

## SEVERE HYPONATRAEMIA: A STEMI MIMIC

*Loi Siew Lin<sup>1</sup>, Aaron Lai<sup>1</sup>, Chan Hiang Chuan<sup>1</sup>*

<sup>1</sup>Emergency and Trauma Department, Sarawak General Hospital, 93586 Kuching, Sarawak, Malaysia.

### ABSTRACT

ST segment elevation can be found in patients presenting with or without chest pain. We reported a case of ST segment elevation caused by severe hyponatraemia. A middle age Malaysian, newly diagnosed pulmonary tuberculosis on treatment, presented with nausea and vomiting for a week. He had no anginal symptoms. He was clinically dehydrated. His electrocardiogram showed ST segment elevation with Q wave at V1-V4 as well as T wave inversion at leads I and aVL. Bedside echocardiography revealed good LV contractility but a hypokinetic septal wall. Cardiac enzymes were within normal limit. He was hydrated with intravenous fluid. The diagnosis was revised to severe hyponatraemia instead of STEMI or TB myopericarditis when the sodium level came back as 98mmol/L. He improved clinically with hydration over days and his ECG normalized when the sodium level normalized. As the treatment for hyponatraemia induced ST segment elevation differ from ST elevation myocardial infarction, it is equally important to identify the exact cause of ST segment elevation.

### Corresponding Author:

Dr Loi Siew Ling

Emergency and Trauma Department,

Sarawak General Hospital,

93586 Kuching, Sarawak, MALAYSIA.

Email: [silynloi@yahoo.com](mailto:silynloi@yahoo.com)

Tel: +6082-276666 ext 5176; +6016-8973080; Fax: +6082-276761

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**Informed consent:** We managed to get only verbal informed consent (reinforced via telephone call) but not the written informed consent. Therefore, the case report was anonymised to make sure no patient data was exposed.

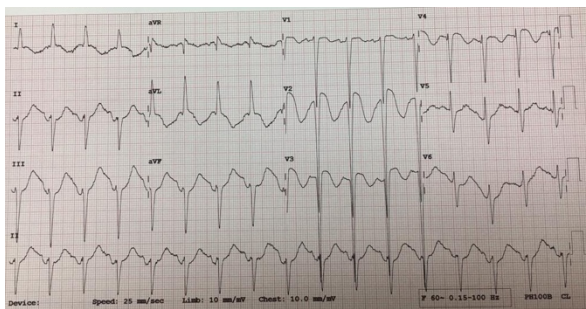
## INTRODUCTION

ST segment elevation (STE) can be found in patients presenting typically with chest pain or atypically without chest discomfort. In emergency medicine, it is always assumed to be an acute myocardial infarction (AMI) until proven otherwise. However, many conditions which alter the cardiac repolarization can produce STE, not necessarily due to an AMI. The purpose of this article is to discuss STE caused by severe hyponatraemia mimicking a STEMI.

## CASE PRESENTATION

We reported a case of middle age Malaysian who presented to our emergency department with nausea and vomiting for 1 week. He was recently diagnosed to have pulmonary tuberculosis on intensive phase treatment day 19. He had no shortness of breath, chest pain, seizures or diarrhoea. He was clinically well perfused but dehydrated. Other systemic examinations were unremarkable. The first electrocardiogram (ECG) showed STE with Q wave at V1-V4 as well as T wave inversion at leads I and aVL. Repeated serial ECGs noted no dynamic changes. Bedside echocardiography revealed good LV contractility but a hypokinetic septal wall. Troponin T levels was not raised.

Image 1: Initial ECG on Presentation



The diagnosis was revised from STEMI to a probable TB myopericarditis based on the patient's risk factors. ACS

treatment regime was not initiated. Blood investigations later on revealed a sodium level of 98 mmol/L (hypovolemic hypoosmolar hyponatremia). Prior to availability of blood results, the patient was hydrated with 1.5L normal saline over 3 hours. The sodium level was slowly corrected not exceeding 12 mmol/L/day. Patient remained pain-free during the admission with resolution of GI losses. Repeated ECG later showed resolution of STE as the sodium normalized. Patient was discharged well with a serum sodium of 127 mmol/L.

## DISCUSSION

The normal cardiac repolarization may be altered by electrolyte imbalance<sup>1</sup>. Traditionally, it is reported that hyponatraemia does not have any major ECG changes<sup>2</sup>. However, severe hyponatraemia may cause STE as depicted in this case. In developing country like Malaysia where coronary angiography is limited in accessibility, we have to treat patient clinically based on the available resources. Luckily this patient was not thrombolysed as per STEMI protocol and he improved markedly after the sodium correction.

## CONCLUSION

Severe hyponatraemia may present with STE on ECG mimicking STEMI. Hence, patient's history, physical examination findings, other features of the ECG and echocardiography should be taken into consideration when evaluating the differential diagnosis of STE.

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