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COVID-19 AND KIDNEY DAMAGE

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ABSTRACT

The review provides data on the frequency and causes of the onset and development of acute kidney injury (AKI) in COVID -19. A review of the available scientific information regarding the occurrence and / or progression of chronic kidney disease with covid -19 was carried out, and an emphasis was placed on rare forms of kidney damage that can develop with covid -19. Attention is paid to aspects of etiopathogenesis and the clinical picture of kidney damage in covid -19. The authors, when writing the article, globally did not touch upon the treatment of renal pathology formed during covid -19, since it is currently mainly reduced, on the one hand, to the treatment of a specific form of kidney disease, on the other hand, to the treatment of the coronavirus infection itself. Understanding the processes of alteration of kidney tissue cells in this disease may help in the future to determine approaches to the prevention of kidney damage and the restoration of their function in patients who are ill or have had covid -19.

Keywords: covid -19, sars - cov -2, coronavirus infection, acute kidney injury, chronic kidney disease, kidney damage, focal segmental glomerulosclerosis

INTRODUCTION

COVID-19 poses a real threat to patients with comorbidities such as diabetes mellitus (DM), hypertension, cardiovascular, renal or hepatic disorders. Kidney damage in people with DM who have been exposed to a new infection seems very likely, and the risk of developing acute kidney injury is associated with mortality. Potential mechanisms for the involvement of the kidneys in the clinical picture of the disease may include cytokine damage, cross-organ damage and systemic effects that determine the treatment strategy. These mechanisms are closely interrelated and are of particular importance for individuals on extracorporeal therapy and kidney transplants. Autopsy data provide evidence of SARS-CoV-2 virus invasion into kidney tissue with damage to tubular epithelial cells and podocytes, erythrocyte aggregation in individuals with severe COVID-19. By including individuals with chronic kidney disease in planned COVID-19 research protocols, an evidence base for effective and safe treatments can be built.

Focal segmental glomerulosclerosis (FSGS) is a form of glomerulopathy, which is characterized by sclerosis of individual segments (segmental changes) in the part of the glomeruli (focal changes); the remaining glomeruli are intact at the onset of the disease. This morphological type of changes is difficult to distinguish from minimal change disease (MCD). It is believed

that these are variants of different severity or different stages of the same disease, united by the term "idiopathic nephrotic syndrome".

Structural disorders of podocytes, disorganization of their actin cytoskeleton, smoothing of the legs, fusion of filtration gaps lead to the development of proteinuria (PU). With prolonged and / or pronounced exposure to a damaging factor, apoptosis mechanisms are activated, podocytes die, lose contact with the glomerular basement membrane (GBM), exfoliate into the urinary space, exposing GBM areas in these places. Having high adhesive properties, the exposed GBM forms synechiae with Bowman's capsule. In places of "fusion" with GBM and in the mesangium, foci of fibrosis are formed. In areas of focal segmental sclerosis, filtration changes its direction towards the interstitium surrounding the glomerulus. As a result, global glomerulosclerosis and interstitial fibrosis are formed. In addition, podocytes in the course of damage undergo transdifferentiation, acquire the properties of fibroblasts and participate in the synthesis of the extracellular matrix, accelerating the formation of fibrosis foci. § Cardiotropin-like cytokine 1 (from the interleukin-6 family), soluble urokinase receptor, hemopexin, etc. are considered as permeability factors. In FSGS and BMI, the activity of circulating permeability factors depends on the balance between the production of these factors (as a result of T-cell dysregulation) and loss with urine of their inhibitors (presumably high density lipoproteins). Proteins of the slit-like diaphragm of podocytes (podocin, nephrin, CD2AP, etc.), which are involved in maintaining the structure and selectivity of the glomerular filter, can be the target of permeability factors. § In virus-induced FSGS, a direct damaging effect of the virus on podocytes is allowed or through the release of inflammatory cytokines that interact with podocyte receptors. § Hemodynamic mechanisms play an important role in damage to podocytes in secondary FSGS associated with a decrease in kidney weight, reflux nephropathy, obesity - adaptive intraglomerular hypertension and hyperfiltration with an increase in glomerular volume, leading to an increase in the mechanical load on podocytes. Hyperproduction of angiotensin II and increased synthesis of TGF-β cause activation of apoptosis, reorganization of the cytoskeleton and dedifferentiation of podocytes.

Acute kidney injury is a sudden decrease in kidney function over several days or weeks, causing a buildup of nitrogenous compounds in the blood (azotemia) with or without decreased urine output. Often this is due to inadequate renal perfusion due to severe trauma, disease, or surgery, but sometimes the cause is rapidly progressive endogenous kidney disease. Symptoms may include anorexia, nausea, and vomiting. Epileptic seizures and coma develop if left untreated. Violations of water, electrolyte and acid-base balance develop rapidly. Diagnosis is based on laboratory examination of renal function, including serum creatinine. Urinalysis, microscopy of the urinary sediment, and often visualization and other investigations (sometimes with kidney biopsy) are needed to determine the cause. Treatment is directed at the cause of the disease, but also includes fluid and electrolyte replenishment and sometimes dialysis.

Diseases of the glomerular apparatus lead to a decrease in the glomerular filtration rate (GFR) and an increase in the permeability of the glomerular capillaries for proteins and erythrocytes; such diseases can be inflammatory diseases (glomerulonephritis) or diseases that develop as a result of vascular pathology - ischemia or vasculitis.

At the tubular level, ischemia and obstruction by cellular debris, protein or crystal deposits, and cellular or interstitial edema may also develop.

Interstitial inflammation (nephritis) usually includes an immunological and allergic component. These mechanisms of tubular injury are complex and mutually dependent, which refutes the earlier term "acute tubular necrosis".

In acute tubular lesions, diuresis can have 3 phases:

- The prodrome usually has normal diuresis and varies in duration depending on the cause (eg, amount of toxin ingested, duration and severity of hypotension).
- During the oliguric period, urine output is usually 50 to 500 ml/day. The duration of the oliguric period is unpredictable and depends on the etiology of AKI and the time to treatment. However, many patients never develop oliguria. Patients without oliguria have lower mortality and morbidity and less need for dialysis.
- Post-oliguric phase - diuresis gradually returns to normal, but serum creatinine and urea levels may remain elevated for several more days. Tubular dysfunction may persist for several days or weeks, manifested by sodium loss, polyuria (possibly massive), vasopressin insensitive, or hypercholeremic metabolic acidosis.

Coronavirus infection (COVID-19) is an infectious disease caused by the SARS-CoV-2 virus.

Most people infected with the virus experience mild to moderate respiratory symptoms and recover without the need for special treatment. At the same time, in some people it occurs in a severe form that requires medical intervention. The severe form of the disease often develops in older people and people with underlying pathologies, in particular cardiovascular, chronic respiratory diseases, cancer and diabetes. Anyone, regardless of age, is at risk of contracting COVID-19, becoming seriously ill or dying from it.

The best way to prevent and slow the transmission of the virus is to educate people about the disease and how it spreads. To protect yourself and others from infection, keep at least 1 m away from others, wear a tight- fitting mask, and wash your hands frequently or use alcohol-based hand rub. Get vaccinated as soon as it's your turn and follow the directions from local authorities.

The source of the spread of the virus can be the mouth or nose of an infected person, from where the virus is ejected with the smallest particles of liquid during coughing, sneezing, talking, singing or breathing. These particles range in size from larger respiratory droplets to finer aerosol dust. It is important to observe respiratory etiquette, in particular, cover your mouth with your elbow when coughing, and if you feel unwell, stay at home and isolate yourself from others until you recover.

Chronic kidney disease (CKD) is a long-term progressive decline in kidney function. Symptoms develop slowly and in advanced stages include anorexia, nausea, vomiting, stomatitis, dysgeusia, nocturia, apathy, chronic fatigue, itching, decreased mental clarity, muscle convulsions and convulsions, fluid retention, malnutrition, peripheral neuropathy, and epileptic seizures. Diagnosis is based on laboratory tests of renal function, sometimes supplemented by renal biopsy. Treatment is primarily directed at the underlying disease, but also includes fluid and electrolyte balance, blood pressure control, anemia management, various types of dialysis, and kidney transplantation.

Chronic kidney disease in the early stages is described as a decrease in renal reserve or renal failure, which can progress (development of end-stage renal disease). Initially, the loss of function of the kidney tissue has almost no obvious pathological manifestations, because the remaining tissue is working hard (functional adaptation of the kidneys).

Decreased renal function correlates with the ability of the kidneys to maintain water and electrolyte homeostasis. In the early stages, the ability of the kidneys to concentrate urine is impaired, and then a decrease in the ability to excrete excess phosphate, acid, and potassium is added. In severe renal failure (glomerular filtration rate ≤ 15 ml / min / 1.73 m2), the ability to effectively dilute or concentrate urine is lost. Thus, urine osmolality is typically around 300-320 mosmol/kg, approaching plasma osmolality (275-295 mosmol/kg), and urine volume does not immediately respond to changes in fluid volume.

The SARS-CoV-2 virus was first detected in December 2019 as a result of nucleic acid analysis in a patient with pneumonia. On December 31, 2019, the World Health Organization was alerted to several cases of viral pneumonia caused by an unknown pathogen. On January 7, 2020, information about the new virus was confirmed, and the virus itself was classified as a coronavirus. The Chinese health services were the first to completely decipher the virus genome, and on January 10 it was made publicly available. Until January 12, 5 genomes were registered in the GenBank database, by January 26, their number increased to 28. With the exception of the earliest genome, genomes are embargoed by GISAID. Phylogenetic analysis is available through Nextrain. On January 20, 2020, human-to-human transmission of the virus was confirmed in the Chinese province of Guangdong.

Coronaviruses, which include SARS-CoV-2, usually cause SARS, but the dangerous viruses SARS-CoV and MERS-CoV, which cause severe acute respiratory syndrome and Middle East respiratory syndrome, respectively, belong to the same family. Coronavirus infection is zooanthroponic, that is, transmission from animals to humans is possible. It was found that the source of SARS-CoV was civets, and MERS-CoV was one-humped camels. It is possible that in the case of SARS-CoV-2, the source of infection is animals - genetic analysis of the virus revealed similarities with coronaviruses common among horseshoe bats, but it is still not known for certain whether they are the original source of infection. Now the main way the virus spreads is person-to-person transmission.

Scientists from different countries have analyzed the genome of the virus and confirm the fact that the virus is very likely to be of natural origin. Various conspiracy theories create an atmosphere of fear, rumors and prejudice, such theories are condemned by the scientific community. Together with the WHO Director-General, scientists are calling for the promotion of scientific evidence instead of disinformation.

According to the latest data, SARS-CoV-2 (like SARS-CoV-1) is able to remain viable outside the body from 3 hours to 4 days, depending on the surface of the object. The virus remains most stable on stainless steel (2 days) and plastic (3 days). During this period, its concentration decreases by more than 3 orders of magnitude. Depending on the conditions, the concentration of the virus decreases by 2 times on stainless steel in the first 3-7 hours, and on plastic - in the first 5.5-9 hours. In the air, the concentration of the virus drops by an order of magnitude in 3 hours under laboratory conditions, while on steel - in 18-19 hours, and on plastic - in 20 hours

and, in rare exceptions, in 22 hours. There is no risk of infection when receiving parcels or letters.

The reproduction index, according to the Chinese Center for Disease Control and Prevention, is estimated between 2 and 3, which by definition of the index corresponds to the number of people who become infected from one infected person, one study estimated the average value as of January 22, 2020. In general, values of this number greater than 1 mean that the epidemic will spread, and measures to counteract the spread of infection help to reduce the number.

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