

DIABETES MELLITUS

An Overview

Compiled by

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CMG Archives

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IMPORTANT

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Introduction

The pancreas, with its function of insulin secretion, is the organ that is often associated with Diabetes Mellitus.

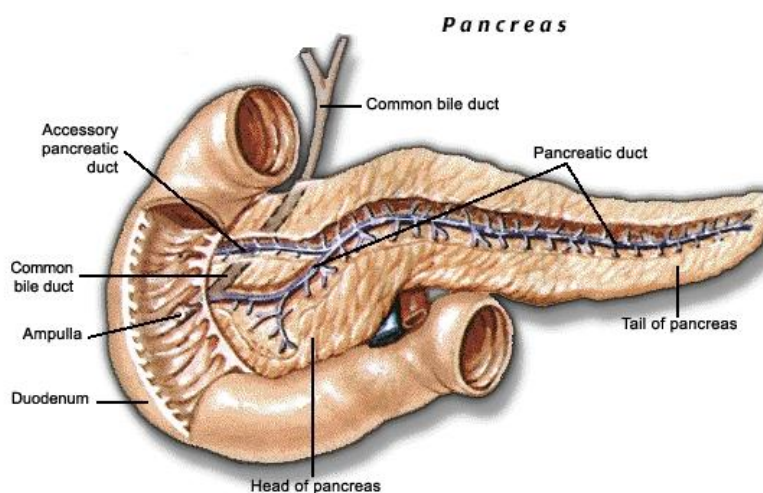
The pancreas

The pancreas is a fish-shaped, greyish-pink gland about 5 inches long that stretches across the back of the abdomen, behind the stomach. With a lumpy surface, the pancreas is divided into three parts – 1) the head, 2) the body, and 3) the tail. The pancreas is the second largest gland in the body and is both an endocrine and an exocrine gland.

(Picture right – the Pancreas)

Exocrine Function

The exocrine function of the pancreas is to produce digestive juices (pancreatic juices/enzymes of digestion) and to release them through a tube, the pancreatic duct, to



the intestine.

Endocrine Function

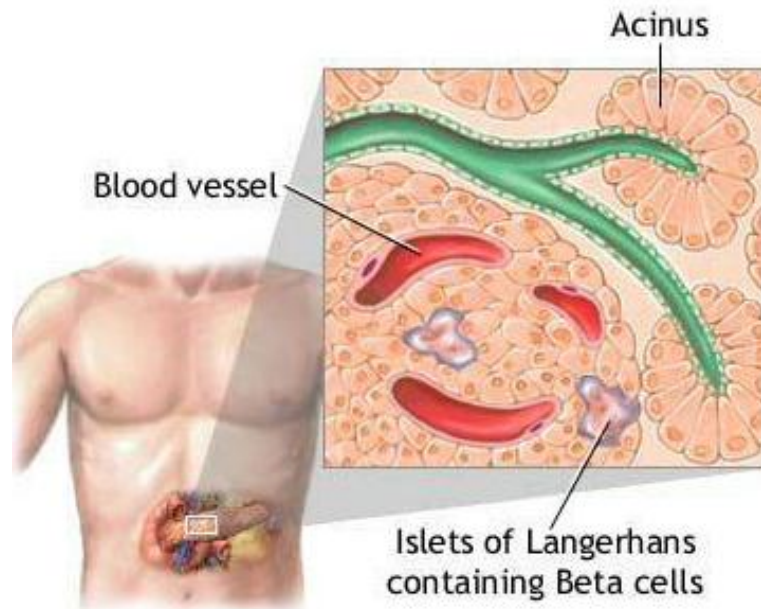
The endocrine function of the pancreas is to control the amount of sugar in the blood by releasing insulin or glucagon.

Islets of Langerhans

Small ducts from the releasing cells empty into the main duct that runs the length of the organ. The main duct empties into the intestine at the same spot as the exit of the common bile duct. The cells that control blood sugar levels are called (there are about 1 million cell units).

(Picture right – Islets of Langerhans)

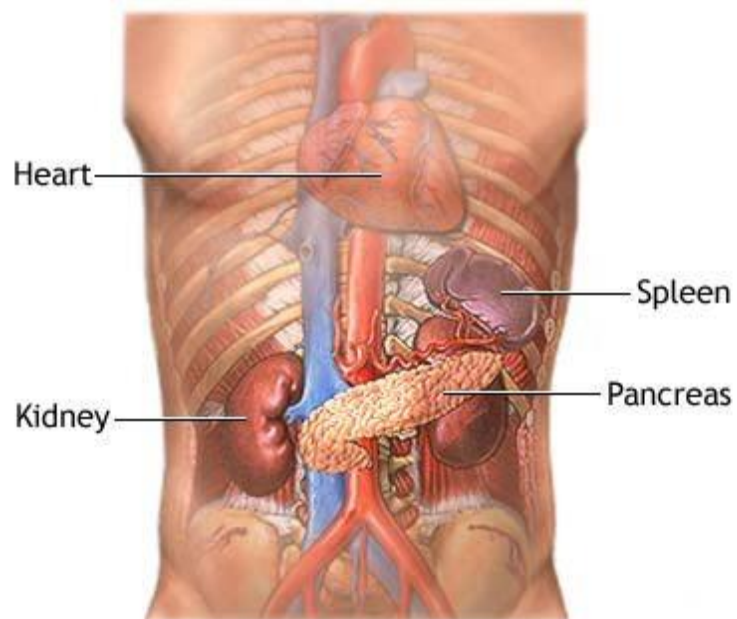
These islets are microscopic clumps of cells scattered throughout the pancreatic tissue among the other pancreatic cells, but are concentrated somewhat in the tail of the pancreas. There are two kinds of cells in the islets, alpha and beta. The alpha cells secrete a hormone called glucagon and the beta cells secrete insulin.



Insulin and glucagon work as a check and balance system regulating the body's blood sugar level. Glucagon accelerates the process of liver glycogenesis (a chemical process by which the glucose stored in the liver cells in the form of glycogen is converted to glucose. This glucose then leaves the liver cells and enters the blood). This process tends to increase the concentration of glucose in the blood. Insulin is an antigen to glucagon. It decreases the amount of blood glucose concentration. Insulin decreases blood glucose by accelerating its movement out of the blood, through cell membranes, and into cells.

(Picture right – Position of the Pancreas)

As glucose enters the cells at a faster rate, the cells increase their metabolism of glucose. All sugary and starchy foods, such as bread, potatoes, and cakes, are broken down into glucose. In this form they can be absorbed by every cell in the body, including the cells in the liver, one of whose major roles is to store sugar. Cells absorb glucose and burn it in structures called mitochondria, using the energy it contains and producing carbon dioxide and water as by-products. This burning up process is the body's



principle source of energy. It cannot take place without insulin.

Diabetes

Diabetes occurs when the pancreas fails to secrete enough insulin and so fails to regulate the glucose concentration in the blood. The normal glucose level for an average adult is about 80 to 120mg of glucose in every 100ml of blood. If the islets of Langerhans secrete too little insulin an excess of glucose develops, a characteristic of diabetes mellitus the most common disorder of the endocrine system.

In summary, beta cells of the islets release insulin, which helps control the body's use of carbohydrate. Alpha cells of the islets release glucagon, which counters the action of insulin. Other units of the pancreas release enzymes that help digest fats and proteins.

Diabetes mellitus

Diabetes Mellitus is the commonest form of diabetes, caused by a deficiency of insulin, in which sugar and starch are not properly metabolized. Symptoms include the blood and urine containing excessive amounts of sugars, causing a risk of convulsions and coma. Diabetes Mellitus is a disease complex characterised by persistent hyperglycaemia caused by insufficient insulin production (e.g. pancreas problems) or resistance to the metabolic action of insulin. Diabetes mellitus (DM) is generally classified as Insulin-Dependent Diabetes Mellitus (IDDM, type I), or Non-Insulin-Dependent Diabetes Mellitus (NIDDM, type II), or Secondary Diabetes Mellitus.

Causes and Incidence of Diabetes Mellitus

The precise causal mechanisms in s are unknown, although genetics and a faulty autoimmune response are thought to play major roles in type I diabetes. Genetics and obesity are risk factors for type II diabetes. Secondary diabetes is caused by underlying primary pathologic abnormalities. Diabetes Mellitus affects approximately 6% of the western population, and it is the leading cause of irreversible blindness and chronic renal failure. However, diabetes is also found world-wide, and its incidence is increasing rapidly.

Type I accounts for 10% to 15% of cases, and the age of onset is primarily childhood or adolescence.

Type II accounts for 85% to 90% of cases and the age of onset generally occurs after age 40. A small number of cases are Secondary Diabetes Mellitus, and the age of onset varies according to the cause of the underlying primary pathologic condition.

Disease Process

Diabetes occurs if the body cannot produce insulin (type I), or if it is unable to use the insulin produced (type II). In either case, the ultimate result is hyperglycaemia and impaired glucose transport.

Type I diabetes is characterised by a genetic predisposition manifested in one of several human leukocyte antigens. Research suggests that the genetic predisposition, coupled with an unknown factor, trigger an ongoing autoimmune process that systematically destroys the beta cells in the pancreas, thereby interfering with the body's ability to produce insulin.

Type II diabetes involves either a defect in the insulin release sites in the pancreas or a resistance to the action of insulin stemming from a decrease in the number of receptor sites in the peripheral tissues. Moreover, this type of Diabetes Mellitus is often associated with obesity.

In both types of Diabetes Mellitus, the result is interference with glucose transport across cell membranes in peripheral muscle and adipose tissue, leading to faulty oxidation and energy production. Metabolism of fat, carbohydrate, and protein is impaired, as are storage of glycogen in the muscle and liver and storage of fatty acids and triglycerides in adipose tissue. Amino acid cell transport is disrupted. Unrestrained gluconeogenic and glycogenolytic processes in the liver cause overproduction of glucose. As the blood glucose level rises, renal tubules fail to reabsorb all the

glucose; this produces glucosuria and osmotic diuresis, with water and electrolyte loss through the urine. Hyperglycaemia also damages myelin nerve coverings, leading to neuropathy. Glycosylation (attaching of glucose to protein molecules) in the capillaries causes thickening of the capillary membrane and microangiopathy. Atherosclerotic processes are accelerated, and vessel elasticity diminishes.

Symptoms

Type I

Abrupt onset with polyuria, polydipsia, polyphagia, weight loss, weakness, fatigue, dehydration

Type II

Usually asymptomatic in early stages, with pruritus vulvae a common presenting symptom in women. Later manifestations include skin infections, cold extremities, fatigue, blurred vision, delayed healing, and polyuria

Potential Complications

Diabetic ketoacidosis is a common acute complication in type I diabetes. If left untreated, it leads to coma and death. Nonketotic hyperglycemic-hyperosmolar coma is an acute complication in type II diabetes. It is frequently accompanied by seizure activity and has a mortality rate of about 50%. Systemic chronic complications include cardiovascular and peripheral vascular disease, retinopathy, nephropathy, neuropathy, dermopathy, and impotence.

Diagnostic Tests

Fasting blood sugar

0.140mg/dl on two occasions

Glucose tolerance test

0.200mg/dl for 2-hour sample and one other sample after administration of 75g of glucose

Blood insulin

Absent in type I; normal or elevated in type II

Plasma C-peptide

Absent in type I; normal or elevated in type II

Treatments

Surgery - Only for chronic complications such as coronary artery grafts, and eye surgery, etc

Drugs - Insulin for type I; oral hypoglycemics for type II

General -

- Dietary control aimed at maintaining stable body weight
- distributing caloric intake into small, evenly spaced loads, avoiding high-fat and high-sugar foods
- weight reduction with obesity
- regular monitoring of blood sugar
- education about disease, complications, medications, diet
- counselling, support for adaptation to long-term disease

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Iridology

Iridology Daisy Iris

The Daisy Iris resembles the petals of a flower. This marking can indicate a genetic disposition.

(Picture right – Daisy Iris (Aust))

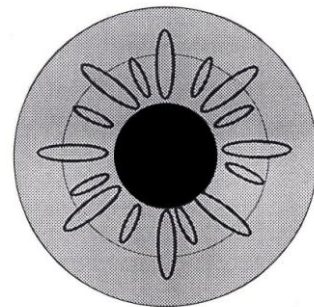
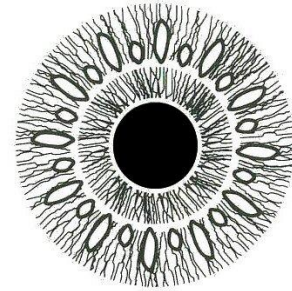
This marking is associated with diabetes and hypoglycaemia.

The Daisy Iris manifests as a 'daisy' pattern over the whole iris, and is commonest in blue-eyed individuals.

The Daisy Iris reflects a glandular tendency, and this often indicates a sugar metabolism problem:

(Picture right – Daisy Iris (BSY))

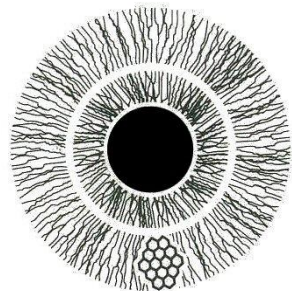
- 1) It can indicate a tendency to develop Diabetes Mellitus.
- 2) it can indicate a tendency to develop hypoglycaemia (low blood sugar levels).



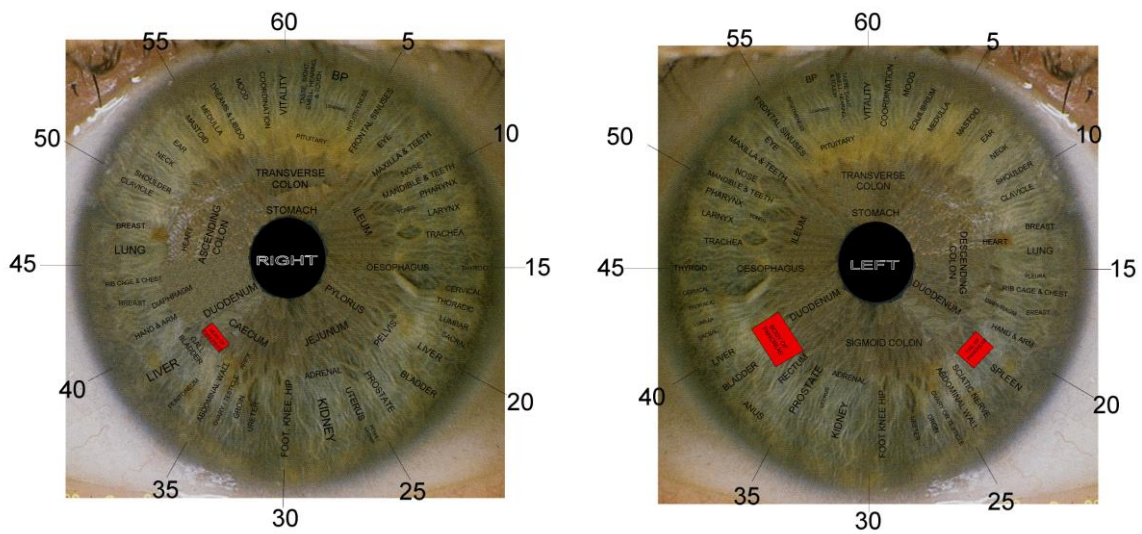
Honeycomb Arrangement of Teased Fibres in Iris

The Honeycomb appears as small hexagonal clusters in a small segment of the iris (Zone 3 to 7), and indicates a problem with the function and metabolic processes of the indicated organ/area. The honeycomb is often seen in the digestive zones, pancreatic area, and in the lungs.

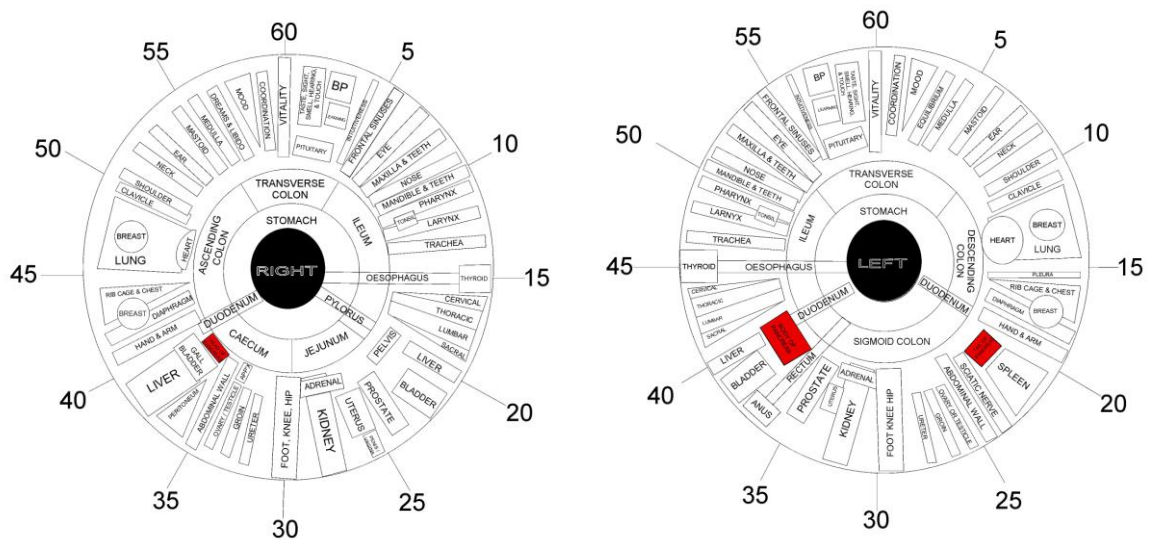
It can also indicate an inherited or genetic problem with the indicated organ. In hormonal or glandular organs, the honeycomb indicates endocrine problems (e.g. diabetes, thyrotoxicosis, etc) (Picture right – Honeycomb).



Iridology is not used to identify specific diseases; however, for indications of diabetes mellitus I would look in the eye area(s) associated with the pancreas. These areas, I have indicated in red on the following diagrams:



Dibetes Mellitus: Right Eye, Seg 38-39 (approx.) - Head of Pancreas; Left Eye, Seg 37-40 (approx.) - Body of Pancreas; Left Eye, Seg 20-22 (approx.) - Tail of Pancreas



Note: The right eye reflects the head of the pancreas (segment 38-39), and the left eye reflects the body and tail of the pancreas (segment 37-40 and 20-22 respectively).

End

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