Diabetes Mellitus Type 2

Objectives:

- Scope of diabetes
- Making the diagnosis
- Pathophysiology
- Disease consequences
- Management
- Conclusion

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Resources: 435 team + Davidson + kumar + Recall questions step up to medicine.

- Editing file
- Feedback

Introduction

★ Diabetes Mellitus:

- Diabetes mellitus is a clinical syndrome characterized by an increase in plasma blood glucose (hyperglycemia). There are many types of diabetes:
  - **Type 1 diabetes:** characterized by a severe deficiency of insulin.
  - **Type 2 diabetes**.
  - **Other specific types**: it could be temporary or permanent but always have a specific cause, **for example**: *Genetic defects of β-cell function, Genetic defects of insulin action and pancreatic disease like pancreatitis, also steroids, cushing syndrome, acromegaly, pheochromocytoma, hyperthyroidism.*
  - **Gestational diabetes mellitus**: diabetes with first onset or recognition during pregnancy, and it goes away after the pregnancy.

21.12 Classical features of type 1 and type 2 diabetes

	Type 1	Type 2
Typical age at onset	< 40 yrs	> 50 yrs
Duration of symptoms	Weeks	Months to years
Body weight	Normal or low	Obese
Ketonuria	Yes	No
Rapid death without treatment with insulin	Yes	No
Autoantibodies	Positive in 80–90%	Negative
Diabetic complications at diagnosis	No	25%
Family history of diabetes	Uncommon	Common
Other autoimmune disease	Common	Uncommon

★ Type 2 Diabetes Mellitus:

- The overall adult prevalence of DM in KSA is 23.7%. underpredicted.
- Type 2 diabetes is characterized by **resistance to the action of insulin and an inability to produce sufficient insulin to overcome this 'insulin resistance'**.
- Type 2 diabetes was previously termed 'non-insulin-dependent diabetes mellitus' (NIDDM) because patients retain the capacity to secrete some insulin but exhibit impaired sensitivity to insulin (insulin resistance) and initially can usually be treated without insulin replacement therapy.
- **Insulin resistance syndrome (Metabolic syndrome/ Syndrome X):**
  - Type 2 diabetes, or its precursor (impaired glucose tolerance), is one of a cluster of conditions thought to be caused by resistance to insulin action. Thus, patients with type 2 diabetes often have associated disorders including **hypertension**, **dyslipidemia**, **non-alcoholic fatty liver** and, in women, **polycystic ovarian syndrome**. This cluster has been termed the '**insulin resistance syndrome**' or 'metabolic syndrome' and is much more common in patients who are obese.
    the more obesity in a community the more T2DM prevalence. (Hallmark marker).

★ DM Risk Factors:

- In both of the common types of diabetes, environmental factors interact with genetic susceptibility to determine which people develop the clinical syndrome.
- **Genetic predisposition:**

- Genetic factors are important in type 2 diabetes, as shown by marked differences in susceptibility in different ethnic groups.
- Genome-wide association studies have identified over 65 genes or gene regions that are associated with type 2 diabetes, each exerting a small effect. **The largest effect is seen with variation in TCF7L2**. Most of the genes known to contribute to risk of type 2 diabetes are involved in β-cell function or in regulation of cell cycling and turnover, suggesting that altered regulation of β-cell mass is a key factor.

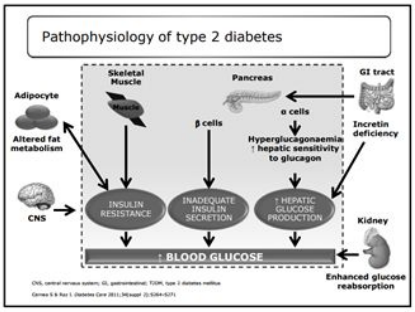
- Environmental and other risk factors:
  - Diet and obesity increase the risk of developing type 2 diabetes
  - The risk of developing DM type 2 increases **tenfold (10x)** in people with a **body mass index (BMI) of more than 30 kg/m2**
  - However, although the majority of patients with type 2 diabetes are obese, only a minority of obese people develop diabetes, as the majority of obese patients are able to increase insulin secretion to compensate for the increased demand resulting from obesity and insulin resistance.
  - **Age**: type 2 diabetes is **more common** in the middle-aged and elderly.

Pathophysiology

★ Basic mechanism of glucose control:

- Before attempting to understand the pathophysiology of DM type 2 you should know the basic mechanism of glucose control, here is a brief reminder:
  - Blood glucose is tightly regulated and maintained within a narrow range by insulin & glucagon. This is essential for ensuring a continuous supply of glucose to the central nervous system.
  - After ingestion of a meal containing carbohydrate, normal blood glucose levels are maintained by:
    - Suppression of hepatic glucose production.
    - Stimulation of hepatic glucose uptake.
    - Stimulation of glucose uptake by peripheral tissues.

- **Incretin effect:**
  - **Insulin**: the primary regulator of glucose metabolism and storage is secreted from **pancreatic β cells** into the portal circulation in response to a rise in blood glucose.
  - Also a number of other factors released from the gut following food intake can **augment (increase) insulin release**, including amino acids and hormones such as **glucagon-like peptide 1 (GLP-1)** and **gastrointestinal peptide (GIP)**.
  - As a result, insulin release is greater when glucose is administered by mouth than when the same rise in plasma glucose is achieved by intravenous



glucose infusion, a phenomenon termed the **(incretin effect)**.

★ Pathophysiology is divided into:

1. **Insulin resistance:**
    - Initially, insulin resistance leads to elevation in insulin secretion to maintain normal blood glucose level. However, in susceptible individuals, the pancreatic β cells are unable to sustain the increased demand for insulin and a slowly progressive insulin deficiency develops. **But the primary cause of insulin resistance remains unclear, so we only have theories.**

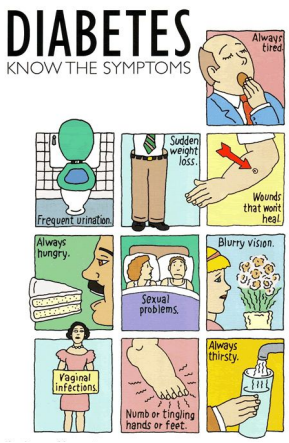
First theory	Second theory
Centered on the Intra-abdominal adipose tissue, how?	**physical activity, how?**
• Central adipose tissue releases large quantities of FFAs, which may induce insulin resistance. • In addition, adipose tissue releases many hormones (adipokines) which act on specific receptors to influence sensitivity of insulin in other tissues. • Several circulating peptides including the cytokines **TNF-α (very imp. overexpressed in obesity & correlates with insulin resistance)** and IL-6, RBP4, and the "adipokines" adiponectin and resistin produced and released from adipose tissue can **modify insulin action**. (lead to insulin resistances) • Also, diabetic patients have some degree of incretin hormones ((GLP-1) and (GIP)) resistance and deficiency (they play a major role in the management).	1. Inactivity is associated with down regulation of insulin-sensitive kinases and may promote accumulation of FFAs within skeletal muscle. 2. Moreover, physical activity allows non-insulin-dependent glucose uptake into muscle, reducing the 'demand' on the pancreatic β cells to produce insulin. • All those changes play a role in development of insulin resistance which is manageable by normal individuals.

2. **Pancreatic β-cell failure:**
    - Some pathological changes are typical of type 2 diabetes, the most consistent of which is **deposition of amyloid in the islets.** Another factors such as **elevated plasma glucose** and **FFAs** also exert toxic effects on pancreatic β cells which will impair insulin secretion.
    - However, while β-cell numbers are reduced, β-cell mass is unchanged and **glucagon secretion is increased**, which may contribute to hyperglycemia.
    - At the time of diagnosis, around 50% of β-cell function has been lost and this declines progressively.

Diagnosis

★ Signs and Symptoms

- Symptoms of hyperglycemia and Clinical examination of patients with diabetes:
  - **Polydipsia** (Thirst, dry mouth).
  - **Polyuria**.
  - **Polyphagia** (excessive eating).
  - **Change in weight** (usually weight loss).
  - Nocturia.
  - Tiredness, fatigue, lethargy.
  - Blurring of vision.
  - Pruritus vulvae, balanitis (genital candidiasis).
  - Nausea.
  - Headache.
  - Mood change, irritability, difficulty in concentrating, apathy.
  - patient present with retinopathy highly suggest T2DM
  - **Neuropathy (Axonal Degeneration).**



★ Investigations

- DM 2 is a diagnosis of <u>exclusion</u> we say it is type 2 when we rule out other types of diabetes, there are two types of diabetes investigations **Blood & Urine**:

Blood testing	Urine testing
1. **Glucose** Laboratory blood glucose testing is cheap and highly reliable. Capillary blood glucose can also be used to monitor diabetes treatment. 2. **Ketones** Whole blood ketone monitoring detects **β-hydroxybutyrate** and is useful in guiding insulin adjustment during intercurrent illness or sustained hyperglycemia to prevent or detect **DKA**. (More common in type 1 DM) 3. **Glycated haemoglobin (HbA1c)** Glycated haemoglobin (Hb) provides an accurate and objective measure of glycaemic control over a period of weeks to months. The rate of formation of **HbA1c** is directly proportional to the blood glucose concentration; a rise of 1% in HbA1c corresponds to an increase of 2	1. **Glucose** Urine dipsticks are used to screen for diabetes. Testing should ideally use urine passed 1–2 hrs after a meal.however, glycosuria can be due to a low renal threshold. This is a benign condition unrelated to diabetes, common during pregnancy and in young people. 2. **Ketones** Ketonuria may be found in normal people who have been fasting, exercising or vomiting repeatedly, or those on a high-fat, low carbohydrate diet. Ketonuria is therefore not pathognomonic of diabetes but, if it is associated with **glycosuria**, diabetes is highly likely. (More common in

mmol/L (36 mg/dL) in blood glucose. **it is most sensitive** to glycaemic control in the past month.	type 1 DM) 3. **Protein** Standard dipstick testing will detect urinary albumin > 300 mg/L but smaller amounts (**microalbuminuria**) require specific sticks or laboratory urinalysis. (diabetic nephropathy → proteinuria)

- HbA1c concentration reflects blood glucose over the erythrocyte lifespan (120 days) that's why it is best for monitoring management but In patients with diseases that reduce red blood cell lifespan, such as hemolytic anaemia or hemoglobinopathies such as sickle-cell disease, a hemoglobin-based A1c test can be misleadingly low.

★ Diagnosing a Newly discovered hyperglycaemia:

- **Glycaemia** can be classified as either **normal**, **impaired (pre-diabetes)** or **diabetes**. **The glucose cut-off that defines diabetes is the level above which there is a significant risk of microvascular complications** (**retinopathy**, **nephropathy**, **neuropathy**). Those with pre-diabetes have a negligible risk of microvascular complications but are at increased risk of developing diabetes. Impaired glucose tolerance: ⅓ reverse, ⅓ continue chronic impairment **(Most common chronic complication)** & ⅓ progress to diabetes after 10 years.
- **What is the GOLD standard for the diagnosis of diabete? Oral glucose tolerance test.**
- **Diagnostic Criteria of Diabetes:**
  - **Plasma glucose in random sample OR 2 hrs value of oral glucose tolerance test (2 hrs after a 75-g glucose load)= ≥ 11.1 (200 mg/dL)**
  - **Fasting plasma glucose= ≥ 7.0 mmol/L (126 mg/dL)**
  - **Glycated haemoglobin (HbA1c)= >6.5% is a diagnostic criterion and is the 📍best test to follow response to therapy over the last several months. (Used for follow up not diagnosis)**

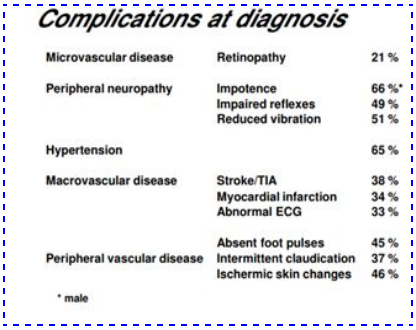
Asymptomatic individuals should have a **second** confirmatory test (repeated on a separate occasion). If the patient was **symptomatic** it is confirmed from the first positive (symptoms + positive diagnostic test = Diabetes type 2)

★ Complications of DM type 2:

Macrovascular	Microvascular
• **Coronary artery disease.** • **Peripheral artery disease.** • **Cerebrovascular disease.** • **Overall lifespan is decreased by 6 years.**	• **Diabetic nephropathy.** • **Diabetic retinopathy.** • **Diabetic neuropathy leads to → diabetic foot**

★ Note that:

- DM complications are present at diagnosis. retinopathy
- DM complications progress with time.
- DM control predicts rate and state of complications.
- Early and sustained control limits complications
- Management is multifaceted and complex.
- insulin resistance lead to macrovascular complications,hyperglycemia leads to both macro & micro
- microvascular complications are more common than Macro



Complications at diagnosis

Microvascular disease	Retinopathy	21 %
Peripheral neuropathy	Impotence	66 %*
	Impaired reflexes	49 %
	Reduced vibration	51 %
Hypertension		65 %
Macrovascular disease	Stroke/TIA	38 %
	Myocardial infarction	34 %
	Abnormal ECG	33 %
Peripheral vascular disease	Absent foot pulses	45 %
	Intermittent claudication	37 %
	Ischermic skin changes	46 %

* male

Management

★ Goals & Aim

Now with your patient diagnosed with DM2 what is your plan ?
The aims of management are to improve symptoms of hyperglycemia and to minimize the risks of long-term microvascular and macrovascular complications.

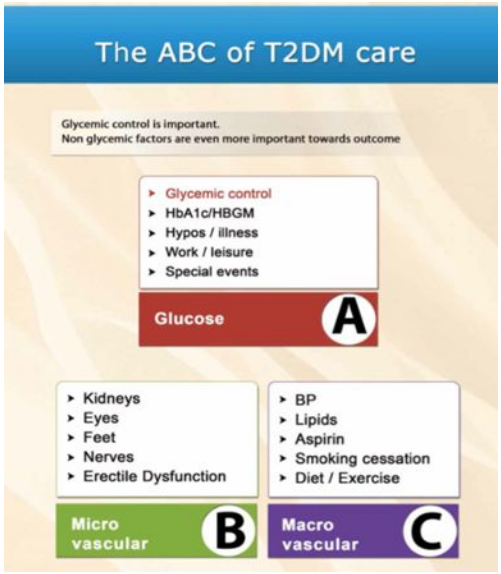
- **Goal:**
  - **Patient education:** This can be achieved by a multidisciplinary team (doctor, dietitian, specialist nurse and podiatrist) in the outpatient setting.
  - **Self-assessment of glycaemic contro**l: Insulin-treated patients should be taught to monitor blood glucose using capillary blood glucose meters, and to use the results to guide insulin dosing and to manage exercise and illness
  - **Therapeutic goal**: The target **HbA1c** depends on the patient. Early on in diabetes (i.e.patients managed by diet or one or two oral agents), a target of 48 mmol/mol (6.5%) or less may be appropriate,(7.5%) may be more appropriate in older patients with pre-existing cardiovascular disease, or those treated with insulin and therefore at risk of hypoglycaemia.

- **MANAGEMENT OF DIABETES:**
  - **In new cases of diabetes, adequate glycemic control can be obtained by diet and lifestyle advice alone in approximately 50%, 20–30% will need oral antidiabetic medication, and 20–30% will require insulin.**
  - Diet and lifestyle.
  - Drugs to reduce hyperglycemia

MANAGEMENT OF DIABETES controlling glycemia is important in preventing microvascular complications	
Diet and lifestyle	• Lifestyle changes, such as taking regular exercise, observing a healthy diet, reducing alcohol consumption and stopping smoking, are important **but difficult for many to sustain** **first line** 1. **Healthy eating** 2. **Weight management** 3. **Exercise**
Drugs to reduce hyperglycemia **(you have more than 1500 combination choice)**	The best **initial** (first-line) drug therapy is with oral **Metformin**. (increases insulin sensitivity & helps in weight loss) Metformin works by blocking gluconeogenesis.(Metformin is **contraindicated** in those with **renal dysfunction** because it can accumulate and cause **lactic acidosis**). Metformin is safe in pregnancy. (other oral anti-diabetics are not)
	Sulfonylureas are not used as first-line therapy because they increase insulin release from the pancreas, thereby driving the glucose intracellularly and increasing obesity.
	Thiazolidinediones (**glitazones**) provide no clear benefit over the other hypoglycemic medications. They are relatively contraindicated in CHF because they increase fluid overload.
	Nateglinide and repaglinide are stimulators of insulin release in a similar manner to sulfonylureas, but do not contain sulfa. They do not add any therapeutic benefit to sulfonylureas.
	Alpha glucosidase inhibitors (**acarbose, miglitol**) are agents that block glucose absorption in the bowel. They add about half a point decrease in HgA1c. They cause flatus, diarrhea, and abdominal pain.They can be used with renal insufficiency.
	Incretins (**exenatide, sitagliptin, saxagliptin, linagliptin**) are part of the mechanism by which oral glucose normally produces a rise in insulin and decreases glucagon levels. These agents also decrease gastric motility and help in weight loss, Exenatide may cause pancreatitis.
	Pramlintide is an analog of a protein called amylin that is secreted normally with insulin. Amylin decreases gastric emptying, decreases glucagon levels, and decreases appetite.
	Insulin is added if the patient is not controlled with oral hypoglycemic agents. Insulin glargine gives a steady state of insulin for the entire day. Dosing is not tested. Glargine provides much more steady blood levels than NPH insulin, which is dosed twice a day. Long-acting insulin is combined with a short-acting insulin such as lispro, aspart, or glulisine. Regular

	insulin is sometimes used as the short-acting insulin. The goal of therapy is HgA1c <7%. (Insulin can be used in pregnancy). First drug to give if patient came with HBA1c >10% is premixed insulin.

★ Considerations when constructing therapy plan at the second follow up visit:

Blood pressure	• ACE inhibitors • ARBS • Beta blockers / CCB • Thiazides	
Proteinuria	• ACE inhibitors • ARBS • BP control • Ref. to Nephrology	
CVS	• Statins • Aspirin • Diet control • Physical activity	
Eyes	• Macula • Retina • Complete eye evaluation • Ref. to Opth	
Feet	• Infection • Vascular status • Deformity • Neuropathy (Diabetic Foot)	

Summary

DM II

- Type 2 diabetes is characterized by resistance to the action of insulin and an inability to produce sufficient insulin to overcome this 'insulin resistance'.
- patients with type 2 diabetes often have associated disorders including hypertension, dyslipidemia, non-alcoholic fatty liver and, in women, polycystic ovarian syndrome. This cluster has been termed the 'insulin resistance syndrome' or 'metabolic syndrome' and is much more common in patients who are obese.

<table>
<tr><td rowspan="3">Risk factor</td><td colspan="2">Genetic predisposition:
- The largest effect is seen with variation in TCF7L2
- altered regulation of β-cell mass is a key factor.</td></tr>
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- The risk increases tenfold (10x) in people with a body mass index (BMI) of more than 30 kg/m2</td></tr>
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more common in the middle-aged and elderly.</td></tr>
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insulin release is greater when glucose is administered by mouth (incretin effect).</td></tr>
<tr><td>Pathophysiology</td><td>1- Insulin resistance:
the primary cause of insulin resistance remains unclear, so we only have theories.
1st: Intra-abdominal adipose tissue FFAs , adipokines , IL-6, RBP4, TNF-α , can modify insulin action.
2nd:Inactivity
- physical activity allows non-insulin-dependent glucose uptake into muscle, reducing the 'demand' on the pancreatic β cells to produce insulin.</td><td>2 - Pancreatic β-cell failure:

- deposition of amyloid in the islets.

- elevated plasma glucose and FFAs

- β-cell numbers are reduced, β-cell mass is unchanged and glucagon secretion is increased, which may contribute to hyperglycemia.</td></tr>
</table>

Signs and Symptoms	polydipsia, polyuria, Polyphagia, Change in weight Nocturia, Tiredness, fatigue, lethargy, Blurring of vision, patient present with retinopathy highly suggest T2DM.	
Investigations	**1- Blood testing** - **Glucose** Laboratory blood. - **Ketones** Whole blood ketone monitoring detects **β-hydroxybutyrate.** - **Glycated haemoglobin (HbA1c) , it is most sensitive** to glycaemic control in the past month.	**2- Urine testing** - **Glucose** Urine dipsticks are used to screen for diabetes. - Ketonuria if associated with **glycosuria**, ,diabetes is highly likely. (More common in type 1 DM) - **Protein** Standard dipstick testing will detect urinary albumin > 300 mg/L but smaller amounts (**microalbuminuria**) require specific sticks or laboratory urinalysis. (diabetic nephropathy → proteinuria)

Diagnosis	- **the glucose cut-off that defines diabetes is the level above which there is a significant risk of microvascular complications** (**retinopathy**, **nephropathy**, **neuropathy**).
	- **the GOLD standard for the diagnosis of diabete is Oral glucose tolerance test.**
	- **Diagnostic Criteria of Diabetes:**
	  - **Plasma glucose in random sample OR 2 hrs value of oral glucose tolerance test (2 hrs after a 75-g glucose load)= ≥ 11.1 (200 mg/dL)**
	  - **Fasting plasma glucose= ≥ 7.0 mmol/L (126 mg/dL)**
	  - **Glycated haemoglobin (HbA1c)= >6.5% is a diagnostic criterion and is the best test to follow response to therapy over the last several months.**	

management	- **Diet and lifestyle:** stopping smoking. - **Drugs to reduce hyperglycemia:** the initial (first-line) **Metformin** (safe in pregnancy ,**contraindicated** in those with **renal dysfunction**, cause **lactic acidosis**)	

Type 1 vs type 2 diabetes mellitus

Variable	Type 1	Type 2
1° DEFECT	Autoimmune destruction of β cells (eg, due to glutamic acid decarboxylase antibodies)	↑ resistance to insulin, progressive pancreatic β-cell failure
INSULIN NECESSARY IN TREATMENT	Always	Sometimes
AGE (EXCEPTIONS COMMONLY OCCUR)	< 30 yr	> 40 yr
ASSOCIATION WITH OBESITY	No	Yes
GENETIC PREDISPOSITION	Relatively weak (50% concordance in identical twins), polygenic	Relatively strong (90% concordance in identical twins), polygenic
ASSOCIATION WITH HLA SYSTEM	Yes (HLA-DR3 and -DR4)	No
GLUCOSE INTOLERANCE	Severe	Mild to moderate
INSULIN SENSITIVITY	High	Low
KETOACIDOSIS	Common	Rare
β-CELL NUMBERS IN THE ISLETS	↓	Variable (with amyloid deposits)
SERUM INSULIN LEVEL	↓	Variable
CLASSIC SYMPTOMS OF POLYURIA, POLYDIPSIA, POLYPHAGIA, WEIGHT LOSS	Common	Sometimes
HISTOLOGY	Islet leukocytic infiltrate	Islet amyloid polypeptide (IAPP) deposits

Questions

1. A 50-year-old woman is 5 ft 7 in tall and weighs 185 lb. There is a family history of diabetes mellitus. Fasting blood glucose (FBG) is 160 mg/dL and 155 mg/dL on two occasions. HgA1c is 7.8%. You educate the patient on medical nutrition therapy. She returns for reevaluation in 8 weeks. She states she has followed diet and exercise recommendations, but her FBG remains between 130 and 140 and HgA1C is 7.3%. She is asymptomatic, and physical examination shows no abnormalities. Which of the following is the treatment of choice?

- **A. Thiazolidinediones such as pioglitazone**
- **B. . Encourage compliance with medical nutrition therapy**
- **C. Insulin glargine at bedtime**
- **D. Metformin**
- **E. Glipizide**

2. A 55-year-old man is seen in the clinic for follow-up of type 2 diabetes mellitus. He feels well, has been exercising regularly, and has had good control of his blood glucose on oral metformin, with HgA1c of 6.4%. He has a history of mild hypertension and hyperlipidemia. Which of the following statements is correct regarding routine testing for diabetic patients?

- **A. Dilated eye examination twice yearly**
- **B. 24-hour urine protein annually**
- **C. Home fasting blood glucose measurement at least once per week**
- **D. Urine microalbumin annually**
- **E. Referral to neurologist for peripheral neuropathy evaluation**

3. Risk factors for type 2 diabetes include all of the following except:

- **A. advanced age**
- **B. obesity**
- **C. smoking**
- **D. physical inactivity**

4. Blood sugar is well controlled when Hemoglobin A1C is:

- **A. less than 7%**
- **B. between 7-8%**
- **C. more than 9%**

5. Which of the following diabetes drugs acts by decreasing the amount of glucose produced by the liver?

A. **Sulfonylureas**

B. **Meglitinides**

C. **metformin**

D. **Alpha-glucosidase inhibitors**

6. A 50-year-old Asian man is referred to the diabetes clinic after presenting with polyuria and polydipsia. He has a BMI of 30, a blood pressure measurement of 137/88 and a fasting plasma glucose of 7.7 mmol/L. The most appropriate first-line treatment is:

A. **Dietary advice and exercise**
B. **Sulphonylurea**
C. **Exenatide**
D. **Thiazolidinediones**
E. **Metformin**

7. A 49-year-old man has recently been diagnosed with type 2 diabetes and is being carefully monitored. He has been advised to maintain a healthier diet and lifestyle, he attends a follow-up clinic and claims to have been following the diet stringently since his last appointment three months ago. The most appropriate investigation is:

A. **Random plasma glucose**
B. **Fasting plasma glucose**
C. **Urine dipstick**
D. **Glycated haemoglobin**
E. **Weight measurement**

8. A 41-year-old man has been recently diagnosed with type 2 diabetes and has been following a plan of lifestyle measures to improve his diet and increase his level of exercise. On returning to clinic, his BMI is 23, fasting plasma glucose 9.0 mmol/L, blood pressure 133/84 mmHg and HbA1c of 7.1 per cent. The most appropriate treatment option is:

A. **Metformin**
B. **Sulphonylurea**
C. **Insulin**
D. **Exenatide**
E. **Further diet and exercise**

9. A 29-year-old man presents to his GP complaining of being constantly thirsty, tired and visiting the toilet more often than usual during the last 4 days. He has noticed his clothes have become more baggy and he now needs to tighten his belt. His parents both have diabetes requiring insulin therapy. A fasting plasma glucose result is most likely to be:

A. 9.0 mmol/L
B. 6.0 mmol/L
C. 16.3 mmol/L
D. 5.0 mmol/L
E. 3.0 mmol/L

Answers:

1.D, 2.D, 3.C , 4.A , 5.C,6.A,7.D,8.B,9.C.