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Extensive Intracranial Calcification

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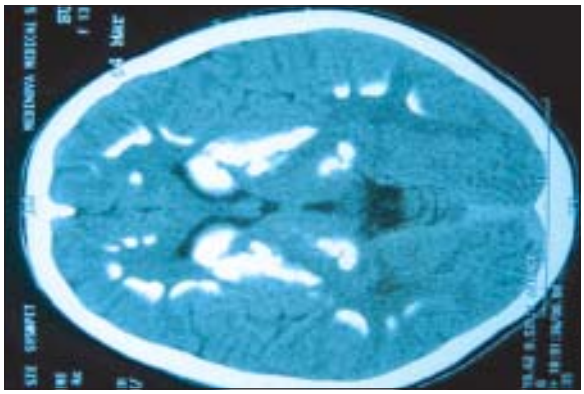

Fig.-1: *Non-contrast enhanced CT scan showing bilaterally symmetrical hyperdense lesions with CT value of calcification in subcortical white matter of bilateral frontal ,parietal and temporal lobes.*

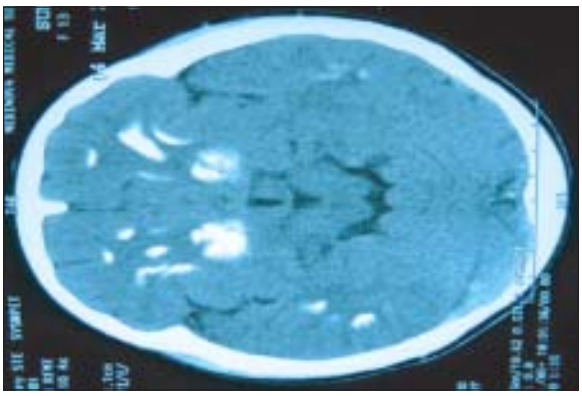

Fig.-2: *Non-contrast enhanced CT scan showing bilaterally symmetrical hyperdense lesions with CT value of calcification in subcortical white matter of bilateral frontal and parietal lobes*

A 13 -year- old girl was admitted with a history of generalized seizures since the age of 4 years and abnormal involuntary movements right leg for 6 months prior to admission. Since then she was diagnosed as a case of epilepsy and was treated with tablet sodium valporate . She born by normal home NVD. Since birth, all her mile stone of development was normal except at the age of 8 month, she developed visual impairment and was treated as corneal ulcer for 4 years. During examination, Trousseau's sign was present, tone of muscle of 4 limbs were increased, hyperreflexia , planter bilaterally extensor and ataxic gaits. Her both cornea were hazy with corneal scar. Investigations revealed hypocalcaemia, hyper-phosphataemia but normal alkaline phosphatase and renal function. A plain computed tomographic (CT) head scan demonstrated extensive bilateral calcification in the region of basal ganglia and cerebral cortex (Figures 1 and 2).

Investigations of the patient

Parameter	Patient
Serum Calcium	3mg/dl. (9-11mgdl)
Serum. Inorganic Phosphate (PO4)-	9.8mg/dl(2.5-5mg/dl)
PTH-	3.4pg/ml(11-67pg/ml)
Serum Magnesium-	2mg/dl.(1.9-2.5mgdl)
Alkaline Phosphate	115 U/L(80-125U/L)
Serum albumin	3.8gm/dl(3.5-5gm/dl)
Serum Creatinine-	0.65mg/dl

The history, clinical examination and investigations were indicative of hypoparathyroidism leading to extensive calcification of the brain.

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.[1] 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. Infections (toxoplasmosis, rubella, cytomegalovirus, cysticercosis, AIDS) give multiple and asymmetric intracranial calcification. Inherited and neuro-degenerative diseases cause symmetrical, bilateral basal

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ganglia calcification which is not related to metabolic disorders.[2] It is also well known that extensive intracranial calcification caused by hypoparathyrodism is rare. These images show extensive intracranial calcification in hypoparathyroidism .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.[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 asymptomatic.[3] The emergence of CT has led to the finding that sporadic calcification is the most common form, present in up to 1.5% of all brain scans.[4]These patients demonstrate extensive calcification bilaterally in basal ganglia, proposed to result from a degenerative vascular process in the extrapyramidal system, initiated possibly by deposition of calcium crystals. This calcification is rarely symptomatic and may not be visualized on plain X-rays of the skull, although readily appreciable in a CT scan. This is possibly because of the thin layering of calcium along blood vessels in basal ganglia.[5] Calcification may rarely extend intracranially beyond the basal ganglia, especially to the cerebellum and frontal lobes of the cortex.[6,7]

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 Basal ganglia calcification is rare before forty years. If it occurs before forty years of age, it should be considered as pathological unless proved otherwise.

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