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ST elevation in the setting of hypercalcemia masquerading myocardial infarction

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Introduction: Seventy eight (78) year old lady presented to a rural setting post a fall. Routine ECG was showing ST elevation of 3 mm in lead V2 and V3 with no corresponding ST depression and T wave changes. There was no history of chest pain or any other cardiac symptoms. Relevant risk factors included; a history of smoking, age, Indigenous demographic and chronic kidney disease stage III b, though there is no history of diabetes mellitus. Serial troponin in the rural setting on I STAT was normal (0.03 and 0.05) which was followed again after transfer to tertiary hospital which was 65 (<10 ng/L) though not significant in the setting of CKD. There was no change in troponin level in serial tests. Other chemistry panel showed a raised corrected calcium level of 3.61 (2.10-2.60 mmol/L) with other electrolyte abnormality of low Phosphate 0.61 (0.75-1.5 mmol/L) hyponatremia (124 mmol/L) and hypokalemia (3.3 mmol/L). She has a Transthoracic Echocardiogram done where there was no regional wall motion abnormality. As there were no symptoms, invasive angiography was not done. IV fluid and IV zoledronic acid was given which has normalized the serum calcium and resolved the ECG changes. On follow up investigations hypercalcemia was due to paraneoplastic effect most likely suggesting of cholangiocarcinoma.

Discussion: Hypercalcemia has been shown with ST elevation changes in different case reports. Characteristic ECG changes include shortened QT interval, prolonged PR interval, lengthened QRS, flattened or inverted T waves and variable degree of heart block. ST elevation changes are rare in the setting hypercalcemia. In true STEMI, ST elevation is seen with concave, convex or obliquely straight morphology with normal QT interval. Benign early repolarization produces modest ST elevations with concave morphology, especially in V2–V5 without reciprocal ST depressions in an asymptomatic patient. Most likely ST elevation occurs as an artifact due to shortening of interval between S wave and end of T wave. Calcium channels act mainly in phase II of the myocardial action potential which is primarily of calcium influx and potassium efflux. During phase III, calcium influx terminates and potassium efflux predominates as the myocyte repolarizes.

Conclusion: ST elevation is a challenging situation in clinical management. We report another case of ST elevation in the setting of hypercalcemia which has been resolved with the prompt treatment of hypercalcemia. Increasing awareness of this relation of hypercalcemia and ST elevation is useful to prevent unnecessary invasive procedures among the physicians.

Biography

Neelabh Sharma MBBS Cardiology Registrar Cairns Hospital, Cairns Qld Australia.
Sharma has been working as a medical registrar at Cairns hospital since 2015. Before persuing the physician training he has worked extensively in Emergency medicine and critical care. He has successfully completed the Royal Australian college of Physician exam and planning his subspecialty training in Cardiology.

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