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Successful Retrial of ECT Two Months After ECT-Induced Takotsubo Cardiomyopathy

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We discuss a case of takotsubo cardiomyopathy in the setting of ECT in a severely depressed patient who had not previously responded to extensive trials of pharmacotherapy but who had responded to a previous course of ECT. Further complicating this case is the fact that the patient's depression was refractory to medication trials after the onset of takotsubo cardiomyopathy, leading to the decision to embark on a second course of ECT after consultation with the cardiology and anesthesiology services.

Takotsubo cardiomyopathy, first described in Japan in the 1990s, is a condition in which the shape of the left ventricle during systole resembles an octopus fishing trap, or *takotsubo* (1). Takotsubo cardiomyopathy is characterized by an acute coronary syndrome-like event in which a patient can experience chest pain and shortness of breath acutely (2, 3). These symptoms generally occur in the setting of severe emotional stress, often after the sudden death of a loved one—hence the alternative term "broken heart syndrome" (4). The patient may have ECG changes, typically T wave inversions and prolonged QT interval, but ST elevations have also been reported (5). A characteristic apical ballooning and hypokinesis developing in the left ventricle are often seen on echocardiography. A small proportion of patients (2.5%) may develop intracavitary thrombus in the affected ventricle, with 0.8% of those having embolic complications, including stroke, renal infarction, and popliteal artery thrombosis (6). Initially the left ventricular ejection fraction diminishes dramatically, causing the symptoms of heart failure; however, recovery of cardiac function is often complete within the first week following the insult. Cardiac enzymes may be mildly elevated, but on cardiac catheterization there is usually no evidence of coronary artery disease, or only "mild luminal irregularities" may be noted (7). Contraction band necrosis may be seen on biopsy in the more serious cases. The cause of takotsubo cardiomyopathy is unknown, but several mechanisms have been suggested, including ischemia from coronary artery spasm, microvascular spasm, and direct myocyte injury (5).

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Case Presentation

Ms. H is a 71-year-old single, employed Caucasian woman with a past psychiatric history of one prior major depressive episode with psychotic features treated successfully with ECT at our hospital in 2003. She had one prior psychiatric hospitalization and no prior suicide attempts and had not been in outpatient psychiatric treatment until 1 month before her first admission.

Ms. H's prior episode of depression began with a decline in mood after the terrorist attacks of September 11, 2001. "I just couldn't get over it," she recounted. She remained functional, however, until 2003, when her symptoms worsened in the setting of persisting financial stress. Ms. H's prominent symptoms included low mood, anxiety, and insomnia. An outpatient psychiatrist prescribed fluoxetine and olanzapine, which brought minimal improvement.

In May 2003 she visited the emergency department five times, concerned that she was having a heart attack, but she was diagnosed with panic attacks. She has a history of intermittent chest pain over the years, thought to be caused by costochondritis. In June 2003, on her second visit to her outpatient psychiatrist, 1 month after being started on medications, Ms. H was escorted to the emergency department for evaluation of her depressive symptoms and agitation. She was hospitalized involuntarily at that time for 2 months for treatment of a major depressive episode with psychotic features. In the hospital her complaints were predominantly somatic; in particular, she was concerned that her intestines were "full" and that she would have urinary incontinence. Her depression was treated with 100 mg/day of sertraline, 75 mg/day of extended-release venlafaxine, and 200 mg/day of quetiapine.

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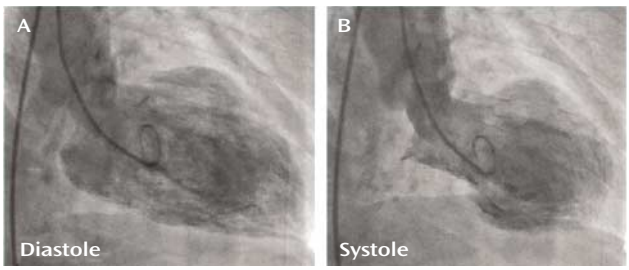
FIGURE 1. Images From Contrast Left Ventriculography Suggesting Takotsubo Cardiomyopathy[a]



[a] In panel A, a normal-appearing left ventricle in diastole. In panel B, ventricular contraction demonstrating apical ballooning ("octopus trap") consistent with takotsubo cardiomyopathy.

Ms. H had no improvement in her depressive symptoms after 2 months of treatment, and she was transferred to our institution in August 2003 and enrolled in an ECT study examining affective and cognitive outcomes of both right unilateral ECT and bilateral ECT with brief-pulse and ultrabrief-pulse stimuli. She received 13 ultrabrief bilateral treatments and showed significant improvement after just a few treatments. Her Hamilton Depression Rating Scale score at completion of treatment was in the normal range. During the period when she was receiving ECT, her cognition was severely impaired, with Mini-Mental State Examination (MMSE) scores as low as 15. However, her cognitive impairment rapidly cleared after cessation of ECT. She was discharged in October 2003 on 600 mg/day of lithium and 75 mg/day of nortriptyline. She remained on this regimen for 4–5 months, then stopped all medications and psychiatric treatment with a complete return to her affective baseline.

Before her current presentation, Ms. H had been euthymic and off all medications for 4 years. In May 2007, in the setting of multiple stressors, she had a return of depressive symptoms, including anhedonia, insomnia, anergia, and low motivation. Her concentration worsened, and she had memory impairment. Additionally, she was preoccupied with her financial losses and was overwhelmed by feeling as though she had "messed up." An outpatient psychiatrist prescribed 50 mg of nortriptyline daily and 300 mg of quetiapine nightly. Her nortriptyline dose had to be decreased to 25 mg because of dry mouth and constipation. Her depressive symptoms were largely unchanged on this regimen, and she was referred to our hospital in July 2007 for reevaluation for ECT.

At the time of her admission, in addition to the aforementioned symptoms, Ms. H had numerous somatic complaints, consistent with her prior major depressive episode. She reported an inability to taste food and a concern that she had "let a urinary tract infection go too long" and would be incontinent. She also had frequent complaints of chest pain (reproducible and atypical), which had been diagnosed by her cardiologist as costochondritis and treated with nonsteroidal anti-inflammatory medications. Her somatic preoccupations bordered on delusional, but she had no other clear psychotic symptoms. She denied suicidal ideation.

During the first few days of admission, while pre-ECT evaluation was taking place, the patient's blood pressure was noted to be significantly elevated (around 180/100 mm Hg) and was managed during this time with 100 mg of extended-release metoprolol and 5 mg of amlodipine, both twice a day, with good effect (systolic blood pressure in the range of 120–140 mm Hg), through the morning of her first ECT.

Three days after admission the patient underwent her first unilateral ECT treatment, after declining the recommended bilateral treatment. Her psychotropic medications at the time were 25 mg of nortriptyline at bedtime, 200 mg of quetiapine at bedtime, and 0.5 mg of lorazepam twice daily. The patient was titrated to seizure threshold and was given three stimulations; the first two were subconvulsive, and the third produced a seizure of 66 seconds in duration, with an EEG time of 81 seconds.

There were no complications, but the patient was noted to be significantly hypertensive immediately after the procedure, with an initial postictal blood pressure of 208/122 (maximum heart rate in the 120s). Subsequent interval monitoring showed a steady descent in blood pressure and heart rate, from 190/103 mm Hg with a pulse of 115 at 5 minutes to 175/96 mm Hg and a pulse of 101 at 25 minutes. Her blood pressure stabilized in the 160s/90s within 1 hour after the procedure.

Several hours later, the patient reported feeling substernal chest tightness. An ECG showed no change, and a troponin test was negative. A second troponin test, however, was positive at 3.28 ng/ml. A repeat ECG showed new changes, including T wave inversions in V1, V2, and V3 and 1-mm ST elevations in I, V2, and V6. On examination, the patient had both reproducible chest pain similar to her costochondritis pain and a deeper, constant substernal chest pain. Her lungs were clear, and her heart rate was regular without murmurs. She was given 325 mg of aspirin and was immediately transferred to the cardiology service with the diagnosis of a non-ST-elevation myocardial infarction. She was treated initially with oxygen, nitroglycerin, and 25 mg of metoprolol every 6 hours. A heparin drip was started, the metoprolol dosage was increased to 50 mg every 6 hours, and aspirin was continued at 325 mg daily. A high-dose statin was added and clopidogrel was loaded at 300 mg, then 75 mg daily was given in anticipation of cardiac catheterization.

Echocardiography revealed a significantly decreased ejection fraction of 20% with an "apical ventricular aneurysm and preserved function of a small cuff of tissue at the base." Cardiac catheterization showed no evidence of significant occlusive coronary artery disease, the usual mechanism for myocardial infarction, but rather showed abnormal left ventricular function (Figure 1) with severe hypokinesis of the anterior, apical, and inferior walls, an overall appearance most compatible with takotsubo cardiomyopathy.

After 1 week of continued cardiac medication and successful management of the patient's hypertension on the cardiology service, a repeat echocardiogram showed that her left ventricular function had dramatically improved (with an ejection fraction of 40%–45%), which is characteristic of takotsubo cardiomyopathy (5).

During this time, the patient was managed psychiatrically by the consultation-liaison service. She remained anxious and dysphoric, preoccupied with finances, worried that she might become incontinent, and showing memory difficulties, but she was not focused on her cardiac condition. She continued treatment with quetiapine and nortriptyline; again, an increase in her nortriptyline

dosage induced side effects, and the low dose was restored. At that time the cardiology team and ECT specialist judged that ECT had precipitated her takotsubo cardiomyopathy and that another trial of ECT would be too risky from a cardiac standpoint.

After 9 days, the patient was transferred back to the inpatient psychiatric unit for further treatment of her depression. Since ECT was deemed too risky, treatment focused on pharmacotherapy. Her medications on transfer included 1 mg of risperidone at bedtime, 150 mg of quetiapine at bedtime, 25 mg of nortriptyline daily, 1 mg of lorazepam twice daily (and an additional 0.5 mg every 6 hours as needed for anxiety), 50 mg of metoprolol twice daily, 325 mg of aspirin daily, 80 mg of atorvastatin daily, 75 mg of clopidogrel daily, and 40 mg of esomeprazole daily, as well as senna, docusate sodium, and a multivitamin.

Initially the team continued her on risperidone, quetiapine, and nortriptyline, and she was noted to be engaged in some activities on the unit and would smile at times spontaneously. She was soon cross-tapered from quetiapine to mirtazapine for sleep and to augment her antidepressant regimen, as there was concern that she had orthostatic hypotension from the quetiapine. Risperidone was also slowly increased to treat her somatic preoccupations.

Because she could not tolerate doses of nortriptyline higher than 25 mg, she was started on venlafaxine at 37.5 mg and titrated up to 150 mg. She was also continued on 2.5 mg of risperidone daily and 45 mg of mirtazapine daily for sleep, and her quetiapine dose was decreased to 50 mg at bedtime. On this medication regimen, she was initially noted to be brighter and was sleeping better. However, within 2 weeks, her somatic preoccupations, which had partially remitted, returned, and she became focused on feeling dirty and losing fluid from her body. Her mood began to worsen, and she expressed passive suicidal ideation, noting that she "wanted it to be over." Her dose of risperidone was limited by the emergence of severe extrapyramidal side effects, and her dose of venlafaxine was limited by increasing blood pressure.

At this time, the psychiatric team became concerned with her neurovegetative symptoms, including decreased oral intake resulting in a 6 lb weight loss over 4 weeks, her inability to get out of bed without significant encouragement and assistance from staff, and the lack of effectiveness of the medication trials in treating her depression. A retrial of ECT was discussed as a possibility approximately 1 month after her initial ECT treatment.

A follow-up echocardiogram 1 month after her cardiac event showed a mildly hypertrophied left ventricle with a normal ejection fraction and normalized wall motion compared to the prior echocardiogram (8 days after the event). The general impression of the consulting cardiologist (W.S.) was that the patient would be able to tolerate further ECT from a cardiac perspective as long as she received adequate beta-blockade during the procedure and for several hours afterward. He recommended that she be maintained on an esmolol drip during the course of the treatment and for several hours after the treatment, with cardiac monitoring and monitoring of her postprocedure troponin levels. The anesthesiologist (E.J.H.) was also consulted for his impressions regarding the safety of the procedure and the appropriate setting in which to conduct the procedure, given the need for the esmolol drip and the postprocedure monitoring. He determined that the procedure would best be carried out in the postanesthesia care unit for adequate cardiac monitoring.

After extensive discussion among the cardiology, anesthesiology, and ECT psychiatry services and the primary treating team, it was decided that a rechallenge of ECT would be the best option for Ms. H in light of her complete response to ECT in the past and the failure of multiple medication trials. Her second ECT trial during this hospitalization was initiated approximately 2 months after the initial treatment. Per our usual protocol, atropine was administered to prevent bradycardia, methohexital for induction of anesthesia, and succinylcholine as a paralytic agent. A nicardipine drip was started to maintain normal blood pressure. In order to reduce the usual catecholamine surge, an esmolol drip was started several minutes before ECT delivery and maintained until several hours afterward.

The patient was monitored in the postanesthesia care unit for 5 hours after the procedure, and her cardiac troponin levels were checked at 4, 8, and 24 hours and were within normal limits. Given the uneventful initial ECT treatment, the team felt that further ECT treatments could be safely administered in the ECT suite on the psychiatric unit with monitoring of pre- and post-ECT troponin levels and using increased beta-blockade with metoprolol.

During this retrial of ECT the patient received 13 ultrabrief unilateral ECT treatments. She was continued during this time on 50 mg quetiapine daily and lorazepam as needed. Risperidone was eventually tapered off because the patient had significant bradykinesia, unsteady gait, and short-term memory impairment. After her first few ECT treatments, Ms. H developed delirium; the etiology was unclear, but the presentation was similar to that of her first ECT course in 2003. It was thought that her delirium may have been caused by ECT itself, by the benztropine that she had been maintained on to counteract the extrapyramidal symptoms from risperidone, or by polypharmacy in general. In addition, around this time she had been started on duloxetine as she was not able to tolerate therapeutic doses of nortriptyline. During the period from ECTs 8 to 13, the patient was found to be more disorganized, with difficulty attending to even simple tasks, and her MMSE score dipped to 21/30 at its lowest. However, this was thought to be due mostly to her depression. Her score on the Quick Inventory of Depressive Symptomatology at this time was 13. The treatment team thought that her symptoms of depression were not improving; she continued to do poorly, having difficulty caring for herself, feeling depressed, not eating much, having continued somatic delusions, and having passive suicidal ideation. Duloxetine was discontinued as there was some concern that it might have been contributing to the delirium. Given the lack of improvement with 13 right unilateral ECT treatments and the patient's past history of successful bilateral treatments, the team decided to try bilateral ECT.

The patient had a total of six bilateral treatments, three of which were bifrontal because of an abrasion with cellulitis at a left temporal probe site. She had significant cognitive effects from the bilateral ECT; although on non-ECT days she could occasionally score up to 28/

30 on the MMSE, on some days she was unable to speak coherently, could not write a sentence, and had severely impoverished thinking. Her score on the Quick Inventory of Depressive Symptomatology had plateaued at 13. To minimize the dose of metoprolol she was on, treatment for hypertension was supplemented with increasing doses of verapamil and lisinopril, although there was no significant change in her depressive symptoms. At this time, the team decided that the benefits of the treatment did not outweigh the risks of continuing, and Ms. H was started on aripiprazole and lamotrigine. After consultation with the cardiology service, nortriptyline was also added to the regimen. Quetiapine was continued throughout for insomnia. She was also started on donepezil in the hope that it would help with her cognitive impairment.

Her last 4 weeks in the hospital were marked by slow improvement in mood and neurovegetative symptoms to a new baseline that included an improved mood, increased speech fluency, increased ability to care for herself (but not independently), and improved gait. While her somatic delusions lessened, she continued to have some odd beliefs (bordering on delusional), including that she was running out of clothes, that she was dirty, and that "food is spilling over from my intestines" at times. At the time of discharge, her mood was improved, her sleep and appetite normalized, and her beliefs that her body was not working properly greatly diminished. She was more energetic and hopeful about the future. Although she was discharged to live with a family member, she hoped to return to independent living and to resume her work.

In her cardiac course during the last 4 weeks of her hospitalization, Ms. H had intermittent chest pain; her troponin tests were always negative, and the pain was thought to be the chronic noncardiac chest pain that she had had for many years. Her hypertension medication regimen was changed multiple times, including a tapering of her beta-blocker because of concerns that it was contributing to her depression and a trial of hydrochlorothiazide, which needed to be stopped as her sodium levels decreased to 132 mmol/liter.

Her medications on discharge were 100 mg of quetiapine at bedtime, 75 mg of lamotrigine daily, 10 mg of aripiprazole daily, 80 mg of nortriptyline at bedtime, 5 mg of amlodipine daily, 0.5 mg of lorazepam twice bid, 40 mg of lisinopril daily, 2.5 mg of donepezil daily, one packet of polyethylene glycol daily, 81 mg of aspirin daily, 40 mg of simvastatin daily, and 40 mg of esomeprazole daily, as well as senna, docusate sodium, and a multivitamin.

Discussion

This case describes a second course of ECT in an elderly female patient 2 months after the diagnosis of ECT-induced takotsubo cardiomyopathy. Dr. Heyer (Professor of Clinical Anesthesiology and Clinical Neurology and Director of Neuroanesthesia, Columbia University Department of Anesthesiology), Dr. Sherman (Associate Clinical Professor, Department of Cardiology), and Dr. Prudic (Associate Clinical Professor, Department of Psychiatry) were integrally involved in the case and participated as discussants at the case presentation.

Dr. Heyer:

The catecholamine surge that we propose caused the takotsubo cardiomyopathy following the first administration of ECT in this case was iatrogenic. Given at least in part the iatrogenic etiology, it is key in this setting to carefully examine the options for limiting the body's exposure to excess catecholamines.

Typically, medications used for induction and paralysis in ECT include methohexital and succinylcholine. Blood pressure and heart rate usually increase after ECT administration, and it is reasonable to administer beta-blockade to limit the increases. Generally, the beta-blocker used is esmolol because it is easily titratable and has a short half-life. In this case, not only was esmolol used extensively as a drip before and after the procedure, but a longer-acting beta-blocker, labetalol, was also used to manage blood pressure during the time surrounding the procedure and metoprolol was used on a more long-term basis to ensure tighter blood pressure control over time.

Beta-blockers are the traditional medication of choice for achieving hemodynamic stability in this and many other perioperative settings. Several alternatives were discussed in this case but ultimately were not tried given the known and respected cardioprotective qualities of beta-blockers. (Interestingly, pindolol has even been shown to enhance response to ECT in depressed patients when given during the first six ECT treatments [8].)

Another general question that arises is whether there is a correlation between cardiac effects and ECT electrode placement. To date, there is limited evidence that electrode placement affects cardiovascular responses. A study by Prudic et al. (9) compared heart rate and blood pressure in bilateral versus unilateral ECT and found no difference. In a small study of 11 patients, Lane et al. (10) found that during the postictal phase, heart rate during bilateral ECT significantly exceeded that during right unilateral ECT despite no differences in peak heart rate (10). Another study (11) found that the magnitude of postictal rate pressure product, an index of myocardial oxygen demand, was greater in bilateral ECT compared to right unilateral ECT. One possible explanation for why bilateral ECT increases postictal heart rate compared to right unilateral ECT is that bilateral ECT releases more circulating catecholamines than right unilateral ECT, although there is no direct evidence that this occurs. It should be emphasized that Ms. H had her cardiac event following one right unilateral ECT treatment and that she subsequently tolerated bilateral ECT.

Dr. Sherman:

One of the most remarkable aspects of this case is that a second attempt at ECT, given known and demonstrated risks, proved to be successful in terms of cardioprotection and safety. The protective elements included close cardiac monitoring, stabilization of blood pressure and heart rate with beta-blockade prior to the procedure, and generous use of beta-blockade during the procedure to further control the known acute physiologic effects of ECT, that is, el-

evated heart rate and blood pressure. This case suggests that ECT can be successfully administered in a patient who has recently (in this case 2 months prior) had a cardiac event such as takotsubo cardiomyopathy.

Given the stressors involved in ECT, it is interesting that takotsubo cardiomyopathy has not been observed more often in this setting. Perhaps the syndrome is quite rare or presents in difficult-to-recognize subclinical forms. While this manuscript was in revision, O'Reardon et al. published a case of takotsubo cardiomyopathy following ECT (12). A sustained course of congestive heart failure has been described in some patients undergoing ECT (13, 14), the pathogenic mechanisms for which have not been elucidated. Based on what is known, it is intriguing, and within reason, to consider takotsubo cardiomyopathy as a possible source of myocardial dysfunction in these patients. Common to both groups of patients is the heightened catecholamine release, induced by emotional trauma and other factors, in the setting of ECT. While the intermediate (and long-term) outcome of patients with takotsubo cardiomyopathy is of recovery of left ventricular function, a small percentage of patients may not recover and as a result emerge from the process with congestive heart failure. In this population, early and serial assessment of left ventricular function would shed light on the mechanisms underlying the chronic condition.

Dr. Prudic:

One question arising from this case is why this course of ECT was not more effective, given the previous successful treatment of the patient's psychotic depression with ECT 4 years earlier. One possible explanation is the extensive use of beta-blockers as well as other psychopharmacology during the second course of ECT.

The consequences of relying heavily on beta-blockade are unknown. Particularly concerning is the relationship between beta-blockers and depression (15–17). In a comprehensive review, however, propranolol was concluded to be problematic only after long-term use (18). Depression after myocardial infarction in the setting of beta-blocker use has also been described. It has been observed in post-myocardial infarction patients that both beta-blocker use and the incidence of depression is high (19). Although van Melle (20) described little depressive effect in patients who used beta-blockers after myocardial infarction, his work focused predominantly on men, and the phenomenon of beta-blocker-induced depression is observed more frequently in women. The lipophilicity of the beta-blocker does not seem to make a difference, as the most extensive review of beta-blockade and depression reported no difference between lipophilic and nonlipophilic agents (21).

In this case, however, the quantity of beta-blocker that the patient received may have been a factor in the outcome of ECT. Not only did the patient take standing metoprolol daily, but she also received a large esmolol bolus and drip immediately surrounding the ECT procedure as well as labetalol for blood pressure management. One study that examined the effect of esmolol on seizure duration determined that it shortened the duration of the motor convulsion and degraded the quality of the ictal regularity, a factor that may have contributed to the patient's lack of response (22).

The literature describes a phenomenon of decreased ejection fraction by up to 30% in patients after one ECT treatment (23). It is unknown how this occurrence might be related to a syndrome such as takotsubo cardiomyopathy, which is also defined by a decreased ejection fraction. It is also unknown how the significant doses of beta-blockers used in this case might affect that phenomenon, perhaps worsening the ejection fraction to the point of causing symptoms and producing the takotsubo syndrome. Cardiac complications in ECT are well described, however (24).

Another question that arises in this case is whether the patient's baseline brain chemistry was significantly different during this second course of ECT. Perhaps the catecholamines, thought to be a causative factor in takotsubo cardiomyopathy, play a role in treatment of the depression and by blocking them so extensively with beta-blockers, we are depriving the patient of a potentially helpful component of ECT. Cases have been described in the literature where catecholamines were depleted in formerly depressed patients in remission. In one study, catecholamine depletion was found to result in an increase in depressive and anhedonic symptoms (25).

Particularly concerning in the second ECT course was the development of significant cognitive impairment. Saber and Cain (26) described impaired learning and performance of spatial skills in rats in the setting of beta-blocker use concomitantly with anticholinergic agents. Perhaps the use of a different cardioprotective agent, with an altogether different mechanism, might have less cognitive effect. One alternative is remifentanyl, a derivative of fentanyl, a very-short-acting opioid that serves effectively as a cardiac depressant, thus helping to lower heart rate. Its opioid qualities have also been useful in providing analgesia, and it has not been shown to have any adverse long-term cognitive effects but has been shown to prolong seizure time, which may be advantageous (27). High doses of naloxone administered immediately before ECT have been shown both to diminish anterograde amnesia and to improve performance on an attention task (28).

Conclusion

Appropriate cardiac management in this case required a close working relationship between the cardiology, anesthesiology, and psychiatry teams. Furthermore, treatment of this patient required thorough consideration of her medication regimen from the perspective of all three disciplines, including weighing the risks of using high doses of beta-blockers and more cardio-neutral psychiatric medications (risperidone and quetiapine). In addition to describing a second course of ECT in a patient who had sustained takotsubo cardiomyopathy in the setting of ECT

only 2 months earlier, this case serves as a nidus for several complex questions, as detailed by our discussants.

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References

1. Dote K, Sato H, Tateishi H, Uchida T, Ishihara M: [Myocardial stunning due to simultaneous multivessel coronary spasms: a review of 5 cases]. J Cardiol 1991; 21:203–214 (Japanese)
2. Pavin D, Le Breton H, Daubert C: Human stress cardiomyopathy mimicking acute myocardial syndrome. Heart 1997; 78: 509–511
3. Tsuchihashi K, Ueshima K, Uchida T, Oh-mura N, Kimura K, Owa M, Yoshiyama M, Miyazaki S, Haze K, Ogawa H, Honda T, Hase M, Kai R, Morii I; Angina Pectoris-Myocardial Infarction Investigations in Japan: Transient left ventricular apical ballooning without coronary artery stenosis: a novel heart syndrome mimicking acute myocardial infarction (Angina Pectoris-Myocardial Infarction Investigations in Japan). J Am Coll Cardiol 2001; 38:11–18
4. Wittstein IS, Thiemann DR, Lima JA, Baughman KL, Schulman SP, Gerstenblith G, Wu KC, Rade JJ, Bivalacqua TJ, Champion HC: Neurohumoral features of myocardial stunning due to sudden emotional stress. N Engl J Med 2005; 352:539–548
5. Wittstein IS: The broken heart syndrome. Cleve Clin J Med 2007; 74(suppl 1):S17–S22
6. de Gregorio C, Grimaldi P, Lentini C: Left ventricular thrombus formation and cardioembolic complications in patients with Takotsubo-like syndrome: a systematic review. Int J Cardiol 2008; 131:18–24
7. Dec GW Jr, Stern TA, Welch C: The effects of electroconvulsive therapy on serial electrocardiograms and serum cardiac enzyme values: a prospective study of depressed hospitalized inpatients. JAMA 1985; 253:2525–2529
8. Shiah IS, Yatham LN, Srisurapanont M, Lam RW, Tam EM, Zis AP: Does the addition of pindolol accelerate the response to electroconvulsive therapy in patients with major depression? a double-blind, placebo-controlled pilot study. J Clin Psychopharmacol 2000; 20:373–378
9. Prudic J, Sackeim HA, Decina P, Hopkins N, Ross FR, Malitz S: Acute effects of ECT on cardiovascular functioning: relations to patient and treatment variables. Acta Psychiatr Scand 1987; 75:344–351
10. Lane RD, Zeitlin SB, Abrams R, Swartz CM: Differential effects of right unilateral and bilateral ECT on heart rate. Am J Psychiatry 1989; 146:1041–1043
11. Mayur PM, Gangadhar BN, Girish K, Prasad KM, Subbakrishna DK, Janakiramiah N: Acute post-ECT cardiovascular response: a comparison of threshold right unilateral and bilateral ECT. J ECT 1998; 14:94–98
12. O'Reardon JP, Lott JP, Akhtar UW, Cristancho P, Weiss D, Jones N: Acute coronary syndrome (Takotsubo cardiomyopathy) following electroconvulsive therapy in the absence of significant coronary artery disease: case report and review of the literature. J ECT 2008; 24:277–280
13. Ring BS, Parnass SM, Shulman RB, Phelan J, Khan SA: Cardiogenic shock after electroconvulsive therapy. Anesthesiology 1996; 84:1511–1513
14. Fuenmayor AJ, el Fakih Y, Moreno J, Fuenmayor AM: Effects of electroconvulsive therapy on cardiac function in patients without heart disease. Cardiology 1997; 88:254–257
15. Pollack MH, Rosenbaum JF, Cassem NH: Propranolol and depression revisited: three cases and a review. J Nerv Ment Dis 1985; 173:118–119
16. Fitzgerald JD: Propranolol-induced depression. Br Med J 1967; 2:372–373
17. Patten SB: Propranolol and depression: evidence from the antihypertensive trials. Can J Psychiatry 1990; 35:257–259
18. Stoudemire A, Brown JT, Harris RT, Blessing-Feussner C, Roberts JH, Nichols JC, Houpt JL: Propranolol and depression: a reevaluation based on a pilot clinical trial. Psychiatr Med 1984; 2: 211–218
19. Schleifer SJ, Slater WR, Macari-Hinson MM, Coyle DA, Kahn M, Zucker HD, Gorlin R: Digitalis and beta-blocking agents: effects on depression following myocardial infarction. Am Heart J 1991; 121:1397–1402
20. van Melle JP, Verbeek DE, van den Berg MP, Ormel J, van der Linde MR, de Jonge P: Beta-blockers and depression after myocardial infarction: a multicenter prospective study. J Am Coll Cardiol 2006; 48:2209–2214
21. Ko DT, Hebert PR, Coffey CS, Sedrakyan A, Curtis JP, Krumholz HM: Beta-blocker therapy and symptoms of depression, fatigue, and sexual dysfunction. JAMA 2002; 288:351–357
22. McCall WV, Zvara D, Brooker R, Arias L: Effect of esmolol pretreatment on EEG seizure morphology in RUL ECT. Convuls Ther 1997; 13:175–180
23. McCully RB, Karon BL, Rummans TA, Black JL, Andreen KM, Oh JK, Seward JB, Tajik AJ: Frequency of left ventricular dysfunction after electroconvulsive therapy. Am J Cardiol 2003; 91: 1147–1150
24. Zielinski RJ, Roose SP, Devanand DP, Woodring S, Sackeim HA: Cardiovascular complications of ECT in depressed patients with cardiac disease. Am J Psychiatry 1993; 150:904–909
25. Hasler G, Fromm S, Carlson PJ, Luckenbaugh DA, Waldeck T, Geraci M, Roiser JP, Neumeister A, Meyers N, Charney DS, Drevets WC: Neural response to catecholamine depletion in unmedicated subjects with major depressive disorder in remission and healthy subjects. Arch Gen Psychiatry 2008; 65:521–531
26. Saber AJ, Cain DP: Combined beta-adrenergic and cholinergic antagonism produces behavioral and cognitive impairments in the water maze: implications for Alzheimer disease and pharmacotherapy with beta-adrenergic antagonists. Neuropsychopharmacology 2003; 28:1247–1256
27. Vishne T, Aronov S, Amiaz R, Etchin A, Grunhaus L: Remifentanil supplementation of propofol during electroconvulsive therapy: effect on seizure duration and cardiovascular stability. J ECT 2005; 21:235–238
28. Prudic J, Fitzsimons L, Nobler MS, Sackeim HA: Naloxone in the prevention of the adverse cognitive effects of ECT: a within-subject, placebo controlled study. Neuropsychopharmacology 1999; 21:285–293