Diabetes Mellitus

Diabetes mellitus (DM) comprises a group of metabolic disorders presenting with hyperglycaemia resulting from insulin deficiency or decreased glucose utilization and increased glucose production.

TYPES OF DIABETES MELLITUS.

TYPE- I DIABETES MELLITUS.

- Type I diabetes mellitus is due to pancreatic *B*-cell destruction leading to insulin deficiency.
- It is more common in children, adolescents and young adults usually below 30 years.

- Insulin is usually required for treatment *i.e.* patients are **insulin dependent**.

- These subjects are genetically susceptible and are prone to develop ketosis.

- Due to the above features, this diabetes is also called **brittle diabetes.**

Type II Diabetes mellitus.

- Type II Diabetes Mellitus is characterized by variable degree of insulin resistance, and impaired insulin secretion.

- It is preceded by a period of abnormal glucose homeostasis classified as impaired fasting glucose (IFG) or impaired glucose tolerance (IGT).

- Typically, it develops with increasing age.

- However, age in no bar and can also occur in obese children and adolescents.

Gestational Diabetes.

- Insulin resistance seen in late pregnancy may lead to impaired glucose tolerance and even frank diabetes called gestational diabetes mellitus.

- Most subjects revert to normal glucose tolerance after delivery but have increased risk (=~60%) of developing diabetes mellitus in future (10-20 years).

METABOLIC CHANGES IN DIABETES MELLITUS.

- Hyperglycemia—

1. overproduction of glucose by gluconeogenesis.
2. under utilization of glucose by the tissues due to insulin deficiency.

- Lypolysis— Low insulin : glucose ratio.

- KETOSIS and Hyperlipidemia- Acetyl-Co A diverted to other pathway of its utilization LIKE .....
- Ketosis, cholesterol synthesis, and fatty acid synthesis.
- Excess ketone bodies produced by Liver , continued if not treated leads to ketoacidosis.

LONG STANDING METABOLIC EFFECT.

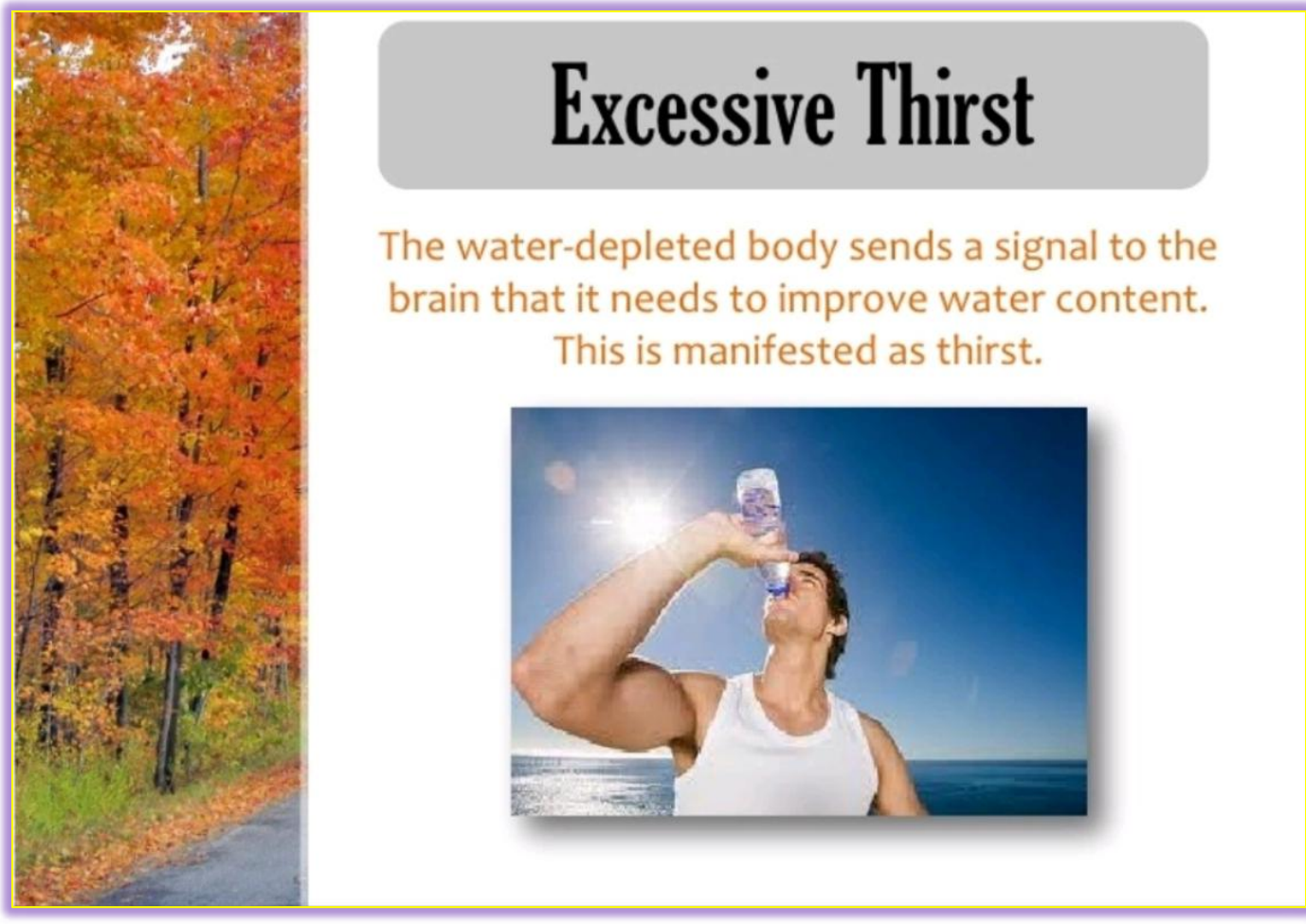
AGEs(advanced glycation end products).

High glucose in tissue leads to non enzymatic attachment of glucose and its metabolites to several protein like Hb, albumin, collagen etc. This process is called glycation. Ultimately leads to the formation of advanced glycation end product which causes cellular dysfunction.one such glycated protein is HbA1c.

CATARACT—(polyol-pathway)—High glucose leads to sorbitol accumulation causing cellular dysfunction. Cataract is believed to be a result of osmotic effect of sorbitol accumulation.

Clinical features of Diabetes Mellitus.

- Polyurea.
- ***Polydipsia.***
- .Polyphagia.
- Fatigue.
- Weight Loss.



Excessive Thirst

The water-depleted body sends a signal to the brain that it needs to improve water content. This is manifested as thirst.

Frequent Urination

Eliminating the extra glucose is through excreting it in the urine.

Sugar attracts water into the urinary tract, causing voiding of great volumes of urine.

Fatigue

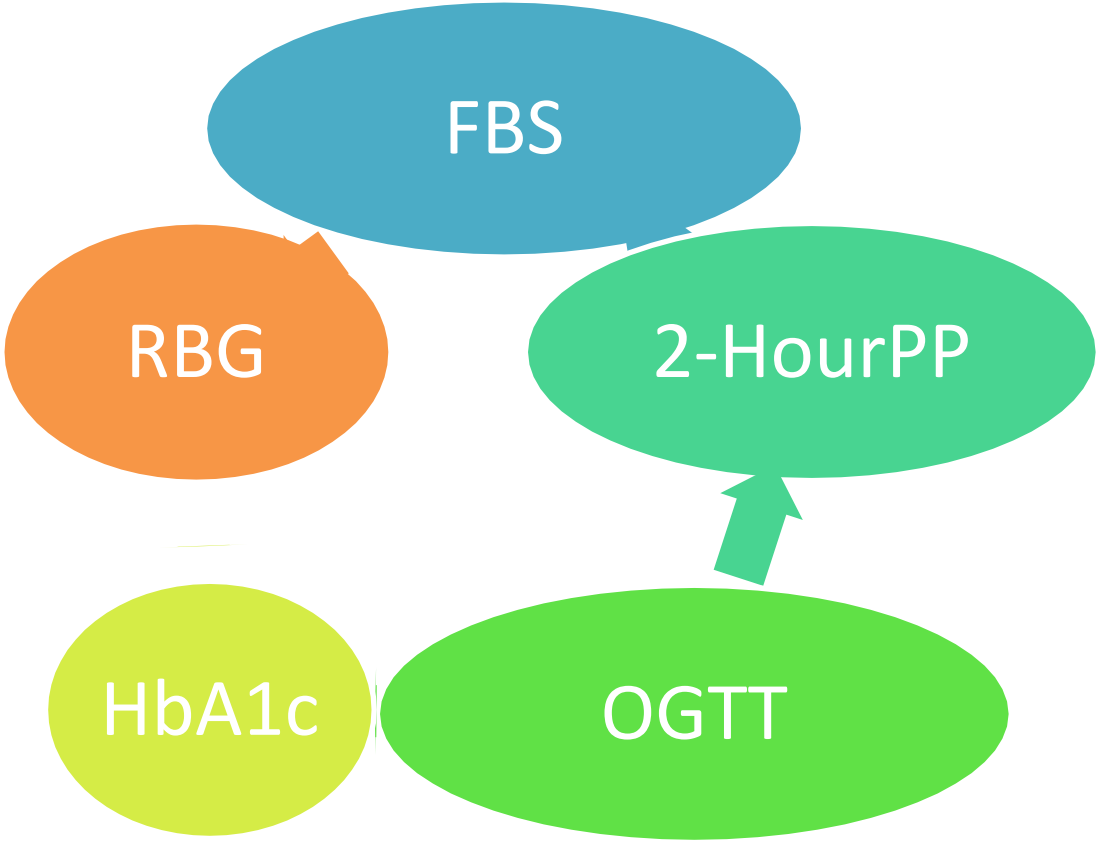
Sugar or glucose is the primary energy source of the cells. Without insulin, glucose is not used properly. The body uses excess fat for energy and moves the metabolism to work harder. In that case you feel more tired than the usual.

DIAGNOSIS OF DIABETES MELLITUS.

WHO guidelines

- FBG– ≥ 126mg%.
- PP-- ≥ 200%.
- RBG—> 200 mg% with symptomps of diabetes mellitus.
- GLYCATED Hb(HbA1c) > 6.5gm%.

DIFFERENT TYPES OF BLOOD GLUCOSE TESTS.



Diabetic profile.

Blood glucose.

4 types :FBS, PPBS, RBS, OGGT.

Urine Analysis.

Urine sugar/urine protein/urine microalbum/ketones.

HbA1C

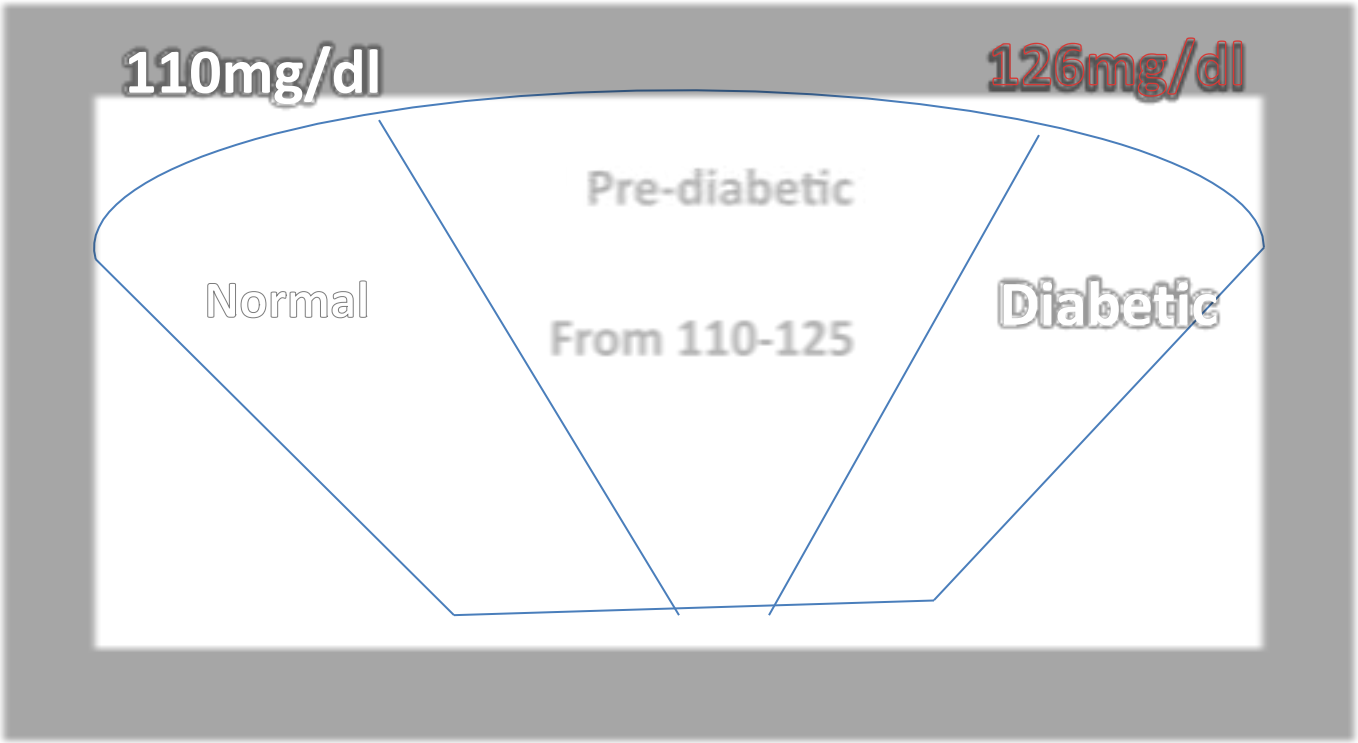
Insulin

ICA (islets cell antibody) for type I

C-peptide

FASTING BLOOD SUGAR.

- Normal level
- 70---110mg/dl.



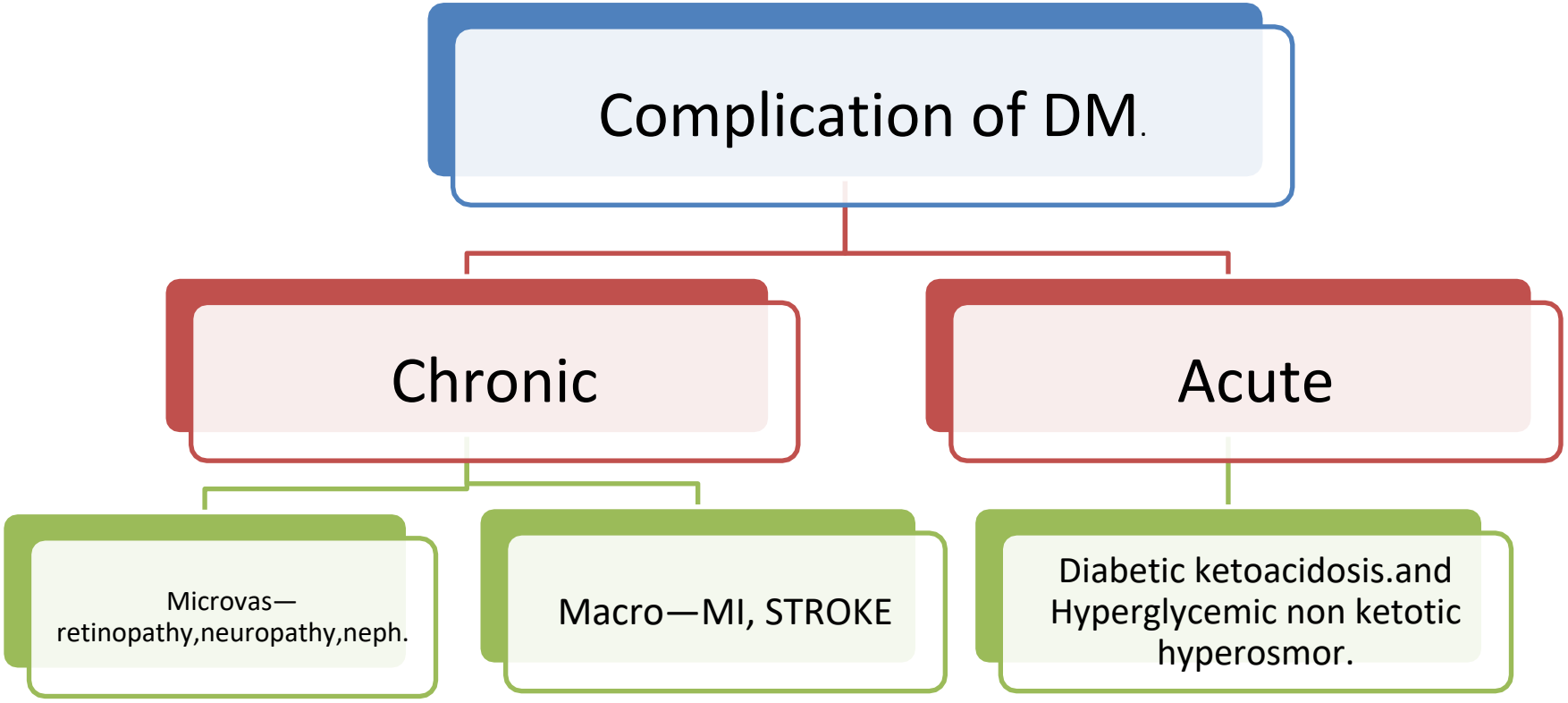
NORMAL BLOOD GLUCOSE LEVEL

- FBG---70 ---110 mg %.
- PP---Less than 140 mg% after 75 mg glucose load.
- HbA1c –below 6.5 gm%.

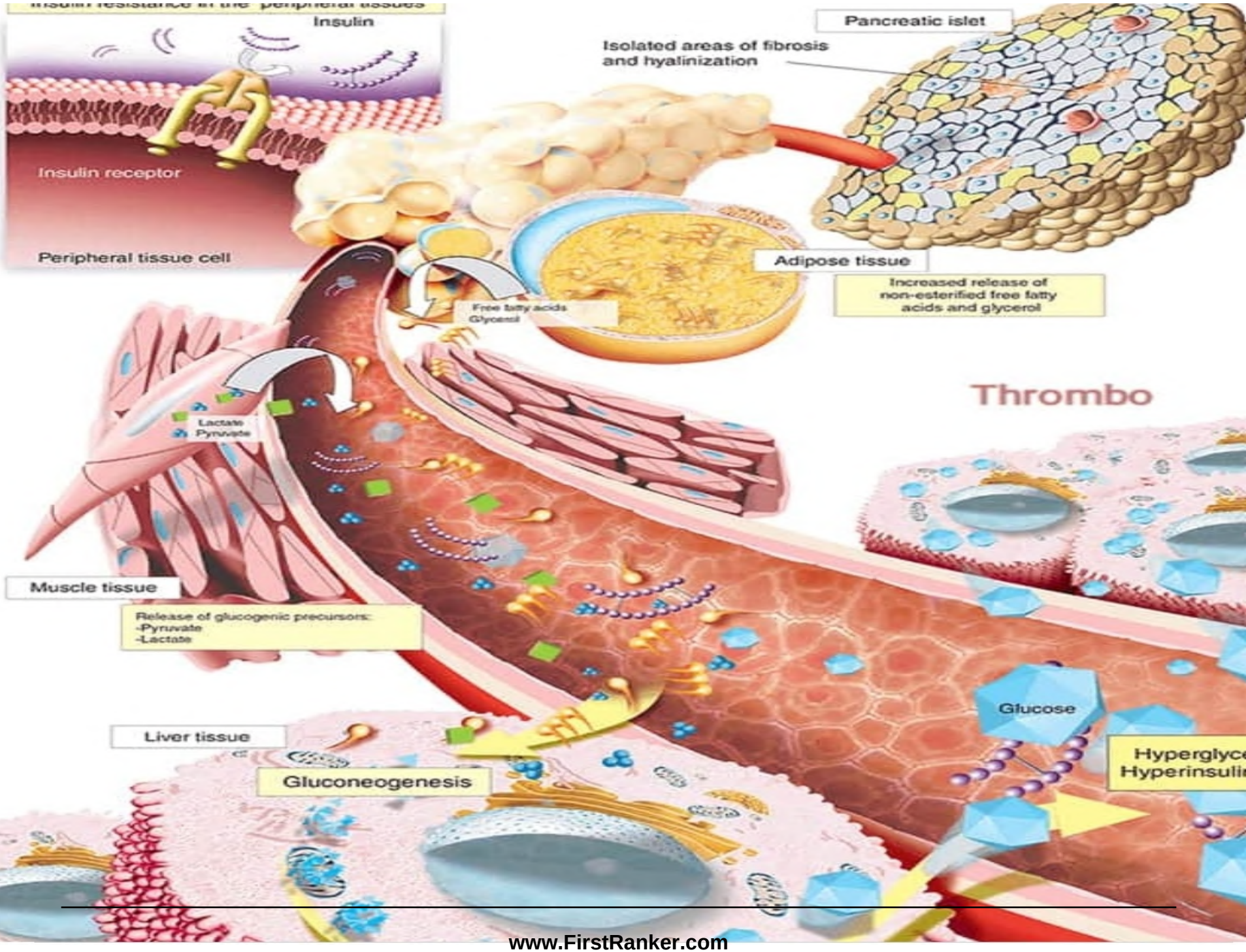
INSULIN RESISTANCE SYNDROME.

- Also known as Metabolic syndrome or the syndrome X, characterised by the following....
- Insulin resistance indicated by ↑blood glucose inspite of ↑insulin level in blood.
- Hypertension.
- Dyslipidemia((↑LDL + TG).
- Central obesity.
- Accelerated cardiovascular disease.
- Insulin resistance syndrome is due to post- receptor signaling defect in target tissues, e.g. Skeletal muscle.

COMPLICATION OF DIABETES MELLITUS.



- Alteration in blood sugar: hyperglycemia and Hypoglycemia.
- Macrovascular(Larege blood vessels)
- Atherosclerosis involving :-
- coronary, cerebral and peripheral arteries.
- Microvascular(small blood vessels):-
- affects basement membrane of small blood vessels and capillaries involving Eyes and Kidneys.



HYPERGLYCEMIA.

- High blood sugar
- DKA
- HHKs
- Dawn phenomenon-
- Rise in blood sugar between 4 a.m. And 8 a.m.
- Not associated with hypoglycemia.
- SOMOGYI phenomenon----
- Combination of hypoglycemia during night with a rebound morning hyperglycemia that may lead to insulin resistance for 12 to 48 hours.

ACUTE COMPLICATION

- Hypoglycemia
- Diabetic ketoacidosis.
- Hyperglycemic hyperosmotic non ketotic coma.
- HYPOGLYCEMIA:--
- Serum glucose < 55 mg/100ml.
- Brain damage develops when the brain is deprived of needed glucose after a dramatic drop in blood sugar.

HYPOGLYCEMIA
SIGN AND SYMPTOMPS.

- MILD:-
-
- Palpitation, pallor , tremors, anxiety
- Parasthesia,
- MODERATE:-
- confusion, cold extremities, blurred vision.
- SEVERE:-
- Seizure, Loss of consciousness,can result in death.

Others complication.

- Cataract.
- Glucoma.
- Infection-Foot ulcer, osteomyelitis.
- Skin infection—candiasis and other fungal infection.

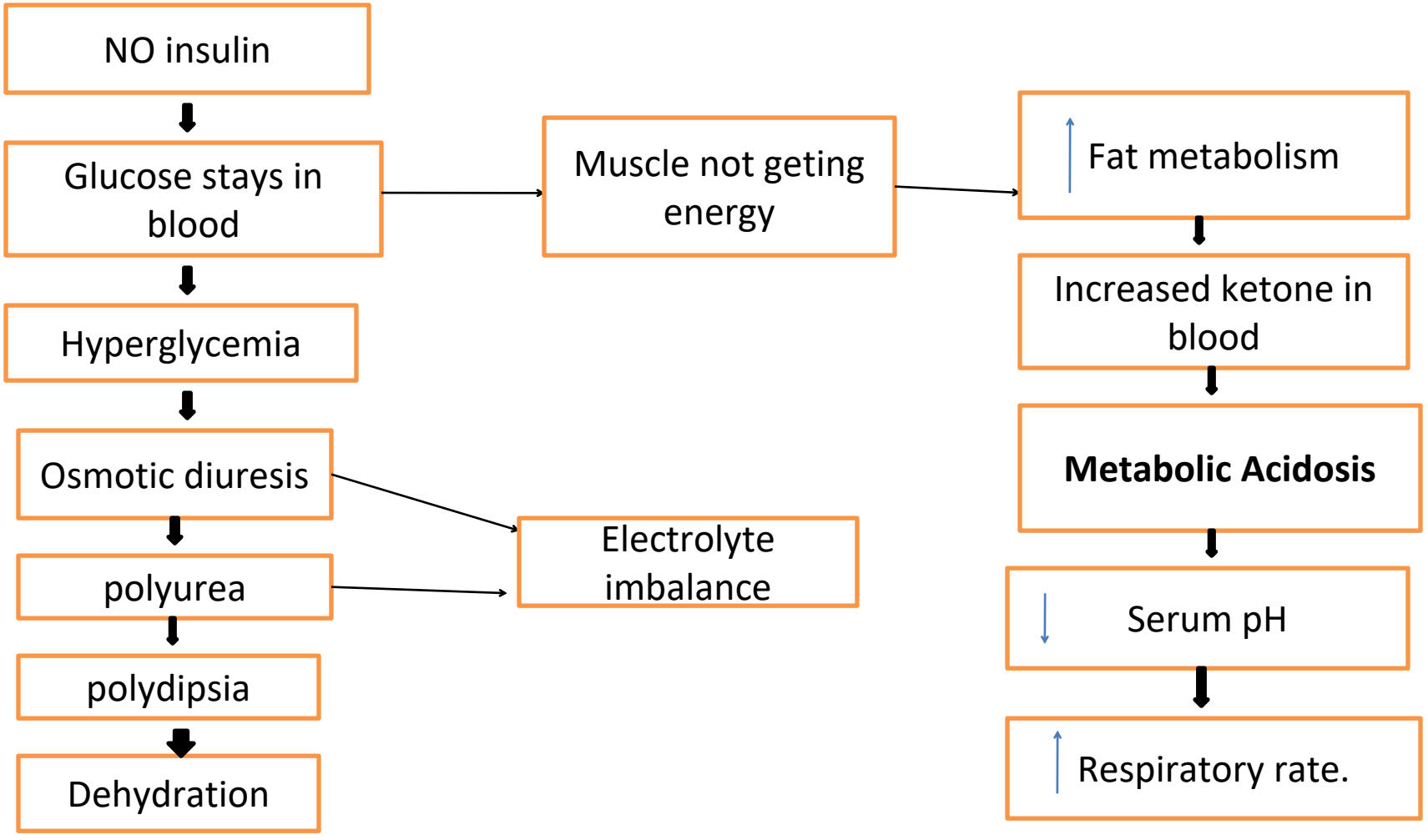
DIABETIC KETOACIDOSIS.

- Diabetic ketoacidosis is the most dreaded acute complication resulting from uncontrolled diabetes mellitus, characterised by
- Severe hyperglycemia-Blood glucose is very high, commonly 400- 500 mg %.
- Dehydration due to polyurea.
- Ketosis—due to increased production of ketone bodies as a result of hyperglycemia.
- Ketone body test is positive in urine.

DKA: 4 main clinical features.

- 1.Hyperglycemia
- 2.Dehydration
- 3.Eectrolyte loss.
- 4.Metabolic Acidosis.

PATHOPHYSIOLOGY DKA.



- Acidosis with increased anion gap---
- PH 6.8—7.3 and anion gap of >15mEq/L.
- Hyperventilaton with fruity odour of breath---
- Acidisis stimulates respiration leading to hyprventilation. Excretion of acetone (ketone bodies) in the breath is responsible for fruity odour in breath.

- Other symptomps include—
- Nausea
- vomiting
- pain abdomen
- letharginess
- Depression.....etc.
- Hyperkalemia and hypontremia.

- It is acute medical emergency.
- Patient is admitted and treated with intravenous insulin, fluids and electrolytes to correct dehydration and electrolyte imbalance.

Glycated Hemoglobin.

- Glycated Hb(HbA1c)---Is formed by non-enzymatic addition of N- terminal Valine of Hb Beta chain due to persistent hyperglycemia.
- Its level in blood gives an estimate of blood glucose over the preceding 6-8 weeks.