World J. Surg. 24, 722–726, 2000
DOI: 10.1007/s002689910116

WORLD
Journal of
SURGERY

Hypomagnesemia and Hypocalcemia after Thyroidectomy: Prospective Study

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Abstract. Hypomagnesemia after total thyroidectomy has not been studied extensively. Our anecdotal experience suggests that it may be important in some patients after thyroid excision. The hypomagnesemic hypocalcemic syndrome has been described in other disease states in which a state of functional hypoparathyroidism exists. This study was designed to determine the incidence of hypomagnesemia after total thyroidectomy and relate it to hypocalcemia and symptoms during the postoperative period. A prospective study of all patients undergoing total thyroidectomy between September 1994 and July 1996 was performed. Patient data, thyroid function, retrosternal extension, initial versus reoperative surgery, operative details, parathyroid resection, and pathology were recorded. Calcium, magnesium, electrolytes, blood count, liver function tests, and albumin were measured prior to surgery and twice daily during the postoperative period. Fifty patients underwent total thyroidectomy: 68% were hypocalcemic, 72% were hypomagnesemic, and 36% were symptomatic during the postoperative period. Hypomagnesemia and gender were associated with hypocalcemia. Volume of fluid and neck dissection were associated with low magnesium levels. Hypomagnesemia and parathyroid resection were risk factors for symptoms after thyroidectomy. No patients developed permanent hypoparathyroidism. Transient hypocalcemia and hypomagnesemia occur frequently after total thyroidectomy. The etiology of this phenomenon is probably multifactorial. Patients are more likely to be symptomatic when both cations are low, and attempting to correct only hypocalcemia may prolong symptoms. It is important to monitor both calcium and magnesium levels after total thyroidectomy and to correct deficiencies to facilitate prompt resolution of symptoms.

Transient asymptomatic hypocalcemia after total thyroidectomy is common [1, 2], whereas clinically significant hypocalcemia, which requires treatment, occurs in up to 50% of patients [3]. Symptoms, such as paresthesia, muscle weakness, tremor, and mental changes usually resolve with calcium (Ca) and vitamin D supplementation. The cause of hypocalcemia after total thyroidectomy is multifactorial, and some of the factors involved include previous thyrotoxicosis, extent of surgery (including completion thyroidectomy), retrosternal extension, removal of more than one parathyroid, macro dilution, medications, and general anesthesia [1, 2].

Hypomagnesemia, long associated with hypocalcemia, can cause the same symptoms with or without coexisting low serum calcium [4]. Magnesium is a cofactor for the activity of most adenosine triphosphatases and is therefore important in many metabolic pathways [5]. In particular, magnesium deficiency has been shown to cause reversible failure of parathormone (PTH) secretion [6–8] and end-organ resistance to PTH and increased peripheral degradation of PTH [9, 10]. Hypomagnesemia occurs in many clinical situations, including malnutrition, malabsorption, and chronic alcoholism, in patients having chemotherapy, and commonly in intensive care patients [5, 7, 9, 11, 12, 13, 14, 15, 16]. Magnesium replacement has been shown to aid in the correction of hypocalcemia and hypokalemia [17]. Although hypomagnesemia has been recognized after parathyroid surgery, it has not been studied extensively after total thyroidectomy [18]. Our own experience suggests that hypomagnesemia occurs after total thyroidectomy and if untreated, particularly in the presence of hypocalcemia, may lead to persistent symptoms.

The main aim of this study was to determine the incidence of hypomagnesemia in patients undergoing total thyroidectomy and to correlate it with calcium levels during the postoperative period. A further aim was to identify clinical risk factors for the development of these metabolic disorders.

Methods

A prospective study of all patients undergoing total thyroidectomy from September 1994 to July 1996 was performed. Data were collected in a prospective database. Age, gender, thyroid functional status, extent of thyroidectomy, length of operation, presence of retrosternal thyroid extension, initial versus reoperative neck surgery, parathyroid resection, and thyroid disease were recorded. It has been our institution's policy to perform total thyroidectomy for patients with multinodular goiter, as lesser procedures may be associated with recurrence [19]. Thyroid function tests were performed at initial presentation; and blood samples were obtained for calcium (Ca), magnesium (Mg), urea, electrolytes, and creatinine assays; a full blood count; and liver function tests. Albumin was measured immediately prior to surgery, at 6, 12, and 24 hours, and twice daily until the patient was discharged. The volume of intravenous fluids, fall in hematocrit, and percentage falls in calcium and magnesium were recorded.

Hypocalcaemia was defined as mild (corrected Ca^{2+} 1.96–2.09 mmol/L) and severe ($Ca^{2+} \leq 1.95$ mmol/L); the normal range for Ca^{2+} was 2.10 to 2.55 mmol/L. Hypomagnesemia was defined as mild ($Mg^{2+} < 0.7$ mmol/L) or severe (< 0.6 mmol/L); normal range was 0.70 to 0.95 mmol/L. Permanent hypoparathyroidism was defined by the requirement for oral calcium or vitamin D

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Table 1. Patient characteristics, surgical details, and pathology of removed thyroid gland.

Parameter	Result
Total no. of patients	50
Male/female ratio	12/38
Age (years), mean (range)	45 (19–75)
Thyrotoxic patients (no.)	7
Operating time (minutes), mean	151
Neck dissections (no.)	6
Completion thyroidectomies (no.)	7
Parathyroid glands removed (no.), mean	0.52
No. with multinodular goiter	26
No. with Graves' disease	3
No. with malignant disease	21

supplementation after 12 months from the time of thyroidectomy to maintain a eucalcemic state [1].

Symptoms of "hypocalcemia" were defined as acroparesthesia, carpopedal spasm, seizures, laryngeal stridor, or cardiac arrhythmias. Patients had Trousseau and Chvostek signs recorded after surgery. Patients with symptomatic hypocalcemia or calcium levels of < 1.95 mmol/L were treated with calcium gluconate infusion (0.67 mmol/hr) followed by calcium carbonate and calcitriol oral supplementation. Patients with severe hypomagnesemia were managed with intravenous magnesium sulfate (10 mmol infusion) followed by oral magnesium supplementation. Patients were followed up after discharge by their consulting surgeon; and information regarding calcium and magnesium supplementation was recorded from their follow-up visits. Patients were followed for a minimum of 6 months after their procedure and longer if symptoms persisted.

Univariate and multivariate analyses were used to determine dependent and independent risk factors for symptoms, respectively.

Results

Fifty patients between September 1994 and July 1996 underwent total thyroidectomy. Patient characteristics and surgical and pathologic details are shown in Table 1. Seven patients were thyrotoxic on presentation; all were euthyroid prior to surgery. Twenty patients had one or more parathyroids found in the specimen after thyroidectomy. Eight patients had significant retrosternal extension at surgery.

No patients had symptoms of hypocalcemia on preoperative examination. Only one patient was hypocalcemic on preoperative testing (calcium level was 2.09 mmol/L, and magnesium was 0.79 mmol/L). The patient became symptomatic on day 1 with magnesium and calcium levels of 0.63 and 2.30 mmol/L, respectively, after total thyroidectomy for papillary thyroid carcinoma. He was not treated with supplementation, and symptoms resolved spontaneously with improvement of magnesium levels.

There were 18 patients (36%) who were symptomatic during the postoperative phase (Table 2); 14 of them had a positive Trousseau sign. There were 34 patients with hypocalcemia (68%) and 13 (26%) with calcium ≤ 1.95 mmol/L; 36 patients were hypomagnesemic, 12 with magnesium levels < 0.6 mmol/L.

All patients who became symptomatic did so within 48 hours after surgery, with 8 of 18 patients developing symptoms on the second postoperative day. Of the 50 patients, 18 (36%) were given intravenous calcium during the postoperative period, 14 of whom were symptomatic. Three patients were given intravenous magnesium, all of whom had severe symptoms. Ten patients received magnesium supplementation overall, 7 of whom were symptomatic.

Table 2. Incidence of calcium and magnesium abnormalities and symptoms.

Parameter	Incidence ($n = 50$)	Symptoms ($n = 18$)
Mild hypocalcemia	21 (42%)	7 (33%)
Severe hypocalcemia	13 (26%)	8 (62%)
Mild hypomagnesemia	24 (48%)	10 (42%)
Severe hypomagnesemia	12 (24%)	7 (58%)
Severe hypomagnesemia and hypocalcemia	6 (12%)	5 (83%)

Criteria: mild hypocalcemia, 1.95–2.10 mmol/L; severe ≤ 1.95 mmol/L (normal 2.10–2.55 mmol/L). Mild hypomagnesemia 0.6–0.7 mmol/L; severe ≤ 0.6 mmol/L (normal 0.70–0.95 mmol/L).

A total of 18 (36%) patients were symptomatic.

Many patients had both calcium and magnesium abnormalities concurrently, so the numbers (incidence, $n = 50$ and symptoms, $n = 18$) do not add up in the table. Some patients who were hypocalcemic were also hypomagnesemic. For example, eight patients in the "severe hypocalcemia" group ($n = 13$) were symptomatic and seven in the "severe hypomagnesemia" group ($n = 12$) were symptomatic. Of these 15 patients, 5 had both severe symptomatic hypocalcemia and severe hypomagnesemia.

Table 3. Correlation of symptoms with possible risk factors.

Parameter	%	Multivariate analysis (p)	Univariate analysis (p)
Hypocalcemia	68	0.015	< 0.001
Hypomagnesemia	72	0.006	< 0.001
Thyrotoxicosis	14	NS	NS
Retrosternal extension	16	NS	NS
Malignant disease	42	NS	NS
Parathyroid excision	40	0.031	0.02
Completion surgery	14	NS	NS
Neck dissection	10	NS	0.04

Of the patients with severe hypomagnesemia, 58% had symptoms compared with an overall incidence of 36% in the study. This compares to 62% of patients with severe hypocalcemia being symptomatic. When severe hypocalcemia and severe hypomagnesemia occurred, five of six (83%) patients became symptomatic ($p = 0.004$). There were 3 of 16 patients with normal calcium levels who were symptomatic, but these patients had associated hypomagnesemia.

There was a trend to lower calcium levels in female patients ($p = 0.039$), but no other risk factors correlated highly with postoperative hypocalcemia. Hypomagnesemia was positively correlated with hypocalcemia ($p = 0.04$), and patients were more likely to be symptomatic when both cations were low ($p = 0.019$). On multivariate analysis, both hypomagnesemia ($p = 0.006$) and hypocalcemia ($p = 0.015$) were associated with symptoms (Table 3). Parathyroid resection ($p = 0.014$) and neck dissection ($p = 0.031$) were suggested as risk factors for symptoms on univariate analysis. Parathyroid resection was an independent risk factor for symptoms on multivariate analysis.

When risk factors for hypomagnesemia after thyroidectomy were analyzed, only the volume of fluid and neck dissection correlated with magnesium levels. Patients with severe hypomag-

nesemia were given an average of 4.93 liters of fluid during the 24-hour perioperative period, compared to 4.69 liters in the mildly hypomagnesemic patients and 3.64 liters in patients with normal magnesium levels ($p = 0.027$). On univariate analysis neck dissection was associated with hypomagnesemia ($p = 0.049$) but not hypocalcemia ($p = 0.167$).

Gender, retrosternal extension, histopathology, parathyroid resection, completion surgery, prior thyrotoxicosis, and a fall in the hematocrit did not correlate with hypomagnesemia. Young patients tended to have lower magnesium levels, but the difference was not significant ($p = 0.098$).

No patients demonstrated permanent hypoparathyroidism after total thyroidectomy. One patient required calcium supplementation for 4 months and one for 8 months.

Discussion

Although most patients with symptomatic hypocalcemia after thyroid or parathyroid surgery respond to calcium and vitamin D supplementation, it has been our anecdotal experience, confirmed by this study, that a small number of patients may also benefit from magnesium replacement. This study has prospectively documented that hypomagnesemia is common in patients undergoing total thyroidectomy, and that up to 10% develop significant hypomagnesemia and hypocalcemia.

The relation between calcium and magnesium metabolism is a complex one and relates mainly to the interaction of these cations with PTH. Levine and Coburn suggested that magnesium may mimic/antagonize calcium by competing with calcium for a binding site on the parathyroid cell [20]. This "calcium" receptor acts by a transmembrane signal transduction system to cause inhibition of PTH secretion in the presence of elevated extracellular calcium levels. In the presence of hypomagnesemia, the relative number of calcium ions available for binding is higher and PTH secretion may be inhibited. Rude et al. [11] showed that administering intravenous magnesium to patients with hypocalcemia secondary to hypomagnesemia resulted in a dramatic rise in serum PTH levels within 1 minute after injection. Thus hypomagnesemia can inhibit PTH secretion.

There is also evidence that hypomagnesemia contributes to increased catabolism of parathormone [9, 21]. Diminished end-organ responsiveness to PTH has also been demonstrated in several studies of magnesium deficiency in humans and experimental animals [9, 22, 23, 24, 25, 26, 27]. Despite this experimental and clinical work, which demonstrates the importance of hypomagnesemia, particularly when it exists with hypocalcemia, there has been little published on the significance of hypomagnesemia after total thyroidectomy. In the present prospective study, when symptoms and magnesium/calcium levels were subjected to multivariate analysis, it was found that both magnesium and calcium were associated with symptoms. This demonstrates that hypomagnesemia may well contribute to postoperative tetany after total thyroidectomy, especially with concomitant hypocalcemia. This finding was supported by Szubin et al. [28], who found that serum magnesium levels should be monitored after total thyroidectomy and corrected if low. However, it is impossible to determine from this study whether calcium or magnesium was mainly responsible for the symptoms we observed when both cations were low. It is possible that administering only calcium to correct severe hypocalcemia in the presence of severe hypomagnesemia may prolong symptoms [14, 18, 29], which has certainly been our anecdotal experience.

The finding that prior thyrotoxicosis and retrosternal goiter were not risk factors for postoperative electrolyte disturbances or symptoms in our study contrasted with the literature regarding extent of surgery and thyrotoxic osteodystrophy [30, 31]. This may be because in the present study there were only three patients with Graves' disease, seven patients in total with thyrotoxicosis, and eight retrosternal goiters; and these risk factors failed to reach statistical significance owing to the small sample size.

We found that neck dissection and parathyroid excision were risk factors for postoperative symptoms but not for hypocalcemia. This is difficult to explain but may be related to the low magnesium levels in patients who had an additional neck dissection or more likely to the small numbers in these two groups. It is also possible that we might be overreporting symptoms, as we were actively testing for neuromuscular irritability rather than letting the patients report their symptoms.

Other studies examining hypocalcemia after thyroidectomy have demonstrated additional possible risk factors for developing symptomatic hypocalcemia after surgery. McHenry et al. [1] found that in their series of unilateral thyroid lobectomy and bilateral thyroid resection 40% and 49% of patients developed transient hypocalcemia, respectively, and 15% of patients were symptomatic. The three independent risk factors for hypocalcemia in their study were thyrotoxicosis at initial presentation, substernal thyroid disease, and carcinoma. Retrosternal goiter and surgery for carcinoma usually involve more extensive surgery, with greater potential for removal of parathyroid glands or impairment of parathyroid blood flow during dissection [2]. Although Henry et al. [32] demonstrated that central node dissection significantly increased the risk of transient hypoparathryoidism, as appeared to be the case in our study, McHenry and coworkers did not show any differences in PTH levels among patients having more or less extensive surgery [1].

Clearly, the mechanism for hypocalcemia after thyroidectomy is more complex. McHenry et al. [1] did not find any correlation between postthyroidectomy hypocalcemia and changes in serum magnesium and phosphorus. Similarly, in a study of 63 subtotal thyroidectomies See and Soo did not find any correlation between postoperative hypocalcemia and hypoparathyroidism [30]. As most of the patients in these series had less than total thyroidectomy, it is difficult to draw direct comparisons between these studies and ours.

The finding that the volume of intravenous fluid related to postoperative magnesium levels partially supports the work of Demeester-Mirkine et al. [33]. They found that transient falls in serum calcium, magnesium, sodium, potassium, osmolality, phosphorus, and protein occurred in both control patients and thyroidectomy patients after surgery, suggesting extravascular flux of proteins and electrolytes. They concluded that postthyroidectomy hypocalcemia was multifactorial, due to a combination of hemodilution, temporary parathyroid insufficiency, and in thyrotoxic patients "hungry bone syndrome."

We do not have a clear-cut explanation for the initial fall in magnesium level in our patients, but similar to the changes in serum calcium it is likely to be multifactorial. Temporary hypoparathyroidism, decreased tubular reabsorption of magnesium in the kidney, and extracellular volume expansion leading to increased magnesium excretion may all play a role.

Conclusions

We have found that transient falls in calcium and magnesium occur frequently after total thyroidectomy, and a proportion of these patients become symptomatic. The etiology of this phenomenon is multifactorial, with physiologic responses to surgery/general anesthesia and hemodilution possibly contributing to changes in electrolyte homeostasis. The finding that hypomagnesemia seemed to be as important as hypocalcemia in postoperative neuromuscular irritability is supported by experimental work demonstrating the membrane-stabilizing properties of magnesium and its effects on PTH secretion, metabolism, and end-organ responsiveness [34]. We also found that hypomagnesemia correlated with hypocalcemia ($p = 0.04$), and that patients were more likely to be symptomatic when both cations are low ($p = 0.019$), supporting the evidence in the literature for the hypomagnesemic hypocalcemic syndrome [34].

We would recommend that serum calcium be measured routinely in all patients after total thyroidectomy, as is the usual practice of many endocrine surgeons. When patients become symptomatic, it is important to monitor both magnesium and calcium levels and correct them when low. This may help to facilitate resolution of symptoms. We propose a further study to investigate the changes in PTH levels after total thyroidectomy and the effect of magnesium supplementation on PTH release during the critical 48-hour postoperative period.

Résumé

Introduction: L'hypomagnésémie après thyroïdectomie totale n'a pas été étudiée en détails. Notre expérience, certes anecdotique, suggère que l'hypomagnésémie puisse être un problème important après thyroïdectomie chez certains patients. Le syndrome d'hypomagnésémie/hypocalcémie a été décrit dans d'autres maladies, dans lesquelles on retrouve une hypoparathyroïdie fonctionnelle. Cette étude a pour but de déterminer l'incidence d'hypomagnésémie après thyroïdectomie totale et de situer celle-ci par rapport à l'hypocalcémie et aux symptômes de la période postopératoire. Méthodes: On a réalisé une étude prospective de tous les patients ayant eu une thyroïdectomie totale entre septembre 1994 et juillet 1996. On a recueilli les renseignements démographiques, évalué la fonction thyroïdienne, noté le degré d'extension rétrosternale, s'il s'agissait d'une chirurgie initiale ou secondaire, les détails opératoires, la résection de la parathyroïde, et la pathologie. On a mesuré les taux de calcium, de magnésie, des électrolytes, les constantes sanguines, la fonction hépatique et l'albumine, avant l'acte chirurgical et deux fois par jour dans la période postopératoire. Résultats: 50 patients ont eu une thyroïdectomie totale. 68% des patients ont eu une hypocalcémie, 72% ont eu une hypomagnésémie dont 36% symptomatiques dans la période postopératoire. L'hypomagnésémie et le sexe étaient associés avec l'hypocalcémie. Le volume de fluide et l'adénolymphadénectomie du cou étaient des facteurs prédictifs d'hypomagnésémie. L'hypomagnésémie et la parathyroïdectomie ont été des facteurs de risque de symptômes après thyroïdectomie. Aucun patient n'a développé une hypoparathyroïdie permanente. Conclusion: Une hypocalcémie et une hypomagnésémie transitoires sont fréquentes après thyroïdectomie totale. L'étiologie de ce phénomène est probablement multifactorielle. Les patients ont plus de chances d'être symptomatiques lorsque les deux cations sont bas, et la correction de la seule hypocalcémie risque de prolonger la symptomatologie. Il importe de monitorer les taux de calcium et de magnésium après thyroïdectomie totale et de corriger les déficits afin de faciliter la résolution rapide des symptômes.

Resumen

Introducción: La hipomagnesemia luego de tiroidectomía total no ha sido estudiada en forma extensa. Nuestra experiencia anecdótica sugiere que la hipomagnesemia puede ser importante en pacientes sometidos a resección tiroidea. El síndrome hipomagnesémico hipocalcémico ha sido descrito en otras entidades clínicas en que existe un estado de hipoparatiroidismo funcional. El presente estudio fue diseñado para determinar la incidencia de hipomagnesemia luego de tiroidectomía total y relacionarla con hipocalcemia y la aparición de síntomas en el periodo postoperatorio. Métodos: estudio prospectivo de todos los pacientes sometidos a tiroidectomía total entre septiembre de 1994 y julio de 1996, registrando la información pertinente al paciente, función tiroidea, extensión retroesternal, cirugía inicial versus reoperatoria, detalles de la operación, resección paratiroidea y patología. Se efectuaron determinaciones de niveles de calcio, magnesio, electrolitos, recuentos globular, pruebas de función hepática y albuminemia antes de la cirugía y dos veces diarias en el periodo postoperatorio. El volumen de los líquidos administrados y la disección cervical se asociaron con bajas concentraciones de magnesio. La hipomagnesemia y la resección paratiroidea fueron factores de riesgo de sintomatología luego de tiroidectomía. Ninguno de los pacientes desarrolló hipoparatiroidismo permanente. Conclusión: la hipocalcemia e hipomagnesemia transitorias se presentan frecuentemente luego de tiroidectomía total. La etiología de este fenómeno es probablemente multifactorial. Los pacientes tienden a ser sintomáticos cuando los niveles de ambos cationes están disminuidos, y cualquier intento orientado a corregir la hipocalcemia solamente puede prolongar la sintomatología. Es importante controlar tanto los niveles de calcio como los de magnesio luego de una tiroidectomía total y corregir las deficiencias a fin de facilitar la pronta resolución de los síntomas.

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