CASE REPORT

Takotsubo cardiomyopathy following electroconvulsive therapy: an increasingly recognised phenomenon

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SUMMARY

Treatment of patients with severe depressive illnesses requiring electroconvulsive therapy (ECT) is challenging. This is compounded by the presence of physical comorbidities and potential complications. We report the case of a patient, on long-term bisoprolol, who developed acute epigastric pain and dyspnoea shortly after receiving ECT for treatment-refractory depression. An ECG showed new-onset ischaemic changes and a troponin-I level was elevated at 12 h. A diagnosis of Takotsubo cardiomyopathy was reached following angiography, which demonstrated left ventricular hypokinesia in the absence of coronary artery disease. With supportive treatment the patient made a good recovery. This report highlights the risk of developing Takotsubo cardiomyopathy following ECT despite β-adrenergic receptor blockade, and adds to a growing number of cases reporting this complication. Clinicians involved in the care of patients undergoing ECT must be aware of this complication and should consider Takotsubo cardiomyopathy in patients who develop atypical chest pain after ECT.

BACKGROUND

Takotsubo cardiomyopathy, also known as broken heart syndrome, apical ballooning syndrome and stress cardiomyopathy, is a condition characterised by transient systolic dysfunction of the mid-segments and apical-segments of the left ventricle.[1] Clinically, it mimics an acute coronary syndrome, but lacks the underlying obstructive coronary artery disease that defines the latter condition.

Takotsubo cardiomyopathy is an increasingly reported condition that is often precipitated by acute, severe physical or emotional stressors, such as major operations or bereavements. In this report, we highlight ECT as an iatrogenic trigger of Takotsubo cardiomyopathy.

CASE PRESENTATION

A 74-year-old Caucasian woman, with no history of ischaemic heart disease, was admitted to our old-age psychiatric unit with severe depression. She had a diagnosis of recurrent major depressive disorder and had deteriorated despite high-dose antidepressant medications (venlafaxine and mirtazapine) in combination with lithium and aripiprazole, an antipsychotic medication. Five years prior to this presentation, she had required 3 months of hospitalisation and eight sessions of ECT for a severe depressive episode. No complications resulted from this course of ECT. Her comorbid physical health conditions were chronic obstructive pulmonary disease and hypertension. For the latter, she was taking the oral β-adrenergic receptor blocking agent, bisoprolol, 2.5 mg once daily and lisinopril, an oral ACE inhibitor, 20 mg once daily. She had stopped smoking 3 years previously, and denied use of alcohol or illicit drugs. She had a strong family history of depression but otherwise had no significant family history.

As recommended by the National Institute for Health and Care Excellence guidelines for refractory depression, the patient was consented for a further course of ECT.[2] Her pre-ECT physical health examination, bloods and ECG were unremarkable, with the latter showing sinus rhythm in the absence of ischaemic changes. The patient remained on her oral bisoprolol prior to ECT and did not receive additional intravenous β-blockers. A standard general anaesthetic protocol was employed, during which she was premedicated with propofol and succinyl choline, before receiving unilateral ECT.

Shortly after the first session of ECT, while in the recovery suite, the patient developed acute epigastric discomfort, without radiation and dyspnoea. A repeat physical examination was unchanged and her vital signs were as follows: blood pressure 120/70 mm Hg; pulse rate 84/min; temperature 35.6°C; oxygen saturations 98% (breathing room air); respiratory rate 18/min.

INVESTIGATIONS

A repeat ECG showed new-onset ST-segment depression and T-wave inversion in leads V5 and V6. A troponin-I level, taken 12 h after the onset of symptoms, was elevated at 2847 ng/L (normal range <40 ng/L). Echocardiography demonstrated mild, mixed systolic and diastolic dysfunction of the left ventricle, with an ejection fraction of 52% (normal range 55–70%). Right ventricular function was preserved and no valvular abnormalities were seen.

A coronary angiogram was performed to further investigate a presumed acute coronary syndrome. This revealed no stenoses or occlusions of the coronary arteries. An angiogram of the left ventricle did, however, show mid-segment and apical hypokinesia in addition to apical ballooning, with preserved function in the basal segment.

DIFFERENTIAL DIAGNOSIS

In a patient with acute-onset chest or epigastric pain, ECG changes and a troponin rise, an acute coronary syndrome should be strongly suspected. Atherosclerotic coronary artery disease is the most frequent underlying cause of an acute coronary syndrome, with other less common causes including

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coronary vasospam, coronary emboli and vasculitis of the coronary arteries. Other differential diagnoses to consider include aortic dissection, acute pericarditis, myocarditis and pulmonary embolism. The combined clinical, biochemical and radiological findings in this case, however, favoured a diagnosis of Takotsubo cardiomyopathy.

TREATMENT

While awaiting her angiogram, the patient was started on aspirin and clopidogrel for a presumed acute coronary syndrome and transferred to a cardiology ward for close monitoring. She remained stable and free from complication throughout this short admission. Her long-term oral bisoprolol and lisinopril were continued at the same preadmission doses and no further interventions or medications were required.

OUTCOME AND FOLLOW-UP

The patient was transferred back to the psychiatric unit. Her mental state improved significantly without further ECT and she was subsequently discharged back to the community with outpatient cardiology follow-up. A repeat left ventricular angiogram, performed after discharge, demonstrated resolution of the apical dilation and hypokinesia.

DISCUSSION

This case report highlights ECT as an iatrogenic trigger of Takotsubo cardiomyopathy. This is an increasingly recognised complication and clinicians performing ECT must be vigilant for signs and symptoms suggestive of an acute coronary syndrome or cardiac failure, including atypical presentations.

Takotsubo cardiomyopathy is thought to represent around 1–2% of all acute coronary syndromes resulting in hospitalisation and frequently occurs in post-menopausal women.[3–5] It is characterised by reversible dysfunction of the mid-segments and apical-segments of the left ventricle, in the absence of angiographically-significant coronary artery disease.[1 6] The pathophysiology of this condition is incompletely understood. It is thought that acute, severe physical or emotional stressors, for example, major operations or bereavements, result in a surge of catecholamines which, in turn, induce coronary artery spasm, microvascular dysfunction and/or direct myocyte damage.[3 4 7 8] In the case of ECT, it has been hypothesised that the induction of seizure activity (and/or the postictal state) precipitates acute catecholamine release, autonomic dysfunction and coronary artery spasm, culminating in Takotsubo cardiomyopathy.[3 4 9–11]

Takotsubo cardiomyopathy as a complication of ECT is an increasingly recognised and reported phenomenon.[3–5 9 12–14] A literature review published in 2011 highlighted seven previously documented cases of Takotsubo cardiomyopathy following ECT, in addition to four cases of myocardial stunning and one case of cardiogenic shock post-ECT.[4] Pre-existing conditions that lead to a high baseline catecholamine level, such as chronic anxiety or depression, may predispose patients to developing Takotsubo cardiomyopathy.[15] It follows that patients selected as candidates for ECT may already be a susceptible population, with the acute iatrogenic stress of ECT acting as the trigger for Takotsubo cardiomyopathy.

Although a reversible condition, Takotsubo cardiomyopathy can be complicated by cardiogenic shock, left ventricular outflow obstruction and embolic strokes secondary to apical thrombi.[7 11 16] The majority of patients recover without complication, however, and the prognosis is favourable relative to that of a myocardial infarction secondary to coronary artery disease, with an estimated inpatient mortality of 1.1%.[3 5] The management of Takotsubo cardiomyopathy is largely supportive. Until a diagnosis of Takotsubo cardiomyopathy has been made, and a myocardial infarction secondary to atherosclerotic plaque rupture excluded, treatment for an acute coronary syndrome should be considered unless otherwise contraindicated; with antiplatelet agents, nitrates, β-adrenergic receptor blockade and serial ECG monitoring playing pivotal roles.[4] Once Takotsubo cardiomyopathy has been diagnosed, continuation of β-adrenergic receptor blockade therapy should be considered, given the adrenergic surge that is thought to play a role in its pathogenesis.[1 4 9] Close observation for complications such as cardiac failure and apical thrombi is required, with prompt treatment as they arise.[1]

The safety of reintroducing ECT after an episode of Takotsubo cardiomyopathy is unclear, and involves weighing the benefits of continuation of ECT in the context of severe psychiatric illness against the potential for a further cardiac insult. Successful reintroduction of ECT in patients with previously documented Takotsubo cardiomyopathy has been reported in a number of cases,[4 11 13] with the suggestion that β-adrenergic receptor blockade may help to prevent recurrence by curtailing the adrenergic surge associated with ECT.[1 4 11 13] Importantly, however, the patient in this case report developed Takotsubo cardiomyopathy despite being on long-term oral bisoprolol (a cardioselective β-blocker). Clinicians performing ECT must therefore remain vigilant for Takotsubo cardiomyopathy in patients already on β-adrenergic receptor blockers.

In conclusion, this report highlights Takotsubo cardiomyopathy as an increasingly recognised complication of ECT—a complication that can arise despite concurrent β-blockade. Clinicians involved in ECT must be aware of this, particularly in view of the proposed susceptibility of this patient group to developing Takotsubo cardiomyopathy. Further research is needed to investigate the incidence and pathophysiology of this complication, and to help guide the prevention and treatment of Takotsubo cardiomyopathy in patients receiving ECT.

Learning points

- Takotsubo cardiomyopathy is an increasingly reported and potentially serious complication of electroconvulsive therapy (ECT).
- Post-menopausal women with chronic depression and anxiety appear to be particularly susceptible to the development of Takotsubo cardiomyopathy after ECT.
- The decision to reintroduce ECT after an episode of Takotsubo cardiomyopathy must weigh the benefits of further ECT against the potential for a further cardiac insult.
- Evidence exists to suggest that β-adrenergic receptor blockade may help in the primary and secondary prevention of post-ECT Takotsubo cardiomyopathy, although, as in this case report, the potential to develop this complication remains in patients on such treatments.

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