LAB 8: THE URINARY SYSTEM

The focus of this week’s lab will be pathology of the urinary system. Diseases of the kidney can be broken down into diseases that affect the glomeruli, tubules, interstitium, and blood vessels. The glomerulus is particularly susceptible to injury and glomerular diseases are often immunologically mediated; interstitial and tubular dysfunction is more likely to be caused by toxic or infectious agents. The structures of the kidney are connected and consequently damage to one component usually adversely affects other structures of the urinary system resulting in chronic renal failure and what is called end-stage kidney disease.

Azotemia is the elevation of blood urea nitrogen and creatinine levels largely related to a decrease in GFR. Azotemia can progress to clinical manifestations and systemic biochemical abnormalities and is then termed uremia. Uremia involves the failure of renal excretory function as well as subsequent metabolic and endocrine alteration of function and secondary gastrointestinal, neuromuscular, and cardiovascular involvement.

Nephritic syndrome is a glomerular syndrome characterized by grossly visible hematuria with dysmorphic RBCs and RBC casts in urine, mild to moderate proteinuria, azotemia, edema, and hypertension. Nephritic syndrome histological changes involve the proliferation of glomerular cells and glomerular infiltration of leukocytes.

Nephrotic syndrome is a glomerular syndrome characterized by heavy proteinuria (>3.5 g protein/day), hypoalbuminemia, severe edema, hyperlipidemia, and lipiduria.

The cases we will cover are:

A. Minimal-Change Disease  
B. Focal and Segmental Glomerulosclerosis  
C. Membranous Nephropathy (Membranous Glomerulonephritis)  
D. Rapidly Progressive (Crescentic) Glomerulonephritis (Goodpasture Syndrome)

The cases for this week will not include virtual slides.
A. MINIMAL-CHANGE DISEASE

CC/HPI: A four year old male presents to his physician after his mother notices that he has put on weight and notices swelling in his ankles and wrists. She reports that he has been more tired than usual and that his urine has been foamy. He had an upper respiratory tract infection one week ago.

PE: Physical exam reveals generalized pitting edema (bilateral) and abdominal distension. Blood pressure is normal. Pulse is normal.

Why is edema present in this patient?

Labs: Urinalysis reveals proteinuria (specifically hyperalbuminuria) (>3.5 g/day) and lipiduria. A blood analysis reveals hypoalbuminemia, hyperlipidemia, normal serum creatinine and BUN.

Does this patient exhibit nephritic or nephrotic syndrome?

Pathology: A renal biopsy is taken and the following image is observed:
The child is treated with a short course of corticosteroid and returns to normal within a week.

**Does this glomerulus look normal?**

**Hypoalbuminemia and proteinuria suggest that what structures shown in the above picture are likely to be damaged?**

**What is the structural defect in this disease (seen in the EM image)?**

**B. FOCAL AND SEGMENTAL GLOMERULOSCLEROSIS**

**CC/HPI:** A sixteen year old female with a history of heroin abuse and STIs presents to her physician with complaints of hematuria (blood in her urine) and weight gain.

**PE:** Pulse is normal, her blood pressure is elevated. Generalized pitting edema is observed.

**Labs:** Urinalysis reveals proteinuria (nonspecific proteins) (>3.5 g/ day) and lipiduria. A blood analysis reveals hypoalbuminemia, hyperlipidemia, normal serum creatinine and BUN.

**Does this patient exhibit nephritic or nephrotic syndrome?**

**Patients with nephrotic syndrome caused by nonselective protein loss (not specifically albumin) are often hypercoagulable—why might this occur?**

**Pathology:** A renal biopsy reveals the following image:

![Trichrome image of glomerulus](image-url)
Does this glomerulus look normal?  
No.

If not, what structures appear altered?

Are all glomeruli affected? Describe the progression of this disease. What is the bright blue staining?

C. MEMBRANOUS NEPHROPATHY

CC/HPI: A 47 year old female with a history of chronic hepatitis B infection complains of weight loss, progressive shortness of breath, and swelling of the lower legs and arms.

PE: She is pale with pitting edema in her extremities; decreased lung sounds are observed. Blood pressure is elevated, pulse is normal.

Labs: Urinalysis reveals proteinuria (nonspecific proteins) (>3.5 g/ day) and lipiduria. A blood analysis reveals hypoalbuminemia, hyperlipidemia, normal serum creatinine and BUN.

Does this patient exhibit nephritic or nephrotic syndrome?

Pathology: A renal biopsy reveals the following image:
Silver stain image showing immune deposits:

Immunofluorescence image staining for IgM:
Does this glomerulus look normal?  
No.

If not, what structures appear altered?

How does the basement membrane become leaky if it appears thickened by light microscopy?
D. RAPIDLY PROGRESSIVE (CRESCENTERIC) GLOMERULONEPHRITIS (GOODPASTURE SYNDROME)

**CC/HPI:** A 25 year old male complains of a chronic cough for several months accompanied by lightheadedness, fatigue, and malaise. Yesterday he coughed up blood. He admits to heavy smoking. He also reports intermittent fever and headaches. He complains that he hasn’t been urinating as frequently and when he does it is dark orange in color.

**PE:** Pulmonary crackles are present bilaterally.

**Labs:** A blood analysis demonstrates azotemia (elevated BUN and creatinine) and the presence of antiglomerular abasement membrane antibodies. A urinalysis demonstrates oliguria, hematuria, and proteinuria.

**Pathology:** A renal biopsy reveals the following image:

PAS stain of glomeruli:

![PAS stain of glomeruli](image)

Trichrome image of glomeruli:
H&E stain:

Immunofluorescence stain for IgG:
Does this glomerulus look normal?

If not, what structures appear altered?

Describe the pathology of this disease. Does this patient exhibit nephritic or nephrotic syndrome?

What are crescents? What cells are they derived from?

What other cells are present in the Bowman’s space? Are these cells usually found here?