OCULAR FEATURES OF HYPOCALCAEMIA IN A 35-YEAR OLD WOMAN - A Case Report

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ABSTRACT
The case of a 35-year-old teacher who developed persistent hypocalcaemia following subtotal thyroidectomy for simple multinodular goitre is presented. She developed carpopedal spasm with positive Trousseau's and Chvostek's signs 10 days after surgery and was found with a calcium level of 1.94mmol/l. Her serum calcium has been persistently below normal levels since then. She developed hypermetropia and early onset of presbyopia 6 weeks after surgery; by this time her lens was clear on examination. She had enjoyed good vision prior to the thyroidectomy surgery. A year later, she had developed a fully mature bilateral presenile cataract. The case illustrates an uncommon cause of rapidly progressive bilateral mature cataract in Nigeria and the difficulty encountered in managing a patient with persistent hypocalcaemia in our environment.

Key words: permanent hypocalcaemia, post thyroidectomy, refractive error, mature bilateral presenile cataract

INTRODUCTION

Hypocalcaemia is one of the metabolic causes of cataract. Other causes include diabetes mellitus, galactosaemia and Wilson's disease.[1, 2] Hypocalcaemia does occur post thyroidectomy and is not uncommon.[3, 4] The reported incidence is widely variable and ranges between 20% and 80%. The majority of post thyroidectomy hypocalcaemia are asymptomatic or transiently symptomatic, requiring no treatment or treatment for only a few days. Fortunately, the incidence of permanent hypocalcaemia is low (0-9%).[3,4,5,6] Various reasons are advanced for hypocalcaemia following thyroidectomy. These include: inadvertent surgical excision of, or damage to the parathyroid glands; release of calcitonin during surgery, and hungry bone syndrome due to post-operative reversal of thyrotoxic osteodystrophy.[7, 8] Hypocalcaemia often causes multicolored crystals or small, discrete, white flecks to develop in the anterior or posterior cortex beneath the lens capsule, usually separated from it by a zone of clear lens.[1, 2] In contrast to true diabetic cataracts, those associated with tetany are said to seldom progress to maturity.[1, 2] Normally a high calcium gradient of 30mM (intracellular) and 2uM(extracellular) is maintained by calcium pump (Ca 2+ -ATPase).[9] This calcium homeostasis is critical to lens metabolism and clearance; the loss of this homeostasis can be highly disruptive to the lens.[10] In hypocalcaemia, the loss in homeostasis results from membrane damage as shown by a study done in rats with induced hypocalcaemia.[11] As shown in the same study, there was no significant change in the level or activity of Ca 2+-ATPase.[11]

Other ocular features of hypocalcaemia, apart from cataracts, include band keratopathy, iridocyclitis and papilloedema [12,13] The general features of hypocalcaemia are paraesthesia of distal extremities, and muscle cramping leading to carpopedal spasm or tetany from increased neuromuscular irritability. Laryngospasm or bronchospasm and seizures may also occur. Other features include fatigue, irritability, and personality disturbance. There may also be increased bone mass with calcification from decreased parathyroid hormone, vitamin D treatment or calcium supplement, when the hypocalcaemia is prolonged. In addition, there may be prolonged QT interval on electrocardiography.[7, 13] Eventually, there could be congestive heart failure from hypocalcaemic myopathy.[14]

Hypocalcaemia occurs in hypoparathyroidism due to an interference in calcium metabolism. Other biochemical imbalances in hypoparathyroidism may include hyper-phosphataemia, hyperkalaemia or hypomagnesaemia.

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CASE REPORT

Mrs NT, a 35-year-old secondary school teacher, was referred to the eye clinic in April 2001. The presenting complaint was difficulty in reading small print and aching of the eyes, which had lasted for one month and which started about two weeks after a subtotal thyroidectomy for a large simple multinodular goitre (euthyroid). She was said to have developed signs of hypocalcaemia (carpopedal spasm, positive Chvostek's and Trousseau's signs) ten days after the surgery. Treatment was initiated with 10mls of 10% calcium gluconate in 500ml of 5% dextrose/saline. The hypocalcaemia persisted, thus necessitating regular treatment with intravenous supplementation due to the non-availability of adequate oral preparations of calcium. Prior to the surgery, she had enjoyed good vision - for both close and distant objects - and had never used glasses. She is not a known diabetic or hypertensive patient. Her preoperative serum calcium level was normal (2.75mmol/l). She is married with four children; she does not smoke or drink alcohol.

On examination, all vital signs showed a healthy young woman. She had a horizontal lower neck scar at the site of the operation. Cardiovascular, respiratory and abdominal findings were essentially normal.

Ocular examination revealed a visual acuity of 6/6 in each eye and a near vision of N36 at 38cm using the Jaeger's chart. Anterior segment was normal, pupillary diameter was 2.5mm, lens was clear and the cup/disc ratio was 0.2 in both eyes. Intraocular pressure was 12mmHg in both eyes. Retinoscopy showed a net result of +1.50 diopters in both eyes for distance vision with an addition of +1.50 diopters. A diagnosis of hypocalcaemia-induced accommodation insufficiency leading to hypermetropia and early presbyopia was made. The possibility of a stress-aggravated pre-existing refractive error was also considered. The error persisted three months after surgery and the reading addition increased to 2.00 diopters. She subsequently defaulted from follow-up both in the eye and surgical outpatient clinic for a period of twelve months, only taking intravenous calcium gluconate at home when the symptoms became severe. At the time she re-presented, she had developed bilateral mature cataract with a visual acuity of counting fingers in both eyes. The patient initially refused to have cataract surgery for fear of complications. She eventually had a right eye extracapsular cataract extraction and a posterior chamber intraocular lens implant in April 2003. Two weeks after the surgery, she had an unaided visual acuity of 6/12.

Her preoperative and postoperative biochemical levels are as shown in tables 1 and 2. Her fasting blood sugar was 2.5mmol preoperative and has remained normal till date. Her T_3, T_4, TSH levels were normal pre and postoperative.

Table 1. Results of investigations pre and post thyroidectomy

Date	Calcium MMOL/L (2.25-2.65)	PO4 MMOL/L (0.6-1.4)	Total protein G/L (60-80)	Albumin G/L (30-50)
*10/2/2000	2.35	1.5	72	38
[1]21/2/2000	1.94	2.2	70	38
[2]21/2/2000	2.97	2.5	70	39
25/2/2000	2.27	2.0	80	39
06/3/2000	2.70	2.04	77	43
14/3/2000	1.86	1.4	81	41
27/3/2000	1.98	-	-	-
29/3/2000	1.02	2.2	80	42
08/8/2001	2.02	1.8	81	43
27/11/2001	1.73	1.6	84	40

*Result preoperatively
[1] Result at onset of symptoms
[2]Result after first correction with intravenous calcium

Table 2. A One-month Laboratory Data 20 months after Thyroidectomy

25/11/02	pre calcium infusion	1.63	1.13	83	39
	post calcium infusion	2.39	0.97	83	36
3/12/02	pre calcium infusion	1.47	1.20	67	34
	post calcium infusion	2.29	0.99	63	33
14/12/03	pre calcium infusion	1.43	1.20	78	43
	post calcium infusion	3.02	0.80	74	41
20/12/02	pre calcium infusion	1.97	1.30	83	45
	post calcium infusion	2.43	0.89	73	40

DISCUSSION

All preoperative investigations carried out, including blood calcium level, were essentially normal. The patient, however, developed permanent hypocalcaemia most likely due to damage to her parathyroid glands during surgery - an uncommon complication and probably the first identified in our centre. Although a parathyroid hormone assay was not done due to non-availability of diagnostic facilities, the presence of hypercalciuria (4.3mmol/24hrs) in the presence of persistent hypocalcaemia was consistent .with hypoparathyroidism. The known preoperative risk factors for the development of hypocalcaemia following thyroidectomy include large thyroid mass with retrosternal extension, elevated free thyroxine pre-operative; thyroid cancer, re-operation and total

thyroidectomy.[1-4] In this patient, a large thyroid mass with retrosternal extension, weighing 950g after surgery, might have contributed to the development of hypocalcaemia. This patient developed a fully mature bilateral cataract within a year of the onset of persistent hypocalcaemia. The only predisposing factor found was the persistently low level of serum calcium as shown in table 1. The thyroid hormone level pre and post-operative were normal (table 2), pre and postoperative fasting blood sugar levels were also within normal limits. The control of the blood calcium level was poor because of the patient's poor compliance with follow-up and recommended medication. For a period of 12 months, she stayed away from the clinic and only went to a medicine store for injections when her symptoms became unbearable. This was compounded by the non-availability of oral calcium in adequate preparation, necessitating the use of intravenous calcium every 3 days when she subsequently re-presented. It has been shown that the development of hypocalcaemia-induced cataract is related to the blood calcium level and not to the duration of the disease. In a series of 38 patients with hypocalcaemia, 32 developed cataract.[9] Cataracts have been reported to develop in some patients 10 years[15] and even up to 36 years[16] after diagnosis of hypocalcaemia. In this patient, the cataract developed and matured within a year. The literature reviewed did not report any refractive error in the patients, but the refractive error seen in this patient may have resulted from hypocalcaemic myopathy.[14] It may, however, be due to a change in the refractive index of the lens during the early phase of cataract formation. It is known that an optimal level of calcium ion is needed for arteriolar vasoconstriction and muscular contractility, while surgery-induced hypocalcaemia can lead to accommodative insufficiency.[8]

RECOMMENDATIONS

All patients with identified risk factors should have preoperative calcium level assessment, a very careful surgery and postoperative calcium level assessment. Routine autotransplantation of at least one parathyroid gland in repeated or total thyroidectomy may reduce permanent hypoparathyroidism to zero[17] The management of patients with permanent hypocalcaemia involves prompt detection and effective control of calcium levels in order to reduce the incidence of ocular and other systemic complications. Calcium level should be maintained at normal or slightly below normal level. Oral calcium and vitamin D should be available in adequate preparation; 2-8g of calcium carbonate per day is needed.[7, 8] Hyperphosphataemia, alkalosis and hypomagnesaemia should be corrected concomitantly when present. Symptomatic treatment of complications such as cataract and band keratopathy should be done. Ocular assessment should be performed periodically when managing a patient with hypocalcaemia.

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