Beneficial Effect of Albumin Therapy Attributable to α1-Acid Glycoprotein?

To the Editor:

Belayev et al1 report that albumin therapy has the beneficial effect of reversing stagnation, thrombosis, and corpuscular adherence in the cortical venules of a rat model of middle cerebral artery occlusion. They cite further studies in which human serum albumin treatment conferred neurological and histological protection in rat stroke models of focal2–4 and global5 cerebral ischemia as well as traumatic brain injury.6

We have previously shown7 that human α1-acid glycoprotein (orosomucoid), an acute phase protein, also has a beneficial effect in a rat model of global cerebral ischemia, even 30 minutes after reperfusion. In our study, human α1-acid glycoprotein was given IV in doses of 50, 200, and 600 mg/kg. Compared with control animals treated with placebo (albumin free of α1-acid glycoprotein), the doses of 200 and 600 mg/kg successfully mitigated brain edema.

The concentration of α1-acid glycoprotein in human plasma is about 0.2 to 1.4 mg/mL. One of its major physiological roles seems to be to maintain permeability of the capillary barrier,8 which is probably achieved by increasing the negative charge of the capillary endothelium9,10 and thus reducing the transvascular transport of polyanionic macromolecules. Increased vascular permeability is a common symptom in various kinds of shock, stroke, etc. A beneficial effect of α1-acid glycoprotein can therefore be anticipated under these pathophysiological conditions. Additionally, we have shown that resuscitation with human α1-acid glycoprotein effectively re-stores cardiac output and stroke volume in a rat model of hemorrhagic/hypovolemic shock11 by tightening the microvessel walls, thereby increasing the intravascular circulating volume.

During early postischemic reperfusion, there is a progressive accumulation of polymorphonuclear leukocytes in regions of low cerebral blood flow.12 It is known from in vitro studies with these leukocytes13,14 that α1-acid glycoprotein inhibits neutrophil aggregation and superoxide anion generation. Furthermore, α1-acid glycoprotein inhibits platelet aggregation15 and enables erythrocytes to pass through micropores,16 which probably improves altered rheologic conditions.

These studies in rat models of stroke point to the possibility that the beneficial effect of albumin treatment is attributable to the α1-acid glycoprotein content of albumin solution.

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