The Effect of Antiplatelet Therapy on the Incidence of Carotid Plaque Hemorrhage

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SUMMARY Hemorrhage into the carotid atheroma has recently been gaining attention with respect to the pathophysiology of cerebrovascular disease. Many patients are currently receiving platelet agents for various vascular diseases. Some researchers have postulated that antiplatelet therapy may be detrimental by possibly inducing intraplaque hemorrhage or by increasing preexisting hemorrhage. This retrospective study was undertaken to determine if the use of antiplatelet therapy increases the incidence of carotid plaque hemorrhage.

Ninety-five consecutive carotid endarterectomies were performed and the atheromas examined microscopically for intraplaque hemorrhage. The atheromas were divided into two groups; those from patients receiving preoperative antiplatelet therapy and those who were not.

Forty-five atheromas were removed from patients receiving preoperative antiplatelet therapy; 39 (87%) of these demonstrated intraplaque hemorrhage. Of the 50 atheromas which were removed from patients not receiving preoperative therapy, 45 (90%) showed intraplaque hemorrhage. We conclude that antiplatelet therapy does not increase the incidence of carotid plaque hemorrhage.

STROKE is a leading cause of morbidity and mortality in the United States. The role of extracranial carotid artery disease in the production of cerebral ischemia has been well accepted. More specifically, hemorrhage into the carotid atheroma has recently been gaining attention with respect to the pathophysiology of cerebral vascular disease.1,2 Also many patients are currently receiving antiplatelet therapy since it has been shown to reduce cerebrovascular ischemic symptoms.3,4 Nonetheless, some investigators have questioned the rationale of administering antiplatelet drugs to patients with hemorrhagic lesions.1,2 With the above information in mind, this retrospective study was undertaken to determine if antiplatelet therapy increases the incidence of carotid plaque hemorrhage.

Materials and Methods

From May to September 1983, 85 consecutive patients who underwent 95 carotid endarterectomies were studied at two large community hospitals in Wichita, Kansas.2 The average age was 68 years. Ten patients had bilateral operations. Of the plaques removed, 44 were from patients with ipsilateral hemi-
removed from patients not receiving antiplatelet agents preoperatively. Of these 50, 45 (90%) demonstrated intraplaque hemorrhage. Eleven (12%) atheromas showed no intraplaque hemorrhage; six (55%) of these were from patients receiving preoperative antiplatelet therapy.

Discussion

Hemorrhage has multiple effects on a carotid plaque. It can increase the plaque size and, consequently, decrease blood flow leading to surface platelet aggregation with platelet embolization or thrombosis. In addition, hemorrhage into a plaque can lead to intimal ulceration and subsequent embolization of hemorrhagic debris. Moreover, Persson and associates have suggested that hemorrhage is significant only when a connection occurs between the arterial lumen and the plaque hemorrhage. These events have been felt to result in the production of cerebrovascular ischemic symptoms. A recent study by this senior author (A.D.A.) demonstrated hemorrhage to be a common finding in most carotid plaques; however, intraplaque hemorrhage was as likely seen in asymptomatic patients as compared with symptomatic patients. On the other hand, research by Lusby, et al and Imparato and colleagues did demonstrate a significant relationship between intraplaque hemorrhage and symptoms.

In 1978, the Canadian Cooperative Study concluded that aspirin was an efficacious drug for men with threatened stroke. In men the risk reduction for stroke or death was 48 percent whereas no significant trend was observed in women. Bousser et al reported similar beneficial effects of aspirin in both men and women. Presently, many symptomatic patients are treated with antiplatelet therapy. Antiplatelet agents are thought to inhibit platelet aggregation on carotid plaque surfaces, thereby reducing embolization and subsequent ischemic symptoms. Some researchers, however, have postulated that antiplatelet agents have the potential of increasing symptoms in some patients by inducing intraplaque hemorrhage or by increasing preexisting hemorrhage. This would explain why antiplatelet therapy could be detrimental.

Results of the present investigation show that hemorrhage was seen as frequently in carotid plaques not exposed to antiplatelet agents as in plaques that were exposed. More importantly, as many nonhemorrhagic plaques were identified in patients on antiplatelet therapy as in those not receiving antiplatelet therapy. Thus, the concept that antiplatelet medications increase the incidence of intraplaque hemorrhage is not supported in this study.

References

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