GLYCOSURIA IN SURGERY

ESSAY

Submitted in Partial Fulfilment for the Degree (M.Sc.)

(SURGERY)

BY

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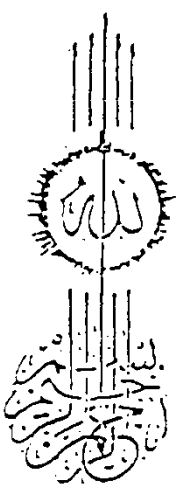

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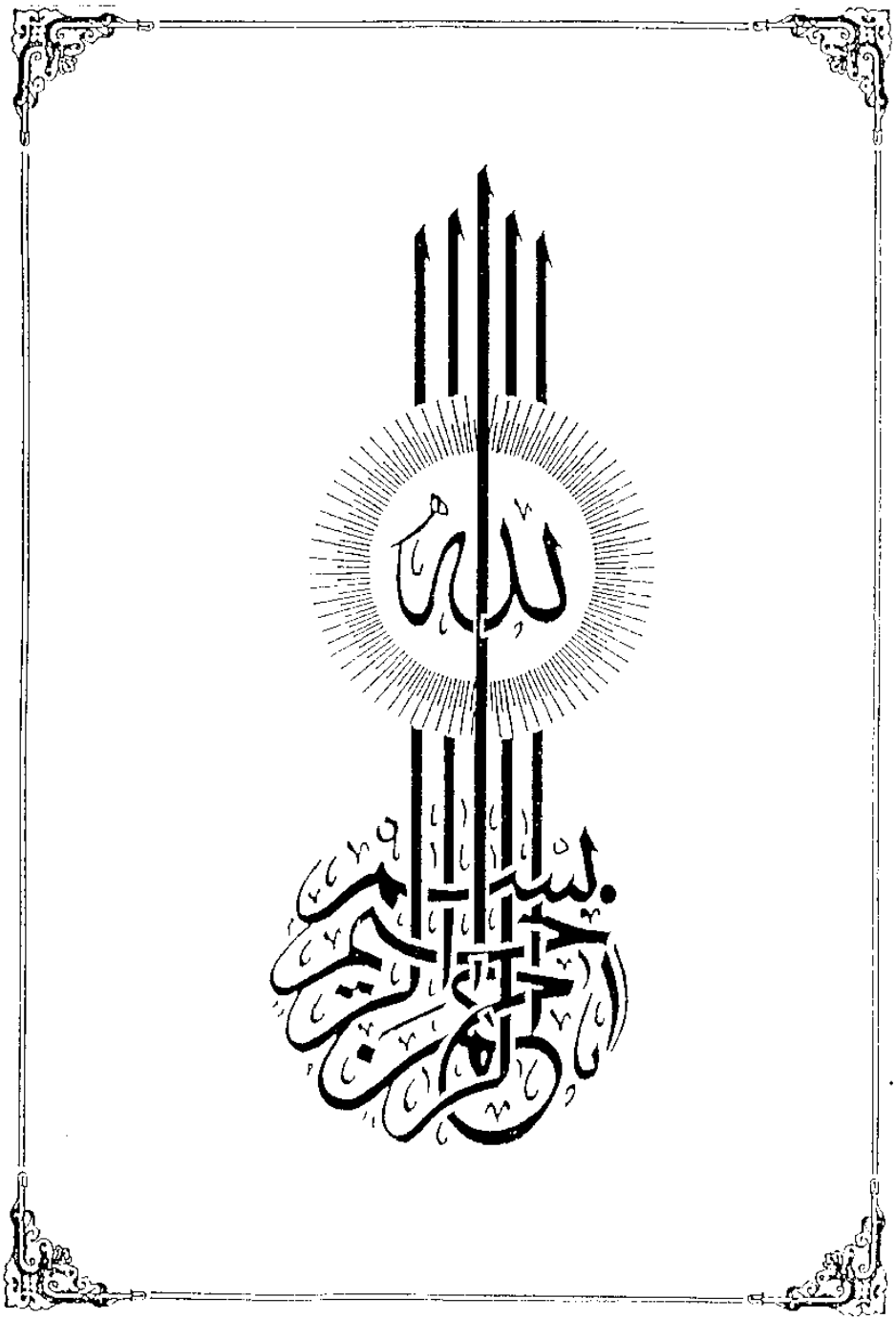

ACKNOWLEDGMENT

It is of great pleasure to me to express my sincere gratitute, deep thanks and apperciation to Dr. ABOU BAKR EL SEDDIK MOSTAFA, professor of general surgery for his faithful supervision guidance and encouragement throughout the accomplishment of this work.

Also I wish too heartly to thank Dr. MOSAAD G. EL BEHIERI, lecturer of general surgery for his kind supervision and encouragement throughout this work .

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C H A P T E R I

INTRODUCTION

CLINICAL TYPES OF GLYCOSURIA

Glycosuria is an important subject for surgeons as they always face it during surgical practice.

Thus the surgeon must be alert to this group of patients.

The glycosuric patients could be glycosuric diabetics or glycosuric non-diabetics.

The glycosuric diabetic patients should be considered as a group of high risk patients that require spacial attention.

The glycosuric non-diabetic patients are considered as normal individuals and they can undergo surgery without problems.

ACCORDING TO DUNCAN 1965 :-

Glycosuria is the presence of sugar in urine which reduces copper to cuprous oxide which may be mistaken for glucose.

If this reducing substance is glucose, it is glucosuria.

Fructosuria, Pentosuria, Lactosuria and Galactosuria are terms used when the sugar is found to be fructose, Pentose, Lactose and galactose respectively in urine. Until recently the metallic oxide reduction test, despite its non specificity, was the most widely used method for detection of melliturie. The sugars capable of prodcing a positive test by this method are glucose, Fructose, Pentose Galactose, Lactose and maltose.

Non sugar reducing substances which may produce a positive reaction include uric acid, nucleoproteins, conjugate glucuronates such as glucuronides of metabolites of salicylates, comphorchloral hydrate, morphine, certain antibiotics and homogentisic acid.

Afalse-positive benidict's test may result when testing the urine of patients receiving certain antiboitics due to excretion of large amounts of glucuronic acid which may produce a green cloudy solution with a small amount of yellow green precipitate.

Inhibition of glucose oxidase by ascorbic acid therapy may prevent the detection of significant degrees of glycosuria.

Urinary sugars :- (According to Duncan 1965)

Urine is examined routinely to detect or determine the presence of glucose, this is done either as screening procedure or as a guide to insulin therapy.

Other sugars may also appear in the urine and interfer with the detection & determination of glucose.

The duration, as well as the degree of hyperglycemia affects the degree of glycosurea.

The rate of urine formation is variable and the degree of glycosuria is a reflection of the blood sugar perior to the time the urine is devoided.

The relationship between the blood sugar value and the urine content may vary in either directions, depnding upon whether the blood sugar is rising or falling prior to time of emptying the bladder.

The renal threshold is lowered in some cases of normal pregnancy. But some patients who have glycosuria only during pregncancy develop clinical diabetis at alater date.

The renal threshold may be elevated because of nephritis and nephrosclerosis and in patients with long standing diabetes.

Thus glycosuria depends on

(1) Arterial blood sugar concentration

(2) Glomerular filteration rate

(3) Tubular reabsorption of glucose

Patients who have glycosuria are those with normal renal threshold, and an elevated blood sugar value.

Incidence :-

The percentage of female positive glycosurics were particularly low compared with those of males.

There is also an increase in the incidence of the diabetic type in recent years. The age distribution of the diabetic type among these subjects showed an increasing number of younger sufferers. (Baba et al 1982)

ETIOLOGY OF GLYCOSURIA :-(According to Zilva and Pannal 1975)

If Glycosuria and hyperglycemia are found, this condition is known as diabetes mellitus. The blood glucose is normally regulated by three hormones, insulin, glucagon and growth hormone, also corticosteroids and adrenalin have an effect. As insulin and glucugen have an opposing effect of blood glucose level, their molar ratio is more improtant than the concentration of either, Ahigh insulin/glucagon ratio lowers glucose concentration.

Scott and Fisher 1938, found that the average amount of insulin in the normal human was 1.7 units per gm in contrast to 0.4 units per gm in the diabetic. Adecreased production of insulin by the pancreas could explain the presence of diabetes in some cases.

(1) Primary hyperglycemic glycosuria :-

The basic lesion may be in the B cells of the pancreas which is present in Juvenile diabetes. The abnormality may be primarily extra pancreatic and long continued antagonism or resistence to insulin action which may lead to exhaustion of B cells. Variants of this theory consider the primary fault in the metabolism of fat, excess free fatty acids antagonizing insulin. This theory may explain "maturityonset diabetes" in obese subjects. There is some evidence that in insulin defficiency, there is increased glycoprotein synthesis and that this may lead to capillary basement membrane thickening. This theory may explain the vascular lesion (Zilva and Bannal 1975)

(2) Secondary hyperglycemic glycosuria :-

Normally, meals are separated by periods of fasting.

Change in secretion of certain hormones results, in order to,

(1) Maintain glucose concentration within relatively narrow limits.

(2) Maintain glycogen stores adequate for emergency needs, while converting excess carbohydrate into fat.

The effect of glucose on insulin secretion is potentiated by amino acids and possibily by gastro-intestinal hormones. A fall of glucagon and growth hormone level in the plasma result in insulin dominance. A fall of blood glucose after two hours is due to that the rate of utilization exceeds the rate of absorption i.e. Blood glucose equal the difference between the tissue needs and body stores in the tiver. Failure of blood glucose concentration to fall below 6.7 m mole/L (120 mg/dL) by two hours may occur in

(1) Insulin defficiency (diabetes mellitus)

(2) Growth hormone excess (acromegaly)

(3) Excess of other hormones antagonizing insulin

- Corticosteroids (Cushing's syndrome and stress)
- Adrenaline (pheochromocytoma)

(Duncan 1965)

I Pancreatic diabetes :-

Diabetes may result from surgical removal or destructive disease of the pancreas. The causes of pancreatic diabetes are :-

(1) Total pancreatectomy

(2) Pancreatitis

a- acute or subacute

b- chronic

(3) Carcinoma of the pancreas

(4) Haemochromatosis.

(Oakly and PYKE 1975)

(A) Pancreatectomy :-

The diabetes which follows total pancreatectomy, contrary to some accounts, is severe, but can always be controlled by relatively small dose of insulin, usually 20-40 units daily. If insulin is withheld there is a rapid onset of insulin Ketoacidosis.

(Oakly and PYKE 1975)

(B) Pancreatitis :- (Acute pancreatitis);-

Acute diabetes is a rare complication of acute pancreatitis. The glucose tolerance test is often ab normal, but impairment of glucose tolerance is almost always temporary. In the established diabetic, acute pancreatitis greatly increases insulin requirement and may precipitate diabetic coma. With recovery from pancreatitis the insulin requirement usually returns to its former level. (Oakly and PYKE 1975)

( ) Chronic Pancreatitis :-

With progressive destruction of the pancreas both exocrine and endocrine function become impaired with the development of diabetes and steatorrhea.

(Oakly and PYKE 1975)

Diabetes like steatorrhea is more common in calcific than in non calcific pancreatitis.

Banks et al 1975, found that, overt diabetes was present in 30% of non calcific and 70% of calcific.

pancreatitis. While lind et al 1977, found that 61% of patients with chronic calcific pancreatitis have overt diabetes, The incidence of diabetes mellitus increased to 50% for non calcific and 90% for calcific pancreatitis if the diagnosis rests with abnormal glucose tolaronce,test rather than overt diabetes.

Criteria of pancreatic diabetes :-

Vascular complications in pancreatic diabetes is rare and this is not fully explained. A defficiency in glucagon in chronic pancreatitis prevents the development of ketonuria by reducing the liver capacity to convert