RENAL DYSFUNCTION

(CHAPTERS 45 AND 46)

QUESTION 1

List some causes of urinary tract obstruction.

In general, urinary tract obstruction can occur due to either blockage **inside** the urinary tract or pressure from **outside** the urinary tract.

Blockage from within the urinary tract typically occurs due to **renal calculi**—a.k.a. "kidney stones."

External pressure can occur due to **tumor** growth near the urinary tract, or during **pregnancy** as the uterus presses on the ureters.

Urinary tract obstruction can also be caused by damage to the **nervous system**, a condition known as **neurogenic bladder**. We'll talk about this in a later question.

QUESTION 2

What is the outcome of urinary tract obstruction?

When the flow of urine out of the body is obstructed, the **production** of urine doesn't slow down—this causes urine to back up into the bladder, first resulting in **bladder distension** and abdominal discomfort.

If the bladder fills to its limit, pressure will either push urine out of the urethra if possible (causing **incontinence**) or up the ureters towards the kidneys (causing **hydronephrosis**.)

QUESTION 3

List the major types of urinary tract stones.

There are **four** major types of renal calculi, categorized based on their **composition**:

calcium stones – most common, usually idiopathic

struvite stones – caused by bacteria that thrive in high-pH urine

uric acid stones – associated with high-purine diet, similar to gout

cystine stones – caused by a rare condition called cystinuria

QUESTION 4

What is the major clinical manifestation of renal calculi?

The classic presentation of **nephrolithiasis**—the presence of calculi in the kidney—is referred to as **renal colic**.

This refers to **flank** and/or **abdominal pain** that occurs due to stones being lodged within the urinary tract.

On assessment, patients will often display **costovertebral angle tenderness** ("CVA tenderness" or simply CVAT,) making this the classic test for calculi.

With positive CVAT, percussing the patient's back where the lower ribs meet the spine will elicit a sharp pain, suggesting nephrolithiasis.

QUESTION 5

List some treatments for renal calculi. What is the goal of these treatments?

The goal of treatment for kidney stones is generally to **restore** or **maintain** patency of the urinary tract while also **preventing** the formation of further calculi.

Intervention ranges from simple lifestyle changes to invasive surgical options, depending on the location of the stone and severity of symptoms.

Asymptomatic nephrolithiasis may resolve on its own with dietary changes and **increased fluid intake** to help pass the stone naturally.

Lithotripsy is a non-invasive procedure which uses high-energy sound waves or lasers to break up larger stones so that they can be passed.

A **nephrolithotomy** may be performed to surgically remove the stone, or a **nephrostomy** inserted to drain urine directly from the kidneys.

QUESTION 6

Describe neurogenic bladder. What is neurogenic bladder commonly associated with?

Neurogenic bladder is a generic term referring to chronic urinary problems caused by a loss of **nervous control** to the bladder.

Remember that the bladder is a **muscle**, and without proper innervation, voiding becomes difficult and inconsistent.

Causes include various neurological disorders, including **Parkinson's**, **multiple sclerosis**, and even **diabetic neuropathy** (nerve damage due to diabetes.)

Alongside symptoms of urinary incontinence and urgency, NB is also associated with **infection** and **nephrolithiasis** due to decreased urine flow.

Patients with neurogenic bladder often require frequent **catheterization** due to urinary retention, which always increases the risk of urinary tract infection.

Remember that straight catheters have **less risk of infection** than indwelling catheters, and always use **sterile technique**!

QUESTION 7

What is the most common renal neoplasm? List the clinical manifestations.

Renal cell carcinoma, a cancer of the primary parenchymal cells of the kidney, represents 85% of renal neoplasms. As with many cancers, **smoking** is a significant risk factor.

Renal symptoms include **flank pain** and **hematuria**, plus general cancer symptoms such as **fatigue** and **rapid weight loss**.

If the tumor grows large enough, there may be a **palpable mass** on the patient's flank, but this is obviously not guaranteed.

QUESTION 8

What is the most common cause of urinary tract infection (UTI?) Which bacteria are involved?

The vast majority of UTIs are **bacterial**, usually caused by normal gut flora such as **E. coli** or **Proteus mirabilis/vulgaris** being spread to the urinary tract.

These bacteria are typically not pathogenic within the GI tract, but cross-contamination of gut bacteria into the urinary system can cause infection.

QUESTION 9

Describe the most common site of urinary tract infection (UTI) and list its types.

Most UTIs fall into the category of **cystitis**, or bladder infection.

This makes sense if you think about it, because UTIs are typically "ascending," i.e. the pathogen comes from **outside** and "ascends" the urethra to the bladder.

Eventually, if untreated, the infection can continue up the ureters and reach the kidneys, causing **pyelonephritis**, or kidney infection.

Cystitis has several forms:

hyperemic – mild inflammatory response to infection

hemorrhagic – damage to the urothelium results in bleeding

suppurative – formation of pus in the bladder

ulcerative – formation of infectious ulcers in the urothelium

gangrenous – serious damage to the urothelium resulting in necrosis

QUESTION 10

Why do women more commonly develop cystitis?

Remember how we said that UTIs typically ascend the urethra from the external environment?

The reason UTIs are more common in women is simple: the bladder is simply **closer** to the urinary orifice in women, meaning a **shorter path** for pathogens to reach the bladder.

Additionally, the relative proximity of the vulva and anus makes it much easier for **gut flora**, such as E. coli, to make their way into the urinary tract.

Clinical practice tip: Make sure that female patients understand the rationale for wiping "front-to-back" after defecation!

QUESTION 11

Describe the usual causative organism in acute pyelonephritis.

Acute pyelonephritis typically occurs as a progression of **untreated cystitis**—bacteria from a bladder infection continue to ascend up one or both ureters, spreading the infection to the kidneys.

Normally, the continuous flow of urine **down** the ureters helps prevent this, but **urinary stasis** or **vesicoureteral reflux** (VUR) can allow bacteria to travel "upstream."

QUESTION 12

How is chronic pyelonephritis different from acute pyelonephritis?

Most cases of pyelonephritis are **acute** in nature and typically resolve with targeted antibiotic therapy.

In some cases, however, an underlying condition such as nephrolithiasis or VUR can result in long-term or repeated infection, which results in **chronic inflammation** and **scarring** of the kidneys.

QUESTION 13

Compare nephrotic and nephritic glomerular diseases.

Nephrotic syndrome and **nephritic syndrome** refer to two distinct sets of symptoms associated with damage to the **glomeruli**—the main "filtering" mechanism of the kidneys.

The glomeruli act like a microscopic sieve, keeping **cells** and large molecules such as **proteins** and **lipids** in the bloodstream, while allowing fluid and electrolytes to pass into the renal tubules.

Nephrotic syndrome (usually caused by **glomerulonephrosis**) is commonly associated with **diabetic nephropathy**, a complication of diabetes in which poorly controlled glucose levels contribute to glomerular damage.

This damage causes the "holes" in the glomeruli to become bigger, allowing proteins to leak into the renal tubules and become excreted in the urine (**proteinuria**.)

This results in **hypoalbuminemia** and therefore **decreased blood osmotic pressure**, leading to **edema**.

Nephritic syndrome (caused by **glomerulonephritis**) is distinct in that the damage to the glomeruli is **inflammatory** and results in **even bigger** "holes" in the glomerular membrane.

These holes are big enough that even **red blood cells** can leak through, resulting in **hematuria**—directly from the bloodstream!

In addition, it can also include some of the same symptoms of nephrotic syndrome due to proteinuria (although usually milder.)

Umm... what? Okay, let's simplify:

glomerulonephr**osis** → nephr**otic** syndrome

glomerular **damage** → bigger holes → **proteinuria**

glomerulonephr**itis** → nephr**itic** syndrome

glomerular **inflammation** → **huge** holes → **hematuria**

QUESTION 14

How do glomerular disorders affect kidney function?

As we mentioned in the last question, the glomeruli are like a "net" intended to keep **big things** in the bloodstream while **small things** are allowed to enter the kidneys.

When the glomeruli suffer damage or inflammation, they no longer filter the big things (like proteins,) which **leak** into the renal tubules and into the urine.

QUESTION 15

What is the most frequent cause of acute glomerulonephritis?

Post-streptococcal glomerulonephritis (PSGN) is another one of those complications of group A strep (GAS) infections that we've been talking about all semester—like rheumatic fever, rheumatic heart disease, etc.

Unlike RF and RHD, PSGN is actually a **type III** hypersensitivity reaction in which strep antigens and their antibodies form immune complexes, which deposit in the glomeruli causing inflammation.

QUESTION 16

Describe Goodpasture syndrome.

Goodpasture syndrome is a rare autoimmune disease in which a type II hypersensitivity reaction causes **rapid deterioration** of the glomerular and respiratory function.

It results in a unique combination of **respiratory** symptoms (e.g. **dyspnea** and **hemoptysis**) plus the symptoms of **nephritic syndrome**.

(Remember "**nephritic**" = **inflammation**!
Huge holes, RBC leakage, and **hematuria**!)

QUESTION 17

Describe chronic glomerulonephritis.

Chronic glomerulonephritis is typically autoimmune in nature, and includes glomerular inflammation due to **Goodpasture syndrome** as well as other disorders such as **systemic lupus erythematosus** (SLE.)

Regardless of the cause, the inflammation causes scarring (**fibrosis**) of the glomeruli, and the increase in glomerular permeability can cause **dilation** of the renal tubules.

QUESTION 18

Describe nephrotic syndrome.

We described this earlier—remember that nephr**otic** syndrome is **non-inflammatory** and results in the glomeruli being permeable to **proteins** and **lipids**, but still retaining **cells** in the bloodstream (so **no** hematuria.)

The key findings in nephrotic syndrome are **proteinuria** and **lipiduria**, often resulting in **edema**. These bigger molecules leak into the kidneys, and the kidneys have no mechanism to reclaim them, so they pass into the urine.

QUESTION 19

At what point does renal failure occur?

Kidney function is indicated using **glomerular filtration rate** (GFR,) which is a measure of how much fluid is passing through the glomeruli and into the kidneys. As kidney failure progresses, GFR goes **down** and urine production **decreases**.

It is typically measured by looking at the **creatinine** levels in the blood, since creatinine is produced at a fairly constant rate by the body and is normally excreted in the urine.

If the serum creatinine level is **high**, this indicates a **low** GFR, as the kidneys are not doing a very good job of clearing creatinine from the blood.

A healthy person's GFR will vary based on age, but the normal range is typically **90–120** ml/min/1.73 m^2 (the units are weird because GFR is adjusted for body size.)

Renal failure is diagnosed when kidney function has decreased to about **25%** of its normal value—generally **25-30** ml/min/1.73 m^2 or lower.

When this reaches **~10%** or lower—**15** ml/min/1.73 m^2 —we call this **end-stage renal disease** (ESRD.)

ESRD patients have such extreme kidney failure that they **must** receive hemodialysis on a regular basis (or undergo a kidney transplant) to survive.

QUESTION 20

Define azotemia and uremia.

Azotemia (from the French "azote" meaning "nitrogen") refers to the buildup of nitrogen-containing compounds such as **urea** and **creatinine** in the blood, caused by renal insufficiency.

While this excess is measurable in the blood, it is not necessarily severe and may be **asymptomatic**.

Uremia occurs when these nitrogen-containing compounds build up to a level that starts producing **symptoms**, such as **fatigue**, **pruritus** (itching,) and **altered mental status**.

This typically begins when GFR reaches around 50–60 mg/ml/1.73 m^2, or roughly **half** of normal kidney function—so **before** true renal failure sets in.

QUESTION 21

Compare prerenal, intrarenal, and postrenal failure.

Let's say we have a patient with a sudden onset of **oliguria** (low urine production.) This is suggestive of an **acute kidney injury** (AKI)... but why is it happening?

The possible answers essentially fall into three categories: **prerenal**, **intrarenal**, or **postrenal**.

Prerenal kidney failure ("before" the kidneys) occurs when there is **hypoperfusion** of the kidneys—they are failing to filter the blood because the blood isn't being **delivered** effectively.

This could be **low blood pressure** due to **dehydration**, **burns**, **hemorrhage**, etc.—or **heart failure**/**cardiogenic shock**.

Intrarenal kidney failure ("in" the kidneys) occurs when the kidneys have enough blood to filter, but aren't filtering it quickly enough.

This could be due to any sort of internal kidney problem, such as **ischemia**, **nephrotoxicity** (drug reaction,) or **post-infective glomerulonephritis**.

Postrenal kidney failure ("after" the kidneys) occurs when the kidneys are filtering the blood fine, but the urine **can't exit** due to **obstruction**.

This could be any of the causes we talked about at the beginning: **calculi**, **tumors**, **pregnancy**, etc.

QUESTION 22

Define oliguria and diuresis (polyuria.)

Oliguria we just defined—a **decrease** in urine output (technically defined as less than **400 ml/day**.)

Beyond this, **anuria** is an **extreme** decrease in urine output, less than 100 ml/day.

Diuresis or **polyuria** is an **increase** in urine output, greater than 3,000-4,000 ml/day.

QUESTION 23

Describe the primary goal of therapy in renal failure.

The kidneys are **important**! The #1 goal in treating kidney failure is simply to keep the patient **alive**!

In AKI, this means until recovery.

In CKD/ESRD, it's for **life**!

One common method for dealing with renal failure is **hemodialysis**: actually **removing** the patient's blood from the body, filtering it in a machine, and putting it back in.

ESRD patients with severe cases often need HD **three times a week** just to stay alive, as this process removes toxins and excess fluids from the body.

Less severe renal impairment, such as azotemia, may be controllable with a **low-protein diet** to reduce urea and creatinine production, thus reducing the workload of the kidneys.

We also need to ensure that fluid intake and output remain balanced, and ensure that electrolyte levels don't become dangerously high or dangerously low.

QUESTION 24

Describe the clinical course of chronic renal failure.

In early **chronic kidney disease** (CKD,) **azotemia** may be present but the patient is often **asymptomatic**, as the body is still compensating adequately.

As GFR decreases, **uremia** gradually sets in, and as the kidneys continue to suffer damage, the GFR eventually reaches the 25–30 threshold at which we begin to call it **renal failure**.

Finally, as the GFR reaches 15 and below, survival without intervention (such as HD or transplant) becomes impossible, which we call **ESRD**.