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REVIEW

Microvascular injury and the kidney in hypertension

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Abstract Renal macrocirculation participates in the development of arterial hypertension. The elevation in systemic blood pressure (BP) can damage the kidney starting in the microcirculation. Established arterial hypertension impinge upon the large arteries and stiffness develops. As a consequence central BP raises and BP pulsatility appear and contribute to further damage renal microcirculation by direct transmission of the elevated BP.

PALABRAS CLAVE
Microcirculación renal;
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Hemodinámica renal

Lesión microvascular y riñón en la hipertensiónlesión microvascular y riñón en la hipertensión

Resumen La macrocirculación renal participa en el desarrollo de la hipertensión arterial. La elevación de la presión sanguínea sistémica (PS) puede producir daños renales, comenzando por la microcirculación. La hipertensión arterial establecida afecta a las grandes arterias, en las que se desarrolla rigidez. Como consecuencia, la PS central se eleva y aparece pulsatilidad, que contribuye al daño adicional de la microcirculación mediante transmisión directa de la PS elevada.

Introduction

The milieu interieur, as defined by Claude Bernard, is maintained constant in every single creature and this stability is

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essential to maintain life.[1] The kidney plays the most relevant role to maintain the milieu interieur in mammals and has unique features that allow the participation of this organ in the regulation of volume and vasoconstriction, otherwise the two main components in the maintenance of systemic blood pressure (BP). The mammalian kidney has unique features such as two types of nephrons with a long and a short-loop that participate differently in renal function and the juxtaglomerular apparatus (JGA). The JGA consists of afferent and efferent arterioles, the macula densa, and extraglomerular mesangium. The main functions of JGA are the control of renin release and the autoregulation of renal blood flow (RBF) and glomerular filtration rate (GFR), that allows the stability of these two parameters that remain constant in the presence of large variations of systemic BP and of salt intake. Fig. 1 is a schematic representation of the pressure natriuresis and autoregulation of renal hemodynamics. As can be seen urinary sodium output can increase or decrease while RBF and GFR remain constant within the limits of autoregulation. The function of short-loop nephrons located in the cortex is to create great amounts of GFR and to reabsorbe the essential elements for life while maintaining autoregulation. Those of the long-loop nephrons are urine concentration and dilution. Medullary autoregulation in the long-loop nephrons located deep in the cortex, is not as good as in cortical nephrons, allowing that an increase in systemic BP results in an increase in medullary blood flow an essential component of the mechanism of pressure natriuresis fundamental to understand the relationship between the kidney and hypertension.

Brief description of renal microcirculation

The renal microcirculation includes the interlobar, arcuate and interlobular arteries. Beyond this point renal microcirculation differs from that of other organs. Afferent arterioles form the glomerular capillaries where plasma filtration takes place, these are not followed, as could be expected, by venules but by the efferent arteriole. The descending vasa recta that ensure blood flow to the renal medulla, emanate from the efferent arterioles of juxtamedullary nephron (long-loop nephrons). Descending vasa recta, containing still arterial blood, continue in the ascending vasa recta where venous blood appears for the first time. The efferent arterioles of midcortical and superficial nephrons form peritubular capillary networks that ensure the return of essential materials previously filtered to the circulation. These capillaries are followed by venules.

Tubulo-glomerular balance ensures the response of the afferent arteriole to changes in renal perfusion pressure and in the amount of sodium detected in the tubuli by the JGA maintaining through vasoactivation the constancy of RBF and GFR. Vasoactivation of the efferent arteriole depends on the levels of angiotensin II that contribute to maintain intraglomerular pressure and GFR.

The kidney and blood pressure regulation. The kidney as culprit of the development of arterial hypertension

The existence of a relation between cardiac hypertrophy and the kidney was first described by Bright in 1836, even before BP could be measured. Mahomed demonstrated a link with BP 40 years later.[3,4] The participation of the kidney in the regulation of blood pressure is determined as previously commented by two main tasks. The first is the regulation of volumen through the excretion of water and electrolytes (mainly sodium) and the second is the participation in the regulation of vasoconstriction through the secretion of renin and the final formation of angiotensin II and also through the secretion of vasodepressor substances.[2] At the simplest level, mean arterial pressure is the result of multiplying cardiac output (representing volumen) by peripheral resistance (representing vasoconstriction) with arterial stiffness contributing as an independent factor to increase systolic BP. Systemic BP can rise as a consequence of either a derangement in volume regulation or an alteration in factors enhancing or opposing vasoconstriction. In both cases the participation of the kidney in the beginning and in the maintenance of the elevation in BP is required.

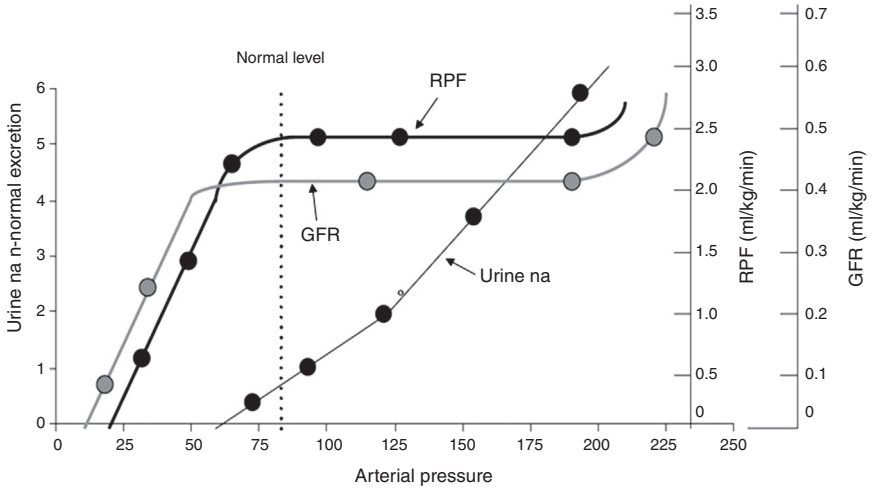

Figure 1 Representation of autoregulation of renal hemodynamics, renal blood flow and glomerular filtration rate (RBF and GFR) and pressure natriuresis. Both mechanisms contribute to the long-term control of blood pressure.