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ORIGIN , PATHOGENESIS OF GLOMERULONEPHRIT DISEASE

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Annotation: Glomerulonephritis is a kind of kidney disease. It involves damage to your glomeruli, tiny filters inside your kidneys. Some people don't show any symptoms. Infections and immune system disorders are one of the many causes. Sometimes, glomerulonephritis is mild and goes away without treatment. Other times it leads to kidney failure and other complications.

Key words : Kidney , nephron, glomerulonephritis.

Each kidney contains about 1 million filtering units (glomeruli). The glomeruli are made up of many microscopic clusters of tiny blood vessels (capillaries) with small pores. These blood vessels are designed to leak fluid from the bloodstream into a system of miniature tubules. The tubules secrete and reabsorb chemicals and substances from the fluid to cause it to become urine. The urine then drains from the tubules into larger and larger tubes until it leaves the kidney. Normally this filtering system permits fluid and small molecules (but almost no protein or blood cells— Asymptomatic Proteinuria and Hematuria Syndrome) to leak into the tubules. Diseases that affect the kidneys can be divided into 3 categories based on the way they affect different parts of the kidneys:

- Glomerulonephritis (or nephritic syndrome) is inflammation of the glomeruli, which causes blood cells and protein to escape from the glomerular capillaries into the urine and is sometimes hereditary (it is then called hereditary nephritis, or Alport syndrome).
- Nephrotic syndrome occurs when damage to the capillaries of the glomeruli causes proteins to leak into the urine.
- Tubulointerstitial nephritis is inflammation of the tubules and/or the tissues surrounding the tubules (interstitium).

Some disorders have features of both glomerulonephritis and nephrotic syndrome.

A separate disorder, called reflux nephropathy due to reflux of urine from the bladder up to the kidneys, increases the risk of urinary tract infection and can cause inflammation and scarring of the kidneys.

Glomerulonephritis, nephrotic syndrome, and tubulointerstitial nephritis are not specific disorders but rather categories of disorders. Many specific disorders fall within each category, and many specific conditions may cause disorders in each category. For example, membranoproliferative glomerulonephritis is inflammation of kidney filtering cells caused by an immune response. The immune response might be caused by any of several autoimmune disorders, such as systemic lupus erythematosus (lupus), or by an infection or even cancer.

In glomerulonephritis, inflammation is often the result of an abnormal immune reaction. Such a reaction can occur in 2 ways:

Antibodies (proteins made by the body to attack specific molecules called antigens) may attach directly to cells of the kidneys or molecules trapped in them, causing inflammation.

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Antibodies attach to antigens outside the kidneys, and these antigen-antibody (or immune) complexes are carried to the kidneys by the bloodstream and get trapped in the glomeruli, causing inflammation.

If enough glomeruli are damaged, kidney function is decreased. As a result, urine production falls and waste products build up in the blood. Also, when damage is severe, inflammatory cells and injured glomerular cells accumulate, compressing the capillaries within the glomeruli and interfering with filtration. Scarring may develop, which also impairs kidney function and reduces urine production. In some cases, tiny blood clots (microthrombi) may form in the small blood vessels, further decreasing kidney function. Rarely, glomerulonephritis can result from a hereditary condition. In other cases, glomerulonephritis is caused by inflammation of the blood vessels (vasculitis).

Nephrotic syndrome causes large amounts of protein to leak from blood into the urine. This leakage can be caused by damage to the glomeruli by inflammatory or noninflammatory processes. In inflammatory processes, red blood cells appear in the urine. Nephrotic syndrome caused by inflammation, therefore, has characteristics similar to those of glomerulonephritis. With noninflammatory processes, no red blood cells appear in the urine. Some forms of nephrotic syndrome can be severe. The glomeruli become scarred, and kidney failure (loss of most kidney function) develops. In less severe forms of nephrotic syndrome, kidney function decreases very little.

Tubulointerstitial nephritis often is caused by a toxic or allergic reaction to a medication. White blood cells or scar tissue appears in the affected kidney. Infection of the kidneys (pyelonephritis) can also cause tubulointerstitial nephritis. When inflammation damages the tubules and surrounding tissues, the kidneys may become unable to carry out their normal functions, such as concentrating urine, resulting in urine that is too dilute. The kidneys may also become unable to eliminate (excrete) waste products from the body or balance the excretion of acid, sodium, and other electrolytes, such as potassium. If the damage is severe and affects both kidneys, the result is kidney failure.

Nephrotic syndrome is a disorder of the glomeruli (clusters of microscopic blood vessels in the kidneys that have small pores through which blood is filtered) in which excessive amounts of protein are excreted in the urine. Excessive protein excretion typically leads to accumulation of fluid in the body (edema) and low levels of the protein albumin and high levels of fats in the blood.

- Medications and disorders that damage the kidneys may cause nephrotic syndrome.
- People feel tired and have tissue swelling (edema).
- Diagnosis is based on blood and urine tests and sometimes imaging of the kidneys, a biopsy of the kidneys, or both.
- People who have disorders that may cause nephrotic syndrome are given angiotensin-converting enzyme (ACE) inhibitors or angiotensin II receptor blockers (ARBs) to slow kidney damage.
- Restriction of sodium intake plus diuretics and statins are also used to treat this disorder.

(See also Overview of Kidney Filtering Disorders.)

Nephrotic syndrome can develop gradually or suddenly. Nephrotic syndrome can occur at any age. In children, it is most common between the ages of 18 months and 4 years, and more males than females are affected. In older people, both sexes are equally affected.

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Excessive protein excretion into the urine (proteinuria) results in low levels of important proteins, such as albumin, in the blood. People also have increased levels of fats (lipids) in the blood, a tendency to increased blood clotting, and a greater susceptibility to infection. The decreased level of albumin in the blood causes fluid to leave the bloodstream and enter the tissues. Fluid in the tissues leads to edema. Fluid leaving the bloodstream causes the kidneys to compensate by retaining more sodium. Nephrotic syndrome can be

Primary, originating in the kidneys

Secondary, caused by a vast array of other disorders

A variety of primary kidney disorders can damage the glomeruli and cause nephrotic syndrome. Minimal change disease is the most common cause of nephrotic syndrome in children.

The secondary causes may involve other parts of the body. The most common disorders causing nephrotic syndrome are diabetes mellitus, systemic lupus erythematosus (lupus), and certain viral infections. Nephrotic syndrome can also result from kidney inflammation (glomerulonephritis). A number of medications that are toxic to the kidneys can also cause nephrotic syndrome, especially nonsteroidal anti-inflammatory drugs (NSAIDs). The syndrome may be caused by certain allergies, including allergies to insect bites and to poison ivy or poison oak. Some types of nephrotic syndrome are hereditary.

Literature :

1. *LI*, 1-6.
2. Maxmudovich, A. X., Raximberdiyevich, R. R., & Nozimjon o'g'li, S. S. (2021). Oshqozon Ichak Traktidagi Immunitet Tizimi. *TA'LIM VA RIVOJLANISH TAHLILI ONLAYN ILMIY JURNALI*, *1*(5), 83-92.An Efficient Algorithm to Find All Small-Size Stopping Sets of Low-Density Parity-Check Matrices Eirik Rosnes, Member, IEEE, and yvind Ytrehus, Senior Member, IEEE 2009.
3. IRE TRANSACTIONS ON IFORMATION THEORY 21 Low-Density parity-Check Codes20056] Achieving the Secrecy Capacity of WiretapChannels Using Polar Codes Hessam Mahdavifar, Student Member, IEEE, and Alexander Vardy, Fellow, IEEE 2011
4. Information-Theoretic Key Agreement: FromWeak to Strong Secrecy for Free Ueli Maurer and Stefan Wolf Computer Science Department, Swiss Federal Institute of Technology (ETH Zurich)7.Physical-Layer security:Combining Error Control Coding and Cryptography Willie.
5. Дусмухамедов, Д. М., Юлдашев, А. А., & Хакимова, З. К. (2020). ОБЩИЙ СТОМАТОЛОГИЧЕСКИЙ СТАТУС У БОЛЬНЫХ ГНАТИЧЕСКИМИ ФОРМАМИ АНОМАЛИИ ОККЛЮЗИИ. *ББК 1 Р76*, 30.
6. Dusmukhamedov, D. M., Dusmukhamedov, M. Z., & Khakimova, Z. K. (2019). ESTIMATION OF MORPHOMETRIC CHANGES OF UPPER RESPIRATORY WAYS IN PATIENTS WITH DENTAL JAW DEFORMITIES. In *Colloquium-journal* (No. 28-3, pp. 5-6). Голопристанський міськрайонний центр зайнятості= Голопристанский районный центр занятости.
7. Дусмухамедов, М. З., Юлдашев, А. А., Дусмухамедов, Д. М., & Хакимова, З. К. (2022). ХИРУРГИЧЕСКОГО ЛЕЧЕНИЯ БОЛЬНЫХ С ВТОРИЧНЫМИ ДЕФОРМАЦИЯМИ

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8. Salomov, S. N. O. G. L., Aliyev, H. M., & Dalimova, M. M. (2022). RECONSTRUCTIVE RHINOPLASTY METHOD WITH EXTERNAL NOSE DEFORMATION AFTER UNILATERAL PRIMARY CHEILOPLASTY. *Central Asian Research Journal for Interdisciplinary Studies (CARJIS)*, *2*(10), 87-90.
9. Shoxabbos, S., & Mahramovich, K. S. M. K. S. (2023). CAUSES OF THE ORIGIN OF CARDIOVASCULAR DISEASES AND THEIR PROTECTION. *IQRO JURNA*