

The Reply

We thank Temtanakitpaisan et al for their interest in our article on reversible myocardial dysfunction in acute cholecystitis. As mentioned, reversible myocardial dysfunction can be precipitated by a variety of stressors, ranging from acute medical illness to emotional or physical stress (Table 1). Of note, reversible myocardial dysfunction has been described in the literature for more than 4 decades, but its pathogenesis remains poorly understood.² It is not known why this disorder is more common in postmenopausal women, and only recently has the predilection for specific myocardial territories (eg, the left ventricular apex) been elucidated.³ Reversible myocardial dysfunction may affect different myocardial segments in the same patient at different time points. This so-called migratory takotsubo cardiomyopathy can be precipitated by rare stressors, such as smoldering cholecystitis, as in our patient.

Reversible myocardial dysfunction clinically reduces myocardial contractile reserve, causing intolerance to volume resuscitation, and it is conceivable that occult ventricular dysfunction in critically ill patients mediates progression toward cardiorespiratory compromise.⁴ Other complications of reversible myocardial dysfunction include ventricular thrombi, posing a risk for systemic embolization.⁵ Overall, reversible myocardial dysfunction increases morbidity and mortality from the underlying disease precipitant, and prompt identification may help prevent some of these adverse consequences. Postulated mechanisms for reversible myocardial dysfunction include catecholamine excess, coronary artery spasm, and microvascular dysfunction. Apical dysfunction in part may be caused by dynamic mid-cavity or left ventricular outflow tract obstruction. Overall, this syndrome may represent a maladaptive response to catecholamine excess during organ failure among critically ill patients.

By contrast, reversible myocardial dysfunction associated with emotional stress is usually a more benign condition that does not affect overall prognosis. If appropriately recognized, this may prevent unnecessary healthcare resource use, including invasive cardiac

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Table 1 Triggers for Reversible Myocardial Dysfunction

Neurogenic Stunned Myocardium

Subarachnoid hemorrhage

Stroke

Subdural hematoma

Cranial trauma

Electroconvulsive therapy

Acute Respiratory Failure

Upper airway obstruction

Asthma

Pulmonary embolism

Acute lung injury

Acute respiratory distress syndrome

Anaphylaxis

Trauma iniuries

Pulmonary contusion

Multiple trauma

Hemorrhagic shock

Blast injury

Burn injuries

Postsurgical pathology transplant

Sepsis

Systemic inflammatory response syndrome

Acute pancreatitis

Acute cholecystitis

Cardiac arrest

Poisoning

Rhabdomyolysis

Pheochromocytoma

Thyroid pathology

Arrhythmias

Hyperthermia/hypothermia

Obstructive jaundice

Emotional stress

Nutrition

Adapted from Bailen et al.6

procedures. Management of reversible myocardial dysfunction is geared toward addressing the underlying stressor and supportive care. In rare cases, patients with reversible myocardial dysfunction may need temporary support with inotropic agents and cardiac assist devices, such as intra-aortic balloon pumps.⁶

Reversible myocardial dysfunction is increasingly recognized and may be triggered by a myriad of noncardiac stressors. Clinicians should maintain a high index of suspicion in patients with noncardiac illnesses presenting with new-onset ventricular dysfunction in the absence of underlying ischemic heart disease risk factors.

Vikas Aggarwal^a
Mori J. Krantz^{a,b}

^aDivision of Cardiology
Department of Internal Medicine
University of Colorado
Aurora

^bDenver Health Medical Center
Cardiology Division
Denver, Colo

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References

 Aggarwal V, Krantz MJ. Migratory takotsubo cardiomyopathy in the setting of cholecystitis. Am J Med. 2012;125:e5-e6.

- Heyndrickx GR, Millard RW, McRitchie RJ, et al. Regional myocardial functional and electrophysiological alterations after brief coronary artery occlusion in conscious dogs. *J Clin Invest*. 1975;56: 978-985.
- Paur H, Wright PT, Sikkel MB, et al. High levels of circulating epinephrine trigger apical cardiodepression in a beta2-adrenergic receptor/Gi-dependent manner: a new model of Takotsubo cardiomyopathy. Circulation. 2012;126:697-706.
- Chockalingam A, Mehra A, Dorairajan S, Dellsperger KC. Acute left ventricular dysfunction in the critically ill. *Chest.* 2010;138: 198-207
- Guest TM, Ramanathan AV, Tuteur PG, et al. Myocardial injury in critically ill patients. A frequently unrecognized complication. *JAMA*. 1995;273:1945-1949.
- Bailen MR. Reversible myocardial dysfunction in critically ill, noncardiac patients: a review. Crit Care Med. 2002;30:1280-1290.