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Current Views on the Diagnosis, Treatment and Prevention of Chronic Kidney Disease

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Annotation: Chronic kidney disease (CKD) is a long-term progressive decline in renal function. Symptoms develop slowly and in advanced stages include anorexia, nausea, vomiting, stomatitis, dysgeusia, nicturia, apathy, chronic fatigue, pruritus, decreased mental clarity, muscle convulsions and seizures, fluid retention, hypotrophy, peripheral neuropathies, 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 normalisation of water and electrolyte balance, control of blood pressure, treatment of anaemia, various types of dialysis and kidney transplantation.

Keywords: Chronic kidney disease, prevention, treatment.

Introduction. The prevalence of CKD (defined as an estimated glomerular filtration rate [rCKF] < 60 mL/min/1.73m2 or urine albumin-to-creatinine ratio [ACR] ≥ 30 mg/g) among adults in the United States from 2015 to 2018 was estimated to be 14.4% (1).

Chronic kidney disease can be caused by any cause due to significant impairment of renal function. The most common causes in the United States, in order of incidence, include the following conditions:

- Diabetic nephropathy
- Hypertensive nephrosclerosis

Various primary and secondary glomerulopathies

Metabolic syndrome, characterised by arterial hypertension and type 2 diabetes mellitus, is a frequent cause of kidney damage with an ever-increasing prevalence.

Chronic kidney disease in its early stages is described as reduced renal reserve or renal failure that may progress (development of terminal renal failure). Initially, loss of renal tissue function has almost no obvious pathological manifestations because the remaining tissue is working hard (renal functional adaptation).

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

Creatinine and urea

Blood concentrations of creatinine and urea (which are highly dependent on glomerular filtration) begin to rise hyperbolically as SCF decreases. Initially, these changes are minimal. When the SCF falls below 15 ml/min/1.73 m2 (normal=90 ml/min/1.73 m2), creatinine and urea levels rise and are usually associated with systemic manifestations (uremia). Urea and creatinine levels are not the main symptoms of uremia; they are markers of many other substances (some of which have not yet been identified) that lead to symptoms.

Sodium and water

Despite a decreased CRP, sodium levels and water balance are well maintained by increased fractional excretion of sodium in the urine and a normal thirst response. Thus, plasma sodium concentration is usually normal and hypervolemia is uncommon, only occurring with restricted or excessive sodium or water intake. Heart failure develops as a consequence of sodium and water overload, particularly in patients with reduced cardiac reserve.

Potassium

For substances whose secretion is controlled primarily through distal nephron secretion (e.g., potassium), renal adaptation usually serum levels are within normal limits, as long as renal failure is not advanced or dietary potassium intake is not excessive. Potassium-saving diuretics, angiotensin-converting enzyme inhibitors, beta-blockers, nonsteroidal anti-inflammatory drugs, cyclosporine, tacrolimus, trimethoprim/sulfomethoxazole, pentamidine, or angiotensin II receptor blockers may contribute to elevated plasma potassium levels in patients with less severe renal insufficiency.

Calcium and phosphate

Disorders of calcium, phosphate, parat hormone (PTH), b and vitamin D metabolism and renal osteodystrophy may develop. Reduced renal production of the vitamin calcitriol (1,25(OH)2D, the active vitamin D hormone) leads to hypocalcaemia. Reduced renal excretion of phosphate results in hyperphosphatemia. Secondary hyperparathyroidism is widespread and may develop in renal failure even before the manifestation of abnormalities associated with changes in calcium or phosphate concentrations. Therefore, PTH monitoring is recommended in patients with moderate CKD, even before the onset of hyperphosphatemia.

Renal osteodystrophy (impaired bone mineralisation due to hyperparathyroidism, calcitriol deficiency, elevated serum phosphate levels or low or normal serum calcium levels) usually results in accelerated bone metabolism due to the bone form of hyperparathyroidism (fibrous osteitis), but may also result in suppression of bone metabolism due to adynamic bone disease (with increased suppression of parathyroid function) or osteomalacia. Calcitriol deficiency may cause osteopenia or osteomalacia.

pH and bicarbonate

Moderate metabolic acidosis is characteristic (plasma bicarbonate level is 15-20 mmol/L). Acidosis causes muscle atrophy due to protein catabolism, bone loss due to acid buffering of bones and accelerated progression of kidney disease.

Anaemia

Anaemia is characteristic of moderately severe to advanced (≥ stage 3) CKD. Anaemia in CKD is normochromic-normocytic, with a haematocrit of 20-30% (35-40% in patients with polycystic kidney disease). It is usually caused by a deficiency in erythropoietin production due to decreased functioning renal mass (see Anaemias due to impaired erythropoiesis page). Other causes include iron, folate and vitamin B12 deficiency.

With a moderate decrease in renal reserve, the course is usually asymptomatic. Even patients with mild to moderate renal impairment may not have symptoms of increased blood urea nitrogen (BUN) and creatinine levels. Nicturia is often observed, especially due to inability to

concentrate urine. Apathy, fatigue, lack of appetite and decreased mental clarity are often the earliest manifestations of uremia.

In more severe renal disease (e.g., estimated glomerular filtration rate [rCF] < 15 mL/min/1.73 m2), neuromuscular symptoms may appear, including marked muscle twitching, peripheral sensory and motor neuropathies, muscle cramps, hyperreflexia, restless legs syndrome, and seizures (usually as a result of hypertensive or metabolic encephalopathy).

Anorexia, nausea, vomiting, weight loss, stomatitis and bad taste in the mouth are very common. The skin may be yellow-brown in colour and/or dry. Sometimes urea crystals with sweat are secreted onto the surface of the skin, forming uremic frosting. Itching can cause severe discomfort. Nutritional deficiency leading to generalised tissue loss is a hallmark of chronic uremia.

With severe CKD, pericarditis, peptic ulcer disease and gastrointestinal haemorrhage may develop. Arterial hypertension is present in more than (>) 80% of CKD patients and is usually associated with hypervolaemia. Heart failure caused by arterial hypertension or ischaemic heart disease or sodium and water retention may lead to secondary oedema and/or dyspnoea.

Electrolytes, blood urea nitrogen concentration (BUN), creatinine, phosphates, calcium, general blood count (GBC)

Urinalysis (including microscopy of urine sediment)

Urine protein quantification (24-hour urine protein excretion or protein/creatinine ratio in a single urine sample).

Ultrasound

Sometimes a kidney biopsy

Chronic kidney disease (CKD) is usually first suspected when serum creatinine levels are elevated. The first step is to determine whether the kidney failure is acute, chronic, or acute to chronic (i.e., an acute illness that further impairs kidney function in a patient with CKD-see Table Distinguishing Acute Kidney Injury From Chronic Kidney Disease). The cause of renal failure is also determined. Sometimes determining the duration of renal failure helps to determine the cause; sometimes it is easier to determine the cause than the duration, and finding out the cause helps to determine the duration.

Examination includes urinalysis with urine sediment microscopy, assessment of electrolyte levels, urea nitrogen, creatinine, phosphate, calcium, and a general blood count. Sometimes special serological tests are needed to determine the cause. In the differential diagnosis of acute renal failure and CKD, a history of creatinine elevation or abnormalities in urinalysis is most helpful. Changes in the urinalysis depend on the underlying disease of PU, but large (>3 leucocytes in diameter) and especially waxy cylinders are usually prevalent in any severe renal failure.

Renal ultrasound is usually helpful in identifying obstructive uropathy and differentiating acute renal failure from CKD on the basis of renal size. With the exception of some diseases (see Table Distinguishing between acute kidney injury and chronic kidney disease), CKD is accompanied by shriveled and shrunken kidneys (usually <10 cm in length) with a thin, hyperechogenic cortical layer. Establishing an accurate diagnosis is particularly difficult in the terminal stage of chronic kidney disease. Renal biopsy is the definitive diagnostic tool, but it is not recommended if ultrasound reveals shrunken sclerosed kidneys; the high risk of the procedure outweighs the low diagnostic value.

Prognosis in CKD

The progression of chronic kidney disease (CKD) is mostly determined by the degree of proteinuria. Patients with nephrotic range proteinuria (>3 g/24 h or urine protein/creatinine ratio

>3) usually have a worse prognosis and more rapid progression to renal failure. Progression may develop even if the underlying pathology is not active. Patients with urine protein < 1.5 g/24 h usually have much slower progression, if at all. Arterial hypertension, acidosis, and hyperparathyroidism are also associated with faster progression.

Treatment of CKD

Treatment of the underlying disease

Potent dietary restriction of protein, phosphate, and potassium

Vitamin D supplements

Treatment of anaemia

Treatment of comorbidities (e.g., heart failure, diabetes mellitus, nephrolithiasis, prostatic hypertrophy)

Dose adjustments of all medications as needed

Haemodialysis if there is a significant decrease in glomerular filtration rate (GFR), if signs and symptoms are not sufficiently controlled by medication intervention

Maintenance of sodium bicarbonate levels at normal levels (23-29 mmol/l)

The underlying pathology and risk factors should be controlled. In particular, control of hyperglycaemia in patients with diabetic nephropathy and control of arterial hypertension in all patients significantly slow the deterioration of glomerular filtration.

Some recommendations for hypertension suggest a target BP < 140/90 mmHg, the American Heart Association recommends a value of 130/80, and some authors continue to recommend a value around 125-130/< 80 mmHg. Angiotensin-converting enzyme (ACE) inhibitors and angiotensin II receptor blockers (ARBs) reduce the rate of decline in CRP in patients with most causes of chronic kidney disease (CKD), especially those with proteinuria. There is increasing evidence that, compared with monotherapy, the combination of ACE inhibitors and BPAs increases the rate of complications and does not slow the decline in renal function, although such use of the drugs significantly reduces proteinuria. Sodium-glucose cotransporter-2 (SGLT2) inhibitors slow the progression of proteinuric CKD in patients with or without diabetes, although these drugs are contraindicated in patients with type 1 diabetes.

Physical activity should not be restricted, although fatigue and lassitude usually limit a patient's abilities when they have chronic renal failure.

Cutaneous pruritus may be managed by restricting phosphate intake and phosphate-binding agents if plasma phosphate levels are increased.

Nutrition

Strict protein restriction in renal disease is controversial. However, moderate protein restriction (0.8 g/kg/day) for patients with a calculated CRP (pCRP) < 60 ml/min/1.73 m2 without nephrotic syndrome is safe and easily tolerated by most patients. Some experts recommend protein restriction to 0.6 g/kg/day for diabetic and nondiabetic patients if the glomerular filtration rate is < 25 mL/min/1.73 m2; Many uremic symptoms are markedly attenuated when protein catabolism and urea synthesis are reduced. The rate of progression of CKD may also be reduced. Adequate amounts of carbohydrates and fats are added to meet energy needs and prevent ketoacidosis. Patients who are advised < 0.8 g/kg/day require constant monitoring by a dietitian.

Because dietary restrictions may reduce essential vitamin intake, patients should take water-soluble vitamins. Prescription of retinol or vitamin E is optional. Vitamins D2 (ergocalciferol) or D3 (cholecalciferol) are not prescribed for use on a regular basis, but are administered based on blood levels of vitamin D 25-OH and PTH.

Dyslipidaemia should also be treated. Dietary changes may be helpful in hypertriglyceridaemia. Statins are effective for hypercholesterolaemia. Fibroic acid derivatives (clofibrate, gemfibrozil) may increase the risk of rhabdomyolysis in patients with CKD, especially when given together with statins, whereas ezetimibe (which reduces cholesterol absorption) is relatively safe. Correction of hypercholesterolaemia is intended to reduce the risk of cardiovascular disease, which is increased in patients with CKD (3).

Mineral and bone disorders

Based on the updated 2017 KDIGO clinical guidelines (3), it is recommended to monitor serum calcium levels, phosphate levels, PTH, vitamin D 25-OH, and check alkaline phosphatase activity, starting monitoring from stage 3a of CKD. The frequency of monitoring depends on the severity of CKD, the severity of the above-mentioned abnormalities and the frequency of therapeutic interventions. Bone biopsy is the most accurate procedure to determine the type of renal osteodystrophy.

Hyperphosphatemia should be treated with a

Dietary intake restriction diets to limit phosphate intake

Phosphate-binding medication

Restriction of dietary phosphate intake to an amount of 0.8-1 g per day is usually sufficient to normalise serum phosphate levels in patients with pSKF < 60 ml/min/1.73 m2. Additional phosphate-binding drugs active in the gut (calcium-containing and non-calcium-containing) may be necessary to adequately control hyperphosphatemia, which is associated with an increased risk of cardiovascular disease. Non-calcium phosphate-binding drugs are preferred for use in patients with hypercalcaemia, suspected adynamic bone disease or evidence of vascular calcification on imaging. If calcium-containing phosphate-binding drugs are prescribed, in general dietary and drug sources of calcium should not exceed 2000 mg/day in patients with pSCF < 60 mL/min/1.73 m2.

In vitamin D deficiency, cholecalciferol (vitamin D3) or ergocalciferol (vitamin D2) should be administered to achieve a serum vitamin D 25-OH level of about 30-50 ng/ml until hyperphosphatemia or hypercalcaemia occurs.

The optimal PTH level in patients with stage 3a to 5 CKD and not on dialysis is not known. However, if PTH levels are progressively elevated or significantly elevated (9 times the upper limit of normal at the time of analysis) despite treatment of hyperphosphatemia or vitamin D deficiency, an active vitamin D analogue (e.g., calcitriol) is recommended. The typical starting dose of calcitriol is 0.25 mcg orally 3 times per week, titrated to maintain PTH levels 2-9 times the upper limit of normal for this assay. PTH levels are not corrected to normal levels because doing so increases the risk of adynamic bone disease.

Water and electrolyte metabolism

Restricted fluid intake is only required if serum sodium concentration < 135 mmol/l or in heart failure or significant oedema.

Restriction of sodium intake < 2 g/day is recommended for patients with CKD with a pSCF < 60 mL/min/1.73 m2 who present with hypertension, volume overload or proteinuria.

Potassium restriction is individually tailored based on serum potassium levels, pSCF, dietary preferences, and use of medications that increase potassium levels (e.g., IPFs, BPAs, or potassium-saving diuretics). Potassium restriction is generally not required when pSCF > 30 mL/min/1.73 m2. Treatment of mild to moderate hyperkalemia (5.1 to 6 mmol/L) entails restricting dietary potassium intake (including avoidance of salt substitutes), correcting metabolic acidosis, and using potassium-reducing diuretics and gastrointestinal cation exchangers. Severe hyperkalemia (> 6 mmol/l) requires urgent treatment.

Metabolic acidosis should be treated until serum bicarbonate levels normalise (23-29 mmol/L) to promote complete muscle recovery or slow muscle atrophy, reduce bone loss and progression of CKD. Acidosis can be corrected by administration of oral alkaline sources such as sodium bicarbonate or an alkalising diet (mainly fruit and vegetable). Sodium bicarbonate is administered 1-2 g orally twice daily with gradual increases until the bicarbonate concentration reaches about 23 mmol/L or until sodium overload prevents the possibility of further therapy. If an alkalising diet is used, serum potassium levels are controlled as fruit and vegetables contain potassium.

Anaemia and coagulation disorders

Anaemia is a frequent complication of CKD from moderate to advanced stages (exceeding stage 3), and when < 10 g/dl, is treated with erythropoiesis-stimulating agents (ESAs) such as recombinant human erythropoietin (e.g. epoetin alfa). Due to the risk of cardiovascular complications including stroke, thrombosis and death, the lowest dose of these drugs needed to maintain Hb levels, 10 to 11 g/dl, is used.

Because of the increase in iron utilisation during stimulation of erythropoiesis, it is necessary to replenish iron stores, which often requires the prescription of parenteral iron preparations. The plasma iron content, iron-binding capacity and ferritin concentration should be continuously monitored. The target transferrin saturation level (TSAT), which is calculated by dividing serum iron by total iron-binding capacity and multiplying by 100%, should be greater than 20%. The target ferritin level in patients not on dialysis is > 100 ng/ml. Haemotransfusion should only be administered in cases of severe (haemoglobin < 8 g/dl) or symptomatic anaemia.

Bleeding in CKD usually does not need therapy. If necessary, cryoprecipitate, erythrocyte transfusion, desmopressin at a dose of 0.3-0.4 mcg/kg (20 mcg maximum) in 20 mL isotonic solution intravenously over 20-30 minutes, or combined estrogens at a dose of 2.5-5 mg 1 orally once daily may be effective. The effect of such treatment lasts 12-48 hours, except for combined estrogens, the effect of which may last for several days.

Heart failure

Symptomatic heart failure is treated by

Restriction of sodium intake

Diuretics

In some cases, dialysis

Loop diuretics, such as furosemide, are usually effective even with significantly reduced renal function, although larger doses may be needed. ACE inhibitors (or BRAs) and beta-blockers (carvedilol or metoprolol) should be prescribed for left ventricular failure. Aldosterone receptor antagonists are recommended for patients with advanced stages of heart failure. Digoxin may be used, but the dosage should be reduced depending on the degree of renal function.

Moderate to severe arterial hypertension requires treatment to prevent its adverse effects on cardiac and renal function. Patients who do not respond to sodium restriction (1.5 g/day) should receive diuretic therapy. Loop diuretics (e.g., furosemide 80-240 mg orally twice daily) may be combined with thiazide diuretics (e.g., chlorthalidone 12.5-100 mg orally once daily, hydrochlorothiazide 25-100 mg orally in one to two doses daily, metolazone 2.5-20 mg orally once daily) if hypertension or oedema cannot be controlled. Even in renal failure, the combination of a thiazide diuretic with a loop diuretic is quite potent and should be used with caution to avoid diuresis.

Occasionally, dialysis may be required to control heart failure. If reduction of extracellular fluid volume does not control blood pressure, conventional hypotensive agents are added. With this treatment, azotemia may increase and necessitate adequate control of heart failure and/or arterial hypertension.

Medications

Reduced renal excretion of drugs should be considered in the management of any patient with renal failure. The most common drugs that require dosage revision are penicillins, cephalosporins, aminoglycosides, fluoroquinolones, vancomycin and digoxin. Haemodialysis reduces the plasma concentrations of a small number of drugs that must be maintained after haemodialysis. It is strongly recommended that physicians refer to drug dosing guidelines for renal failure before prescribing them to a vulnerable group of patients (4, 5, 6).

Most experts recommend avoiding the use of NSAIDs in patients with CKD because they can worsen renal function, exacerbate hypertension and cause electrolyte imbalances.

Certain drugs should be completely avoided in patients with chronic kidney disease and pSKF < 60 ml/min/1.73m2. These include nitrofurantoin and phenazopyridine. In the past, the MRI contrast agent gadolinium has been associated with the development of nephrogenic systemic fibrosis in patients with a calculated CRP < 30 ml/min/1.73m2. Recently, class II gadolinium preparations are considered safer and are preferred when gadolinium is indicated in patients with pSFC < 30 or on dialysis (7).

Dialysis

Dialysis usually stimulates the onset of one of the following:

Uraemia symptoms (e.g., anorexia, nausea, vomiting, weight loss, pericarditis, pleurisy)

Poorly controlled hypervolaemia, hyperkalemia or acidosis with medications and lifestyle interventions

These problems usually occur when the SCF reaches ≤ 10 ml/min in a patient without diabetes or ≤ 15 ml/min in a patient with diabetes; patients whose estimated SCF values are approximately equal to these values should be monitored so that these signs and symptoms are recognised as early as possible. It is best to make provision for dialysis, and preparations can be made to avoid emergency haemodialysis catheter placement. Such preparations can usually begin when the patient is in early to mid-stage 4 CKD; preparation allows time for patient education, selection of the type of dialysis, and timely creation of an arteriovenous fistula or insertion of a dialysis catheter.

Begin preparation for dialysis, renal transplantation or palliative care in early to mid-stage 4 CKD to allow sufficient time for patient education and treatment selection, as well as any associated preparatory procedures.

Conclusions: Thus, chronic renal failure is a syndrome resulting from irreversible progressive decline in renal function due to a decrease in the mass of their functioning parenchyma with associated metabolic disorders and the development of pathology of a number of organs and systems.

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