

## Case Report

## Open Access

# A Rare Double Trigger for Takotsubo Cardiomyopathy

Anna Mengoni, Andrea Cardona, Giuseppe Ambrosio\*

University Hospital Santa Maria della Misericordia, cardiology, Italy

\*Corresponding author: Giuseppe Ambrosio, University Hospital Santa Maria della Misericordia, Cardiology, Piazzale Menghini, Perugia. Italy. Tel: 0755782394. E-mail: giuseppe.ambrosio@ospedale.perugia.it

Rec date: January 26, 2016, Acc date: February 12, 2016, Pub date: February 19, 2016

Copyright: © 2016 Mengoni A. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution and reproduction in any medium, provided the original author and source are credited.

## Abstract

A woman admitted to the hospital for a massive Pulmonary Embolism, developed, at stable emodynamic condition, a complex clinical scenario characterized by Crohn disease exacerbation, chest pain and worsening dyspnea. The electrocardiogram presented non-specific repolarization changes. Echocardiogram showed new Left Ventricle akinesia of the apex and medium-apical segments, hyperkinetic basal segment with reduced ejection fraction. Coronarography showed not significant coronary artery disease and typical left ventricle apical ballooning. Left ventricle dysfunction recovered spontaneously in few days. This is the first case of Takotsubo Cardiomyopathy in course of pulmonary embolism and Crohn disease exacerbation.

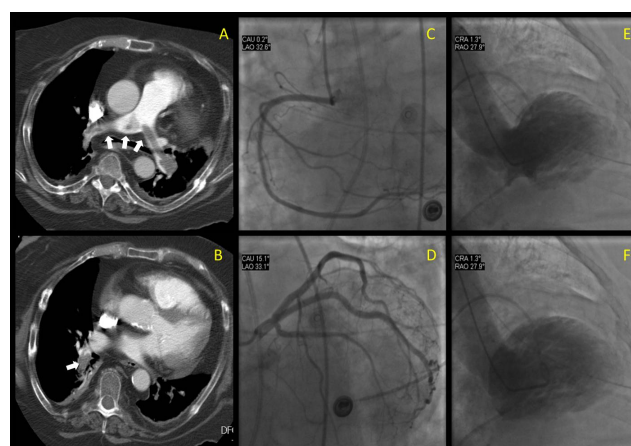
**Summary:** Takotsubo Cardiomyopathy is a syndrome characterized by acute regional systolic dysfunction of the left ventricle, frequently related to psycho-physical acute stress and usually reversible, in the absence of significant coronary artery disease. No cases have been reported of Takotsubo Cardiomyopathy associated with pulmonary embolism in the context of Crohn disease reactivation.

## Case Report

We report the case of a 71 years old lady referred to our hospital for tachycardia, asthenia and worsening dyspnea at rest. Clinical history was relevant for severe obesity, diabetes mellitus and history of poorly controlled Crohn Disease (CD) treated with long-term corticosteroid therapy. A few days before admission, she had decided on her own to reduce corticosteroid therapy. On admission, blood pressure was 90/55 mmHg, heart rate 105 b/min, respiratory rate 26 breaths/min and oxygen saturation 98% (on and O<sub>2</sub>). Emogasanalysis showed respiratory alkalosis; sinus tachycardia and diffuse ST-segment depression were present on electrocardiogram (EKG). The echocardiogram (TTE) showed a D-shaped left ventricle in the short-axis view with preserved global and regional systolic function (Ejection Fraction ((EF)) Simpson 55%). The Right Ventricle (RV) was dilated and hypokinetic with a tricupid annular plane systolic excursion (TAPSE) of 12 mm. Mild systolic pulmonary artery hypertension (38 mmHg) was also present. Laboratory analyses showed elevated D-dimer (1130 ng/mL) and LDH (2016 UI/L) and normal Troponin I concentrations.

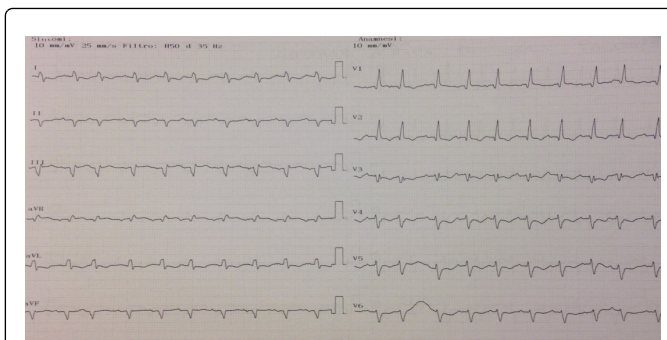
Pulmonary Embolism (PE) was suspected; urgent chest Computer Tomography (CT) showed a massive thrombus involving the main pulmonary artery at the bifurcation (Figure 1, panels A-B).

Therapy with enoxaparin and rTPA was started, leading to recovery. After five days of stable emodynamic conditions, the patient presented a complex clinical scenario characterized by abdominal pain, nausea, vomiting, and diarrhea. Laboratory exams showed neutrophilia, high ESR (58), and CRP (35.8 mg/dl), worsening anemia, low total protein count, and hypocalcemia (6.2 mg/dl). CT of the abdomen showed multiple air-fluid levels in the colon and small bowel and gastric dilation. The suspect of reactivation of underlying CD was confirmed by high levels of faecal calprotectin (918 mg/kg).



**Figure 1:** Panels A-B: computer tomography showed thrombus involving the main pulmonary artery at the bifurcation thrombus. Panel C: Angiogram showing normal right coronary. Panel D: angiogram showed left coronary without significant lesion. Panel E: left ventriculography during systole. Panel F: left ventriculography during diastole (see text for further details).

Just a few hours after the onset of abdominal symptoms, the patient experienced dyspnea and chest pain. The EKG showed diffuse T wave inversion not present at the previous EKG (Figure 2), while the TTE showed akinesia of the apex and mid-segments of LV, hyperkinesia of basal segments and depression of EF (30%). RV showed normal systolic function.



**Figure 2:** Electrocardiogram during chest pain.

For these reasons she was transferred to our Cardiology department. Markers of myocardial necrosis were elevated (peak TnI 1.32 ng/ml). Urgent coronary angiography was performed, showing normal coronary arteries and LV dysfunction with characteristic apical ballooning of the apex and hyperkinesia of basal segments (Figure 1, panels C-F). LV contractile function fully recovered a few days later. The patient was discharged in good clinical conditions, asymptomatic.

This clinical context led to the most likely diagnosis of Takotsubo Cardiomyopathy (TCM) developing in the complex scenario of PE and CD reactivation.

## Discussion

TCM, also known as transient apical ballooning syndrome, is a form of non-ischemic cardiomyopathy that resembles an acute coronary syndrome, but that is typically characterized by the absence of coronary artery obstruction and spontaneous recovery of LV function [1]. Clinical and instrumental presentation is characterized by acute chest pain or dyspnea at rest, extremely wide range of EKG presentations [ST-Segment Elevation, T-wave inversion, or nonspecific alterations], rise of myocardial necrosis markers, and characteristic LV apical akinesia with compensatory hyperkinesia of basal segments [apical ballooning] [2]. Spectrum of hemodynamic involvement ranges from only mild impairment to severe cardiogenic shock, but typically LV function recovers spontaneously in a few hours or days [1,2].

TCM seems to be triggered by emotional or physical stressors, but its pathophysiology remains yet to be completely ascertained [3]. Possible explanations range from coronary vasospasm to microcirculatory dysfunction and to hyper-catecholaminergic status [3]. The latter mechanism in particular seems to play a central role in TCM pathophysiology, albeit it is very likely that just one theory cannot univocally explain the whole clinical scenario of TCM.

This patient had at least two important triggers that may have led to TCM. PE, with its attendant hypoxemia and hyperadrenergic status, may have played an initial role; subsequently, the situation could have been precipitated by reactivation of CD, caused by abrupt reduction of corticosteroid therapy: malabsorption syndrome, in turn, may have induced electrolyte alterations that may have further contributed to impaired myocardial contractility, coronary artery dysfunction, and angina [4]. Endothelial dysfunction that seems involved in the pathogenesis of CD, may be another mechanism for TCM [5-7]. This is the first case of TCM in the setting of PE and acute CD exacerbation.

## References

1. Prasad A, Lerman A, Rihal CS (2008) Apical ballooning Syndrome [Takotsubo or stress cardiomyopathy]: a mimic of acute myocardial infarction. *Am Heart J* 155: 408-417.
2. Bybee KA, Kara T, Prasad A, Lerman A, Barsness GW (2004) Systematic review: transient left ventricular apical ballooning: a syndrome that mimics ST-segment elevation myocardial infarction. *Ann Intern Med* 141: 852-65
3. Pelliccia F, Greco C, Vitale C, Rosano G, Gaudio C, et al. (2014) Takotsubo Syndrome [Stress Cardiomyopathy]: An Intriguing Clinical Condition in Search of its Identity. *Am J Med* 127: 699-704
4. Chmielecki M, Strozyk A, Jaguszewski M, Gruchala M, Rynkiewicz A (2012) Takotsubo cardiomyopathy related to compound electrolyte aberration and anemia in Crohn's disease. *International Journal of Cardiology* 157: e57-9
5. Roifman I, Sun YC, Fedwick PJ, Panaccione R, Buret AG, et al. (2009) Evidence of Endothelial Dysfunction in Patients with Inflammatory Bowel Disease. *Clinical gastroenterology and hepatology* 7: 175-82
6. Gesualdo M, Scicchitano P, Carbonara S, Ricci G, Principi M, et al. (2015) The association between cardiac and gastrointestinal disorders: casual or causal link? *J Cardiovasc Med* 2015 Dec 23
7. Ciccone MM, Principi M, Lerardi E, Di Leo A, Ricci G, et al. (2015) Inflammatory bowel disease, liver diseases and endothelial function: is there a link? *J Cardiovasc [Med Hagerstown]* 16: 11-21