In this lecture the doctor continued the pituitary gland and started with the adrenal gland. (it's really important to refer to the slides).

First the pituitary gland deficiency:

- A) <u>Panhypopituitarism</u>: "pan = all ", this term means deficiency of all hormones including posterior pituitary hormones (ADH, oxytocin).
 - The general effects of the adult panhypopituitrasim are:
 - 1) **ADH**: deficiency of ADH causes loss of high amounts of water due to lack of the reabsorption which lead to diabetes insipidus.
 - 2) **Oxytocin**: this hormone causes contraction of the pregnant uterus which helps in delivery, a deficiency of this enzyme is not very serious and will not severely affect the labor process it will just make it a little bit harder.
 - 3) **Gonadotropin**: deficiency of this hormone will decrease the libido "sexual desire" in both sexes, and will cause aspermia "the complete lack of semen", and loss of facial and body hair in males, while deficiency of this hormone in females will lead to amenorrhea.
 - 4) **TSH**: a deficiency of this hormone will lead to hypothyroidism due to the lack of secretion of the thyroid hormone from the thyroid gland.
 - 5) **ACTH** :a deficiency of this hormone will affect the secretion of adrenal cortex's hormones.
 - 6) **MSH**: a human with deficiency of this hormone will have a pale skin
 - 7) **GH**: a deficiency of this hormone will cause dwarfism (in general all the physical parts of the body develop in appropriate proportion in to one another in dwarfism but the rate of the development Is greatly decreased), hypoglycemia.

B) Anterior pituitary deficiency:

- Three types: severe, moderate, mild.

Notes:

- 1) In person with dwarfism the abdominal portion is smaller than the thorax .
- 2) In severe ,moderate and mild anterior pituitary deficiency <u>refer</u> to the slides the doctor just read them but he pointed at some points :
 - in moderate deficiency ACTH is partially deficient and the GH is normal.
 - in mild deficiency only gonadotropins are deficient (notice that gonadotropins are deficient in all pituitary gland deficiencies).
- 3) Gonadotrpins deficiency in child lead to delayed puberty (deficient growth preclude eunchoid habitus).
 - eunchoid habitus: The condition of being a eunuch or of having undeveloped sexual organs in which testicular hormones are not produced (from the internet).

Hyper secretion of GH: two conditions

- A- <u>Giantism or gigantism</u>: it occurs before adolescence, all body tissues will grow rapidly including the bones because the epiphysis of the long bones have not fused with the shafts.
 - These individuals will:
 - Be 8-9 feet height
 - Have Hyperglycemia
 - 10% develop diabetes mellitus
 - All parts of the body develop in appropriate proportion in to one another

If the giant remain without treatment they will develop panhypopituitarism.

- B- <u>Acromegaly</u>: after adolescence, after the fusion of the long bones, the person can't grow taller but the soft tissue can continue growing and the bones can grow in thickness. These individuals will:
- Suffer from enlargement of the small bones of hands, feet, cranium, nose, forehead, supraorbital ridges, the lower jaw bone and portions of the vertebrae.
- Have the liver ,tongue, kidneys greatly enlarged.

second the adrenal gland

We have two glands (right and left)above the kidneys, they are unique glands, very important and essential for life .one indication for their importance is their extensive blood supply .

Each gland consist of two parts:

- Adrenal medulla which is functionally related to sympathetic nervous system and release epinephrine and norepinephrine.
- Adrenal cortex: is essential to life because their hormones:
 - 1- control mineral and water metabolism.
 - 2- control the metabolism of fat, protein, and carbohydrate.
 - 3- participate in the response to stress.

the doctor said that we will talk about the adrenal cortex only.

Adrenal cortex

Produces hormones known as corticosteroid which are synthesized from cholesterol, the corticosteroid include: mineralocorticoids, glucocorticoids, and androgens. These hormones are not stored in the gland, they're released

immediately, so a new synthesis is needed if there is an urgent need for the hormones.

The cortex consist of three main zone:

A- Zona glomerulosa(12%): produce hormones called corticoids (Aldosterone), which function in the mineral metabolism.

B- Zona fasciculata(65%): produce hormones called glucocorticoids (cortisol), which function in glucose metabolism. May produce a little of androgens.

C-Zona reticularis(23%): produce androgen and estrogen and a little bit of glucocorticoids.

They say that after certain age the glomerulosa cells migrate down to reticularis and change their shape and function. (unknown reason).

Adrenal cortex mainly produces cortisol more than aldosterone. In the slides, figure 3.5 there is a comparison between the two hormones and from it we notice that:

- In glucocorticoids activity the cortsiol is more potent. while in the mineralocorticoid activity the aldosterone is more potent.
- we also can see that the cortsiol secretion rate is higher than that of aldosterone, and that's give the cortsiol a significant effect on mineral corticoids

Both hormones bind with the same receptors but the affinity differ.

Adrenal cortex and ACTH

ACTH is the main stimulus for the adrenal cortex, it helps in the growth of cortex cells as well as in the synthesis and secretion of hormones. the main target of ACTH is the **cortisol**.

The regulation of ACTH secretion regulate the growth and secretion of adrenal cortex, and even though the regulation of ACTH is very complicated -with the regulation mechanism being unclear-, CRH (corticotropin-releasing hormone) and ADH play an important role in its regulation.

ACTH secretion also response to stressful stimuli, which is a critical response for survival.

Extra adrenal actions for ACTH are lipolysis and MSH like action.

Figure 49.5 in slides represent a diurnal/circadian rhythm (secretion during 24 hours) of ACTH and cortsiol. we can notice that when there is a variation in ACTH secretion there is a variation in the cortisol secretion(parallel secretion).

Cortisol

- It affect almost all organs of the body but mainly it has four main functions:
- Gluconeogenesis.
- Increase the responsiveness of blood vessels.
- -Fat metabolism.
- -Modulation of the CNS function.
 - Cortisol does not break glucagon by itself, it plays a permissive role with it (facilitate the action of glucagon). it's also essential for life especially in fasting humans and animals, and a deficiency of cortisol may lead to chronic or acute fasting leading to death of animals or humans.
 - 90% of the cortisol bind to corticosteroid binding protein and 6% bind with albumin, it also can bind to aldosterone receptors but with less affinity.
 - sometimes the cortisol synthesis may be blocked which lead to the increase of the corticosterone concentration.

- The role of cortisol in fetal life.
 - 1) Maturation of the lungs: some new born may have problem with respiration so the doctor inject them with cortisol.
 - 2) liver enzymes.
 - 3)Development of hypothalamic function and the thyroid-pituitary axis.
 - 4) The sequential changes of placental structure and ionic composition of amniotic and allanotic fluids during development.
 - 5) initiation of endocrine changes.
- There is natural and synthetic cortisol, the synthetic are used as drugs against any disease related to blood, these drugs may function or not depending on the presence of receptors.

Aldosterone

- Short half life.
- 20% bind to the corticosteroid receptor, 40% with albumin, and it may bind to the cortisol receptor.
- There is natural and synthetic aldosterone.
- Aldosteone is very important for life, main function of it to normalize the extracellular fluid volume affecting the blood volume consequently the blood pressure. It also important in glucose metabolism.
- Aldosterone is stimulated by:
 - 1) Angiotensin II: (main stimulus) for example in hemorrhage or something like that, extracellular fluid decreases, stimulating the release of renin(from kidneys)which stimulate the conversion of angiotensinogen(from liver) to angiotensin I which is converted to angiotensin II that stimulate the secretion of aldosterone.

people with hypertension tend to take drugs to inhibit the enzyme that convert the angiotensin I into angiotensin II, such drugs are called angiotensin converting enzyme inhibitors.

- 2) Increase in the plasma potassium.
- 3) ACTH.

Angiotensin II

- Stimulate the aldosterone secretion.
- stimulate sodium reabsorption.
- increase the reabsorption of water.

angiotensin II is a vasoconstrictor of the efferent tubule which has two effects:

- 1) increase glomular filtration rate → plasma decreases→ hydrostatic pressure in peritubular capillaries decrease→ water reabsorption increase.
- 2) Filtration increase→ plasma decrease (plasma protein concentration does not change)→ osmotic pressure increase→ water reabsorption increase.

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14-3-2013	physiology 4	Dr.saleem
	8	