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Editorial Note

Editorial: One Step Forward Towards The Advancements In Treating Chronic Kidney Diseases

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Despite being a "silent epidemic" disease, chronic kidney disease (CKD) is considered one of the major causes of mortality, together with its main complication, the cardiovascular disease, which contributes to the poor prognosis of these patients. Inflammation has been recognized as an essential part of CKD and is closely linked to cardiovascular complications. The identification of novel biomarkers using omics technologies is rapidly advancing and could improve the early detection in renal diseases. Omics approaches, including proteomics, could provide novel insights into disease mechanisms, identifying at the same time accurate inflammatory biomarker panels with an essential role in disease monitoring and follow-up. Recent advances highlight the gut microbiota as an important source of inflammation in kidney diseases. An increasing body of evidence reveals the cross talk between microbiota and host in CKD; in addition, gut dysbiosis may represent an underappreciated cause of inflammation and subsequently could lead to malnutrition, accelerated cardiovascular disease and CKD progression. This chapter discusses the relationship between inflammation and CKD and highlights the novel approaches regarding microbiota involvement in CKD pathology, as well as their potential to facilitate improving the quality of life.

To confirm that you have a kidney infection, you'll likely be asked to provide a urine sample to test for bacteria, blood or pus in your urine. Your doctor might also take a blood sample for a culture — a lab test that checks for bacteria or other organisms in your blood. Other tests might include an ultrasound, CT scan or a type of X-ray called a voiding cystourethrogram. A voiding cystourethrogram involves injecting a contrast dye to take X-rays of the bladder when full and while urinating.

Antibiotics for kidney infections

Antibiotics are the first line of treatment for kidney infections. Which drugs you use and for how long depend on your health and the bacteria found in your urine tests.

Usually, the signs and symptoms of a kidney infection begin to clear up within a few days of treatment. But you might need to continue antibiotics for a week or longer. Take the entire course of antibiotics recommended by your doctor even after you feel better.

Your doctor might recommend a repeat urine culture to ensure the infection has cleared. If the infection is still present, you'll need to take another course of antibiotics.

Treatment for recurrent kidney infections

An underlying medical problem such as a misshapen urinary tract can cause you to get repeated kidney infections. In that case, you might be referred to a kidney specialist (nephrologist) or urinary surgeon (urologist) for an evaluation. You might need surgery to repair a structural abnormality.

The presence of albuminuria or proteinuria constitutes a sign of kidney damage and, together with the estimation of glomerular filtration rate, is based on the evaluation of chronic kidney disease. Proteinuria is a strong marker for progression of chronic kidney disease, and it is also a marker of increased cardiovascular morbimortality. Filtration of albumin by the glomerulus is followed by tubular reabsorption, and thus, the resulting albuminuria reflects the combined contribution of these 2 processes.

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The integrity of the glomerular filtration barrier depends on its 3-layer structure (the endothelium, the glomerular basement membrane, and the podocytes). Increased intraglomerular hydraulic pressure or damage to glomerular filtration barrier may elicit glomerular or overload proteinuria. The mechanisms underlying glomerular disease are very variable and include infiltration of inflammatory cells, proliferation of glomerular cells, and malfunction of podocyte-associated molecules such as nephrin or podocin.

Albumin is filtered by the glomeruli and reabsorbed by the proximal tubular cells by receptor-mediated endocytosis. Internalization by endocytosis is followed by transport into lysosomes for degradation. The multiligand receptors megalin and cubilin are responsible for the constitutive uptake in this mechanism. Albumin and its ligands induce expression of inflammatory and fibro genic mediators resulting in inflammation and fibrosis resulting in the loss of renal function as a result of tubular proteinuria. TGF-β, which may be induced by albumin exposure, may also act in a feedback mechanism increasing albumin filtration and at the same time inhibiting megalin- and cubilin-mediated albumin endocytosis, leading to increased albuminuria. Urinary proteins themselves may elicit proinflammatory and profibrotic effects that directly contribute to chronic tubulointerstitial damage. Multiple pathways are involved, including induction of tubular chemokine expression, cytokines, monocyte chemotactic proteins, different growth factors, and complement activation, which lead to inflammatory cell infiltration in the interstitium and sustained fibrogenesis. This tubulointerstitial injury is one of the key factors that induce the renal damage progression.

Therefore, high-grade proteinuria is an independent mediator of progressive kidney damage. Glomerular lesions and their effects on the renal tubules appear to provide a critical link between proteinuria and tubulointerstitial injury, although several other mechanisms have also been involved. Injury is transmitted to the interstitium favoring the self-destruction of nephrons and finally of the kidney structure.