Response to Letter Regarding Article by Banki et al, “Acute Neurocardiogenic Injury After Subarachnoid Hemorrhage”

We were very interested to read Dr Gnecchi-Ruscone’s comments regarding our article.1 Similar to stress cardiomyopathy (SC), elevated catecholamine levels are frequently observed after subarachnoid hemorrhage (SAH).2 In our series, the mean plasma epinephrine and norepinephrine levels were 129±102 pg/mL and 372±494 pg/mL, respectively.3 The majority of the levels are lower than those reported for SC,4 though the difference may be explained by the fact that our samples were drawn an average of 5 days after SAH symptom onset; catecholamine levels may have been higher closer to the time of hemorrhage.

In our cohort, we observed 2 strikingly different patterns of regional wall motion abnormalities (RWMAs) of the left ventricle (LV). Some patients with SAH do have extensive apical RWMAs consistent with those observed in SC. Others, however, have RWMAs that affect only the base of the LV and have hyperdynamic function of the apex.5 In the majority of cases, both patterns of RWMA resolve during the hospitalization. Why neurological injury and/or psychological stress result in 2 such divergent LV phenotypes is currently unclear, though the apex-sparing pattern matches the typical distribution of the myocardial sympathetic nerves (less innervation at the apex).

Similar to what is seen in SC, women are more likely then men to have neurocardiogenic injury after SAH.6 The rate of cardiac troponin I release (>1 mg/dL) was 25% for women versus 9% for men. After adjusting for relevant covariates, the odds ratio for a cardiac troponin I level >1 for female gender was 35 (95% confidence interval, 2.5 to 495, P=0.009). The higher rate of cardiac injury among women with both SC and SAH does suggest that women are more susceptible to cardiac dysfunction in the setting of increased sympathetic activation.

Disclosures

None.

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