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Arterial hypertension and the kidney: The fatal duo of chronic noncommunicable diseases

ABSTRACT

Chronic noncommunicable diseases are the leading cause of death in the world. Among them, cardiovascular diseases occupy first place in mortality and the main risk factor is hypertension, which is usually asymptomatic until the harmful effects manifest themselves. There is a strong relationship between hypertension and the kidney, since on the one hand hypertension is the major risk factor for the initiation and progression of kidney disease and at the same time hypertension is the result of kidney disease itself. In this review, different aspects of arterial hypertension were analyzed in a general way, such as prevalence, classification, etiology, heritability and relationship with chronic noncommunicable diseases and the importance of the kidney in the regulation of blood pressure; as well as renal disease and its relationship with hypertension and the deleterious consequences for health.

Keywords: hypertension; kidney; chronic kidney disease; chronic noncommunicable diseases.

INTRODUCTION

Chronic noncommunicable diseases are conditions of slow evolution and prolonged duration that lead to bodily deterioration (Barba Evia, 2018). Among them are cancer, diabetes, respiratory, renal and cardiovascular conditions. According to the World Health Organization, in 2016 70% of deaths worldwide corresponded to chronic noncommunicable diseases (Global Burden of Disease Study 2016 Risk Factors Collaborators, 2017). Hypertension is one of the seven disorders that make up the entity known as cardiovascular diseases (CVD). Epidemiological data have shown that hypertension is the main risk factor for cardiovascular disease (Perumareddi, 2019). In Mexico, the prevalence of arterial hypertension is 25.5% (Campos Nonato, Hernández-Barrera, Pedroza-Tobías, Medina, & Barquera, 2018).

Chronic kidney disease (CKD) is related to several chronic noncommunicable conditions, mainly arterial hypertension. In Mexico, the epidemiological data on morbidity and mortality for CKD are very alarming (Méndez-Durán, Méndez-Bueno, Tapia-Yáñez, Muñoz-Montes, & Aguilar-Sánche z, 2010). A close relationship between hypertension and CKD has been demonstrated, since the former is the main risk factor for the onset and progression of CKD and, at the same time, hypertension may be the outcome of CKD. Therefore, in this review we will analyze in a general way aspects of arterial hypertension, such as its prevalence, classification, etiology, heritability and relationship with chronic noncommunicable diseases and the importance of the kidney in the regulation of blood pressure; as well as chronic kidney disease and its relationship with hypertension and the deleterious consequences for health.

Arterial hypertension

When there is an imbalance between vasoconstrictor and vasodilator factors in the body, hypertension occurs, which consists of a sustained and chronic elevation of blood pressure due to various factors and is characterized by an increase in systolic pressure >140 mm Hg and diastolic pressure >90 mm Hg (Kapil &Lobo, 2014; SSA, 2012). Environmental, genetic and epigenetic factors are involved in this imbalance. The latter are the connection whereby environmental factors (diet) directly intervene with genes and regulate their expression (Poulter, Prabhakaran, & Caulfield, 2015).

Blood pressure and hypertension can be classified according to blood pressure levels (Table 1) (Kapil & Lobo, 2014; SSA, 2012).

There is also the term prehypertension, named by the Seventh Report of the Seventh Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure, to define a group of people at increased risk for CVD who had blood pressure readings not considered by physicians to be - significant. The defined blood pressure range for prehypertension is 120-139 mmHg systolic/80-89 mmHg diastolic (Kalaitzidis & Bakris, 2010).

Table 1

Blood pressure classification. Definition of the different categories of blood pressure according to systolic and diastolic pressure levels (Kapil & Lob◦, 2014; SSA, 2012).

Category	Systolic Pressure (mmHg)	Diastolic Pressure (mmHg)
Optimum	<120	<80
Normal blood pressure	120 a 129	80 a 84
High normal blood pressure	130 a 139	85 a 89
Grade 1 hypertension	140 a 159	90 a 99
Grade 2 hypertension	160 a 179	100 a 109
Grade 3 hypertension	>180	≥110
Masked hypertension	≤140	≤90

Note: Own elaboration.

It is known that the relationship between blood pressure level and CVD risk events are continuous, consistent, and independent of other risk factors. Observational studies with more than 1 million people indicate that death from ischemia, heart disease, and stroke increase linearly from blood pressure levels as low as 115 mmHg systolic and 75 mmHg diastolic upwards. Epidemiological studies also support the hypothesis that in the relationship between blood pressure range and CKD risk, the progression is linear (Kalaitzidis & Bakris, 2010). Epidemiological studies for prehypertension are associated with an intermediate level of CVD risk, higher than for normotensive patients, but lower than for patients with grade 1 hypertension (Gu, Burt, Paulose-Ram, Yoon, & Gillum, 2008).

Etiology of arterial hypertension

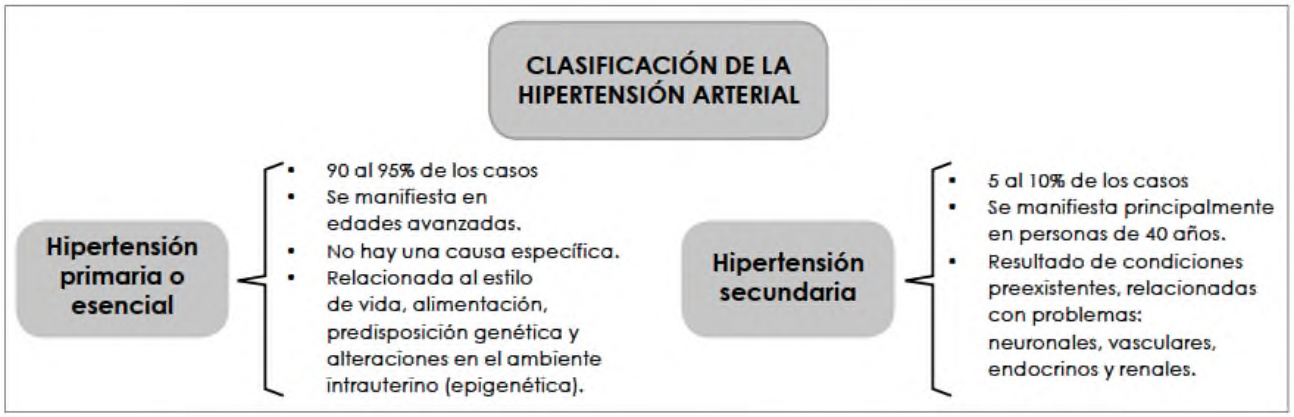
According to its etiology, hypertension can be classified into two major groups (Figure 1):

a) Essential or primary, where there is no specific medical cause and represents 90 to 95% of the diagnosed cases and manifests mainly in advanced ages. It is strongly related to sedentary lifestyle habits and a diet rich in fats, carbohydrates, few vegetables and fruits. There are also several factors that can cause it, such as anti-inflammatory drugs, steroids, medications, salt (sodium chloride), alcohol and female replacement hormones (Kapil & Lobo, 2014).

b) Secondary, it is the result of pre-existing conditions, mainly categorized into problems: renal (tubular monogenic syndrome, subcapsular compression, reninoma, polycystic disease and chronic kidney disease); vascular (aortic coarctation, atherosclerotic renal stenosis and fibromuscular dysplasia); endocrine (hyperaldosteronism, hypercortiso- lemia, pheochromocytoma, acromegaly, steroid synthesis disorders, hyperthyroidism and hypothyroidism) and neuronal

(obstructive sleep apnea and autonomic failure). It accounts for 5-10% of all cases and manifests mainly in people in their 40s (Kapil &Lobo, 2014; Poulter et al., 2015).

Blood pressure also has a hereditary trait; it has been estimated that 30% of the variation in blood pressure is related to genetic factors. A common feature of most forms of hypertension with Mendelian inheritance is that they have alterations in sodium homeostasis. Genome studies have identified more than 65 loci that affect blood pressure, most of these include genes that would not have been expected to affect blood pressure based on knowledge of the pathophysiology of hypertension (Munroe, Barnes, & Caulfield, 2013). In recent years, it has been proposed that hypertension may also originate from intrauterine metabolic programming (Poulter et al., 2015).



CLASSIFICATION OF ARTERIAL HYPERTENSION	
Primary or essential hypertension - 90 to 95% of cases - It manifests itself in advanced ages. - There is no specific cause. - Related to lifestyle, diet, genetic predisposition and alterations in the intrauterine environment (epigenetics).	Secondary hypertension - 5 to 10% of cases - It occurs mainly in people over 40 years of age. - Result of pre-existing conditions, related to neurological, vascular, endocrine and renal problems.

Figure 1. Classification of arterial hypertension. According to its etiology, hypertension can be classified into: 1) Primary or essential hypertension. 2) Secondary hypertension.

Own elaboration.

Epidemiological data in humans and experimental data in animals have shown that alterations in the intrauterine environment due to various factors, such as alterations in maternal nutrition, are associated with the onset of arterial hypertension in adults and animals exposed to these during pregnancy (Velázquez, Fleming, & Watkins, 2019). One approach to understanding the relationship between fetal metabolic programming and the development of hypertension in the adult is through epigenetics (Burton & Lillycrop, 2019). This studies how environmental factors (nutrients) chemically modify DNA without changing its sequence, can be inherited, and generates a specific phenotype (Britten & Davidson, 1969).

The difficulty in interconnecting all the - mechanisms that regulate blood pressure leads to the fact that even if there is a main factor responsible for

originating hypertension, others could be responsible for its maintenance. Most theories agree that the disorder of its regulation is due to endogenous or exogenous factors. Endogenous factors are multifactorial, including genetic factors. Exogenous factors are those that trigger the genetic propensity and include mostly high salt intake, inadequate diet (foods rich in saturated fat and carbohydrates) and some psychogenetic factors (stress) (Kasko, Budaj, & Hulin, 2012).

Hypertension often presents a set of additional risk factors related to chronic noncommunicable diseases, such as insulin resistance, diabetes, obesity, and dyslipidemia, which can cause hypertension. For example, increased body weight is related to sodium retention, due to increased activity of the renin-angiotensin-sina-aldosterone system. Also in obese people, the increase in triglyceride concentration favors the appearance of atheromas that reduce the vascular lumen and increase blood pressure. Similarly, insulin resistance produces a compensatory increase in insulin to maintain adequate blood glucose levels and hyperinsulinemia has been reported to be an important risk factor for atherosclerosis that contributes to elevated blood pressure due to decreased vascular lumina (Anari, Amani, Latifi, Veissi, & Shahbazian, 2017; Schrauben et al., 2019).

Arterial hypertension is characterized by - endothelial dysfunction related to a decrease in vasorelaxant factors, such as nitric oxide (NO), bradykinin and prostacyclins, and an increase in vasoconstrictor factors such as adrenaline, serotonin, endothelins, thromboxane A2 and reactive oxygen species (Plavnik, Ajzen, Chris- tofalo, Barbosa, & Kohlmann, 2007). There is also an inability of blood vessels to modify their structure in response to he-modinamic and mechanical changes due to hyperstimu-lation of the renin-angiotensin-aldosterone system and hypersensitivity of the sympathetic nervous system (Feihl, Liaudet, Waeber, & Levy, 2006; Poulter et al., 2015). Also increased PAI-1 (plasminogen activator inhibitor-1) has an important role remodeling the structure of the vascular endothelium and participates in blood vessel thrombosis (De Taeye, Smith, & Vaughan, 2005).

The fact that endothelial dysfunction is a promoter of hypertension highlights the relationship between hypertension and other diseases that disrupt endothelial function such as diabetes, obesity, and dyslipidemia (Landmesser & Drexler, 2007). Also prospective studies have shown that chronic stress and anxiety cause an impact on the development of hypertension by constant activation of the sympathetic system (Ettner, Etner, & White, 2012).

Arterial hypertension is a disease that can be asymptomatic until non-reversible consequences manifest themselves, which is why it is known as the silent killer. The deleterious effects of hypertension are related to damage in several organs, mainly the heart, brain and kidney. In the heart, cardiac output is modified, which produces heart failure and is manifested by intolerance to effort, fatigue and renal dysfunction. When hypertension damages the blood vessels of the brain, it causes what is known as stroke, thrombosis, embolism or ictus. Depending on the part of the brain affected, stroke can lead to paralysis, blindness, memory loss, language problems or even death. Similarly, hypertension induces damage to the kidneys, originating destruction of nephrons leading to renal failure and ultimately death (Gargiulo, Suhail, & Lerma, 2015; Poulter et al., 2015).

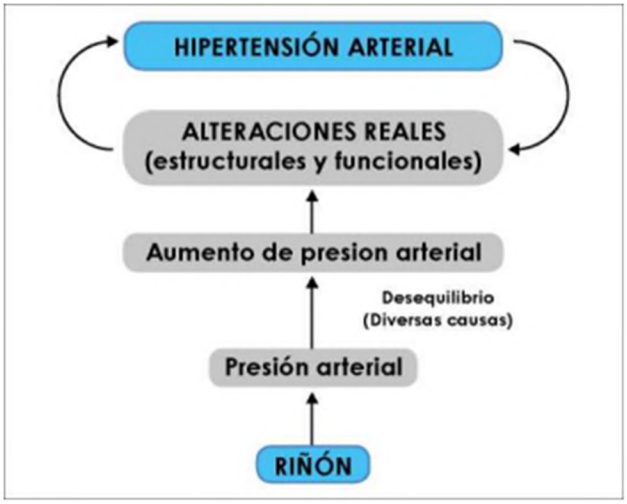
Kidney and blood pressure control

Anatomically each kidney is covered by a tough, fibrous, rigid capsule of connective tissue that serves to limit the abrupt changes in volume that occur in response to an elevation in blood pressure. The kidney is divided into two main regions: cortex and medulla; it can be further divided into four zones: cortex, outer fringe of the outer medulla, inner fringe of the outer medulla, and inner medulla (Ahmeda & Alzoghaibi, 2016). The functional and structural unit of the kidney is the nephron, which contains a cluster of capillaries called the glomerulus. Large amounts of blood are filtered in it and the filtered fluid becomes urine on its way to the pelvis of the kidney (Guyton & Hall, 2007; Schnaper, 2014).

The kidney plays a very important role in the control of blood pressure by controlling the excretion and reabsorption of water-sodium and through the synthesis and release of hormones that regulate two major systems: the renin-angiotensin-al-dosterone and the adrenergic system (Guyton & Hall, 2007). All these mechanisms also feed back on each other; therefore, when there is an imbalance, structural and functional renal alterations occur that have cumulative consequences, which are related to the genesis and maintenance of arterial hypertension (Figure 2).

The kidneys have a preponderant role in the long-term control of blood pressure, since they excrete large amounts of water and sodium. This control is related to fluid volume homeostasis in the body. When blood volume increases and vascular capacitance does not change, it generates an increase in blood pressure. If the pressure increases too much, the kidney will excrete more fluid into the urine and the blood pressure will normalize. When blood pressure decreases, the kidney will excrete less fluid than is ingested and this fluid retention will increase blood pressure. Renal elimination of water is called pressure diuresis. The increase in blood pressure also causes an increase in sodium elimination, which is known as pressure natriuresis (Guyton & Hall, 2007).

The kidneys can also regulate blood pressure in the short term, by means of the renin-an- giotensin-aldosterone system. This system contributes to its control by regulating sodium reabsorption by aldosterone and through the synthesis of angiotensin II (Ang II). Ang II increases myocardial contractility, stimulates the release of aldosterone, catecholamines from the adrenal medulla and sympathetic nerve endings, which increases sympathetic nervous system activity and water reabsorption in the kidney (Miller & Arnold, 2018). In addition, acute stimulation with Ang II regulates water-sodium homeostasis and vasoconstriction, which modulates blood pressure; whereas chronic stimulation promotes dysfunction of vascular smooth muscle cells, cardiac muscle and increased renal fibrosis (Pérez-Día z, Hiriart, Olivares-Reyes, & Robles-Día z, 2006).



ARTERIAL HYPERTENSION
ACTUAL ALTERATIONS (structural and functional)
Increase in blood pressure
Imbalance (Various causes)
Blood pressure
KIDNEY

Figure 2. Relationship between the kidney and arterial hypertension. The kidney regulates blood pressure; when there is an imbalance, there is an increase in blood pressure, which conditions the appearance of renal alterations (structural and functional) that favor the appearance of arterial hypertension, which in turn produces greater renal alterations that favor the maintenance and progression of arterial hypertension. Own elaboration.

Hypertension and chronic kidney disease

Chronic kidney disease is defined by the Kidney - Disease Improved Global Outcomes as a decrease in renal function, with a glomerular filtration rate (GFR) less than 60 ml/min/1.73 m^2 SC for more than three months and with histological alterations, albuminuria or proteinuria greater than 30 mg/dl. Chronic noncommunicable diseases are important susceptibility factors for kidney disease, mainly diabetes and hypertension (Barba Evia, 2018).

Whether renal dysfunction is the result of chronic hypertension or the first cause of the hypertensive state

continues to be under discussion (Ah- meda & Alzoghaibi, 2016; Hsu & Tain, 2019; Webster, Nagler, Morton, & Masson, 2017). However, what is not up for debate is the involvement of the kidney in the genesis and maintenance of arterial hypertension (Weir, 2009). There is a view that changes in renal function are due to structural factors and functional alterations caused by exposure of the kidney to increased perfusion pressure.

It has been shown that increased basal blood pressure can exacerbate structural changes in the kidney of hypertensive patients, resulting in secondary hypertension. Similarly, increased total peripheral resistance due to various causes has also been shown to induce increased renal vascular resistance, modify kidney function, and give rise to arterial hypertension (Ahmeda & Alzoghaibi, 2016; Weir, 2009).

The first studies that related hypertension with renal changes were carried out by Guyton et al (1972). They postulated that alterations in renal function are predisposing factors for any type of arterial hypertension, since in order to achieve natriuresis it is necessary to increase arterial pressure, with the aim of increasing glomerular filtration pressure to increase the filtered load and eliminate sodium (Guyton et al., 1972; Zehnder, 2005). Subsequently Brenner, Garcia and Anderson (1988) proposed that a decrease in the number of nephrons is also related to hypertension, since a decrease produces a compensatory glomerular hyperfiltration to maintain global glomerular filtration and sodium filtration. This effect is generated by the increase of Ang II, which determines efferent vasoconstriction, increase of glomerular pressure and hypertension (Brenner et al., 1988). Another study was that of Rettig, which consisted of transplanting kidneys from rats genetically conditioned to develop hypertension (SHR, Spontaneous Hypertensive Rats) to compatible normotensive recipient rats, which developed hypertension. It was concluded that the kidneys of the hypertensive rats had intrinsic damage that caused hypertension (Rettig et al., 1990).

On the other hand, Curtis conducted studies in individuals where he evaluated whether kidney transplantation from a healthy donor (normotensive) prevents hypertension in a recipient with arterial hypertension. He found that essential hypertension disappeared when hypertensive individuals received a transplant from a normotensive donor (Curtis et al., 1983). Although these studies point to the kidney as the cause of hypertension, renal structural alterations alone cannot be considered as the sole factor in causing hypertension.

It has been shown that hypertension can aggravate injury to the glomeruli and renal blood vessels and is a major factor in end-stage renal disease. In contrast, abnormalities of renal function can lead to arterial hypertension. The relationship between hypertension and nephropathy conditions the development of a vicious cycle, as primary renal injury increases blood pressure, which in turn further injures the kidneys, further increases blood pressure, and so on, until it gives rise to terminal nephropathy (Barba Evia, 2018; Webster et al., 2017). Vascular disorders of the kidneys related to arterial hypertension include partial or complete occlusion of vessels of various caliber, which affects the glomeruli and as a consequence progressive renal failure develops; thus, arterial hypertension further increases hypertension (Gekle, 2017; Guyton & Hall, 2007). Hypertension is highly prevalent in patients with chronic kidney disease, playing an important role in the high cardiovascular morbidity and mortality in this population (Morgado & Leão Neves, 2012; Mulè et al., 2017).

In general, two mechanisms have been proposed to explain renal damage in patients with hypertension:

1. Changes in the renal macro- and microvasculature, leading to loss of renal autoregulation and elevated glomerular capillary pressure and consequent damage by glomerular hyperfiltration.

2. Renal endothelial dysfunction and loss of endogenous vasodilators, which favor ischemic vascular injury, leading to activation of the renin-angiotensin-al- dosterone system and increased release of cytokines and growth factors, which in turn leads to the recruitment of inflammatory cells that stimulate apoptosis, causing nephron loss and

an increase in extracellular matrix synthesis, leading to renal fibrosis (Morgado & Leão Neves, 2012).

In addition, renal regulation of the renin-angiotensin-aldosterone system, mediated by local ischemia, is associated with an increase in agiotensin-converting enzyme (ACE) activity in the proximal and peritubular tubules of the interstitium, which increases Ang II production and, therefore, vasoconstriction and changes in vascular structure (Mehta & Griendling, 2007; Vío & Jeanneret, 2003). Ang II regulates cell growth in the kidney and its increase favors the - development of glomerulosclerosis and tubulointerstitial fibrosis. In addition, it stimulates endothelin I synthesis and decreases nitric oxide synthesis, which enhances its vasoconstrictor effect. Hypertension, in turn, causes afferent arteriolar sclerosis with tubular ischemia, interstitial inflammation and Ang II release, which contributes to renal fibrosis and functional deterioration (Zehnder, 2005).

CONCLUSIONS

Chronic non-communicable diseases are a major health problem, both in Mexico and in the world. Among them, arterial hypertension and chronic kidney disease have a high prevalence of morbidity and mortality. In addition, arterial hypertension in most cases does not manifest symptoms that allow it to be diagnosed in time, which has serious consequences as it is a risk factor for myocardial infarction and chronic kidney disease, making it the silent killer of chronic diseases. It is concluded that regardless of the mechanism that causes arterial hypertension, renal alterations will contribute to maintain arterial hypertension and exacerbate it.

Regardless of the origin of CKD, hypertension will contribute to worsening CKD and in turn lead to health complications, which can be life-threatening. The outlook for a possible decrease in the prevalence of hypertension and chronic kidney disease is bleak. Therefore, it is very important to educate patients suffering from either of these two diseases about the care they should take, because the appearance of one conditions the appearance of the other, leading to a progressive deterioration of health with a high probability of death.

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