DELAYED ONSET OF POSTTHYROIDECTOMY HYPOPARATHYROIDISM

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Introduction

Hypocalcemia may follow thyroid and parathyroid surgery. It is more common with total thyroidectomy than hemithyroidectomy [1]. Onset of hypoparathyroidism can be delayed after surgery [2]. Diagnosis may be missed for years [3] in an epileptic patient who is on antiepileptic drugs, which masks hypocalcemic symptoms [4]. Here we report a case of postsurgical hypoparathyroidism associated with hypothyroidism who presented with seizure with altered consciousness following total thyroidectomy, in whom diagnosis was delayed for 19 years.

Case report

SSN, 42 year old male, was found unconscious following generalised tonic-clonic seizure at railway station. He was admitted to a civil hospital and subsequently transferred to this centre with diagnosis of renal failure (hemoglobin 10.5 gm/dl, blood urea 90 mg/dl, serum creatinine 3.3 mg/dl with normal serum sodium and potassium). History from relatives revealed that he was a case of

seizure for last 14 years on irregular treatment and had undergone neck operation in the past, details of which were not available. He was drowsy, confused, dehydrated, responded to painful stimuli and looked pale. There was no icterus, cyanosis, clubbing or edema. Skin was dry and rough. There was alopecia and seborrhoeic dermatitis of scalp. Neck revealed well healed supple transverse scar. Pulse was 60/min regular, blood pressure 104/80 mm of Hg and respiratory rate was 16/min. He was afebrile. Pupils were round, regular, reacting and equal on both sides along with normal fundus. Motor system examination revealed generalised rigidity with hyporeflexia and plantar flexor. He was moving all limbs in bed. Chovstek's and Trousseau's sign were strongly positive (Grade 4). A diagnosis of hypocalcemic seizure due to hypoparathyroidism with hypothyroidism was considered. He was treated with intravenous calcium bolus and infusion, and thyroxin replacement therapy. Investigation showed hemoglobin 8.9 gm/dl, total leukocyte count 8,600/cumm (4000-11000), blood glucose random 104 mg/dl (60-100), blood urea 55 mg/dl (20-40), serum creatinine 1.6 mg/dl (0.1-1.0), serum uric acid 2.9 mg/dl, serum sodium 140 meq/L (135-150), serum potassium 4.1 mg/dl (3.5-5.0), serum calcium 5.2 mg/dl (9.0-11.0), serum phosphate 6.1 mg/dl (2.5-4.5), serum cholesterol 282 mg/dl (160-250). Urine examination was normal. Urinary spot sodium was 17 meq/L. Liver function tests were normal except elevated ALT (142 IU/L) and AST (396 IU/L). Electrocardiogram showed prolonged Q-T inter-

Fig. 1 : Extensive intracranial calcification of cerebral white matter

Fig. 2 : Extensive intracranial calcification of basal ganglia

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val (0.53 sec). CT scan brain revealed extensive intracranial calcification of basal ganglia, cerebellum and cerebral white matter (Figs. 1,2).

Patient responded remarkably with treatment. Detailed history revealed that he had undergone total thyroidectomy for papillary carcinoma of thyroid in 1978. He was prescribed thyroxin replacement therapy, which he took for two years and stopped without any medical advice. In 1983, he developed generalised tonic-clonic seizure and was treated with phenobarbitone 180 mg daily in divided doses, which controlled the seizure partially. He also had symptoms suggestive of hypothyroidism. Slit lamp examination showed early cataract. Serum triiodothyronine, thyroxin and thyroid stimulating hormone levels were 0.1 ng/ml (80-210), 0.77 µg/dl (4.5-13.5) and 88.9 mIU/L (0.5-6.5) respectively (Kit supplied by BHARC, Bombay). Echocardiography examination detected minimal pericardial effusion. Electroencephalogram was normal. Parathyroid hormone level was less than 1.0 pg/ml (12-72). (Automated Chemiluminescent Immunoassay System, DPC-USA). 24-hour urine calcium and phosphate were 102 mg and 207 mg respectively.

Discussion

Our case presented with seizure, altered consciousness, pallor, seborrheic dermatitis, hyporeflexia, rigidity, positive Chovstek's and Trousseau's sign with hypocalcemia, hyperphosphatemia, hypercalciuria, low PTH indicating hypoparathyroidism. Thyroid function test was consistent with primary hypothyroidism.

Hypothyroidism can be mistaken for renal failure [5] due to presence of pallor, dry skin and elevated blood urea and serum creatinine. In our case deranged blood urea and serum creatinine can be explained by associated dehydration leading to prerenal azotemia, diagnosis of which is supported by bland urinary sediments, low urinary spot sodium and response to intravenous fluid therapy [6]. Elevated liver cell enzymes are known in hypothyroidism [5]. Hypothyroid coma was considered as differential diagnosis, however absence of hypothermia, hypoventilation, bradycardia and hyponatremia excluded it.

Postsurgical hypoparathyroidism after total thyroidectomy occurs in about 10-33% of patients [7]. Convulsions have been reported in 70-76% of patients with idiopathic hypoparathyroidism, however in surgical hypoparathyroidism the occurrence of seizure ranged from less than 5% [8] to nearly 35% [9]. In many epileptic patients who were under antiepileptic treatment, diagnosis of hypoparathyroidism was established after many years and response to drugs varied from failure to poor control [3]. The presence of recurrent seizure in our patient lead to initiation of therapy with anticonvulsant drugs, which is known to delay the recognition of hypocalcemia as a cause of seizure by masking signs of tetany [4]. Forgetfulness, a common feature of associated hypothyroidism, in our case might have contributed to delayed diagnosis. Central

nervous system manifestations in postoperative hypoparathyroidism usually occur early but rarely many years after the surgery [2], with longest duration reported as 33 years [9]. In our case it was delayed for 5 years after surgery. Hypocalcemia that is overlooked results in its deleterious effect (as in our case) such as cataract, cerebral calcifications and trophic changes in skin. Extraparathyroidal features like progressive rigidity, tremors, propulsive gait, cogwheeling, choreiform and athetotic movement has been reported in long standing post surgical hypoparathyroidism [10] with or without basal ganglia calcification. In our case only feature noticed was generalised rigidity, which has improved after treatment.

Hypocalcemia, by increasing the excitability of central cortical centres, is most likely the principle cause of convulsion and altered consciousness in our case, though associated hypothyroidism may also have contributed to it. Seizure patterns reported in hypoparathyroidism, are usually associated with tetany, however old age, alkalosis and anticonvulsant drugs can mask features of tetany [10]. High index of suspicion and simple investigations can establish diagnosis in a given patient of seizure.

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