Letters to the Editor

Infection and Brain-Induced Immunodepression After Acute Ischemic Stroke

To the Editor:

We read with great interest the recent review article by Chamorro et al.1 Understanding whether an anti-inflammatory response after acute ischemic stroke facilitates poststroke infection is clearly an important question. Poststroke infection is in itself an important independent contributor to poor outcome, and the potential introduction of new treatments modifying the inflammatory response—for example, anticytokine therapies2—requires that we take into account anything that might influence immunity.

We recently published clinical data3 providing evidence of systemic inhibition of the production of cytokines associated with the innate immune and inflammatory responses. After lipopolysaccharide stimulation of whole blood sampled between admission and 5 to 7 days poststroke, we found significantly reduced interleukin (IL)-1β, tumor necrosis factor-α and IL-6 production relative to controls. Minimum in vitro cytokine production in the first week correlated significantly with poorer clinical outcome and was lower in 12-month nonsurvivors than survivors. Although the mechanism involved was not defined, we previously reported plasma cortisol concentration to be markedly elevated within 12 hours of ischemic stroke4 and observed a strong inverse correlation between cortisol concentration and minimum in vitro cytokine production.

Our clinical observations are consistent with experimental data describing a systemic anti-inflammatory response, characterized by reduced endotoxin stimulated whole blood tumor necrosis factor-α and increased IL-10 production, after infusion of IL-1β into the brain, via stimulation of the hypothalamo-pituitary-adrenal axis and the sympathetic nervous system.5 Both catecholamines and corticosteroids suppress endotoxin-induced cytokine production by whole blood from healthy volunteers.6,7 Such regulatory responses, and increases in inhibitory cytokines such as IL-1ra, may be responsible for increased vulnerability of stroke patients to infections.

Disclosures

None.

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Stroke. 2008;39:e7; originally published online November 29, 2007;
doi: 10.1161/STROKEAHA.107.500447
Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the
World Wide Web at:
http://stroke.ahajournals.org/content/39/1/e7

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