# **Anesthetic Management of Endocrinal Emergencies**

An essay submitted for partial fulfillment of

Master Degree in anesthesia by

## Shaimaa Sayed Ahmed Rashed

M.B.B.ch., AinShamsUniversity Under the supervision of,

## Prof. Samir Abdulrahman Elsebaay

Professor of Anesthesia and Intensive care Faculty of Medicine

AinShamsUniversity

## Dr. Sanaa Farag Mahmoud

Lecturer of anesthesia and Intensive care Faculty of Medicine

AinShamsUniversity

## Dr.Heba Abdulazem Labib

Lecturer of anesthesia and Intensive care Faculty of Medicine

AinShamsUniversity

Faculty of Medicine Ain-Shams University 2015

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#### Essay

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## Shaimaa Sayed Ahmed Rashed M.B.,B.Ch., Faculty of medicine – Ain Shams University

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## Prof. Dr. Samir Abdulrahamn Elsebaay

Professor of Anesthesia and Intensive care Faculty of Medicine - Ain Shams University

#### **Dr. Sanaa Farag Mahmoud**

Lecturer of Anesthesia and Intensive care Faculty of Medicine - Ain Shams University

### Dr. Heba Abdulazem Labib

Lecturer of Anesthesia and Intensive care Faculty of Medicine - Ain Shams University

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#### List of Abbreviations

ACTH..... Adrenocorticotropic hormone

AI..... Adrenal insufficiency

**BP**...... Blood pressure

CHF ...... Congestive heart failure

CT...... Computerized Tomography

**DKA.....** Diabetic ketoacidosis

**E**..... Epinephrine

ECF..... extracellular fluid

**ECG**..... Electrocardiographic

**GA.....** ganarel anesthesia

GIT..... Gastrointestinal tract

**HHS** ...... Hyperosmolar hyperglycemic state

**HPA** ...... hypothalamic-pituitary-adernal

**I** 131 ..... Iodine-131

ICU..... Intensive care unit

IM..... Intramuscular

IV..... Intravenous

LMA ..... larengeal mask airway

MI..... Myocardial infarction

MRI..... Magnetic Resonance Imaging

NE...... Norepinephrine

NS...... Normal saline

**PA.....** pituitary apoplexy

PCC..... Phaeochromocytoma

PTU..... Propylthiouracil

**RAIU** ...... Radioactive iodine uptake

Sc..... Subcutaneous

**T3**..... Tri-iodothyroxine

T4..... thyroxine

TIVA...... Total intravenous anesthesia

**TRH** ...... Thyrotropin-releasing hormone

**TSH.....** Thyroid-stimulating hormone

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#### INTRODUCTION

nesthetic management of endocrine surgical patients should Consider not only the organ of interest but also the end-organ consequences of the endocrine dysfunction and possible rare syndromes (Akhtar, 2012).

The ability to electively manage an anesthetic emergency by using combination of knowledge, skills and experience is vital. There are many causes of endocrine crisis during anesthesia. These can be broadly divided into: thyroid disease (thyroid storm and myxedema), diabetes mellitus (DKA, hypoglycemia), addisonian crisis, carcinoid crisis, Pheochromocytoma and parathyroid storm (*Alarifi et al.*, 2001).

Patients with endocrinopathies frequently present to the operating room. Although many of these disorders are managed on chronic basis, patients may have acute changes in the perioperative period that if left unrecognized, can have a negative effect on the perioperative morbidity and mortality. It is imperative that anesthesiologist understand the implications of surgical stress response on hormonal flux (Kohl and Schwartz, 2010).

Endocrine emergencies pose unique challenges for the attending anesthesiologist while managing critically ill patients. Besides taking care of primary disease state, one has to divert an equal attention to the possible associated endocrinopathies also. One

of the common reasons for inability to timely diagnose an endocrinal failure in critically ill patients being the dominance of other severe systemic diseases and their clinical presentation (Bajwa and Jindal, 2011).

Careful evaluation of clinical history and a high degree of suspicion are the corner stone to diagnose such problems. Aggressive management of the patient is equally important as the complications are devastating and can prove highly fatal (Goldberg and Inzucchi, 2003).

## **AIM OF THE STUDY**

To give a framework to think thorough when faced with the patient presenting with their endocrinal diseases in the theatre.

#### 100 (

## Perioperative Diabetes Mellitus <u>Emergencies</u>

### **Anatomy**

The pancreas, named for the Greek words *pan* (all) and *kreas* (flesh), is a 12-15–cm long J-shaped (like a hockey stick), soft, lobulated, retroperitoneal organ. It lies transversely, although a bit obliquely, on the posterior abdominal wall behind the stomach, across the lumbar (L1-2) spine (*Lewis*, 2000).

The endocrine pancreas consists of the islets of Langerhans, which are small endocrine glands scattered throughout the pancreas. The beta cell synthesizes pro-insulin which is converted to insulin and C-peptide after proteolytic cleavage. Both C-peptide and insulin are released in the circulation in equomolar amounts. Insulin's half-life is 3-5 minutes and about 50% of it is cleared in a single pass through the liver. Approximately 1/3-1/2 of total daily insulin is basal insulin, which is secreted in the fasting state. The rest is secreted as bolus (stimulated) insulin in response to exogenous stimuli. The main stimulus for insulin release is circulating glucose. Other stimuli for insulin release including: amino-acids, ketoacids, beta-catecholamines, and certain gut hormones (*Ordovas et al.*, 2003).

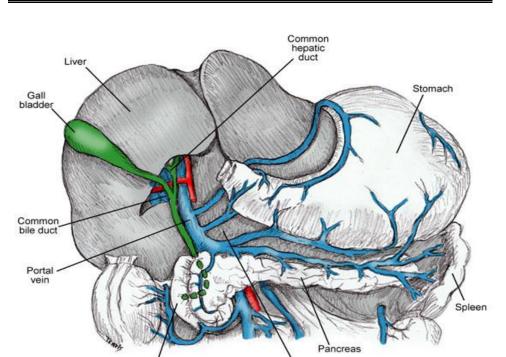


Fig. (1): Pancreas and surrounding anatomical structures (*Lewis*, 2000).

#### **Definition and description of diabetes mellitus**

Diabetes is a group of metabolic diseases characterized by hyperglycemia resulting from defects in insulin secretion, insulin action, or both. The chronic hyperglycemia of diabetes is associated with long-term damage, dysfunction, and failure of different organs, especially the eyes, kidneys, nerves, heart, and blood vessels (*Genuth et al.*, 2003).

Several pathogenic processes are involved in the development of diabetes. These range from autoimmune destruction of the  $\beta$ -cells of the pancreas with consequent insulin deficiency to abnormalities that result in resistance to insulin action. The basis of the

abnormalities in carbohydrate, fat, and protein metabolism in diabetes is deficient action of insulin on target tissues. Deficient insulin action results from inadequate insulin secretion and/or diminished tissue responses to insulin at one or more points in the complex pathways of hormone action (*Genuth et al.*, 2003).

The majority of cases of diabetes fall into two broad etiopathogenetic categories. In one category, type 1 diabetes, the cause is an absolute deficiency of insulin secretion. Individuals at increased risk of developing this type of diabetes can often be identified by serological evidence of an autoimmune pathologic process occurring in the pancreatic islets and by genetic markers.

In the other, much more prevalent category, type 2 diabetes, the cause is a combination of resistance to insulin action and an inadequate compensatory insulin secretory response. In the latter category, a degree of hyperglycemia sufficient to cause pathologic and functional changes in various target tissues, but without clinical symptoms, may be present for a long period of time before diabetes is detected. During this asymptomatic period, it is possible to demonstrate an abnormality in carbohydrate metabolism by measurement of plasma glucose in the fasting state or after a challenge with an oral glucose load (American Diabetes Association 2012).

Identification and management of emergencies in diabetes is important to prevent mortality. Three major life-threatening