

# Intracranial bleeding mimicking an extensive acute myocardial infarction with reversible apical ballooning and systolic left ventricular dysfunction. A case report

## *Infarto miocardico acuto esteso con asinergia apicale e disfunzione ventricolare sinistra reversibile associata a sanguinamento intracranico. Un caso clinico*

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**ABSTRACT:** *Intracranial bleeding mimicking an extensive acute myocardial infarction with reversible apical ballooning and systolic left ventricular dysfunction. A case report. A. D'Aloia, E. Vizzardi, P. Faggiano, C. Fiorina, L. Dei Cas.*

A novel syndrome with transient asynergy of the apical segments of the left ventricle, also known as tako-tsubo cardiomyopathy, has been recently described and presents characteristics and effects similar to acute myocardial infarction. We report the case of a 69-year-old woman presenting with chest pain typical of myocardial ischemia, electrocardiographic abnormalities typical of antero-lateral transmural myocardial infarction, and unstable clinical and hemodynamic condition. Trans-thoracic echocardiography showed a left ventricular dysfunction with a markedly decreased ejection fraction.

The patient received heparin and abciximab bolus but coronary angiography demonstrated the absence of coronary stenoses, thrombi, and coronary spasm. Due to impaired neurological conditions the patient underwent brain tomography that showed subarachnoid haemorrhage secondary to a right vertebral artery aneurysm rupture. The possible occurrence of a reversible cardiomyopathy mimicking an acute coronary syndrome in presence of intracranial bleeding should be always considered.

*Keywords: tako-tsubo cardiomyopathy, acute myocardial infarction, intracranial bleeding, subarachnoid haemorrhage, aneurysm rupture.*

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### Introduction

A novel syndrome with transient asynergy of the apical segments of the left ventricle, also known as "tako-tsubo" cardiomyopathy, has been recently described both in Japanese and Caucasian patients [1-3]. This case reports on a woman admitted with chest pain and ECG modifications suggestive of acute myocardial infarction (diagnosis of acute coronary syndrome), treated by the glycoprotein antagonist abciximab and heparin but that finally fulfilled the diagnostic criteria of transient apical ballooning with normal coronary arteries associated to the presence of a cerebrovascular accident.

The clinical implications of this case are important to avoid possible misdiagnosis and harmful therapeutic treatments.

### Case report

A 69-year-old woman was admitted to the emergency department of our Hospital because of general discomfort started at home with chest pain, di-

aphoresis, without a previous significant emotional stress. She had only a history of hypertension not treated in the last year.

At the admission the patient was awake collaborative, complaining general malaise and dyspnoea; pupils were isocyclic and isochoric, and no focal evidences of neurological deficit was observed.

Blood pressure was 170/80 mmHg, heart rate was 100 beats /min; temperature was 37 °C, skin was cold and wet. On chest auscultation there was an S3 and S4 double gallop and widespread crackles in both lungs. Arterial gas analysis showed hypoxemia (pO<sub>2</sub>: 64 mmHg, sat O<sub>2</sub>: 92%), hypocapnia (pCO<sub>2</sub>: 35 mmHg), pH: 7.4; HCO<sub>3</sub>: 22 mmol/l.

Soon she developed acute pulmonary oedema requiring oro-tracheal intubation because of unstable clinical and hemodynamic condition.

Cardiac enzymes at the admission (after 1 hour from the starting symptoms) were negative: troponin I: 0.02 (normal 0.04 ng/mL) - creatinine kinase levels and MB fraction was normal. Electrocardiogram revealed normal sinus rhythm with ST-segment elevation in leads V2 through V6, D1, aVL, QT intervals was 392 ms (fig. 1A).

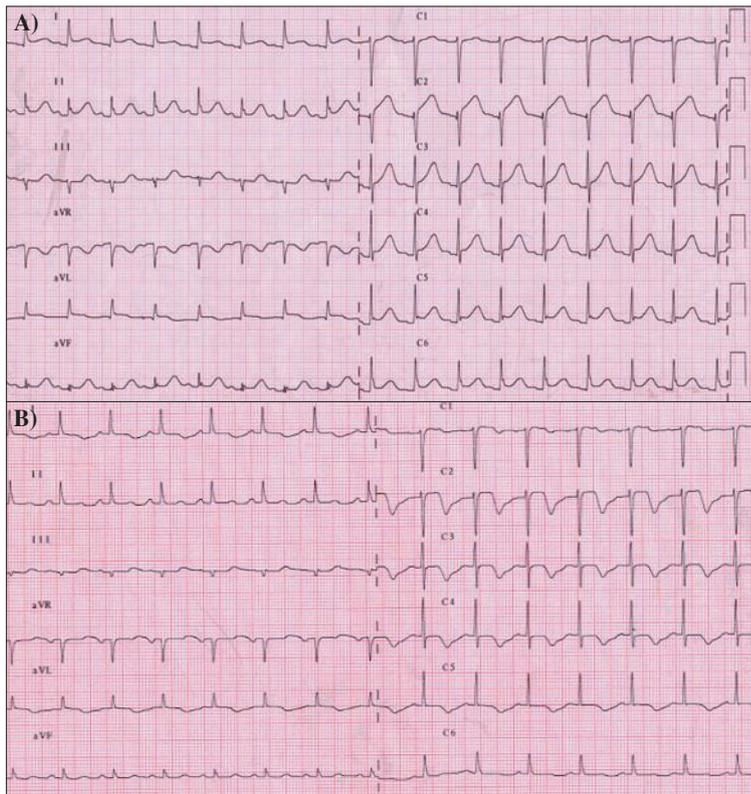


Figure 1.

Transthoracic echocardiography showed a mild ventricular hypertrophy with a moderate to severe left ventricular systolic dysfunction (LVEF 35%) due to a marked dyskinesia of mid-distal septal antero-lateral and infero-posterior wall/segments. Left ventricular filling pattern on transmitral and pulmonary venous flow was restrictive ( $E/A > 1.6$ ; deceleration time 150 msec  $S/D < 1$ ), indicating high left atrial pressure. No intracardiac thrombus was observed, no valvular abnormalities. Right ventricle was normal in size and function (fig. 2).

Based on the clinical presentation and features of acute coronary syndrome (with ST segment elevation) the patient received oxygen, nitrates, aspirin, heparin and gpIIb/IIIa-abciximab bolus and urgent coronary angiography was planned.

Coronary angiography demonstrated the absence of coronary stenoses, thrombi, and coronary

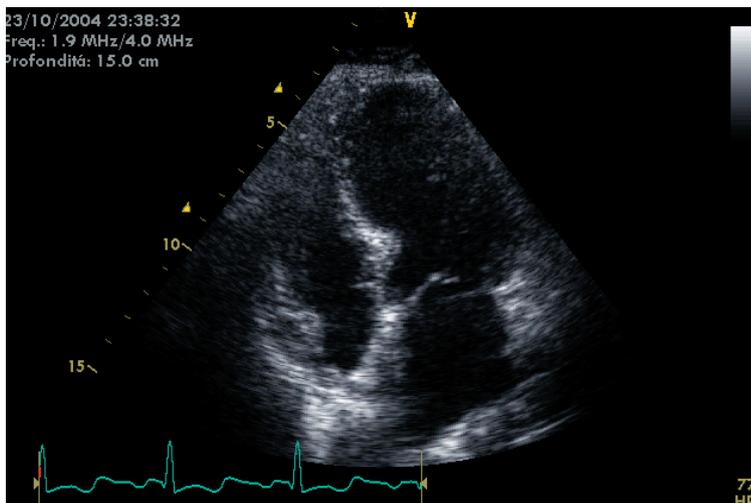


Figure 2.

spasm; in particular, the right artery was dominant and the left anterior descending artery passed beyond the apex.

Cardiac enzymes had a modest rise with troponin I peak was of 5.63 ng/ml and the maximal level of creatine kinase (280 U/L) was reached 12 hours upon admission.

Thereafter, for the presence of unconsciousness, the patient underwent brain tomography that showed bilateral subarachnoid haemorrhage at silvian level and right lateral pons with initial sign of a sovratentorial hydrocephalus (fig. 3A).

The angiography showed a right vertebral aneurism with a diameter of 4 mm at the level of postero-inferior cerebellar artery origin (fig. 3B).

The patient was then treated urgently with haemorrhage drainage and then with clips positioned percutaneously by femoral arterial approach to isolate the aneurism. The clinical course showed a rapid improvement of her status.

A second electrocardiogram taken after 6 hours from the admission resulted completely normal while the echocardiographic abnormalities persisted.

The next day the electrocardiogram showed symmetric negative T waves (inversion) in the precordial and antero-lateral leads, heart rate of 95 beats/min, QT interval of 364 msec ( $QTc = 458$  msec) (fig. 1B).

Electrocardiogram and echocardiogram showed, after three days, complete regression of ST/T segments modifications and LV regional and global systolic function, respectively.

The patient was discharged without neurological deficit in good general conditions on beta blocker and ace-inhibitor.

## Discussion

Takotsubo-like cardiomyopathy is a recently described acute cardiac syndrome characterized by transient systolic dysfunction involving the left ventricular apex, appearing at echocardiography as an apical “ballooning”, associated with symptoms, electrocardiographic abnormalities and myocardial enzymatic release resembling an acute myocardial infarction. However, coronary angiography usually does not demonstrate vascular abnormalities [4].

Dote *et al.* [5] firstly described this syndrome in a Japanese population, but recently it has been observed also in Europe [3] and in the United States [6]. Previous observations have suggested a strong female gender predominance with spanning age from 62 to 75 years.

Typically this syndrome is precipitated by sudden emotional and physical stress such as accidents of family members, relative's death or funeral, epileptic seizure, cerebrovascular accidents, bronchial asth-

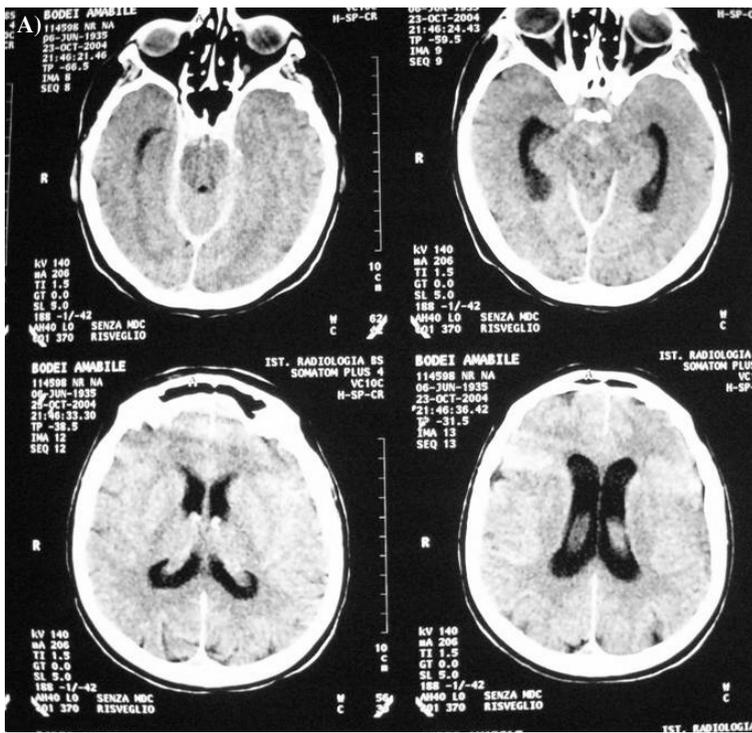


Figure 3A.



Figure 3B.

ma, diabetic coma, acute pancreatitis and various invasive procedure like intubation, cholecystectomy and others [1].

There is no consensus about the mechanisms of the disease: many have hypothesized coronary vasospasm or thrombosis to be the cause [7, 8]. Japanese authors support the theory of myocardial perfusion abnormalities related to fatty acid metabolism impairment [9].

The Mayo Clinic criteria for the clinical diagnosis of transient left ventricular apical ballooning syndrome include the absence of recent significant head trauma, intracranial bleeding, pheochromocytoma, myocarditis, hypertrophic cardiomyopathy [10] but as recently demonstrated by Wittstein *et al.* [11], the pathophysiology of the disease seems to involve stressors with a sympathetic activation; subarachnoid hemorrhage is associated with an enhanced sympathetic stimulation and a stress-induced cardiomyopathy [12-18].

Our paper confirms that cerebrovascular accidents may result in a unique pattern of apical left ventricular dysfunction associated with QT prolongation and deep precordial T wave inversion on electrocardiogram, known as "T cerebral waves".

We hope that the increasing reports of this new syndrome will allow to identify the clinical features useful for differentiate diagnosis from myocardial infarction in order to avoid treatment with anticoagulants and antiplatelets, potentially dangerous particularly in the group of patients with hemorrhagic cerebral accident.

### Riassunto

*Recentemente è stata descritta una nuova sindrome, anche conosciuta con il nome di "cardiomiopatia di tako-tsubo", caratterizzata da asinerzia*

*transitoria dei segmenti apicali del ventricolo sinistro, e che presenta caratteristiche ed effetti simili ad un infarto miocardico acuto. Si riporta un caso di una donna di 69 anni giunta alla nostra osservazione con dolore toracico tipico di ischemica miocardica, anomalie elettrocardiografiche tipiche di un infarto miocardico transmurale antero-laterale, e condizioni cliniche ed emodinamiche instabili. L'elettrocardiogramma transtoracico mostrava disfunzione ventricolare sinistra con una marcata riduzione della frazione di eiezione. Alla paziente veniva somministrato un bolo di heparina e abciximab ma l'angiografia coronarica evidenziava assenza di stenosi coronariche, trombi, e spasmo coronarico. Migliorato il quadro neurologico, la paziente veniva sottoposta ad esame TC del cranio che evidenziava emorragia subaracnoidea secondaria a rottura di un aneurisma dell'arteria vertebrale destra. La possibilità che si verifichi una cardiomiopatia reversibile che mima una sindrome coronarica acuta in presenza di sanguinamento intracranico deve essere sempre tenuta in considerazione.*

*Parole chiave: cardiomiopatia di Tako-tsubo, infarto miocardico acuto, sanguinamento intracranico, emorragia subaracnoidea, rottura di aneurisma.*

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