International Journal of Current Advanced Research
ISSN: O: 2319-6475, ISSN: P: 2319 – 6505, Impact Factor: SJIF: 5.995
Available Online at www.journalijcar.org
Volume 6; Issue 6; June 2017; Page No. 4204-4205
DOI: http://dx.doi.org/10.24327/ijcar.2017.4205.0463

Review Article

BASAL GANGLIA CALCIFICATION A RARE FINDING OF HYPOPARATHYROIDISM

Pauline Sheela Priya S

Sree Balaji College of Nursing, BIHER, Chennai, India

A R T I C L E I N F O

Article History:
Received 19th March, 2017
Received in revised form 8th April, 2017
Accepted 24th May, 2017
Published online 28th June, 2017

Key words:
Basal ganglia calcification, Hypoparathyroidism, hypocalcemia, hypomagnesemia

A B S T R A C T

Basal ganglia calcification is due to various causes, such as metabolic disorders, infectious and genetic diseases. Hypoparathyroidism and pseudo hypoparathyroidism are the most common causes of pathological basal ganglia calcification. It is also well known that extensive intracranial calcificatio n caused by hypoparathyrodism is rare. This report presents a case of basal ganglia due to hypoparathyroidism presented with seizures. CT brain showed bilateral symmetrical Basal ganglia calcification .Investigations revealed low serum calcium, low magnesium and low parathoromone levels. Patient was treated with calcium supplementation and is on regulartreatment.

INTRODUCTION

Physiological intracranial calcification occurs in about 0.3-1.5% of cases. It is asymptomatic and detected incidentally by neuroimaging. Pathological basal ganglia calcification is due to various causes, such as: metabolic disorders, infectious and genetic diseases. Hypoparathyroidism and pseudo hypoparathyroidism are the most common causes of pathological basal ganglia calcification. Inherited and neurodegenerative diseases cause symmetrical, bilateral basal ganglia calcification which is not related to metabolic disorders. Since adequate treatment of hypoparathyroidism may lead to marked clinical improvement, serum concentration of calcium, phosphorus, and parathyroid hormone (PTH) is suggested to be determined in all individuals with calcification of the basal ganglia to rule out hypoparathyroidism.

Case Study

A43year old male presented to the casualty to be drowsy, but arousable, oriented to time, place and person. He had no recollection f his episode of seizure. His only complaint was bad headache, drowsy and wanted to sleep. Patient had no history of seizures. On clinical examination vitals were stable.CNS function did not reveal any dysfunction. higher motor functions were intact. Motor power was 5/5 on four limbs with no sensory deficit. Basic investigations were normal. Serum electrolytes, ABG, LFT, normal ECG was within normal limits. CT brain showed bilateral symmetrical calcifications in the basal ganglia, thalami, peri ventricular white matter and dentate nuclei likely represent fahr's disease. MRI brain plain showed flair hyper intensities noted in caudate, lentiform, pulvinar areas due to calcifications.

Corresponding author:* **Pauline Sheela Priya S
Sree Balaji College of Nursing, BIHER, Chennai, India

Causes of Basal Ganglia Calcification

- Post anoxic encephalopathy
- Viral encephalitis
- Methhemogolbinemia
- Down's syndrome
- Tuberous sclerosis
- Pseudo hypoparathyroidism
- Cockayne syndrome
- Fahr's syndrome
- Parkinsonism -dementia complex
- Hypoparathyroidism
- Fetal hypoxia
- Carbon monoxide poisoning
- Lead encephalopathy
- Mitochondrial disorders

In this case the cause of basal ganglia calcification was hypoparathyroidism due to hypomagnesemia.

Idiopathic

- Ageing: common, globus pallidus most commonly affected
- fahr's disease

Basal ganglia calcification is common and is seen in approximately 1% of all CT scans of the brain, depending on the demographics of the scanned population. It is seen more frequently in older patients and is considered a normal

incidental and idiopathic finding in an elderly patient but should be considered pathological in persons younger than age 40 years unless proved otherwise.

Neuropathology

Gross pathologic examination shows accumulation of granular material and solid nodules in the striatum,internal capsule,white matterand cerebellum.circumcized calcium deposits may also be seen in the thalamus and the cerebral cortex.histologic examination of affected area shows concentric calcium deposits within the walls of small and medium sized arteries and less frequently in veins.on electron microscopy the mineral deposits appear as amorphous or crystalline material surrounded by basal membrane. Calcium granules are seen within the cytoplasm of neuronal and glial cells

Hypoparathyroidism is an endocrine disorder caused as result of congenital disorders, iatrogenic causes (eg, drugs, removal of the parathyroid glands during thyroid or parathyroid surgery, radiation), infiltration of the parathyroid glands (eg, metastatic carcinoma, Wilson's disease, sarcoidosis), suppression of parathyroid function such as in hypomagnesemia, HIV/AIDS, or idiopathic mechanisms. Idiopathic Hypoparathyroidism is diagnosed when all the possible causes of hypoparathyrodism are ruled out. Hypoparathyroidism by any cause is well known to basal ganglia calcification in most of the patients. It is also well known that extensive intracranial calcification caused by hypoparathyrodism is rare.

The predilection for basal ganglia calcification in the recognized disorders associated with intracranial calcifications may be due to disorders of calcium metabolism, increased vascular permeability, preferential perfusion of grey matterand high rate of blood flow to the basal gangalia. Further more, alkaline phosphatase activity may be regionally elevated in the basal gangalia in patients developing intracranial calcifications.

Investigations

Serum calcium, phosphorous, magnesium, PTH. ECG may show abnormal heart rythms, alkaline phosphatase.routine hematologic and biochemical investigations.

- Total count:leukocyte 13,100 cells/mm3neutrophils-85%
- Lymphocytes-13%
- Hemoglobin 9.5g/dl RBC count-3.3million/mm3 pcv-28%
- ESR (westgren)half hour:22mm 1 hour 34mm
- Blood urea:39mg/dl serum creatinine1.0mg/dl
- Random blood sugar:120mg/dl
- USG abdomen: normal study
- Chest x-ray normal study
- Serum calcium -6.5mg/dl
- Serum phosphorus-5.0mg/dl
- Serum magnesium-0.8 mg/dl
- 1,25 dihydroxy vitamin d- 40pg/dl
- Serum albumin - 4.0g/dl
- Serum parathyroid hormone(PTH)-8.50pg/ml
- Serum calcium-6.3mg/dl

Treatment

- Calcium carbonate
- Vitamin D supplements

Patient was treated with calcium and magnesium supplements and anti epileptics. There were no further seizure episodes and patient is on regular follow-up

CONCLUSION

Since adequate treatment of hypoparathyroidism may lead to marked clinical improvement and due to its rarity, it is warranted to do serum concentration of calcium, phosphorus, and parathyroid hormone in all individuals with calcification of the basal ganglia to rule out hypoparathyroidism

References

1. Rizvi I, Ansari NA, Beg M. Widespread intracranial calcification, seizures, and extrapyramidal manifestations in a case of hypoparathyroidism. *N Am J Med Sci* 2012; 4:369-372.
2. Verulashvili IV, Glonti LS, Miminoshvili DK, *et al*. Basal ganglia calcification: Clinical manifestations and diagnostic evaluation. *Georgian Med News* 2006; 140:39-43.
3. Basak R. A case report of basal ganglia calcification: A rare finding of hypoparathyroidism. *Oman Med J* 2009; 24:220-222.
4. Abe S, Tojo K, Ichida K, *et al*. A rare case of idiopathic hypoparathyroidism with varied neurological manifestations. *Intern Med* 1996;35:129-134
5. Lewis, Medical Surgical nursing, II South Indian Edition, Elsivier, India. pp- 1392 - 1303.
6. B.Venkatesan, text book of Medical surgical Nursing, I Edition, EMMESS Medical Publication, Bangalore, pp- 430 - 432.

How to cite this article:

Pauline Sheela Priya S (2017) 'Basal Ganglia Calcification A Rare Finding Of Hypoparathyroidism', *International Journal of Current Advanced Research,* 06(06), pp. 4204-4205.DOI: http://dx.doi.org/10.24327/ijcar.2017.4205.0463

****