Brain is susceptible to acute MI, chronic heart failure

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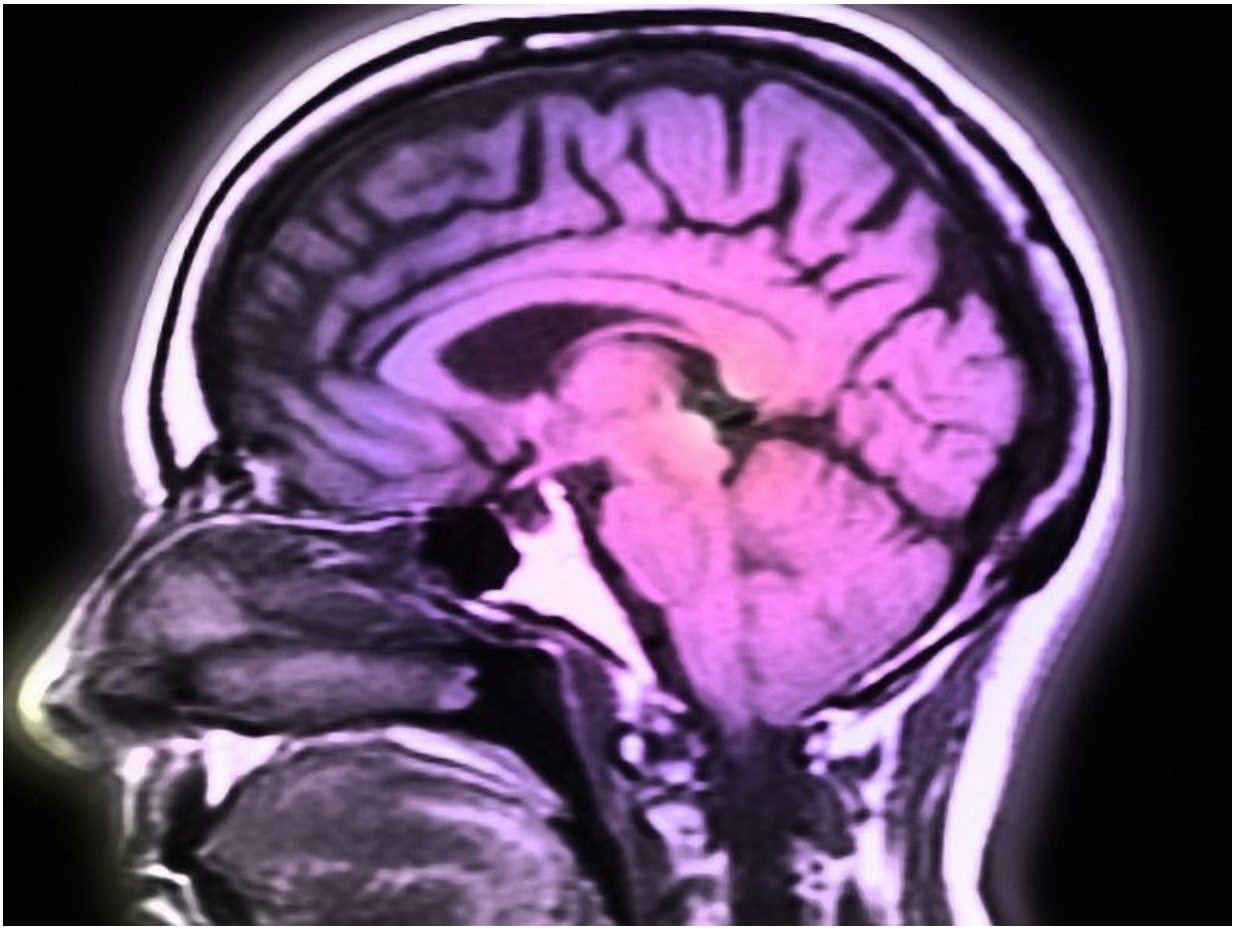

(HealthDay)—Acute myocardial infarction (MI) and chronic heart

failure have effects on the brain, according to a study published in the Jan. 23 issue of the *Journal of the American College of Cardiology*.

James T. Thackeray, Ph.D., from Hannover Medical School in Germany, and colleagues examined the influence of MI on cardiac and brain inflammation using noninvasive positron emission tomography (PET) of the heart-brain axis. Forty-nine mice underwent serial whole-body PET imaging of the mitochondrial translocator protein (TSPO) after coronary artery ligation or sham surgery. In addition, three patients after acute MI were compared with nine healthy controls.

The researchers found that, compared with sham mice, infarct mice exhibited elevated myocardial TSPO signal at one week, localized to activated CD68+ inflammatory cells in the infarct. Early TSPO signal predicted remodeling of the left ventricle at eight weeks. Brain TSPO was elevated in parallel at one week, localized to activated microglia. Progressive heart failure precipitated a second wave of neuroinflammation after interval decline at four weeks. At eight weeks, TSPO was concurrently up-regulated in remote cardiomyocytes without inflammatory cell infiltration. Treatment with angiotensin-converting enzyme inhibitor reduced acute inflammation in the heart and brain, and improved late cardiac function. Cardiac TSPO signal elevation was also seen in the infarct territory in patients, paralleled by neuroinflammation versus controls.

"Immune activation may interconnect heart and brain dysfunction, a finding that provides a foundation for strategies to improve heart and brain outcomes," the authors write.

Agents for the synthesis of the TSPO marker were provided by GE Healthcare.

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