

All needed to know on chronic kidney disease from prevention to treatment

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Chronic kidney disease (CKD) is the progressive loss of kidney function over years resulting in the buildup of wastes, electrolytes, and fluid. It may eventually lead to permanent kidney damage or end-stage kidney disease. The most common etiologies are high blood pressure (hypertension) and diabetes. Other causes include pyelonephritis, polycystic kidney disease, glomerulonephritis, systemic lupus erythematosus (SLE), blockage e. g. from kidney stones, and long-term use of NSAIDs and lithium. This research paper aims at describing the clinical presentations of CKD, explaining how to diagnose it including the role that patient history, physical exams, and diagnostics play in diagnosis. It also explains the implications of treatment options including prescribed drug. It finally describes how patient factors might impact the diagnosis and treatment of a patient with CKD.

The signs and symptoms of CKD include anemia due to reduced synthesis of erythropoietin by the kidney, swollen hands, feet, and ankles due to water retention, breathlessness, insomnia, itchy skin which may be persistent, and muscle cramps. Additionally, patients present with decreased appetite and weight, hypertension, nausea, diarrhea, and vomiting, erectile dysfunction in men and amenorrhea in women, restless leg syndrome, bone pain due to metabolic bone disease, paresthesia due to polyneuropathy (Kumar & Clark, 2012).

In the patient history, the duration of symptoms may help to differentiate between acute and chronic kidney disease. The drug history including NSAID agents, analgesics, and herbal treatments may give a clue if it is drug-induced. Past medical and surgical history may point to diseases such as diabetes and hypertension as the causes. Previous kidney function tests, <https://assignbuster.com/all-needed-to-know-on-chronic-kidney-disease-from-prevention-to-treatment/>

imaging, and urine findings may point to the diagnosis of CKD. A detailed family history may be used to exclude a hereditary kidney disorder (Arici, 2014).

On physical examination, signs of uremia may point to CKD. They include short stature as seen in patients who had CKD in their childhood, manifestations of fluid overload e. g. swollen feet. Scratch marks may point to uremic itching, pallor due to anemia, and brown discoloration of the nails. Moreover, pericardial friction rub and flow murmurs may indicate volume overload and point to CKD. Other physical findings like retinopathy in diabetes, peripheral vascular disease, and cutaneous vasculitis may show underlying conditions that led to CKD. Assessments e. g. skin turgor, blood pressure, and peripheral circulation may also point to CKD (Kumar & Clark, 2012).

Diagnostics also assist in CKD diagnosis. A urinalysis with hematuria, proteinuria, and glycosuria may suggest CKD. Radiological tests e. g. MRI, CT, and ultrasound show changes that occur in CKD. Biochemical tests may show elevations in urea and creatinine and suggest CKD. The creatinine clearance shows the severity of CKD. Urine microscopy with red cells and casts can indicate CKD. Hematological tests and immunological tests may be performed to indicate CKD (Kumar & Clark, 2012).

Various treatment options are available for CKD. ACE inhibitors e. g. captopril and enalapril lower blood pressure and preserve kidney function. Side effects (S. E) include a persistent dry cough, fatigue, headaches, and dizziness.

ARBs are an alternative and include losartan and candesartan. S. E include

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dizziness, stuffy nose, nausea, and vomiting. Statins are drugs that lower cholesterol levels e. g. atorvastatin and simvastatin. S. E comprise constipation, diarrhea, abdominal pain, headaches, pain, tenderness, and weakness(Katzung, Masters & Trevor, 2009). Erythropoietin injections help to eliminate anemia resulting from CKD. Examples include epoetin and darbepoetin. S. E comprise aggravation of hypertension, influenza-like symptoms, increased platelet count, hyperkalemia, and skin reactions. Diuretics e. g. furosemide and hydrochlorothiazide reduce edema associated with CKD. Their S. E include muscle cramps, headache, thirst, raised blood sugar, skin rash, and joint pain. Other treatments include calcium and vitamin D supplements to protect bones, lifestyle changes such as stopping smoking, low-fat diet, reduced salt intake, and exercise can help control CKD(Daugirdas, 2012).

Genes e. g. APOL1, UMOD, MMP20, and SHROOM3 are associated with CKD. Inheriting them increases the risk for CKD development. Women are more prone to CKD than men because estrogen and some estrogen receptors increase the expression of fibrotic and inflammatory pathways involved in kidney injury. African-Americans, American Indians, Hispanics, and people from South Asia have are more prone to CKD because of high rates of diabetes and hypertension (Bomback & Bakris, 2010). Those aged above 65 are more likely to develop CKD due to reduced GFR. Additionally, smoking damages arteries and causes high blood pressure and increases the risk of CKD. Understanding the risk factors helps to know the pathogenesis of the condition and can assist to diagnose and facilitate treatment(Davison, 2005)