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THE RENIN-ANGIOTENSIN-ALDOSTERONE SYSTEM AND ITS ACTION IN HYPOTENSION BY VOLEMIC LOSS

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Abstract: GOAL: Conduct a literature review and observe evidence of performance of the renin-angiotensin aldosterone system (RAAS) in stabilizing blood pressure (BP) during volume loss. **METHOD:** a search was performed in bibliographic databases using the keywords: hypotension, hypovolemia, renal system, renin-angiotensin-aldosterone system. A search was also carried out in books and in databases such as Lilacs, Scielo and Medline, selecting abstracts of articles that met the initial selection criteria and discarding those that had little to do with the main object of study. **RESULT:** The RAAS is of great importance in the electrolyte balance, in the acid-base balance of the human body, especially in the control of intravascular volume and blood pressure. **CONCLUSION:** The study clarified the body's reaction, the origin of the signs and symptoms presented by the patient in the face of the activation of the renin-angiotensin-aldosterone system and the need for a very detailed evaluation, taking into account external factors that may generate doubts at the time of evaluation. However, it is worth emphasizing the importance of science that each case must be seen differently, given that each situation is unique, whose signs and symptoms may not necessarily appear all at the same time, but sometimes only one of them.

Keywords: Hypotension; hypovolemia; Renal System; Renin-Angiotensin-Aldosterone System.

INTRODUCTION

The RAAS represents a great importance in the electrolyte balance, in the acid-base balance of the human organism. It is a set of peptides, enzymes and receptors involved especially in the control of intravascular volume and BP. Briefly, macroscopic anatomophysiological aspects and their microscopic components will be addressed so that we can have a clearer understanding of the functioning of this system, which is of paramount importance for the maintenance of life.

This research is extremely important due to the theme being present in the daily life of health professionals, both pre-hospital and intra-hospital and, even so, it brings with it some dubious aspects due to the range of physiological varieties that may present one or more more signs according to the physiological response of each patient's body, the cause, the intensity, the response time for the beginning of the treatment, the environment, among other aspects.

Through a direct and didactic approach with accessible language also for lay people on the subject, who have little or no contact with such situations, this bibliographic review aims to observe the performance evidences of the RAAS in BP stabilization during a volume loss in order to clarify any doubts regarding the evaluation of a patient and the signs and symptoms of hypovolemia that he may present. Sweating in a hot climate country can mimic hypovolemia, just as cyanosis can do in cold climate regions.

Using the keywords: hypotension, hypovolemia, renal system, renin-angiotensin-aldosterone system, a search was performed in bibliographic databases. A search was also carried out in books and in databases such as Lilacs, Scielo and Medline, selecting abstracts of articles that met the initial selection criteria and discarding those that had little to do with the main object of study.

THEORETICAL FOUNDATIONS

The RAAS is of great importance in electrolyte homeostasis. It is a set of peptides, enzymes and receptors involved especially in the control of intravascular volume and the patient's BP. However, some prolegomena are necessary before studying it further.

RENAL/URINARY SYSTEM

Set of organs responsible for filtering blood, producing urine and controlling blood pressure and the body's water balance. The organs that make up this system are: kidneys, ureters, bladder and urethra.

KIDNEY

Retroperitoneal organ located between the L1 and L4 vertebrae, measuring approximately 12 cm in length. Divided into cortex and medulla, it has anterior and posterior surfaces, lateral and medial margins, superior (adrenal or adrenal gland) and inferior poles, surrounded by a connective tissue membrane and has approximately 700,000 to 1.2 million nephrons in each kidney, responsible for blood filtration.

Nephrons

The nephron is the functional part of the kidney and has the functions of blood filtration, reabsorption, secretion and excretion of substances, being essential in the process of urine formation . Composed of a longilinear tubule called the nephric tubule and the renal corpuscle (glomerulus + Bowman's capsule) and the juxtaglomerular apparatus formed by 3 types of cells: macula densa, which are sensor cells for urine osmolarity; juxtaglomerular granule cells which are modified smooth fibers of the afferent and efferent arterioles, secreting renin; and mesangial cells that have contractile properties and, according to Yones-Ibrahim (2013) "participate in the control of intraglomerular capillary flow, the glomerular ultrafiltration area and, consequently, the glomerular filtration rate in each nephron.".

The nephric tubule is divided into three parts:

- Proximal convoluted tubule: this is the region where most of the substances filtered by the glomerulus are reabsorbed in the process of urine formation ;
- Loop of Henle: Divided into thick portion and thin descending portion or straight portion of the proximal tubule (*pars recta),* thin portion and thick ascending portion or straight portion of the distal tubule. This is where water reabsorption from the filtrate takes place. This region has numerous channels formed by proteins, which make the site suitable for osmotic function due to tissue permeability;
- Distal convoluted tubule: region that regulates pH and K^+ (potassium) concentration and NaCl (sodium chloride) in the body. It follows to a larger collecting duct or tubule and carries the filtrate to the renal pelvis. The collecting duct is where urine is formed from the final processing of the filtrate;
- Collecting duct: it is the most distal (distant) portion of the nephron, responsible for several physiological processes such as bicarbonate reabsorption, hydrogen secretion, potassium reabsorption and secretion, ammonia secretion, water reabsorption, among others, being, sometimes, difficult to separate them from the functions of the distal convoluted tubule;

URETER

According to Marques (2015), they are tubular organs measuring approximately 25 cm in length connecting the renal pelvis to the urinary bladder. Its wall is composed of 3 layers: internal mucosa, consisting of transitional epithelial tissue; middle layer, formed by muscle fibers that allow peristaltic movements leading urine to the bladder; outer or adventitious layer, formed by connective

tissue protecting the other layers.

URINARY BLADDER

Muscular organ whose function is to store urine until its elimination. Its shape depends on the amount of urine contained. When full it has an oval shape and when empty it has a pyramid shape (MARQUES, 2015). It can store about 1 liter of urine, but when it reaches 200 to 400 ml, the spinal cord receives impulses that trigger the need to urinate by relaxing the internal and external sphincters.

URETHRA

Tubular duct that connects the bladder to the outside of the body. The female urethra measures about 4 cm. The male urethra measures about 20 cm, which in addition to expelling urine, has the function of eliminating seminal fluid (MARQUES, 2015).

RENIN-ANGIOTENSIN-ALDOSTERONE SYSTEM

The renin-angiotensin-aldosterone system is of extreme importance in electrolyte homeostasis. It is a set of peptides, enzymes and receptors involved, primarily in the control of intravascular volume and blood pressure (BP).

The RAAS acts to reverse the tendency to arterial hypotension by inducing peripheral arteriolar vasoconstriction and increasing blood volume through retention of sodium (by aldosterone) and water (by the release of antidiuretic hormone - ADH-vasopressin).

The activation of this system occurs in the decrease of renal perfusion detected by baroreceptors, which are "small nerve receptors that detect changes in pressure within the blood vessels and transmit this information to the central nervous system (CNS)" (BRUNNER, 2006).

In response to the central nervous system (CNS), the kidneys release renin into the bloodstream. This release occurs due to several factors such as: stimulation by pressure drop of baroreceptors located in the wall of afferent arterioles; decrease in NaCl concentration at the beginning of the distal convoluted tubule, causing also a decrease in the macula densa. Ex.: Athletes who replaced liquid ingesting only water, however, also lost mineral salts during a test, now, ingest isotonic (liquid + mineral salts).

RENIN

Renin is a proteolytic enzyme produced in Polkissen cells located in the juxtaglomerular apparatus within the nephron. With renin in the blood plasma, the liver is induced to produce and release angiotensinogen, a primarily inactive substance that, in contact with renin, is converted into Angiotensin I.

ANGIOTENSIN I

Formed from the action of the enzyme renin on angiotensinogen, it is a decapeptide that, when in contact with the angiotensin-converting enzyme (ACE), is transformed into angiotensin II.

ANGIOTENSIN II

Active hormone, resulting from the transformation of angiotensin I by the angiotensin-converting enzyme (ACE) that is produced and secreted in the endothelial cells of pulmonary capillaries, liver and kidneys in an integrated action. Its main function is vasoconstriction resulting in increased blood pressure.

ALDOSTERONE

Also controlled by renin, aldosterone is a hormone synthesized and released by the adrenal cortex whose main function is the renal retention of sodium. The drop in pressure in the renal arterioles is detected by the baroreceptors, in parallel, the decrease in

sodium to be absorbed is perceived by the chemoreceptors in the afferent and efferent arterioles, but mainly in the afferent ones. Along with sodium retention is water retention, increasing the volume of intravascular fluid.

ANTIDIURETIC HORMONE (ADH)

Hormone secreted by the posterior pituitary in response to stimulation from the sympathetic and parasympathetic nervous system that causes the kidneys to reabsorb more water, aiding in water retention already initiated by aldosterone through sodium retention.

RESULTS AND DISCUSSION

Current studies recognize two renin-angiotensin systems, which are intra-renal circulating. In the circulating system, the synthesis of angiotensin II occurs in a combined action of the renal, hepatic and pulmonary endothelium portions. In the second case, angiotensin II is formed locally in the kidney. In both cases, angiotensin II is responsible for the vasoconstriction of pre- and post-glomerular vessels. Evidence suggests that this effect occurs primarily in afferent arterioles to the detriment of efferent arterioles. However, in the juxtamedullary cortex these actions have been shown to be similar.

For Monteiro and Helou (2003), there are two intra-renal mechanisms that are the detection of hypotension, baroreceptors that detect changes in pressure in the wall of renal arterioles and in the heart, the chemoreceptors that are specialized cells to measure the concentration of NaCl in the blood and an extrarenal mechanism that are baroreceptors located in the cerebellum.

Activation of the system begins when baroreceptors detect a decrease in cardiac output, therefore, a reduction in renal perfusion, where the kidney releases the renin produced inside the nephrons by Polkissen cells commanded by the CNS, as seen above, this activates the liver, inducing it to release angiotensinogen, which, in contact with renin, is cleaved and transformed into angiotensin I. With Angiotensin I in the bloodstream, ACE is released by the adrenal glands, transforming it into Angiotensin II.

From there, the system begins to be effective because Angiotensin II, as a powerful vasoconstrictor, increases systemic vascular resistance. Hence the appearance of one of the signs of hypovolemia, cyanosis of the extremities. In response to *feedback* from the chemoreceptors in the loops of Henle, aldosterone is released by the endothelial cells of the pulmonary capillaries, exerting the function of decreasing renal sodium excretion, maintaining adequate capillary perfusion, thus bringing with it the water and fluids that are retained. also thanks to the action of ADH, produced by the hypothalamus that responds to the negative *feedback* of the neuroendocrine center, manifesting in the patient one of the signs of volume loss, thirst. The vasoconstriction of peripheral vessels affects various systems of the body, also compressing sebaceous and sweat glands, resulting in the appearance of other signs of hypovolemia, sweating, cold skin with a sticky appearance (body fat).

The combination of vasoconstriction and fluid reabsorption by both aldosterone and ADH results in an increase in blood pressure. As soon as blood pressure is readjusted to normal levels, either by the effectiveness of the renin system, or by an external action - in the case of hypovolemic shock, a volume resuscitation - the RAAS stops the hormonal cascade to which the organism is submitted, activating the system. parasympathetic nervous system to inhibit the production and release of renin.

The reader's understanding of how the

renin-angiotensin-aldosterone system works in practice will become simpler by following the flowchart in figure 1.

FINAL CONSIDERATIONS

After a brief explanation of the renal system and the organs involved, a description regarding the mechanisms of RAAS activation, the physiological process itself in a general and specific view, both macro and microscopic, the substantial logic that governs the functioning of the system it is to hemodynamically reestablish the body's functions in order to avoid volume reduction and, consequently, a reduction in systemic tissue perfusion.

By understanding the process in the microscopic scope, it will become easier to understand the genesis, the origin of signs and symptoms in a patient in the process of volume loss and/or hypovolemic shock, thus facilitating the choice of the most appropriate treatment for each case. , providing the reversal of the pathophysiological state, stabilization and recovery of the patient. Especially in a country with a tropical climate like ours, where due to the heat the body tends to work so that the body temperature remains within normal parameters where sweat is the result of thermoregulation and can sometimes confuse the professional during an appointment, letting it go by a possible situation of hypovolemia is unnoticed, whether arising from a trauma or a clinical process.

The study brought to light answers to some dubious questions, clarifying to the reader the origin of the most common signs and symptoms in a patient who presents a picture of hypovolemia, helping him in a more assertive evaluation, providing the patient with a more adequate treatment aiming at stabilization of the clinical picture and recovery, however, it is necessary to continuously search for information and research, given that each patient and each situation is unique, taking into account different factors such as the environment, climate, response time, mechanism of injury or pathology. between others.

Stimuli for Renin Secretion

Decreased renal perfusion pressure and/or decreased NaCl release to the renal tubules.

1. Examples: Bleeding, heart failure, cirrhosis, loop diuretics, salt intake. diminuída.

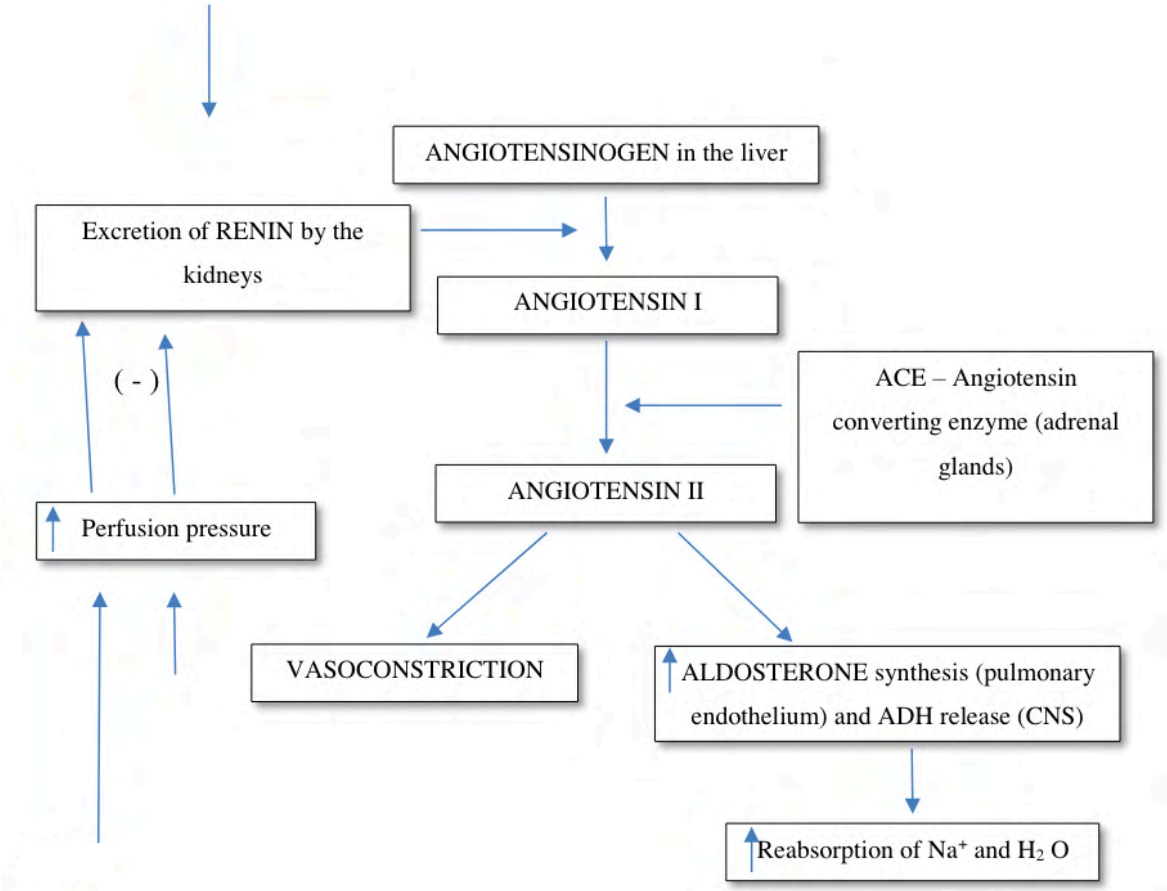

Fig. 1

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