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Hypoparathyroidism: A Rare Case, Late Diagnosed

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Abstract: *Chronic hypocalcemia due to hypothroidism (an endocrine disorder) could be a result of acquired or hereditary hypoparathyroidism. Patient may manifest in the form of lethargy, personality changes, blurred vision, features of parkinsonism, seizures, muscle cramps, irritatibility and tetany. Here we report a case of hypoparathyroidism with features of parkinsonism with seizures and altered sensorium. He had cataract of left eye. Investigations revealed hypocalcaemia, hyperphosphotaemia and extensive intra cranial calcification in NCCT brain. He was treated with high doses of Vit D combined with elemental calcium in addition to other symptomatic treatment to which he responded well.*

I. Introduction

Acute hypoparathyroidism with hypocalcaemia results into tetany, muscle cramps, irritability, convulsions and carpopedal spasms etc. while in chronic cases the patient may be lethargic with personality changes, mental retardation, seizures and features of parkinsonism. The causes could be acquired or hereditary. As PTH is responsible for minute to minute regulation of plasma calcium;chronic hypocalcaemia means failure of PTH response. The prolonged hypocalcemia with hyperphosphataemia can cause calcification of basal ganglia, grandmal epilepsy, cataract and psychosis.

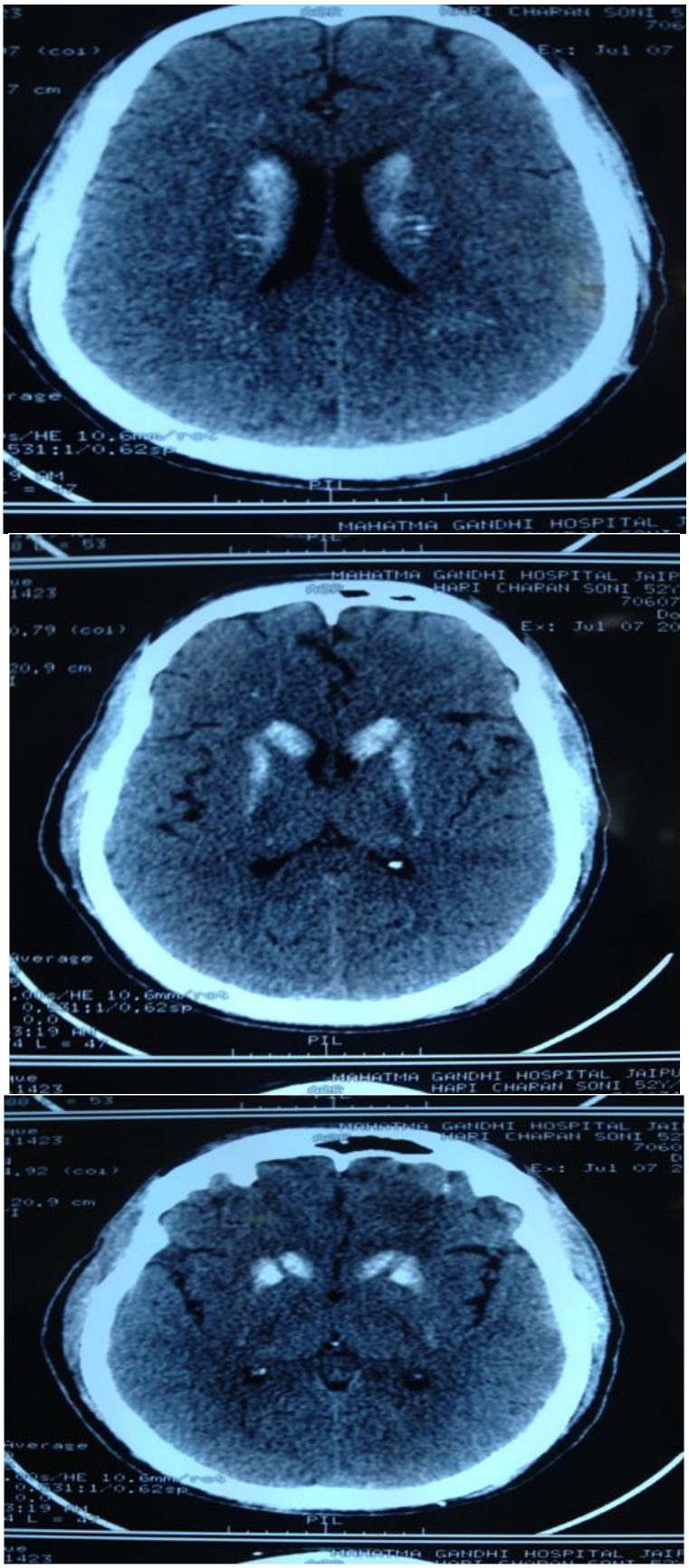
The commonest causes of acquired chronic hypoparathyroidism are thyroid surgery with parathyroid damage, glandular damage in haemochromatosis , wilson's disease and radiation induced damage. Hypoparathyroidism could also be hereditary with genetic defects or complex hereditary autoimmune syndrome involving failure of other glands like ovaries and adrenals etc. It could also be due to abnormalities in calcium sensing receptors .

II. Case Report

A 50 years old male was diagnosed as a case of parkinsonism with recurrent seizures disorder was put on levodopa plus carbidopa and anticonvulsant therapy by a practitioner physician several years back prior to arrival in our hospital . Patient kept deteriorating who had poor compliance of medication. He was brought to our hospital in a state of altered sensorium with stiffness of whole body. He also had multiple episodes of generalised tonic-clonic seizures prior to onset of altered sensorium. He was evaluated in detail. On examination mild pallor was present. He was normotensive, demensic, cataract of left eye and signs of parkinsonism were present. Latent tetany was demonstrable by eliciting chvostek's and trousseau's signs.

On investigations: CBC was normal except Hb 10gm % with low haematocrit, S. Calcium (ionised) was 2.2mgm% & total S. Calcium 4.5mgm%, phosphate 8.4mgm% and magnesium 2mgm%. PTH 4.9pgm/ml (N-16 to 75pgm/ml) ,25 (OH) D (Vit D) 7.13ngm/dl, S. Copper 80mgm/dl. The 24 hour urinary calcium excretion was with in normal range . Renal and liver function tests were within normal range. S. Iron and ferritin level were also in normal range. Total protein and albumin were normal, ECG was evident of prolonged QTc interval with unduly prolonged ST segment. CT brain showed B/L symmetric calcification in multiple areas of brain as shown. All acquired causes of chronic hypocalcaemia like thyroid surgery in the past, radiation therapy, hemochromatosis, wilsons disease and autoimmune polyglandular failure etc. were excluded.

Patient was treated with injectable calcium gluconate, followed by high doses of Vit D and calcitriol and high oral calcium intake, doses adjusted according to S. Calcium levels and urinary excretion.Patient responded well clinically.



NCCT Brain : B/L Symmetrical calcification seen in b/l centrum semiovale, corona radiata , b/l caudate nuclei, b/l putamen and globus pallidus.

III. Discussion

This patient was initially treated for several years as a case of parkinsonism and seizures and was not evaluated in detail by initial treating physician. Hypoparathyroidism with chronic hypocalcaemia whether hereditary or acquired, has a number of common components, although the onset of hereditary hypoparathyroidism is more gradual and often associated with developmental defects as well.

All secondary causes of hypoparathyroidism like infiltrative disorders of gland with iron or copper, metabolic deposits, thyroid surgery with gland damage or radiation induced damage were excluded in our case.Pseudohypoparathyroidism could not be the cause in this case in view of low PTH level. S. Magnesium and S. Albumin were with in normal range. There was no clinical evidence of polyglandular failure. In other common causes of hypocalcaemia like chronic kidney disease ,Vit D deficiency or intestinal malabsorption the PTH level is raised which is not present in this case. Thus, this patient was diagnosed as a case of hereditary hypoparathyroidism who otherwise remained undiagnosed for a long period.

IV. Conclusion

Since adequate treatment of hypoparathyroidism may lead to marked clinical improvement .Due to its rarity ,it is warranted to do serum calcium, phosphorus and PTH level in all individuals with calcification of basal ganglia to rule out hereditary or acquired hypoparathyroidism.