Anesthetic Management of A diabetic Child

Essay

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By

Maii Ali Mohammed Saadallah

M.B.B.CH

Under supervision of

Prof. Dr./ Nabila Mohamed Abdel-Aziz Fahmy

Professor of Anesthesiology and Intensive Care
Faculty of Medicine-Ain Shams University

Dr./ Mostafa Gamal-Eldin Ahmed Mahran

Lecturer of Anesthesiology and Intensive Care
Faculty of Medicine-Ain Shams University

Dr./ Ahmed Mohammed Elsayed Ibrahim

Lecturer of Anesthesiology and Intensive Care
Faculty of Medicine-Ain Shams University

Faculty of Medicine
Ain Shams University
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List of Abbreviations

ACTH	**:**	Adereno-corticotrophic hormones
ADA	**:**	America diabetes association
DKA	**:**	Diabetic ketoacidosis
DM	**:**	Diabetes mellitus
DPP-4	**:**	Dipeptidyl peptidase-4
FPG	**:**	Fasting plasma glucose
GA	**:**	General anesthesia
GIP	**:**	Glucose-dependent insulin tropic peptide
GLP	**:**	Glucagon-like peptide
HgbAlc	**:**	Glycosylated hemoglobin
HLA	**:**	Human leucocyte antigen
IAA	**:**	Insulin autoantibodies
ICA	**:**	Islet cell autoantibodies
IDDM	**:**	Insulin-dependent diabetes mellitus
IFG	**:**	Impaired fasting glycemia
MODY	**:**	Maturity onset diabetes of youth
NPH	**:**	Neutral protamine Hagedorn
OGTT	**:**	Oral glucose tolerance test
SC	**:**	Subcutaneous

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Introduction

Surgical operations needing general anesthesia carry a greater risk to the child with diabetes than to the child without diabetes. Every such surgical procedure and anesthesia should be taken seriously and meticulously to prevent preoperative hypoglycemia, hyperglycemia, or electrolyte disturbance ***(Rhodes et al., 2005).***

Surgical operations for children with diabetes include elective surgeries. About half of these are for dental procedures and the rest mostly for ear, nose, and throat operations. They also include emergency operations such as appendectomy or trauma ***(Golden et al., 2009).***

Goals of anesthetic management of diabetic child are to minimize physiological stress, to maintain euglycemia, to avoid ketoacidosis and to minimize the risk of postoperative infection ***(Canadian diabetes association, 2003).***

The peri-operative plan should be developed in consultation with a pediatric endocrinologist, as management of these patients is becoming increasingly complex due to the complexity and variability of current diabetes treatment options ***(Cruse and Foord, 2003).***

Anesthesiologist must carefully consider not only the pathophysiology of the disease, but also each child's specific diabetes treatment regimen, glycemic control, child metabolic state, age, pubertal development, intended surgery and its length, and anticipated postoperative care when devising an appropriate peri-operative management ***(Glister and Vigersky, 2003).***

Optimal management should maintain adequate hydration and near to normal glycemic ctrol, while minimizing the risk of hypoglycemia. The stress of surgery may cause acute hyperglycemia, which increases the risk of postoperative infection ***(Van et al., 2003).***

Centers performing surgical procedures on children with diabetes should have available written protocols for post-operative management of diabetes on the wards where children are admitted ***(Pomposilli et al., 2003).***

Aim of the Essay

The aim of our study is to discuss the proper anesthetic management of a diabetic child to maintain euglycemia and minimize the physiological stress of surgery, thus avoiding the possible complication in the form of hyperglycemia, hypoglycemia and ketoacidosis.

Pathophysiology of Diabetes Mellitus

Pancreas

The pancreas is a gland in the digestive and endocrine system of vertebrates **(Fig. 1).** It is both an endocrine gland producing several important hormones, including insulin, glucagon, and somatostatin, as well as an exocrine gland, secreting pancreatic juice containing digestive enzymes that pass to the small intestine. These enzymes help in the further breakdown of the carbohydrates, protein, and fat in the chime ***(Medvei and Victor, 2003).***

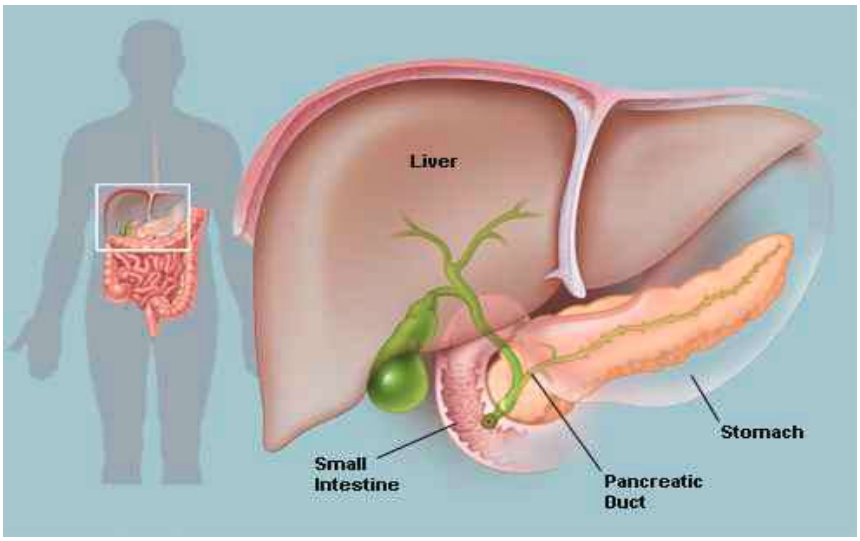

Fig. (1): Anatomy of the pancreas ***(Cabrera et al., 2006).***

The pancreas is a dual-function gland, having features of both endocrine and exocrine glands:

Endocrine function:

The part of the pancreas with endocrine function is made up of approximately a million cell clusters called islets of Langerhans. Four main cell types exist in the islets. They can be classified by their secretion: α cells secrete glucagon (increase Glucose in blood), β cells secrete insulin (decrease Glucose in blood), δ cells secrete somatostatin (regulates/stops α and β cells), and pancreatic polypeptide cells secrete pancreatic polypeptide ***(Rother, 2007)***.

Exocrine function:

In contrast to the endocrine pancreas, which secretes hormones into the blood, the exocrine pancreas produces digestive enzymes and an alkaline fluid (referred to as pancreatic juice), and secretes them into the small intestine through a system of exocrine ducts in response to the small intestine hormones secretin and cholecystokinin. Digestive enzymes include trypsin, chymotrypsin, pancreatic lipase, and pancreatic amylase, and are produced and secreted by acinar cells of the exocrine pancreas. Specific cells that line the pancreatic ducts, called centroacinar cells, secrete a

bicarbonate- and salt-rich solution into the small intestine ***(Lawrence et al., 2008).***

Glucose control:

Glucose regulation is an exquisite orchestration of many hormones, both pancreatic and gut, that exert effect on multiple target tissues, such as muscle, brain, liver, and adipocyte. While health care practitioners and patients have had multiple therapeutic options for the past 10 years, both continue to struggle to achieve and maintain good glycemic control ***(Stephen et al., 2004).***

Plasma glucose concentration is a function of the rate of glucose entering the circulation (glucose appearance) balanced by the rate of glucose removal from the circulation (glucose disappearance). Circulating glucose is derived from three sources:

1. Intestinal absorption during the fed state
2. Glycogenolysis
3. Gluconeogenesis

The major determinant of how quickly glucose appears in the circulation during the fed state is the rate of gastric emptying. Other sources of circulating glucose are derived chiefly from hepatic processes: glycogenolysis, the breakdown of glycogen, the polymerized storage form of glucose; and gluconeogenesis, the formation of glucose

primarily from lactate and amino acids during the fasting state. Glycogenolysis and gluconeogenesis are partly under the control of glucagon, glycogenolysis is the primary mechanism by which glucose is made available, and Glucagon facilitates this process and thus promotes glucose appearance in the circulation. Over longer periods of fasting, glucose produced by gluconeogenesis is released from the liver ***(Stephen et al., 2004).***

Glucoregulatory hormones include insulin, glucagon, amylin, Glucagon-like peptide-1 (GLP-1), glucose-dependent insulinotropic peptide (GIP), epinephrine, cortisol, and growth hormone.

Insulin and amylin are derived from the β-cells, glucagon from the α-cells of the pancreas, and GLP-1 and GIP from the L-cells of the intestine. Those glucoregulatory hormones of the body are designed to maintain circulating glucose concentrations in a relatively narrow range. In the fasting state, glucose leaves the circulation at a constant rate. To keep pace with glucose disappearance, endogenous glucose production is necessary. For all practical purposes, the sole source of endogenous glucose production is the liver. Renal gluconeogenesis contributes substantially to the systemic glucose pool only during periods of extreme starvation ***(Defeo et al., 2001).***