PHYSIOLOGY ASSIGNMENT by DR OLANIYI

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Q1. Discuss the role of kidney in glucose homeostasis?

Glucose is completely reabsorbed in the proximal convoluted tubule. It is transported by secondary active transport (sodium cotransport) mechanism. Glucose and sodium bind to a common carrier protein in the luminal membrane of tubular epithelium and enter the cell. The carrier protein is called sodium-dependent glucose cotransporter 2 (SGLT2). From tubular cell glucose is transported into medullary interstitium by another carrier protein called glucose transporter 2 (GLUT2).

Renal threshold for glucose is 180 mg/dL in venous blood. When the blood level reaches 180 mg/dL glucose is not reabsorbed completely and appears in urine.

Q2. Discuss the process of micturition?

INTRODUCTION Micturition is a process by which urine is voided from the urinary bladder. It is a reflex process. However, in grown up children and adults, it can be controlled voluntarily to some extent. The functional anatomy and nerve supply of urinary bladder are essential for the process of micturition.

MICTURITION REFLEX Micturition reflex is the reflex by which micturition occurs. This reflex is elicited by the stimulation of stretch receptors situated on the wall of urinary bladder and urethra. When about 300 to 400 mL of urine is collected in the bladder, intravesical pressure increases. This stretches the wall of bladder resulting in stimulation of stretch receptors and generation of sensory impulses. Pathway for Micturition Reflex Sensory (afferent) impulses from the receptors reaches the sacral segments of spinal cord via the sensory fibers of pelvic (parasympathetic) nerve. Motor (efferent) impulses produced in spinal cord, travel through motor fibers of pelvic nerve towards bladder and internal sphincter. Motor impulses cause contraction of detrusor muscle and relaxation of internal sphincter so that, urine enters the urethra from the bladder. Once urine enters urethra, the stretch receptors in the urethra are stimulated and send afferent impulses to spinal cord via pelvic nerve fibers. Now the impulses generated from spinal centers inhibit pudendal nerve. So, the external sphincter relaxes and micturition occurs. Once a micturition reflex begins, it is self-regenerative, i.e. the initial contraction of bladder further activates the receptors to cause still further increase in sensory impulses from the bladder and urethra. These impulses, in turn cause further increase in reflex contraction of bladder. The cycle continues repeatedly until the force of contraction of bladder reaches the maximum and the urine is voided out completely. During micturition, the flow of urine is facilitated by the increase in the abdominal pressure due to the voluntary contraction of abdominal muscles. Higher Centers for Micturition Spinal centers for micturition are present in sacral and lumbar segments. But, these spinal centers are regulated by higher centers. The higher centers, which control micturition, are of two types, inhibitory centers and facilitatory

centers. Inhibitory centers for micturition Centers in midbrain and cerebral cortex inhibit the micturition by suppressing spinal micturition centers. Facilitatory centers for micturition Centers in pons facilitate micturition via spinal centers. Some centers in cerebral cortex also facilitate micturition.

Q3. Explain juxtaglomerular apparatus?

DEFINITION Juxtaglomerular apparatus is a specialized organ situated near the glomerulus of each nephron (juxta = near).

STRUCTURE OF JUXTAGLOMERULAR APPARATUS Juxtaglomerular apparatus is formed by three different structures

1. Macula densa
2. Extraglomerular mesangial cells
3. Juxtaglomerular cells.

MACULA DENSA is the end portion of thick ascending segment before it opens into distal convoluted tubule. It is situated between afferent and efferent arterioles of the same nephron. It is very close to afferent arteriole. Macula densa is formed by tightly packed cuboidal epithelial cells.

EXTRAGLOMERULAR MESANGIAL CELLS are situated in the triangular region bound by afferent arteriole, efferent arteriole and macula densa. These cells are also called agranular cells, lacis cells.

JUXTAGLOMERULAR CELLS are specialized smooth muscle cells situated in the wall of afferent arteriole just before it enters the Bowman capsule. These smooth muscle cells are mostly present in tunica media and tunica adventitia of the wall of the afferent arteriole. Juxtaglomerular cells are also called granular cells because of the presence of secretary granules in their cytoplasm. Polar Cushion or Polkissen Juxtaglomerular cells form a thick cuff called polar cushion or polkissen around the afferent arteriole before it enters the Bowman capsule.

FUNCTIONS OF JUXTAGLOMERULAR APPARATUS

Primary function of juxtaglomerular apparatus is the secretion of hormones. It also regulates the glomerular blood flow and glomerular filtration rate.

SECRETION OF HORMONES

Juxtaglomerular apparatus secretes two hormones:

1. Renin
2. Prostaglandin.

1. Renin

Juxtaglomerular cells secrete renin. Renin is a peptide with 340 amino acids. Along with angiotensins, renin forms the renin-angiotensin system, which is a hormone system that plays an important role in the maintenance of blood pressure

Stimulants for renin secretion: Secretion of renin is stimulated by four factors:
i. Fall in arterial blood pressure
ii. Reduction in the ECF volume
iii. Increased sympathetic activity
iv. Decreased load of sodium and chloride in macula densa.

Renin-angiotensin system When renin is released into the blood, it acts on a specific plasma protein called angiotensinogen or renin substrate. It is the α2 -globulin. By the activity of renin, the angiotensinogen is converted into a decapeptide called angiotensin I. Angiotensin I is converted into angiotensin II, which is an octapeptide by the activity of angiotensin-converting enzyme (ACE) secreted from lungs. Most of the conversion of angiotensin I into angiotensin II takes place in lungs. Angiotensin II has a short half-life of about 1 to 2 minutes. Then it is rapidly degraded into a heptapeptide called angiotensin III by angiotensinases, which are present in RBCs and vascular beds in many tissues. Angiotensin III is converted into angiotensin IV, which is a hexapeptide.

Actions of Angiotensins

Angiotensin 1 is physiologically inactive and serves only as the precursor of angiotensin II. Angiotensin II is the most active form. Its actions are:

On blood vessels:

i. Angiotensin II increases arterial blood pressure by directly acting on the blood vessels and causing vasoconstriction. It is a potent constrictor of arterioles. Earlier, when its other actions were not found it was called hypertensin.

ii. It increases blood pressure indirectly by increasing the release of noradrenaline from postganglionic sympathetic fibers. Noradrenaline is a general vasoconstrictor.

On adrenal cortex: It stimulates zona glomerulosa of adrenal cortex to secrete aldosterone. Aldosterone acts on renal tubules and increases retention of sodium, which is also responsible for elevation of blood pressure.

On kidney:

i. Angiotensin II regulates glomerular filtration rate by two ways:

a. It constricts the efferent arteriole, which causes decrease in filtration after an initial increase b. It contracts the glomerular mesangial cells leading to decrease in surface area of glomerular capillaries and filtration ii. It increases sodium reabsorption from renal tubules. This action is more predominant on proximal tubules.

On brain:

i. Angiotensin II inhibits the baroreceptor reflex and thereby indirectly increases the blood pressure. Baroreceptor reflex is responsible for decreasing the blood pressure

ii. It increases water intake by stimulating the thirst center

iii. It increases the secretion of corticotropin-releasing hormone (CRH) from hypothalamus. CRH in turn increases secretion of adrenocorticotropic hormone (ACTH) from pituitary

iv. It increases secretion of antidiuretic hormone (ADH) from hypothalamus. Other actions: Angiotensin II acts as a growth factor in heart and it is thought to cause muscular hypertrophy and cardiac enlargement.
Angiotensin III increases the blood pressure and stimulates aldosterone secretion from adrenal cortex. It has 100% adrenocortical stimulating activity and 40% vasopressor activity of angiotensin II.
Angiotensin IV It also has adrenocortical stimulating and vaso pressor activities.
2. Prostaglandin Extraglomerular mesangial cells of juxtaglomerular apparatus secrete prostaglandin. Prostaglandin is also secreted by interstitial cells of medulla called type I medullary interstitial cells.
SECRETION OF OTHER SUBSTANCES
1. Extraglomerular mesangial cells of juxtaglomerular apparatus secrete cytokines like interleukin-2 and tumor necrosis factor
2. Macula densa secretes thromboxane A2.
REGULATION OF GLOMERULAR BLOOD FLOW AND GLOMERULAR FILTRATION RATE Macula densa of juxtaglomerular apparatus plays an important role in the feedback mechanism called tubuloglomerular feedback mechanism, which regulates the renal blood flow and glomerular filtration rate.

Q4 Discuss the role of kidney in regulation of blood pressure?

Kidneys play an important role in the longterm regulation of arterial blood pressure. When blood pressure alters slowly in several days/months/years, the nervous mechanism adapts to the altered pressure and loses the sensitivity for the changes. It cannot regulate the pressure any more. In such conditions, the renal mechanism operates efficiently to regulate the blood pressure. Therefore, it is called longterm regulation. Kidneys regulate arterial blood pressure by two ways:
1. By regulation of ECF volume
2. Through reninangiotensin mechanism.
 BY REGULATION OF EXTRACELLULAR FLUID VOLUME: When the blood pressure increases, kidneys excrete large amounts of water and salt, particularly sodium, by means of pressure diuresis and pressure natriuresis. Pressure diuresis is the excretion of large quantity of water in urine because of increased blood pressure. Even a slight increase in blood pressure doubles the water excretion. Pressure natriuresis is the excretion of large quantity of sodium in urine. Because of diuresis and natriuresis, there is a decrease in ECF volume and blood volume, which in turn brings the arterial blood pressure back to normal level. When blood pressure decreases, the reabsorption of water from renal tubules is increased. This in turn, increases ECF volume, blood volume and cardiac output, resulting in restoration of blood pressure.
THROUGH RENIN-ANGIOTENSIN MECHANISM: Source of renin secretion, formation of angiotensin and conditions when renin is secreted. Actions of Angiotensin II When blood pressure and ECF volume decrease; renin secretion from kidneys is increased. It converts angiotensinogen into angiotensin I. This is converted into angiotensin II by ACE (angiotensin converting enzyme). Angiotensin II acts in two ways

to restore the blood pressure:

i. It causes constriction of arterioles in the body so that the peripheral resistance is increased and blood pressure rises. In addition, angiotensin II causes constriction of afferent arterioles in kidneys, so that glomerular filtration reduces. This results in retention of water and salts, increases ECF volume to normal level. This in turn increases the blood pressure to normal level.

ii. Simultaneously, angiotensin II stimulates the adrenal cortex to secrete aldosterone. This hormone increases reabsorption of sodium from renal tubules. Sodium reabsorption is followed by water reabsorption, resulting in increased ECF volume and blood volume. It increases the blood pressure to normal level.

Actions of Angiotensin III and Angiotensin IV Like angiotensin II, the angiotensins III and IV also increase the blood pressure and stimulate adrenal cortex to secrete aldosterone.

Q5. Discuss the role of Kidney in Calcium homeostasis?

Kidneys play a role in the regulation of blood calcium level by activating 1, 25-dihydroxycholecalciferol into vitamin D. Vitamin D is necessary for the absorption of calcium from intestine. Calcitriol is a steroid hormone synthesized in kidney. It is the activated form of vitamin D. Its main action is to increase the blood calcium level by increasing the calcium absorption from the small intestine. Calcium is absorbed from duodenum by carrier mediated active transport and from the rest of the small intestine, by facilitated diffusion. Vitamin D is essential for the absorption of calcium from GI tract