



## **An Unusual Case of Acute Myopericarditis Possibly Due to Food Poisoning**

**Annapaola Zito<sup>1</sup>, Fiorella Devito<sup>1</sup>, Annamaria Dachille<sup>1</sup>, Rosa Carbonara<sup>1</sup>,  
Francesco Giardinelli<sup>1</sup>, Ottavio Di Cillo<sup>2</sup> and Marco Matteo Ciccone<sup>1\*</sup>**

<sup>1</sup>*Cardiovascular Diseases Section, Department of Emergency and Organ Transplantation (DETO),  
University of Bari, Piazza G. Cesare 11, 70124 Bari, Italy.*

<sup>2</sup>*Chest Pain Unit, Cardiology Emergency, University of Bari, Italy.*

### **Authors' contributions**

*This work was carried out in collaboration between all authors. Authors AZ and FD wrote the draft of the manuscript. Authors AD, RC and FG managed the literature searches. Author ODC designed the figures, managed literature searches and contributed to the correction of the draft. Author MMC provided the case, the figures and supervised the work. All authors read and approved the final manuscript.*

### **Article Information**

DOI: 10.9734/IJMPCR/2015/12942

#### Editor(s):

(1) Nurhan Cucer, Medical Biology Dept., Faculty of Medicine, Erciyes University, Turkey.

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Complete Peer review History: <http://www.sciencedomain.org/review-history.php?iid=705&id=38&aid=6586>

**Case Study**

**Received 24<sup>th</sup> July 2014**  
**Accepted 4<sup>th</sup> October 2014**  
**Published 22<sup>nd</sup> October 2014**

### **ABSTRACT**

The manuscript describes the case of a 23 year-old woman, with acute myopericarditis associated with food poisoning without other cardiovascular disease. The patient had a history of hypothyroidism, treated with levothyroxine 75 µg/d, however she was clinically and biochemically euthyroid. We present a discussion of this rare association, followed by clinical presentation and treatment. This case report underlines the importance of considering possible cardiac complications when treating patients with food poisoning.

\*Corresponding author: E-mail: [marcomatteo.ciccone@uniba.it](mailto:marcomatteo.ciccone@uniba.it);

*Keywords: Acute myopericarditis; food poisoning; salmonella; stool culture.*

## 1. CASE PRESENTATION

A 23 year-old woman, with a history of hypothyroidism, in treatment with levothyroxine 75 µg/d (however she was clinically and biochemically euthyroid), came to the first aid of our Hospital because of an onset of heartburn associated with several diarrheal discharges, fever, asthenia and syncope. The day before the patient had eaten raw fish. Due to syncope, head Computed Tomography (CT) examination was performed and it showed some calcifications of the epiphyseal gland. In the emergency room evaluation, there was evidence of orthostatic blood pressure drop, that could explain the syncope.

Because of the rise of cardiac markers (troponins), the patient was admitted to the Chest Pain Unit. At the admission, the blood pressure was 100/65 mmHg, cardiac and pulmonary examinations were normal, Murphy's sign was positive. The electrocardiogram described slight non-specific abnormalities of ventricular repolarization.

Echocardiography examination was in the standard. Laboratory tests showed an increase in troponins (4,160 ng /ml), D-dimer (1.05 mg/L), reactive C-protein (31.6 mg/L) and transaminases (aspartate amino-transferase: 81 U/L; alanine amino-transferase: 126 U/L).

We performed also a stool culture but there were no abnormal bacteria (Campylobacter, Salmonella, etc.) or other germs in the sample. Abdominal echography examination was normal and thorax, abdomen and pelvis CT highlighted the presence of thoracic parenchymal nodules of small size of uncertain nature, biliary sand in the gallbladder, a modest effusion in the Douglas cavity, numerous lymph nodes (>2 cm in diameter) at the mesenteric fold.

Pulmonary consultation advised therapy with hydration and determination of markers of hepatitis, anti-thyroid antibodies, Mantoux skin test, research on Koch's bacillus, quantiferon test, and viral panel (Cytomegalovirus, Epstein-Barr virus, etc.). All these tests resulted negative. The patient did not perform blood or allergy test for Anisakis. We performed the detection of Toxoplasma-specific antibodies, that was negative.

The following day, in the morning, the patient continued to suffer mild heartburn, troponins decreased (2.2 ng/ml), but at the second determination, after 12 hours from the first one, troponins increased again (8.03 ng/ml). The echocardiography evaluation showed a medium amount of pericardial effusion with a length-diameter of 1.2 cm (Figs. 1a, 1b, 1c).

Then we treated the patient with appropriate therapy for pericarditis. Therapy was ibuprofen 600 mg (1 sachet/three times a day), esomeprazole (1 vial/two times a day) and physiologic solution (500 ml/two times a day). During the hospitalization, there was an improvement in abdominal pain symptoms, a progressive decrease of troponins and the other abnormal values, up to their normalization. The echocardiography evaluation, performed in the last day of hospitalization, showed a reduction of pericardial effusion with a length-diameter of 0.5 cm (Fig. 1d).

We discharged the women after eight days of hospitalization with a diagnosis of "Likely food poisoning with acute myopericarditis. Acknowledgement of small pulmonary nodules of CT examination of uncertain determination. Hypothyroidism in treatment."

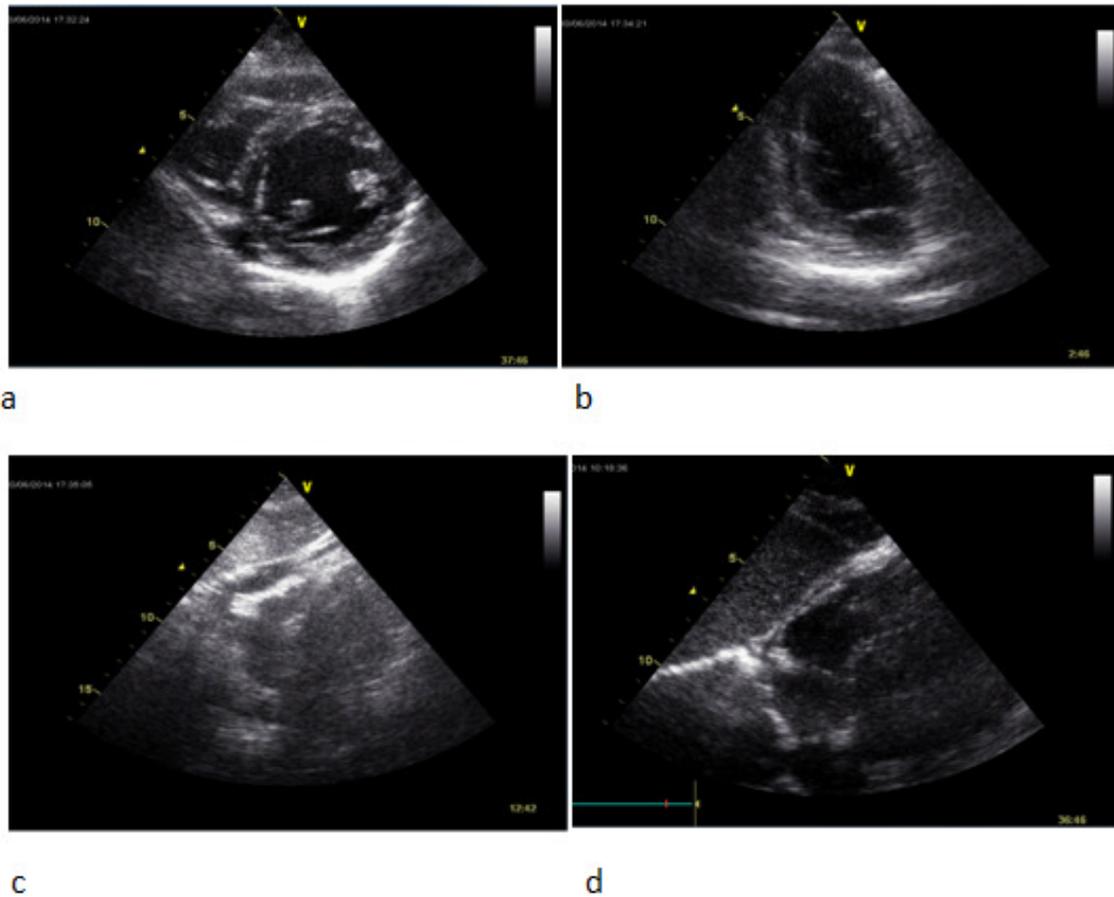
We recommended the following home-therapy: Ibuprofen 400 mg/twice a day, Levothyroxine sodium 25 ug/daily, Esomeprazole 40 mg/daily vial.

After fifteen days we re-evaluated the patient. The examination showed a disappearance of the symptoms and the echocardiography evaluation demonstrated a disappearance of the pericardial effusion. We also performed a new determination of troponins, that was in the standard.

## 2. DISCUSSION

The manuscript describes the case of a 23 year-old woman, with acute myopericarditis (the troponin elevation suggests myocardial inflammatory involvement) associated with food poisoning without other cardiovascular disease.

Only two cases of Campylobacter -associated myocarditis are known [1-2], moreover in patients with other comorbidities.



**Fig. 1A. Parasternal short axis echocardiography examination at the admission in Chest Pain Unit, B. Parasternal long axis echocardiography examination at the admission in Chest Pain Unit, C. Subcostal echocardiography examination at the admission in Chest Pain Unit, D. Subcostal echocardiography examination at the discharge**

However, in the present case report, we cannot exclude the possibility of Salmonella food poisoning. Infact we performed only a single stool culture, that was negative for Salmonella but according to “Guidelines for the investigation and management of food handlers during non-typhoidal Salmonella outbreaks” [3], multiple negative stool cultures are required to prove that you don’t have Salmonella infection.

It is difficult to reliably exclude infection with Salmonella due to intermittent excretion in stool and decreased test sensitivity at low levels of excretion. In literature is reported a case of three subjects with Salmonella typhimurium food poisoning [4], in which bacteraemia was confirmed only in two patients, in one of whom no organisms were isolated from stool cultures, as in our case report. Moreover this patient

developed myopericarditis, like in our case [4]. This outbreak illustrates that salmonella gastroenteritis, although usually fairly mild and self-limiting, can be a virulent disease resulting in serious complications [4].

Pancarditis is well documented in literature with non-typhoidal Salmonellae since the 1930s and this may explain the paucity of more recent reports [5-7]. It is a known complication of intestinal infections with these organisms, albeit occurring in <2% of cases. Most cases of cardiovascular complications occur due to infection with nontyphoidal species, such as Salmonella dublin [8], as a result of their propensity to cause bacteremia. Salmonella has the ability to adhere to damaged endothelium, predisposing individuals to complications rarely seen with other Gram-negative organisms.

Potential complications include endocarditis, myocarditis and pericarditis.

The present report highlights the importance to be aware of the ability of Salmonella to infect pericardium, especially in individuals with risk factors for atherosclerosis and also with negative stool culture.

### 3. CONCLUSION

It is reasonable to think that other cases have gone undetected because of a clinical picture. This case underlines the importance of considering possible cardiac complications when treating patients with food poisoning.

### CONSENT

All authors declare that written informed consent was obtained from the patient for publication of this case report and accompanying images.

### ETHICAL APPROVAL

All authors declare that written informed consent was obtained from the patient for publication of this case report and accompanying images.

### COMPETING INTERESTS

Authors have declared that no competing interests exist.

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