

# Extensive Intra-Cerebral Calcification and Uncontrolled Seizures due to Hypo-Parathyroidism

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## ABSTRACT

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Calcifications in brain parenchyma is a routine CT Scan curiosity.<sup>1,2,3,4</sup> It can occur in several clinical conditions and hypoparathyroidism is one.<sup>4,5,6</sup> We are reporting a case of hypoparathyroidism following total thyroidectomy manifesting as extensive intracerebral calcifications and uncontrolled seizures. The role of routine calcium estimation in seizure disorders is highlighted. A low Ca and high P in the absence of renal failure and massive tissue destruction invariably mean hypoparathyroidism. Selective involvement in Basal Ganglia and other areas are due to high neurotransmitter sensitivity and metabolic rate. Calcium is merely laid upon as fundamental organic matrix from mucopolysaccharides and glycoprotein. This type of calcification contains hydroxyapatite different from usual calcifications. DPH by their effect on Vit.D and calcium metabolism can aggravate hypocalcaemia and seizure; may impair 25-hydroxylation by inactivating microsomal enzymes in liver; have direct negative effect on intestinal calcium transport and bone metabolism. DPH and Pheno. may delay the diagnosis by suppressing the peripheral manifestations of neuro-muscular irritability in hypo para-thyroidism. Sodium valproate is the preferred anti-convulsant.

**Keywords:** Calcification, Intracerebral, Seizures, Hypoparathyroidism.

\*See End Note for complete author details

## MATERIALS AND METHODS

Mrs.S aged 60, admitted with recurrent seizures of tonic clonic type, associated with carpo pedal spasm during inter-ictal period. She gave history of total thyroidectomy 15 years ago without adequate and persistent replacement. Seizure was tonic and clonic generalized with status epilepticus. She was managed with IV Dilantin and Midazolam.

## INVESTIGATIONS

Urinalysis, erythrocyte sedimentation rate, and complete blood count were normal. Results of blood chemical tests were also not remarkable: Creatinine 0.9 mg/dl; Urea nitrogen 22 mg/dl; RBS- 126 mg/dl, Calcium 6.4 mg/dl; Inorganic phosphorus 7.1 mg/dl; Alk.Phos- 174 IU, Magnesium 1.9 mEq/L; Iron 146 µg/dl; Copper 78 µg/dl; Ceruloplasmin 29 mg/dl; T3-51ng (70-200), T4 -1.9µg (4-11), TSH-11.06 µU/dl (0.5-4.5), Parathyroid hormone-3.6pg/dl ECG-QTC prolongation, EEG- Abnormal, CT-scan showed extensive calcification involving Corpus striatum, Basal Ganglia, Putamen, Thalamus, Cerebellum and Cortex. (Figure 1).

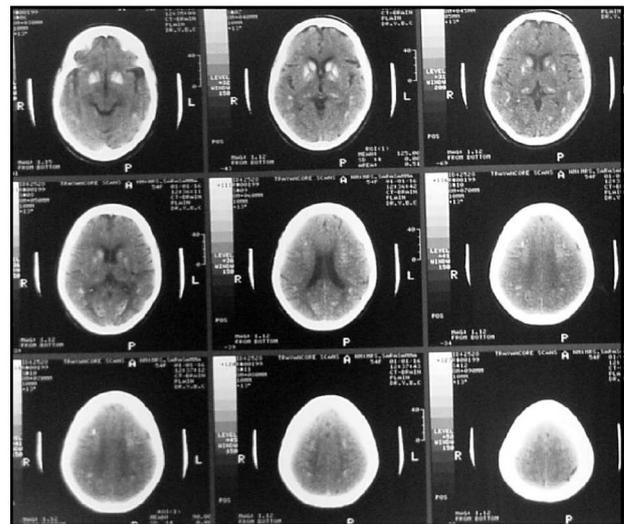


Figure 1. CT Scan Head showing extensive calcification

## DIAGNOSIS

Hypothyroidism, Hypoparathyroidism, Extensive Brain Calcifications, Uncontrolled seizures. She was managed with Na.Valproate, high dose calcitriol and calcium.

## Causes of Intra Cerebral Calcification

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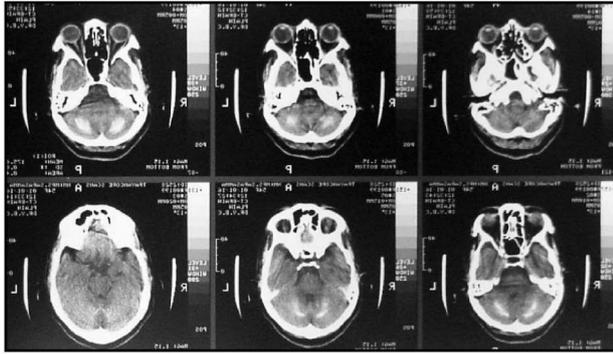


Figure 2. CT Scan Head showing extensive Cerebellar calcification

Idiopathic- Commonest, Familial, Hypoparathyroidism, Pseudo hypo parathyroidism-Albright's, Fahr's syndrome-Idiopathic Familial CV Ferro calcinosis, Cockayne's syndrome, Sturge Weber syndrome, CO poisoning, Lead poisoning, Toxoplasmosis, Torchs, Neurocysticercosis, Mineralizing microangiopathy, Sec. hyper parathyroidism, Mitochondrial cytopathy, Osteopetrosis, RTA, AIDS, Childhood meningo encephalitis

### Causes of Hypoparathyroidism

#### Hereditary

Multiple end organ endocrinopathy, Defective development of PT and Thymus-Di George, Autoimmune polyglandular deficiency and as isolated entity.

#### Acquired

Surgical removal,<sup>8,9,10</sup> Cutting of vascular supply, Radiation, Hypomagnesemia, Hemosiderosis, Haemochromatosis,

PTH ineffective- CRF- Vit-D deficiency, Defective Vit-D metabolism, Vit-D ineffectiveness, Pseudo hypoparathyroidism

Functional classification of hypocalcemia

PTH Absent- Hereditary, Acquired, Hypo Mg, PTH Ineffective. -CRF, Active Vit-D lacking-decreased intake, defective metabolism due to anticonvulsant, Vit-D dependent rickets type-1,

Active Vit-D ineffective- Intestinal malabsorption, Vit D dependent Rickets Type 2. Pseudo hypo parathyroidism.

PTH overwhelmed- Severe acute hyperphosphatemia, Tumor lysis, acute renal failure. Rhabdomyolysis, Osteitis fibrosa after parathyroidectomy.

Critically ill patients, severe sepsis, burns, ARF, transfusions with citrated blood, hypoalbuminemia cause

decrease in ionized calcium. Alkalosis by increasing Ca binding to protein decrease ionized Ca. Protamine, Glucagon, and Heparin can cause transient hypocalcemia; acute pancreatitis often causes hypocalcemia, which determines the severity.

Chronic hypocalcemia is usually symptomatic and requires treatment;

Neuromuscular and neurological manifestations are muscle spasms, carpopedal spasms, facial grimacing, laryngeal spasms, convulsions, respiratory arrest, increased ICT, papilloedema, mental changes, irritability, psychosis, OTC prolongation, digitalis ineffectiveness, intestinal cramps, malabsorption. Chvostek or Trousseau's sign are used to confirm latent tetany.

### DISCUSSION

Delacour- first described this ICC in 1850, and Lowenthal- described association with hypo- calcemia and hypoparathyroidism. Calcification of the brain is found in approximately 0.3 to 1.2% of subjects undergoing routine CT examination.<sup>1,2,3</sup> Brain calcification exists mainly in the bilateral basal ganglia and less frequently in the cerebellar dentate nucleus.<sup>4,5</sup> The frequency of basal ganglia calcification increases with advancing age, but most patients have no neurologic symptoms.<sup>4,5,6,7</sup> although many disorders, such as hypoparathyroidism, pseudohypoparathyroidism, Down's syndrome, and mitochondrial encephalopathy are associated with brain calcification,<sup>2</sup> idiopathic cases are common. The majority of cases of brain calcification occur sporadically. Various names have been given- Striatal vascular calcification, Bil. Idiopathic non- arteriosclerotic vascular calcification, cerebral calcinosis, Sriato-palido dentate calcifications, Calcinosis nucleorum cerebri, Basal ganglia calcifications. PTH is responsible for minute to minute regulation of plasma calcium concentration and therefore the occurrence of hypocalcemia must mean a failure of the homeostatic action of PTH and failure can occur due to hereditary or acquired parathyroid gland failure, ineffectiveness etc.

This case was presented mainly to highlight the role of routine calcium estimation in seizure disorders. Hypomagnesaemia is associated with both deficient PTH release and impaired responsiveness to the hormone. Selective involvement in Basal Ganglia and other areas are due to high neurotransmitter sensitivity and metabolic rate.<sup>7,8</sup> Mechanism of calcification is due to dystrophic or metastatic, increased phosphate and P-Ca ratio in the setting of hypoxia. Calcium is

merely laid upon as fundamental organic matrix from muco polysaccharides and glycoprotein. This type of calcification contains hydroxyapatite different from usual calcifications seen in kidney and pancreas. Diphenylhydantoin (DPH) by their effect on Vit.D and calcium metabolism, can aggravate hypocalcaemia and seizure; may impair 25- hydroxylation by inactivating microsomal enzymes in liver; anti convulsants have direct negative effect on intestinal calcium transport and bone metabolism. DPH and Phenobarbitone may delay the diagnosis by suppressing the peripheral manifestations of neuromuscular irritability in hypo parathyroidism. Sodium valproate is the preferred anti-convulsant.

## TREATMENT

Normal dose of Vit-D is 200 u /day. In hypoparathyroidism the requirement is 20000 to 40000u/ day but conversion to 1-25 (OH) D is difficult. When withdrawn it takes weeks to get eliminated. High dose elemental calcium is required to maintain the calcium balance. Calcitriol is needed in doses of 1 to 1.5 microgram / day. Correction of hypo Mg is mandatory.

## CONCLUSION

A case of Primary hypoparathyroidism following total thyroidectomy manifesting as extensive intracerebral calcifications and uncontrolled seizures. The role of routine calcium estimation in seizure disorders is stressed. Relevant literature is reviewed.

## END NOTE

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**Conflict of Interest:** None declared

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