
Nephrotic Syndrome

Description

Nephrotic syndrome has a course of remissions and exacerbations that usually lasts for months.

- Nephrotic syndrome, or nephrosis, is defined by the presence of nephrotic-range proteinuria, edema, hyperlipidemia, and hypoalbuminemia.
- While nephrotic-range proteinuria in adults is characterized by protein excretion of 3.5 g or more per day, in children it is defined as protein excretion of more than 40 mg/m²/h or a first-morning urine protein/creatinine of 2-3 mg/mg creatinine or greater.

Pathophysiology

In a healthy individual, less than 0.1% of plasma albumin may traverse the glomerular filtration barrier. Amounts above 500 mg/day point to glomerular disease.

- The glomerular capillaries are lined by a fenestrated endothelium that sits on the glomerular basement membrane, which in turn is covered by glomerular epithelium, or podocytes, which envelops the capillaries with cellular extensions called foot processes.
- In between the foot processes are the filtration slits; these three structures—the fenestrated endothelium, glomerular basement membrane, and glomerular epithelium—are the glomerular filtration barrier.
- Filtration of plasma water and solutes is extracellular and occurs through the endothelial fenestrae and filtration slits.
- The importance of the podocytes and the filtration slits is shown by genetic diseases: in congenital nephrotic syndrome of the Finnish type, the gene for nephrin, a protein of the filtration slit, is mutated, leading to nephrotic syndrome in infancy; similarly, podocin, a protein of the podocytes, may be abnormal in a number of children with steroid-resistant focal glomerulosclerosis.
- The glomerular structural changes that may cause proteinuria are damage to the endothelial surface, the glomerular basement membrane, or the podocytes; one or more of these mechanisms may be seen in any one type of nephrotic syndrome.
- Albuminuria alone may occur or, with greater injury, leakage of all plasma proteins (ie, proteinuria) may take place.
- There are two current hypotheses for the formation of edema in nephrotic syndrome: the *underfill* hypothesis holds that the loss of albumin leading to lower plasma colloid pressure is the cause; the *overfill* hypothesis states that the edema is due to primary renal sodium retention.

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Didier Musso



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