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Hypercalcemia Simulating A Myocardial Infarction with St-Segment Elevation

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Introduction

Finding ST-segment elevation is essential whenever myocardial ischemia is suspected since patients with this type of myocardial infarction should undergo urgent angiography.[1] Sadly, ST-segment elevation is a generic finding; on angiography, a coronary lesion with a TIMI is discovered in around 75% of patients who come with chest pain and ST-segment elevation.[2] Furthermore, it is discovered that the coronary arteries of about 2.8% of individuals with suspected STEMI are angiographically normal.[3] On an electrocardiogram, hyperkalemia and hypercalcemia are examples of electrolyte disorders that can cause ST-segment elevation and a pseudo-infarction pattern.[4] It is frequently necessary to decide whether to continue with invasive angiography before test data are available in patients who present with suspected STEMI.[5]

Case presentation

A 70-year-old woman arrived at the emergency room complaining of near syncope, nausea, vomiting, and exhaustion during the past few days. Past history of a lumpectomy and radiation treatment for left breast cancer was remote 15 years ago.

She was only responding to discomfort at the emergency room and had changed mental status. Her ECG presented with fresh ST-segment elevation in V1 through V3, which is suggestive of STEMI. Her symptoms did not point to a myocardial infarction, but the ECG did. An emergency bedside transthoracic echocardiography was performed, and the results showed normal motion of the anterior wall and an ejection fraction of 55% without any anomalies in segmental wall motion. She was deemed unlikely to have STEMI based on her clinical history and the echocardiographic results, thus she did not have coronary angiography.

Most importantly, when her troponin levels were checked three, twelve, and hours after admission, they were all less than 0.10 ng/mL, ruling out myocardial infarction as the reason for the alterations in her ECG. Her ionized calcium was 2.6 mmol/L, and her serum calcium was high at > 15.0 mg/dL. The diagnosis of humoral hypercalcemia of malignancy was confirmed by the fact that her parathyroid hormone was markedly high and her parathyroid hormone-related peptide was at the low end of the normal range. It's interesting to note that she had no history of hypercalcemia and that her metabolic profile, evaluated eight days before admission, showed a normal serum calcium of 8.9 mg/dL.

After receiving brain computed tomography and magnetic resonance imaging, there was no indication of a stroke, intracranial bleeding, or metastatic illness. The vigorous injection of intravenous pamidronate, calcitonin, furosemide, and 0.9% normal saline was used to treat her hypercalcemia. An ECG showed normalizing serum calcium levels and resolving ST-segment elevation. After receiving brain computed tomography and magnetic resonance imaging, there was no indication of a stroke, intracranial bleeding, or metastatic illness. The vigorous injection of intravenous pamidronate, calcitonin, furosemide, and 0.9% normal saline was used to treat her hypercalcemia. An ECG showed normalizing serum calcium levels and resolving ST-segment elevation.

Discussion

Hypercalcemia, an often under-recognized condition, can manifest with an uncommon electrocardiographic anomaly: ST segment elevation.[6] Previous reports have linked this occurrence to various conditions such as vitamin D toxicity, milk-alkali syndrome, hyperparathyroidism, and malignancy. In the bloodstream, around 40% of calcium exists in an active ionized form, while the rest binds to proteins or forms complexes with phosphate or citrate.[7] Remarkably, this case report marks the first instance of linking ST-segment elevation directly to plasma ionized calcium levels. Hypercalcemia, particularly in malignancy, frequently presents with markedly elevated serum calcium levels, where values exceeding 15 mg/dL significantly escalate the risk of death within a month.[8] The largest documented case series observed 15 instances of ST-segment elevation over 15 years, showcasing serum calcium levels ranging from 11 mg/dL to 20.4mg/dL, with approximately half attributed to underlying malignancy. Predominantly, electrocardiographic alterations are evident in the precordial leads.[9] One proposed theory suggests that hypercalcemia-induced shortening of the QaTc interval permits a high take-off of the ST segment, mimicking acute myocardial infarction.[10] This hypothesis aligns with our patient's findings, exhibiting a notably shortened QaTc interval on the ECG amidst a substantially elevated ST segment. Notably, hypercalcemia can induce other ECG abnormalities mimicking myocardial ischemia, including inverted, biphasic, notched, or flattened T waves. Our patient's ECG depicted a characteristic flattened T-wave as the ST segment elevation resolved. While hypercalcemia stands out as a significant cause of ST segment elevation, it's crucial to consider other pertinent etiologies for this electrocardiographic finding.

Conclusion

Severe hypercalcemia is known to cause ST segment elevation, and doctors should be aware that elevated calcium levels might cause an ECG pseudo-infarct pattern. When assessing the ECG for symptoms of ischemia, it is important to take into account the likelihood of electrolyte-induced alterations, especially in

individuals who have a history of cancer and may be particularly susceptible to hypercalcemia.

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