

Original Research Article

Hypoparathyroidism, an innate risk of thyroidectomy

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ABSTRACT

Background: The identification and preservation of parathyroid glands (PT) with its intact blood supply is of utmost importance during thyroid surgery. To preserve the PT with intact blood supply, a medial to lateral dissection is advocated, with plane of dissection along the thyroid capsule. One of the earliest and feared complications of thyroidectomy is hypoparathyroidism (HPT). Aim was to study the incidence of temporary and permanent hypoparathyroidism during thyroidectomy.

Methods: This was a hospital based prospective study, from 2008 to 2015. 472 cases who underwent thyroidectomy (182 near total and 290 total thyroidectomies) for any indications in general surgery department of a tertiary care centre were included.

Results: The incidence of permanent and transient HPT after total thyroidectomy was 3.8 and 09%, where as it was 2.75 and 3.85% after near total thyroidectomy. The results of present study were comparable with other studies.

Conclusions: Meticulous dissection, absolute hemostasis, and a thorough knowledge of neck anatomy are the key in reducing the post thyroidectomy complications. Transient or permanent hypo parathyroidism is due to inadvertent gland removal or injury to its vascular pedicle. Dissection close to the thyroid capsule and ligation of capsular branches of thyroid avoiding the main trunk of inferior thyroid artery holds the key.

Keywords: Hypocalcaemia, Hypoparathyroidism, Parathyroid, Tetany, Thyroidectomy complication

INTRODUCTION

The identification and preservation of parathyroid glands (PT) with its intact blood supply is of utmost importance during thyroid surgery.¹ The PT glands are four in number which arise from the third branchial pouch (inferior PT) and fourth branchial pouch (superior PT), and are seen as small bean shaped structures with yellow tan to caramel colour (Figure 1).² The superior PT are most consistently located within 1cm superior to the intersection of recurrent laryngeal nerve (RLN) and inferior thyroid artery (ITA). The inferior PT are variable

in location.³ The blood supply of parathyroid are by ITA with some variable component from superior thyroid artery (STA).⁴ To preserve the PT with intact blood supply, a medial to lateral dissection is advocated, with plane of dissection along the thyroid capsule. One of the earliest and feared complications of thyroidectomy is hypoparathyroidism (HPT). The PT produce parathyroid hormone (PTH) which is intimately involved in the regulation of serum calcium. PTH acts to increase serum calcium level by causing bone resorption, increased renal absorption of calcium, and stimulating the synthesis of the biologically active form of vitamin D (1,25-dihydroxy vitamin D). 1,25- dihydroxy vitamin D increases serum

calcium level by a number of mechanisms, including increasing intestinal absorption of calcium.⁵ PTH also increases renal phosphorus excretion. So low PTH result in high serum phosphorus levels. Inadequate production of PTH leads to hypocalcaemia. Hypoparathyroidism and the resulting hypocalcaemia may be transient or permanent. The rate of permanent HPT is 0.4-13.8% and of temporary HPT is 2-53%. This may occur due to direct trauma to the PT, devascularisation of the glands, or removal of them during surgery.⁶ The cause of transient HPT is not clearly understood. It may be attributable to reversible ischemia to the PT, hypothermia, or release of endothelin 1. Endothelin 1 is an acute phase reactant known to suppress PTH production, and have been found to be elevated in patients with transient HPT. The type of thyroidectomy performed also influences the risk of postoperative hypocalcaemia.

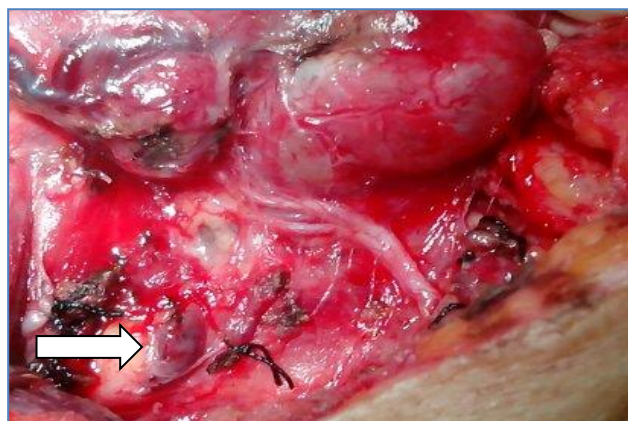


Figure 1: Right superior parathyroid with intact vascular pedicle.

Aim was to study the incidence of temporary and permanent hypoparathyroidism during thyroidectomy.

METHODS

This is a hospital based prospective study, from 2008 to 2015.

472 cases who underwent thyroidectomy (182 near total and 290 total thyroidectomies) for any indications in general surgery department of a tertiary care centre were included.

Data collection

A detailed history was taken and a physical examination was done for all patients apart from the routine investigations like thyroid function tests (TFT), indirect laryngoscopy (IL), serum calcium. Intra operative documentation of duration of surgery, bleeding, identification and safeguarding of PT were done. Post operatively the patients were monitored for any features of hypoparathyroidism. Apart from routine post-operative

investigations, serum calcium was checked routinely 24 hour after surgery in the morning and corrected for serum albumin levels. A fall in corrected serum calcium concentration below 8mg/dL, and/or the need for calcium supplementation was defined as temporary hypoparathyroidism. The need for oral vitamin D and/or calcium supplements six months following surgery to maintain a normal serum calcium concentration was branded as permanent hypoparathyroidism.

RESULTS

472 cases (290 total and 182 near total thyroidectomy) were analyzed during the study period. There was no intra or post-operative mortality in this study. The incidence of permanent and transient HPT after total thyroidectomy was 3.8 and 09%, where as it was 2.75 and 3.85% after near total thyroidectomy (Table 1). The incidence of both temporary and permanent hypoparathyroidism was more in females which may be due to more number of patients belonging to that sex. Most of the patients developed transient HPT within 48 hours after surgery. Few developed hypocalcemic symptoms before 48 hours especially those who were treated for toxic symptoms preoperatively.

Table 1: Incidence of permanent and transient hypoparathyroidism after total and near total thyroidectomy.

Hypoparathyroidism	Total thyroidectomy	Near total thyroidectomy
Transient	3.8%	2.75%
Permanent	9%	3.85%

DISCUSSION

Most patients who are hypocalcemic after thyroidectomy are initially asymptomatic. Symptoms and signs of hypocalcaemia include circumoral parasthesia, mental state changes, tetany, carpopedal spasm, laryngospasm, seizures, QT prolongation on ECG, and cardiac arrest.⁷ Evaluate ionized calcium in the perioperative period in patient undergoing total thyroidectomy (TT). If suspicion of iatrogenic hypoparathyroidism exists, close follow up care is warranted for at least 72 hours or until serum calcium demonstrate that parathyroid function is intact. Chovstek and Trousseau signs may be both be elicited at bedside in the setting of hypocalcaemia. The Chovstek sign is elicited by tapping the facial nerve in the preauricular area and observing for facial contractions; Trousseau's sign by induction of carpal spasm upon inflation of blood pressure cuff above the systolic value.^{8,9}

The best way to reserve the PT function is the deliberate identification of them and safeguarding its blood supply. In a cadaveric study, 77% of the superior PT was at the cricothyroid junction, intimately associated with the RLN. 22% were on the posterior surface of the upper part

of the thyroid gland. About 1% of the superior PT were located behind the junction of hypopharynx and upper esophagus.¹⁰ The position of inferior PT is more variable. 42% were found on the anterior or lateral surfaces of the lower lobe of the thyroid, often hidden by vessels or creases in the thyroid. 39% were located within the superior tongue of the thymus, 15% extrathyroidal and lateral to thyroid, 2% in mediastinal thymus and another 2% in carotid sheath.¹¹

The inferior PT receive the blood supply from ITA. The superior PT also receive the blood supply from ITA, but in some cases they receive the supply from superior thyroid artery (STA), the anastomotic loop between STA and ITA or from direct branches from thyroid. So during thyroidectomy ligation of only the capsular branches of ITA is the key to prevent the HPT. Recognition of the PT which appear in a variety shades is critical. When they lose the vascularity, they turn black. The devascularised gland (confirmed by frozen) should be removed and put in ice saline, loafed into small bites and reimplanted into muscle pockets in sternomastoid or brachioradialis.¹² Their location should be marked with a metal clip or nonabsorbable suture. Patients who have asymptomatic hypocalcaemia should not be treated with supplemental calcium. There is a school of thought that hypocalcaemic state stimulates the stunned PT to produce PTH.

Patients who have symptomatic hypocalcaemia in early postoperative period or whose calcium level continue to fall, require treatment. In a symptomatic patient, 10 ml 10% solution of calcium gluconate may be given i.v. over 10-20 minutes under cardiac monitoring.¹³ A calcium drip at the rate of 1-2mg/kg/hour may also be started in severe cases. Elemental oral calcium at the rate of 1-2 gms should be given daily. Calcium carbonate at a dose of 1250mg provides 500mg of elemental calcium, therefore a patient with symptomatic hypocalcaemia, should take 2500-5000mg of calcium carbonate a day. They may also need concomitant vitamin D replacement in the form of calcitriol at a dose of 0.25-1mcg/dl. Assistance from an endocrinologist to ensure close monitoring of calcium levels for the medical management of the sequel of HPT.

In 1-2 months, an attempt to wean the patient off oral calcium may be made to reveal if the HPT is temporary. Dependence on calcium supplementation for longer than 6 months probably indicate permanent HPT. According to Asari R et al patients with iPTH levels of 15 pg/mL or less and sCa levels of 1.9 mmol/L or less were at increased risk of developing postoperative hypoparathyroidism. Observation of sCa and iPTH levels independently showed different sensitivity and specificity on different postoperative days. Therefore, on the first 2 postoperative days, interpretation of iPTH levels in combination with sCa levels seems an optimal strategy for predicting patients at risk of hypoparathyroidism.¹⁴ The overall incidence of transient and permanent hypoparathyroidism was 7.3% and 1.5%, respectively in

a study by Thomusch O et al.¹⁵ Filho GJ demonstrated that there was transient hypocalcaemia in 27.5%, permanent hypocalcaemia in 5.1%.¹⁶ Ozbas S et al in his study showed that after subtotal thyroidectomy 8.2% developed transient hypocalcaemia and 2.4% and 0.6% patients developed transient and permanent RLN palsy respectively. In NTT group 12.2% developed transient hypocalcaemia and 0.6% had transient voice disturbances. None of the patients experienced permanent complications. In total thyroidectomy group, 30% had transient hypocalcaemia whereas only 0.4% suffered permanent hypoparathyroidism.¹⁷

According to Lee YS et al the most common surgical complication was symptomatic hypoparathyroidism, of which 28.4% of cases were transient and 0.3% permanent.¹⁸ Efremidou EI et al had permanent hypocalcaemia in 0.3% and temporary hypocalcaemia occurred in 7.3% of patients.¹⁹ In a study by Sreejayan et al the incidence of transient and permanent hypoparathyroidism was 21.87% and 3.25 respectively.²⁰

The results of present study are comparable with other available literature. The identification and preservation of all parathyroids were attempted especially the constant superior ones. Meticulous dissection and absolute haemostasis are the keys in this crucial step. Special care is taken to leave the branches to the parathyroids and ligate only the capsular branches of the inferior thyroid artery (ITA).

CONCLUSION

Meticulous dissection, absolute hemostasis, and a thorough knowledge of neck anatomy are the key in reducing the post thyroidectomy complications. Transient or permanent hypo parathyroidism is due to inadvertent gland removal or injury to its vascular pedicle. Dissection close to the thyroid capsule and ligation of capsular branches of thyroid avoiding the main trunk of inferior thyroid artery holds the key.

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