Objectives:

• Basics about diabetic late complication.
• Diabetic Nephropathy & management.
• Diabetic Retinopathy & management.
• Diabetic foot.
Basic pathophysiology – Theory 1:

1. Increase Glucose level. 
   Increase formation of glu-derived precursors (glyoxal, methylglyoxal, 3-deoxyglucosone)

2. Non-enzymetic reaction with amino acids of intracellular & extracellular proteins. 
   e.g. (hemoglobin, collagen, LDL & tubulin of Peripheral nerves)

3. Result in formation of Advanced glycation end products (AGEs) 
   The natural rate of AGEs formation enhanced by the hyperglycemia.
Diabetic nephropathy:

• Types of damages:
  A. Glomerular damage \(\rightarrow\) Glomerulosclerosis.
  B. Ischemia resulting from hypertrophy of afferent or efferent arteries.
  C. Pyelonephritis.
  D. Nephron hypertrophy \(\rightarrow\) as result of high glucose reabsorption.

• Major points in the management:
  A. High BP aggravates the kidney damages \(\rightarrow\) should be controlled.
  B. Glomerulosclerosis \(\rightarrow\) filtration membrane damage \(\rightarrow\) proteinuria \(\rightarrow\) edema, hypotrienemia \(\rightarrow\) must be controlled.
  C. Hyperglycemia is the key player \(\rightarrow\) glycemic control.
  D. Infection???
Diabetic foot:

- 60 – 70% have neuropathy only.
- 15 – 20% have vascular problem only.
- 15 – 20% have combined problems.

- Sensation of lower limb is lost \(\rightarrow\) unawareness of any object, injury or abnormal high pressure on the foot.

- If it severe enough \(\rightarrow\) ulceration \(\rightarrow\) infection \(\rightarrow\) amputation.
4- Wound environment:

- Dressings are used to absorb or remove exudate, maintain moisture & protect from contaminating agents.

- Expensive new dressings containing growth factors & biological active agents → future.