CASE REPORT

Case Report: From Irregular Hiccups to Acute Myocardial Infarction

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ABSTRACT

Introduction: The electrocardiogram (ECG) is the key player in the diagnosis of an acute coronary syndrome. In the light of normal ECG findings, the diagnosis can be missed. The high-sensitive troponin is a necessary laboratory value for patients with uncommon symptoms.

Case Presentation: A 53-year-old man without a history of coronary heart disease initially presented to his general practitioner with persistent hiccups for 3 weeks. In the emergency department, the patient complained of nausea and burping. The high-sensitive troponin T was 989 pg/mL and led to the diagnosis of an acute coronary syndrome.

Conclusion: The troponin algorithm helps to identify this patient group, especially in the setting of elevated creatine kinase and lactate dehydrogenase. Awareness of these symptoms can help lead to a timely reperfusion therapy and thus improved outcomes. The 2015 European Society of Cardiology algorithm for troponin may not only help for the initial diagnosis, but rather should be regarded as crucial.

INTRODUCTION

Acute coronary syndrome (ACS) is a collective name for thoracic complaints resulting from an underlying coronary insufficiency. It includes ST segment elevation myocardial infarction, non-ST segment elevation myocardial infarction, and unstable angina pectoris. The diagnosis relies on a patient’s history, the 12-lead electrocardiogram (ECG), and the troponin level. Yet, the ECG is the key player in differentiating between ST segment elevations versus depressions. Establishing the diagnosis of ACS with normal ECG findings is difficult and can result in the delay of a definitive diagnosis and subsequent treatment. Autonomic symptoms often accompany ACS and include sweating, nausea, vomiting, and unrest. Nausea is present in 25% of patients and is more prevalent among female patients.1

We report the case of a 53-year-old man who initially presented to his general practitioner with persistent hiccups for 3 weeks. In the emergency department, the patient complained of nausea and burping and denied any history of vomiting.

CASE PRESENTATION

A 53-year-old man was sent to the emergency department by his general practitioner. He reported a 3-week history of persistent hiccups. At the time of presentation, the patient’s symptoms were consistent with frequent burps, which occurred irregularly every 1 to 5 minutes; however, there was no evidence of hiccups. The patient was a poor historian and did not have a list of his medications. Previous medical records were unavailable. The medical team was suspicious that the patient’s symptoms were nonpathologic and self-induced. Of note, his symptoms were accompanied by tremor and cold sweats. The patient’s medical history was significant for 2 cardiac risk factors: arterial hypertension and a history of smoking (active, 40 pack-years). He did not have any other comorbidities or prior hospitalizations. While obtaining the family history, the patient mentioned his brother’s heart attack a year ago at the age of 50. His initial vital signs were: temperature, 98.42°F (36.9°C); pulse, 104 beats/min; blood pressure, 125/85 mmHg; 16 respirations per minute; and oxygen saturation, 98% on room air.

Physical examination revealed a man with moderate tenderness to palpation of his upper left chest. The patient had a regular heart rate and rhythm without murmurs. The patient was treated with ramipril 2.5 mg every day, metoprolol 47.5 mg every day, atorvastatin 40 mg every day, as well as 40 mg of pantoprazole every day. As part of the standard procedure in our emergency room, a 12-lead ECG was obtained (Figure 2). There were no previous ECGs for comparison. The ECG displayed sinus tachycardia, left axis deviation, a left anterior hemiblock, and S-persistence through V6, without ST changes. With an increased lactate dehydrogenase of 482 U/L and no clear clinical lead for another pathology cardiac markers were obtained. The results were significant for high-sensitive troponin T, 989 pg/mL (normal range > 14 pg/mL).

Therapeutic Intervention and Treatment

The patient received intravenous aspirin 500 mg and 5000 IE heparin. Because of his lack of chest pain or anxiety, no further medication was given. An ECG was obtained immediately, which revealed normal cardiac output with hypokinesia of the anteroseptal wall. The patient was taken urgently to the cardiac catheterization laboratory (Table 1).
Results of the coronary angiogram demonstrated moderate ectatic angiopathy of the left anterior descending artery and left circumflex artery with a subtotal stenosis of ramus marginalis sinister (RMS), suspected to be the culprit lesion. Also, a 50% to 60% stenosis of the Arteria coronaria dextra (segment 2) was present (Figure 1). Successful percutaneous transluminal coronary angioplasty of the proximal RMS lesion was performed. During the catheterization and inflation of the balloon in the RMS, the patient began to burp and complained of nausea and cold sweats. Seconds after the stent placement, these symptoms ceased. Krysiak et al reported a similar case, yet without the immediate cessation of the symptom.\(^2\) The patient was admitted to the intensive care unit for 24-hour observation. During this time,
no further episodes of hiccups occurred. The patient was discharged on hospital day 3, with improving cardiac markers.

Pathophysiology

The reflex arc of hiccups consists of the afferent limb composed of the phrenic, vagus, and sympathetic nerves followed by a central processor in the mid-brain and finishes with the efferent limb composed of the phrenic nerve supplying the diaphragm.3 This sole presentation of this autonomic symptom could be explained by 2 mechanisms. On the 1 hand, the inflammatory markers released by the myocardium may trigger the pathway, thereby producing hiccups. On the other hand, the close proximity of the heart to the phrenic nerve may lead to either the irritation of the vagus nerve supplying the pericardium or the phrenic nerves, which innervates the diaphragm. Whether the trigger comes from the direct effect on the nerve or the markers remains unclear.4 Involuntary burps may follow the same pathophysiology in the light of myocardial damage.

CONCLUSION

Discussions of this phenomenon were first published in 1939 and 1958.5 Despite the known relationship between hiccups and ACS, without prominent ECG findings, these symptoms can be overlooked and lead to a delay in treatment.5 Rarely burps or hiccups are the sole symptom of an ACS. As in this case, they often present secondary to an inferior myocardial infarction.

The 2015 troponin algorithm by the European Society of Cardiology helps to identify this patient group, especially in the setting of elevated creatine kinase, creatine kinase-MB, and lactate dehydrogenase. Awareness of these symptoms and subsequent high-sensitive troponin T testing can help lead to a timely reperfusion.
Table 1. Case timeline

<table>
<thead>
<tr>
<th>Time</th>
<th>Workup (findings)</th>
<th>Treatment</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>3 wks before</td>
<td>Onset of symptoms</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>admission</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Arrival at 8:30 o’clock</td>
<td>ECG, laboratory: increased lactate</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td></td>
<td>dehydrogenase</td>
<td></td>
<td></td>
</tr>
<tr>
<td>9:15 o’clock</td>
<td>Hs Troponin 989 pg/mL</td>
<td>5000 IE heparin, aspirin 500 mg</td>
<td>None</td>
</tr>
<tr>
<td>9:45 o’clock</td>
<td>Echocardiography</td>
<td>None</td>
<td>Normal cardiac output with hypokinesis of the anteroseptal wall</td>
</tr>
<tr>
<td>10:15 o’clock</td>
<td>Reperfusion therapy: angiopathy of the</td>
<td>Successful percutaneous transluminal coronary</td>
<td>Cessation of persistent hiccoughs, involuntary burps, Tremor, Sweats an</td>
</tr>
<tr>
<td></td>
<td>left anterior descending artery and</td>
<td>angioplasty of the proximal RMS lesion was</td>
<td>nausea</td>
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<tr>
<td></td>
<td>left circumflex artery with a subtotal</td>
<td>performed</td>
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<tr>
<td></td>
<td>stenosis of RMS, suspected to be the</td>
<td></td>
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<tr>
<td></td>
<td>culprit lesion. Also, a 50% to 60%</td>
<td></td>
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<tr>
<td></td>
<td>stenosis of the Arteria coronaria dextra</td>
<td></td>
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<tr>
<td></td>
<td>(segment 2) was present</td>
<td></td>
<td></td>
</tr>
<tr>
<td>24-h observation</td>
<td>None</td>
<td>None</td>
<td>None</td>
</tr>
</tbody>
</table>

ECG, electrocardiogram; RMS, ramus marginalis sinister.
therapy and thus improved outcomes. Thus, the troponin algorithm may not only help in the initial diagnosis, but is also crucial.

The differential diagnosis for persistent hiccups should not only include gastroenterological etiologies, but rather, should be viewed from a general internal medicine point of view, with multiple potential culprits such as myocardial infarction.

Disclosure Statement
The author(s) have no conflicts of interest to disclose.

Authors’ Contributions
Kamiar Rueckert, MD, participated in the critical review, drafting, and submission of the final manuscript. Adrian Willersinn, MD, participated in the review and drafting of the final manuscript. All authors have given final approval to the manuscript.

How to Cite this Article

References