



HYPOVITAMINOSIS D AS A CAUSE OF SEVERE HYPOCALCAEMIA IN A FEMALE NIGERIAN: A CASE REPORT

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BACKGROUND

Severe hypocalcaemia is a metabolic emergency condition which is life threatening and must be identified and treated properly to prevent mortality. Causes of hypocalcaemia range from parathyroid hormone-related, electrolytes deficiencies such as hypomagnesemia, hypophosphatemia, medication effects, surgical complications, liver and kidney diseases to nutritional problems such as hypovitaminosis D. Hypovitaminosis D can be classified into mild, moderate and severe vitamin D deficiency. The objective of this presentation is to highlight a rare finding of severe hypocalcaemia due to vitamin D deficiency in a Nigerian female on anti-tuberculous therapy.

CASE PRESENTATION

A 30-year-old Nigerian female who was constantly in purdah presented to the medical emergency on account of perioral numbness and generalised muscle spasms of a week, with generalised malaise, difficulty with breathing, palpitation and significant weight loss. Further enquiry revealed that she had been on first line anti-tuberculous agents for 32days following diagnosis of pulmonary tuberculosis complicated by massive left sided pleural effusion which has been drained once.

General physical examination revealed an asthenic woman with BMI of 17.0kg/m^2 and left axillary lymphadenopathy. Chest examination revealed features in keeping with left massive pleural effusion. She had positive Chvostek's and Trousseau's signs. Investigations revealed haemoglobin of 11g/dL with hypokalaemia (2.9mmol/L) and hypomagnesaemia of 0.34mmol/L ($0.7-1.15$). She had low total calcium 1.66mmol/L ($2.2-2.6$), low corrected calcium of 2.04mmol/L ($2.2-2.6$), low serum albumin (25g/dL) Serum vitamin D was 8.4ng/ml (>30) and normal alkaline phosphatase 116u/L ($40-120$).

A diagnosis of severe hypocalcaemia due to severe vitamin D deficiency worsened with hypomagnesaemia was made on a background of complicated pulmonary tuberculosis. She was treated with multiple intravenous calcium and magnesium, oral calcium, and calcitriol. During her hospital stay, she had chest tube drainage for the massive left pleural effusion, continued on anti-tuberculous therapy. Her clinical state improved and was discharged home in good general condition.



FIGURE 1

DISCUSSION

Severe hypocalcaemia is a metabolic medical emergency not commonly encountered in our day to day practise. It is sometimes misdiagnosed as a seizure disorder when patient present with generalised muscle spasms as in this index case. The causes of hypovitaminosis D identified in the index patient are prolonged use of purdah and rifampicin use. It is recommended for individuals in the tropics with dark skin to have about 20 to 30mins of sunlight exposure in order to make sufficient amount of vitamin D for use per day. Consistent use of purdah prevents sunlight rays from getting to the skin. The precise mechanism of how rifampicin contributes in depleting active form of vitamin D is unknown. However, association has been documented between prolonged use of rifampicin and low circulating vitamin D levels. Rifampicin is an inducer of enzymes of the cytochrome P450 which catalyse the bioactivation of vitamin D in the liver and kidney. Rifampicin also causes alkalemia which increases calcium binding to albumin, therefore decreasing ionised calcium. Other factors contributing to the manifestation of severe hypocalcaemia in this patient include hypoalbuminaemia and hypomagnesemia.

CONCLUSION

Prolonged use of rifampicin for the treatment of tuberculosis is a cause of hypovitaminosis D. Combination of other aggravating factors such as prolonged use of purdah could potentiate severe hypocalcaemia in individuals on anti-tuberculous therapy.

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