In the October 24, 2000, issue of Circulation, Ojio et al reported 20 cases of acute myocardial infarction in which the patients had had angiograms shortly before acute myocardial infarction. They found evidence of plaque rupture/thrombi in 60% of cases at least 3 days before infarction. Because one half of the patients developed stable angina within a month before infarction, considerable time might elapse between plaque rupture and infarction. On the basis of these findings, Ojio and colleagues postulated that infarction occurs in 2 steps: initial plaque rupture and/or thrombi with mild to moderate stenoses and later progression to occlusion and infarction.

I suggest that this study casts doubt on the accepted view that plaque rupture/thrombi are the direct cause of infarction and other acute coronary syndromes (ACS), and I also suggest that the delay between plaque rupture and infarction can be explained by the spasm-of-resistance-vessels (S-RV) concept. This theory asserts that S-RV directly initiates the ACS and that thrombi develop secondarily. The S-RV concept avers that plaque rupture, which frequently is asymptomatic, is involved in infarction through 3 factors: stenoses, stasis, and platelet emboli. First, 95% of Ojio’s cases had significant stenoses shortly before infarction because of plaque rupture and/or nonocclusive thrombi. It is well known that severe ischemia causes S-RV, and the S-RV concept asserts that stenoses favor S-RV and infarction through severe ischemia. Second, once S-RV occurs, S-RV would cause upstream stasis, which would foster occlusive thrombi; thrombi would tend to occur in intimal irregularities of ruptured plaques. Third, platelet emboli secondary to intimal irregularities would favor S-RV through release of vasoconstrictive substances.

A recent review analyzed the role of plaque rupture/thrombi in ischemic heart disease, demonstrating that multiple findings do not support primary plaque rupture/thrombi. Infarction can occur in the absence of coronary artery disease and thrombi, and the incidence of thrombi rises over time with infarction—a finding inconsistent with primary thrombi. Also, most types of ACS have incidences of thrombi of ~25%, and reliable studies found relatively low incidences of thrombi in transmural infarction.

Furthermore, triggers of ACS generally are considered to operate through acute plaque rupture/thrombi, but this mechanism cannot explain the triggering of silent myocardial ischemia and variant and effort angina; if these syndromes are not triggered by plaque rupture/thrombi, perhaps acute plaque rupture/thrombi do not directly trigger the ACS. Additionally, ACS occur in the absence of irregular intimal lesions, and irregular intimal lesions occur in the absence of ACS.

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