Research Paper

Volume : 3 | Issue : 2 | February 2014 • ISSN No 2277 - 8179

A Case Report of Basal Ganglia Calcification - A Rare Finding of Hypoparathyroidism

Medical Science

KEYWORDS :

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ABSTRACT

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 Pseudohypoparathyroidism are the most common causes of pathological basal ganglia calcification. Besides tetany and seizures this condition is presented by parkinsonism and dementia. 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

INTRODUCTION:

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[1]. Idiopathic Hypoparathyroidism is diagnosed when all the possible causes of hypoparathyrodism are ruled out. Hypoparathyroidism by any cause is well known to cause basal ganglia calcification in most of the patients[2]. It is also well known that extensive intracranial calcification caused by hypoparathyrodism is rare. This report presents a case of idiopathic hypoparathyroidism which presented with extensive intracranial calcification.

CASE REPORT:

A 45-year-old hindu male labourer presented in the emergency department of Civil Hospital Ahmedabad, with generalized tonic-clonic seizure followed by altered sensorium. His general examination showed pulse 90 per min, Blood pressure 126/80 mmHg and pallor without icterus. Chvostek's and Trousseau's signs were positive. He had no dysmorphic features.

Systemic examination showed exaggerated DTR including jaw jerk, plantar were bilateral extensor, he could localize pain but due to altered sensorium other higher functions could not be assessed.Fundoscopy was normal;there were no KF rings seen nor was there presence of band keratopathy.

Once the patient retained consciousness he had both cerebellar as well as extra-pyramidal signs.

His laboratory investigations are presented in Table 1.

MDCT (16 Slice) scan of brain showed extensive calcification noted at subcortical and deep white matter, basal ganglia and cerebellar hemispheres.

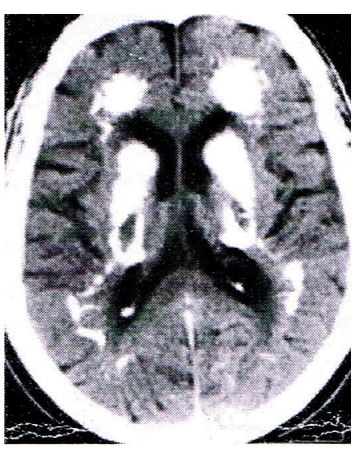

FIGURE 1:

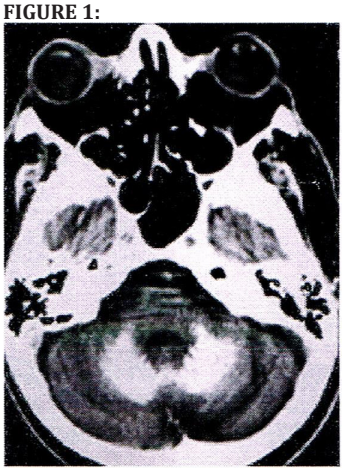

FIGURE 2:

With these investigations and after ruling out of other causes of

hypoparathyrodism the possibility of idiopathic hypoparathyrodism was suggested.

The patient was treated with intravenous calcium gluconate, anti-convulsants (valproate), acetazolamide, glycerol, oral cholecalciferol (60,000units/week).

The patient was discharged with oral calcium carbonate, cholecalciferol and valproate as anticonvulsant.

The patient returned for follow up after a week - was in much better position and able to carry out daily activities.

DISCUSSION:

Hypoparathyroidism is an endocrine disorder caused as a result of congenital disorders, iatrogenic causes, infiltration of the parathyroid glands, suppression of parathyroid function, or idiopathic mechanisms[1]. In these cases either there will be apparent deficiency of PTH secretion or end-organ failure.Idiopathic hypoparathyroidism is an uncommon condition of unknown etiology. Acquired and congenital hypoparathyroidism would have either normal or undetectable PTH levels with hypocalcaemia whereas in idiopathic hypoparathyroidism both calcium and PTH levels are low[2]. In pseudo hypoparathyroidism PTH levels will be high with hypocaicaemia. In pseudopseudohypoparathyroidism with increased PTH level both serum calcium and phosphorus levels are normal[2]. Prevalence of hypoparathyroidism is equal in men and women and occurs in all age groups. A literature review of the clinical presentations of basal ganglia calcification revealed that there are diverse presentations, the most common including seizures, mental deterioration, and disorders of cerebellar or extra-pyramidal function. Movement disorders, chorea, or parkinsonism are present in 20 - 30% of patients with basal ganglia calcification, while some patients are a symptomatic.[3]

Radiologically hypoparathyroidism causes calcification most often in bilateral basal gangiia4. The most common site is often globus pallidus5. Calcification can also occur in the cerebellum, sub cortical white matter, corona radiata and the thalamus.

The immediate treatment for all types is calcium supplement with supplementation of PTH in cases of acquired hypoparathyroidism. Also one has to treat symptomatically for the seizures and other changes. This disease has a good prognosis if detected early and treated13. Since adequate treatment of hypoparathyroidism may lead to marked clinical improvement, determination of serum calcium, phosphorus, and parathyreoid hormone is mandatory in all individuals with calcification of the basal ganglia to rule out hypoparathyroidism.

TABLE- 1 : IMPORTANT LABORATORY INVESTIGATIONS.

TEST		DATE
	24/03/2013	25/03/2013
Hemoglobin (g/dl)	10.7	10.4
Serum creatinine (mg/dl)	0.6	0.5
Serum Sodium (mEq/l)	137	136
Serum Potassium (mEq/l)	3.60	3.40
Serum Billirubin (T/D/l) (mg/dl)	0.9/0.4/0.5	0.8/0.4/0.4
Serum SGPT (IU/l)	32	43
ESR	11mm /1st hr	
SERUM CALCIUM (mg/dl)	5.4	
SERUM PTH (pg/dl)		5.31
SERUM PHOSPHORUS (mg/dl)		4.55
SERUM MAGNESIUM (mg/dl)		2.2
SERUM ALBUMIN		3.8
Vitamin B-12 (pg/ml)	250	
ANA, RA, VDRL, HIV, HBsAG	Negative	
2D ECHO	Normal	

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