

## Chapter 2

# Definition and Characteristics of Hypertension Associated with Chronic Kidney Disease: Epidemiological Data

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### Introduction

The prevalence of hypertension appears to be around 30–45% of the general population and it is increasing with age [1]. The kidneys play such a vital role in long-term blood pressure [2]. Chronic kidney disease (CKD) is one of the most common causes of secondary hypertension. The prevalence of hypertension is higher among patients with CKD than in general population, and its frequency increases progressively with the severity of CKD [2–4]. According to US Renal Data System Annual Data Report of 2010, hypertension occurs in 23.3% of individuals without CKD, while in 35.8% of patients with CKD stage 1, in 48.1% with stage 2, in 59.9% with stage 3, and in 84.1% with CKD stages 4–5 [5]. However, the frequency of hypertension may vary in different CKD causes including renal artery stenosis (93%), diabetic nephropathy (87%), and polycystic kidney disease (74%) [2, 6]. The pathogenesis of hypertension associated with chronic kidney disease (CKD) is complex and multifactorial [7, 8]. Numerous studies confirmed the association between renal defects and essential hypertension in humans. As early as in 1983, Curtis et al. [9] demonstrated a remission of essential hypertension after renal transplantation from

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normotensive donors. Moreover, Widgren et al. [10] study revealed that salt loading in normotensive individuals with family history of hypertension is associated with lower natriuresis and higher blood pressure than in those with no family history. Additionally, the autopsy of hypertensive victims of fatal accidents demonstrated decreased amount of nephrons [11]. It is estimated that half of patients with chronic kidney disease die of cardiovascular causes before they reach end-stage renal disease.

The pathogenesis of hypertension in chronic kidney disease is multifactorial and can be associated with diabetic nephropathy, glomerulonephritis, nephropathy in the course of connective tissue disorders, vasculitis, pyelonephritis, and obstructive, analgesic, and reflux nephropathy as well as congenital diseases such as polycystic kidney [12]. It is estimated that only 5–10% of all cases of hypertension is associated with secondary causes. Renal parenchymal hypertension is present in 5–6% of cases of secondary hypertension, while renovascular hypertension is diagnosed in 1% of cases. Simple screening for secondary forms of hypertension should comprise the analysis of clinical history (renal disease, urinary tract infection, hematuria, analgesic abuse) and family history of renal disease, physical examination, and routine laboratory tests [13]. The presence of secondary hypertension is suggested by sudden onset of hypertension, severe increase in blood pressure, and problems to lower blood pressure with the use of drug therapy [13]. It has been believed that hypertension in CKD is associated with excessive intravascular volume or excessive activation of the renin–angiotensin system due to sodium/volume imbalance (renin-dependent hypertension) [14–16]. Recently, the role of the following factors has been confirmed: enhanced activity of sympathetic nervous system sodium and potassium retention, disorders of divalent ion metabolism, disturbances in parathyroid hormone (PTH) secretion, decreased amount of endothelium-related dilating factors accompanied by the increase in vasoconstrictive factors (endothelin), baroreceptors dysfunction, oxidative stress, structural changes of the arteries, renal ischemia, and sleep apnea in the development of hypertension in chronic kidney disease [12, 14]. Moreover, it has been suggested that also iatrogenic factors, such as erythropoietin, cyclosporine, steroids, divalent ions, and vitamin D, sympathomimetic agents, and nonsteroidal anti-inflammatory drugs (NSAIDs) may influence the onset and progression of hypertension in CKD [14].

## Diagnosis of Hypertension and Chronic Kidney Disease

According to the European Society of Hypertension (ESH) and of the European Society of Cardiology (ESC), the distinction between normotension and hypertension on the basis of cutoff BP values is difficult due to the continuous association between BP and CV and renal events [1]. However, in practice the cutoff BP values are used to simplify the diagnostic approach and to facilitate the decision about treatment. The recommended definition of hypertension remained the same as 2003 and 2007 ESH/ESC guidelines. According to them hypertension is diagnosed when

**Table 2.1** Stages of kidney disease

Stage	GFR [mL/min/1.73 m <sup>2</sup> ]	Description
1	> 90	Normal kidney function; urine tests results, structural abnormalities, or genetic conditioning suggest kidney disease
2	60–89	Mild reduction in kidney function; urine tests results, structural abnormalities, or genetic conditioning suggest kidney disease
3A	45–59	Moderate reduction in kidney function
3B	30–44	
4	15–29	Severe reduction in kidney function
5	<15 or on dialysis	Very severe or end-stage renal disease (ESRD)

Adapted from [18]

systolic blood pressure (SBP) values  $\geq 140$  mmHg and/or diastolic blood pressure (DPD) values  $\geq 90$  mmHg. The same classification is used in young, middle-aged, and elderly subjects [1]. Moreover, according to the Guidelines of Polish Society of Hypertension (2015), the diagnosis of hypertension in patients with BP values below 160/100 mmHg should be confirmed by ambulatory blood pressure monitoring (ABPM) or by home BP measurements. In the case of patients with BP values  $\geq 180/\geq 110$  mmHg, the diagnosis of hypertension can be made during the first visit after the exclusion of influence of factors leading to acute BP elevation, such as anxiety, pain, or alcohol intake [17]. 2013 ESH/ESC guidelines for the management of arterial hypertension comprises also the grading of hypertension. High normal blood pressure is diagnosed in patients with a systolic BP of 130–139 mmHg and/or a diastolic BP of 85–89 mmHg, grade 1 hypertension - in persons with a BP of 140–159 and/or 90–99 mmHg, grade 2 hypertension - in those with BP 160–179 and/or 100–109 mmHg, grade 3 hypertension - in persons with BP  $\geq 180$  and/or  $\geq 110$  mmHg, and isolated systolic hypertension - in individuals with BP  $\geq 140$  and  $< 90$  mmHg.

Chronic kidney disease is classified using estimated glomerular filtration rate (eGFR) calculated by abbreviated “modification of diet in renal disease” (MDRD) formula, Cockcroft–Gault formula, or Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) formula [1]. The stages of renal disease are presented in Table 2.1.

## Hypertension in Chronic Kidney Disease

Hypertension in chronic kidney disease is primarily associated with sodium retention. Hypervolemia associated with the disturbances with sodium and water excretion with urine results in increase in blood pressure in order to enhance excretion to maintain isovolemia. Kidney ischemia related to renal fibrosis and scarring occurring in CKD patients results in the increase in renin–angiotensin–aldosterone

system activity and elevations in blood pressure. Also secondary hyperparathyroidism leading to the increase in intracellular calcium concentration is associated with vasoconstriction and hypertension [12, 19, 20].

## Diabetic Nephropathy

Hypertension is common among patients with diabetes mellitus (DM1 and DM2), and its prevalence in these groups of patients is twice as high as in general population. According to studies, high blood pressure correlates with the presence of diabetic nephropathy [12]. Diabetic nephropathy, being one of the chronic complications of diabetes of microangiopathic nature, is defined as a condition characterized by the presence of proteinuria, elevated arterial BP, and diminished GFR. Hypertension is present in 15–25% of patients with microalbuminuria and even in 75–85% with diabetic nephropathy, but the prevalence of HA in diabetes varies across different ethnic, racial, and social groups. Results of other studies demonstrated that the incidence of hypertension in diabetic nephropathy increased with worsening kidney function, reaching 90% in ESRD patients [21].

In patients with diabetic nephropathy, hypertension is defined as systolic blood pressure  $\geq 130$  mmHg or a diastolic blood pressure  $\geq 80$  mmHg [21]. Diabetic nephropathy, characterized by albuminuria, glomerulosclerosis, and decline in glomerular filtration rate (GFR), is the most common cause of hypertension in patients with type 1 diabetes. According to Lago et al. [22], in patients with type 2 diabetes, hypertension occurs mainly without abnormal renal function and is frequently associated with central obesity. In the early stages of diabetic nephropathy, the increase of mesangium and the thickening of the glomerular basement membrane occur due to the accumulation of extracellular matrix, which in consequence leads to the hypertrophy and glomerulosclerosis [23]. Diabetic nephropathy is diagnosed on the basis of the presence of albuminuria  $>300$  mg/d, coexistence of diabetic retinopathy, and lack of clinical or laboratory evidence of renal and urinary tract disease [23]. The activation of local (renal) RAAS, hyperinsulinemia, overhydration, arterial stiffness as well as obesity, endothelium dysfunction, autonomic nervous system disturbances, oxidative stress, and abnormal NO metabolism are the risk factors for hypertension in diabetic nephropathy. Volume expansion due to increased renal sodium reabsorption and peripheral vasoconstriction are the main reasons for hypertension in diabetes [21]. The activation of RAAS, elevated concentration of endothelin-1, decreased level of nitric oxide, and increased oxidative stress result in the development of hypertension and accelerate kidney disease due to the stimulation of vasoconstriction in vascular smooth muscle cells (VSMC); induction of aldosterone released from the adrenal cortex; enhancement of production of superoxide by activation of NADPH oxidase in the systemic vasculature, heart, and kidney; and augmented sodium reabsorption at the renal proximal tubule

[21]. Increased oxidative stress associated with hyperglycemia and the presence of mediators of both RAAS and endothelial dysfunction contributes to hypertension-enhanced vasoconstriction. As it was mentioned above, also increased activity of sympathetic nervous system (SNS) plays an important role in the pathomechanism of hypertension in patients with diabetic nephropathy. Results of studies suggest that insulin resistance may pose a possible link between SNS activation and hypertension. In diabetic nephropathy, autoregulatory functions of the afferent arteriole responsible for maintaining constant glomerular pressures despite variations in systemic blood pressure are impaired, and thus elevated systemic blood pressure is directly transmitted to the renal microvasculature and glomeruli leading to glomerular hypertension and activation of local mediators that induce inflammation, fibrosis, and further injury [21].

## Glomerulonephritis and Vasculitis

Systemic vasculitis is characterized by the presence of inflammatory infiltrates and necrosis within arterial walls. Changes in large and medium renal vessels result in organ ischemia and the development of hypertension [23]. Patients with glomerulonephritis tend to accumulate fluids due to enhanced sodium retention which in consequence results in volume overload and blood pressure increase. In these patients also the suppression of renin–angiotensin system and the increase in atrial natriuretic peptide (ANP) release are observed. The prevalence of hypertension in glomerulonephritis is various and depends on the type of disease [24]. According to studies, hypertension occurs most frequently in patients with membranoproliferative GN (57%), rapidly progressive GN (52%), and endocapillary (acute) GN of poststreptococcal origin (51%), while less frequently in patients with focal sclerosis GN (34%), mesangioproliferative GN (34%), and perimembranous GN (30%). Symptoms of hypertension are aggravated in advanced glomerulonephritis; however, elevated blood pressure is also seen in patients with creatinine concentration within normal range [12]. Mechanisms of hypertension development in acute glomerulonephritis comprise sodium and water retention due to glomerular lesions [24] as well as renin–angiotensin–aldosterone system activation resulting from suppression inadequate to the degree of sodium and water retention. According to studies, in chronic GN with minimal glomerular alterations, the development of hypertension may be preceded by vascular changes [24]. It seems interesting that elevated blood pressure is observed even in patients with confirmed complete recovery from this disease [24].

Clinical symptoms of immunologically caused vasculitis, depending on its severity and type of organ involved, comprise arterial hypertension, hemoptysis, arthralgia, muscle pain, palpable purpura, hematuria, proteinuria, and renal failure [25]. In patients with vasculitis, hypertension is mainly associated with renal ischemia accompanied by the activation of renin–angiotensin–aldosterone system.

## Renovascular Hypertension

Ischemia of renal parenchyma associated with renal artery stenosis is the cause of renovascular hypertension. The stenosis of renal artery due to atherosclerosis (75%; mainly elderly population) or fibromuscular dysplasia (25%; most common in young adults) is the cause of 95% of renovascular hypertension. It is believed that atherosclerotic renovascular disease is associated with hastened and more severe target organ injury than essential hypertension [7]. According to Medicare studies in patients with newly identified renovascular disease, the rate of cardiovascular event (including coronary events, myocardial infarction, and heart failure) development is higher than in those without renovascular disease [7].

Characteristic features of renovascular hypertension comprise sudden onset of disease, lack of hypertension risk factors and obesity, lack of family history, high values of blood pressure ( $>160/100$  mmHg) resistant to the treatment with three hypotensive drugs including diuretic, sudden raise in blood pressure in people with well-controlled hypertension, malicious course of disease with signs of organ damage, sudden increase in creatinine level ( $>30\%$  above the baseline level) following the ACE or sartan treatment, recurrent episodes of pulmonary edema or heart failure with unknown etiology, and the presence of asymmetric or cirrhotic kidney as well as general atherosclerosis. The symptoms of renal artery stenosis include the presence of abdominal bruit with lateralization, hypokalemia, polyglobulia, and progressive decline in renal function [13]. Occlusion in renal artery reducing renal perfusion pressure intensifies sodium retention by slowing blood flow and filtration and increasing peritubular forces resulting in solute reabsorption. Sodium retention is further enhanced by the activation of the renin–angiotensin–aldosterone system. Angiotensin II directly increases sodium transport, while aldosterone stimulates distal sodium retention through the activation of sodium–potassium ATPase resulting in the diminished sodium excretion in the post-stenotic kidney and in consequence to hypertension [7, 26]. Moreover, angiotensin II promotes the hypertrophy of both vascular smooth muscle cells and heart [23]. It also enhances oxidative stress further aggravating imbalance between vasoconstrictive and vasodilatory substances and endothelial dysfunction. Decrease in renal perfusion is also associated with overproduction of renin by juxtaglomerular apparatus, which in consequence leads to the constriction of afferent arteriole and increased sodium reabsorption. High concentration of renin in one kidney hampers its secretion by the second kidney [27].

Renovascular hypertension diagnosis is made on the basis of the demonstration of structural and functional occlusion of the renal vessels. Ultrasound determination of the longitudinal diameter of the kidney is used as a screening procedure. Color Doppler sonography with calculation of peak systolic velocity and resistance indices, MR angiography, CT angiography, or intra-arterial angiography is utilized for the visualization of renovascular lesions. The difference of over 1.5 cm in length between the two kidneys is usually the confirmation of renal artery stenosis. However, this abnormality is present in only 60–70% of such patients, and thus

color Doppler sonography or spiral computed tomography with iodine-containing contrast media is used to detect stenoses [13, 28]. According to Vasbinder et al. [29], the analysis of renal vasculature with the use of breath-hold three-dimensional, gadolinium-enhanced magnetic resonance angiography with sensitivity of 95% will be the diagnostic tool of the future.

## Polycystic Kidney Disease

Autosomal dominant polycystic kidney disease (ADPKD) is a systemic, hereditary kidney disease. Hypertension occurs early in the course of ADPKD (between the age of 30 and 34) and is associated with increased patient morbidity and mortality and the progression to ESRD [30]. Arterial hypertension is one of the main symptoms of polycystic kidney disease and is observed in 59–79% of patients with various stages of this disease. Results from large ADPKD registry demonstrated that in children with autosomal dominant polycystic kidney disease, blood pressure was higher by 4–6 mmHg in comparison to unaffected age- and gender-matched controls [30, 31]. Moreover, in ADPKD children with hypertension, greater kidney volume and increased number of cysts were observed in comparison to age-matched normotensive ADPKD children [30, 31]. In hypertensive adults with ADPKD, greater LVMI in comparison to matched essential hypertensive men was observed, and it has been found that both LVMI and left ventricular hypertrophy aggravate along with the progression of kidney disease toward renal failure [30]. Early diastolic dysfunction has been demonstrated in this group of patients [32]. Impaired endothelium-dependent relaxation in small resistance vessels was observed in young normotensive patients. Along with the progression of disease, intima-media thickness of carotid arteries increases, and fibromatous areas in carotid walls and important alterations in large arteries appear [32]. Moreover, in hypertensive ADPKD patients, sclerosis of renal arterioles and global glomerulosclerosis is observed. Analysis of renal specimens demonstrated advanced sclerosis of preglomerular vessels, interstitial fibrosis, and tubular atrophy even in patients with normal renal function or early renal failure [33]. The prevalence of target organ damage is also higher in hypertensive ADPKD than in other age-matched hypertensive patients [32]. Greater albuminuria in ADPKD is associated with higher mean blood pressure as well as severe renal cystic development. However, in ADPKD patients, glomerular filtration rate for a long time does not seem to be affected by the progression of renal structural abnormalities due to compensatory hyperfiltration [32]. Numerous studies demonstrated higher rate of increase in kidney volume, enhanced proteinuria, and decreased renal blood flow in hypertensive ADPKD patients with normal renal function in comparison to normotensive patients [30, 34, 35]. Reduced renal blood flow resulting from renal cysts enlargement and concomitant compression of renal vasculature leading to intra renal ischemia, reduction of renal vasculature, and intrarenal activation of the renin-angiotensin-aldosterone system (RAAS) is a characteristic feature of hypertension in ADPKD [30]. It has been suggested



that the activation of renin–angiotensin–aldosterone system plays a role in the association between hypertension and increased kidney volume. This hypothesis was confirmed by the observation of the increase in both renin activity and plasma levels of aldosterone in ADPKD patients in comparison to age-, sex-, and kidney function-matched patients with essential hypertensive [32]. Local activation of RAAS leading to hyperplasia of the juxtaglomerular apparatus has also been demonstrated. Results of studies suggest that RAAS inhibition may prove beneficial in the control of blood pressure level, simultaneously limiting renal cyst growth and renal enlargement as well as slowing down the progression to ESRD [32]. Increased concentration of erythropoietin (due to intrarenal ischemia/hypoxia) is another factor involved in the development of hypertension in ADPKD. Moreover, intrarenal ischemia influences renal tubular sodium handling and enhances sympathetic nervous system activity. Hypertension in ADPKD patients may be also associated with the imbalance between vasoconstrictor and vasodilatation factors. High levels of circulating vasopressin and endothelin-1 and diminished activity of nitric oxide synthase are observed in this group of patients [32].

## **Analgesic Nephropathy**

The abuse of painkillers may result in the damage of parenchyma and the development of interstitial nephritis. According to the National Kidney Foundation, analgesic nephropathy (AN) is defined as “a disease resulting from the habitual consumption over several years of a mixture containing at least two anti-pyretic analgesics and usually codeine or caffeine” [36, 37]. Among the main symptoms of analgesic nephropathy, there are arterial hypertension and renal failure. Progressive kidney failure is related to kidney papillary necrosis and chronic interstitial nephritis. Earliest changes in kidneys comprise sclerosis of vasa recta capillaries and patchy tubular necrosis, and they are followed by papillary necrosis and secondary focal segmental glomerulosclerosis, cortical scarring, and interstitial fibrosis [37]. The pathogenesis of hypertension in AN has not been fully elucidated. It seems that the decreased production of vasodilatory substances within renal papilla and sodium and water retention due to the hampering of vasodilatory prostaglandins and bradykinins secretion may play an important role in the development of hypertension [23].

## **Hypertension in End-Stage Renal Disease Patients**

Hypertension is diagnosed in 50–90% of hemodialysis patients and only in 30% of those on peritoneal dialysis. There are no recommendations concerning the optimal blood pressure values for dialysis patients. Among hypertension risk factors in



dialysis patients, there are decreased excretion of sodium and water, increased concentration of endothelin, vessel calcification, and overhydration [12]. During hemodialysis, hypertension occurs less frequently due to the better control of volemia than in patients with end-stage renal disease. Among the risks of hypertension in hemodialysis patients, there are overhydration, decreased secretion of sodium and water, increased level of vasoconstrictive endothelin-1, and vessel calcification [38, 39]. Overhydration present in hemodialysis patients negatively influences cardiac output and peripheral resistance. It was shown that lowering of sodium concentration in dialysate, removal of excess water, and the achievement of dry weight can improve interdialytic BP, reduce pulse pressure, and limit hospitalizations. According to the National Kidney Foundation/Disease Outcomes Quality Initiative (NKF/DOQI), optimal blood pressure for dialysis patient should be 135/90 mmHg during day and 120/80 mmHg at night [40].

The use of erythropoietin in end-stage renal disease patients is also associated with the possibility of hypertension development. The exact mechanism of blood pressure increase in response to erythropoietin in patients with chronic uremia is complex and not fully explained. According to studies, increase in systolic and diastolic BP was an average approximately 5–8 mmHg in SBP and 4–6 mmHg in DBP. The incidence of hypertension is Epo dose-dependent. It was demonstrated that the administration of 40, 80, and 120 U/kg of Epo, three times a week for 49 weeks, was associated with hypertension in 28%, 32%, and 56% of treated subjects, respectively [41]. Erythropoietin may increase blood pressure due to its direct vasoconstrictive and mitogenic effects and enhancement of blood viscosity [23]. Clinical studies results suggest that Epo-induced hypertension may be associated with its effect on red blood cell mass and viscosity. Moreover, erythropoietin stimulates both the release of endothelin-1 and enhanced mitogenic response in endothelial cells. Additionally, Epo inhibits extrarenal eNOS/NO production and impairs both NO action and vasodilatory response to endothelial NO. Erythropoietin also enhances adrenergic sensitivity. It has been demonstrated that in hemodialysis patients, angiotensin II infusion during Epo treatment was associated with higher elevation of blood pressure in comparison to pre-Epo condition [41].

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