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Association Between Stress and prolactin hormone levels: A Mini-Review

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REVIEW ARTICLE

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Association Between Stress and prolactin hormone levels: A Mini-Review

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Abstract

The physiological functions of prolactin are highly varied, despite the fact that it is most commonly recognized for its participation in the inhibition of reproduction and the regulation of breastfeeding. The conditions known as hyperprolactinemia can have a variety of origins, some of which are physiological and others of which are pathologic. The following are examples of physiological causes: pregnancy, breastfeeding, sleep-related, stimulation of the nipples and sexual orgasm, stimulation or trauma to the chest wall. Another significant physiological factor that contributes to hyperprolactinemia is stress, and the clinical importance of this factor is currently being investigated. This paper aims to elucidate the physiology of prolactin, the influence of stress on its secretion, and the overarching clinical strategy for managing hyperprolactinemia.

Keywords: Galactorrhea, Hyperprolactinemia, Prolactin, Stress

1. Introduction

1.1. Structure of prolactin

The endocrine hormone known as prolactin is the primary hormone that regulates the beginning and continuation of lactation [1]. It is a peptide hormone composed of 198 constituent amino acids. It has been demonstrated that both monomeric and polymeric forms of prolactin can be found in the bloodstream. Chromosome 6 contains the copy of the prolactin gene. The molecular weight (MW) of the monomeric form of prolactin in circulation is 22 kilodaltons (KDA), the molecular weight (MW) of the polymeric form known as "big prolactin" is between 50 and 60 kilodaltone (KDA), and the molecular weight (MW) of the large polymeric form known as macroprolactin is greater than 100 kba. Eighty to ninety –five percent of the prolactin that is detected in the circulation is located in the smallest form, which is physiologically active. Despite the fact that complexed forms of pituitary hormone prolactin, particularly macro-prolactin, have a longer clearance time due to their large size, they do not contribute to the symptoms of hyperprolactinemia because of their lack of biological activity [2, 3]. The very name of this hormone comes from the fact it has the ability to stimulate lactation in response to the stimulation of a baby's sucking. Prolactin, on the other hand, is frequently referred to as the "stress hormone" due to the fact that its levels rise in reaction to a variety of stresses. This is the reason why it is frequently referred to as the stress hormone [4].

1.2. Metabolism of prolactin

The normal range for serum prolactin levels in adults varies across sexes. For women, the range is 10–25 μg/L, and firm men, it is 10–20 μg/L. prolactin production is a process that occurs in a pulsatile manner, much like the secretion of many other hormones. during rapid eye movement (REM) sleep, most secretion happens. The usual peaks happen between

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4.00 and 6.00 AM [5]. The liver and kidneys eliminate prolactin. Circulation prolactin has a half-life of 20–50 minutes, as was previously reported [6].

1.3. Prolactin physiology

Prolactin shares structural similarities with growth and placental lactogen hormones. They are characterized by their shared protein structure as a helix bundle protein, which is characteristic of the prolactin/growth hormone/placental lactogen family. There is a common ancestor gene for all hormones in this family. While most people know that prolactin is produced and secreted by the anterior pituitary and is subject to dopamine-mediated hypothalamic regulation, they may not know that prolactin can also be produced by the central nervous system, the immune system, the uterus, and the mammary glands. These tissues can start producing prolactin in response to stimulation of the nipple, light, smell, or stress [7].

2. Etiology of Hyperprolactinemia

2.1. Stress physiology

The classification of hyperprolactinemia according to etiology is shown in Table 1. Stress is a significant contributing element to the development of depressive illnesses. Stress induces the production of corticotrophin-releasing hormone (CHR) in the paraventricular nucleus (PVN), which stimulates the secretion of adrenocorticotrophic (ACTH) from the pituitary gland. Adrenocorticotropic hormone (ACTH) stimulates the adrenal glands to release glucocorticoids in response. Additionally, a variety of stresses cause the pituitary gland to release PRL [8]. When homeostasis is disrupted by any physical or psychological stimulus, a stress reaction occurs as a combination of the physiological and behavioral changes that occur as a result from exposure to stressors is referred to as the stress response. The sympathetic-adreno-medullar (SAM) axis, the hypothalamic-pituitary-adrenal (HPA) axis, and the immune system are activated by a stress response that is mediated by a complex interaction of neurological, endocrine, and immunological systems [9].

2.2. Relationship linking Prolactin and Stress

The primary players in the hypothalamic-pituitary-adrenal axis are readily identifies as stress hormones. They include corticotrophin-releasing hormone, which stimulates the pituitary gland to release adrenocorticotropic hormone. The latter, in turn, stimulates the synthesis of glucocorticoids by the adrenal cortex. The crosstalk that they have with the adrenal-medullary system is essential to the activation of a response that preserves life, such as the fight-or-flight response [10, 11]. As part of the stress response, prolactin is released. This is clinically relevant because there is a great deal of indications that prolactin plays a major role in the pathology caused by stress. These involve stress-induced dysfunction of epithelial barrier of the trachea, stress-induced dysfunction of intestinal epithelial barrier cardiomyopathy, and psychological stress in the development of cardiovascular pathology [12, 13]. Gaining a more thorough comprehension of the role that prolactin plays in the modulation of emotional stress is relevant and can give better understanding on systemic diseases attributable to hyperprolactinemia. There is a certain amount of evidence to suggest that neuroendocrine changes in dopamine and serotonin that are brought about by stress could be the cause of functional hyperprolactinemia, which in turn could impact prolactin release [14]. Therefore, it is possible that stress has complicated effects that require further research and investigation, and these effects are outside the scope of this review [15]. It has been discovered via psychological study that hyperprolactinemia is more prevalent among those who have mental illnesses than it is among the general population [16]. It is hypothesized that the elevation of dopamine which takes place in psychosis is, in part, a regulatory

Table 1. Classification of hyperprolactinemia according to Etiology.

Pituitary Disease	Hypothalamic Disease	Medications	Neurogenic	Other
•Prolactinomas •Acromegaly •Clinically nonfunctioning pituitary adenomas •Empty Sella syndrome •Hypophysitis	•Meningiomas •Germinomas •Other tumors •Sarcoidosis •Langerhans cell histiocytosis •Neuroaxis irradiation	•Phenothiazines •Butyrophenones •Atypical Antipsychotics •Tricyclic Antidepressants •Serotonin Reuptake Inhibitors •Reserpine •Methyldopa •Verapamil •Metoclopramide	•Chest wall/breast lesions •Spinal cord lesions	•Pregnancy •Breast-feeding •Exercise •Stress •Epileptic seizure •Hypothyroidism •Renal Insufficiency •Hepatic failure •Adrenal insufficiency

response, which down regulates the elevation of prolactin which is produced by stress [17]. Furthermore, women who were exposed to an alcoholic, violent, or missing father throughout their infancy years may be more likely to develop hyperprolactinemia later in life [18].

2.3. Clinical manifestations

Hyperprolactinemia is characterized by galactorrhea, but galactorrhea is not always present in people with hyperprolactinemia. Somewhere between 30% and 80% of individuals with hyperprolactinemia also have galactorrhea [19]. In addition, around 50% of women with galactorrhea have prolactin levels that are within normal limits. Breast atrophy, which is shown in women who are postmenopausal or who have amenorrhoea as a result of gonadotropin deficiency or primary ovarian failure, does not occur in women who have amenorrhea owing to hyperprolactinemia. Gynaecomastia is typically not present in male patients who have hyperprolactinemia, although milk may be released from a male breast that is of a completely normal size. Galactorrhea, which is defined as the production of milk in the absence of pregnancy or breastfeeding, is a relatively uncommon condition in men who have hyperprolactinemia. It affects less than 30% of men with the condition, meaning that it is significantly less prevalent than it is in women. However, the presence of galactorrhea in a male who also has a tumour on their pituitary gland is a significant clinical indication that the individual may have hyperprolactinemia and that there may be prolactinomas [20]. When hyperprolactinemia develops in children who are prepubertal or peripubertal, it may manifest itself through symptoms such as growth arrest, headaches, visual field abnormalities, or delayed or halted puberty. Particularly in boys, prolactinomas that are larger and more aggressive are frequently observed in children and adolescents [21]. Due to the fact that genetic anomalies in lactotroph tumours are found to be highly prevalent in this population, a recent consensus for pituitary adenomas in children and adolescents suggests that genetic testing be provided to all pediatric patients in order to facilitate therapy and family surveillance [22].

3. Conclusion

Although researchers are still trying to pin down exactly how prolactin works, they do know that it rises in response to psychosocial stress-though the exact amount of this response varies greatly from person to person. PRL affects a wide range of physiological systems and is implicated in a number of disorders.

Ethical approval

Not Applicable

Conflict of interest

The authors declare that there is no conflict of interest.

Author's contribution

- **Sara salah:** Conceived the idea for the review article, organized the structure, and contributed to writing the manuscript.
- **Huda S. Abdulghani:** Conducted the literature search, synthesized the findings, and assisted in writing and editing the manuscript.

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