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Case Report

Post- Surgical Hypoparathyroidism-Challenges in Management.

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Abstract

Post-surgical hypoparathyroidism is not frequently reported in Africa. The communication reports seizure disorder as a rare complication of post total thyroidectomy in a Nigerian.

A 49-year-old woman who presented with 18 days history of multiple tonic-clonic seizures preceded by tingling sensation in the fingers and around the mouth noticed three weeks post total thyroidectomy. She had a healing scar on her neck with Chvostek's and Trousseau's sign positivity. There was presence of transmitted breathe sounds. Blood chemistry confirmed severe hypocalcemia, hypomagnesemia and hypoparathyroidism. There was leukocytosis with neutrophilia and thyroid profile was in keeping with hypothyroidism.The findings were compatible with Hypocalcemia seizures secondary to post-surgical hypoparathyroidism following total thyroidectomy complicated by aspiration pneumonitis. She was admitted to the High Dependency Unit (HDU) and commenced on intravenous infusion of calcium gluconate, magnesium sulphate, anti seizures medications and discharged 37 days post admission.

Keywords: Hypocalcemia, Hypoparathyroidism, Seizures

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Introduction

Post-surgical hypoparathyroidism (PH) and hypocalcemia was first described as postoperative complications of thyroid surgery from the days of Kocher and Bilroth.[1]It can also occur as a sequalae of surgical procedures involving the neck ranging from total thyroidectomy, laryngectomy among others due to direct trauma of the parathyroid glands, devascularization, or their accidental removal.[2,3]with presentation that can be transient or permanent. Post-surgical hypoparathyroidism remains a major complication with high-risk morbidity and mortality if not promptly identified and managed.

The prevalence of transient hypoparathyroidism post thyroid surgery is about 6.9% and [4-7] while permanent hypoparathyroidism varies between 0.4% and 33%.[7-9]Studies have shown the risk of acute symptomatic hypoparathyroidism increases in patients with a serum postoperative PTH level less than 15 pmol/L while serum PTH level of less than 10 pmol/L measured at 4 h to 24 hours after surgery indicates an impaired parathyroid metabolism with increasing risk of permanent hypoparathyroidism.[10,11] A study by Galy-Bernadoy C et al. reported that postoperative 4–6-h serum PTH level of less than 7 ng/L has a 100% positive predictive value of permanent hypoparathyroidism.[12]

The exact occurrence of post-surgical hypoparathyroidism in Nigeria is unknown to the best of my knowledge. We report a case of hypocalcemic seizures in a woman who recently had total thyroidectomy who was found to have PH complicating thyroidectomy.

Case Report

Mrs A.O, 49-year-old Nigerian was referred to the Emergency on account of recurrent seizures following total thyroidectomy three weeks prior to presentation.

She presented with 18 days history of multiple generalized tonic clonic seizures. Each episode lasting about two to five minutes with regain of consciousness after each episode. The symptoms that preceded the onset of seizures were tingling sensation in the fingers and around the mouth with involuntary painful spasms of the fingers associated occasional lip smacking. She had about three to six episodes of seizures in 24 hours. There was no accompanying unilateral limb weakness, no headache or any focal neurological deficit. Prior to the surgery, she was managed for Graves' disease with Graves Ophthalmopathy. Details as regards her thyroid profile, serum calcium, magnesium prior to presentation were unknown.

On examination, there was the presence of healing scar on her neck, febrile with temperature of 38-degree Celsius. There were no signs of meningeal irritation with normal and reactive pupils. Other clinical findings were bilateral proptosis, Chvostek's and Trousseau's signs with presence of transmitted breath sounds. Her pulse and blood pressure were normal, although she is a known hypertensive diagnosed eight years ago on oral Amlodipine 10mg daily. There was no other known chronic medical condition.

Her investigation results confirmed severe hypocalcemia with hypomagnesemia and hypoparathyroidism (see table 1). The complete blood count showed leukocytosis with neutrophilia while her thyroid profile was in keeping with Hypothyroidism. The diagnosis of severe hypocalcemia was based on the measurement of serum calcium (albumin-adjusted calculated calcium or ionized calcium). Other laboratory investigations were all within normal limits (see table 1). Her Brain MRI result was essentially normal. The findings were compatible with Hypocalcemia seizures secondary to post-surgical hypoparathyroidism following total thyroidectomy complicated by aspiration pneumonitis.

Results of investigations

Table 1: Serum Electrolyte, urea, creatinine and Serum intact parathyroid hormone

Parameters	Result (Day 1)	Normal range	Remarks
Sodium	135.1	135-155	Normal
Potassium	4.98	3.0-5.3	Normal
Chloride	96.9	96-106	Normal
Bicarbonate	26	23-34	Normal
Phosphorus (mg/dl)	3.27	2.7-4.5	Normal
Total Calcium (mmol/L)	1.24	2.2-2.9	Low
Albumin (g/L)	30.7	35-50	Low
Corrected Calcium (mmol/L)	0.63	1.1-1.4	Low
Magnesium(mg/dl)	0.51	1.7-2.2	Low
Urea (mg/dl)	32	10-55	Normal
Creatinine (mg/dl)	0.6	0.5-0.9	Normal
Intact Parathyroid hormone (pg/mL)	5.11	15-68.3	Critically Low

Table 1 shows the patient's results for serum electrolytes with low calcium and magnesium. The serum intact parathyroid hormone is critically low.

Table 2: Serum thyroid function, glucose, lipid profile alkaline phosphatase and urinary calcium results

Analyte (units)	Result Day 1	Normal range	Remarks
TSH (uIU/ml)	9.40	0.35-4.94	High
Free T3 (pmol/L)	<0.7	2.8-7.1	Low
Free T4 (pmol/L)	9.40	7.2-16.4	Normal
Alkaline phosphate (U/L)	68	65-108	Normal
24-hour urinary calcium	0.9	2.5-7.5	Low

(mmol/L)			
Random plasma glucose	114mg/dl	<140mg/dl	Normal
Total cholesterol	176mg/dl	<200mg/dl	Normal
Low Density Lipoprotein (LDL)	98mg/dl	<130mg/dl	Normal
High Density Lipoprotein (HDL)	54mg/dl	>50mg/dl	Normal
Triglyceride	80mg/dl	<150mg/dl	Normal

Table 2 shows the patient's results for glucose, lipid profile and thyroid function. The thyroid function test showed Hypothyroidism.

She was immediately transferred to the High Dependency Unit (HDU) and commenced on intravenous infusion of 10mls of 10% calcium gluconate to run over 15 minutes, this was repeated. Due to the continuous seizures, she was commenced on calcium gluconate infusion at 1.5mmol/kg/hour to run over 8 to 10 hours with commencement of oral calcium carbonate at 1250mg daily and 12 hourly monitoring of serum calcium levels. Also, oral calcitriol was commenced at 0.5ug daily and the doses were progressively increased until she attained normal low levels of serum calcium.

Additional treatment modalities were antiseizures medications with sodium valproate and levetiracetam at 1g twice daily, antibiotics, intravenous ceftriaxone and paracetamol infusion. Hypomagnesemia was corrected with intravenous 4g of magnesium sulphate and oral magnesium while serially monitoring serum calcium and magnesium levels. She was also placed on thyroid hormone replacement with levothyroxine 100mcg daily an hour before breakfast and other oral medications.

Her management was particularly challenging in optimizing serum calcium and magnesium levels after cessation of seizures. The oral calcium was increased to 3.1g daily while oral calcitriol was increased to 2ug daily before she could achieve a lower limit of normal serum calcium levels.

The aspects of management we focused on in our index patient were on the correction of hypocalcemia and hypomagnesemia, thyroid hormonal replacement, control of seizures and management of sepsis. Due to the association of hypocalcemia with depression, she was reviewed by the Neuropsychiatric team. Her management was multi-disciplinary with involvement of Neuropsychiatric, cardiologist, neurologist teams. Multidisciplinary care with involvement of cardiologist and neurologist teams.

She was educated and counselled on the implications of her diagnosis and need for follow up care in the Endocrine clinic. She was also counselled on need for regular monitoring of her serum electrolytes and parathyroid hormone levels to prevent Iatrogenic hypercalcemia and Nephrocalcinosis.

Discussion

Acute symptomatic hypocalcemia such as hypocalcemia seizures in the index patient is an endocrine emergency, if not proper managed can be fatal.

In a prospective study at the University of Benin Teaching Hospital (UBTH) of 108 patients who had thyroidectomy between 1986 to 1990's , this study showed that one patient had overt hypoparathyroidism while six patients had transient hypoparathyroidism with an overall incidence of 6.5%.[13]This complication is commoner during total thyroidectomy as in our index case.[14]

The risk factors associated with post-surgical hypoparathyroidism are female sex, surgery for Grave's disease, older age, bilateral neck surgery, thyroid malignancy and retrosternal and huge goiter.[15]Serum calcium level preoperatively is a risk factor for the development of transient hypocalcemia.[16] Like in the index patient, the serum calcium and magnesium levels preoperatively were unknown. Post-surgical hypoparathyroidism is commoner in females compared with males as our index patient was a female.

They can present both asymptomatically or with symptoms suggestive of hypocalcemia such as numbness or tingling sensation in the face and extremities, laryngospasm, tetany and in severe cases seizures as in our index case. They could also present with neuropsychiatric symptoms of delirium, confusion and depression.

The serum calcium level is mainly regulated by the action of parathyroid hormone and vitamin D on the kidneys, bones, and gastrointestinal tract.The diagnosis is based on the measurement of serum calcium (albumin-adjusted calculated calcium or ionized calcium), phosphate and PTH levels less than or equal to 15pg/L within 24 hours post-surgery.[17]There are various factors that affect the ratio of ionized calcium to bound calcium, being mainly the serum albumin concentration.[18] The ionised serum calcium is the biologically active calcium, hence the calculation of albumin-adjusted calculated calcium is crucial in the diagnosis of hypocalcemia.[18]The diagnosis is mainly biochemical with or without clinical symptoms hypocalcemia.[17]Post-surgical hypoparathyroidism could be transient or permanent. The diagnosis of permanent PH is usually made after six months of persistent low PTH levels.

The management of hypocalcemic seizures requires acute intravenous administration of calcium dextrose infusion.[19] The target is to raise the ionized serum calcium concentration into the low normal range (approximately 1.0 mmol/L) and also to control the patient's symptoms. Calcium gluconate is preferred over calcium chloride because of risk of hyperchloremic metabolic acidosis and preferably given via a central vein.[19]

The prognosis of PH is usually good if promptly managed. A regular follow up monitoring of serum and urinary calcium including the renal function test is essential.[19] Our index patient was reviewed by team of specialists including Neuropsychiatric, Neurologist and Cardiologist are referred to see the ophthalmologist. She was discharged 37 days after admission to see at follow up clinic with results of thyroid profile, serum calcium, albumin, magnesium, urea and creatinine, parathyroid hormone and 24-hour urinary calcium.

Conclusion

Good clinical history and laboratory back up with high index of suspicion is needed to avoid a misdiagnosis. Hypocalcemia has deleterious effect on the neurological, muscular, cognitive and cardiac systems. It can present as acute symptoms as in the index patient or as asymptomatic hypocalcemia. Follow up care and investigations are needed to avoid Iatrogenic hypercalcemia and Nephrocalcinosis.

Authors contributions

All the authors were involved in conceptualization, literature search and writing of the manuscript.

Conflict of interest

There is no conflict of interest

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