Recurrent Right Ventricular Takotsubo Syndrome in a Diabetic Patient With Dysautonomia

To the Editor:
I read with interest the abstract by Sta. Cruz and Codolosa published recently in CHEST (March 2014), about a diabetic patient with recurrent isolated right ventricular Takotsubo syndrome in the setting of diabetic ketoacidosis and pulmonary aspiration. The authors ascribe the recurrent Takotsubo syndrome in their patient to dysautonomia, as inferred from the bradycardia and the “elevated pressures of the upper and lower esophageal sphincters suggestive of increased vagal tone.” Do the authors imply that there was diminished autonomic sympathetic activity and a resultant increased influence of the parasympathetic autonomic nervous system? If this is the position of the authors, why was there an enhanced “hypercontractility of the basal segments” of the right ventricle, a response that customarily should be attributed to the contractility-enhancing sympathetic component of the autonomic nervous system? Alternatively, one could hypothesize that the different parts of the right ventricle were under the influence of the two different components of the autonomic nervous system.

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Response

To the Editor:
I thank Dr Madias for his interest in our abstract. He requested clarification on whether we implied that there was “diminished autonomic sympathetic activity and a resultant increased influence of the parasympathetic autonomic nervous system” responsible for Takotsubo cardiomyopathy (TC) in the patient.

We refer to autonomic dysfunction as a disruption of the delicate balance between the sympathetic and parasympathetic arms of the autonomic nervous system, a recognized phenomenon in diabetes. With loss of normal regulatory feedback mechanisms, dysregulated autonomic reflexes may predominate at any point. We mentioned this patient’s episodic bradycardia and aspiration from high esophageal sphincter pressures as evidence of dysregulated parasympathetic hyperactivity or autonomic dysfunction and not a result of diminished sympathetic activity. In fact, it could be the recurrent aspiration that served as physical stress and precipitated recurrent TC in this patient.

The exact pathophysiology of TC is still a mystery. It usually presents as apical ballooning and basal hyperkinesis of the left ventricular myocardium as precipitated by emotional and physical stress, which is why it remains a special interest for neurocardiologists. Using radioactive metaiodobenzylguanidine (MIBG), a chemical structurally similar to norepinephrine, Akashi and colleagues in 2004 hypothesized that TC might be caused by neurogenic myocardial stunning due to autonomic imbalance. Initial MIBG myocardial scintigraphy depicted a unique pattern of ventricular asynergy with decreased uptake of MIBG during the acute phase of TC isolated in the apex, reflecting cardiac sympathetic denervation. This may explain why, despite high levels of catecholamines in the blood, the left ventricle would demonstrate apical hypokinesis with basal hyperkinesis. Three years later, Akashi and colleagues sought to prove this hypothesis clinically by analyzing domains of heart rate variability, parameters considered as measures of cardiac autonomic function. They found out that time domain heart rate variability parameters increased and coincided with decreased left ventricular

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wall motion during the acute phase of TC. These parameters significantly improved as left ventricular wall motion returned to normal, supporting their previous hypothesis.

Regarding the possible mechanism of the atypical presentation of TC in this patient, the recurrent right ventricular basal hyperkinesis and apical ballooning may be explained by the differences of regional sympathetic innervation and catecholamine receptor density in the ventricular myocardium. We hope this answers Dr Madias’ query and sheds some light on the mystery of TC.

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