ESC European Society of Cardiology

European Heart Journal - Case Reports (2019) **3**, 1–5
doi:10.1093/ehjcr/ytz138

CASE REPORT
Cardiac imaging

A case report of a giant hiatal hernia mimicking an ST-elevation myocardial infarction

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Received 5 March 2019; first decision 1 April 2019; accepted 5 August 2019; online publish-ahead-of-print 19 August 2019

Background Acute coronary syndrome (ACS) can be a life-threatening condition. However, identification of patients with ACS can be challenging, especially among women, and clinical presentation can often overlap with other medical entities.

Case summary A 61-year-old woman with a history of stable bronchial asthma presented with worsening dyspnoea for spiroergometry. During bicycle exercise testing, she developed acute chest pain and her electrocardiogram showed significant ST-segment elevations. High-sensitivity cardiac troponin was elevated and a coronary angiography was performed showing normal coronary arteries. Cardiac magnetic resonance imaging showed no signs of myocardial infarction, myocarditis or Takotsubo cardiomyopathy but the incidental finding of a giant hiatal hernia impeding the filling of the left atrium. The giant hernia was surgically corrected, and the patient's exertional dyspnoea fully relieved during follow-up.

Discussion Hiatal hernia might compress cardiac structures, cause exertional dyspnoea and mimic ST-elevation myocardial infarction.

Keywords Giant hiatal hernia • Acute coronary syndrome • ST-elevation myocardial infarction • Electrocardiogram • Cardiac compression • Case report

Learning points

- Gastrointestinal symptoms caused by hiatal hernias can cause angina equivalent symptoms.
- Hiatus hernias may cause compression of the cardiac chambers and result in exertional dyspnoea, cause abnormalities on the electrocardiogram including ST elevation and cause raised serum biomarkers of acute coronary syndromes.
- A variety of non-cardiac aetiologies should be considered in the differential diagnosis of patients with chest pain and ST-segment elevation on the electrocardiogram, in particular, in the absence of obstructive coronary disease and alternative cardiac abnormalities.

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Handling Editor: Gianluigi Savarese

Peer-reviewers: Francesca Musella and John Kanakakis

Compliance Editor: Mohammed Majid Akhtar

Supplementary Material Editor: Peysh A. Patel

Introduction

Acute coronary syndromes (ACS) can be a life-threatening condition. However, identification of patients with ACS can be challenging. Women are known to present with atypical symptoms.[1–5] Frequently, clinical presentation can be confusing and other important diseases can be misdiagnosed.

One of the most frequent atypical symptoms for an ACS is dyspnoea, which is considered as an equivalent of angina pectoris. The term dyspnoea refers to a wide variety of subjective perceptions, which can be caused by many underlying conditions including cardiac diseases (as ACS, acute heart failure, pulmonary embolism, cardiac tamponade), pulmonary diseases (as respiratory infections, chronic obstructive pulmonary disease, asthma, pneumothorax) but also many other conditions as obesity, anaemia, several neurological disorders (Guillain-Barré, amyotrophic lateral sclerosis, multiple sclerosis, muscular dystrophy), and gastrointestinal disorders including gastro-oesophageal reflux disease, neoplasia, and hernias.[6]

Similarly, ST-segment elevations are a pathognomonic sign for an ST-segment elevation myocardial infarction (STEMI), which often represents an acute vessel occlusion and is a time-dependent medical emergency.[7] Nevertheless, other cardiac entities as myocardial infarction with non-obstructive coronary arteries (MINOCA), myocarditis, Takotsubo cardiomyopathy, aortic dissection, pericarditis, ethnic variant, and hypertension with strain, repolarization abnormalities, and ventricular aneurysm can mimic the electrocardiographic signs of STEMI. Furthermore, non-cardiac related disorders (such as metabolic disturbance, stroke, intracranial haemorrhage) can also present with similar electrocardiogram (ECG) abnormalities.[8] Finally, arrhythmias such as atrial tachycardia, atrial fibrillation, supraventricular tachycardia, paroxysmal atrial flutter, and electrocardiographic changes such as T-wave inversion have been reported with large hiatal hernias in previous case reports.[9–13]

Timeline

Case presentation

A 61-year-old white woman with an 8-year history of stable bronchial asthma under medical treatment developed worsening exertional dyspnoea for 8 months and therefore presented for pneumological assessment including cardiopulmonary exercise testing (CPET). Lung function test showed no evidence of obstruction [FEV_1 of 2.1 L (80% of expected) and FEV_1/VC_{max} 70.7%]. During CPET, the patient developed acute retrosternal chest pain at 59 W (59% of age-predicted Watt) with worsening dyspnoea and progressing diffuse ST-segment elevation, more pronounced in the anterolateral ECG leads with maximal elevation of 2 mm (*Figure 1*). Due to exacerbation of her symptoms and the ECG changes, the CPET had to be prematurely terminated after 6 min and the patient was referred to the cardiology department. The 12-lead ECG showed multiple ST-segment elevations, more pronounced in the anterolateral leads (*Figure 1*). Findings on physical examination were unremarkable including no auscultatory signs of a pulmonary obstruction. Laboratory tests revealed elevated levels of N-terminal pro B-type natriuretic peptide (NT-pro-BNP) (349 ng/L, normal range <170 ng/L) and high-sensitivity cardiac Troponin T (hs-cTnT) (33 ng/L, normal range <14 ng/L) with slight dynamic changes in the first hours (after 1 h: 37 ng/L, after 3 h: 36 ng/L) and elevated white blood cell count (15.15 × 10S9/L, normal range 3.5–10 × 10S9/L) with a normal C-reactive protein. Bedside echocardiography revealed a normal left and right ventricular function without regional wall motion abnormalities, signs for a diastolic dysfunction (Grade I) and high normal levels of pulmonary artery pressure.

The first clinical suspicion was a STEMI. However, since the patient's symptoms improved gradually after termination of the CPET and because of the lack of relevant dynamic changes of hs-cTnT, despite no resolution of ECG changes, a coronary angiography was performed only on the next day. It revealed normal coronary arteries, with normal left ventricular function and increased end-diastolic pressure (16 mmHg) but no explanation for the ST elevations.

Time	Events
January 2010 to December 2018	Stable history of bronchial asthma, oligosymptomatic under treatment
January 2018 to September 2018	Progressive exertional dyspnoea, without clear pneumological reason
Day 0	Spiroergometry to discern between cardiac or pneumological aetiology of dyspnoea: • No pneumological signs for worsening asthma with a reduced exercised capacity (max. 59 W) • Progressive global significant ST-segment elevation in several leads, therefore premature termination Elevated cardiac troponin levels Echocardiography showing normal left ventricular function and a diastolic dysfunction Grade I
Day 1	Angiographically normal coronary arteries, elevated left ventricular end-diastolic pressure
Day 2	Cardiac magnetic resonance imaging showed no signs of myocardial infarction, myocarditis or Takotsubo cardiomyopathy but a giant hiatal hernia impeding the filling of the left atrium
Day 6	Discharge from hospital
Day 22	Surgical correction of the giant hiatal hernia (fundoplication)
Day 81	Full relief of the exertional dyspnoea

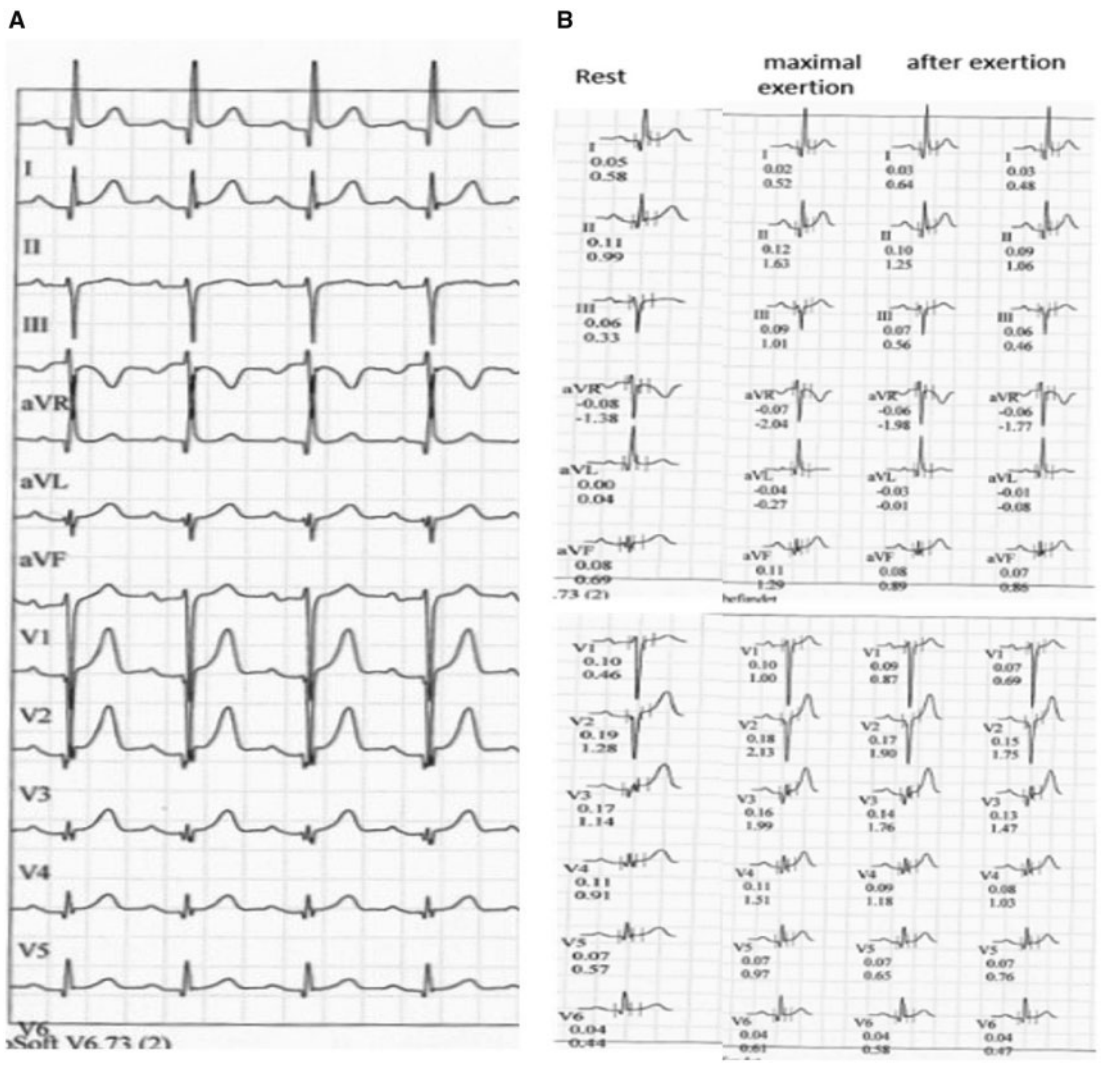

Figure 1 Electrocardiographic changes. (*A*) Rest-electrocardiogram showing non-specific ST-segment elevations in all leads, more pronounced anterolateral. (*B*) Evolution of ST-segment elevations during exertion with a maximal exertion at 59 W showing more prominent ST-segment elevations compared to rest-electrocardiogram.

Due to the unclear symptoms of the patient, ECG abnormalities and elevated blood biomarkers (hs-cTnT and NT-proBNP values) in a young female, a cardiac magnetic resonance imaging (MRI) was performed: the MRI showed normal biventricular dimensions with normal biventricular global and regional function. There was no evidence of a myocardial oedema on T2 mapping images. Late gadolinium enhancement images did not show any subendocardial, transmural or other focal myocardial or pericardial enhancement. There was no evidence of diffuse myocardial fibrosis as shown by normal extracellular volume of the myocardium of 24% (using native and post-contrast T1 mapping). In conclusion, the MRI excluded MINOCA and there was no evidence of an acute (peri-)myocarditis, Takotsubo cardiomyopathy, or other cardiomyopathy (*Figure 2*).

However, a translocation of more than 50% of the stomach to the thoracic cavity (giant hiatal hernia) impeding the filling of left atrium was detected (*Figure 2*, Supplementary material online, *Video S1*). Additionally, a pre-operative computed tomography was performed (Supplementary material online, *Video S2*) confirming the giant hiatal hernia.

After consultation with the visceral surgeons, the patient's giant hiatal hernia was successfully laparoscopically corrected by fundoplication during a second hospitalization (Supplementary material online, *Video S3*). The post-operative course was uneventful.

In a follow-up appointment 2 months later, the patient's exertional dyspnoea was no longer present. Her ECG repolarization disorders however still persisted at three-months (*Figure 3*).

Discussion

We report the case of a 61-year-old woman with typical chest pain and significant ECG repolarization abnormalities under exertion mimicking STEMI. Although the coronary angiogram showed normal

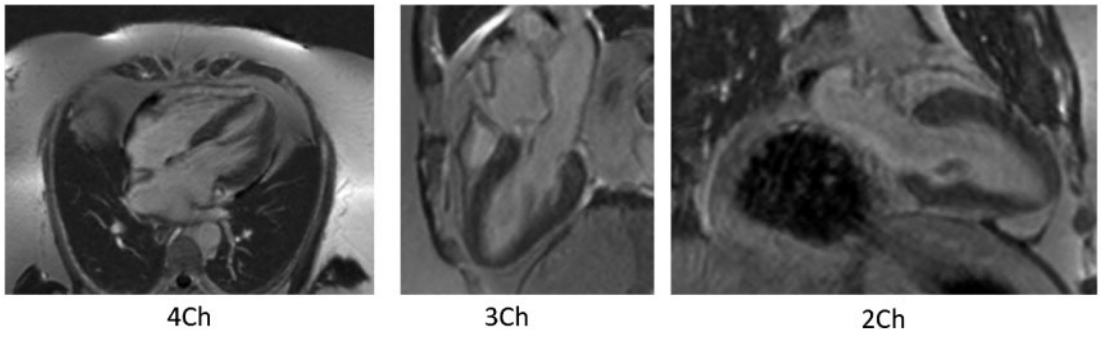

Figure 2 Heart-magnetic resonance imaging. Heart-magnetic resonance imaging displaying no late gadolinium enhancement, showing no evidence of myocardial infarction or myocarditis but a giant hiatal hernia leading to a haemodynamic compromise of the filling of left atrium.

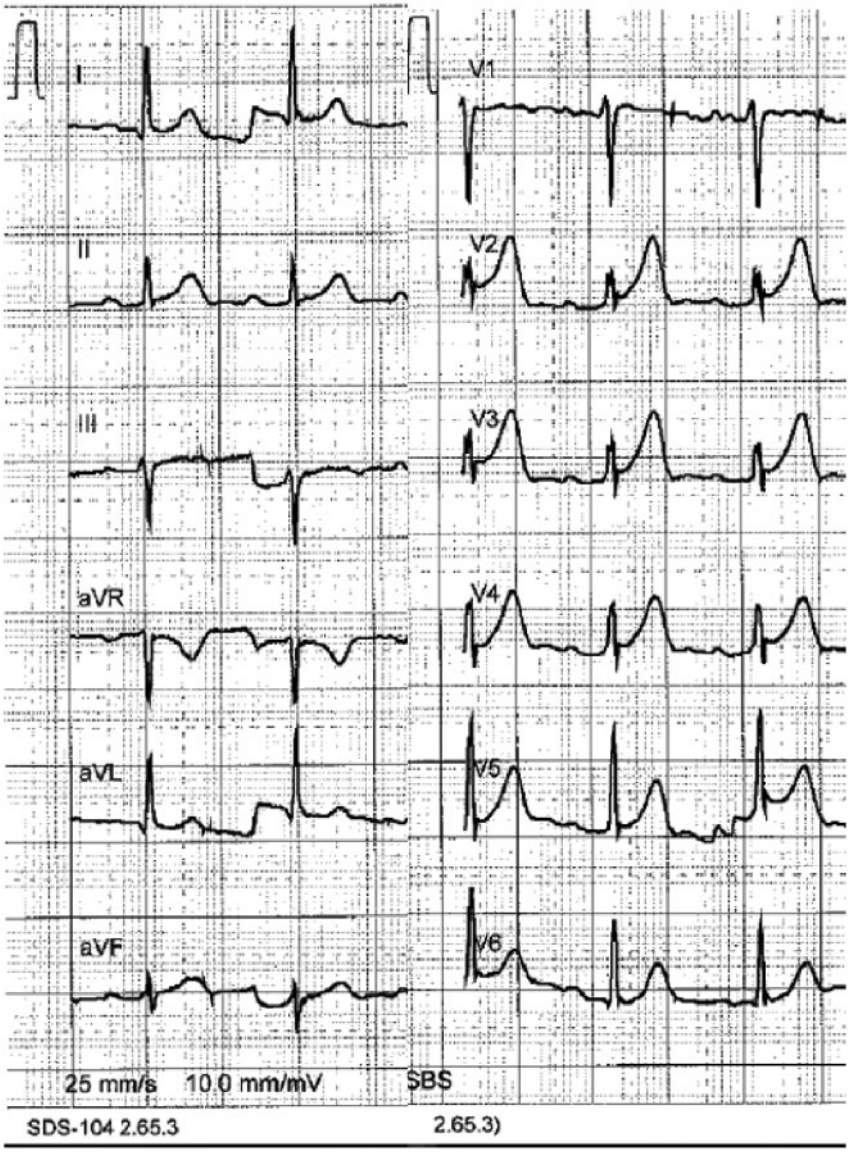

Figure 3 Electrocardiographic changes. Rest-electrocardiogram at Day 81 showing persistence of non-specific ST-segment elevations in all leads.

coronary arteries, we decided to further examine the patient by an MRI scan and thereby excluded other cardiac-related disorders and MINOCA. With current late gadolinium enhancement (LGE) techniques MRI is able to detect as little as 1 g of infarcted myocardium.[14] Nevertheless, some patients with normal MRI scans may still have too little myonecrosis to be detected by MRI but currently only by high-sensitivity cardiac troponin assays. Also necrotic myocytes may be distributed over a larger area with no contiguous island of cell death of sufficient size to be detected by LGE imaging.[15]

Incidentally, a giant hiatal hernia was diagnosed by cardiac MRI impeding the filling of the left atrium. After surgical correction, the patient's symptoms fully were relieved.

Few cases have been reported where a gastrointestinal disorder was the cause of exertional dyspnoea and dynamic ST-segment elevation due to a haemodynamic cardiac compromise.[9–13]

To the best of our knowledge, this is the first described case, where clinic, laboratory findings and ECG mimicked a STEMI in a young woman with a giant hiatal hernia.

The exact mechanism of electrocardiographic changes in patients with gastrointestinal disorders is not well understood. Several hypotheses have been raised. First, an increase in direct or indirect pressure to the global surface of the heart might cause electrical alternation seen on the ECG.[16] Second, hiatal hernia may cause compression of the vagal innervation to the heart causing electrocardiographic changes.[17] Finally, ECG changes might be caused by pericardial irritation.[12] The pathophysiological reasoning behind the non-resolving ECG disorders in this case 3 months after the operation remains speculative but might be due to a pericardial irritation, still persistent several weeks after her operation.

Nevertheless, in patients with exertional dyspnoea and normal coronary arteries, a further assessment using non-invasive cardiac imaging should be considered in order to exclude other cardiac disorders or even extra-cardiac disorders affecting the cardiovascular system. Although it is a rare entity, hiatal hernia might mimic a STEMI and should be considered in the differential diagnosis of acute chest pain and exertional dyspnoea.

Lead author biography

Maria Rubini Gimenez completed clinical training in internal medicine and cardiology at the University Hospital Basel. Currently Fellowship in interventional cardiology at the Heart Center in Leipzig. Research in acute cardiac care biomarkers and gender studies.

Supplementary material

Supplementary material is available at *European Heart Journal - Case Reports* online.

Acknowledgements

We thank Dr Karsten Murray for his critical grammatical revision as an English native speaker.

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

Consent: The author/s confirm that written consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: M.R.G. has received research grants (outside the submitted work) from the Swiss National Science Foundation (P400PM 180828) and Swiss Heart Foundation.

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