بسم الله الرحمن الرحيم

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بقسم التوثيق الإلكتروني بمركز الشبكات وتكنولوجيا المعلومات دون أدنى مسئولية عن محتوى هذه الرسالة.

ملاحظات:
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Meta-analysis of predictors of post-thyroidectomy hypocalcaemia

Thesis

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General Surgery

By

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Mahmoud Abdallah Bostamy El-kharadly

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List of Abbreviations

Abbreviation	Full term
RLN	recurrent laryngeal nerve
ITA	inferior thyroid artery
STA	superior thyroid artery
T3	triiodothyronine
T4	thyroxine
TH	Thyroid hormone
TRH	thyrotropin releasing hormone
TSH	thyroid stimulating hormone
Ca	Calcium
PTGs	Parathyroid glands
PTH	parathyroid hormone
P	Phosphorus
Mg	magnesium
CaSRs	calcium-sensing receptors
CBC	Complete Blood count
FBS	Fasting blood sugar
FNAC	Fine needle aspiration cytology
US	Ultrasound
PPV	positive predictive value
NA	Not available
CLND	Cervical lymph node dissection

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INTRODUCTION

Hypocalcemia is the most frequent complication after thyroid surgery.

Thyroid surgeries are among the most common in the world **(Karamanakos et al., 2010)**. Thyroid surgery is the definitive management option for thyroid malignancies, and also for benign diseases such as multinodular goiter with compression symptoms **(Shaheen et al., 2011)**.

Hypocalcemia following total thyroidectomy is a relatively frequent complication, which is sometimes difficult to correct. Temporary hypocalcemia occurs in 50–68% of post total thyroidectomy patients **(Rosato et al., 2004)**, while permanent hypocalcemia occurs in 3% of post total thyroidectomy patients **(D'Ajello et al., 2010)**.

Temporary hypocalcemia is defined by various authors as a post-surgery decrease in calcium (Ca) lasting for 6 to 12 months; permanent hypocalcemia is a post total thyroidectomy decrease in Ca lasting for more than 12 months **(Stack et al., 2015)**.

Post total thyroidectomy hypocalcemia depends on a number of factors, including biochemical blood parameters before and after surgery, clinical effects and factors related to surgery, surgical technique, surgeon's experience, the patient, and the disease **(Edafe et al., 2014)**.

Post-thyroidectomy hypocalcemia arises because of parathyroid removal, devascularization and damage which induce a state of transient or permanent hypoparathyroidism **(Sands et al., 2011)**.

Additional mechanisms, such as vitamin deficiency, an acute increase in calcitonin serum levels (because of gland handling during surgery) or an "hungry bone syndrome" are believed to contribute to this process **(Glinoer et al., 2000)**.

Etiological considerations include post-operative alkalosis-induced hypocalcemia resulting from hyperventilation triggered by postoperative pain, and dilution hypocalcemia **(Ozemir et al., 2016)**.

Eventhough the perfect knowledge of thyroid anatomy regarding the embryological origin of parathyroid glands is the most concrete element to decrease incidence of postoperative hypocalcemia **(Del et al., 2010)**.

Although the rate of hypocalcemia has decreased as parathyroid preserving techniques have developed, the rates of transient hypocalcemia still range between 6.9 and 49.0% of patient undergone thyroid surgery **(Kakava et al., 2016)**.

Surgeon's ability to predict the onset of post-thyroidectomy hypocalcemia is very important for postoperative management **(Goncalves et al., 2005)**.

Early detection of any risk of developing hypocalcemia will reduce the hospital stay length and eliminate unnecessary laboratory examinations **(Tomusch et al., 2003)**.

When hypocalcemia is predicted, treatment with prophylactic calcium and vitamin D supplements can prevent the development of hypocalcemia symptoms and premature discharge of patients **(Cranshaw et al., 2007)**.

Several studies tried to identify risk factors related to early hypocalcemia development after thyroid surgery, with different results **(Calò et al., 2017)**.

Postoperative hypoparathyroidism remains a clinical challenge for thyroid surgeons because of its frequency and the limited number of established preoperative predictors **(Rosato et al., 2004)**.

AIM OF THE WORK

This study aims to clarify factors related to the patient as well as surgical methods to find a significant correlations between them and early hypocalcemia development.

Chapter 1

ANATOMY OF THE THYROID AND PARATHYROID

Thyroid gland

A. Embryology

The thyroid gland formed from a pocket (marked by the foramen cecum) that separates from the surface of the boundary endoderm in the floor. Cells originate on the surface of the floor and descend into mesoderm above aortic sac and into the hypopharyngeal eminence as "cords". These cells continue to descend until they reach their final destination in the neck adjacent to the thyroid cartilage. This pathway forms a temporary duct (thyroglossal duct). There are abnormalities of incomplete or excessive descent of these thyroid precursor cells. The thyroid is one of the earliest endocrine organs to differentiate and has an important hormonal role in embryonic development. The early bundle of cells then forms the thyroid by first dividing to form 2 lobes separated by a narrow connecting isthmus**.** In the first trimester, the developing fetus is initially dependent upon maternal thyroid hormone crossing the placental barrier. Around week 16 the fetal thyroid becomes active enough to support the fetal requirements for neural development **(James et al., 2007) Figure (1).**

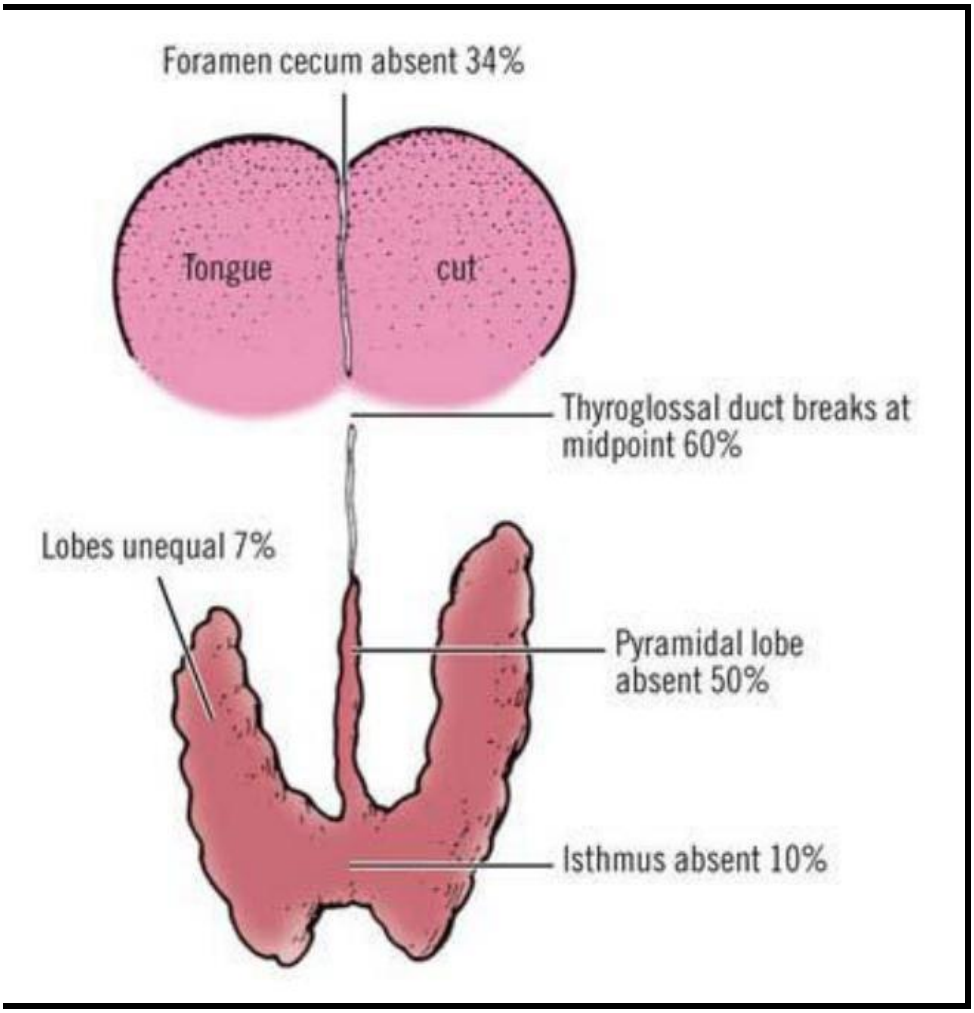

Figure (1): Vestiges of thyroid gland development **(Skandalakis et al., 2009).**

B. Morphology

The thyroid gland is a butterfly-shaped organ and composed of two cone-like lobes or wings, right lobe and left lobe, which are connected together by the isthmus **(Yalçin and Ozan, 2006).**