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One-Hour PTH after Thyroidectomy Predicts Symptomatic Hypocalcemia

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Abstract

Background—A major morbidity following total thyroidectomy is hypocalcemia. While many clinical factors and laboratory studies have been correlated with both biochemical and symptomatic hypocalcemia, the ideal use and timing of these tests remains unclear. We hypothesize one-hour (PACU) PTH will identify patients at risk for symptomatic hypocalcemia.

Methods—This prospective study evaluated 196 patients undergoing total thyroidectomy. Serum calcium and PTH levels were measured one hour after surgery and on postoperative day 1 (POD1). Performance of a central compartment lymph node dissection, parathyroid autotransplantation, indication for procedure, pathology, and presence of parathyroid tissue in the pathology specimen were recorded.

Results—Of 196 patients, 9 (4.6%) developed symptomatic hypocalcemia. 34 (17.3%) had a 1-hour PACU PTH ≥ 10 pg/dL while 31 (15.8%) had a POD1 PTH of ≥ 10 . Five (56%) of the nine symptomatic patients underwent central compartment lymph node dissection, 4 (44%) had parathyroid autotransplantation and 4 (44%) had a PACU PTH ≥ 10 . PACU and POD1 PTH levels were correlated ($R^2=0.682$). Multivariate regression identified central compartment dissection,

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autotransplantation, and PACU or POD1 PTH correlated with symptomatic hypocalcemia. PACU PTH, POD1 PTH, PACU Ca, malignant final pathology, and Age ≥ 45 years correlated with biochemical hypocalcemia.

Conclusion—1-hour postoperative PACU PTH is equivalent to POD1 PTH in predicting the development of symptomatic hypocalcemia. Biochemical hypocalcemia was not predictive of symptoms in the immediate post-operative period. Lymph node dissection and parathyroid autotransplantation correlated with symptomatic hypocalcemia and improve the sensitivity of biochemical screening alone.

Keywords

Thyroidectomy; PTH; Calcium; Hypocalcemia; Symptomatic Hypocalcemia

Introduction

One of the most common complications after total thyroidectomy is hypocalcemia. This can be a result of manipulation of the parathyroids during surgery, devascularization of the parathyroids, or from inadvertent removal of the parathyroid gland with the thyroid specimen. While improvements in surgical technique have made thyroidectomy a safe operation, temporary hypocalcemia still occurs. For routine cases, roughly 5-10% of patients will develop symptoms of temporary hypocalcemia (1, 2). While in more complex cases, such as those requiring lymph node dissection or with a diagnosis of Graves' disease, up to 30% of patients will experience symptoms of temporary hypocalcemia (3-7). The incidence of permanent hypocalcemia is considerably less (0-2%) (8-11).

In recent years there has been increasing pressure for early discharge of patients undergoing total thyroidectomy. While some surgeons place all patients on calcium postoperatively, others use this medication more selectively with biochemical and/or clinical factors guiding their decision making. Postoperative parathyroid hormone (PTH) and calcium levels, as well as clinical factors, such as lymph node dissection, autotransplantation, and indication for thyroidectomy have been studied to anticipate the need for calcium supplementation and predict the development of hypocalcemic symptoms. While biochemical studies of post thyroidectomy patients have demonstrated PTH levels at various times correlate with the development of symptomatic hypocalcemia, the optimal timing remains unclear. Postoperative serum calcium on day one has not been shown to consistently correlate with symptomatic hypocalcemia (1, 2, 12-18).

We sought to compare serum Calcium and PTH drawn at two time points, one hour postoperatively (PACU) and on postoperative day 1 (POD1) to identify which test is the best predictor of symptomatic and biochemical hypocalcemia. Additionally, we aimed to determine whether combining known clinical factors associated with hypocalcemia could improve the sensitivity of biochemical tests alone in predicting postoperative hypocalcemia after total thyroidectomy.

Methods

This was a prospective study evaluating all patients undergoing total thyroidectomy with and without lymph node dissection between July 2012 and December 2013 at our single institution. Total serum calcium (normal range: 8.4-10.2 mg/dL) and PTH (normal range: 15-75 pg/dL) levels were measured one hour after surgery in the PACU and on the morning of POD1. All patients were admitted for overnight observation following their procedure. The timing and whether to initiate calcium and/or calcitriol supplementation based on day of surgery and POD1 serum levels was performed at the discretion of the attending surgeon. Albumin was not used to correct calcium during the study period. Vitamin D was not routinely repleted prior to operative intervention.

Parathyroid glands seen during the operation were left in situ. Any parathyroid gland inadvertently removed at the time of surgery or appearing devascularized was autotransplanted into the ipsilateral sternocleidomastoid muscle. All lymph node dissections performed included a central compartment dissection with or without dissection of the lateral nodes. All central compartment dissections were completed following the American Thyroid Association Working Group Definition (19).

All patients undergoing total thyroidectomy during the study period were recorded. Patients with known hyperparathyroidism or renal failure at the time of surgery were excluded. Data collected included age, sex, medical history, and pathology results (including notation of any parathyroid tissue discovered in the specimen). Instances of symptomatic hypocalcemia were defined as circumoral numbness, tingling of the fingers and toes, or intense anxiety (20). Instances of symptomatic hypocalcemia were recorded on post-operative day one as well as at a 2 week post-operative visit. Patient contact was not initiated by the surgical team during this interval, patients developing symptoms during that time were, however, recorded. Any patient who reported symptoms of hypocalcemia during this post operative period were recorded as having symptomatic hypocalcemia. Postoperative calcium and PTH levels measured at one hour and on postoperative day one were recorded.

Risk factors for symptomatic postoperative hypocalcemia were derived from the literature. Biochemical cutoffs were defined as POD1 PTH ≥ 10 pg/dL and serum Calcium of ≤ 8.0 mg/dL. Clinical factors were chosen based on previously reported associations with postoperative hypocalcemia and included parathyroid autotransplantation, lymph node dissection, diagnosis of Graves' Disease and presence of parathyroid tissue in the pathology specimen (1, 2, 4-7, 10, 12-18, 21, 22).

Statistical analysis was performed using STATA software (version 13, College Station, TX) (23). Biochemical correlation between PACU and POD1 calcium and PTH levels was performed using linear regression analysis. Univariate analysis of individual risk factors was performed using student's t-test and case control odd's ratio calculator. Multivariate logistic regression analysis was performed and a forward stepwise variable selection algorithm with an α_{crit} of 0.30 applied to identify significant biochemical and clinical factors associated with symptomatic or biochemical hypocalcemia. The model conformed to standard assumptions for logistic regression. Testing of our model did not identify any confounders

or effect modifiers within the covariables. The study received approval from the University of Chicago institutional review board.

Results

During the study period, 229 patients underwent total thyroidectomy with or without neck dissection at our institution by one of six surgeons. Thirty-two patients with a preoperative diagnosis of hyperparathyroidism and/or renal failure were excluded. One patient was excluded for a postoperative diagnosis of hyperparathyroidism. The final study cohort consisted of 196 patients with a mean age of 47.1 ± 16.3 years (range, 10-82). One hundred fifty-eight (80.6%) of the patients were female. Preoperative diagnoses included symptomatic nodular goiter (31.1%), indeterminate thyroid nodule (28.1%), hyperthyroidism (19.4%), and malignancy (21.4%). Final pathology was benign for 93 (47.4%) of patients and malignant for 103 (52.6%) patients (see Table 1). In total 29 lymph node dissections were performed. All 29 lymph node dissections included a central compartment dissection, 3 had an additional selective lymph node dissection, 3 were unilateral modified radical neck dissections, and seven were bilateral modified radical neck dissections.

In total, nine (4.6%) patients developed symptomatic hypocalcemia following their procedure (Table 2). These patients' primary complaint was tingling/numbness of their extremities. One patient's symptoms were relieved with the initiation of calcium supplementation, five were relieved with an increased dose of calcium and three were relieved with an increased dose of calcium and calcitriol. No patients required IV calcium. Sixty-three (32.1%) patients developed biochemical hypocalcemia on post-operative day #1 AM lab draw. Univariate risk factors for the development of symptomatic hypocalcemia and post operative day #1 biochemical profiles are summarized in Table 3.

Calcium supplementation was initiated in 181 (92.3%) patients. Seven (3.9%) were prescribed 500 mg of calcium carbonate BID, 83 (45.8%) were prescribed 500 mg of calcium carbonate TID, and 91 (50.3%) were prescribed 1,000 mg of calcium carbonate TID. Calcitriol supplementation was initiated in 25 (12.8%) patients; 3 (12%) received 0.25 ug daily, 7 (28%) received 0.25 ug BID, and 15 (60%) received 0.5 ug BID. Of the 9 patients that developed symptoms of hypocalcemia, 8 (88.9%) had received calcium supplementation prior to developing symptoms 6 (66.7%) had received 500mg, 1 (11.1%) had received 1,000 mg and 1 (11.1%) had received 1,000mg and 0.25 ug of Calcitriol. Those developing symptomatic hypocalcemia were significantly more likely to have received calcium supplementation prior to developing symptoms ($p<0.01$) Of the 63 patients who developed biochemical hypocalcemia, 28 (44.4%) had not received calcium supplementation prior to their POD1 lab draw. Those with biochemical hypocalcemia on post operative day one significantly less likely to have received calcium supplementation ($p<0.01$). Calcium supplementation did not significantly affect the change in serum calcium levels noted between the PACU and POD1 ($p=0.79$).

The mean time to PACU calcium and PTH lab draw from surgery end time was 1.2 ± 0.5 hrs. The mean time to POD1 lab draw from surgery end time was 14.7 ± 1.2 hrs. Univariate

analysis of clinical and biochemical factors is summarized in Table 3. Significant biochemical predictors of symptomatic hypocalcemia on univariate analysis included PTH in PACU and on POD1. Linear regression demonstrated correlation between these two values ($R^2=0.682$) (Figure 2). The regression coefficient with constant suppression demonstrated that POD1 PTH was systematically lower than one-hour PTH (regression coefficient $\beta=0.80$; 95% CI={0.76-0.84}). This remains true in the subset of patients who did not receive calcium supplementation (regression coefficient $\beta=0.79$; 95% CI={0.75-0.84}) as well as those that did receive calcium supplementation (regression coefficient $\beta=0.83$; 95% CI={0.77-0.89}). Clinical risk factors identified on univariate analysis included performance of a central compartment lymph node dissection, and performance of a parathyroid autotransplantation. Indication for procedure, parathyroid noted in the pathology specimen, and gender were not statistically significantly associated with the development of symptomatic hypocalcemia.

Forward stepwise variable selection identified the following factors associated with symptomatic hypocalcemia: either POD1 or PACU PTH, along with central compartment lymph node dissection, and performance of a parathyroid autotransplantation. Notably, collinearity between POD1 and PACU PTH precluded them from being modeled together, suggesting these values are interchangeable. On multivariate analysis stepwise variable selection identified significant factors as PACU or POD1 PTH ≥ 10 ($p=0.14$), parathyroid autotransplantation ($p=0.11$), and performance of a central compartment lymph node dissection ($p=0.02$). This multivariate model predicted symptomatic hypocalcemia with odds ratio of 4.68 (95% CI: 1.26-17.35). Retroactively applying these risk factors identified on multivariate regression demonstrates that the utility of a postoperative PTH ≥ 10 at one hour or on post-operative day one predicts symptomatic hypocalcemia with a 44.4% sensitivity, 85.6% specificity with an AUC of 0.65 (95% CI 0.48-0.82) (Figure 3). The addition of clinical risk factors (central compartment lymph node dissection and parathyroid autotransplantation) improves this to 67% sensitivity, 70.1% specificity with an AUC of 0.75 (95% CI 0.55-0.95) (Figure 3) ($p=0.20$). Our final screen would only miss 3 cases of symptomatic hypocalcemia in the first 24 hours out of 196 patients (98% negative predictive value, 9.7% positive predictive value).

Clinical and biochemical factors associated with a decrease in serum calcium levels on POD1 were evaluated and summarized in Table 3. PTH at either one hour or on POD1 were associated with a statistically significant drop in POD1 serum calcium. Clinical factors associated with a decreased serum calcium on POD1 included age ≥ 45 years, the performance of a central compartment lymph node dissection, and parathyroid autotransplantation. Notably indication for procedure and presence of a parathyroid tissue in the pathology specimen were not associated with a change in POD1 serum calcium levels. On multivariate regression, factors associated with biochemical hypocalcemia (defined as a POD1 serum Ca ≤ 8.0 mg/dL) were PACU Ca ≤ 8.0 mg/dL ($p=0.139$), PACU or POD1 PTH ≥ 10 pg/dL ($p<0.01$), age ≥ 45 ($p<0.01$), and malignant final pathology ($p=0.16$). This multivariate model predicted biochemical hypocalcemia with an odds ratio of 7.44 (95% CI: 3.88-14.25) and 76.2% sensitivity, 30.1% specificity, 72.7% negative predictive value, and 34.0% positive predictive value with an AUC of 0.67 (95% CI (0.59-0.75) improving upon PTH at one-hour alone AUC=0.59 (95% CI 0.53-0.66) ($p=0.02$) (Figure 4).

Discussion

In this study, we prospectively examined and quantified the utility of biochemical and clinical risk factors associated with postoperative symptomatic and biochemical hypocalcemia. By measuring calcium and PTH levels one-hour after surgery and on POD1, we were able to demonstrate the utility of a one-hour post-operative PTH in predicting either symptomatic or biochemical hypocalcemia. Additionally, we included an evaluation of clinical risk factors previously identified as being associated with hypocalcemia in a multivariate model in order to increase the sensitivity of a biochemical test. By doing so, we are able to maximize our ability to identify patients at risk for developing symptomatic hypocalcemia.

We found PTH at one hour postoperatively or on POD1, along with the performance of a central compartment lymph node dissection and/or a parathyroid autotransplantation is associated with the development of symptomatic hypocalcemia. This demonstrates a single PTH within one hour of surgery is the best biochemical predictor of symptomatic and biochemical hypocalcemia after total thyroidectomy. The addition of any further laboratory studies does not add any clinical value to this single PTH level. Additionally, clinical risk factors such as a central compartment dissection or parathyroid autotransplantation should be taken into account. We hypothesize that the increased surgical manipulation of the parathyroid glands intra-operatively during either a central compartment lymph node dissection or parathyroid autotransplantation contributed to this increased risk of symptomatic hypocalcemia. The mechanism of this relationship in the setting of a normal PTH remains, however, unclear as those patients included in our screen using clinical factors alone have normal PTH values.

We postulated the indication for thyroidectomy may be a risk factor, given that Graves' disease and high levels of circulating thyroid stimulating hormone have been previously shown to be associated with postoperative hypocalcemia (6). Our subgroup analysis, however, demonstrated Graves' disease, hyperthyroidism, and preoperative TSH levels were not significant risk factors for symptomatic hypocalcemia in our cohort.

Many surgeons prescribe calcium repletion for all patients undergoing total thyroidectomy as a cost effective and uniform means of prophylaxing against this complication (24-26). Others, including our own group, strive for a more selective and still cost effective approach to preventing symptomatic hypocalcemia. Furthermore, as outpatient thyroidectomy becomes increasingly common, it is important to develop maximally sensitive screening tools to identify patients that may need closer observation such as a 23 hour observation admission (24). While the utilization of PTH has been well studied in recent years, the inclusion of clinical factors should not be overlooked. Although lacking statistical significance, the ability of clinical factors to improve this test's sensitivity in predicting symptomatic hypocalcemia is demonstrated in Figure 3. Utilizing a one-hour PTH supplemented with clinical risk factors provides a maximally sensitive algorithm which may facilitate day of surgery discharge in appropriate patients as well as initiate early calcium supplementation in patients at high risk for postoperative symptomatic hypocalcemia.

Limitations of this study include variability in the postoperative administration of calcium and/or calcitriol. This supplementation was based on surgeon judgment with respect to serum calcium and PTH levels as well as clinical factors felt to be significant. However, even the most aggressive supplementation would not have occurred prior to the one hour lab draw. At most one to two doses of calcium and/or calcitriol were given prior to the POD1 lab draw and did not affect the difference between PACU and POD1 levels ($p=0.79$ data not shown). This may, however, have affected rates of symptomatic hypocalcemia. Additionally, because the rate of symptomatic hypocalcemia in this cohort was comparatively low our screening algorithm, retroactively applied only missed three patients giving a negative predictive value of 98%. Despite this fact, only 67% of symptomatic patients were caught by this screening algorithm. Further prospective randomized study will be required to better describe these associations with larger cohorts of patients, especially those with symptoms of hypocalcemia. While our optimized screen was not highly sensitive, this work demonstrates no additional benefit to further laboratory studies, identifies pertinent clinical factors associated with symptomatic hypocalcemia, and would only under treat or not identify 1.5% of patients developing symptomatic hypocalcemia.

In conclusion, we found PACU PTH measured at 1 hour postoperatively was clinically equivalent to the POD1 PTH in predicting the development of hypocalcemic symptoms within 24 hours. Considering the advantages of having an equally predictive PTH level at 1-hour postoperatively, we believe the PACU PTH level is the best test and is the only necessary lab value to check postoperatively. When combining this value with central compartment lymph node dissection or parathyroid autotransplantation, the ability to predict symptomatic hypocalcemia is improved. Further studies with larger cohort of patients, however, will be needed to further delineate the significance of the addition of clinical factors to this screening algorithm for symptomatic hypocalcemia. We have utilized this algorithm within our own institution to create a clinical algorithm improving the selection of patients receiving calcium supplementation. We hope these results will help to lower overall hospital costs by minimizing lab draws and facilitating early discharge of low risk patients.

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Figure 1.

Post-operative calcium levels (mg/dL) measured in PACU and on POD1 listed by those with a PACU PTH of ≤ 10 pg/dL and those with a PACU PTH > 10 pg/dL

Figure 2.
PACU versus POD1 PTH levels. PACU PTH was found to correlate highly with POD1 PTH ($R^2=0.682$)

Figure 3. Receiver operating characteristic curve of the ability to predict symptomatic hypocalcemia for PTH alone AUC=0.65 (95% CI 0.48-0.82) and PTH with clinical factors AUC=0.75 (0.55-0.95) ($p=0.20$).

Figure 4. Receiver operating characteristic curve of the ability to predict biochemical hypocalcemia for PTH alone AUC=0.59 (95% CI 0.53-0.66) and PTH with clinical factors AUC=0.67 (0.59-0.75) ($p=0.02$).

Table 1

Cohort Demographics

	Total (n=196)
Age (mean)	47.1 ± 16.3
Gender	
Male	38 (19.4%)
Female	158 (80.6%)
Preoperative Laboratory Values	
PTH (n=24)	38.7 ± 15.9
Calcium (n=90)	9.2 ± 0.5
Vitamin D (n=10)	31.8 ± 15.4
Indication	
Multinodular Goiter or Symptomatic Nodule	51 (31.1%)
Indeterminate Nodule	55 (28.1%)
Cancer	42 (21.4%)
Graves' Disease or Toxic Multinodular Goiter	38 (19.4%)
Time to 1-hour Lab Draw (mean)	69 ± 34 min
Time to POD1 Lab Draw (mean)	881 ± 187 min
Final Pathology	
Benign	93 (47.4%)
Malignant	103 (52.6%)

Table 2

Patients Developing Symptomatic Hypocalcemia

	Age	M/F	Indication	PACU PTH	POD1 PTH	PACU Ca	POD1 Ca	LNDx [*]	Autotrxp [†]	Para in specimen	Supplementation on Discharge
Patient 1	54	F	goiter	2	2	8.7	8.7	no	yes	2	1000 TID CaCO ₃
Patient 2	43	F	malignancy	4	5	8.4	7.2	yes	yes	1	1000 TID CaCO ₃ 0.5 BID Vit D
Patient 3	17	F	malignancy	6	8	8.5	7	yes	no	1	1000 TID CaCO ₃ 1.25 BID Vit D
Patient 4	27	F	malignancy	9	8	8.8	7.9	yes	yes	2	1000 TID CaCO ₃ 0.5 BID Vit D
Patient 5	38	M	nodule	18	17	8.8	9	yes	yes	0	500 TID CaCO ₃
Patient 6	70	F	nodule	22	18	8.4	8.3	no	no	1	1000 TID CaCO ₃
Patient 7	78	F	goiter	36	24	8.7	7.9	yes	no	0	500 TID CaCO ₃
Patient 8	34	F	goiter	49	41	8.2	8.6	no	no	0	500 TID CaCO ₃
Patient 9	38	F	nodule	55	28	8.4	9	no	no	0	500 TID CaCO ₃

* LNDx - central compartment lymph node dissection performed

† Autotrxp - parathyroid allotransplantation performed

Table 3

Univariate Analysis of Predictors of Hypocalcemia

					POD 1			
	Sx	No Sx	OR	p	Calcium	p	PTH	p
Age				0.17		<0.01		0.84
0-45	6	81	2.62	-	8.13 ± 0.49	-	23.6 ± 13.8	-
>45	3	106	-	-	8.37 ± 0.55	-	23.9 ± 11.8	-
Gender				0.52		0.43		0.39
Female	8	150	1.97	-	8.25 ± 0.55	-	23.4 ± 13.0	-
Male	1	37	-	-	8.33 ± 0.48	-	25.4 ± 11.3	-
PACU Ca				0.36		0.13		0.69
8.0	0	16	0	-	8.07 ± 0.67	-	26.8 ± 15.4	-
> 8.0	9	171	-	-	8.28 ± 0.52	-	23.5 ± 12.4	-
PACU PTH				0.03		<0.01		<0.01
10	4	30	4.19		7.91 ± 0.63		6.9 ± 2.3	
> 10	5	157	-	-	8.33 ± 0.49	-	27.0 ± 11.2	-
POD1 Ca				0.41		NA		<0.01
8.0	4	59	1.73	-	NA	-	19.8 ± 12.4	-
> 8.0	5	128	-	-	NA	-	25.9 ± 12.3	-
POD1 PTH				0.02		<0.01		NA
10	4	27	4.74	-	7.91 ± 0.63	-	NA	-
> 10	5	160	-	-	8.33 ± 0.49	-	NA	-
LN dissection				<0.01		0.04		<0.01
performed	5	24	8.54	-	8.30 ± 0.52	-	25.4 ± 12.6	-
not performed	4	163	-	-	8.07 ± 0.58	-	14.1 ± 7.6	-
Parathyroid in specimen				0.09		<0.01		0.01
present	5	54	3.08	-	8.01 ± 0.52	-	25.8 ± 12.0	-
not present	4	133	-	-	8.37 ± 0.51	-	19.1 ± 12.9	-
Parathyroid Autotransplant				0.01		0.38		0.01
yes	4	24	5.25	-	8.18±0.54	-	24.8 ± 12.4	-
no	5	163	-	-	8.27±0.54	-	17.9 ± 13.0	-
Pathology				0.85		0.68		0.16
Malignant	5	98	1.14	-	8.25 ± 0.58	-	25.1 ± 1.4	-
Benign	4	89	-	-	8.25 ± 0.52	-	22.6 ± 1.2	<0.01