

## Takotsubo cardiomyopathy following ischemic stroke: a cause of troponin elevation

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Dear Sirs,

Elevation of cardiac troponin was reported to occur in up to 34% of patients with acute ischemic stroke [1, 2]. Troponins are valuable cardiac biomarkers as they reflect myocardial injury with high sensitivity. Preceding acute myocardial infarction (AMI), renal failure or stroke-induced myocytolysis are regarded to be possible causes of troponin elevation in stroke patients [1, 3]. However, the cause of myocardial injury in the individual stroke patient is unclear leading to diagnostic and therapeutic uncertainty.

An 82-year-old woman presented to our emergency department with sudden onset of expressive aphasia and right-sided central facial paresis. MRI revealed occlusion of the M2 segment of the left middle cerebral artery with a small area of restricted diffusion including the insular cortex (Fig. 1a, b). Laboratory measures showed an elevated troponin (0.085 µg/l) and normal retention parameters (creatinine = 0.83 mg/dl). Electrocardiogram (ECG) showed atrial fibrillation without signs of AMI. The heart rate ranged between 93 and 160 per min. The patient did not report dyspnea, chest pain or palpitations and no

clinical signs of heart failure were present at that time. During the first night on the stroke unit the woman complained about moderate chest pain and dyspnea. Troponin further increased to 0.100 µg/l. ECG still showed no signs of AMI. Chest X-ray demonstrated pleural effusion. Symptoms resolved within a few hours after diuretic therapy. Coronary angiography (CAG) was performed and showed normal flow without signs of significant arteriosclerosis (Fig. 1c). The left ventriculography showed the typical pattern of left ventricular apical ballooning and a mildly decreased left ventricular function with an ejection fraction of 51% (Fig. 1d). The diagnosis of Takotsubo cardiomyopathy (TTC) was established according to proposed Mayo criteria [4]. The systolic dysfunction was completely resolved within 14 days in echocardiographic follow up showing a normalized left ventricular ejection fraction of 72%.

TTC is a reversible type of stress cardiomyopathy, which is characterized by symptoms mimicking AMI combined with troponin elevation or ECG alterations. In contrast to AMI, signs of obstructive coronary artery disease are missing in the coronary angiogram [4].

In the largest case series by Yoshimura et al. TTC occurred in 1.2% of ischemic stroke patients [5]. Troponin elevation is observed in most of the patients with TTC and is usually modest compared to AMI [4]. In our patient, alternative causes of troponin release like AMI or renal insufficiency were ruled out via CAG and laboratory measures. For many years, atrial fibrillation has been recognized as a cause of global decrease in left ventricular function [6]. However, the typical pattern of wall motion abnormalities sparing the basal segments is usually not seen in tachycardia-induced cardiomyopathy and on the other hand, rhythm disturbances are reported in 27% of TTC cases [7]. Therefore, atrial fibrillation might have

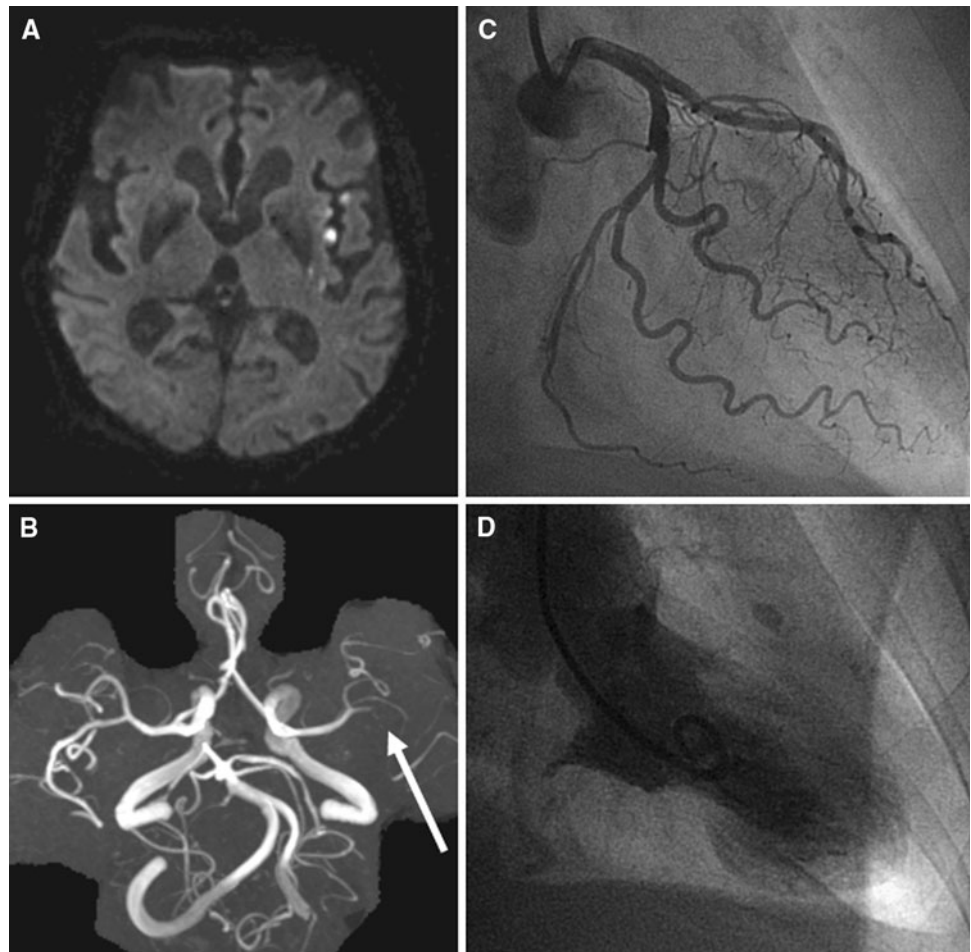
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**Fig. 1** Diagnostic findings of the presented case: **a** Diffusion-weighted MRI on admission confirming involvement of the left insular cortex. **b** Arterial occlusion in the M2 segment of the middle cerebral artery (*white arrow*). **c** Angiography of the left coronary artery showing normal flow and no culprit lesion. **d** Left ventriculography demonstrating left ventricular apical ballooning during systole



added to the clinical presentation of mild acute heart failure and troponin release in our patient, but does not commonly cause the ventriculographic finding of left ventricular apical ballooning which is a typical feature of TTC.

The etiology of TTC is controversial. A catecholamine-mediated development of TTC was proposed, because most cases described stressful events as a trigger. Moreover, elevated catecholamine levels and morphological changes in cardiomyocytes suggestive of toxic catecholamine overload were reported in patients with TTC [8]. The majority of the patients documented by Yoshimura et al. had stroke in the insula or close to it [5]. This is consistent with our case and might underline the pathophysiological concept of a catecholamine-induced origin of TTC, because the insular cortex is regarded to contribute to cortical control of autonomic cardiac function [9, 10].

TTC was also described as a cardioembolic cause of stroke via left ventricular thrombus formation [11, 12]. Therefore, stroke may also be the effect of TTC rather than the cause and it might be difficult in some cases to establish a definite cause-effect relationship. In our patient, the time-course with chest pain following stroke argues in favor of

insular stroke as a trigger of TTC, and rather, atrial fibrillation can be assumed the cardioembolic source of stroke.

In conclusion, the reported case indicates that (a) troponin elevation in ischemic stroke may originate from TTC, (b) TTC may be a complication of stroke affecting the insular cortex and (c) findings of increased troponin in strokes with insular involvement may hint at evolving TTC and warrant cardiac monitoring and further investigation with echocardiography or CAG, if feasible.

**Conflict of interest** None.

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