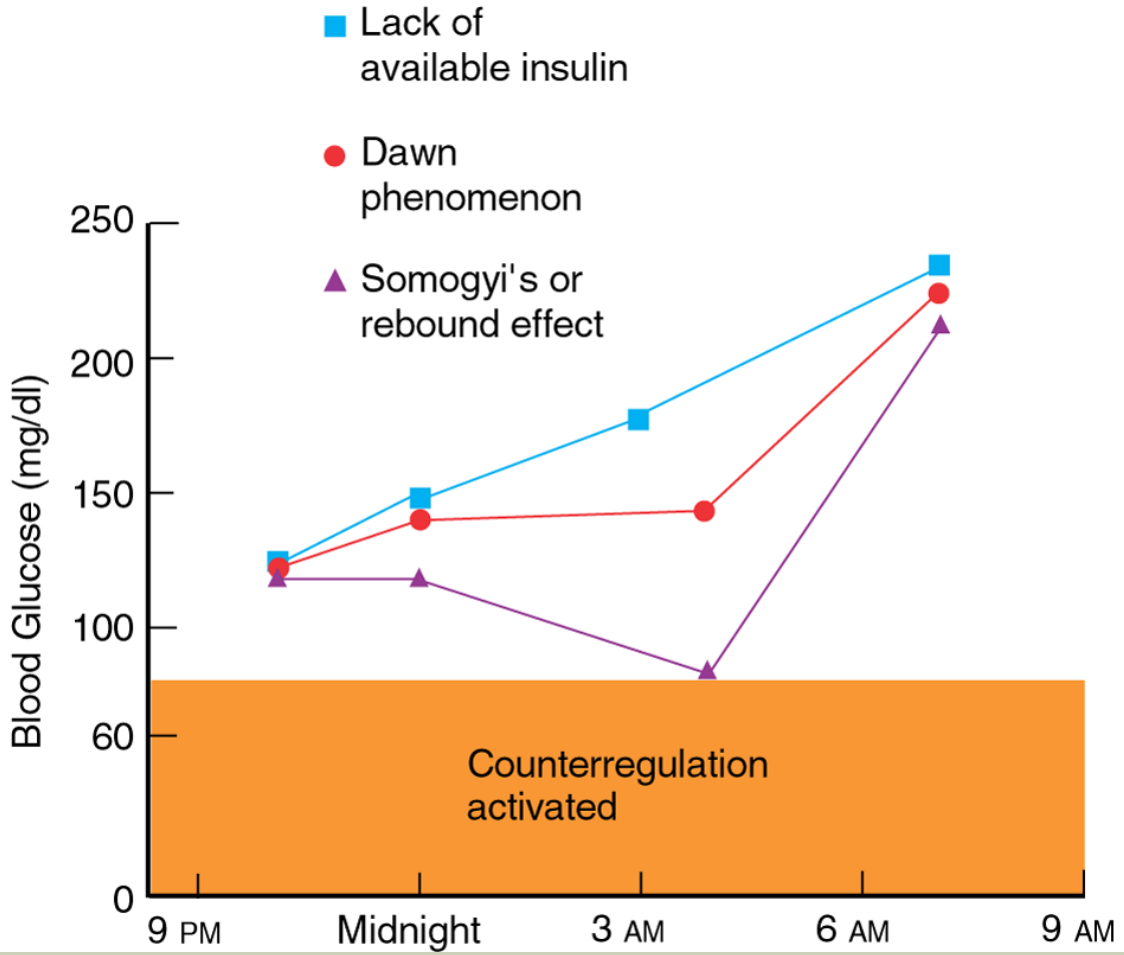
COMPLICATIONS OF DIABETES MELLITUS

- Alterations in blood sugars: hyperglycemia and hypoglycemia
- Macrocirculation (large blood vessels)
  - Atherosclerosis occurs more frequently, earlier in diabetics
  - Involves coronary, peripheral, and cerebral arteries
- Microcirculation (small blood vessels)
  - Affects basement membrane of small blood vessels and capillaries
  - Involves tissues affecting eyes and kidneys

HYPERGLYCEMIA

- high blood sugar
- DKA
- HHKS
- Dawn phenomenon:
  - rise in blood sugar between 4 am and 8 am
  - not associated with hypoglycemia (associated with Diabetes Type 1 and 2)
- Somogyi effect:
  - combination of hypoglycemia during night with a rebound morning hyperglycemia that may lead to insulin resistance for 12 to 48 hours

THREE BLOOD GLUCOSE PHENOMENA IN DIABETIC CLIENTS



ACUTE COMPLICATIONS

ACUTE COMPLICATIONS OF DM

- Hypoglycemia
- Diabetic Ketoacidosis
- Hyperglycemic, Hyperosmotic Non Ketotic Coma

HYPOGLYCEMIA

HYPOGLYCEMIA

- Serum glucose level < 55 mg/100ml
- brain damage develops when the brain is deprived of needed glucose after a dramatic drop in blood sugar
- insulin reaction, insulin shock, “the lows”
- Mismatch between insulin dose, carbohydrate availability and exercise

- Causes:
  - Excess or overdose of insulin or OHA (oral hypoglycemic agents)
  - Skip meal or omitting a meal
  - Overexertion/ stress
  - Under-eating
  - Eating late
  - Unplanned exercise

HYPOGLYCEMIA: SIGNS & SYMPTOMS

- Mild
  - Diaphoresis
  - Pallor
  - Paresthesia
  - Palpitations
  - Tremors
  - Anxiety

- ANS/Adrenal Medulla

Note: Clients taking medications, such as beta-adrenergic blockers may not experience manifestations associated with autonomic nervous system

HYPOGLYCEMIA: SIGNS & SYMPTOMS

- Moderate:
  - Confusion/ disorientation
  - Behavioral Changes
- cold clammy extremities,
- yawning,
- tremors,
- blurred vision

- Severe
  - Seizures
  - Loss of Consciousness
  - Shallow respirations
  - **Severe hypoglycemia can result in death**

HYPOGLYCEMIA: DIAGNOSIS

- Signs & Symptoms
- SMBG
- FSBG or FSBS

MANAGEMENT: MILD

- Simple Sugars p.o (15 gm of rapid-acting sugar)
  - 8 oz fruit juice
  - 8 oz of skim milk
  - 3 glucose tablets
  - 3-4oz regular soft drink
  - 3-4 pieces hard candy (life savers)
  - 1 tbsp sugar
  - 5 ml pure honey


Raisins
• I.D. bracelet
• Sugar source (glucose tablets or raisins)
A.D.A.M.


Dex4
Fast Acting Glucose
Raises Low Blood Sugar Fast and Boosts Energy!
Sour Berry
60 Bits


GLUCOSE TABLETS
10 TABLETS
GLUCOSE TABLETS
10 TABLETS
GLUCOSE TABLETS
10 TABLETS
GLUCOSE TABLETS
10 TABLETS


LIFESAVERS
FIVE FLAVOR

RULES TO REMEMBER

- Do not add sugar to OJ
- Recheck FSBS/CBG q 15 min until WNL
- Avoid high fat → slows absorption of glucose
- Instruct: carry fast sugar
- If meal is >1 hr away, follow with a protein and complex carbohydrate
- NPO if "unconscious" or confused

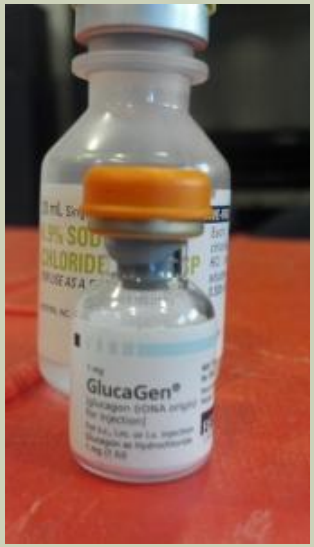
- **15/15 rule**: wait 15 minutes and monitor blood glucose; if still low (BG<80), client should eat another 15 gm of sugar
- Continue until blood glucose level has returned to normal
- Client should contact medical care provider if hypoglycemia occurs more than 2 or 3 times per week

PROTEIN SOURCES

- 1 Tbsp peanut butter
- 1 oz cheese
- 1 oz meat

HYPOGLYCEMIA TREATMENT UNCONSCIOUS

- Glucagon 1 mg Subq, IM, IV; follow with oral or intravenous carbohydrate
  - Action: (hormone) → raises BS levels
  - Onset:10 minutes
  - Duration 25 minutes
  - S/E: N/V
- Position: side lying



HYPOGLYCEMIA TREATMENT UNCONSCIOUS

- Or give 25mL of D50 as IV push
- followed by infusion of 5% dextrose in water

HYPOGLYCEMIA GERONTOLOGICAL CONSIDERATION

- Cognitive deficits →
  - not recognize S&S
- Decreased renal function →
  - oral hypoglycemic meds stay in body longer
- More likely to _____a meal
  - Skip
- Vision problems →
  - inaccurate insulin draws

HYPOGLYCEMIA
NURSING MEASURES

- Follow protocol
- Teach
  - Carry simple sugar at all times
  - S&S or hypoglycemia
  - How to prevent Hypoglycemia
  - Check FSBS if you suspect → NOW!

HYPOGLYCEMIA
NURSING MEASURES

- Encourage to wear ID bracelet
- Teach family that belligerence is sign of hypoglycemia

DIABETIC KETOACIDOSIS (DKA)

DKA

- Serious complication of hyperglycemia due to lack of insulin
- Usually occurs with type I DM(> 250mg/dl)

DKA

- Results from breakdown of fat and overproduction of ketones by the liver and loss of bicarbonate

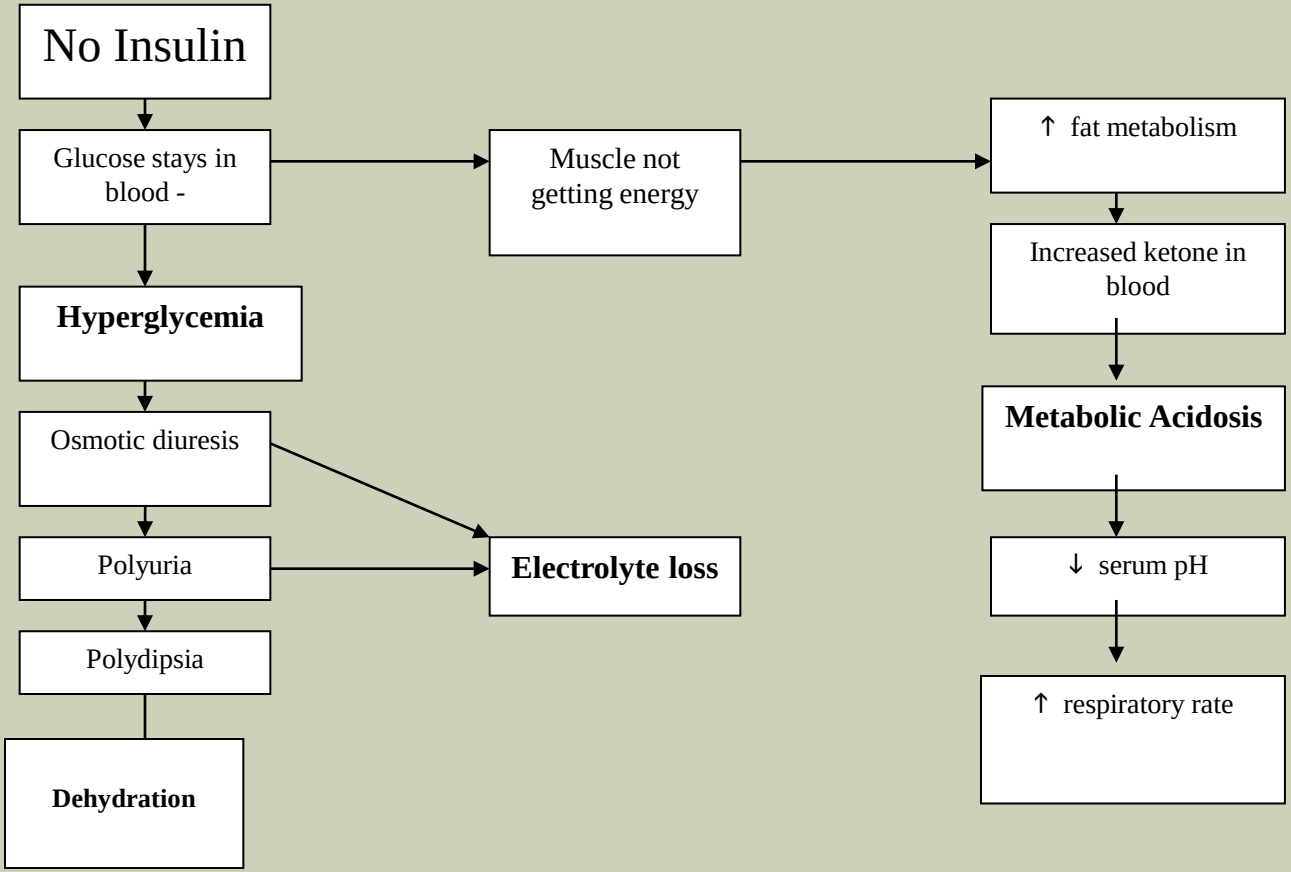
DKA: ETIOLOGY

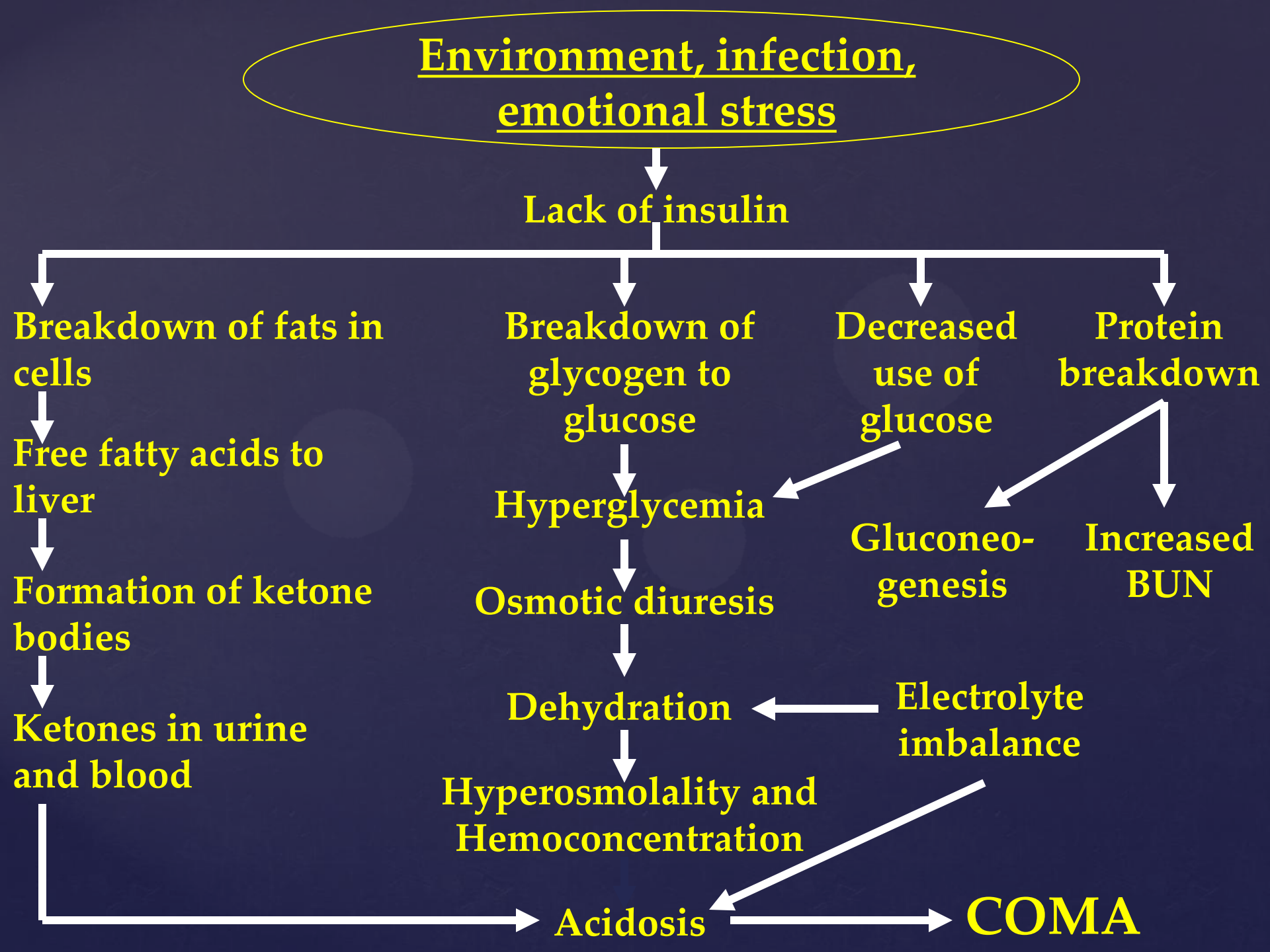
- #1 cause: illness, infection, stress (physical or emotional stress)
- surgery, trauma, pregnancy
- absence or inadequate insulin
  - taking too little insulin
  - omitting doses of insulin
- known diabetic has increased energy needs,
- Initial or undiagnosed diabetes
- developing insulin resistance

DKA: 4 MAIN CLINICAL FEATURES

1. Hyperglycemia
2. Dehydration
3. Electrolyte loss
4. Metabolic Acidosis

PATHOPHYSIOLOGY DKA




Environment, infection, emotional stress
Lack of insulin
Breakdown of fats in cells
Free fatty acids to liver
Formation of ketone bodies
Ketones in urine and blood
Breakdown of glycogen to glucose
Hyperglycemia
Osmotic diuresis
Dehydration
Hyperosmolality and Hemoconcentration
Decreased use of glucose
Protein breakdown
Gluconeo-genesis
Increased BUN
Electrolyte imbalance
Acidosis
COMA

S&S OF DKA

- Hyperglycemia
  - ↑ blood glucose
  - Tired
  - Polyphagia
  - Decreased attention, confusion
  - N/V, abdominal pain
  - Blurred vision

S&S OF DKA

- Dehydration
  - Polydipsia
  - Polyuria
  - Dry/flushed skin
  - Orthostatic hypotension
  - Tachycardia
  - Headaches
  - Decreased Na+ and K+ levels

S&S OF DKA

- Acidosis
  - ↑ Resp. rate → Kussmaul's
  - Fruity breath, acetone breath
  - Serum pH
    - Decreased
      - Normal Serum pH 7.35 – 7.45
      - ↓ pH = acidic / acidosis
      - ↑ pH = alkaline/ alkalosis

DKA: DIAGNOSIS

- Blood
  - Serum Osmolality
    - ↑
    - thick
  - Ketones
    - +
  - bicarbonate
    - ↓
    - less than 15 mEq/L
- pH
  - ↓
  - less than 7.3
- sugar levels
  - Elevated
  - greater than 250 mg/dL
- BUN Blood Urea Nitrate
- increased = dehydration

DKA: DIAGNOSIS

- Urine
  - Ketones
    - +
  - Glucose
    - +
  - Specific gravity of urine
    - ↓

DKA: DIAGNOSIS

- Hemoglobin
  - Normal
    - Female : 12-16 g/dL
    - Male: 14-18 g/dL
  - Elevated
    - Dehydration
    - COPD
  - Decreased
    - Anemia, hemorrhaging, over-hydration

DKA: DIAGNOSIS

- Hematocrit
  - Normal
    - Female: 37-47%
    - Male 42-52%
  - Elevated
    - Dehydration & COPD
  - Decreased
    - Anemia, leukemia

DKA: DIAGNOSIS

- Serum Potassium levels
  - Normal levels
    - 3.5-5.5 mEq/L
    - Increased K^+ levels = Hyperkalemia
    - Decreased K^+ levels = Hypokalemia
  - Purpose of K^+
    - Skeletal & cardiac muscle activity
  - DKA → decreased K^+ levels

HYPOKALEMIA S&S

- Fatigue
- Anorexia N/V
- Muscle weakness
- Leg cramps
- Dysrhythmias
- ↑ sensitivity to digitalis

MANAGEMENT OF DKA

- Focus on the four main clinical features
  - Hyperglycemia
  - Dehydration
  - Electrolyte loss
  - Acidosis

MANAGEMENT OF DKA

- Hyperglycemia
  - Give insulin → IV

MANAGEMENT OF DKA

- Dehydration
  - Rehydrate
    - IV, push fluids
    - I&O
    - Check vital signs
    - Check Lung sounds
    - Monitor lab values

MANAGEMENT OF DKA

- Electrolyte loss
  - Polyuria → loss of K^+
  - Treatment of DKA dehydration → drop in K+

5 K / 1 ml serum

5.0 mEq/L

K K K K K

5 K / 2 ml serum

2.5 mEq/L

K K K K K

MANAGEMENT OF DKA

- Electrolyte loss
  - Replace K+
  - Monitor lab values closely

MANAGEMENT OF DKA

- Acidosis
  - Reversed with insulin
    - Insulin →
    - glucose enters muscles →
    - ↓ fat metabolism →
    - ↓ in Ketones →
    - acidosis reversed

PREVENTION OF DKA

- #1 cause of DKA?
  - Illness
- Sick Day Rules

SICK DAY PROTOCOL/RULES

- Never omit insulin
- If you are unable to eat normally, DO NOT stop taking insulin
- Sliding scale
- Test blood sugar every 3-4 hours
- Test urine for ketones every 3-4 hours
- Take liquid/fluids q hour

SICK DAY PROTOCOL/RULES

- If you can not eat your usual meal, substitute soft foods
- Have “sick day” food in house
- If vomiting, diarrhea or fever persists, take liquids q half hour
- If miss or replace 4 meals with fluids, call MD

SICK DAY PROTOCOL/RULES

- Go to bed and keep warm
- Friends: good to have someone around who understands and knows about insulin reactions and diabetes

HYPERGLYCEMIA HYPEROSMOLAR NONKETONIC SYNDROME (HHNK)

HHNK

- occurs when there is insufficient insulin to prevent hyperglycemia, but there is enough insulin to prevent Ketoacidosis
- Occurs in all types of diabetes
  - Esp Diabetes Type 2

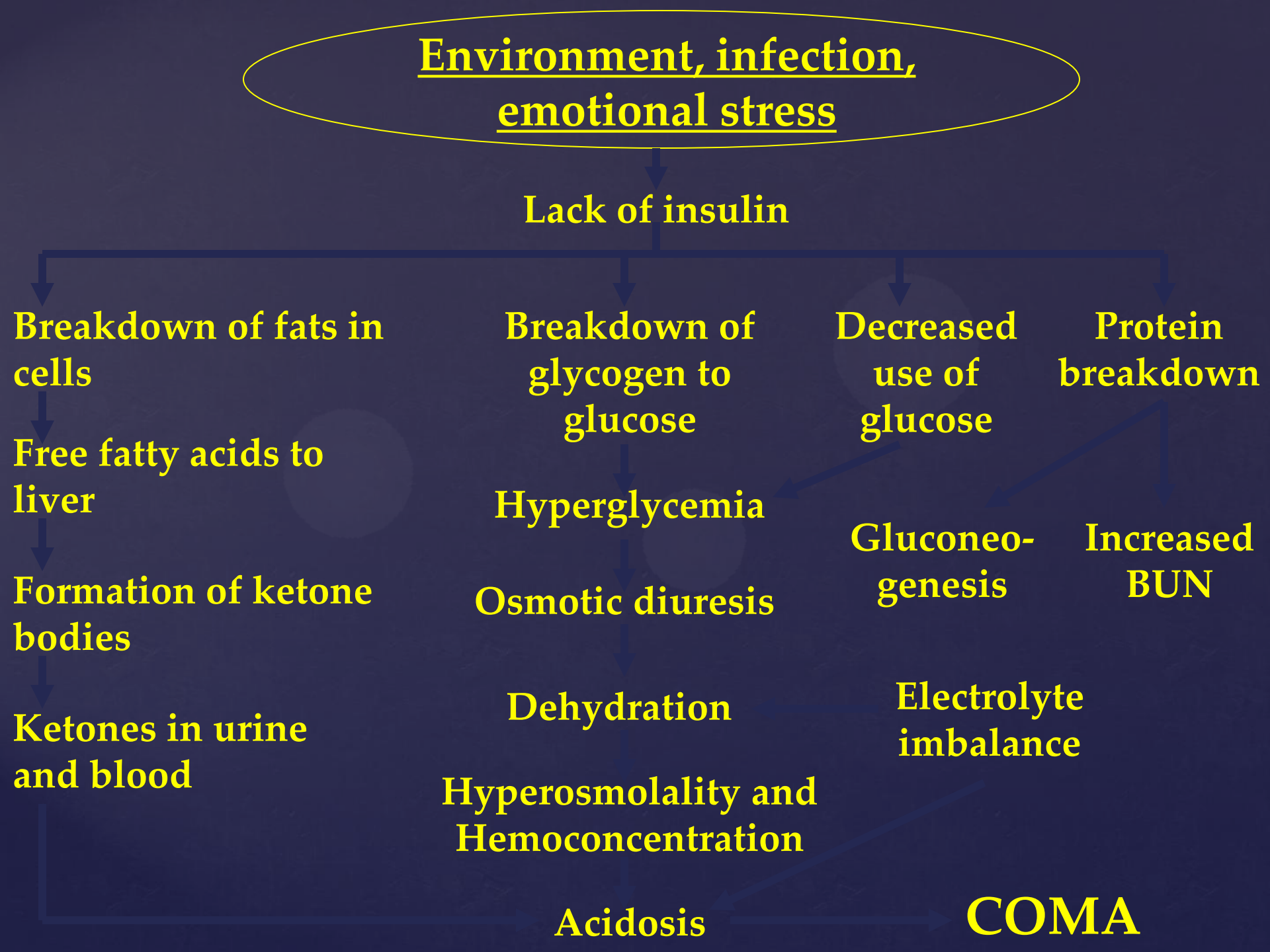
- Life threatening medical emergency, high mortality rate
- Characterized by
  - Plasma osmolarity 340 mOsm/L or greater (normal: 280 -300)
  - Blood glucose severely elevated, 600 - 1000 or 2000 (normal 70-110)
  - Altered level of consciousness

HHNKS

- extreme hyperglycemia (800-2000mg/dl)
- undetectable ketonuria
- absence of acidosis
- major difference from diabetic ketoacidosis is the lack of ketonuria because there is some residual ability to secrete insulin in NIDDM.

PRECIPITATING FACTORS

- Infection (most common)
- Therapeutic agent or procedure
- Acute or chronic illness
- Overeating
- Stress
- Too little insulin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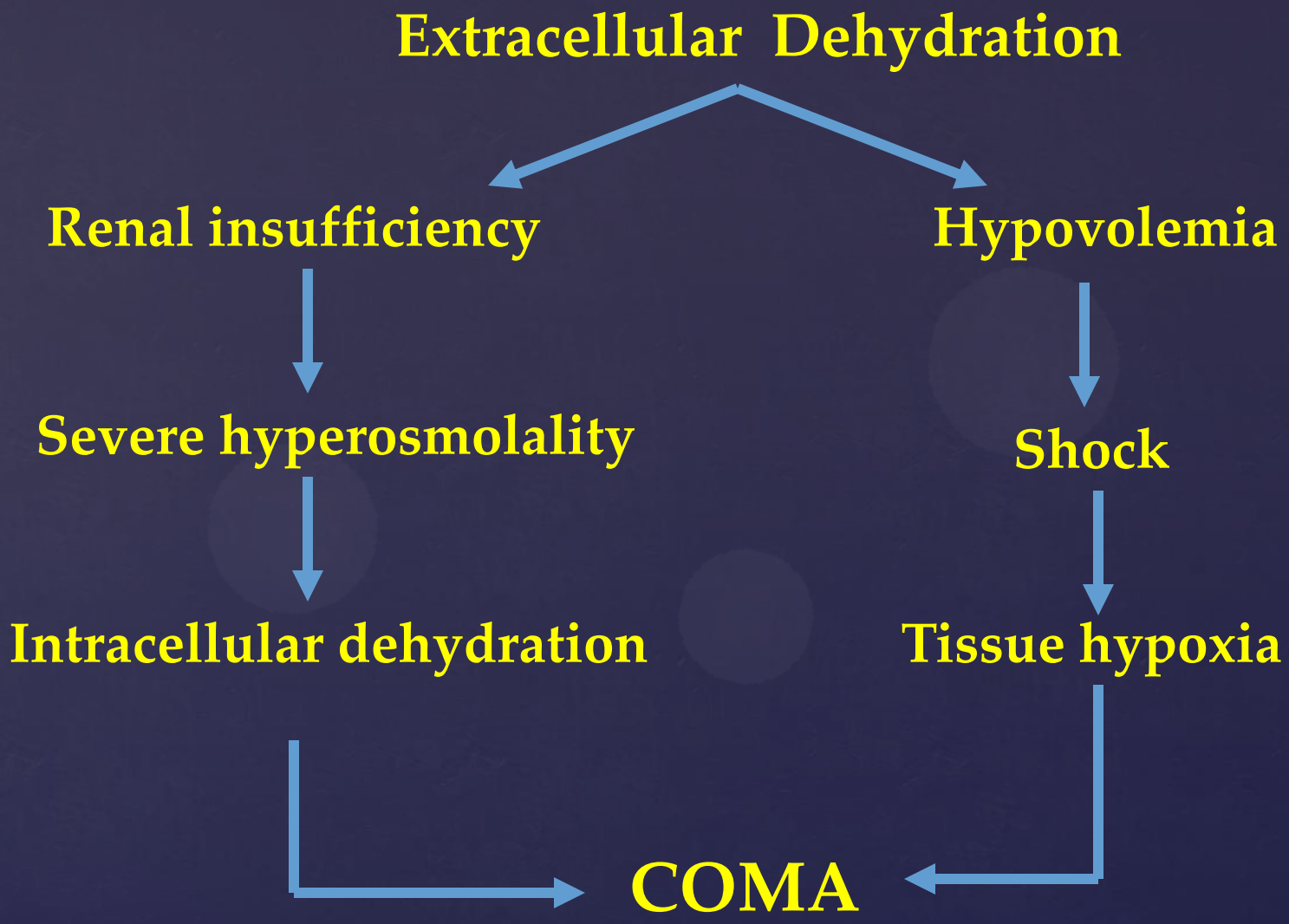
Increased BUN
Electrolyte imbalance
COMA


Extracellular Dehydration
Renal insufficiency
Hypovolemia
Severe hyperosmolality
Shock
Intracellular dehydration
Tissue hypoxia
COMA

COMA

S&S OF HHNK SYNDROME

- Altered level of consciousness (lethargy to coma)
- Neurological deficits: hyperthermia, motor and sensory impairment, seizures
- Dehydration: dry skin and mucous membranes, extreme thirst

MANAGEMENT

- Usually admitted to intensive care unit of hospital for care since client is in life-threatening condition: unresponsive, may be on ventilator, has nasogastric suction
- vigorous fluid replacement
- give insulin IV
  - Lower glucose with regular insulin until glucose level drops to 250 mg/dL
- potassium, sodium chloride given IV
- dextrose is given when blood sugar reaches around 250mg/100ml to prevent hypoglycemia
- Treat precipitating factors

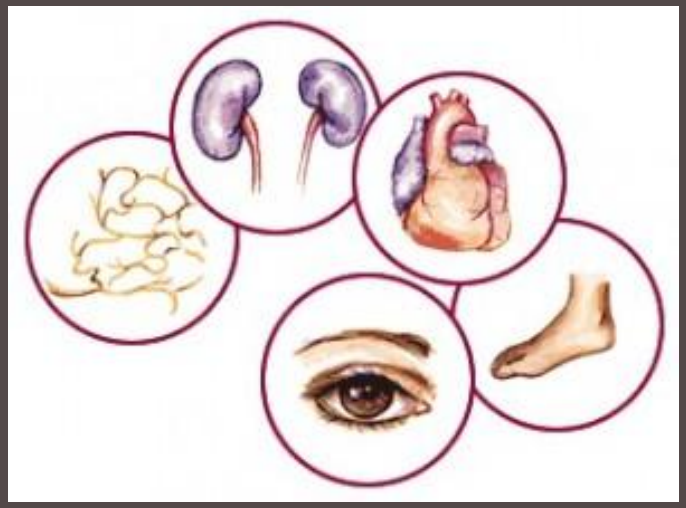
NURSING RESPONSIBILITY

- Same as with DKA
  - Insulin
  - Hydration
  - Electrolyte replacement and monitoring
  - Treat underlying cause

SUMMARY

- Acute complications of DM
  - Hypoglycemia
  - Diabetic Ketoacidosis
  - Hyperglycemia Hyperosmolar Non-ketonic Syndrome

CHRONIC COMPLICATIONS



COMPLICATIONS

Macrovascular complications

- Arteriosclerosis
  - Characterized by thickening and loss of elasticity of the arterial walls "hardening of the arteries".
- Coronary Artery Disease
- Cerebrovascular Disease
- Peripheral vascular disease

Microvascular complications

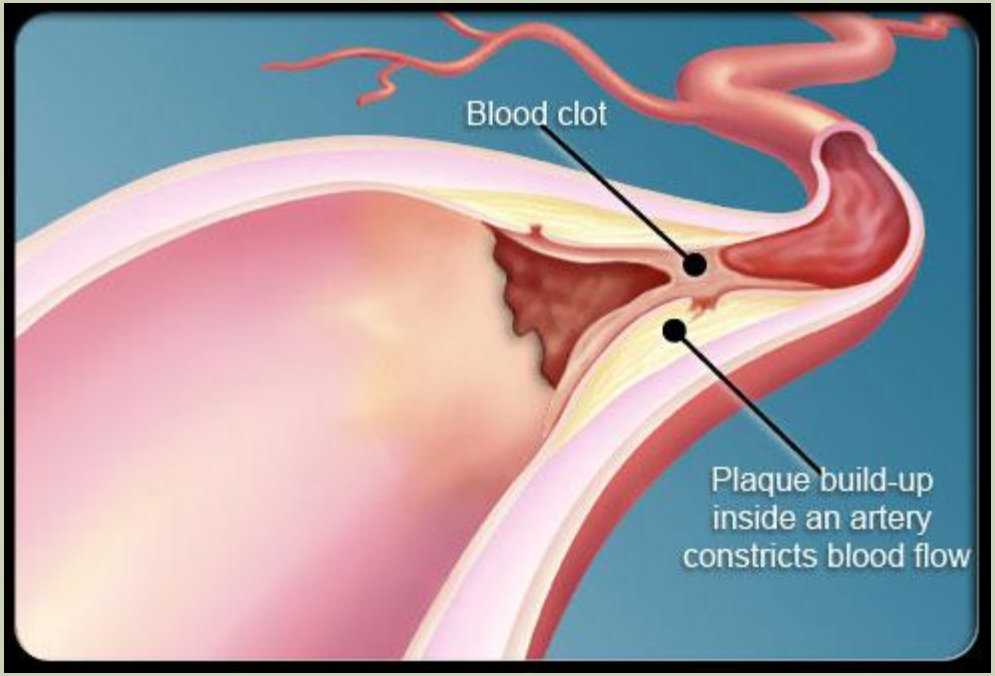
- Characterized by basement membrane thickening
- Effects smallest blood vessels
- Due to hyperglycemia

CARDIOVASCULAR DISEASE

- major source of mortality in patients with type 2 DM.
- Approximately two thirds of people with diabetes die of heart disease or stroke.
- Men with diabetes face a 2-fold increased risk for CHD, and women have a 3- to 4-fold increased risk
- Diabetics more likely to develop MI, Congestive Heart Failure

ATHEROSCLEROSIS

- About two out of three people with diabetes die of heart disease
- two to four times higher risk for stroke.

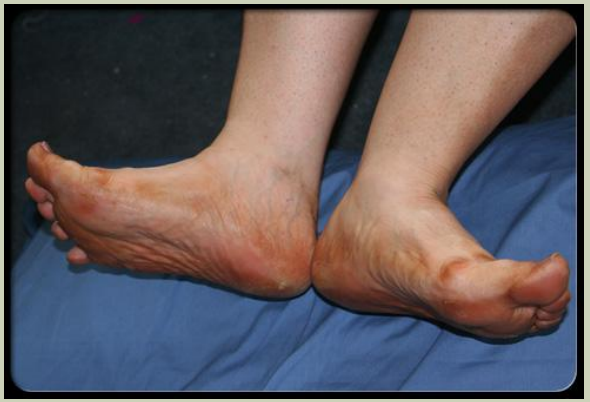


HYPERTENSION

- Affects 20 – 60 % of all diabetics
- Increases risk for retinopathy, nephropathy

PERIPHERAL VASCULAR DISEASE

- Increased risk for Types 1 and 2 diabetics
- Development of arterial occlusion and thrombosis resulting in gangrene
- Gangrene from diabetes most common cause of non-traumatic lower limb amputation



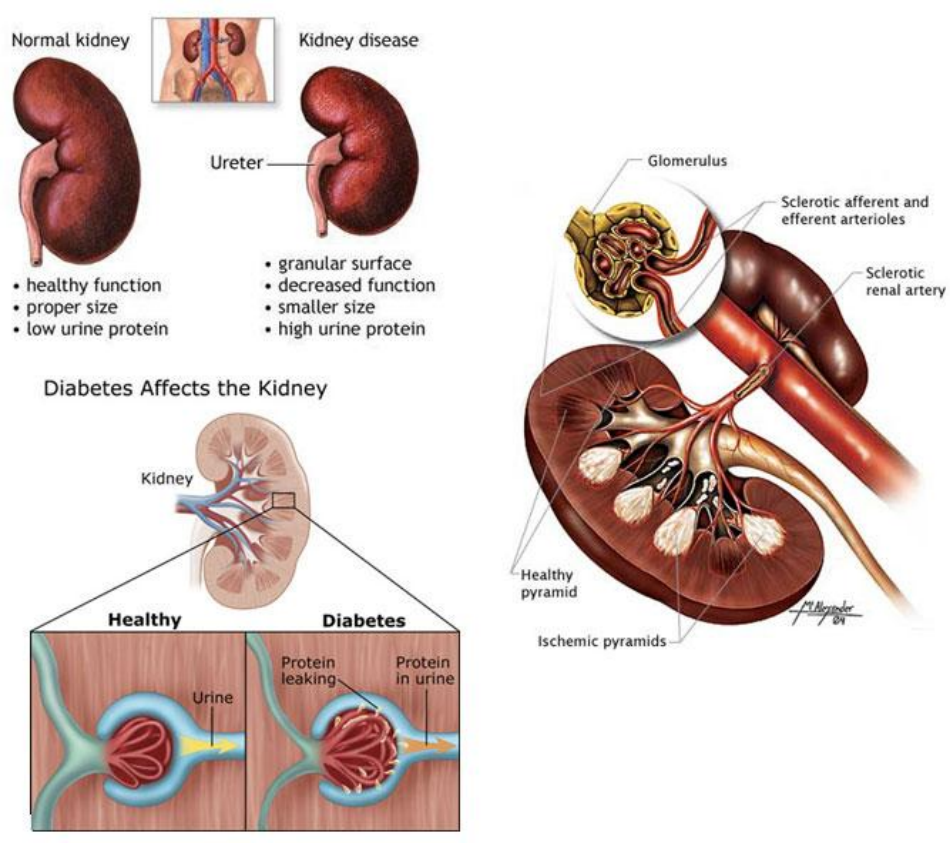
DIABETIC NEPHROPATHY

DIABETIC NEPHROPATHY

- Diabetes is the leading cause of kidney failure, accounting for 44% of new cases in 2008.
- Most common cause of end-stage renal failure in U.S.

DIABETIC NEPHROPATHY

- glomerular changes in kidneys of diabetics leading to impaired renal function



DIABETIC NEPHROPATHY

- Aka: Kimmelstiel-Wilson syndrome
  - is a kidney condition associated with long-standing diabetes
  - glomerulosclerosis associated with diabetes
- First indicator: microalbuminuria

DIABETIC NEPHROPATHY

- Diabetics without treatment go on to develop hypertension, edema, progressive renal insufficiency
  - In type 1 diabetics, 10 – 15 years
  - May occur soon after diagnosis with type 2 diabetes since many are undiagnosed for years

- Damage to the tiny blood vessels within the kidney.
- Due to
  - Hyperglycemia
- ↑ glucose levels
  - Stress kidney's filtration mechanism
- Blood protein leaks into urine
- Pressure in blood vessel of kidney ↑
- Kidney failure

NEPHROPATHY: PATHOPHYSIOLOGY

Normally

- Kidneys filter blood
- Small molecules & waste squeeze through kidneys → urine
- Big stuff (I.e. protein, RBC), stay in blood where they belong

NEPHROPATHY: PATHOPHYSIOLOGY

- Diabetes damages the system
- Filters start to leak
- Protein and RBC lost in urine
  - Microalbuminuria
  - Macroalbuminuria
  - Proteinuria
- Filters collapse
- Lose of filtering ability
  - Kidney failure
  - ESRF / ENRD
- Waste products build up in blood

S&S / DX

- Proteinuria / albuminuria
- ↓ urine output
- Edema
- BUN & Creatinine ↑
- ↑ BP

NEPHROPATHY: MANAGEMENT

- Tight glucose control
- Anti-hypertensives
  - Calcium-channel blockers
  - Alpha blockers
  - ACE inhibitor
- Dialysis
- Transplant

PREVENTION

- Control BG
- Control HTN
- Tx UTI
- No nephrotoxic substances
- ↓ Na
- ↓ Protein

DIABETIC RETINOPATHY

DIABETIC RETINOPATHY

- Retinal changes related to diabetes
- DM is the major cause of blindness in adults aged 20-74 years in US
- accounts for 12,000-24,000 newly blind persons every year.
- Affects almost all Type 1 diabetics after 20 years
- Affects 60 % of Type 2 diabetics

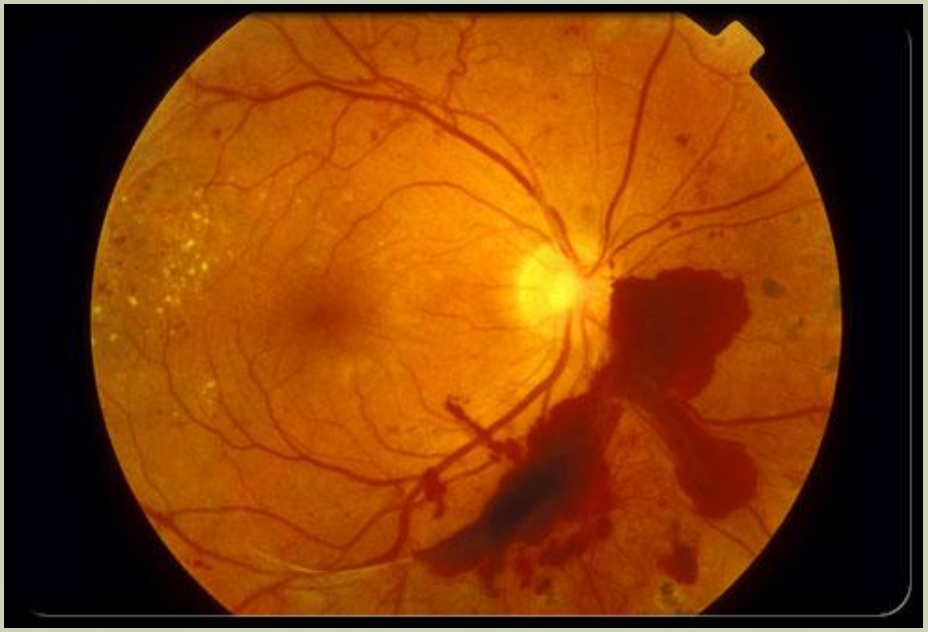
Diabetics also have increased risk for cataract development

DIABETIC RETINOPATHY

- due to hyperglycemia
- Damage to the tiny blood vessels that supply the eye →
- Small hemorrhages occur
- Leads to retinal ischemia and breakdown of blood-retinal barrier

DIABETIC RETINOPATHY

- progressive, irreversible vision loss.
- Pools of blood, or hemorrhages, on the retina of an eye are visible in this image.

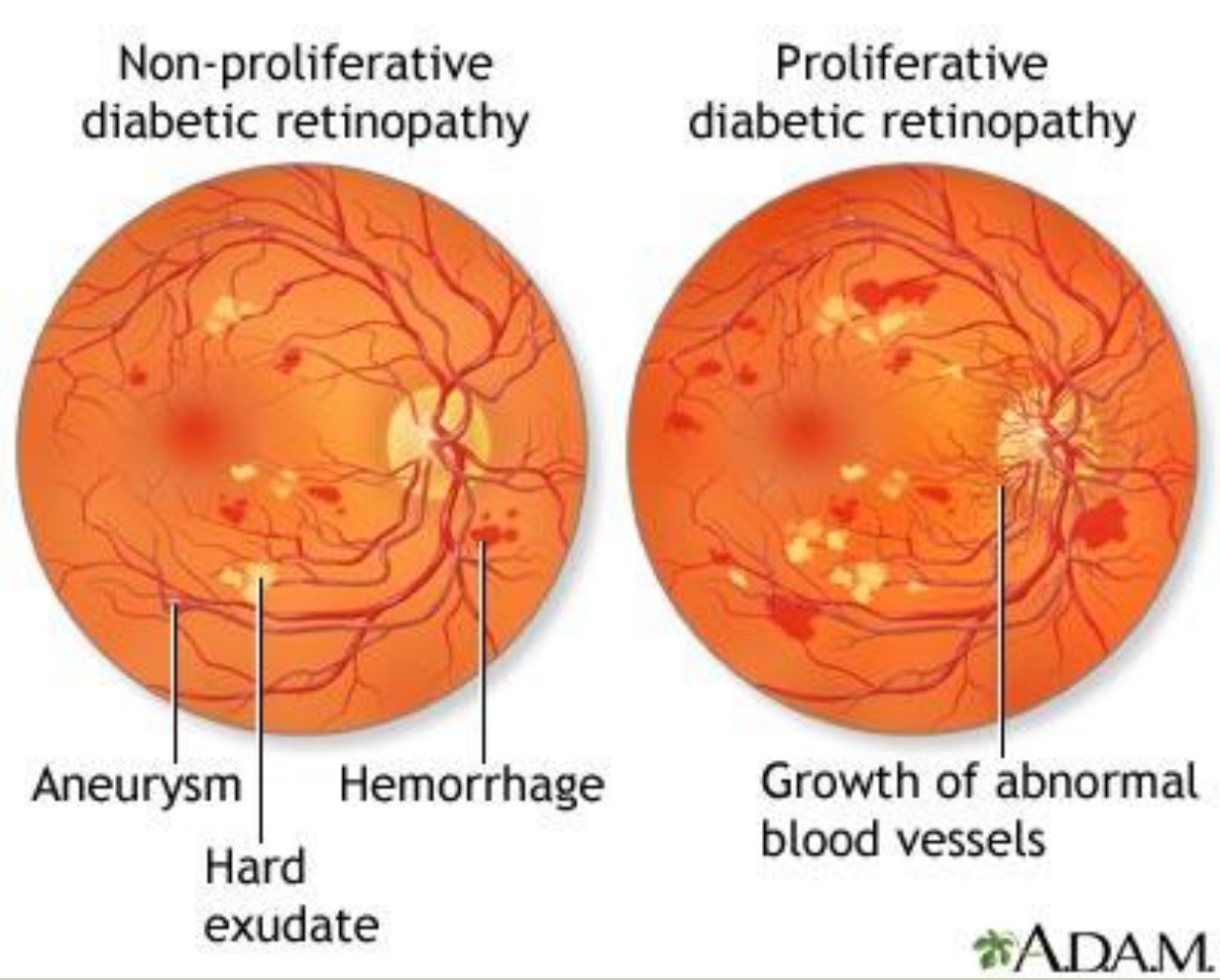


PREVENTION

- Control
  - Glucose
  - BP
- No straining
- Use laxatives
- Avoid lowering head
- Avoid lifting above shoulders

- Diabetics should be screened for retinopathy

DIABETIC RETINOPATHY



RETINOPATHY

Medical Management

- Photocoagulation "laser" treatment
- Control hypertension
- Control blood glucose
- No smoking

Nursing Considerations

- Expected
- Odds are good
- Frequent eye exams
- Bilateral but uneven

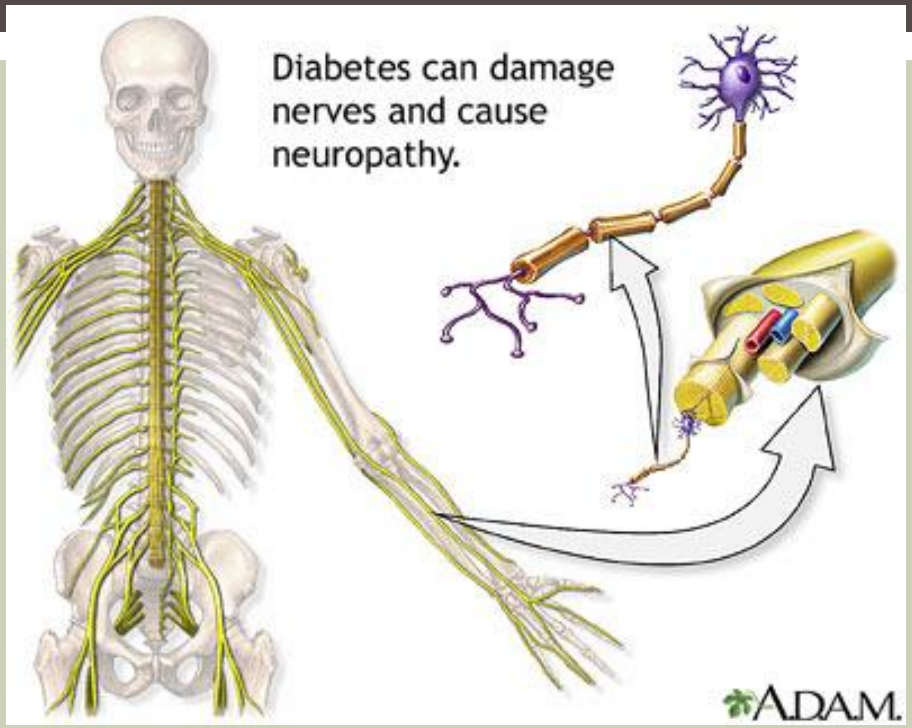
OTHER OPTIC COMPLICATIONS

- Cataracts
- Lens Changes
- Extraocular muscle palsy
- Glaucoma

DIABETIC NEUROPATHY

- Damage to the Nerves due to hyperglycemia
- Most common complication
- Various Types of Neuropathies…
  - Sensory-Motor Polyneuropathy
  - Autonomic neuropathy

DIABETIC NEUROPATHY



SENSORY-MOTOR POLYNEUROPATHY

s/s

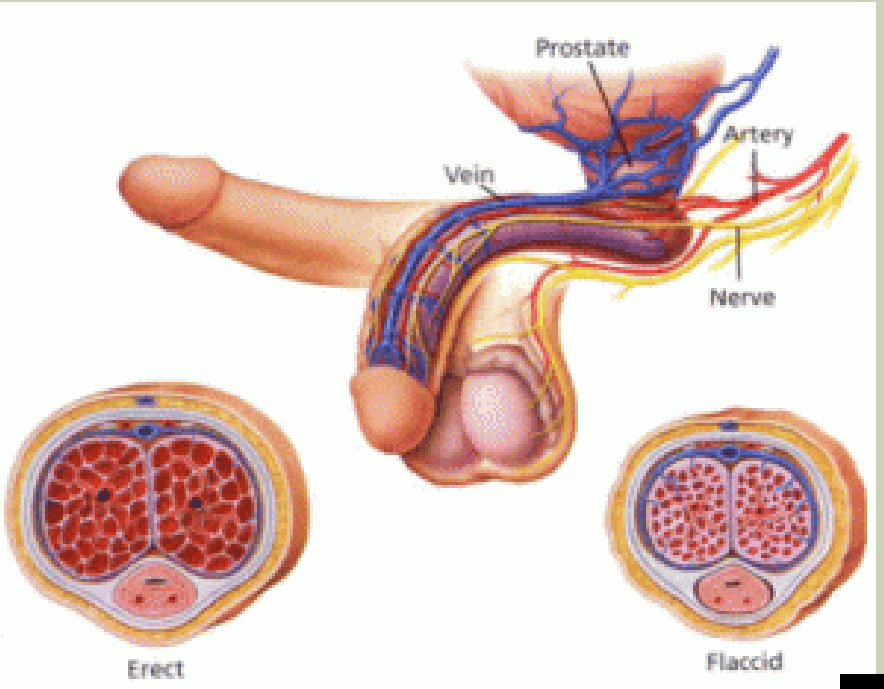
- AKA peripheral neuropathy
- Paresthesias: primarily lower extremities
- ↓ deep tendon reflexes
- Numb feet
- ↓ proprioception
- ↓ sensation
- Unsteady gait
- ↑ risk foot injury

AUTONOMIC NEUROPATHY

- Autonomic NS
- Can affect almost any system
  - Cardiovascular
    - Tachycardia
    - Orthostatic hypotension
    - MI
  - Gastro-intestinal
    - Delayed gastric emptying
    - Constipation
    - Diarrhea
  - Urinary
    - Retention
    - Neurogenic bladder
  - Reproductive
    - Male impotence
  - Adrenal Gland
    - "Hypoglycemic Unawareness"
    - Adrenal Medulla
    - Adrenergic symptoms
    - No longer feel S&S
    - Strict BG control & frequent monitoring
  - Sudomotor neuropathy
    - No sweating
    - Anhidrosis
    - → dry feet
    - → foot ulcers

MANAGEMENT

- Control serum glucose levels
- Pain control
  - Analgesics (non-narcotic)
  - Tri-cyclic antidepressants
  - Anticonvulsants


Prostate
Artery
Vein
Nerve
Erect
Flaccid

OTHER COMPLICATIONS FROM DIABETES

DIABETES MELLITUS

- Increased susceptibility to infection
  - Predisposition is combined effect of other complications
  - Normal inflammatory response is diminished
  - Slower than normal healing
- Periodontal disease
- Foot ulcers and infections: predisposition is combined effect of other complications

INFECTIONS

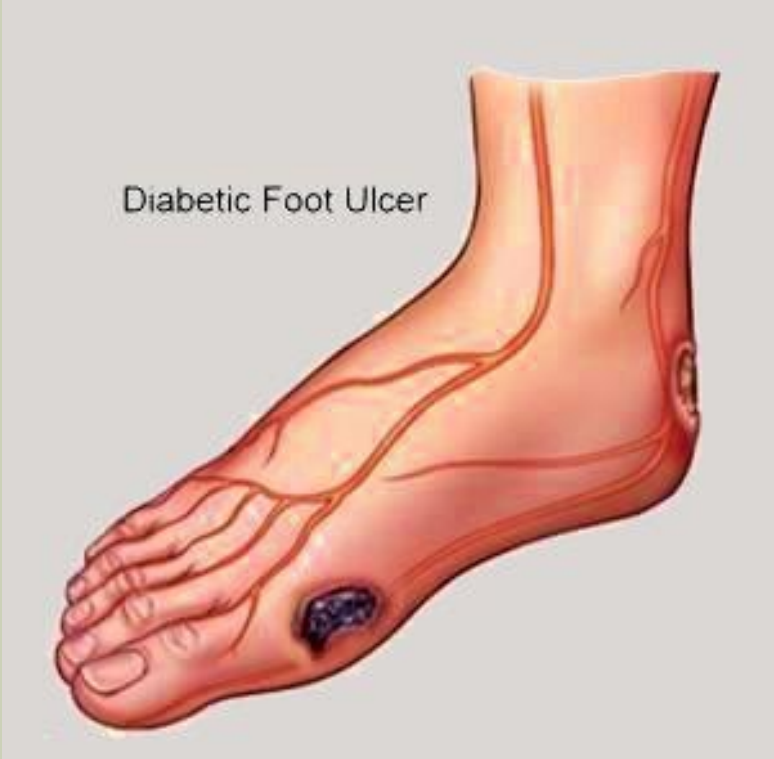
- High risk of foot infections
  - Neuropathy
    - Pain sensation
      - ↓
    - Pressure sensation
      - ↓
    - Dryness
      - ↑
    - Fissures
      - ↑
- Peripheral vascular disease
  - Circulation
    - ↓
  - WBC
    - ↓
  - Oxygen
    - ↓
  - wound healing
    - Poor
  - Antibiotics
    - ↓
  - Gangrene

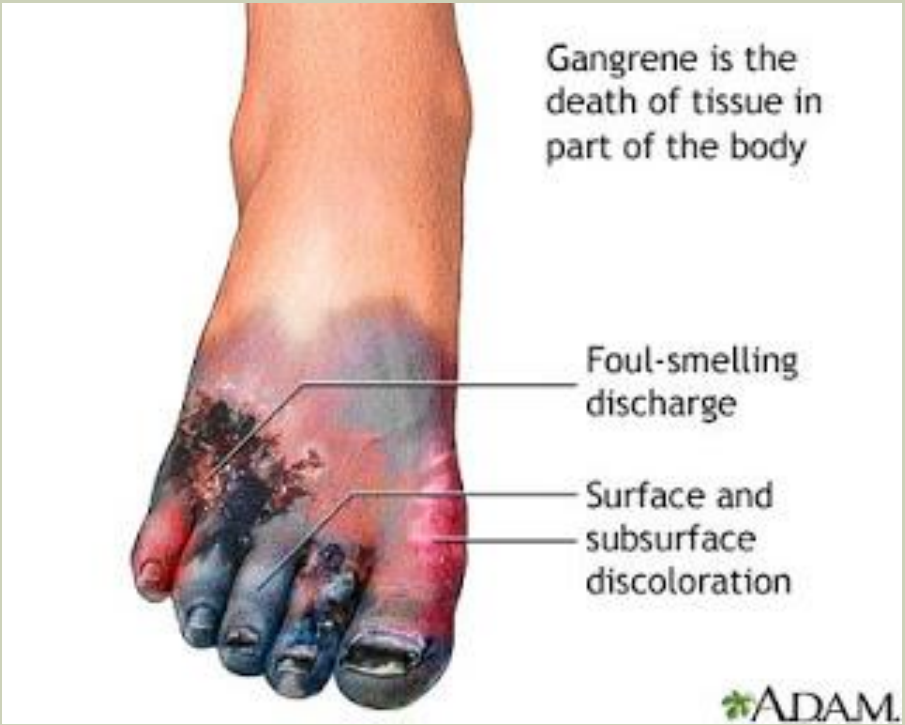
- Immuno-compromised
  - WBC + hyperglycemia = sluggish WBC's
- Once they occur → difficult to treat
  - Poor circulation
  - Antibiotic not get there
  - Sluggish WBC's
  - Unknown wounds
- Particular concern
  - Foot infections/wounds

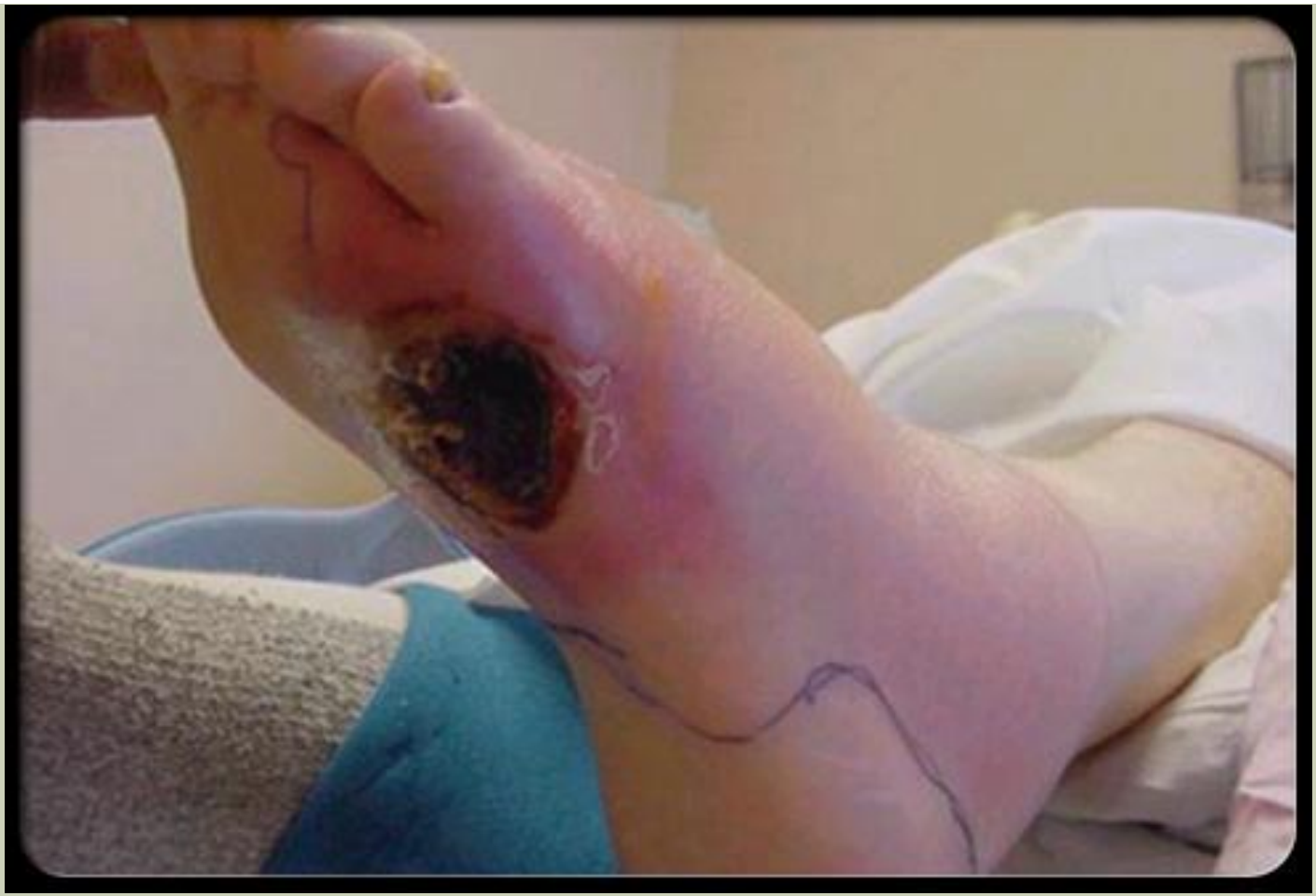
HIGH RISK FOR FOOT INFECTIONS

- Duration of diabetes
- ↑ Age
- Smoking
- ↓ Peripheral pulses
- ↓ Sensation
- Deformities/pressure areas
- Hx of foot ulcers

- Progression of events
  - Soft tissue injury →
  - Injury not sensed →
  - Infection →
  - Drainage, swelling, redness →
  - Gangrene →


Diabetic Foot Ulcer


Gangrene is the death of tissue in part of the body
Foul-smelling discharge
Surface and subsurface discoloration
A.D.A.M.



Boils:

- AKA: "furuncles"
- round, pus-filled bumps on the skin
- D/T: Staphylococcus aureus bacteria

Cellulites

- noncontagious inflammation of the connective tissue of the skin,
- D/T bacterial infection
- Treatment
  - Antibiotics
  - Analgesics

INFECTIONS OF CONCERN

- UTI's
- Yeast Infections
- Periodontal disease

Gangrene

- term to describe the decay or death of an organ or tissue
- d/t ↓ blood supply.

MANAGEMENT OF INFECTIONS

- Bed rest
- Antibiotics
  - Topic vs. IV
- Debridement
- Control Glucose levels
- ? Amputation
- Teach foot care
  - prevention
- Teach wound care

CANCER

- people with type 2 diabetes are at an increased risk for many types of cancer
  - 2010 Consensus Report from a panel of experts chosen jointly by the American Diabetes Association and the American Cancer Society

PREVENTION OF COMPLICATIONS

1. Managing diabetes
2. Lowering risk factors for conditions
3. Routine screening for complications
4. Implementing early treatment