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Case Report

Title: An Unexpected Case of Postparathyroidectomy Normocalcaemia

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ABSTRACT

The following case study involved a sixty years old post-menopausal lady presenting to the Department of Biochemistry, in a tertiary care hospital, with non-specific weakness and tingling of fingers and toes following elective parathyroidectomy, two days previously. She had stage III hypertensive nephropathy and was under immobilization for last two months due to hip surgery. Biochemical investigation revealed normal serum calcium and phosphate levels. However, she was found to be hypoproteinemic and also exhibited severe deficiency of 25-OH vitamin D. This low level of Vitamin D may be the result of parathyroidectomy in the setting of stage III nephropathy and prolonged immobilization. This along with loss of functional parathyroid glands also accounted for normal phosphate levels. Hence normal biochemical levels of minerals may well belie underlying abnormal physical condition. So, very cautious evaluation of such patients is advocated for their management.

Introduction:

A sixty years old postmenopausal lady presented to the department of Biochemistry in a tertiary care hospital with muscle weakness and tingling sensation of limbs, following elective parathyroidectomy, two days back. She had a seven-year history of hypertension and had been diagnosed with stage III hypertensive nephropathy, three years back. Laboratory investigations revealed normal serum calcium and phosphate levels. Since such normal values were unexpected in a patient who had undergone parathyroidectomy two days back and yet not on calcium supplements, the patient was further evaluated, to see if these values could be attributable to derangement of various calcium and phosphate regulatory mechanisms.

Case report:

Blood sample of the patient was sent to the Biochemistry laboratory two days after parathyroidectomy. Since the last year, she had generalized weakness, anorexia, nausea, constipation. She has had a seven-year history of hypertension and had been diagnosed with stage III hypertensive nephropathy, three years back for which she was being treated in the Department of Nephrology of this Institution. However, she was non-diabetic. There was no other significant history except surgery for a fracture of the right femur three months back requiring bed rest for last two months.

A year back, in response to the manifestation of symptoms, she was thoroughly evaluated and found to have a serum calcium level of 15.9 mg/dl (normal 8.7 -10.2 mg/dl), serum phosphate 2.2 mg/dl (normal 2.5-4.3 mg/dl) and serum intact parathyroid hormone (iPTH) 3518 pg/ml (normal 8.0-51.0 pg/ml). She was referred to the Department of Endocrinology where she was diagnosed with an adenomatous nodule in the right inferior parathyroid gland. Serum creatinine level of the past year revealed rise from 1.6 mg/dl to 2.7 mg/dl (normal 0.5 -0.9 mg/dl in females). Haemoglobin and albumin were low at 7.6 g/dl; (normal 12.0-15.8 g/dl in females) and 2.3mg/dl (normal 4.0-5.0 mg/dl) respectively. Proteinuria (+2) was also present.

After full work-up, she underwent an uneventful parathyroidectomy. Two days postoperatively, she developed non-specific weakness and tingling of fingers and toes suggestive of hypocalcaemia. However, Trousseau and Chvostek's signs were negative and there was no perioral tingling. Her serum calcium and phosphate were measured in the Biochemistry department (by Roche AVL 9180 Electrolyte Analyzer) using ion-selective electrodes, following the principle of direct potentiometry. Both were within normal reference intervals (calcium 9.5mg/dl, phosphate 3.1 mg/dl).

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However, considering the sinister implications of parathyroidectomy in a patient with underlying advanced Chronic Kidney Disease (CKD) and prolonged immobilization, some other relevant parameters were estimated including serum albumin; 2.8 gm/dl; serum creatinine 4.7 mg/dl; serum urea nitrogen; 40.1 mg/dl (normal 7.0-20.0 mg/dl), all measured in automated clinical chemistry analyser iMOLA, (RANDOX, India) based on the principle of spectrophotometry. Serum vitamin D level was measured to be 14.9 ng/ml (normal 30.0-50.0 ng/ml) using a commercially available kit EUROIMMUN, (Lübeck, Germany) based on enzyme-linked immunosorbent assay (ELISA).

Discussion:

The current protocol of parathyroidectomy involves mere resection of the abnormal gland, while preserving adjacent normal tissues. This ensures maintenance of normal calcium homeostasis while avoiding development of surgical complications and precludes the necessity of administering calcium supplements postoperatively. [1] However, in hyperparathyroidism the abnormal gland or nodule remains hyper functioning leading to suppression of non-adenomatous parathyroid tissue. Hence immediately after parathyroidectomy, this long suppressed normal tissue fails to maintain calcium homeostasis and serum calcium falls. [2] As this tissue regains its function, calcium level gradually becomes normal. [3] One recent study demonstrated hypocalcaemia in 52% of postparathyroidectomy patients in early postoperative period, among whom 51% were asymptomatic. [4] Along with the various risk factors for development of postparathyroidectomy hypocalcaemia like high preoperative calcium and iPTH values, hypoalbuminemia, the patient had an underlying CKD which tends to further lower blood calcium level. Hence, hypocalcaemia was highly expected in her. Paradoxically, however, her blood calcium and phosphorus levels were found to be within normal limits. However, this does not preclude abnormal calcium and phosphate homeostasis.

Normal phosphate levels can be explained by the fact that, parathormone decreases serum phosphate. Hence, after parathyroidectomy phosphate levels rise. However, she had concurrent vitamin D deficiency probably because of three factors. First, due to deficiency of 1 alpha hydroxylase activity, due to co-existent nephropathy 1, 25 dihydroxy vitamin D levels fall. Secondly, hypo-parathyroidism (postoperatively) causes vitamin D deficiency as PTH is also needed for the 1 alpha hydroxylation. Thirdly, fibroblast growth factor is increased in CKD which suppresses vitamin D levels. The net result is a normal blood phosphate level in such individuals.

Vitamin D deficiency and hypoparathyroidism both cause hypocalcaemia, further compounded by concurrent CKD. Moreover, following parathyroidectomy, calcitonin is released from the thyroid gland, adding to this. However, the patient was normocalcaemic. One hypothesis which could be put forward is that since the patient had deteriorating kidney function as evidenced by increasing serum creatinine, ultimately reaching 4.5 mg/dl, along with decreased haemoglobin and albumin; likely there was also concomitant diminished glomerular filtration, resulting in calcium retention. Prolonged non-ambulation following surgery also resulted in more mobilization of calcium from bone stores. However, she did not become overtly hypercalcaemic, probably as a consequence of co-existing vitamin D deficiency and postoperative hypoparathyroidism, both of which contributed to normal serum calcium levels.

In some cases of secondary hyperparathyroidism, particularly in the setting of CKD, persistent stimulation of parathyroid tissue leads to development of autonomous parathyroid function, unresponsive to medical therapy. This so-called tertiary hyperparathyroidism and requires surgical intervention. Herein lies the importance of early medical management to decrease the proliferative response of parathyroid cells which leading to irreversible genetic changes can result even in malignancy. [5] This patient also demonstrated a striking similarity with tertiary hyperparathyroidism. She had stage III CKD when she developed symptoms suggestive of hypercalcaemia, attributed to an adenomatous parathyroid nodule.

One shortcoming of this case is that there is no iPTH report available of this patient before the development of symptoms of hypercalcaemia. So it is not known whether there was any secondary hyperparathyroidism due to CKD, against which background the autonomous nodule developed or whether it started simply as a case of primary hyperparathyroidism superimposed on CKD.

Conclusion:

This case highlights the necessity of judicious interpretation of apparently normal values of biochemical parameters, since normal level of a parameter does not necessarily mean absence of underlying pathology. There may be multiple underlying interrelated mechanisms in existence, even if the patient comes with a single presenting feature. Hence there is a need to have a full ringside view of all the possible biochemical mechanisms, which may be at play before drawing any conclusions, which in turn will ensure appropriate and effective therapeutic management of such individuals.

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