The Importance of Timely Detection of Risk Factors and Complications in Chronic Kidney Disease in Mexico

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Chronic kidney disease is considered a pathology of multiple etiologies that impairs the patient's overall physical condition by failing to adequately perform its function of filtering, absorbing, and excreting substances entering the body. That is why this disease has been well defined within the healthcare sector for prompt prevention, diagnosis, and treatment.

Chronic kidney disease is defined as structural or functional kidney damage evidenced by diagnostic blood tests (albumin/creatinine ≥ 30 mg/g or albuminuria ≥ 30 mg/g/24 h), urine tests (abnormal urinary sediment such as hematuria, leukocyturia, casts, or tubular epithelial cells), or a period of three months or more accompanied by a glomerular filtration rate (GFR) below 60 mL/min/1.73 m²; it is classified according to the degree of renal function, with both GFR and albuminuria used to stage the disease.

In Mexico, 9,184.9 out of every 100,000 inhabitants suffer from this condition. At its highest concentration within the population, it involves common risk factors that can trigger the onset of the disease. It is estimated that 40% of diabetics are susceptible to developing this condition, in addition to the large proportion of the population who already have other risk factors. As a consequence of impaired renal function, the disease can develop, along with various long-term systemic complications.

Risk factors allow the identification of patients who are more susceptible to the disease. Type 2 diabetes mellitus, arterial hypertension, obesity, dyslipidemia, and hyperuricemia are among the most important factors in our environment; their identification is essential for proper prevention.

Diabetes causes kidney damage through hyperfiltration, which generates advanced glycation end products and/or reactive oxygen species. Among the molecules involved are various cytokines, growth factors, and hormones, which contribute to changes associated with diabetic nephropathy at the glomerular, interstitial, and vascular levels. In the first phase, also known as the preclinical phase, damage is mediated by hyperglycemia, which triggers thickening of the glomerular basement membrane, increased mesangial matrix, vasoconstriction, arterial hyalinosis, and tubulointerstitial fibrosis; these changes result in albuminuria, increased proteinuria, and a progressive decline in glomerular filtration rate.

Arterial hypertension is one of the most common causes (60–80%). Elevated blood pressure causes vasoconstriction of the afferent arteriole as a protective mechanism for the glomerulus; however, this results in glomerular ischemia.

Subsequently, progressive damage occurs with loss of renal mass, and adaptive mechanisms emerge to compensate for the loss of renal function, such as the disappearance of preglomerular vasoconstriction, which increases pressure in the glomerular capillaries, leading to hypertrophy of the intact nephrons and hyperfiltration. This entire set of events leads to the development of glomerular sclerosis and proteinuria.

As in many other diseases, obesity predisposes to systemic damage by inducing hemodynamic changes that lead to hyperfiltration, as well as increasing vasoactive and fibrogenic substances, such as angiotensin II, insulin, leptin, TGF-β, VEGF, and fibronectin. Likewise, the predisposition to proteinuria is greater in this condition, and focal segmental glomerulosclerosis (FSGS) is often a common finding.

Dyslipidemia is a significant risk factor in chronic kidney disease, characterized by normal or slightly elevated LDL cholesterol levels, low HDL cholesterol levels, and elevated triglyceride levels. LDL particles are more atherogenic due to the oxidation and carbamylation they undergo through processes associated with uremia.

The presentation of hyperuricemia in chronic kidney disease (CKD) is characterized by decreased urinary excretion of uric acid, which secondarily leads to reduced renal perfusion due to smooth muscle proliferation in the afferent arteriole. As a consequence of these pathophysiological events, patients are at increased risk of hypertension, development of diabetic nephropathy, and cardiovascular morbidity and mortality, which can accelerate the progression of CKD.

LAnother aspect to consider is the complications associated with this condition: anemia, electrolyte and bone-mineral metabolism disturbances, as well as an increased predisposition to cardiovascular risk.

Their importance lies in the significant impact they have on patients' quality of life, coupled with the increased difficulty in covering necessary expenses, such as the rising cost of medications, the use of hemodialysis or dialysis, and the consideration of a kidney transplant.

Anemia is one of the most common complications in these patients, resulting from a deficiency of erythropoietin, a hormone produced in the kidneys that stimulates erythropoiesis. Anemia is evaluated by measuring ferritin levels below 100 mg/mL and/or transferrin saturation index levels below 20%.

Evidence of electrolyte disturbances in patients with chronic kidney disease is generally less frequent; however, it is often significant due to the presence of metabolic acidosis—secondary to the kidney's reduced ability to excrete non-volatile acids—along with low plasma bicarbonate concentrations.

Although each nephron can increase ammonium synthesis, the reduction in nephron number fails to compensate; therefore, the net fraction of acid excreted as ammonium decreases, leading to elevated tissue ammonium levels with consequent activation of the alternative complement pathway, stimulation of the renin–angiotensin–aldosterone system, and increased endothelin- 1 levels, resulting in greater interstitial inflammation and,

subsequently, more extensive renal tissue fibrosis, which is associated with accelerated disease progression.

Decreased renal function is accompanied by reduced phosphorus excretion due to its deposition in the renal tubules and interstitium, as well as by a deficiency in vitamin D (calcitriol) production, which stimulates the secretion of parathyroid hormone (PTH), leading to secondary hyperparathyroidism with a rapid increase in the synthesis of components of the renin–angiotensin system.

The association between increased cardiovascular risk and chronic kidney disease should not be overlooked, as higher all-cause and cardiovascular mortality has been demonstrated in the population with a glomerular filtration rate < 60 mL/min/1.73 m². This promotes sodium and water retention, equivalent to a volume overload, which is accompanied by activation of the renin–angiotensin–aldosterone system and the sympathetic nervous system, thereby increasing vascular pressure.

Categoría según filtrado glomerular (mL/min/1,73 m²)	Categoría según albuminuria (mg/g)		
	A1 (< 30)	A2 (30-299)	A3 (≥ 300)
G1 (>90)			
G2 (60-89)			
G3a (45-59)			
G3b (30-44)			
G4 (15-29)			
G5 (<15)			

C.2.1. Green: low risk; yellow: moderate risk; orange: high risk; red: very high risk. Adapted from the KDIGO 2012 guidelines.

EIt is not unusual to find a relationship between urea and kidney damage, as the presence of urea and other electrolytes can trigger arrhythmias, especially supraventricular and ventricular arrhythmias, which are most commonly associated with hemodialysis sessions. The increase in cytokines at the vascular level predisposes cardiomyocytes to apoptosis and necrosis, accompanied by the aforementioned complications such as anemia. In the final stages of the disease (stages 4 and 5), with the presence of uremic toxins, left ventricular hypertrophy secondary to volume and pressure overload is commonly observed, with an increased predisposition to the development of congestive heart failure, ischemic heart disease, intradialytic hypotension, and arrhythmias.

It is important to highlight the onset of atherosclerosis, caused by increased pulse pressure combined with an inflammatory process that leads to atheromatous plaques in the intima of medium- and large-sized arteries, along with an occlusive syndrome at various levels that can progress to coronary artery disease, cerebrovascular disease, peripheral vascular disease, and aortic atherosclerosis. In addition, another finding to consider is albuminuria ≥ 30 mg/g, which is associated with an increased risk of mortality.