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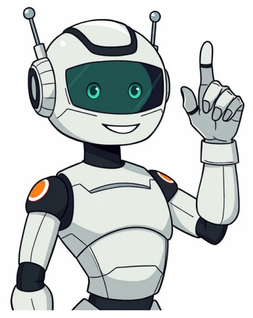

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However, constant changes in information resulting from continuing research and clinical experience, reasonable differences in opinions among authorities, unique aspects of individual clinical situations, and the possibility of human error in preparing such an extensive text mean that other sources of medical information may differ from the information on this site. The information on this site is not intended to be professional advice and is not intended to replace personal consultation with a qualified physician, pharmacist, or other health care professional. The reader should not disregard medical advice or delay seeking it because of something found on this site. Content in the Manuals reflects medical practice and information in the United States. Outside of the United States, clinical guidelines, practice standards, and professional opinion may differ and the reader is advised to also consult local medical sources. Please note, not all content that is available in English is available in every language. Nephrotic syndrome is urinary excretion of > 3 g of protein/day due to a glomerular disorder plus edema and hypoalbuminemia. It is more common among children and has both primary and secondary causes. Diagnosis is by determination of urine protein/creatinine ratio in a random urine sample or measurement of urinary protein in a 24-hour urine collection; cause is diagnosed based on history, physical examination, serologic testing, and renal biopsy. Prognosis and treatment vary by cause.(See also Overview of Glomerular Disorders.) Nephrotic syndrome occurs at any age but is more prevalent in children (primarily minimal change disease), with a mean age of diagnosis of 5 to 6 years (1, 2). Congenital nephrotic syndromes appear during the first year of life. At younger ages (< 8 years), boys are affected more often than girls, but both are affected equally at older ages. Causes differ by age (see table Glomerular Disorders by Age and Presentation) and may be primary or secondary (see table Causes of Nephrotic Syndrome).The most common primary causes are the following:Important secondary causes, more common in adults than children, includeDiabetic nephropathySystemic lupus erythematosusPreeclampsia (3)Amyloidosis is an underrecognized cause.HIV-associated nephropathy is a type of focal segmental glomerulosclerosis that occurs in patients with advanced HIV infection. 1. El Bakkali L, Rodrigues Pereira R, Kuik DJ, Ket JC, van Wijk JA. Nephrotic syndrome in The Netherlands: a population-based cohort study and a review of the literature. Pediatr Nephrol 2011;26(8):1241-1246. doi:10.1007/s00467-011-1851-82. Parikh RV, Tan TC, Fan D, et al. Population-based identification and temporal trend of children with primary nephrotic syndrome: The Kaiser Permanente nephrotic syndrome study. PLoS One 2021;16(10):e0257674. Published 2021 Oct 14. doi:10.1371/journal.pone.02576743. Kodner C. Diagnosis and Management of Nephrotic Syndrome in Adults. Am Fam Physician 2016;93(6):479-485.Proteinuria occurs because of changes to capillary endothelial cells, the glomerular basement membrane (GBM), or podocytes, which normally filter serum protein selectively by size and charge.The mechanism of damage to these structures is unknown in primary and secondary glomerular diseases, but T cells may upregulate a circulating permeability factor or downregulate an inhibitor of permeability factor in response to unidentified immunogens and cytokines. Other possible factors include hereditary defects in proteins that are integral to the slit diaphragms of the glomeruli, activation of complement leading to damage of the glomerular epithelial cells and loss of the negatively charged groups attached to proteins of the GBM and glomerular epithelial cells. The disorder results in urinary loss of macromolecular proteins, primarily albumin but also opsonins, immunoglobulins, erythropoietin, transferrin, hormone-binding proteins (including thyroid-binding globulin and vitamin D-binding protein), and antithrombin III. Deficiency of these and other proteins contribute to a number of complications (see table Complications of Nephrotic Syndrome); other physiologic factors also play a role.Primary symptoms include anorexia, malaise, and frothy urine (caused by high concentrations of protein).Fluid retention may cause:Dyspnea (pleural effusion or laryngeal edema)Arthralgia (hydrarthrosis)Abdominal pain (ascites or, in children, mesenteric edema)Corresponding signs may develop, including peripheral edema and ascites. Edema may obscure signs of muscle wasting and cause parallel white lines in fingernail beds (Muehrcke lines).Other symptoms and signs are attributable to the many complications of nephrotic syndrome (see table Complications of Nephrotic Syndrome).Urine random (spot) protein/creatinine ratio ≥ 3 or proteinuria ≥ 3 g/24 hoursSerologic testing and renal biopsy unless the cause is clinically obviousDiagnosis is suspected in patients with edema and proteinuria on urinalysis and confirmed by random (spot) urine protein and creatinine levels or 24-hour measurement of urinary protein. The cause may be suggested by clinical findings (eg, systemic lupus erythematosus, preeclampsia, cancer); when the cause is unclear, additional (eg, serologic) testing and renal biopsy are indicated.A finding of significant proteinuria (3 g protein in a 24-hour urine collection) is diagnostic (normal excretion is < 150 mg/day). Alternatively, the protein/creatinine ratio in a random urine specimen usually reliably estimates grams of protein/1.73 m2 body surface area (BSA) in a 24-hour collection (eg, values of 40 mg/dL protein and 10 mg/dL [884 micromol/L] creatinine in a random urine sample are equivalent to the finding of 4 g/1.73 m2 in a 24-hour specimen). Calculations based on random specimens may be less reliable when creatinine excretion is high (eg, during athletic training) or low (eg, in cachexia). However, calculations based on random specimens are usually preferred to 24-hour collection because random collection is more convenient and less prone to error (eg, due to lack of adherence); more convenient testing facilitates monitoring changes that occur during treatment.Besides proteinuria, urinalysis may demonstrate casts (hyaline, granular, fatty, waxy, or epithelial cell). Lipiduria, the presence of free lipid or lipid within tubular cells (oval fat bodies), within casts (fatty casts), or as free globules, suggests a glomerular disorder causing nephrotic syndrome. Urinary cholesterol can be detected with plain microscopy and demonstrates a Maltese cross pattern under crossed polarized light; Sudan staining must be used to show triglycerides.Adjunctive testing helps characterize severity and complications.Blood urea nitrogen (BUN) and creatinine concentrations vary by degree of kidney function impairment.Serum albumin often is < 2.5 g/dL (25 g/L).Total cholesterol and triglyceride levels are typically increased.It is not routinely necessary to measure levels of alpha- and gamma-globulins, immunoglobulins, hormone-binding proteins, ceruloplasmin, transferrin, and complement components, but these levels may also be low.The role of testing for secondary causes of nephrotic syndrome (see table Causes of Nephrotic Syndrome) is controversial because yield may be low. Tests are best performed as indicated by clinical context. Tests may include the following:Test results may alter management and preclude the need for biopsy. For example, demonstration of cryoglobulins suggests mixed cryoglobulinemia (eg, from chronic inflammatory disorders such as systemic lupus erythematosus, Sjögren syndrome, or hepatitis C virus infection), and demonstration of a monoclonal protein on serum or urine protein electrophoresis suggests a monoclonal gammopathy (eg, multiple myeloma), especially in patients > 50 years who have anemia.Renal biopsy is indicated in adults to diagnose the disorder causing idiopathic nephrotic syndrome. Idiopathic nephrotic syndrome in children is most likely minimal change disease and is usually presumed without biopsy unless the patient fails to improve during a trial of corticosteroids. Specific biopsy findings are discussed under the individual disorders.Treatment of causative disorderCorticosteroids in children with presumed minimal change diseaseAngiotensin inhibitionSodium restrictionStatinsDiuretics for excessive fluid overloadRarely, nephrectomyTreatment of underlying disorders may include prompt treatment of infections (eg, staphylococcal endocarditis, malaria, syphilis, schistosomiasis), and stopping medications (eg, gold, penicillamine, nonsteroidal anti-inflammatory drugs [NSAIDs]); these measures may cure nephrotic syndrome in specific instances.Treatment of underlying disorders may include prompt treatment of infections (eg, staphylococcal endocarditis, malaria, syphilis, schistosomiasis), and stopping medications (eg, gold, penicillamine, nonsteroidal anti-inflammatory drugs [NSAIDs]); these measures may cure nephrotic syndrome in specific instances.Angiotensin inhibition (using angiotensin-converting enzyme [ACE] inhibitors or angiotensin II receptor blockers [ARBs]) is indicated to reduce systemic and intraglomerular pressure and proteinuria. These medications may cause or exacerbate hyperkalemia in patients with moderate to severe chronic kidney disease.Protein restriction is not recommended because of lack of demonstrated effect on progression.Sodium restriction (< 2 g sodium, or approximately 100 mmol/day) is recommended for patients with symptomatic edema.Loop diuretics are usually required to control edema but may worsen preexisting chronic kidney disease and hypovolemia, hyperviscosity, and hypercoagulability and thus should be used only if sodium restriction is ineffective or there is evidence of intravascular fluid overload. In severe cases, of nephrotic syndrome, IV albumin infusion followed by a loop diuretic may also be given to control edema.Loop diuretics are usually required to control edema but may worsen preexisting chronic kidney disease and hypovolemia, hyperviscosity, and hypercoagulability and thus should be used only if sodium restriction is ineffective or there is evidence of intravascular fluid overload. In severe cases, of nephrotic syndrome, IV albumin infusion followed by a loop diuretic may also be given to control edema.Statins are indicated for dyslipidemia.Limitation of saturated fat and cholesterol intake is recommended to help control dyslipidemia.Anticoagulants are indicated for treatment of thromboembolism, but few data exist to support their use as primary prevention. Rarely, bilateral nephrectomy is necessary in severe nephrotic syndrome because of persistent hypoalbuminemia. The same result can sometimes be achieved by embolizing the renal arteries with coils, thus avoiding surgery in high-risk patients. Dialysis is used as necessary.1. Centers for Disease Control and Prevention: Vaccines and Immunizations: Altered Immunocompetence. Updated August 1, 2023. Accessed February 13, 2025.2. Rubin LG, Levin MJ, Ljungman P, et al. 2013 IDSA clinical practice guideline for vaccination of the immunocompromised host [published correction appears in Clin Infect Dis 2014 Jul 1;59(1):144]. Clin Infect Dis 2014;58(3):e44-e100. doi:10.1093/cid/cit6843. Beck L, Bomback AS, Choi MJ, et al. KDOQI US commentary on the 2012 KDIGO clinical practice guideline for glomerulonephritis [published correction appears in Am J Kidney Dis. 2017 Mar;69(3):485. doi: 10.1053/j.ajkd.2017.01.008.]. Am J Kidney Dis 2013;62(3):403-441. doi:10.1053/j.ajkd.2013.06.002Nephrotic syndrome is most common in young children, is usually idiopathic, and is most often minimal change disease.Consider nephrotic syndrome in patients, particularly young children, with unexplained edema or ascites.Confirm nephrotic syndrome by finding spot protein/creatinine ratio ≥ 3 or urinary protein ≥ 3 g/24 hours.Assume minimal change disease if a child with idiopathic nephrotic syndrome improves after treatment with corticosteroids.In adults, nephrotic syndrome is often secondary, most often to diabetes, systemic lupus erythematosus, or preeclampsia.Test for secondary causes and perform renal biopsy selectively, based on clinical findings.Treat the causative disorder with disease-specific therapy.Administer angiotensin inhibition, sodium restriction, and often diuretics and/or statins to manage proteinuria, edema, and hyperlipidemia.Test your KnowledgeTake a Quiz! This figure shows hand position during subclavian venipuncture (infraclavicular approach).