

Finally, left-to-right shunting secondary to restrictive CTD and a patent foramen ovale or ASD might lead to central cyanosis and symptoms consistent with increased portal venous pressure, such as ascites, coagulopathy, hepatic dysfunction, and protein-losing enteropathy.⁵ In such cases, ASD closure should be undertaken after surgical or percutaneous balloon correction of CTD.⁶

*Efrén Martínez-Quintana, MD, PHD,
Cardiology Service,
Insular-Materno Infantil University Hospital; and
Fayna Rodríguez-González, MD,
Dr. Negrín University Hospital of Gran Canaria;
Las Palmas de Gran Canaria, Spain*

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Myocardial Apical Hypertrophy and Takotsubo Cardiomyopathy

To the Editor:

I appreciated the article by Roy and colleagues¹ in the *Texas Heart Institute Journal*. To the authors' knowledge, theirs were the first reported cases in which apical-variant hypertrophic cardiomyopathy was masked by apical ballooning from stress-induced cardiomyopathy. The authors added information about 5 reported cases of patients with hypertrophic cardiomyopathy (HCM) who had experienced an episode of takotsubo cardiomyopathy, all of whom had the obstructive HCM "with asymmetric septal hypertrophy, not apical-variant HCM." What was actually observed is a recently detected phenomenon of apparent left ventricular (LV) apical hypertrophy,^{2,3} which occasionally is seen in the

subacute and chronic phase of convalescence from takotsubo cardiomyopathy. It is caused by transient myocardial edema.⁴ Kato and colleagues³ observed apical hypertrophy of the LV at approximately 3 weeks after onset, when the wall motion had improved; the ventricular wall gradually became thinner, and the transient apical hypertrophy was attributed to hypertrophic signaling in the myocardium, which was stimulated by catecholamines.³ Myocardial edema with a hypertrophic LV apex has been reproducibly detected on cardiac magnetic resonance images⁴ and echocardiograms. Whether the LV hypertrophy¹ represents apical HCM or takotsubo-induced myocardial edema can be resolved by observing subsequent electrocardiograms of these patients for chronically persisting giant negative T waves⁵ and R waves⁶ in the mid-precordial leads, and by comparing old and follow-up echocardiograms.

*John E. Madias, MD, FACC,
Icahn School of Medicine at Mount Sinai,
New York City; and
Division of Cardiology,
Elmhurst Hospital Center,
Elmhurst, New York*

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