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POST-THYROIDECTOMY HYPOCALCAEMIA

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Temporary post-thyroidectomy hypocalcaemia is a relatively common complication, due to removal, injury or devascularization of the parathyroid glands. It may also be secondary to hungry bones due to postoperative reversal of thyrotoxic osteodystrophy, reactive hypoparathyroidism due to relative hyper-calcaemia in thyrotoxic patients or calcitonin release due to operative manipulation of the thyroid glands.
62 patients, 38 with nodular toxic goitres and 24 with simple nodular goitres, undergoing subtotal thyroidectomies were included in this study. 88.7% had at least three parathyroid glands preserved. All had postoperative levels of parathyroid hormone measured.
The incidence of biochemical hypocalcaemia was 42% and that of symptomatic
hypocalcaemia was 24%. No patient was hypoparathyroid. The incidence of hypocalcaemia was 50% (19/38) in the toxic group compared with 16.6% (4/24) in the simple goitre group.
Hypoparathyroidism may not be the cause of post-thyroidectomy hypocalcaemia and 'hungry bone syndrome' could be considered particularly in toxic patients

Keywords: Hypocalcaemia, Thyroidectomy

INTRODUCTION

Hypocalcaemia may occur after thyroid surgery due to removal, injury or devascularization of the parathyroid glands. It may also be secondary to hungry bones[1] due to postoperative reversal of thyrotoxic osteodystrophy[2], calcitonin release due to operative manipulation of the thyroid glands[3,4] or reactive hypoparathyroidism due to relative hyper-calcaemia in thyrotoxic patients[5]. The symptoms of clinical hypocalcaemia are often distressing when severe[6] and the sooner the clinical manifestations, the more serious the prognosis is[1]. The condition presents dramatically 2-5 days postoperatively and the delay of the appearance of hypocalcaemia to 2-3 weeks is very rare. Parathyroid insufficiency may be temporary or permanent[7]. The incidence of permanent hypocalcaemia ranged from 0.5%7 and 0·2–1·9%6 to 2.8% and 3.4% in subtotal and total thyroidectomies respectively[8]. Reported incidence of temporary hypocalcaemia ranged from 5.2%8 and 6·9%6 to 30%9 and many patients require prolonged treatment before calcium levels return to normal[6].

The aim of this study is to determine the significance of hypoparathyroidism as a cause of hypocalcaemia after subtotal thyroidectomy with respect to postoperative parathyroid hormone (PTH) levels

PATIENTS AND METHODS

62 patients, 38 with nodular toxic goitres and 24 with simple nodular goitres, undergoing subtotal thyroidectomies were included in this study. In each case, bilateral subtotal thyroidectomy was performed with a remnant equal to a normal thyroid lobe left dorsally. As many parathyroid glands were identified and preserved as possible and both inferior thyroid arteries were not ligated in all thyroidectomies.

In all patients, the postoperative PTH level was measured as well as the serum calcium level preoperatively and on the first and third postoperative days.

Biochemical hypocalcaemia was defined as a total calcium concentration less than 8.4 mg/dl (normal range

8.4-10.2 mg/dl) and clinical hypocalcaemia by the presence of one or more of the following: numbness or paraesthesia of the face, fingers or toes, or positive Chvostek's or Trousseau's signs. Normal PTH values were defined as 5.26–34.74 pmol/l (C-terminal radioimmunoassay). Statistical analysis was done considering P>0.1 to be not statistically significant and P<0.05 to be statistically significant

RESULTS

The 62 patients (38/62 toxic nodular goitres & 24/62 simple nodular goitres) were 55/62 females (88.7%) and 7/62 men (11.3%). The mean age was 31 years (range 25-44 years).

The mean time for toxic patients to be euthyroid before surgery was 8 weeks.

In the subtotal thyroidectomies, the average dimensions of the dorsally left remnant were approximately 4x3x2 cm and the number of identified and preserved parathyroid glands was 3 or more in 88.7%. 4 were identified and preserved in 24 cases, 3 in 31 cases, 2 in 6 cases and in one case the parathyroids were not identified. The upper parathyroids were commonly encountered at the posterolateral aspect of the upper 2/3 of the thyroid lobes from the termination of the inferior thyroid artery to the upper pole, while the lower at the intersection of inferior thyroid artery and recurrent laryngeal nerve or at the lower pole.

No parathyroids were detected histopathologically in the removed specimens.

All patients were normocalcaemic before operation, with mean serum calcium level of 9.16 (range 8.56-10.08) mg/dl. 26/62 patients (42%) developed biochemical hypocalcaemia and 15/62 patients (24%) developed temporary clinical manifestations of hypocalcaemia.

Table (1) compares between the pre and postoperative serum calcium levels in patients with toxic and simple goitres. The mean postoperative level was lower than the mean preoperative level in the two groups. The mean decrease was greater in patients with toxic goitres but the difference was not statistically significant (P 0·1). The incidence of hypocalcaemia was 50% (19/38 patients) in the toxic group compared with 16.6% (4/24) in the simple goitre group (P 0·05). (Table 2) compares between the raised and normal postoperative PTH levels in patients with toxic and simple goitres. The postoperative PTH level was raised in 6 patients, all with toxic goitres and there was no difference in postoperative PTH level in 56 patients with toxic and simple goitres. No patients with postoperative hypoparathyroidism were encountered and the mean postoperative PTH level was 19·6 (range 5.62–39.46) pmol/l. (Table 3) compares between the pre and postoperative serum calcium levels in patients with raised and normal PTH. The decrease in serum calcium level after operation was not significant in patients of both groups. (Table 4) compares between the hypo (26/62) and normocalcaemic (36/62) patients in patients with raised (6/62) and normal (56/62) postoperative PTH levels. The PTH level was not related to postoperative hypocalcaemia.

Table (1): ***Serum calcium levels***

	Serum calcium (mg/dl) mean (range)	
	Toxic goitre	Simple goitre
Preoperative	9.20(8.56-10.08)	9.12(8.76-9.52)
Postoperative	8.60(6.24-9.64)	8.96(7.92-9.40)
Difference	0.60	0.16

Table (2): ***Postoperative PTH levels***

	No. of Patients	
	Toxic goitre	Simple goitre
Raised PTH	6	0
Normal PTH	32	24

Table (3): ***Serum calcium levels in patients with raised & normal postoperative PTH levels***

	Serum calcium (mg/dl) mean (range)	
	Raised PTH	Normal PTH
Preoperative	9.12(8.76-9.44)	9.20(8.56-9.72)
Postoperative	8.64(7.92-9.36)	8.76(6.24-9.64)
Difference	0.48	0.44

Table (4): *Hypocalcaemia in patients with raised & normal postoperative PTH levels*

	No. of Patients	
	Raised PTH	Normal PTH
Hypocalcaemic	2	24
Normocalcaemic	4	32

DISCUSSION

In this study, 88.7% of the patients undergoing subtotal thyroidectomy had at least three parathyroid glands preserved at operation, both inferior thyroid arteries were not ligated and the parathyroids were not detected histopathologically in the removed specimens. In spite of this, the incidence of biochemical and temporary clinical hypocalcaemia was 42% and 24% respectively while the incidence of biochemical hypoparathyroidism was nil. Parathyroid insufficiency as a postoperative complication of bilateral subtotal thyroidectomy, is due to removal of parathyroid glands, or infarction through damage to its end artery[10] but a recent article stated that truncal ligation of the inferior thyroid arteries has no effect on the incidence of postoperative hypocalcaemia[11].

This study does not support hypoparathyroidism as an aetiological factor in post-thyroidectomy hypocalcaemia although it is the most widely accepted[12-14].

Among the other causes of post-thyroidectomy hypocalcaemia, Calcitonin release proved to be wrong[2,15]. 'Hungry bone syndrome' was doubted, based on the fact that most thyrotoxic patients undergoing thyroidectomy became euthyroid before operation, thus reversing the osteodystrophy. Similarly, the relative hypercalcaemia would have been reversed before surgery[5,16,17].

In this study, the incidence of hypocalcaemia was significantly higher in thyrotoxic patients, 6 of them were with raised postoperative PTH levels. There was no significant difference in the preoperative and postoperative serum calcium levels between these and patients with normal postoperative PTH levels. The results of this study could be explained by the fact that there is an increase in bone catabolism in thyrotoxicosis, and that the degree of thyrotoxic osteodystrophy increases proportionately with toxicity [5,16,17]. Reversal of this osteodystrophy may be involved and hyperparathyroidism may be secondary to this reversal as a compensatory mechanism to prevent post-thyroidectomy hypocalcaemia[18].

Although preoperative preparation by antithyroid drugs should reverse osteodystrophy, this reversal takes up to 20 weeks[2,16], whereas most surgical patients are prepared for a much shorter period (6–10 weeks)[18]. The 'hungry bone syndrome' could be considered as a cause of post-thyroidectomy hypocalcaemia particularly in toxic patients.

REFERENCES

1. Ramzy AF. The Parathyroid glands. Hypoparathyroidism. In Kasr El-Aini Introduction to Surgery (2nd edn), Galal SA, Barsoum MS, Mohsen A, Fawzy T, Abdel-Halim S (eds). University Book Centre: Cairo, 2000; 383-9.

2. Michie W, Duncan T, Hamer-Hodges DW et al. Mechanism of hypocalcaemia after thyroidectomy for thyrotoxicosis. Lancet 1971; i: 508–13.

3. Watson CG, Steed DL, Robinson AG, Deftos LJ. The role of calcitonin and parathyroid hormone in the pathogenesis of post-thyroidectomy hypocalcemia. Metabolism 1981; 30: 588–9.

4. Wilkin TJ, Eyles TE, Paterson CR et al. Post-thyroidectomy hypocalcaemia: a feature of the operation or the thyroid disorder? Lancet 1977; i: 621–3.

5. Golding SR, Krane SM. Organ system manifestations of thyrotoxicosis: the skeletal system. In: Ingbar SH, Braverman LE, eds. The Thyroid. Philadelphia, Pennsylvania: JB Lippincott, 1986: 558–615.

6. Falk SA, Birken EA, Baran DT. Temporary post-thyroidectomy hypocalcemia. Arch Otolaryngol Head Neck Surg 1988; 114: 168–74.

7. Kenawy M, Moussa A. The Thyroid gland. Surgery for Thyrotoxicosis. In Kasr El-Aini Introduction to Surgery (2nd edn), Galal SA, Barsoum MS, Mohsen A, Fawzy T, Abdel-Halim S (eds). University Book Centre: Cairo, 2000; 356-83.

8. Songun I, Kievit J, Wobbes T, Peerdeman A, Van de Velde CJH. Extent of thyroidectomy in nodular thyroid disease. European Journal of Surgery 1999; 165: 839-42.

9. Percival RC, Hargreaves AW, Kanis JA. The mechanism of hypocalcaemia following thyroidectomy. Acta Endocrinol 1985; 109: 220–6.

10. Wade JSH. Parathyroid insufficiency. In Bailey and Love's Short Practice of Surgery (21st edn), Mann CV, Russell RCG. Chapman & Hall: London, 1992; 756.

11. Dolapç M, Doanay M, Reis E, Kama NA. Truncal ligation of the inferior thyroid arteries does not affect the incidence of hypocalcaemia after thyroidectomy. European Journal of Surgery 2000; 166: 286-8.

12. Ramus NI. Hypocalcaemia after subtotal thyroidectomy for thyrotoxicosis. Br J Surg 1984; 71: 589–90.

13. Sugrue DD, Drury MI, McEvoy M, Heffernan SJ, O'Malley E. Long term follow-up of hyperthyroid patients treated by subtotal thyroidectomy. Br J Surg 1983; 70: 408–11.

14. Wingert DJ, Friesen SR, Iliopoulos JI, Pierce GE, Thomas JH, Hermreck AS. Post-thyroidectomy hypocalcemia: incidence and risk factors. Am J Surg 1986; 152: 606–10.

15. Sawers JSA, Kellett HA, Brown NS et al. Does calcitonin cause hypocalcaemia after thyroidectomy? Br J Surg 1982; 69: 456–8.

16. Mosekilde L, Melsen F, Bagger JP, Myhre-Jensen O, Schwartz-Sorensen N. Bone changes in hyperthyroidism: interrelationships between bone morphometry, thyroid function and calcium–phosphorus metabolism. Acta Endocrinol 1977; 85: 515–25.

17. Laitinen O. Hypocalcemia after thyroidectomy. Lancet 1976; ii: 859–60.

18. See ACH, Soo KC. Hypocalcaemia following thyroidectomy for thyrotoxicosis. Br J Surg 1997; 84: 95-7.