LETTER TO THE EDITOR

Age Bias and Individual Variability in Stress Cardiomyopathy Syndromes: Mere Observations or Answers?

To the Editor:—In their detailed review of the Takotsubo cardiomyopathy, the authors¹ did not include a discussion on the age-distribution and phenotypic variation seen in the syndrome that may be crucial in context of the proposed catecholamine-mediated mechanism.

The majority of the reported patients with stress cardiomyopathy syndromes have mean age >50 years at presentation. The answer, perhaps, lies in the mechanism itself. Many physiological observations in apparently healthy subjects point towards age-related vulnerability to sympathetic perturbations. For example, aging of healthy women is associated with a decrease in the parasympathetic modulation of the heart in the setting of a preserved sympathetic tone leading to a net predominance of sympathetic control.² Decreased baroreflex buffering ability is also seen with advancing age.² Moreover, imaging studies after adjustment for perfusion changes in healthy hearts have shown that cardiac uptake 1 activity decreases with aging.³ These observations corroborate well with the known evidence on the increased cardiac norepinephrine spillover during normal aging⁴ and potentially make older hearts appear pre-disposed to catecholamine exposure in a given stressful situation.³

Why is it that while stress is ubiquitous, transient cardiomyopathy syndromes are not, and why are differences in pattern of contractile abnormalities seen? These points are worth exploring in the review for they have wide implications. A recent prospective genotype study of 182 patients of subarachnoid hemorrhage (SAH) by Zaroff and colleagues provides valuable insights in this regard.⁵ They demonstrated that certain adrenoceptor polymorphisms that are known to increase presynaptic catecholamine release or are associated

with increased heart failure/adverse cardiovascular events in elderly are also associated with an increased risk of wall motion abnormalities, degree of troponin release and systolic dysfunction in SAH. Thus, apart from providing a basis for individual susceptibility and variable wall motion abnormality, indirect evidence on a unifying mechanism (neurocardiogenic) of the stress cardiomyopathy syndromes was also highlighted in the important study and warrants future research.

Nishith K. Singh, MD, Southern Illinois University School of Medicine, 1617 Westchester Blvd#2, Springfield, IL 62704, USA; (e-mail: nishith_singh2007@yahoo.com).

REFERENCES

1. Sealove BA, Tiyyagura S, Fuster V. Takotsubo Cardiomyopathy. *J Gen Intern Med*. 2008 Aug 8.
2. Lavi S, Nevo O, Thaler I, et al. Effect of aging on the cardiovascular regulatory systems in healthy women. *Am J Physiol Regul Integr Comp Physiol*. 2007;292:R788–93.
3. Li ST, Holmes C, Kopin LJ, Goldstein DS. Aging-related changes in cardiac sympathetic function in humans, assessed by 6–18F-fluorodopamine PET scanning. *J Nucl Med*. 2003;44:1599–603.
4. Esler MD, Turner AG, Kaye DM, et al. Aging effects on human sympathetic neuronal function. *Am J Physiol*. 1995;268:R278–85.
5. Zaroff JG, Pawlikowska L, Miss JC, et al. Adrenoceptor polymorphisms and the risk of cardiac injury and dysfunction after subarachnoid hemorrhage. *Stroke*. 2006;37:1680–5.

J Gen Intern Med 24(2):286
DOI: 10.1007/s11606-008-0862-z
© Society of General Internal Medicine 2008

Published online December 17, 2008