



Adrenal Glands Hormone Deficiency Causes Different Symptoms

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Abstract

There are two adrenal glands, located above the kidneys in the lower back, and they produce several types of hormones. The inner part of each gland produces adrenaline and noradrenaline, which are responsible for the cause-and-effect reaction that occurs during danger and emotional stress. The outer part produces aldosterone, which regulates the body's salt balance; cortisol, which is essential for the metabolism of proteins, fats and carbohydrates; and male sex hormones. In some diseases of the adrenal glands, cortisol and aldosterone cannot be produced, usually due to the lack of enzymes necessary for their production. The hypothalamus detects low levels of these hormones in the blood and stimulates the pituitary gland, which then tries to stimulate the adrenal glands to produce sufficient amounts of cortisol and aldosterone. The adrenal glands enlarge to more than 10 to 20 times their normal size due to constant stimulation from the hypothalamus and pituitary gland, but are still unable to produce cortisol and aldosterone.

Keywords: adrenal glands; ACTH; hormones; ai; health

The adrenal glands are important endocrine organ that produces corticosteroids counting glucocorticoids, mineralocorticoids and androgens [1]. These are important hormones that direct sodium maintenance, blood pressure, fluid volume, the resistant framework, metabolism and conduct. The adrenals too apply negative feedback component within the hypothalamus and pituitary through the hormone cortisol. High levels of cortisol smother the pituitary hormone ACTH though moo cortisol invigorates ACTH discharge by expanding the discharge of hypothalamic hormones CRH and AVP. The adrenal medulla, which is found within the central portion of the adrenal, shapes portion of the thoughtful apprehensive framework and is alluded as the sympatho-adrenomedullary system. It insider facts epinephrine (adrenaline), norepinephrine (noradrenaline) and dopamine in reaction to incitement by the thoughtful anxious framework.

Anatomy

The adrenal glands weigh 4–5 g each in a typical sound adult and sit on the upper end of the correct and left kidneys [1]. The middle of the adrenal gland, the medulla, weighs around 1 g. The external layer, the zona glomerulosa (zG), is composed of ovoid-shaped cells. The zona fasciculata shapes the majority of the adrenal cortex and is sorted out in fascicles or bundles. There's no anatomical demarcation between medulla and cortex. The medulla is composed of chromaffin cells and contains small vesicles 100–300 nm in distance across in which catecholamines (adrenaline and noradrenaline) are put away and released. Within the human, adrenaline accounts for 80% of adrenal catecholamine release into the circulation system and has effects on numerous organs.

The adrenal cortex synthesises corticosteroids, counting over 50 unmistakable steroid hormones. Beneath typical condition within the nonappearance of push, the grown-up adrenal cortex produces approximately 10–15 mg of cortisol per day.

The adrenal glands are exceedingly vascular, with three basic supply routes providing each adrenal gland. The predominant suprarenal course, the prevalent suprarenal artery and the center suprarenal artery. Blood streams into the adrenal cortex and drains into the adrenal medulla some time recently entering the second rate vena cava through the central vein on the correct adrenal. On the cleared out, the adrenal vein blood channels into the cleared out renal vein. The waste system of the adrenal gland plays a complex part in steroid blend and direction. The adrenal gland is additionally well innervated. The nerve supply starts from the coeliac plexus and thoracic splanchnic locale of the thoughtful autonomic apprehensive system, as well as a few parasymphathetic commitments from the phrenic and vagal nerves. The nerve supply also comes to the chromaffin cells within the medulla, and the innervation has been proposed to reach the cortisol arteriolar and capillary bed to control cortisol blood flow. Chromaffin cells are too found within the vagus nerve, carotid supply routes, bladder, prostate and liver. Other than emitting catecholamine and dopamine, the adrenal medulla too secretes other stretch hormones such as enkephalins and neuropeptide Y. One of the foremost vital activities of catecholamine is the fight-or-flight reaction which leads to an increment in breath, heart rate, blood pressure and blood vessel constriction (in the skin and gut).

Cortisol Deficiency

Circulating cortisol concentrations are impacted by the presence of cortisolbinding globulin (CBG), too known as transcortin or serpin A6, an α -globulin encoded by the SERPINA6 gene [2]. On the off chance that hepatic blend of CBG is lacking due to lack of healthy sustenance, malabsorption, or liver malady, or from protein misfortune due to nephrotic disorder, add up to cortisol concentrations may decay underneath the reference interim, however the organically dynamic free cortisol concentration is satisfactory. Then again, hoisted CBG concentrations can raise add up to cortisol levels without expanding free cortisol movement. Ordinarily, around 70% of circulating cortisol is bound to CBG, 20% is bound to albumin, and 10% of add up to cortisol is unbound and organically dynamic. Numerous hormones are exceedingly protein-bound, either to particular hormone-binding proteins or albumin, or most commonly, a combination of both. The proteins buffer the concentration of free, dynamic hormone, preventing unexpected changes in hormonal movement that may have unpleasant, or indeed life-threatening physiological consequences.

There's awesome intrigued in measuring free cortisol. In seriously care settings, where patients are as often as possible malnourished, low CBG has the potential to incite the overdiagnosis of Addison illness. Measuring CBG permits calculation of a free cortisol index, but there's contradiction over the unwavering quality of calculated free cortisol estimates. Coordinate estimation of free cortisol concentration by equilibrium dialysis or ultrafiltration is awkward and once in a while done. Be that as it may, two choices exist for assessing the free cortisol concentration: salivary cortisol and urinary free cortisol (UFC).

Salivary cortisol reflects free cortisol concentrations within the blood. The spit example is regularly collected on a wipe and communicated into a test tube some time recently filtration and investigation by either immunoassay or mass spectrometry. In expansion to cortisol, there's intrigued in measuring other steroids in spit. A later report portrays the estimation of classical and 11-oxygenated androgens in human spit utilizing fluid chromatography-tandem mass spectrometry (LC-MS/MS). Steroid profiling by LC-MS/MS, at one time restricted generally to research facilities testing competitors for performance-enhancing drugs, is getting to be progressively common for clinical research facility appraisal of endocrine clutters.

Hormones

Over 150 years ago, Brown-Sequard illustrated that the adrenal organs are fundamental for life [3]. Within the late nineteenth and twentieth centuries, the hormones emitted by the adrenal cortex and medulla were disconnected, purified, and synthesized for helpful utilize. In addition, their receptors, stimulators, inhibitors, co-activators, corepressors, intracellular signaling cascades, and downstream effectors have been depicted. Within the twenty-first century, the molecular hereditary qualities basic numerous adrenal disorders have been revealed.

In spite of the fact that physiologic levels of adrenal hormones are fundamental for the right working of all tissues and organs and basic for the stress response, pharmacologic levels of these hormones have obliterating impacts on numerous organ frameworks. Clinical considers over the past 20 a long time have uncovered that numerous cases of so-called "idiopathic" hypertension are due to basic adrenal hypersecretory disorders. A number of review thinks about over the past decademonstrate that any degree of hypercortisolism is pernicious to bone, metabolic, and cardiovascular health. Propels in radiologic imaging, especially



the presentation of the CT filter within the early 1970s, driven to the plague of “adrenal incidentalomas”—incidentally found adrenal masses, a substance that is especially predominant in more seasoned people. Detailed hormonal testing of patients with adrenal incidentalomas has uncovered that numerous are related with gentle hypercortisolism. At the other conclusion of the range, Cushing’s disorder due to ectopic ACTH produces extreme and possibly lifethreatening hypercortisolism. The cortisol overabundance must be quickly tended to, indeed in patients with metastatic cancer, as diminishment in cortisol levels makes strides dismalness and mortality and allows the understanding to get cancer medications. Over the top catecholamine discharge from pheochromocytomas, indeed on the off chance that as it were verbose, can result in sudden death. Pheochromocytomas are considered “ticking time bombs” that require careful preoperative and perioperative administration in arrange to guaranteed a secure result.

ACTH

The adrenal corticotropic hormone (ACTH), which is additionally called corticotropin or adrenocorticotropin, controls hormone generation by the cortex of the adrenal glands [4]. This family of pituitary peptides incorporates α -, β -, and γ -melanocytostimulating hormones (MSH), β - and γ -lipotropin (LPH), and β -endorphin, but ACTH is the as it were item of corticotropes with an set up physiological role in people. The MSHs, which disperse melanin color in melanocytes within the skin of lower vertebrates, are not emitted in significant amounts by the human pituitary organ, but these compounds are created in melanocytes and keratinocytes within the skin where they act in a paracrine or autocrine way to affect pigmentation. Be that as it may, ACTH and both β - and γ -lipotropin contain the grouping of seven amino acids that produces the melanocyte fortifying impact of MSH. It is likely accounts for the obscuring or bronzing of the skin when these hormones are emitted in overabundance. α - and β -MSH too are delivered by neurons within the arcuate core, and play an critical part in control of nourishment admissions. β -LPH is named for its stimulatory effect on mobilization of lipids from fat tissue in rabbits, but the physiological significance, on the off chance that any, of this activity in people is questionable. The e 91-amino-acid chain of β -LPH contains at its carboxyl conclusion the total amino corrosive arrangement of β -endorphin (from endogenous morphine), which energizes the same receptors as morphine in different cells.

The ACTH related peptides constitute a family because (1) they contain locales of homologous amino corrosive groupings, which may have emerged through exon duplication, and (2) since they all are encoded within the same quality. The quality item is called pro-opiomelanocortin (POMC) and comprises of 239 amino acids after evacuation of the 26 amino corrosive flag peptide. The atom contains 10 doublets of fundamental amino acids (arginine and lysine in different combinations), which are potential locales for cleavage by subtilisin-like endopeptidases, called prohormone convertases (PCs). At slightest five other chemicals deliver other posttranslational adjustments. Tissue-specific contrasts in expression of preparing chemicals account for the contrasts in lastsecretory items of corticotropes and the other cells that express POMC. Corticotropes express PC 1, while POMC communicating neurons and skin cells contain both PC1 and PC2. As a result, corticotropes deliver less, but bigger parts. The overwhelming items of human corticotropes are ACTH and β -LPH. Since last preparing of POMC happens within the secretory granule, β -LPH is discharged along side ACTH. Cleavage of β -LPH moreover happens to some degree in human corticotropes, so that a few β -endorphin may moreover be discharged, especially when ACTH discharge is brisk.

Adrenal Cortex

The cortex of the adrenal glands creates from the embrional mesodermal layer, while the medulla emerges from the neuroectodermal layer and incorporates portion of the chromaffin system [5]. The adrenal glands are more often than not found over the kidneys and their weight, in adults, ranges from 4.0 to 4.2 g \pm 15%. Histological thinks about of the adrenal cortex illustrate a partition into three layers. The zona glomerulosa speaks to 5–10% of the cortex with irregular, subcapsular totals of little cells. This zone is less wealthy in cytoplasm than the other cortical zones and contains the chemicals that synthesize aldosterone and the other mineralocorticoids, beneath the control of the renin-angiotensin system. The zona fasciculata, which is shaped by spiral ropes of expansive columned cells, with inexhaustible lipid-filled cytoplasm covers 70% of the cortex. It produces glucocorticoids beneath the impact of the adrenocorticotropin hormone (ACTH) delivered by the pituitary organ. The zona reticularis, which sums to almost 20% of the cortex, is composed of cells orchestrated in strings, with compact, finely granular, eosinophilic cytoplasm, and synthesizes the adrenal androgens (such as dehydroepiandrosterone). The adrenal blood supply determines from the three fundamental supra-renal supply routes, which puncture the organ surface, isolating into 50–60 little branches and shaping a subcapsular plexus, which grasps the cell clusters within the zona glomerulosa, and after that runs along the cellular lines within the zona fasciculata. The capillary branching from the arterial system shapes a

microvascular arrange around the zona reticularis, which channels into the medullary sinusoids by implies of generally few little venules, which in the long run frame a expansive central vein. This move from the arterial to the capillary framework is so sharp that it shapes a sort of utilitarian “vascular dam”. In expansion, the medullary veins appear an unpredictable musculature, which encourages the development of pockets of localized stasis when the bundles contract. The vein compression is additionally advanced by the tall catecholamine concentration at this level.

The homeostasis of the glucocorticoids is controlled by a input instrument; firstly through the hypothalamus by implies of a corticotropin-releasing hormone (CRH), at that point through the pituitary organ by implies of ACTH, and final through the adrenal cortex by implies of cortisol. On the opposite, the mineralocorticoids are primarily directed by the renin-angiotensin system and by certain particular serum electrolytes (potassium and sodium).

The circadian movement of the hypothalamic-pituitary-adrenal (HPA) pivot is made up from an fundamental oscillatory cadence of ACTH and glucocorticoid pulses that shift in plentifulness but not recurrence over the 24 h [6]. This oscillatory action isn’t the result of a hypothalamic oscillator but rises as a normal result of the feedforward:feedback interaction between the pituitary corticotropes and the glucocorticoid-secreting cells of the adrenal cortex. This oscillatory action has come about in adjustments within the way tissues perused their ‘digital’ ligand flag. The adrenal cortex is relatively insensitive to steady signals of ACTH but reacts briskly to the identical sum of ACTH managed in a pulsatile way. So also glucocorticoid-responsive tissues such as the brain and the liver are able to studied the swaying signals of cortisol or corticosterone emission, with differential biochemical and useful reactions to distinctive designs of ligand introduction. Amid a drawn out intense stretch there’s a major alter within the pituitary-adrenal relationship, with a stamped increment within the affectability of the adrenal to little changes in ACTH, so that taking after cardiac surgery little motions in ACTH result in enormous swings in cortisol. This reaction shows up to be due to a alter both within the ACTH flagging pathway and within the endogenous activators and inhibitors of glucocorticoid union.

Functions

The adrenal gland is composed of an external cortex and an internal medulla [7]. The external cortex is isolated in three parts: the external zona glomerulosa, the mid zona fasciculata and an internal zona reticularis. Mineralocorticoids which are created by the external zona glomerulosa are transcendently included in sodium and potassium adjust. Glucocorticoids, which are delivered by the fasciculata, are overwhelmingly included in carbohydrate metabolism but moreover have a few impact in salt and electrolyte adjust. The zona reticularis produces adrenal androgens which are metabolized incidentally to testosterone and dihydrotestosterone. The adrenal gland is under hypothalamic/pituitary control to deliver glucocorticoids and the renin angiotensin system to deliver mineralocorticoids.

The adrenal cortex is mindful for the generation of three major classes of steroid hormones: glucocorticoids, mineralocorticoids and androgens [1]. The zona glomerulosa incorporates the mineralocorticoids aldosterone, the zona fasciculata produces cortisol, whereas the inward layer zona reticularis secretes androgen steroids such as dehydroepiandrosterone (DHEA), dehydroepiandrosterone sulfate (DHEAS), androstenedione and 1 β -hydroxyandrostenedione. Glucocorticoids, such as cortisol, are discharged in high amounts around 10–50 mg/day while mineralocorticoids, such as aldosterone, are much less at around 100–200 μ g/day. Shockingly, DHEAS is additionally discharged in huge amounts.

All of the items emitted by adrenal glands have significant impacts on the kidneys, both in physiological and neurotic conditions [8]. Steroids hormones, i.e., glucocorticoids, mineralocorticoids, and sex hormones, are discharged by the outer cortex and are included in metabolism, inflammation, and fluid homeostasis. Adrenal medulla primarily secretes catecholamines and having a diverse formative beginning is additionally distinctive in terms of function and structure. Catecholamines (adrenaline and noradrenaline) control blood weight, blood stream direction, and breathing rate. The interaction between the kidney and mineralocorticoids is based on the RAAS (renin-angiotensin-aldosterone system) system. Angiotensinogen is delivered by the liver and the arrangement of angiotensin I is catalyzed by renin (angiotensinogenase). Renin is discharged from the juxtaglomerular cells of the kidney and has autocrine and paracrine impacts. The control of the production of renin is based on the action of baroreceptors that identify variety in arterial blood pressure, on the conveyance of sodium to the distal tubule that’s detected by the macula densa, and on the activity of the thoughtful anxious system. Angiotensin I is at that point changed over within the vasoactive peptide angiotensin II by the angiotensin-converting enzyme (Ace). Ace is found in lungs but too within the renal tubular epithelial brush border. Angiotensin II receptor type 1 (AT1) is communicated within the kidney and other tissues, though Angiotensin II receptor type 2 (AT2) is found within the adrenal medulla, brain, and gonads. The secretion of the mineralocorticoid aldosterone, which could be a steroid hormone, is primarily actuated by the generation of Angiotensin II caused by hypotension but too by



hoisted levels of potassium in plasma. Aldosterone increments the blood pressure because it has an antinatriuretic impact, advancing sodium and water retention primarily acting on the receptors of the distal tubule and collecting conduit of the nephron. It too invigorates potassium discharge, hence evacuating the abundance of potassium in case of hyperkalemia, and hydrogen excretion.

The impacts of tall levels of mineralocorticoids incorporate an expanded extracellular liquid volume, hypokalemia, and alkalosis. On the inverse, hypocorticism causes a diminished liquid extracellular volume with hyponatremia and hyperkalemia.

Glucocorticoids and mineralocorticoids have distinctive capacities. Glucocorticoids increment renal stream, glomerular filtration rate, and diuresis. Cortisol increments sodium maintenance and potassium excretion but not hydrogen. The utilize of corticosteroids as drugs is common in renal diseases, and complications include nephrocalcinosis and renal stones formation.

Androgens

Adrenal androgens are orchestrated from cholesterol through a arrangement of intracellular and extracellular activities including oxidases and dehydrogenases [1]. Not at all like glucocorticoids, adrenal androgens have small movement at androgen receptors. Instead, they work as antecedent particles prepared to be metabolized incidentally to powerful androgens such as DHT or aromatised to oestradiol. The C19 steroids incorporate DHEA, DHEAS, androstenedione and 11 β -hydroxyandrostenedione. They are delivered in adrenal gland and gonads in both men and women. Circulating levels of dehydroepiandrosterone (DHEA), DHEA-sulphate (DHEAS), androst-5-ene-3 beta, 17 beta-diol (5-diol), 5-diolsulphate, 5-diol-fatty acid esters and androstenedione in both men and women were found to decay from the age of 20–80 a long time ancient. The majority of DHEA and DHEAS comes from the adrenals, counting 50% of emitted androstenedione. The change to powerful androgens such as testosterone or dihydrotestosterone occurs in tissues that express HSD3-B1 and HSD17-B1/5 such as adipose tissue and the skin

The adrenal gland secretes a little sum of testosterone. In men, testosterone is overwhelmingly created by Leydig cells in the testes. In ladies, DHEA can be taken up by ovarian theca cells to orchestrate testosterone and may be changed over to dihydrotestosterone in fringe tissues once changed over to androstenedione, without requiring change to testosterone. In solid men, circulating testosterone levels are around ten times higher than women.

Testosterone has a coordinate affect in body composition of men by increasing lean body mass and this leads to extend affront affectability. Moo testosterone autonomously influences affront resistance. Androgen replacement treatment makes strides affront affectability, diabetes and the metabolic disorder in hypogonadal men.

High levels of testosterone relate with tall androstenedione levels in women with polycystic ovary disorder who were found to have an antagonistic metabolic phenotype.

On the other hand, patients with adrenocortical carcinoma frequently show with Cushing's syndrome and androgen overabundance counting DHEA/DHEAS, androstenedione and testosterone. High testosterone in women may cause hirsutism, skin break out, developing of the voice, hair misfortune and amenorrhoe. Be that as it may, testosterone overabundance in men raised haematocrit, seborrhoea, skin break out and a few disposition changes.

AI

Adrenal insufficiency (AI) could be a common lifethreatening endocrine condition [9]. It is caused by the inability of the adrenal glands to produce cortisol, a hormone fundamental for life, either due to failure of the adrenals (primary AI), or due to maladies influencing the hypothalamus or the pituitary which control the adrenals (secondary AI). Patients with AI require lifelong glucocorticoid (GC) substitution treatment and expanded GC measurements during periods of intercurrent sickness or other major psychological and physical stress to imitate the typical increment in physiological cortisol reaction to such circumstances. Insufficient GC substitution for day by day support and expanded dosages amid sickness, can accelerate an adrenal emergency (AC) an adrenal emergency which can be fatal in case the prompt organization of parenteral hydrocortisone is postponed.

The predominance of essential AI is 93–140 patients/million populace and of auxiliary AI is 150–280/per million. Standard mortality rate for patients with AI is more than twofold compared to the common populace agreeing to review clinic information. AI has noteworthy affect on patients' quality of life, and problematic GC substitution (over- or under-replacement) can lead to intense and long term complications such as osteoporosis and type 2 diabetes.

AI envelops a wide assortment of restorative analyze and can be an unrecognized fundamental condition veiled by another determination in both paediatrics and adults. There ought to be a increased sense of doubt within the introduction of any seriously unwell neonate, child or grown-up where an unexplained introduction, weakening of an intercurrent ailment or other push (e.g. surgery or significant trauma) may have accelerate an AC.

The diagnosis of AI brings numerous challenges for children, parents, adult patients and their families with the affect of a numerous every day

pharmaceutical schedule, and the require for debilitated day reconnaissance and management, and for vigilance to detect potential sickness and conceivable occasions which may be life undermining. Wellbeing experts have to be give satisfactory progressing psychological support and instruction for patients and families long term as they adjust to their health needs of their condition and consolidate treatment plans into their every day lives. Understanding of the instruction handle is crucial and one of the foremost one vital angles of the part of the endocrine nurture.

The determination of adrenal inadequate in the hospitalized patients is challenging not as it were since of the lack of particular indications or clinical signs but moreover due to the challenges setting up cutoff values for cortisol levels in intensely sick patients [10]. Anticipated cortisol levels change with the sort and seriousness of illness, making it troublesome to characterize typical ranges. Patients conceded to the clinic don't have the classical circadian rhythms with higher cortisol levels early in the morning and lower levels at night. Numerous edge levels have been proposed for the definition of an inadequately cortisol level (measured at any time of day) during acute illness, but none is totally palatable. In fundamentally sick patients, cortisol levels are more often than not elevated, and a serum cortisol esteem of ≥ 18 mcg/dL (497 nmol/L) effectively rules out adrenal insufficiency. Patients with central adrenal insufficiency have low cortisol levels with inappropriately normal or low ACTH levels. Given the need of a cutoff esteem for irregular cortisol levels, an ACTH incitement test can be performed to affirm the conclusion of adrenal lacking in the event that basal levels are dubious. Cosyntropin, a engineered ACTH comparing to amino acids 1–24 of ACTH that has full biologic potency, is utilized to assess the capacity of the adrenal organ to deliver cortisol. The ACTH incitement or cosyntropin test comprises of measuring serum cortisol instantly some time recently and 30 and 60 minutes after intravenous or intramuscular infusion of 250 mcg (85 nmol or 40 worldwide units) of cosyntropin. Serum cortisol concentration ≥ 18 to 20 mcg/dL (500 to 550 nmol/L) after the infusion shows typical adrenal function. It is critical to highlight that patients with unused onset of central adrenal insufficiency may have an appropriate response to cosyntropin incitement since the adrenal organ will react to an ACTH boost regularly. Hence, a ordinary reaction to cosyntropin test does not run the show out central adrenal lacking, and physicians will have to be depend on basal cortisol and ACTH levels and clinical judgment. Patients with adrenal decay resulting from chronically moo incitement by endogenous ACTH will have an unusual reaction within the cosyntropin test.

Conclusion

Adrenal hormone deficiency causes different symptoms, depending on the deficiency of which hormone is involved. If the formation of aldosterone is reduced, too much sodium is excreted in the urine, as a result of which the pressure drops and the level of potassium in the serum rises. If the formation of cortisol is significantly reduced, and especially if the production of aldosterone is blocked, within a few days or weeks, life-threatening adrenal gland failure can develop with a severe drop in blood pressure, accelerated heart rate and impaired function of numerous organs.

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