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Takotsubo Syndrome and Cerebral Cardioembolism: Case report to redefine the short-term prognosis

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Takotsubo cardiomyopathy is characterized by transient hypokinesis of the left ventricular apex or midventricular segments, without significant stenosis affecting coronary arteries[1]. This condition is often associated to emotional[2] or physical stress[3]. Although the underlying pathophysiology remains unclear[4], alterations in erythrocyte membranes and endothelial integrity could determine micro-vascular hypo-perfusion, favouring the occurrence of left ventricular ballooning[5].

Recent findings showed that this cardiomyopathy is well known to be related to cerebral infarction[6], although scarce data describe the real timing of this adverse event.

We report the case of a woman experienced Takotsubo cardiomyopathy, and developed cardiogenic cerebral embolism on the fourth day from the onset of symptoms.

A 69-year-old woman was admitted to the Internal Medicine Department of our Hospital, after clinical stabilization of acute hearth failure occurring the day before. At the onset of symptoms, she complained of chest pain; electrocardiography showed a decreased ST segment in all leads and X-ray evidenced pulmonary vascular congestion, compatible with acute pulmonary oedema. Echocardiography showed a global reduction in kinetic of the left ventricular and the apex, associated to the ballooning morphology typical of Takotsubo syndrome; no other pathological findings were found. Ventricular coronary arteriography did not show significant stenosis on epicardial coronary arteries. Markers of myocardial necrosis were slightly elevated.

Therefore, at the admission to our Unit, she was diagnosed with Takotsubo cardiomyopathy and treated with antithrombotic prophylaxis, by LMWH and antiplatelets, and drugs to sustain heart function, as recommended from guidelines.

On her third hospital day, instrumental examinations showed that cardiac function was slowly, but significantly, recovering. The day after she became less responsive with neurological signs, compatible with cerebral stroke. We observed left hemiplegia, left sided neglect, oral and ocular deviation. The Glasgow Coma Scale was 10 (E4V1M5) and The National Institutes of Health Stroke Scale score was 16. In the acute phase, electrocardiography showed ST decrement in absence of arrhythmias; at echocardiography, hypokinesia was nearly resolved.

We performed a Computed Tomography imaging, diagnosing a cerebral stroke, sided in cortico- subcortical area of right insular lobe (fig 1A); in particular, we showed a hyperdensity in

Figure 1:
Computed Tomography imaging. Cerebral stroke sided in cortico-subcortical area of right insular lobe (fig 1A); hyperdensity in the transition between M1 and M2 of the right middle cerebral artery (fig 1B).

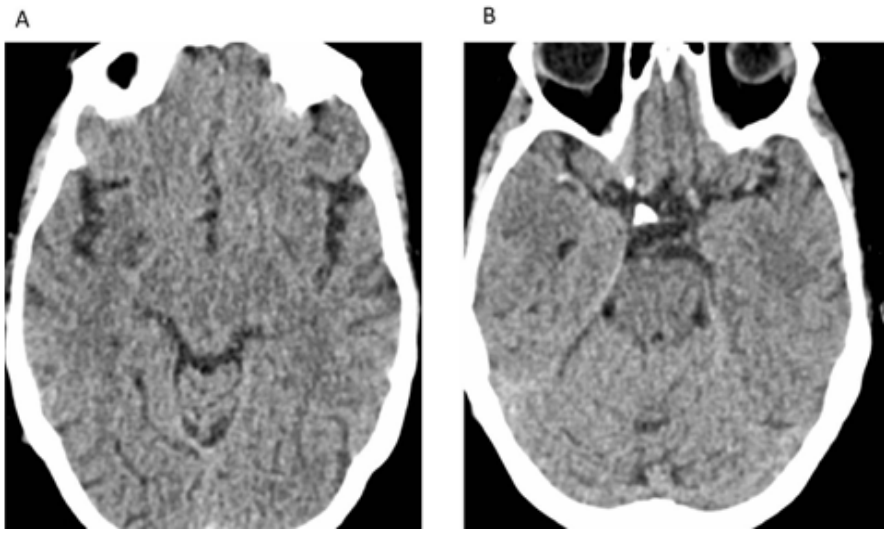

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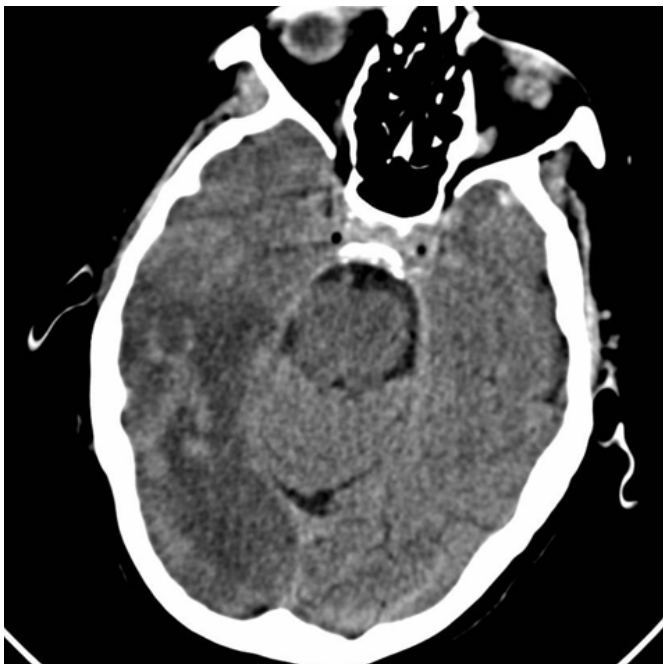
Figure 2:
Computed Tomography imaging. At 48-hour CT control the cerebral lesion became haemorrhagic. Haemorrhagic lesion sided in the cortical-subcortical area of right insular, temporal, occipital lobes.



the transition between M1 and M2 of the right middle cerebral artery(fig 1B).

The same day, in order to investigate a cardio-embolic source, we performed a trans-oesophageal echocardiography, showing a 3-cm mobile thrombus.

At the 48-hour CT control, we appreciated that the cerebral lesion became haemorrhagic (fig 2); thus, we stopped the anticoagulation therapy.

Patient survived the cerebral accident and, gradually, neurological findings attenuated and she showed a complete recovery of her motility. At the control trans-oesophageal echocardiography, we appreciated the ventricular thrombus reduction; at the CT control, the lesion was regressed, and the patient was eligible to life-long treatment by oral anticoagulants. After discharge from our Unit, she started a rehabilitation program, in order to consolidate her motility.

Takotsubo cardiomyopathy had a favourable prognosis related to the complete recovery of cardiac function after the acute onset[1]; however, several reports described the occurrence of cardio-embolic cerebral embolism[6-9], an event that could dramatically change the clinical perspective.

At the Hospital admission, the patient did not show any condition promoting ventricular thrombosis, with the exception of the global hypokinesia related to Takotsubo syndrome. Accordingly to literature[10], our clinical findings suggested that the initial recovery of contractile function, occurred on the third day from the admission, could be the critical factor involved in to cerebral embolization, despite we provided antithrombotic prophylaxis, accordingly to current guidelines.

These data contribute to the hypothesis that Takotsubo patients could be effectively at high risk for stroke; therefore, in order to prevent cerebral cardio-embolic accidents, our clinical management should pay attention to 1) perform imaging assessments to rule out ventricular thrombosis; 2) consider patients eligible to anticoagulant therapy from the onset of symptoms; 3) revise Takotsubo prognosis because its complications.

Statement of ethical publishing

The authors agree to abide by the requirements of the "Statement of publishing ethics of the International Cardiovascular Forum Journal"[11].

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The authors have nothing to declare.

Conflict of interest:

The authors declare that there is no conflict of interest.

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