


Granulocytes, Platelet Activating Factor, and Stroke

To the Editor:

Saito et al.1 report increased leukotriene-like immunoreactivity (C4 and D4) in gerbil brain following bilateral common carotid occlusion and reduced leukotriene levels as a result of busulfan-induced granulocytopenia. Their results support the view that granulocytes may be involved in the pathogenesis of cerebral infarction.

In addition to being the source of leukotrienes, granulocytes, particularly basophils, produce platelet-activating factor (PAF, 1-o-alkyl-2-o-acetyl-sn-glycero-3-phosphorylcholine),4 which is a potent inducer of platelet activation, thrombosis, and ischemia.5,6 Apart from the platelet-mediated effects, PAF has also been shown to cause direct neuronal damage,5 cerebral vasoconstriction,6 and cerebral hypoperfusion,7 possibly mediated by specific PAF binding sites in the brain.8,9 In addition, we have reported an increased sensitivity to PAF-induced platelet activation in patients with acute cerebral infarction.10 We therefore suggest that the generation of platelet-activating factor may be another mechanism by which granulocytes participate in the pathogenesis of cerebral infarction.

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References


Pulsed Doppler Assessment of Arterial Obstructive Disease

To the Editor:

The article by Drs. Rautenberg and Hennerici in Stroke raised these questions for the authors in my mind:

1. How were the asymptomatic patients selected?

2. What were the features of the symptomatic transient ischemic attacks (TIAs)?

3. Were blood pressures obtained in both arms, and were there significant inequalities?

4. In those patients who received surgery to bypass the innominate artery obstructive diseases?

5. Finally, is it correct that some of the asymptomatic patients underwent surgery?

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Reference


The following is in reply:

To the Editor:

We appreciate the letter by Dr. Perron. His five questions are answered as follows:

1. The asymptomatic patients were investigated because of carotid bruits, risk factors for atherosclerosis, or coexisting peripheral arterial disease or coronary artery disease (cf. reference 1).

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2. Two patients had amaurosis fugax attacks of the right eye, and five reported brainstem TIAs.

3. Blood pressure was measured bilaterally, with significant differences found in eight patients. In six patients with bilateral subclavian steal, no differences were seen.

4. After surgery, the TIAs stopped.

5. Among the 10 operated patients, seven were symptomatic. Three asymptomatic patients underwent surgery prior to aortic-coronary bypass surgery during the same thoracotomy.

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Reference

Hemorrhagic Transformation of Cardioembolic Stroke

To the Editor:

Two recent reports in Stroke provide interesting information and prompt further speculation about hemorrhagic transformation of cardioembolic stroke. 1,2 Secondary hemorrhagic transformation of presumed cardioembolic stroke is usually not associated with recognized clinical worsening. 3,4 Definition of the temporal window of hemorrhagic transformation has been based on retrospective case series in which computed tomography (CT) data were collected at nonstandard time intervals, perhaps in patients with late hemorrhagic transformation that was undetected and therefore not included. 5-7 The single prospective study using serial CTs up to 3 weeks after stroke reported an extraordinarily high prevalence of hemorrhagic infarction (43% of all supratentorial infarcts, 61% of presumed cardioembolic infarcts). 8 While initial case collections suggested that the great majority of spontaneous hemorrhagic transformation occurred within 2-4 days of cardioembolic stroke (Figure 1), 1,2,5 multiple case reports have since documented later occurrence. 9-9 In short, the exact limits of the window of spontaneous secondary hemorrhagic transformation remain ill-defined. There is clearly a delay between stroke onset and the development of hemorrhagic transformation detected by CT. In the CT/autopsy series of Loder et al., only 10% (221) of the infarcts had definitely transformed before 24 hours, and 43-90% transformed after 24 hours. Among 28 cases of hemorrhagic transformation collected by the Cerebral Embolism Study Group, CTs done before 6 hours (n = 7) showed no cases of hemorrhagic transformation while those done between 6 and 18 hours (n = 11) showed hemorrhagic transformation in 45%. 5

The potential safety of acute fibrinolytic therapy in cardioembolic stroke may be influenced by this initial delay in hemorrhagic transformation. When Mori et al. infused intracarotid urokinase into patients with acute middle cerebral artery occlusion, hemorrhagic transformation was linked to presumed cardioembolic sources and the severity of initial deficit rather than to observed recanalization. While Mori et al hypothesized that the volume of the embolus explained the differential recanalization rate of cardioembolic (29%) versus noncardioembolic (75%) middle cerebral artery occlusions (p = 0.048, Fisher's exact test), the age of the embolic fragment may also affect recanalization. 2,10 Left atrial thrombi are often many weeks old, firm, and well-organized and may be less susceptible to lysis than recently formed intracarotid thrombi. These recent reports suggest that

FIGURE 1. Timing of hemorrhagic transformation (HT) defined by computed tomography (n = 34) or autopsy (n = 14) from two combined case series. 1,5 The area indicated as (?HT) reflects the interval between nonhemorrhagic CT [(-)HT] and diagnosis of hemorrhagic infarct [(+)HT] within which the exact time of HT is uncertain. For example, at 24 hours after stroke onset, at least 25% and possibly as many as 77% of patients had undergone HT.

while very early infusion of fibrinolytic agents (<6 hours) in cardioembolic stroke may safely precede the period of hemorrhagic transformation, recanalization may be less frequently achieved in thrombus of left atrial origin.

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References
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