COMPLICATION OF DM

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Stay Tuned : World Diabetes Day

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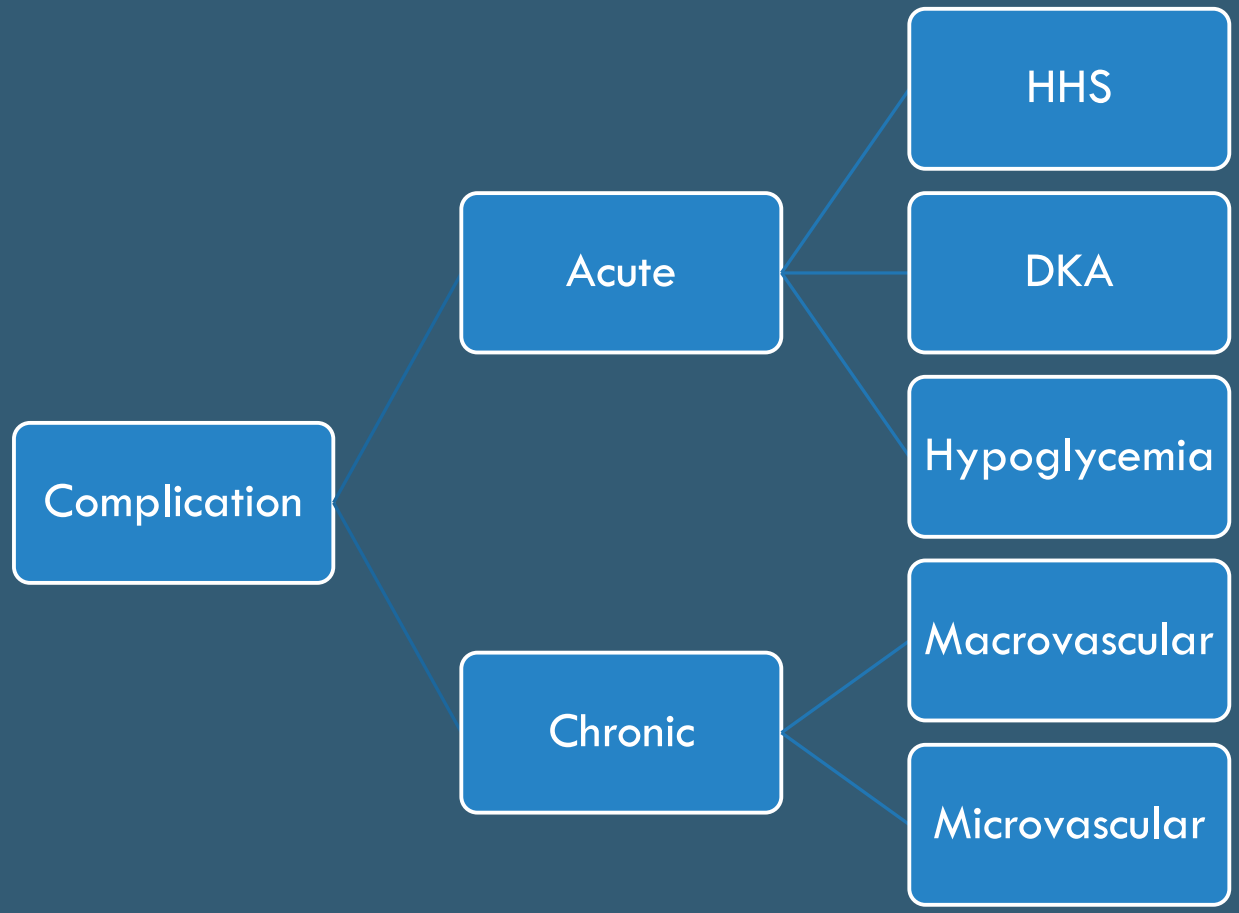
INTRODUCTION

Diabetes-related complications affect many organ systems and are responsible for most of the morbidity and mortality associated with the disease.

For many years in the United States, diabetes has been a leading cause of new blindness in adults, renal failure, and non traumatic lower extremity amputation and is a leading contributor to coronary heart disease (CHD).

DM complications are classified into :

CLASSIFICATION



DIABETIC KETOACIDOSIS (DKA)

* DKA is an acute complication of Diabetes that is a consequence of ABSOLUTE insulin deficiency .

* DKA most commonly occurs in people with type 1 diabetes and can be the first presentation of the condition .

* It can also occur in type 2 diabetes, particularly if it has been longstanding or in individuals who are ketosis-prone (have significant ß-cell dysfunction) or who have been misclassified as type 2 rather than type 1 diabetes .

In established diabetes, DKA may be precipitated by an underlying illness physiological stress , medication .

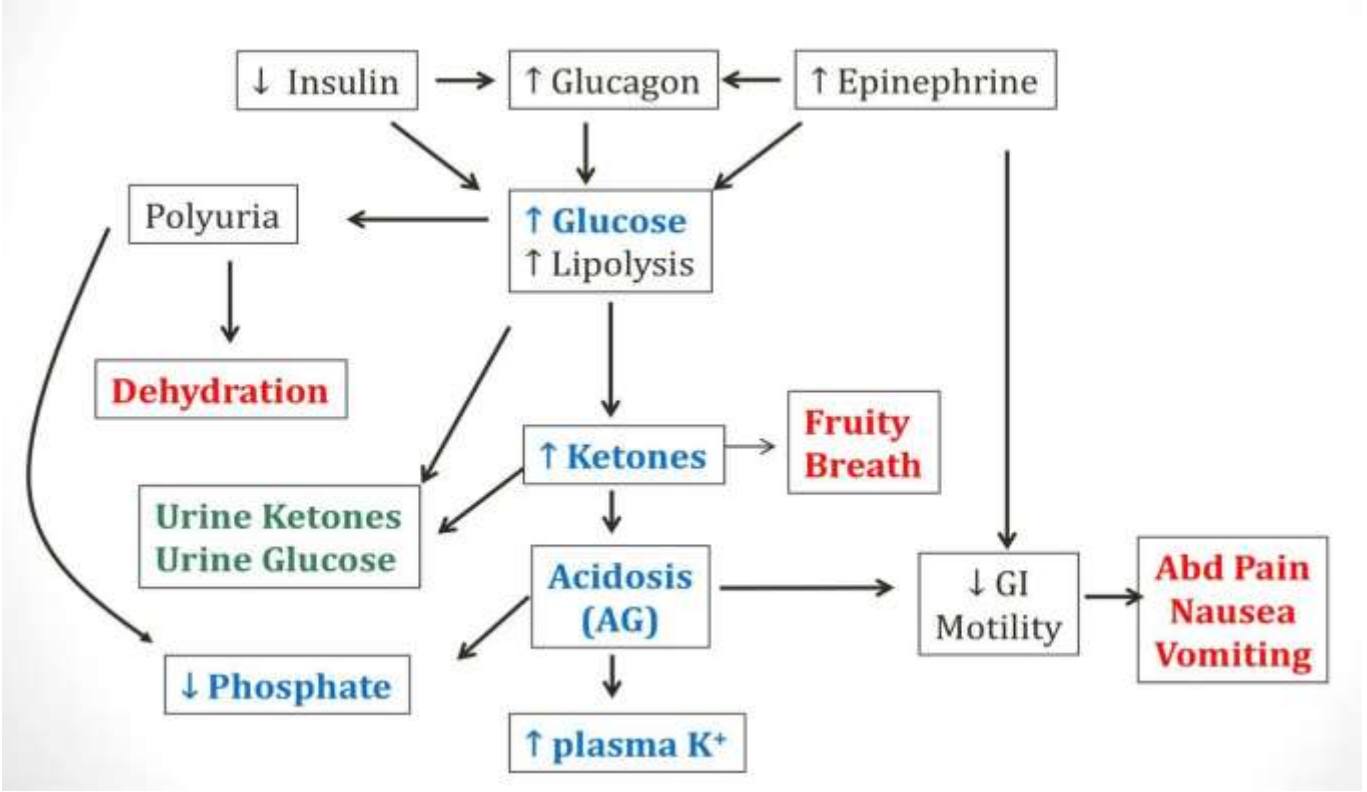
Infection is the most common of these: pneumonia and urinary tract infection are often implicated .

Acute myocardial infarction, cerebrovascular disease and pancreatitis are other common examples of a precipitating illness .

Physiological stress such as surgery, trauma and pregnancy can also be implicated.

The second most common cause of DKA is insulin deficiency due to discontinuation (accidental or deliberate)

or inadequate delivery (error in administration, insulin pump failure, pen malfunction, out of date or inadequate storage) of insulin .



PATHOPYSIOLOGY

CONT . . .

Insulin deficiency, together with excess glucagon (partly due to loss of insulin's normal suppressive effect), leads to a marked increase in glucose and ketone production. Other counter-regulatory hormones, including catecholamines, cortisol, and growth hormone, further amplify these effects.

A ↓ insulin / glucagon ratio stimulates:

- ↑ Lipolysis and proteolysis → increased gluconeogenic substrates (FFA, amino acids)
- ↑ Gluconeogenesis and glycogenolysis
- ↓ Peripheral glucose uptake

The net result is hyperglycemia (**plasma glucose > 11.1 mmol/L or > 200 mg/dL**, often with prior history of diabetes), leading to osmotic diuresis → polyuria → dehydration.

In parallel, excessive gluconeogenesis depletes oxaloacetate (required for entry of acetyl-CoA into the TCA cycle). As a result, acetyl-CoA is diverted toward ketogenesis, producing β-hydroxybutyrate, acetoacetate, and acetone (→ fruity breath odor). **Ketosis (β- hydroxybutrate concentration > 3.00 mmol/L OR more than 2+ on urine dipstick .** Accumulation of ketoacids increases hydrogen ion concentration, consumes bicarbonate, and results in high anion gap metabolic acidosis **(pH < 7.3 and/or HCO_3^- < 18 mmol/L .**

CONT . . .

There are significant deficit of water and electrolytes in DKA.

A- Serum K

Plasma potassium may even be raised initially due to :

disproportionate loss of water

catabolism of protein and glycogen

displacement of potassium from the intracellular compartment by H+ ions (depletion of intracellular K)

Furthermore with Osmotic diuresis will increase urine K LOSS > TOTAL body depletion of Potassium .

B- Serum Na (Hyponatremia)

Usually Secondary to :
Hyperglycemia leads to Increase osmotic flux of water from intracellular to extracellular space .

Obligate Sodium loss with Ketonuria .

CLINICAL FEATURES

* The clinical features of ketoacidosis usually progress rapidly over the course of several hours.

DKA should be considered if there are the typical presenting symptoms of diabetes (e.g 3Ps) in association with nausea, vomiting, abdominal pain (mimic acute abdomen) , hyperventilation, dehydration or reduced consciousness .

In a severe and rapidly progressing case, the striking features are

volume depletion , (sunken eyes), dry axillae, decreased

skin turgor , reduced jugular venous pressure, tachycardia and hypotension.

Breathing may be deep and sighing (**Kussmaul's breathing**) which may have the sweet smell of acetone, similar to nail polish remover .

21.11 Clinical features of diabetic ketoacidosis

Symptoms

- Polyuria, thirst
- Weight loss
- Weakness
- Nausea, vomiting
- Leg cramps
- Blurred vision
- Abdominal pain

Signs

- Dehydration
- Hypotension (postural or supine)
- Cold extremities/peripheral cyanosis
- Tachycardia
- Air hunger (Kussmaul's breathing)
- Smell of acetone
- Hypothermia
- Delirium, drowsiness, coma (10%)

EUGLYCEMIC DKA

Eu-DKA is a Rare form of diabetic ketoacidosis characterized by metabolic acidosis with elevated ketone bodies, **but normal or only mildly elevated blood glucose levels** (often < 250 mg/dL).

Prominent trigger: use of SGLT2 inhibitors (sodium-glucose co-transporter-2 inhibitors) in patients with diabetes, which enhance glucosuria and reduce plasma glucose levels, thereby masking hyperglycemia typical of DKA.

Other triggers are also reported .

Eu-DKA may be under-recognized because clinicians may rely on marked hyperglycemia as a hallmark of DKA; the absence of that may delay diagnosis and treatment.

Management :

- Approach similar to classic DKA: fluid resuscitation, insulin therapy, correction of electrolyte disturbances (especially potassium), treat precipitating cause.
- In patients on SGLT2 inhibitors: discontinue the SGLT2 inhibitor when eu-DKA suspected.

DIAGNOSIS

Table 6.8–Diagnostic criteria for DKA

DKA	
Diabetes/hyperglycemia	Glucose ≥200 mg/dL (11.1 mmol/L) or prior history of diabetes
Ketosis	β-Hydroxybutyrate concentration ≥3.0 mmol/L or urine ketone strip 2+ or greater
Metabolic acidosis	pH <7.3 and/or bicarbonate concentration <18 mmol/L

☐ Venous blood: for urea and electrolytes, glucose, bicarbonate and acid–base status to confirm the presence of **hyperglycaemia** and **acidosis. Hyponatraemia and hyperkalaemia are common.**

N.B : Serum amylase is usually elevated in DKA, but rarely indicates coexisting pancreatitis.

☐ Blood (or urine if unavailable) analysis for ketones .

☐ Electrocardiogram (ECG) to look for evidence of acute myocardial infarction or electrolyte abnormalities.

☐ Chest X-ray to look for evidence of lung consolidation or pulmonary oedema.

☐ Infection screen: full blood count, blood and urine culture, C-reactive protein . (N.B. leucocytosis occurs commonly in DKA, but generally represents a stress response rather than infection.)

☐ Pregnancy test: in all women of childbearing age.

TREATMENT

GOALS :

Restore circulatory volume and tissue/organ perfusion

Resolve ketoacidosis

Correct electrolyte imbalances, particularly potassium.

Fluid Replacement:

Severe hypovolemia : 0.9% Nacl or other crystalloid 1 L/h .

Mild hypovolemia : 0.9% Nacl or other crystalloid at a clincally appropriate rate aiming to replace 50% of the estimated fluid deficit in the first 8-12 hours.

Cardiac compromise : Hemodynamic monitoring / pressors.

Insulin Therapy

IV regular insulin **: 0.1 units/ kg IV bolus ,** then start a continuous IV infusion 0.1 units /kg per hour.

Continue the drip **until plasma ketone < 0.6 mmol/L and Venous pH > 7.3 OR bicarbonate > 18 mmol/L and patient is able to tolerate food .**

Transition to subcutaneous insulin 1-2 hours before stopping IV insulin to prevent **rebound hyperglycemia** .

Electrolyte Management:

Monitor and replace potassium—patients with DKA have a large total body K+ deficit. Potassium should be given as long as it is less than 5.0 mmol/L.

Otherwise , start insulin therapy and keep monitoring of K level every 2h .

Maintain serum potassium between 4-5 mEq/L .

Bicarbonate therapy :

generally not recommended unless pH <7.0.

HYPERGLYCAEMIC HYPEROSMOLAR STATE (HHS)

Hyperglycameic Hyperosmlar State (HHS) is a metabolic emergency characterized by hyperglycemia and hyperosmlarity **WITHOUT KETOACIDOSIS** in a DM -2 patient .

Mortality rates are higher than DKA (up to 20%) .

WHAT HAPPENS ?

Relative insulin deficiency and inadequate fluid intake are the underlying causes of HHS. Insulin deficiency increases hepatic glucose production (through glycogenolysis and gluconeogenesis) and impairs glucose utilization in skeletal muscle .

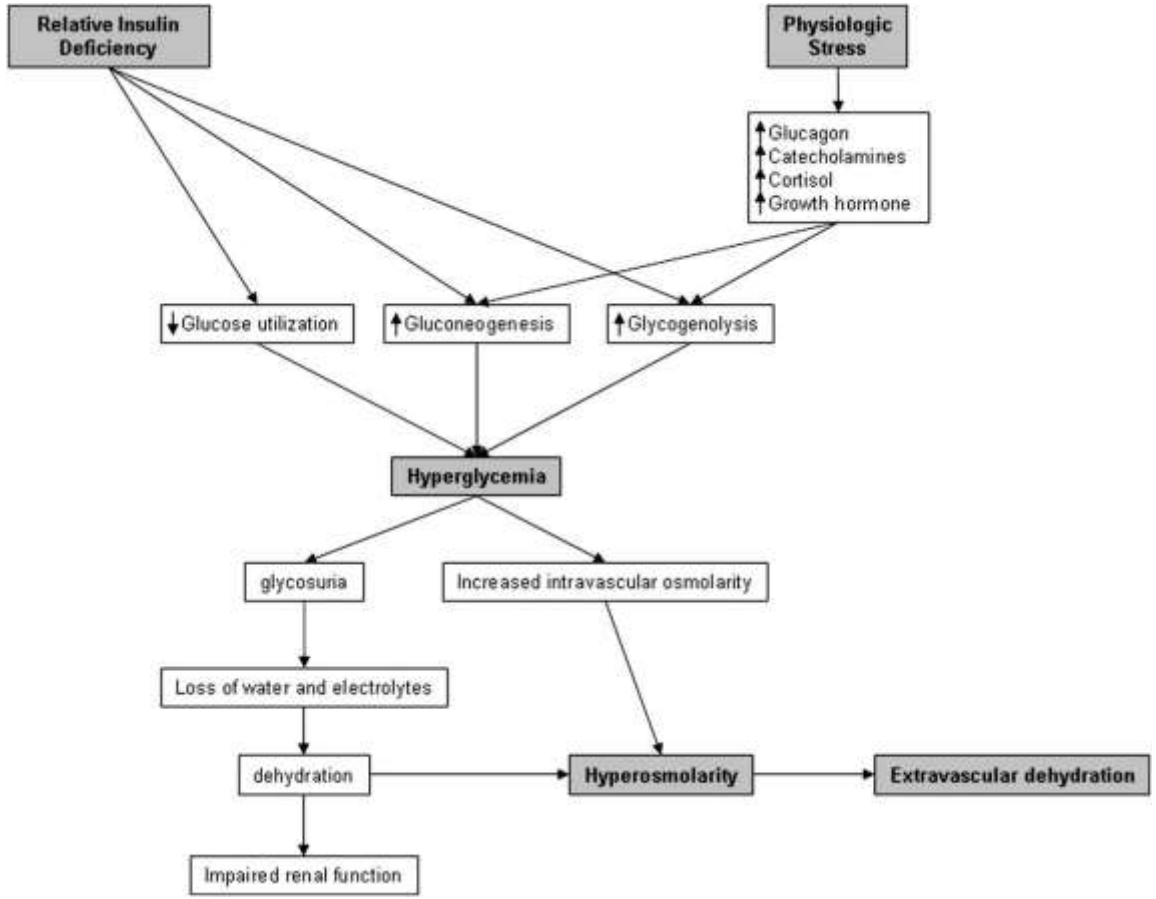
Hyperglycemia induces an osmotic diuresis that leads to intravascular volume depletion, which is exacerbated by inadequate fluid replacement.

The absence of ketosis in HHS is not understood.

Presumably, the insulin deficiency is only relative and less severe than in DKA .

Lower levels of counterregulatory hormones and free fatty acids have been found in HHS than in DKA in some studies.

It is also possible that the liver is less capable of ketone body synthesis or that the insulin/glucagon ratio does not favor ketogenesis



CLINICAL FEATURES

The most common presentation of HHS is an elderly individual with type 2 DM, with a several-week history of **polyuria, weight loss,** and **diminished oral intake** that culminates in **mental confusion , lethargy , or coma** .

The physical examination reflects profound dehydration and hyperosmolality and reveals hypotension, tachycardia, and altered mental status.

Notably absent are symptoms of nausea, vomiting, and abdominal pain and the Kussmaul respirations characteristic of DKA.

HHS is often precipitated by a serious, concurrcrent and other serious infections are frequent precipitants and should be sought.

In addition, a debilitating condition (prior stroke or dementia) or social situation that compromises water intake usually contributes to the development of the disorder.

DIAGNOSIS

Most notable findings are the **marked hyperglycemia :**

(plasma glucose may be > 600 mg/dL)

hyperosmolality :

Effective serum osmolality (>300 mOsm/kg)

total serum osmolality (> 320 mOsm/kg)

In contrast to DKA, **acidosis and ketonemia are absent or mild**. A **small anion-gap metabolic acidosis** may be present secondary to increased lactic acid (anaerobic respiration) .

Moderate ketonuria, if present, is secondary to starvation .

HHS	
Hyperglycemia	Plasma glucose ≥600 mg/dL (33.3 mmol/L)
Hyperosmolarity	Calculated effective serum osmolality >300 mOsm/kg (calculated as [2×Na^+ (mmol/L) + glucose (mmol/L)] or total serum osmolality >320 mOsm/kg [2×Na^+ (mmol/L) + glucose (mmol/L) + urea (mmol/L)]
Absence of significant ketonemia	β-Hydroxybutyrate concentration <3.0 mmol/L OR urine ketone strip less than 2+
Absence of acidosis	pH ≥7.3 and bicarbonate concentration ≥15 mmol/L

MANAGEMENT

Appropriate resuscitation with attention to the principle of Airway, Breathing, Circulation (ABC) should be initiated .

Patients with HHS can present with **altered mental status** as a result of significant fluid depletion and decreased cerebral perfusion .

Fluid Replacement is crucial , as mentioned previously ; with caution of co-morbidities .

Check for potassium level ; If K more than 5 mmol/L ; **START INSULIN THERAPY** otherwise K administration is indicated (establish adequate renal function) .

Normalization of Serum osmolality (< 320 mOsm/kg) .

TO SUM UP !

21.13 Typical characteristics of HHS and DKA

	HHS	DKA
Age	Older	Younger
Classification	Established pre-diabetes/type 2 diabetes or first presentation	Established type 1 diabetes or first presentation
Duration of onset	Days to weeks	Hours to days
Abdominal pain	Uncommon	Common
Volume depletion	10%–20%	5%–10%
Insulin deficiency	Relative	Absolute
Glucose	Usually >30 mmol/L	Usually >11.1 mmol/L
Ketonaemia	<3.0 mmol/L	>3.0 mmol/L
Acidosis	Bicarbonate >15 pH >7.3	Bicarbonate <15 pH 7.0–7.3

(DKA = diabetic ketoacidosis; HHS = hyperglycaemic hyperosmolar state)

HYPOGLYCEMIA

Hypoglycaemia has been defined by the American Diabetes Association in three distinct levels : **level 1** (alert value), **level 2** (clinically significant) and **level 3** (severe).

Hypoglycaemia seldom occurs in people without diabetes, but is very common in people with diabetes, mainly due to **insulin** and (to a lesser extent) **sulphonylurea or Meglitinides therapy** (i.e. complication of treatment) .

21.14 Levels of hypoglycaemia

- Level 1 hypoglycaemia (*alert value*) as <3.9 mmol/L but ≥3.0 mmol/L (<70 mg/dL but ≥54 mg/dL)
- Level 2 hypoglycaemia (*clinically significant*) is defined as <3.0 mmol/L (<54 mg/dL). This level is unequivocally low and suggests clinically important biochemical hypoglycaemia
- Level 3 hypoglycaemia (*severe*) is defined as any low blood glucose level leading to cognitive impairment and the need for external assistance to provide glucose, glucagon or other corrective activity

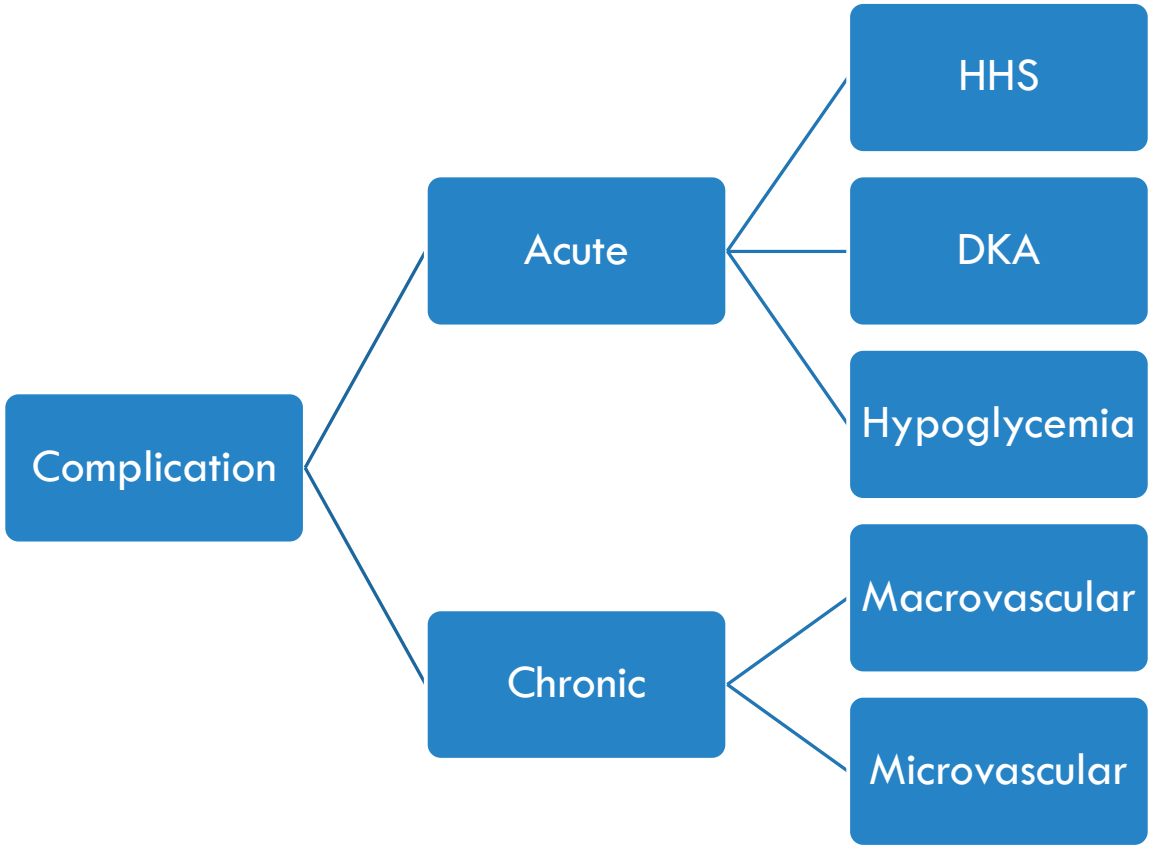
CLINICAL FEATURES AND MANAGEMENT

Neurogenic (autonomic) symptoms: diaphoresis, tachycardia, tremor, anxiety, hunger. Allow perception of reduced glucose (hypoglycemia awareness) .

Neuroglycopenic symptoms: altered mental status, seizures, death due to insufficient glucose in CNS. May occur in the absence of preceding neurogenic symptoms in patients with attenuated autonomic response (hypoglycemia unawareness).

For symptomatic hypoglycemia, the patient should take 15–20 g of fast-acting carbohydrate, then recheck glucose after 15 minutes and repeat if still ≤70 mg/dL (3.9 mmol/L), followed by a longer-acting carb snack or meal to prevent recurrence. **Severe hypoglycemia** requires help from another person: if IV access is available, give 25 g of IV dextrose (50%); if IV access is not available, treat with glucagon (subcutaneous, intramuscular, or intranasal).

CLASSIFICATION



CHRONIC COMPLICATION OF DM

Chronic hyperglycemia is the important etiologic factor leading to complication that are likely multifactorial with an emerging hypothesis that hyperglycemia leads to epigenetic changes that influence gene expression in affected cells.

Chronic hyperglycemia leads to formation of **advanced glycosylation end products** (AGEs; e.g., pentosidine, glucosepane, and carboxymethyllysine), which bind to specific cell surface receptor and/or the nonenzymatic glycosylation of intra- and extracellular proteins, leading **to cross-linking of proteins, glomerular dysfunction, endothelial dysfunction, altered extracellular matrix composition, and accelerated atherosclerosis .**

* **In TYPE 1 DIABETES ,** the attained age and the duration of diabetes appear to correlate with the degree of complication usually (5-10 years after diagnosis) .

* **In TYPE 2 DIABETES ,** the complication itself may be present at the time of diagnosis as the first manifestation (especially **MACROVASCULAR COMPLICATIONS**) .

MACROVASCULAR COMPLICATION

ATHEROSCLEROSIS accounts for approximately 80% of all diabetic mortality (75% from coronary atherosclerosis and 25% from cerebral or peripheral vascular disease) and

more than 75% of all hospitalizations for diabetic complications.

More than 50% of patients with newly diagnosed type 2 diabetes already have coronary heart disease.

so **CAD IS THE MOST COMMON CAUSE OF DEATH IN DM PATEINTS** .

Clinical presentation :

1. **Coronary artery disease** may present with chest pain and/or silent ischemia

2. **Peripheral arterial disease (PAD)** may present with claudication or nonhealing extremity ulcers.

3. **Cerebrovascular disease** may present **as transient ischemic attacks (TIA) or stroke;** carotid bruits on physical exam may indicate underlying cerebrovascular disease.

Screening:

Annual screening for dyslipidemia (ASK FOR FULL LIPID PANEL – BY ADA) and hypertension. Exercise stress test and ECG .

Treatment (MAINLY EARLY GLYCEMIC CONTROL) :

aggressive management of hypertension (ACEIs . ARBs)and dyslipidemia (STATINS) , smoking cessation, andaspirin prophylaxis. Statin should be given for all diabetics >40-year-old regardless of LDL level

2. NEPHROPATHY

- Diabetic nephropathy is an important cause of morbidity and mortality in both type 1 and type 2 diabetes.

- **20%-30%** of patients with type 1 or type 2 diabetes develop evidence of nephropathy, but a smaller fraction progress to end-stage renal disease (ESRD) in type 2 diabetes.

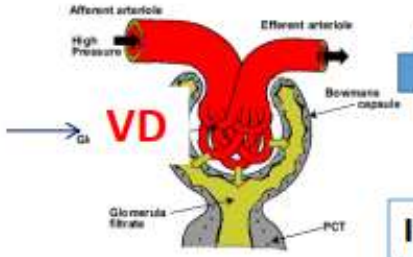
- The pathophysiology is not fully understood and there are several postulated mechanisms by which hyperglycemia causes the pathological changes seen in diabetic nephropathy. **The central features are activation of the renin–angiotensin system, leading to both intrarenal and systemic effects, as well as direct toxic effects of prolonged hyperglycemia, leading to renal inflammation and fibrosis.** ʕʕʕʕʕʕʕʕʕʕʕʕ

1 Decrease in contractile ability of meningeal cells

2 Immune Dysfunction

3 Non enzymatic glycosylation of proteins

4 Exceeded the capacity of glycolysis



VD

↑GFR

Pathogenesis of DM

Infections

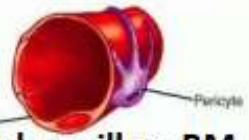
AGEs

Attached to collagen in vessel wall

Impair interaction between lamina propria and proteoglycans → Increase capillary permeability → Proteinuria

Irreversible cross linkage to LDL → Atherosclerosis

Irreversible cross linkage to Albumin → Thick capillary BM

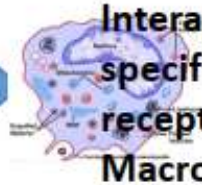


Microangiopathy

Retinopathy

Neuropathy

Interact with specific receptors on Macrophages → IL1 TNF



Polyol pathway → Sorbitol → Eye damage

Sorbitol → Neuropathy

DIABETIC KIDNEY DISEASE

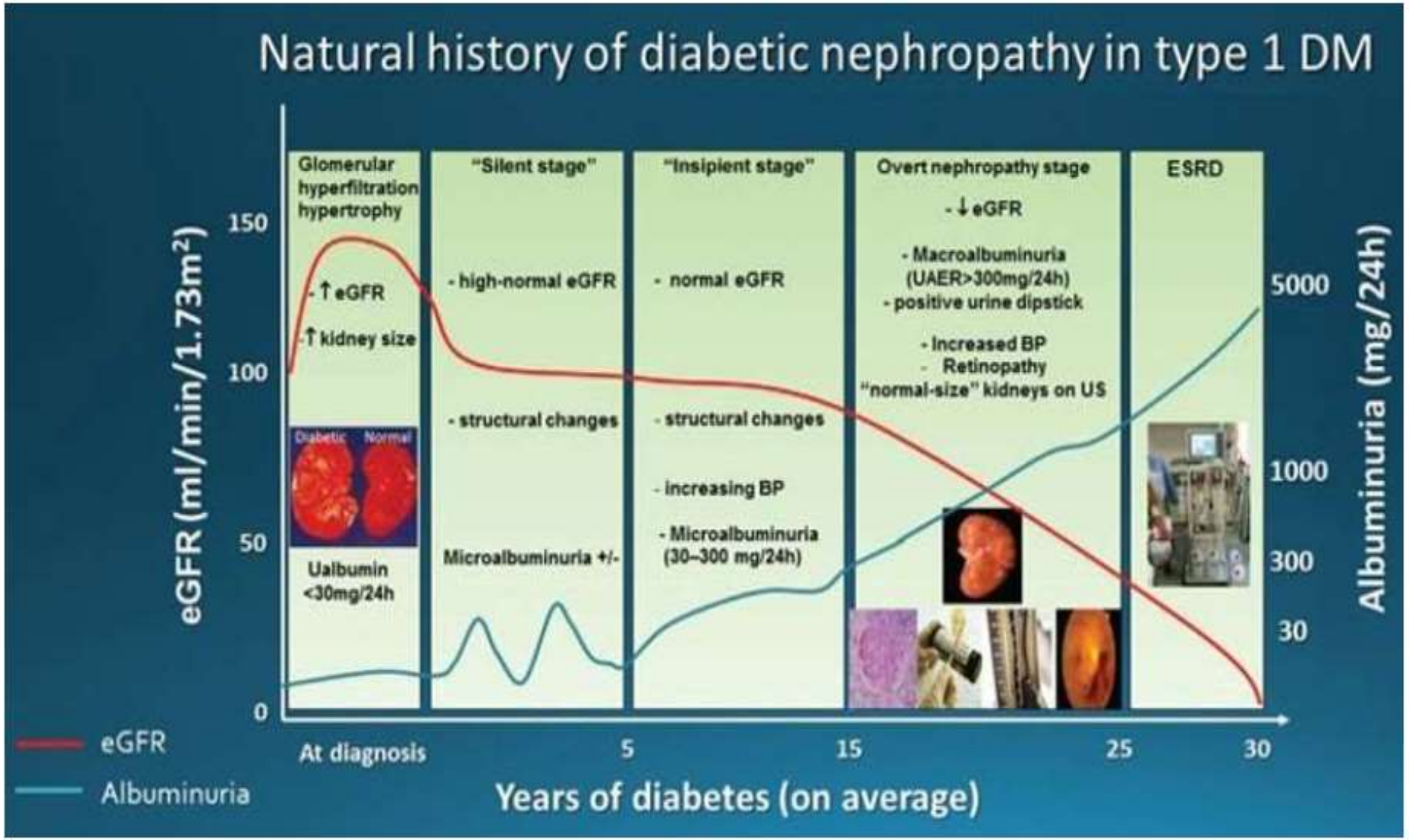
- ❶Many factors play a role in the pathogenesis of Diabetic nephropathy.
These include:

- Hyper-filtration injury (due to GFR)- Atherosclerosis- InfectionsVUR (due to autonomic neuropathy)- and others.

- ❷The condition can range from mild proteinuria to End Stage Renal Failure (ESRF).
- (Proteinuria is considered to be the main feature of this condition)

- ❸Early proteinuria could be diminished by the use of ACE-I

- Stage I: Increase GFR-increase kidney sizemicro albuminuria more than 15ug/minute BUT less than 100 ug/minute-NO increase in incidence of Hypertension.
- Stage II: Declining GFR-increasing proteinuria that may reach nephrotic range within years (Hypertension and Edema?!)
- Stage III: Rapid decrease in GFR that leads to Azotemia-Hypertension and oedema worsens…Finally, Hypoproteinemia and widespread microangiopathy.
- Stage IV: ESRF


Natural history of diabetic nephropathy in type 1 DM
eGFR (ml/min/1.73m²)
150
100
50
0
Albuminuria (mg/24h)
5000
1000
300
30
Glomerular hyperfiltration hypertrophy
- ↑ eGFR
- ↑ kidney size
Diabetic
Normal
Ualbumin <30mg/24h
"Silent stage"
- high-normal eGFR
- structural changes
Microalbuminuria +/-
"Insipient stage"
- normal eGFR
- structural changes
- increasing BP
- Microalbuminuria (30–300 mg/24h)
Overt nephropathy stage
- ↓ eGFR
- Macroalbuminuria (UAER>300mg/24h)
- positive urine dipstick
- Increased BP
- Retinopathy
"normal-size" kidneys on US
ESRD
eGFR
Albuminuria
At diagnosis
5
15
25
30
Years of diabetes (on average)

CLINICAL PRESENTATION: Patients usually asymptomatic until late in course. Characterized by proteinuria and rising creatinine

- **SCREENING:** urine albumin measurement should be performed **annually** to screen for diabetic nephropathy **after 5 years of disease duration** in patients with type 1 diabetes and at diagnosis in patients with type 2 diabetes. **Albumin/creatinine ratio is the ideal.**
- **TREATMENT:**
- 1. **ACE inhibitors** is recommended for all hypertensive and non-hypertensive patients with type 1 diabetes and albuminuria.
- 2. **Angiotensin II receptor blockers (ARBs)** are the agents of first choice in hypertensive type 2 diabetic patients with albuminuria.
- **Hypertension goal: < 130/80 mmHg**

RETINOPATHY

RETINOPATHY

- **DIABETIC RETINOPATHY** is the **MOST COMMON** pattern of **EYE DISEASE** and the leading cause of blindness in adults ages 20 to 74 years, and its presence is strongly related to the duration of diabetes (mostly after 20 years of DM with prevalence is **75%)**

- **The prevalence of DR increases with duration of diabetes**, and almost all individuals with type 1 diabetes and the majority of those with type 2 diabetes will **have some degree of DR after 20 years.**

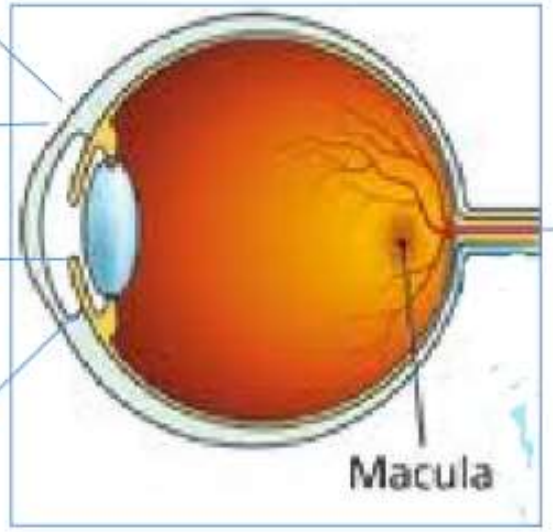
DIABETIC EYE DISEASE

Recurrent sties

Xanthlasma

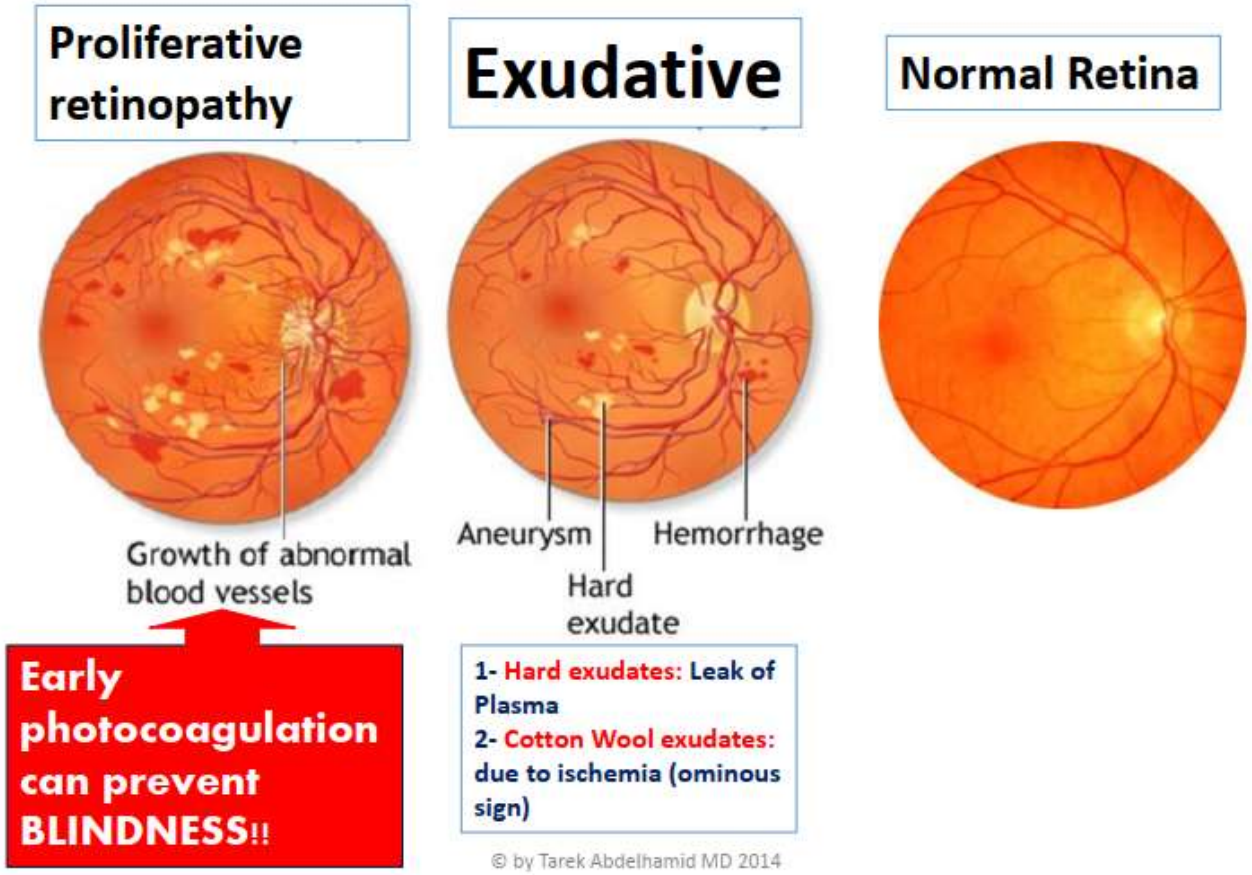
Cataract

Glaucoma

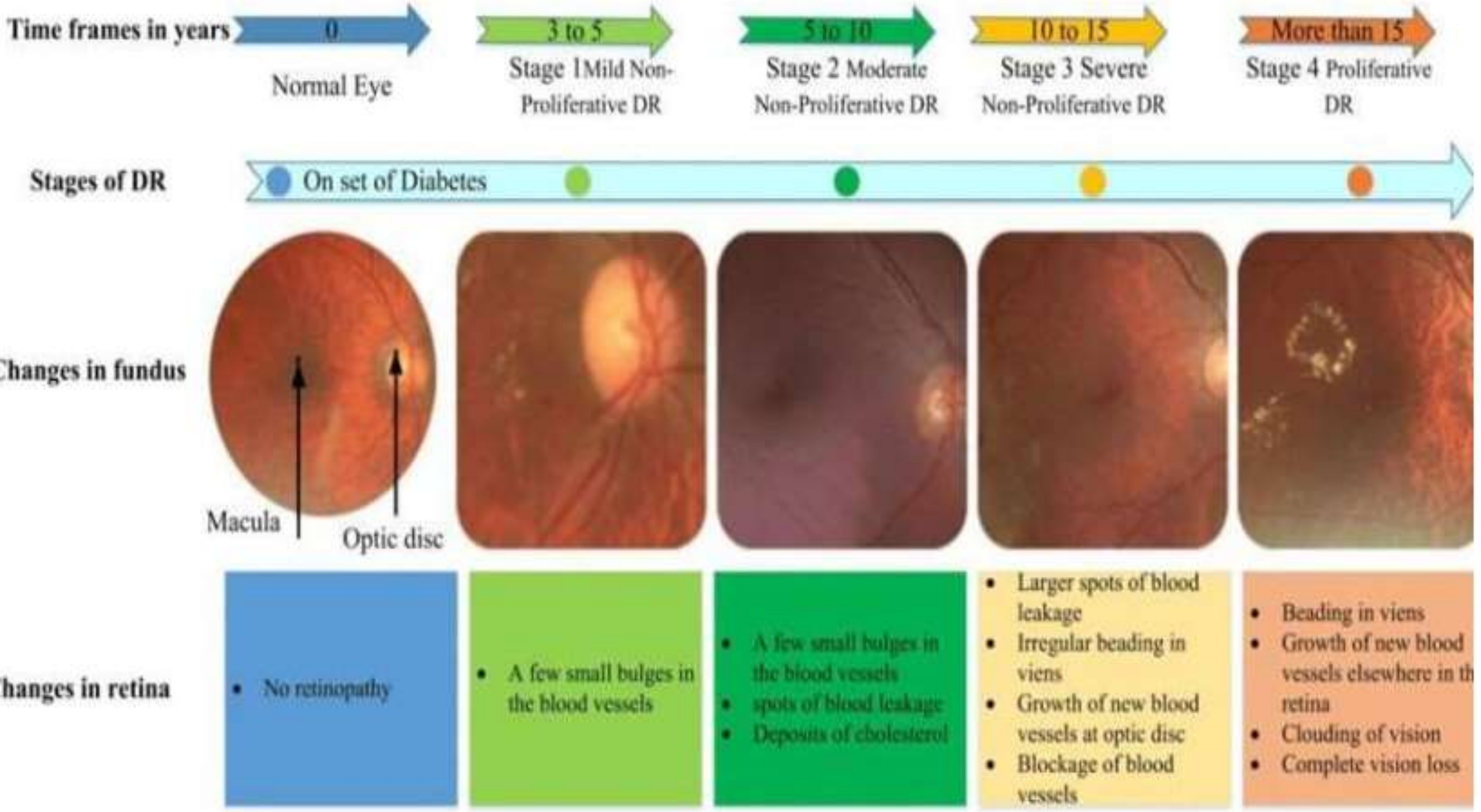


Retinopathy

DIABETIC RETINOPATHY



- Clinical presentation:
- asymptomatic unless a serious pathophysiology occure (causing visual loss).
- Screening:
- ANNUALLY Ophthalmologic evaluation should be performed starting 5 years after diagnosis of type 1 diabetes and on initial diagnosis of type 2 diabetes. Less frequent exams (every 2 to 3 years) can be considered in those with several normal eye exams.
- Treatment:
- Refer to ophthalmologist
- 1- The lipid-lowering drug ,FENOFIBRATE, has been shown to slow the progression of retinopathy.
- 2- Intraocular anti-VEGF agents
- 3- Laser photocoagulation
- 4- Vitreoretinal surgery


Time frames in years
0
Normal Eye
3 to 5
Stage 1 Mild Non-Proliferative DR
5 to 10
Stage 2 Moderate Non-Proliferative DR
10 to 15
Stage 3 Severe Non-Proliferative DR
More than 15
Stage 4 Proliferative DR
Stages of DR
On set of Diabetes
Changes in fundus
Macula
Optic disc
Changes in retina
No retinopathy
A few small bulges in the blood vessels
A few small bulges in the blood vessels
spots of blood leakage
Deposits of cholesterol
Larger spots of blood leakage
Irregular beading in viens
Growth of new blood vessels at optic disc
Blockage of blood vessels
Beading in viens
Growth of new blood vessels elsewhere in th retina
Clouding of vision
Complete vision loss

MICROVASCULAR COMPLICATIONS

3. NEUROPATHY

About **HALF OF ALL PEOPLE WITH DIABETES** have some form of nerve damage. It is more common in those who have had diabetes for many years and can lead to various health problems down the line, impacting your quality of life .

NEUROPATHY INTITIES :

1 PROXIMAL NEUROPATHY
2 MONO - NEUROPATHIES
3 AUTONOMIC NEUROPATHY
4 PERIPHERAL NEUROPATHY
5 OTHER ADDITIONAL SUPTYPES

1- PERIPHERAL NEUROPATHY

MOST COMMON

"Distal symmetric polyneuropathy"
It affects the nerves in the hands, feet, legs, and arms. It generally starts in the feet, and it tends to start in both feet at once.

- "**STOCKING-GLOVE**" sensory loss
- Progressive loss of sensation: distal → proximal
- Severe cases: motor weakness

- ***THE CLINICAL MANIFESTATIONS** of the diabetic peripheral neuropathies vary with the location of the lesion:

1. **Tingling**
2. **Pain or increased sensitivity**
3. **Numbness or weakness**
4. **Muscle and bone deformity**

	Large Myelinated Fibers	Small Myelinated Fibers
Function	Proprioception/Pressure	Pain
Symptoms	Numbness, loss of balance	Burning, electric shocks
Exam Findings	Reduced ankle reflexes Reduced vibration Reduced proprioception	Loss of pinprick Loss of hot/cold discrimination

#DIAGNOSIS
Foot exams / Nerve conduction studies and electromyography (EMG)

#Painful diabetic neuropathy –hypersensitivity to light touch ,sever burning pain ,especialy at night that can be difficult to tolerate

-Treatment is with GABAPENTIN

2- AUTONOMIC NEUROPATHY is damage to nerves that control your internal organs.

- UG system **(MOST COMMON):** bladder dysfunction , erectile dysfunction.
- GI: gastroparesis is diagnosed when there is an objectively measured delay in gastric emptying in the absence of mechanical obstruction.) (The main symptoms are chronic nausea, vomiting (especially of undigested food), abdominal pain and a feeling of fullness/early satiety)
- CV: orthostatic hypotension, SILENT ISCHEMIA ?!

3- MONO - NEUROPATHIES Focal neuropathies are conditions in which you typically have damage to single nerves, most often in your hand, head, torso, and leg.

***"The cause is often acute thrombosis or ischemia of the vasa nervorum, leading to nerve infarction—commonly seen in conditions such as :

1 ulnar neuropathy
2 common peroneal neuropathy
3 median nerve neuropathy (carpal tunnel syndrome)

- Course: Usually reversible within 1–3 months.

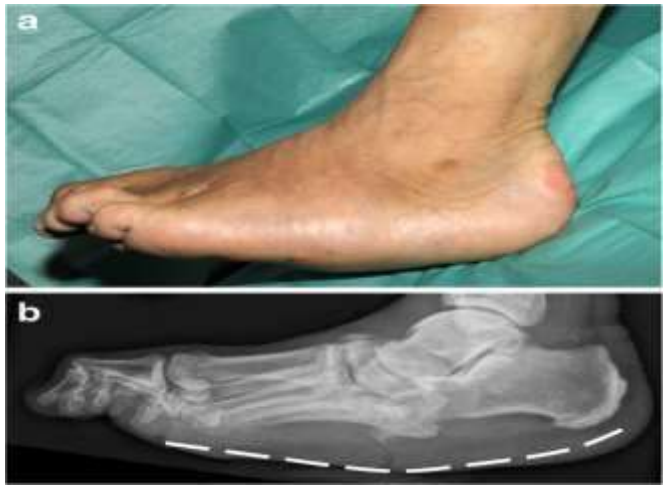
4-PROXIMAL NEUROPATHY Proximal neuropathy is a rare and disabling type of nerve damage in your hip, buttock, or thigh.(diabetic lumbosacral plexopathy)

This type of nerve damage typically affects one side of your body and may rarely spread to the other side.

Proximal neuropathy often causes severe pain and may lead to significant weight loss

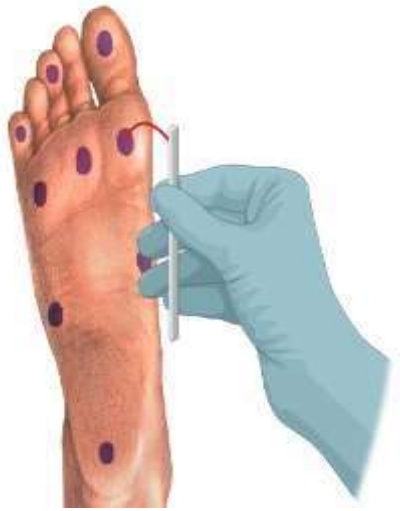
QUIQ NEUROPHATY RECAB :

SYMPTOMS: Burning pain, electrical or stabbing sensations, paresthesia, hyperesthesia, deep aching pain occurring in the feet and lower limbs; symptoms worse at night.



COMPLICATIONS: repetitive injury to the foot, ulcers and Charcot joints. May ended with amputation of the foot.

SCREENING: Careful clinical exam with annual screening by examining pinprick, temperature, and vibration perception (using 128- Hz tuning fork), 10-g monofilament pressure sensation at distal halluces, and ankle reflexes.



TREATMENT: optimal blood glucose control with TCAs, SSRIs, capsaicin cream and gabapentin for NEUROPATHIC PAIN.

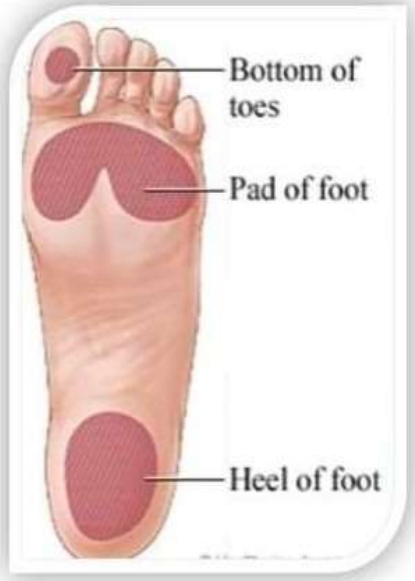
DIABETIC FOOT ULCER

- Distal symmetric neuropathy is a major risk factor for foot ulcers in 25% of DM patients.
- * Unaware of the constant trauma to the feet caused by poorly fitting shoes, or infections.
- * Motor neuropathy with weakness of the intrinsic muscles of the foot may result in foot deformities, which lead to focal areas of high pressure a foot ulcer.

- * **Common sites of trauma** are the back of the heel, the plantar metatarsal area, or the great toe, where weight is borne during walking.

SO … It's Common problem in diabetes :

- Loss of sensation → tissue damage
- Vascular disease → impaired healing
- May be painless → WORSINIG → infection / ulcerations / gangrenes / amputations ***SO… Patients should check feet daily.



CLICAL PRESENTATIONS :

Skin discoloration , temperature changes and delayed of any injury healing

Generally NOT REVERSIBLE (**EARLY management PREVENT further progression**)

- Glucose control slows progression

- **RELIVING AGENTS :**
- Pain in feet (burning or stabbing)
- SNRIs: duloxetine or venlafaxine
- TCAs: amitriptyline, desipramine or nortriptyline
- AEDs: pregabalin or gabapentin

- **SCREENING / FOLLOW UP :**
- **# Annual foot exam and sensory testing**
- **MONOFILAMENT TESTING (PRESSURE)**
- Vibratory testing
- Pinprick testing
- Ankle reflexes

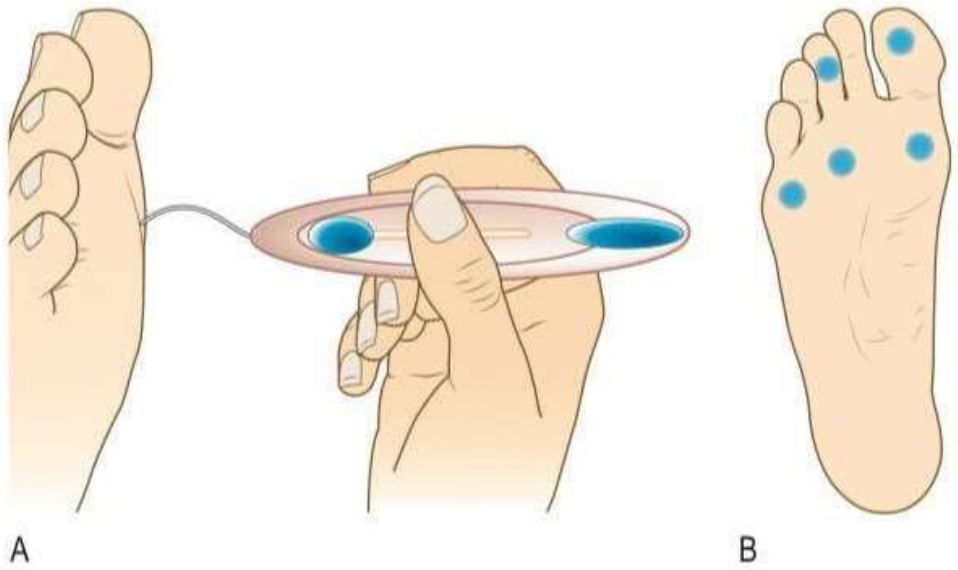
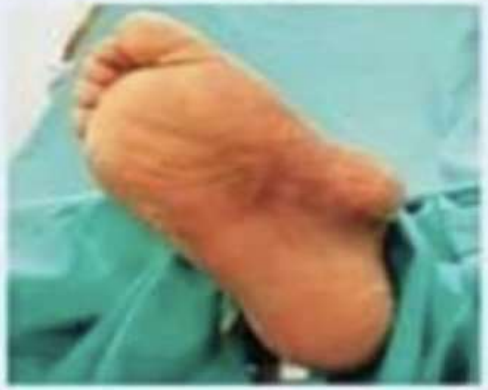
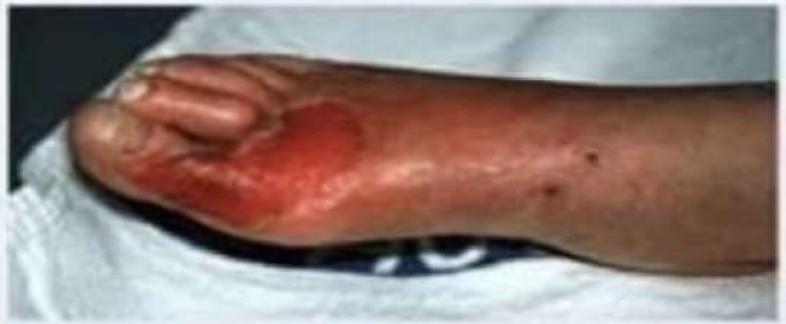
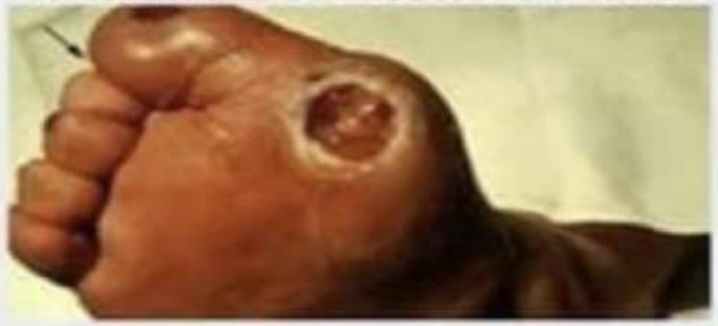
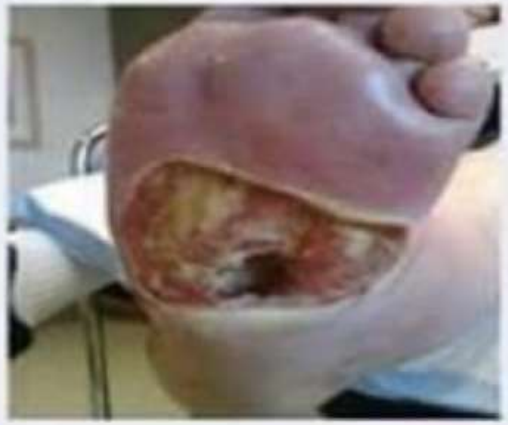
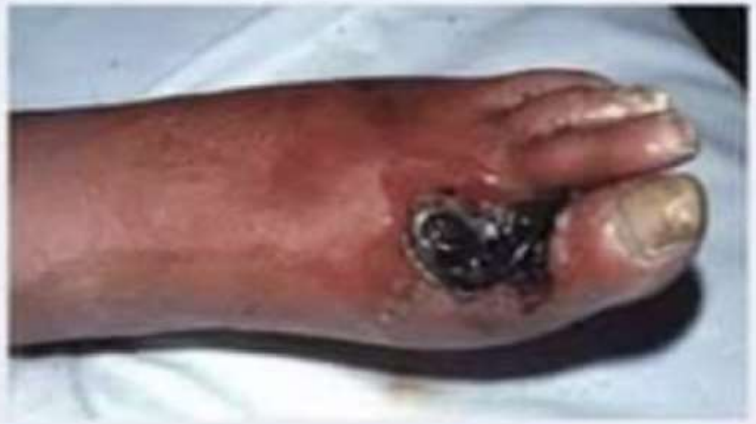


Fig. 10.17 Monofilament sensory testing of the diabetic foot.
A Apply sufficient force to allow the filament to bend. B Sites at highest risk (toes and metatarsal heads).

Wagner classification of diabetic foot ulcers

Grade 0	Grade 1	Grade 2
No ulcer in a high-risk foot 	Superficial ulcer involving the full skin thickness but not underlying tissues 	Deep ulcer, penetrating down to ligaments and muscle, but no bone involvement or abscess formation 
Grade 3	**Grade 4**	**Grade 5**
Deep ulcer with cellulitis or abscess formation, often with osteomyelitis 	Localized gangrene 	Extensive gangrene involving the whole foot 

10.8 Risk assessment of the diabetic foot

Level of risk	Definition	Action required
Low	No sensory loss, peripheral vascular disease or other risk factors	Annual foot screening can be undertaken by any trained healthcare professional
Moderate	One risk factor present, e.g. absent pulses or reduced sensation	Annual foot screening should be undertaken by a podiatrist
High	Previous ulceration or amputation, or more than one risk factor present	Annual screening should be undertaken by a specialist podiatrist
Active foot disease	Ulceration, spreading infection, critical ischaemia or an unexplained red, hot, swollen foot	Prompt referral to a multidisciplinary diabetic foot team is required

INFECTIONS

Certain types of infections occur with increased frequency in people with diabetes:

1. Soft tissue infections of the extremities
2. Osteomyelitis
3. Urinary tract infections and pyelonephritis
4. Candida infections of the skin and mucous surfaces
5. Dental caries and infections
6. Tuberculosis *Suboptimal response to infection in a person with diabetes is caused by the presence of chronic complications, such as vascular disease and neuropathies, and by the presence of hyperglycemia and altered neutrophil function.

*** HYPERGLYCEMIA AND GLYCOSURIA MAY INFLUENCE THE GROWTH OF MICROORGANISMS AND INCREASE THE SEVERITY OF THE INFECTION**


DISEASE
DIABETES
BLOOD SUGAR
HORMONE
INSULIN
BLOOD GLUCOSE
PANCREAS
HEALTH
A1C
TYPE 1
TYPE 2
Thank you