Chronic kidney disease

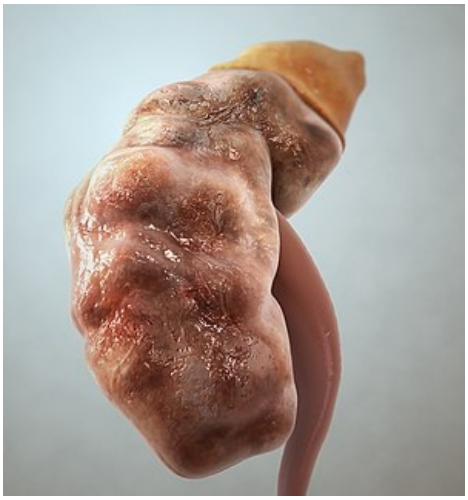
Chronic kidney disease (CKD) is defined as damage to the structure or function of the kidneys lasting at least 3 months, which has consequences for the patient's health. [1]

Sometimes we can also meet the names "chronic renal insufficiency", in the case of the last stage of the disease (G5, see below) also "chronic renal failure". However, this is not the official name of the disease.

Characteristics of the disease

Evidence for chronic kidney disease:

- signs of kidney damage:
  - albuminuria (*albumin excretion rate* ≥ 30 mg/24 hours; or ratio albumin to creatinine, *albumin to creatinine ratio* ≥ 3 mg/mmol),
  - abnormal finding in urine sediment,
  - electrolyte and other abnormalities caused by tubule damage,
  - abnormal histological findings,
  - abnormal finding on imaging methods (structural kidney damage),
  - kidney transplant in the anamnesis,
- reduced glomerular filtration below 60 ml/min/1.73 m^2



chronic kidney disease

The classification of chronic kidney disease is based on the values of three parameters[2][3]:

- Cause,
- determination of glomerular filtration (grades G1-G5),
- determination of albuminuria (grades A1-A3).

The following table shows the classification of diseases based on glomerular filtration:

CKD category based on GF (according to KDIGO 2012[1])

Categories	Glomerular filtration (ml/min/1.73 m^2)
G1	≥ 90
G2	60-89
G3a	45-59
G3b	30-44
G4	15-29
G5	< 15

The categories of chronic kidney disease according to albuminuria are shown in the following table:

Category of CKD based on albuminuria (according to KDIGO 2012[1])

Category	Albuminuria (mg albumin/24 hours)	Proportion of albumin and creatinine in urine collection (mg albumin/mmol creatinine)
A1	< 30	< 3
A2	30-300	3-30
A3	> 300	> 30

Epidemiology

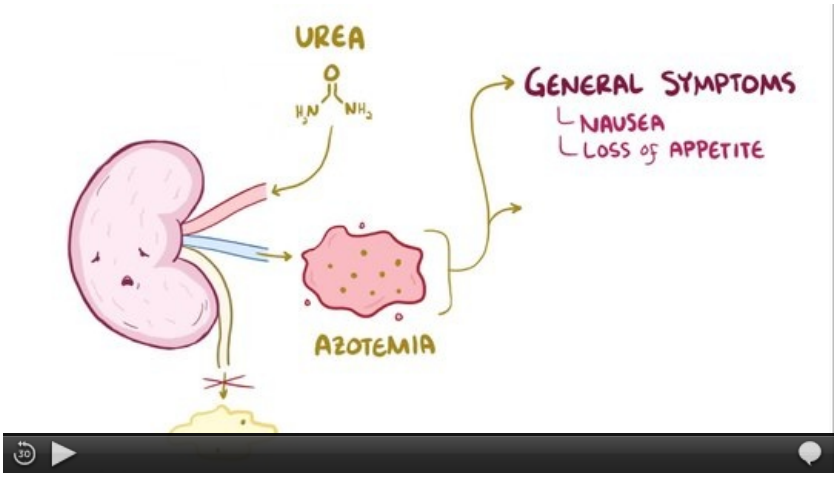
Prevalence of CKD of all categories (G1-G5) is estimated to be about 13% worldwide [4]. Incidence of patients in the last stage of the disease (G5) treated with one of the methods of renal replacement therapy (*renal replacement therapy, RRT*, i.e. dialysis methods and transplantation) in Europe is around 129 new patients per 1 million inhabitants per year [2]. In the Czech Republic, the incidence of new patients in the hemodialysis program is around 200 new patients per 1 million inhabitants[5].

Etiopathogenesis

The causes of chronic kidney disease can be many and can be combined. These are most often the consequences of other diseases, especially diabetes mellitus, arterial hypertension or glomerulonephritis[2][4]. Other causes are hereditary kidney diseases (eg polycystic kidney disease, tuberous sclerosis, von Hippel-Lindau disease, Alport syndrome, Bartter syndrome, Gitelman syndrome), tubulointerstitial nephritis, hydronephrosis and the like.

In the initial phase of chronic kidney disease, kidney tissue is damaged by the underlying disease (arterial hypertension, diabetes mellitus, etc.). In the course of the disease, there is a gradual loss of functional nephrons. Residual nephrons initially undergo adaptive changes (hypertrophy, dilation of the afferent arteriole, increased plasma flow, hyperfiltration). After the majority of nephrons are damaged (data in the literature are around 75% - 85%), the residual nephrons are exposed to excessive metabolic load, structural and metabolic changes occur leading to the development of glomerulosclerosis and fibrotization in tubulointerstitium[2][4].

Summary video



Video in English, definition, pathogenesis, symptoms, complications, treatment.

Clinical picture

The clinical picture of chronic kidney disease depends on disorders of individual kidney functions[4].

Decreased concentration function of the kidneys is manifested mainly by swelling or polyuria. As a result of kidney disease, the electrolyte balance is also disturbed. We can especially encounter hyperkalemia, hyperphosphatemia and hypocalcemia, which can clinically manifest in the form of arrhythmias, [[Hyperparathyroidism|hyperparathyroidism]], convulsions or a disorder of bone metabolism (renal osteopathy). A build-up of uric acid (hyperuricemia) can result in urolithiasis or gouty arthritis.

Part of kidney function is the production of erythropoietin. In the case of chronic kidney disease, this production is reduced and is manifested by progressive (often normochromic normocytic) anemia and anemic syndrome. Another product of the kidneys is 1,25-dihydroxycholecalciferol or calcitriol, the reduced production of which is manifested by hypocalcemia, hyperparathyroidism and a disorder of bone metabolism.

Other manifestations of chronic kidney disease can be various cardiovascular, hematological, neurological, endocrine, gastrointestinal or infectious complications. These include, for example, metabolic acidosis, bleeding conditions (uremia caused by thrombocytopathy), uremic encephalopathy, peripheral neuropathy, dyslipidemia, dyspepsia , malnutrition, gastrointestinal bleeding, uremic colopathy, vascular calcification, uremic pericarditis, pruritus and skin discoloration. arterial hypertension can also be a consequence of impaired kidney function, which in itself further worsens the course of the disease.

Diagnostics

The diagnosis of chronic kidney disease is based on the examination of glomerular filtration and albuminuria and on auxiliary examinations, which aim to find out the cause of the disease and to distinguish chronic kidney disease from acute kidney damage.

In chronic kidney disease, findings of anemia are common, imaging methods often demonstrate wrinkled kidneys with parenchyma reduction and the possible presence of cysts. Laboratory findings may include hypocalcemia, hyperphosphatemia, elevated parathyroid hormone and metabolic acidosis. If a histological sample is taken, glomerulosclerosis or tubulointerstitial fibrosis can be demonstrated.

In contrast, with acute kidney damage, an ultrasound finding of enlarged kidneys is more likely, and the above-mentioned laboratory findings are not so pronounced.

Treatment

One of the first steps in treatment is the administration of antihypertensives. Combination therapy, especially ACEI and sartans, is most often given, as they have a renoprotective effect. Arterial hypertension is not only a common cause of chronic kidney disease, but also one of the most common complications. Antihypertensive drugs can reduce intraglomerular pressure by vasodilating the "vas efferens". In addition, ACEIs slow down glomerular hypertrophy.

Other important points of therapy are:

- low-protein diet with possible supplementing with ketoanalogues of essential amino acids,
- treatment of dyslipidemia, most often by administration of statins,
- limiting the intake of salt and potassium in food,
- diuretic therapy with loop diuretics, e.g. furosemide (spironolactone or amiloride can cause hyperkalemia),
- treatment of obesity,
- smoking cessation,
- avoiding non-steroidal antiphlogistics,
- monitoring of uric acid level and possible correction of hyperuricemia,
- monitoring of acid-base balance and possible correction of metabolic acidosis (e.g. $NaHCO_3$ is administered),
- correction of disorders of calcium phosphate metabolism,
- correction of anemia by administration of recombinant erythropoietin.

If the disease continues to progress, from stage G3b it is necessary to administer hepatitis B vaccination and to monitor a sufficient titer of HBsAg antibodies. In addition to this mandatory vaccination, it is also recommended to receive a vaccination against influenza and against pneumococci.

From stage G4, the patient should be regularly monitored by a nephrologist and preparation for RRT (*renal replacement therapy*), i.e. methods to replace kidney function, is in place. These are dialysis methods and kidney transplantation in indicated cases.

Patient preparation for RRT includes:

- patient education,
- creation of an AV shunt for hemodialysis treatment, possibly insertion of a catheter for peritoneal dialysis,
- identification of the living donor,
- performing examinations necessary to be included in the transplant waiting list.

Prognosis

The prognosis depends both on the degree of kidney disease and on the complications that accompany the disease (i.e. anemia, disorders of electrolyte and hormonal balance disorders, cardiovascular complications and others).

The indicative risk assessment based on glomerular filtration and degree of albuminuria is shown in the following table:

Stages of CKD according to KDIGO 2012 [1]

Prognosis of CKD based on GFR and albuminuria				**Albuminuria**		
				A1p	**A2**	**A3**
				normal or slightly elevated	moderately elevated	severely elevated
				< 30 mg/24 hours	30-300 mg/24 hours	> 300 mg/24 hours
GFR (ml/min/1.73 m^2)	**G1**	high or normal	≥ 90	low risk	medium risk	high risk
	G2	slightly reduced	60-89	low risk	medium risk	high risk
	G3a	slightly to moderately reduced	45-59	medium risk	high risk	very high risk
	G3b	moderately to severely reduced	30-44	high risk	very high risk	very high risk
	G4	severely reduced	15-29	very high risk	very high risk	very high risk
	G5	kidney failure	< 15	very high risk	very high risk	very high risk

Links

References

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