



RENAL VEIN THROMBOSIS

Presented by:

Dr. M. Hassanian

آذر ماه 1402



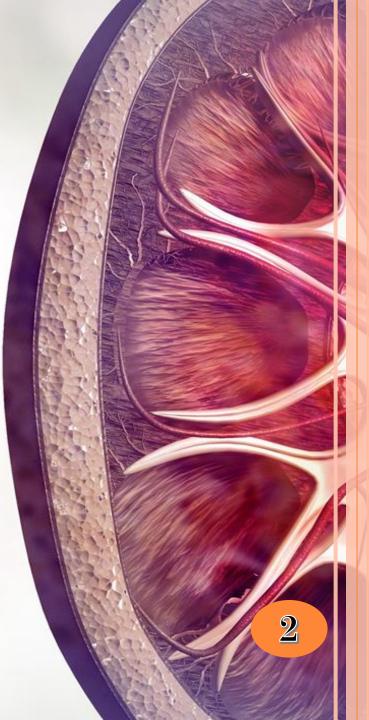
RENAL VEIN THROMBOSIS



- Introduction
- > Etiology
- Epidemiology
- > Pathophysiology
- > History and Physical
- Evaluation
 - **Treatment / Management**
- Differential Diagnosis
- Prognosis



- Renal vein thrombosis (RVT), the presence of thrombus in the major renal veins or its tributaries, is a rare clinical entity.
- It can present acutely or go unnoticed and can result in acute kidney injury or chronic kidney disease.
- Rayer, was the first to describe RVT and its association with proteinuria in the 1840s.



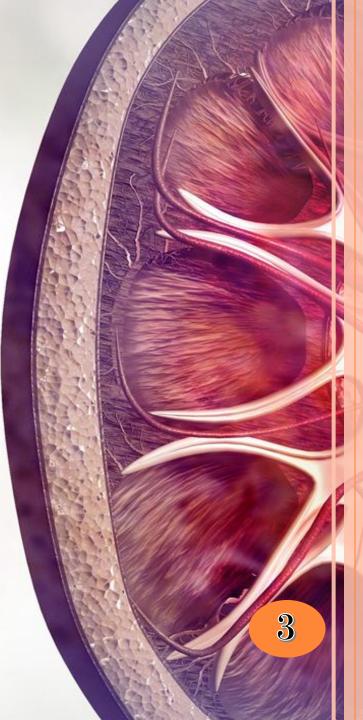


- Bilateral renal vein involvement Two-thirds of patient
- Left renal vein: more commonly involved due to its extensive venous

network compared to the right.

■ May extend from the vena cava into the peripheral venules or may

originate in the peripheral veins and propagate to the main renal vein.





• The severe passive congestion that develops causes the kidney to swell and become engorged, leading to degeneration of nephrons and causing symptoms of flank pain, hematuria, and decreased urine output



ETIOLOGY



• The most common: Nephrotic syndrome as it results in a hyper-coagulable state.

• In patients who are nephrotic, the most common underlying nephropathy associated with RVT is membranous nephropathy, followed by minimal change disease, and membrano proliferative glomerulonephritis

ETIOLOGY Systemic

- Primary hypercoagulability disorders (antithrombin III deficiency, protein C or S deficiency, factor VLeiden mutation, prothrombin G20210A mutation)
- Antiphospholipid syndrome
- Postrenal transplant/allograft rejection
- Renal vasculitis
- ✓ Sickle cell nephropathy



ETIOLOGY Systemic

- Systemic lupus erythematosus (SLE)
- Amyloidosis
- Diabetic nephropathy
- Pregnancy or estrogen therapy
- ✓ Behcet syndrome
- Severe dehydration or prolonged hypotension (especially in neonates)
- Infection



ETIOLOGY Local

• The malignant renal tumors, typically renal cell carcinoma that extends into the renal veins, are associated with thrombus in 4% to 25% of cases

• Extrinsic compression of the renal vein or IVC due to a tumor, lymph nodes, retroperitoneal disease, or an aortic aneurysm

• Blunt abdominal trauma or trauma during venography





• There is scarce data on the exact US prevalence of RVT in adults as it is often asymptomatic with spontaneous resolution

• There is also a high degree of variability as the reported number of cases of RVT in patients with nephrotic syndrome, and membranous nephropathy is 5% to 60%.



• In intrauterine life, RVT can occur mostly in the presence of factor V Leiden.

• In neonates, RVT is one of the mostfrequent causes of venous thrombosis and usually follows severe dehydration or prolonged periods of hypotension.



• No specific numbers are available for the frequencies of RVT by gender. However, since RVT is commonly associated with membranous nephropathy, a male predominance is reported. This is because membranous nephropathy has a 2:1male to female ratio.



- Age is a factor in RVT only to the extent as any age-related risk of glomerular disease (membranous nephropathy peaks in the fourth through the sixth decade, making RVT likely in this specific age group).
- Lastly, there is no racial predilection.



PATHOPHYSIOLOGY



□ The pathogenesis of RVT is based on Virchow's triad which is

due to one or more of the following:

- > Vascular endothelial damage due to an injury of the vessel wall or
 - in cases of homocystinuria where high levels of homocysteine cause

spontaneous microtrauma to the endothelium



PATHOPHYSIOLOGY

- Stasis of blood flow(in severe dehydration/volume depletion in infants)
- A hypercoagulable state such as in patients with a nephrotic syndrome where excessive urinary protein loss, decreased antithrombin III levels, a relative excess of fibrinogen, and changes in other clotting
 - factors; all leads to the propensity of a clot



HISTORY AND PHYSICAL



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- Clinical presentation of renal vein thrombosis varies by the rapidity of venous occlusion and development of venouscollaterals.
- Chronic RVT has an insidious onset and is usually asymptomatic.
- It may present as peripheral edema.

HISTORY AND PHYSICAL

• Acute renal vein thrombosis usually presents with:

- Renal infarction(flank pain, flank tenderness)
- Rapid deterioration of renal function
- Worsening proteinuria
- Micro or macroscopic hematuria
- Nausea, vomiting or fever may be present



HISTORY AND PHYSICAL

• In a renal transplant, RVT usually presents within 48 hours of surgery manifesting as sudden anuria and tenderness over the transplanted kidney. • In neonates and infants, acute RVT may present with gross hematuria, flank mass, signs and symptoms of dehydration or shock, and oligo-anuria. • Left-sided RVT can lead to gonadal vein thrombosis manifested as pelvic congestion syndrome in females and whereas in males with painful swelling of the left testis and varicocele.



EVALUATION



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- » RVT has an insidious onset, producing minimal to no symptoms, and is usually diagnosed incidentally during imaging for some other reason.
- > Renal venography is the gold standard diagnostic test for RVT and has the additional advantage of a therapeutic procedure.
 - > Rarely used in current practice due to the availability of lesser invasive tests.
- Renal ultrasonography(USG): is a safe, non-invasive test, usually not sensitive enough to make a diagnosis of RVT.

EVALUATION

- Renal USG shows an enlarged kidney and hyper-echogenic kidney in approximately 90% of the patients in the early phase of acute RVT.
- Color Doppler or contrast-enhanced ultrasonography may yield better results.
- Computed tomography (CT) angiography: Choice for diagnosing RVT which can demonstrate thrombus in the renal vein and, at times, in the vena cava.
 - > The sensitivity and specificity of CT angiography are almost 100%.

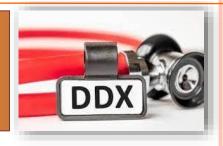


EVALUATION

- Contrast-enhanced, magnetic resonance venography (3D-CE-MRV) is assensitive as CT and has the major benefit of avoidance of radiation and intravenous (IV) contrast.
 - * Both CT and MRV can help detect RVT as well as the presence of renal cell cancer.
- No specific laboratory studies are indicated for renal vein thrombosis (RVT), except those specific for the underlying etiology (e.g., for nephrotic syndrome or coexisting hypercoagulable state).

TREATMENT / MANAGEMENT

DIFFERENTIAL DIAGNOSIS



- Acute RVT presenting with loin pain and hematuria can mimic renal colic, pyelonephritis, renal papillary necrosis, or renal infarction from renal artery thrombosis or embolism.
- Renal cyst in adult polycystic kidney disease is an other condition to consider.
- * Rarely, a tumor thrombus (extension of a tumor into the vein) may be confused with RVT.

PROGNOSIS

□ Prognosis is favorable with treatment.

- However, the morbidity and mortality of RVT depend on its underlying cause. For example, in cases of RVT secondary to malignancy, complications can arise from the malignancy itself or result in thromboembolism at other sites such as DVT or PE. Prognosis of RVT due to nephrotic syndrome as per a retrospective cohort study showed a 40% mortality at 6 months in 27 patients with RVT.
- Survivors appeared to have stable renal function and resolution of nephrotic syndrome.
- RVT that develops after a renal transplant has a poor prognosis and usually results in graft failure, particularly in the first month post-transplant

