Commentary

The deleterious effects caused by the degradation products of an intracerebral hemorrhage have been reported clinically and have been proven in animal models. Hemoglobin breakdown products following an intracerebral hemorrhage can result in free radical formation, oxidative brain damage and delayed cerebral edema. Prostaglandin E1 (PGE1) is a vasodilator that has been shown to increase cerebral blood flow in animal and human studies. PGE1 has been used in recent clinical trials to evaluate its ability to alleviate the imaging and clinical presentations of an intracerebral hemorrhage. Although the authors did include thirteen references, a substantial amount of additional references are necessary to support this theory and improve the presentation of this manuscript.

Cao et al. convincingly demonstrated that bolus injections of PGE1 are able to reduce the size of hematoma and perihematomal ischemic tissue, restore regional cerebral blood flow, and alleviate the patient's neurological deficits, all without observable adverse effects. There were, however, some flaws in the methodology of their study. Although it was stated as a randomized study, the patients were randomly assigned into two cohorts according to their admission dates. In reality, it is extremely rare to obtain a near-perfect distribution of patient parameters. Instead of using volumetry, which is a readily available technique to better define the volume of a hematoma and ischemic tissue, the authors chose to compute the volume using 1/2ABC methods. Additionally, the region of interest the authors chose to obtain Ra from might vary greatly even within the same hematoma from the same patient. Proximal and distal ischemic regions of the hematoma and areas of ischemia are always irregular. Nevertheless, their study clearly revealed the beneficial effects of PGE1 to increase rCBF and decrease edema in the brain. Whether or not the rapid resolution of the hematoma is related to PGE1 requires further investigated. This study has observed an effect, but the actual mechanism has not been explained. We welcome further molecular studies to investigate the mechanisms behind the benefits of PGE1 in improving the imaging and clinical consequences of intracerebral hemorrhages.

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