Prevention and Management of Post Thyroidectomy Hypocalcaemia

Essay

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In General Surgery

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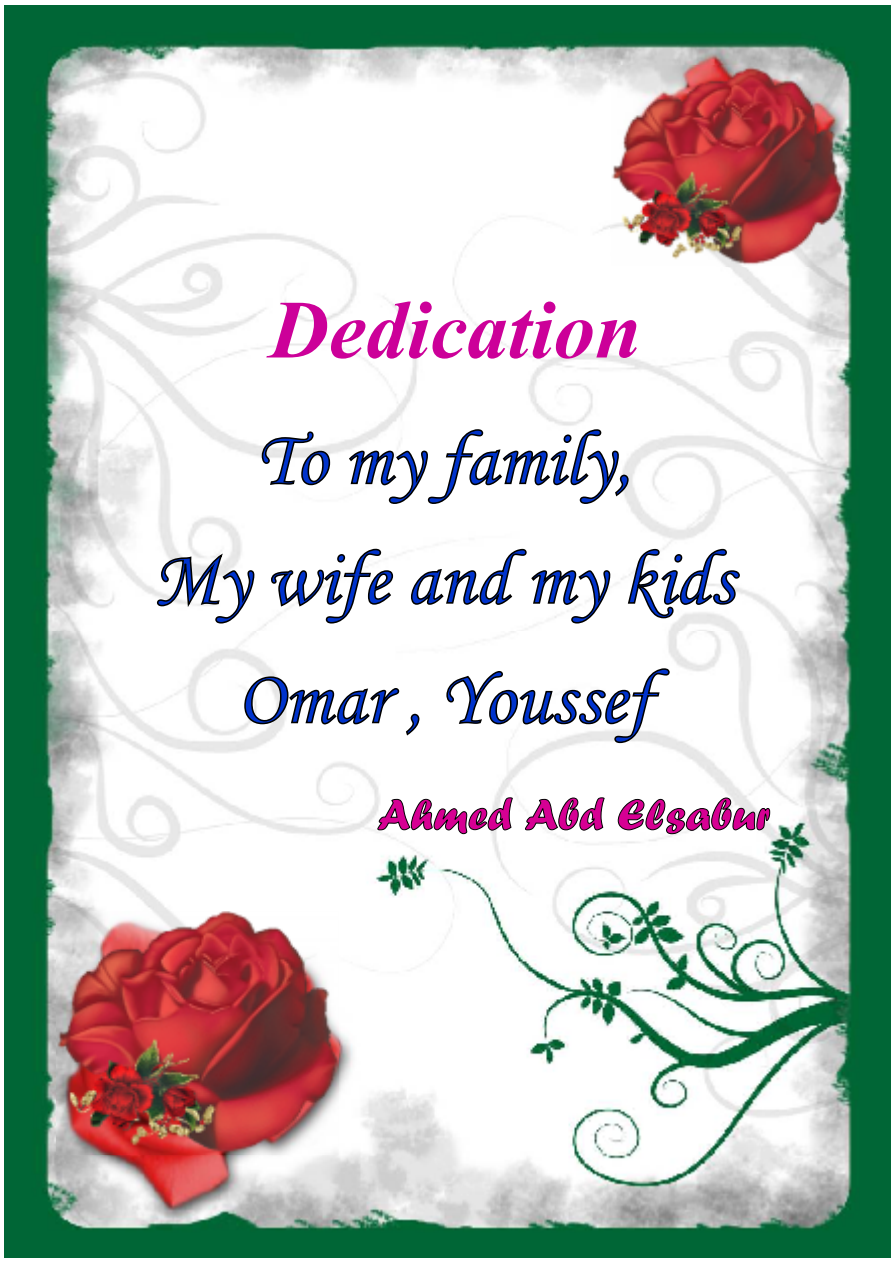
Dedication
To my family,
My wife and my kids
Omar , Youssef
Ahmed Abd Elsabur

First of all, Thanks Allah who allowed and helped me to accomplished this work.

I would like to express my deepest thanks and gratitudeTo **Prof. Dr. Abd Elghany Mahmoud Elshamy,** *Professor of Surgery Faculty of Medicine, Ain Shams University, for his interest, meticulous supervision, constant guidance.*

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Ahmed Abd Elsabur

بسم الله الرحمن الرحيم

قَالُوا سُبْحَانَكَ لاَ عِلْمَ لَنَا
إِلاَّ مَا عَلَّمْتَنَا إِنَّكَ أَنتَ
الْعَلِيمُ الْحَكِيمُ

صدق الله العظيم

سورة البقرة آية (٣٢)

List of Abbreviations

ACE	Angiotensin converting enzyme.
AHO	Albright hereditary osteodystropy.
ALS	Amyotrophic lateral sclerosis.
ALT	Alanine amino transferees.
ATP	Adenosine triphosphate.
CAMP	Cyclic adenosine monophosphate
CaSR	Calcium sensing receptor.
CHF	Congestive heart failure.
DIT	Diiodotyrosin
ECF	Extra cellular fluid.
ECG	Electro cardiography.
EDTA	Ethylene diamine tetra acetic acid.
GABA	Gamma aminobutyric acid.
GFR	Glomerular filtration rate.
Gs	Regulatory proteins
iPTH	Immunoreactive parathyroid hormone
MCT	Mono carboxylate transporter.
MIT	Monoiodotyrosin
NSAIDS	Nonsteroidal anti inflammatory drugs.
OATP	Organic anion transporting polypeptide.
PBi	Protein bound iodine
PTH	Parathyroid hormone.
rhPTH	Recombinant human parathyroid hormone

T٣	Tri iodothyronine
T٤	Tetra iodothyronine
TBG	Thyroxine binding globulin
TGB	Thyroglobulin
TPO	Thyroid peroxidase
TRH	Thyrotropin releasing hormone
TRS	Thyroid receptors
TSH	Thyroid stimulating hormone.
TTR	Transthyretin

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Introduction

Post operative hypocalaemia is one of the complications of thyroidectomy. It occurs more frequently after total thyroidectomy than after other more conservative thyroidectomies. The reported incidence of transient hypocalcaemia ranges, from ١.٦ % to ٩,٣٪ after subtotal thyroidectomy and from ٦,٩٪ to ٤٢٪ after total thyroidectomy. In contrast, permanent hypocalcaemia has been reported in ٠,٢ % to ٣ % of patients after subtotal thyroidectomy and in ٠,٤٪ to ٢٩٪ of patients after total thyroidectomy ***(Glinoer and andry,*** ***).***

Temporary hypocalcaemia may be attributed to calcitonin release due to operative manipulation of the thyroid gland ***(See and Soo,*** ***).***

The etiology of post thyroidectomy hypocalcaemia is still a debatable topic. Many factors, such as excision, de vascularization and infarction of parathyroid gland are involved. Moreover, the influence of surgical experience on the incidence of post thyroidectomy hypocalcaemia may be relevant. The importance of preserving the parathyroid glands and their blood supply can not be overstated ***(Cakmkli et al.,*** ***).***

Clinically significant postoperative hypocalcaemia could be predicted by the slope of change in calcium levels drawn within ٢٤ hours of surgery. Apositive slope in these calcium levels guaranteed that the patient would remain normocalcemic,whereas astrongly Negative slope fortold hypocalcemia. For thyroid surgery, calcium levels in the immediate postoperative perio (< ٢ hours) are not as useful for predicting hypocalcaemia as those occurring later (> ٨ hours) ***(Luu et al.,).***

Recently, patients are given prescriptions for oral calcium and calcitriol to minimize hypocalcemia. This treament has resulted in shorter hospitalization and fewer symptoms of tetany, but it has also resulted in imprecise assessment of outcome and over prescription of these medications. If the symptoms of hypocalcemia are severe and the patient appears to be on the verge of tetany the clinician may need to treat with i.v. calcium. These symptoms can usually be rapidly corrected by the infusion of ٢ mg/kg of elemental calcium over ١٥ min, while monitoring ECG and respiration, symptoms return unless a longer infusion is used. Approximately ١٥ mg/kg of elemental calcium is then infused over ٢٤h, with half the total amount administered in the initial ٦h. Serum levels of calcium should be monitored closely during the infusion, and infusion rates and amounts may be adjusted accordingly ***(Le and Norton,).***

Aim of the Work

To discus the different etiological and risk factors leading to post thyroidectomy hypocalcaemia and presentation as well as prevention and management protocols.

Thyroid Physiology and Calcium Metabolism

A - Thyroid Physiology

The thyroid gland develops in the first trimester of pregnancy, beginning around the fifth week of gestation, and its development is completed by the tenth week of gestation. It develops from median and paired lateral anlages. The median anlage arises in the midline oropharynx at the fourth to fifth gestational week and gives rise to follicular thyroid tissue, which will ultimately secrete hormones. The lateral anlages are believed to arise from the ultimobranchial bodies, which in turn are derived from the fourth and fifth branchial pharyngeal pouches at around the fifth week of gestation. They give rise to the parafollicular C cells that are thought to be derived from the neural crest ***(LiVolsi,)***.

The parafollicular cells ultimately secrete calcitonin. By the tenth week inutero, the right and left lateral anlages fuse with the median anlage, resulting in the bilobed thyroid gland ***(Pintar, ٢٠٠٧)***.

The thyroid develops from the endoderm in the floor of the pharynx and migrates downwards, anterior to the primitive gut. Faults in the process cause thyroglossal cyst and fistula and

the presence of thyroid tissue in abnormal situations, such as the posterior part of the tongue and retrosternally ***(Koutras and Alexander,*** ***).***

The gland is composed of a uniform cluster of follicles enclosed by athin fibrous capsule surrounded by capillaries. The follicles are the structural, functional, and secretory units of the thyroid gland ***(Rousset and Dunn,*** ***)***.

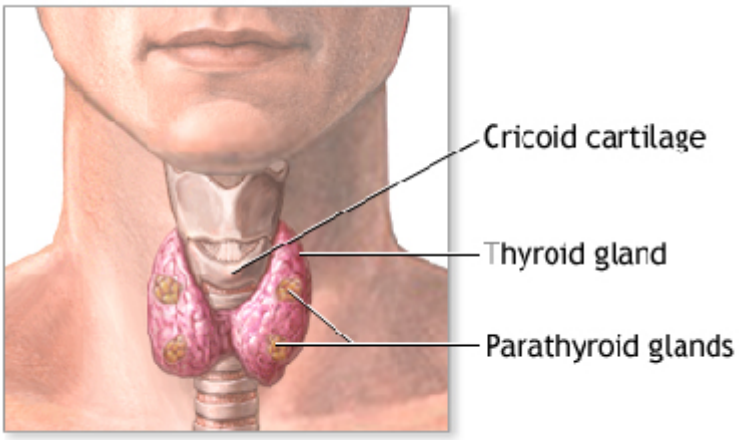

Figure (١): Thyroid and parathyroid glands ***(Russell and Edis,*** ***).***

Synthesis and Release of Thyroid Hormones

Iodine is a critical component of thyroid hormones and composes ٦٥٪ of T٤ weight and ٥٨٪ of T٣ weight. T٣ is the active hormone (٣ times the metabolic potency of T٤), and T٤ is the prohormone, broken down in the tissues to form T٣ as needed ***(Durmont and Corvilain,*** ***).***

Man requires ٠-١ mg per day to replace the iodine lost from the body, mainly in the urine, an amount which can be obtained by eating two average servings of sea fish aweek. The other main dietary sources of iodine are milk and eggs, but these contain little when produced in those island areas where water and soil are deficient in the element. In many countries potassium iodide is added to table salt ***(Koutras and Alexander,*** ***).***

In the alimentary tract dietary iodine is converted into iodide which is readily absorbed from the stomach and upper part of the small intestine. Iodide is concentrated within the thyroid and several other tissues. Normally in man the thyroid/serum iodide ratio is about ٥٠/١ but increases up to ٤٠٠/١ during treatment with antithyroid drugs. The iodine pump mechanism is located in the peripheral border of the follicular cells. The thyroid contains very little free iodide because iodide is very rapidly oxidized to nascent iodine, a