CASE REPORT

Takotsubo cardiomyopathy and transient global amnesia: a shared aetiology

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SUMMARY

Takotsubo cardiomyopathy (TTC) is associated with acute, reversible left ventricular (LV) dysfunction, while transient global amnesia (TGA) is a reversible disorder of the brain characterised by anterograde amnesia. We report an unusual case of TTC occurring concurrently in a patient with TGA, and propose that catecholamine surge induced cerebral venous congestion and cardiotoxicity is the shared aetiology that leads to the concurrent manifestation of these conditions. TTC and TGA are reversible disorders that can occur concurrently in a subset of patients due to a unifying aetiology, catecholamine excess, leading to pathophysiological changes within the brain and the myocardium.

BACKGROUND

Transient global amnesia (TGA) and Takotsubo cardiomyopathy (TTC) share common features including onset in the sixth decade, a reversible natural history and similar precipitants. Due to similar pathophysiological mechanisms, concurrent TGA and TTC may occur in a subset of patients as a result of a catecholaminergic surge resulting in cerebral venous congestion and cardiotoxicity. We report the unusual case of a patient with simultaneous TGA and TTC.

CASE PRESENTATION

A 60-year-old woman presented to the emergency department with acute memory loss and sudden onset chest pain. The patient's partner described her waking from sleep with central chest heaviness and confusion, but did not note any focal muscle twitching, incontinence or tongue biting. She asked repetitive questions regarding the day's events and could not recollect her overseas holiday 2 weeks prior. She was disoriented to time and date with anterograde amnesia. The day had particular significance to the patient, as it was the anniversary of the death of a loved one.

On examination, vital signs including temperature were within normal limits and cardiovascular examination was unremarkable. Anterograde amnesia persisted with preserved immediate recall with no other focal neurological signs. She had a history of hypertension, irritable bowel syndrome and fibromyalgia.

INVESTIGATIONS

Routine haematological and biochemical investigations were normal. However, high

sensitivity troponin T (hsTnT) was elevated at 82 ng/L (normal <14 ng/L). Initial ECG showed sinus rhythm with no abnormalities. Chest radiography, cerebral CT and MRI were normal. An electroencephalogram was not performed. After a complete neurological review, a diagnosis of TGA was made.

A repeat ECG performed 12 hours later for recurrence of chest pain showed diffuse T wave inversions (figure 1). Coronary angiography revealed normal coronary arteries, left ventriculography and transthoracic echocardiogram revealed apical ballooning with basal sparing and moderate left ventricular (LV) dysfunction with an ejection fraction of 30% consistent with TTC (figure 2,3). Investigations for other causes of high catecholamine release such as pheochromocytoma were not performed as the history was suggestive of TGA and the stress from the death of a loved one was the likely precipitating cause of her cardiomyopathy.

OUTCOME AND FOLLOW-UP

The patient's memory impairment resolved over 16 hours and she was discharged 3 days later. A repeat echocardiogram 1 month following discharge showed normalisation of LV function with no regional wall motion abnormalities. A residual memory gap of approximately 10 hours remained.

DISCUSSION

TTC is characterised by transient regional LV dysfunction that mimics acute myocardial infarction in the absence of obstructive coronary artery disease¹. Despite marked ventricular dysfunction in the acute phase, the vast majority of patients make a full recovery with normalisation of LV function.^{1,2} Although the pathogenesis of TTC is still unclear, catecholamine excess is thought to play a significant role.³ Numerous neurological conditions such as stroke, epilepsy and traumatic brain injury have previously been associated with TTC, highlighting the spectrum of conditions that constitute the so called 'brain heart disorders'.⁴

TGA is a reversible disorder characterised by anterograde amnesia as a result of disrupted formation and retrieval of new episodic memories due to dysfunction of the mesial temporal lobes and the diencephalon.⁵ This manifests as repetitive questioning often referred to as the 'broken record' phenomenon and typically lasts less than 24 hours. Postulated aetiologies for TGA include arterial ischaemia, epileptic phenomena and cerebral venous

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Figure 1 ECG on presentation showed widespread T wave inversions: a typical pattern observed in Takotsubo cardiomyopathy.

insufficiency.^{6,7} Antecedent emotional stress and somatic triggers that promote sympathetic activity, including strenuous physical exertion, Valsalva manoeuvre and sexual intercourse have been known to trigger TGA.^{5,7} Increase in sympathetic tone leads to an elevated central venous return with a resultant rise in cerebral venous pressure. The subsequent venous congestion of the hippocampi and diencephalon leads to transient disruption in memory formation and retrieval which corresponds to changes on diffusion-weighted MRI.⁸ This signifies the importance of cerebral venous congestion in the pathophysiology of TGA.

Both TGA and TTC share common features including onset in the sixth decade, a reversible natural history and with psychosomatic and physiological stress the likely precipitant in a significant proportion of the cases.^{1,8} In our patient, both conditions developed concurrently; this has been reported two times in literature.^{9,10} Similarly, a case of cardiac enzyme elevation without contractile dysfunction in the context of TGA has been reported.¹¹ Although a transient decrease in ejection fraction during an event may explain acute confusion, it is unlikely that a prolonged decrease in cerebral perfusion would persist without MRI changes.

The presence of shared pathophysiological and epidemiological features indicates that both entities may manifest due to a common precipitant—sympathetic overactivity. We propose that concurrent TGA and TTC occur in a subset of patients as a result of a catecholaminergic surge resulting in cerebral venous congestion and cardiotoxicity. This results in a spectrum of cardiac

Figure 2 Left ventriculogram showing apical hypokinesis with basal sparing.

Figure 3 Transthoracic echocardiogram showing apical and mid segment left ventricular hypokinesis with basal sparing.

presentations that range from isolated troponin elevation to overt LV dysfunction typical of TTC. Despite the complexity of the presentation, these patients appear to make a complete recovery both from a cardiac and neurological perspective.

Learning points

- ▶ Both transient global amnesia (TGA) and Takotsubo cardiomyopathy (TTC) share common features including onset in the sixth decade, a reversible natural history, and similar precipitants.
- ▶ Concurrent TGA and TTC may occur in a subset of patients as a result of a catecholaminergic surge resulting in cerebral venous congestion and cardiotoxicity.
- ▶ Patients with TGA should undergo serial ECGs and measurement of cardiac enzymes to monitor for the development of TTC and to ensure appropriate therapy is commenced.

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REFERENCES

- Templin C, Ghadri JR, Diekmann J, et al. Clinical features and outcomes of Takotsubo (Stress) Cardiomyopathy. *N Engl J Med* 2015;373:929–38.
- Gianni M, Dentali F, Grandi AM, et al. Apical ballooning syndrome or takotsubo cardiomyopathy: a systematic review. *Eur Heart J* 2006;27:1523–9.
- Ono R, Falcão LM. Takotsubo cardiomyopathy systematic review: pathophysiologic process, clinical presentation and diagnostic approach to Takotsubo cardiomyopathy. *Int J Cardiol* 2016;209:196–205.
- Finsterer J, Wahbi K. CNS disease triggering Takotsubo stress cardiomyopathy. *Int J Cardiol* 2014;177:322–9.
- Pantoni L, Bertini E, Lamassa M, et al. Clinical features, risk factors, and prognosis in transient global amnesia: a follow-up study. *Eur J Neurol* 2005;12:350–6.
- Wittstein IS, Thiemann DR, Lima JA, et al. Neurohumoral features of myocardial stunning due to sudden emotional stress. *N Engl J Med* 2005;352:539–48.
- Sander K, Sander D. New insights into transient global amnesia: recent imaging and clinical findings. *Lancet Neurol* 2005;4:437–44.
- Lewis SL. Aetiology of transient global amnesia. *Lancet* 1998;352:397–9.
- Quick S, Speiser U, Richter N, et al. Transient global amnesia and broken heart syndrome: two faces of one pathology. *Clin Auton Res* 2015;25:189–91.
- Bobinger T, Köhrmann M, Raaz-Schrauder D, et al. Lost memories can break your heart: a case report of transient global amnesia followed by takotsubo cardiomyopathy. *Clin Res Cardiol* 2013;102:693–6.
- Jalanko M, Forsström F, Lassus J. Cardiac troponin T elevation associated with transient global amnesia: another differential diagnosis of 'troponosis'. *Eur Heart J Acute Cardiovasc Care* 2015;4:561–4.

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