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PLATELET ACCUMULATION IN CAROTID ATHEROSCLEROTIC LESIONS: SEMIQUANTITATIVE ANALYSIS WITH INDIUM-111 PLATELETS AND TECHNETIUM-99M HUMAN SERUM ALBUMIN.

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Atheroma at the carotid bifurcation is one of the common causes of stroke from obstruction of the internal carotid artery. Moreover, transient ischemic attacks of cerebral ischemia (TIA)may result from microemboli released from thrombi deposited on atheromatous lesions. Platelet activation on the vascular wall is therefore an important event in the initiation arterial thrombus. If the vascular endothelium is injured, adherence and aggregation of platelets occur on the exposed connective tissue. Atherosclerosis is thus promoted by interaction between platelets and endothelium. However, the in vivo platelet accumulation and/or activation in atherosclerotic lesions has not been fully demonstrated. In the field of nuclear medicine, indium-111 platelets have been used to identify carotid atherosclerosis. In these studies, however, quantitative analysis was not performed. In the present study, we evaluated the accumulation of platelets on carotid atherosclerotic lesions semiquantitatively by means of a dual-tracer technique, using In-111 platelets and Tc-99m human serum albumin (HSA). With this approach, the background radioactivity of In-111 platelets in the blood pool was, in effect subtracted using a Tc-99m HSA blood-pool image. Furthermore, the ratio of radioactivity in In-111 platelets deposited on the vascular wall to those circulating in the bllod pool at the carotid bifurcation, the platelet accumulation index (PAI) was determined for semiquantitative analysis. The purpose of present study was (1) to compare the results of semiquantitative analysis with those of visual analysis, and (2) to compare the degree of platelet accumulation on carotid bifurcation (PAI) with angiographic findings. Twelve normal subjects and 25 patients with ischemic cerebrovascular disease (CVD) were examined. The mean age was 58.5 yr (range 39-81) in normal subjects and 61.5 yr (47-73) in CVD patients. Angiographic abnormalities were observed at 34 of 50 carotid bifurcations in the CVD patients. The mean PAI value was significantly higher at the carotid bifurcations with angiographic abnormality than at the normal ones ($P < 0.001$). Furthermore, elevations of mean PAI were prominent at the lesions with severe stenosis or ulcertions. The mean PAI value was significantly reduced with aspirin therapy (660 mg daily). The degree of platelet accumulation was well demonstrated by this technique, which can also yield information on thrombogenicity and efficiency of antiplatelet therapy in carotid atherosclerotic disease.