DM Complications

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Complications of diabetes mellitus

Acute complications:

ketoacidosis

Hyperglycemic hyperosmolar non ketotic syndrome

Hypoglycemia

Chronic complications:

Disorders of the microcirculation

Neuropathies

Nephropathies

Retinopathies

Macrovascular complications

Foot ulcers

Diabetic ketoacidosis (DKA)

*Most commonly occurs in type 1 diabetes, lack of insulin leads to mobilization of fatty acids from adipose tissue, increase in fatty acid levels leads to ketone production by the liver.

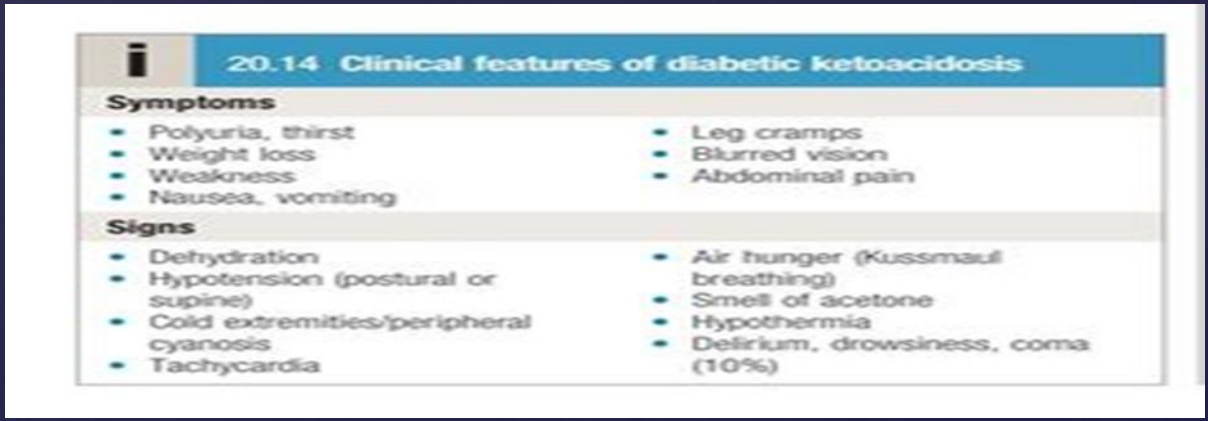
* DKA often is preceded by physical or emotional stress, such as infection, pregnancy, or extreme anxiety. In clinical practice, ketoacidosis also occurs with the omission or inadequate use of insulin.

20.15 Indicators of severe diabetic ketoacidosis

- Blood ketones >6 mmol/L
- Bicarbonate <5 mmol/L
- Venous/arterial pH <7.0 (H^+ >100 nmol/L)
- Hypokalaemia on admission (<3.5 mmol/L)
- Glasgow Coma Scale score <12 (p. 194) or abnormal AVPU scale score (p. 188)
- O_2 saturation <92% on air
- Systolic blood pressure <90 mmHg
- Heart rate >100 or <60 beats per minute
- Anion gap >16 mmol/L

Manifestation

Typically history of 1 or 2 days of polyuria, polydipsia, nausea, vomiting, and marked fatigue , stupor , coma.
Abdominal pain and tenderness may be present without abdominal disease. (glucose waste product)
The breath has a characteristic smell because of the presence of the volatile ketoacids. The rate and depth of respiration increase (i.e., Kussmaul's respiration) as the body attempts to prevent further decreases in pH
Hypotension may be present because of a decrease in blood volume.



20.14 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 breathing)
- Smell of acetone
- Hypothermia
- Delirium, drowsiness, coma (10%)

Treatment

The goals in treating DKA are:

* To improve circulatory volume and tissue perfusion
* To decrease serum glucose
*To correct the acidosis and electrolyte imbalances
*Accomplished through the administration of insulin and intravenous
fluid and electrolyte replacement solutions.
*Identification and treatment of the underlying cause.

The hyperglycemic hyperosmolar nonketotic (HHNK) syndrome

HHNK is characterized by
* Hyperglycemia (blood glucose>600 mg/dL),
*Hyperosmolarity (plasma osmolarity>310 mOsm/L) and dehydration
*Absence of ketoacidosis
* It is seen most frequently in people with type2 diabetes.
* Two factors appear to contribute to the hyperglycemia that precipitates the condition:
* An increased resistance to the effects of insulin
* An excessive carbohydrate intake.

The most prominent manifestations are

*Dehydration

* Neurologic signs and symptoms:
  Generalized seizures
  Hemiparesis
  Aphasia
  Muscle fasciculations
  Hyperthermia
  Visual field loss
  Nystagmus
  Visual hallucinations

* Excessive thirst

* The onset of HHNK syndrome often is insidious, and because it occurs

most frequently in older people, it may be mistaken for a stroke.

Treatment

The goals of treatment are same as DKA but need more intensive circulatory volume.
* Judicious medical observation and care because water moves :: brain cells during treatment :: cerebral edema.
* Extensive K+ losses occurred during the diuretic phase of the disorder require correction.

Hypoglycemia

*relative excess of insulin in the blood and is characterized by below-normal blood glucose levels.

* It occurs most commonly with insulin injections and some oral hypoglycemic agents (i.e., beta cell stimulators).

* Many factors precipitate an insulin reaction in a person with type1 diabetes, including:

Error in insulin dose

Failure to eat

Increased exercise

Decreased insulin need after removal of a stress situation

Alcohol decreases liver gluconeogenesis, and people with diabetes need to be cautioned about its potential for causing hypoglycemia.

Brain function relies on blood glucose (energy source)

*Hypoglycemia produces behaviors related to altered cerebral function:

* Headache

* Difficulty in problem solving

* Disturbed or altered behavior

* Coma

* Seizures

* At the onset of the hypoglycemic episode, activation of the parasympathetic nervous system often causes hunger:: if not corrected:: parasympathetic response is followed by activation of the sympathetic nervous system; this causes anxiety, tachycardia, sweating, and constriction of the skin vessels (i.e., the skin is cool and clammy).

The most effective treatment

*Immediate ingestion of a concentrated carbohydrate source, such as sugar, honey, candy , or orange juice.

* Alternative methods when the person having the reaction is unconscious or unable to swallow:

* Glucagon may be given intramuscularly or subcutaneously.

* In situations of severe or life-threatening hypoglycemia, it may be necessary to administer glucose intravenously.

Chronic complications

These disorders occur in the insulin-independent tissues of the body tissues that do not require insulin for glucose entry into the cell:: intracellular glucose concentrations in many of these tissues approach or equal those in the blood.

* Chronic complications can be reduced by intensive diabetic treatment.

Peripheral neuropathy

Two types of pathologic changes with diabetic peripheral neuropathies.

* The first is a thickening of the walls of the nutrient vessels that supply the nerve, leading to the assumption that vessel ischemia plays a major role in the developmentof these neural changes.

*The second finding is a segmental demyelination process that affects the Schwann cell. This demyelination process is accompanied by a slowing of nerve conduction.

*The clinical manifestations of the diabetic peripheral neuropathies vary with the location of the lesion

Peripheral neuropathy

Peripheral neuropathy is nerve damage that typically affects the feet and legs and sometimes affects the hands and arms.

Autonomic neuropathy

Autonomic neuropathy is damage to nerves that control your internal organs. Autonomic neuropathy can lead to problems with your heart rate and blood pressure, digestive system, bladder, sex organs, sweat glands, eyes, and ability to sense hypoglycemia.

Focal neuropathies

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

Proximal neuropathy

Proximal neuropathy is a rare and disabling type of nerve damage in your hip, buttock, or thigh. 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.

Diabetic nephropathy

* Cause of end-stage renal disease, accounting for 40%-50% of new cases.

*The term diabetic nephropathy is used to describe the combination of lesions that often occur concurrently in the diabetic kidney :: Glomerular changes may occur, including capillary basement membrane thickening, diffuse glomerular sclerosis, and nodular glomerulosclerosis.

*Among the suggested risk factors for diabetic nephropathy are:

1. Genetic and familial predisposition
2. Elevated blood pressure
3. Poor glycemic control
4. Smoking
5. Hyperlipidemia
6. Microalbuminuria

Pathogenesis

Hyperglycemia increases the expression of (TGF-beta) in the glomeruli and of matrix proteins specifically stimulated by this cytokine. TGF-beta may contribute to the cellular hypertrophy and enhanced collagen synthesis observed in persons with diabetic nephropathy :: renal hemodynamic alterations, patients with overt diabetic nephropathy (dipstick-positive proteinuria and decreasing (GFR) generally develop systemic hypertension.

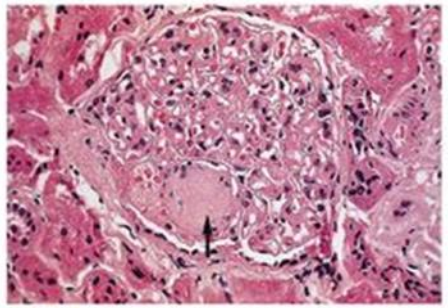


Fig. 20.22 Nodular diabetic glomerulosclerosis. There is thickening of basement membranes, mesangial expansion and a Kimmelstiel–Wilson nodule (arrow), which is pathognomonic of diabetic kidney disease.

Diabetic retinopathy

Diabeticretinopathy is the most common pattern of eye disease. It is characterized by
* abnormal retinal vascular permeability
* microaneurysm formation
* neovascularization
* hemorrhage, scarring, and retinal detachment.
* Risk factors associated with diabetic retinopathy are poor glycemic control, elevated blood pressure, and hyperlipidemia
* Another condition called macular edema :: damaged blood vessels leak fluid and lipids into the macula, the part of the retina. The fluid makes the macula swell, which blurs vision.

Pathogenesis

Diabetic retinopathy :: microvascular retinal changes.

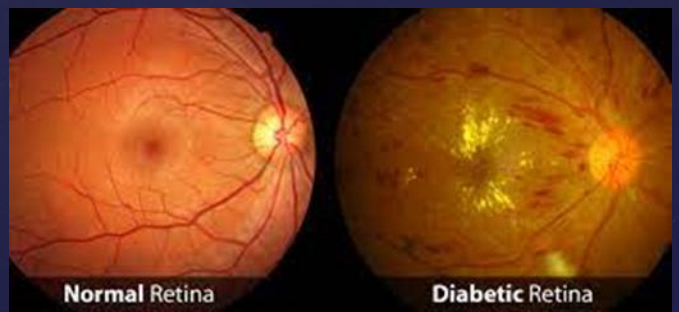
* These damages change the formation of the blood-retinal barrier and also make the retinal blood vessels become more permeable.

* The lack of oxygen in the retina causes fragile, new, blood vessels to grow along the retina and in the clear, gel-like vitreous humour that fills the inside of the eye.

* Without timely treatment, these new blood vessels can bleed, cloud vision, and destroy the retina.

*Fibrovascular proliferation can also cause retinal detachment.

* The new blood vessels can also grow into the angle of the anterior chamber of the eye and cause neovasculargl aucom



Macrovascular complications

Diabetes mellitus is a major risk factor for coronary artery disease,

cerebrovascular disease, and peripheral vascular disease.

* Multiple risk factors for vascular disease

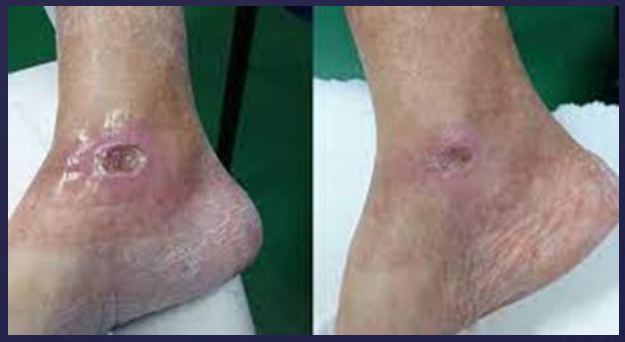
obesity, hypertension, hyperglycemia, hyperlipidemia, altered platelet function, and elevated fibrinogen levels.

* In people with type 2 diabetes, macrovascular disease may be present at the time of diagnosis.

* In type 1 diabetes, the attained age and the duration of diabetes appear to correlate with the degree of macrovascular disease.

Diabetic Foot Ulcer

* Distal symmetric neuropathy is a major risk factor for foot ulcers.
* Unaware of the constant trauma to the feet caused by poorly fitting shoes, improper weight bearing 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.



i 20.44 Clinical features of the diabetic foot

	Neuropathy	Ischaemia
Symptoms	None Paraesthesiae Pain Numbness	None Claudication Rest pain
Structural damage	Ulcer Sepsis Abscess Osteomyelitis Digital gangrene Charcot joint	Ulcer Sepsis Gangrene

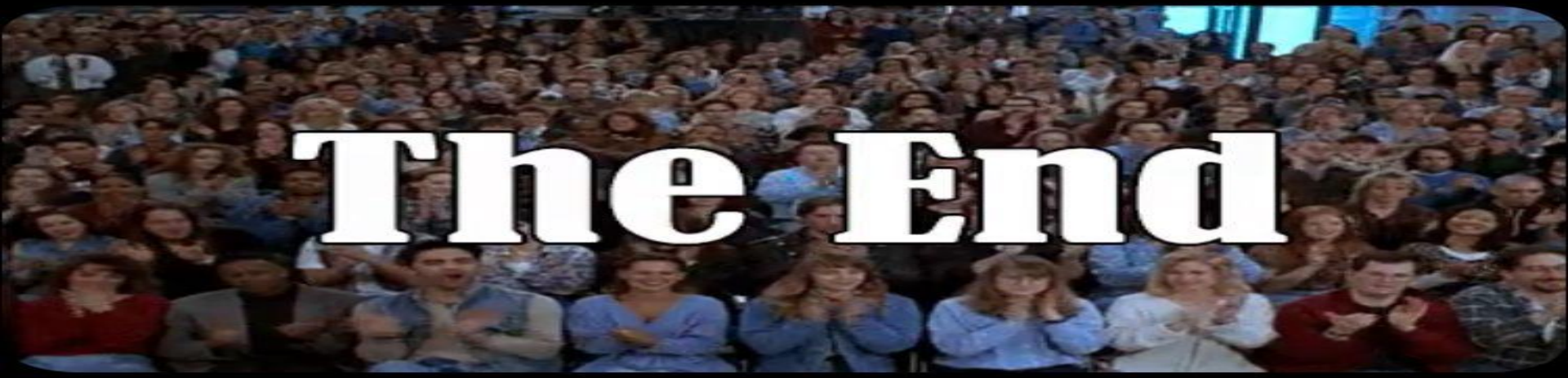
Infectios

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. Candidal 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.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