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CUSHING SYNDROME – DISEASE OF BOTH BODY AND MIND. LITERATURE REVIEW

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ABSTRACT

Introduction and purpose: Cushing's syndrome is a complex clinical condition characterized by a constellation of signs and symptoms resulting from prolonged exposure to elevated levels of glucocorticoids. Although it is relatively rare, the disorder is associated with numerous serious complications affecting multiple organ systems. The aim of this study is to present current state of knowledge on clinical features of Cushing Syndrome.
A Brief description of the state of knowledge: Due to hypercortisolemia and potential disturbances in other hormonal networks, Cushing's syndrome may lead to a wide range of both physical and psychological symptoms and signs. Characteristic abnormalities in laboratory findings may also be observed. Symptoms may involve multiple organ systems, including the cardiovascular, immune, reproductive, musculoskeletal systems, as well as the skin. Complications are mainly cardiovascular, thrombotic and infectious. A broad range of neuropsychiatric disturbances may also occur, including, among others, depression and psychosis. It is therefore important to note, that mortality in Cushing's syndrome is not solely attributable to somatic complications but is also associated with an increased risk of suicide. Distinct differences in the clinical presentation of Cushing's syndrome may be observed in pediatric patients. Likewise, elderly may exhibit clinical features that differ from those typically seen in younger adults.
Materials and methods: The comprehensive literature review was conducted using PubMed and Google Scholar databases. The analysis incorporated review papers, case studies, and original research articles addressing possible symptoms, signs, complications, and laboratory abnormalities observed in the course of Cushing's syndrome. Both somatic and psychiatric manifestations of the condition were considered in the selection and evaluation of sources. The analysis encompassed publications available in Polish or English, with a publication date ranging from 2015 to 2025. This time restriction was implemented to ensure the inclusion of the most recent studies reflecting the current state of knowledge.
Conclusion: Cushing syndrome may present with a wide spectrum of clinical manifestations and can lead to multiple complications. Continued medical follow-up is crucial even after the resolution of hypercortisolism, as not all somatic and psychiatric symptoms fully resolve following treatment. Further research is needed to understand the course of CS and potential long-term consequences of the disease. This is essential for improving patient care, as well as prolonging survival and enhancing quality of life.

KEYWORDS

Cushing Syndrome, Symptoms, Signs, Complications, Comorbidities, Laboratory Tests

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1. Introduction and background

Cushing's syndrome refers to a constellation of clinical symptoms resulting from prolonged exposure to excessive levels of glucocorticoids. Although the overall prevalence of Cushing's syndrome in the general population is relatively low, the condition is associated with numerous serious complications. It may occur in both adults and children. When the source of cortisol overproduction is a pituitary adenoma secreting adrenocorticotropic hormone (ACTH), the condition is specifically referred to as Cushing's disease.

The causes of Cushing's syndrome can be classified as either exogenous or endogenous. The most common form is exogenous Cushing's syndrome, which results from prolonged administration of glucocorticoids used to treat various medical conditions. Endogenous causes can be further divided into ACTH-dependent and ACTH-independent forms.

A thorough medical history and physical examination play a fundamental role in the diagnosis of hypercortisolism. Recognizing specific signs and symptoms is crucial, as they often serve as the first indicators pointing toward the need for further diagnostic evaluation. Therefore, a solid understanding of the possible clinical manifestations of Cushing's syndrome is essential, as it may prompt timely and appropriate investigations. In addition to clinical assessment, laboratory testing, imaging studies, and, when necessary,

invasive procedures contribute significantly to establishing the diagnosis. Time to diagnosis can differ for subtypes of CS, remains to be long and requires to be improved [1]. Diagnostic delay in children is not dissimilar to adult population [2].

Due to the systemic effects of cortisol, Cushing's syndrome may present with a wide range of clinical manifestations, particularly related to metabolic dysfunction. It is also important to note that dysregulation of other endocrine axes may occur, including the hypothalamic–pituitary–thyroid and hypothalamic–pituitary–gonadal axes [3] [2]. Importantly, mortality in Cushing's syndrome results not only from acute, life-threatening complications, but also from increased long-term mortality due to multisystem comorbidities associated with the disease [4].

The primary goals of treating patients with Cushing's syndrome are to normalize serum cortisol levels and to alleviate or, if possible, completely eliminate the symptoms and consequences of hypercortisolism. Depending on the underlying cause of the hypercortisolism, treatment strategies may vary.

2. Description of the state of knowledge

2.1. Physical symptoms and signs

2.1.1. Metabolic and cardiovascular symptoms

The metabolic impact of cortisol often manifests in characteristic physical features such as increased body weight, predominant fat deposition in the abdominal region and dorsocervical area (commonly known as the 'buffalo hump'), as well as facial rounding typically described as a 'moon face' [5] [6]. Metabolic disturbances may include diabetes mellitus and insulin resistance, as well as dyslipidemia, characterized by elevated levels of LDL cholesterol, triglycerides, and total cholesterol. These alterations may contribute to the development of atherosclerosis and hepatic steatosis [5] [6][7] [8]. Interestingly, fat accumulation may also occur in the epicardial and pericardial regions, which is clinically relevant due to its potential contribution to the development of cardiomyopathy [9].

Possible complications of hypercortisolism include cardiovascular disorders, among which arterial hypertension is one of the most significant [10] [11]. Moreover, there is an increased risk of cerebrovascular stroke, myocardial infarction, cardiac arrhythmias and heart failure [12] [13] [8] [7][5] [6] [14] [15]. Possible cardiac structural and functional alterations are left ventricular hypertrophy, concentric remodeling, dilated cardiomyopathy, as well as systolic and diastolic dysfunction [6] [7] [16]. Importantly, an adverse cardiovascular disease risk profile may persist even after successful treatment, cardiovascular abnormalities do not reverse completely, and most patients remain overweight or obese even after remission [6][7]. It should be emphasized, that atherosclerotic cardiovascular and cerebrovascular diseases are the main causes of death in Cushing syndrome [17] [6].

Cushing's syndrome (CS) is associated with a markedly increased incidence of venous thromboembolism (VTE) compared to the general population. Hypercoagulability and thromboembolic events are therefore another clinically significant features of CS, especially in the perioperative period and in patients with malignant ectopic ACTH syndrome or adrenocortical carcinoma [13]. There are also independent risk factors of VTE, such as advanced age or reduced mobility [18]. Hypercortisolism provokes a hypercoagulability state because of different underlying mechanisms. They include increased platelets, but also increase in pro-coagulation factors (particularly factor VIII and von Willebrand factor), increased thrombin, fibrinogen and thromboxane 2, shortened activated partial thromboplastin time (aPTT), rapid clot formation, impaired fibrinolysis and prolonged clot lysis time [13] [19] [7]. Compensatory increase in anticoagulation factors such as protein C and S may also occur [19] [7]. It should be emphasized, though, that these changes are not observed in every CS patient [19].

2.1.2 Immunological complications and blood count changes

Cushing's syndrome is associated with an increased incidence of infections, particularly opportunistic ones, as well as an elevated risk of their severe clinical course. Infectious processes can involve various organs and systems, and the potential for sepsis should not be overlooked. Both severe systemic infections as well as chronic or recurring mild infections may be observed [20]. The severity of infection is correlated with the degree of cortisol excess [13]. The main species of opportunistic pathogens reported in patients with Cushing's syndrome are Aspergillus fumigatus, Cryptococcus neoformans, Pneumocystis jirovecii and Nocardia asteroides [13].

Moreover, individuals demonstrate heightened susceptibility to skin damage and a predisposition to the development of poorly healing wounds, which can also serve as portals of entry for infections [21] [22]. For instance, superficial fungal infections of the skin are common in CS [23].

The increased risk of infections has a multifactorial etiology, including the immunosuppressive effects of cortisol, as well as the contribution of coexisting diabetes and vascular insufficiency. Importantly, infections represent a significant cause of mortality in patients with Cushing's syndrome, what highlights the need to prevent and manage them in addition to treating hypercortisolism [17] [13]. By acknowledging the immune alterations, including a more thorough evaluation of signs, symptoms, and predisposing factors and developing strategies to prevent the risk of infections, it may be possible to improve the prognosis, quality of life, and outcome of patients with hypercortisolism [24].

Attention must also be paid to other immunological consequences of Cushing's syndrome. For instance, the remission phase of CS can be associated with the unmasking of autoimmune diseases elicited by reactivation of the previously suppressed immune system [25][24] [26]. The most common autoimmune conditions associated with CS are autoimmune thyroid diseases, but celiac disease, lymphocytic hypophysitis, rheumatoid arthritis, seronegative arthritis, sarcoidosis, systemic lupus erythematosus, vitiligo, atopic dermatitis, psoriasis and others are also possible [24] [27][25][26].

It should be noted that in the course of Cushing's syndrome, changes in both the complete blood count and the differential blood count may be observed. These may include an increased number of leukocytes, neutrophils, and platelets; elevated or decreased levels of hemoglobin and erythrocytes; as well as a reduction in lymphocyte, eosinophil and monocyte count [28] [29] [30] [24] [11]. It is worth highlighting that polycythaemia is considered a more typical symptom than anaemia [28]. Glucocorticoids exert immunomodulatory effects at multiple levels of the immune system, influencing not only immune cells but also modulating proinflammatory and antiinflammatory cytokines [24]. What is worth highlighting, alterations in the blood count are not only laboratory findings but may also have significant clinical implications. For instance, leukocytosis predicts a poorer remission prognosis in CD [30]. Furthermore, lymphopenia associated with Cushing syndrome, accompanied by an altered CD4/CD8 ratio, contributes to increased vulnerability to infections. Elevated neutrophil levels further exacerbate the clinical risk by promoting a prothrombotic state and facilitatating the development of sepsis [24].

2.1.3. Skin lesions

The skin in the course of Cushing's syndrome is thin, dry and atrophic. As a consequence of both epidermal and dermal atrophy, a characteristic 'cigarette paper' appearance of the skin may be observed on the elbows and the dorsum of the hands [22]. Possible cutaneous manifestations of hypercortisolism also include telangiectasias, facial erythema, wide red or reddish-purple striae, and also easily induced petechiae and ecchymoses (resulting from increased capillary fragility) [21] [23]. The changes may involve not only the skin but also its appendages, including manifestations such as hair loss [31]. Primarily due to metabolic changes in ACTH-dependent forms, appearance of skin may manifest as acanthosis nigricans, hiperpigmentation of the skin and tendency to discoloration (particularly in sun-exposed areas)[22][21][23]. As a result of increased adrenal androgen and cortisol secretion, facial acne and hirsutism may appear [22] [23] [11].

2.1.4. Other physical symptoms

Decreased intestinal calcium absorption, along with increased urinary calcium excretion, may contribute to the development of hypocalcemia [32] [33]. Other potential mineral homeostasis disturbances include hypokalemia, hypophosphatemia, and hypernatremia [10] [11] [26]. Cushing syndrome is also associated with an elevated risk of nephrolithiasis [34]. Due to the fundamental role of cortisol in calcium metabolism and its inhibitory effect on osteogenesis, patients may experience bone pain, osteopenia, osteoporosis, as well as an increased susceptibility to fractures, particularly involving the pelvis, ribs, and vertebral bodies [35] [32] [7]. One of the most characteristic manifestations of Cushing syndrome is also skeletal muscle weakness resulting from glucocorticoid-induced myopathy [32] [11]. The most common form is proximal lower limb myopathy, which primarily presents as difficulty climbing stairs and straightening up [36].

It should be noted that the clinical manifestations of CS may arise not only from the direct effects of cortisol excess and adrenal dysfunction, but also from dysregulation of other components of the endocrine system. For instance, the hypothalamus-pituitary-thyroid axis activity can be impaired by the inappropriate cortisol secretion, moreover some drugs used for the medical control of hypercortisolism are associated with alterations in the thyroid function [3].

Elevated cortisol levels, along with concurrent hyperandrogenism, may also adversely affect sexual function. Clinical manifestations may include erectile dysfunction in men, reduced libido, menstrual irregularities (oligo- or amenorrhea), anovulation, virilization, gynecomastia, pubertal hypogonadism, as well as previously mentioned hirsutism and acne [2] [21]. The severity and manifestation of these symptoms may

differ substantially across age groups. Older females with CS tend to report hyperandrogenism-related signs rarely, unlike younger female patients who more often seek medical advice because of them [12].

Symptoms of Cushing's syndrome also include, among others, impaired exercise tolerance, polydipsia, polyuria, and headaches [11]. Among the rare manifestations, gastrointestinal tract perforation has been reported [10].

2.2. Neuropsychiatric symptoms

Hypercortisolism can lead not only to somatic symptoms but also to a range of neuropsychiatric disturbances, including depression, emotional lability, anxiety, irritability, aggression, psychosis, and suicidal thoughts [37] [38] [2] [39] [40]. These symptoms are observed in a substantial proportion of patients with Cushing's syndrome across various age groups. A particularly important issue, especially in elderly patients, is cognitive impairment [12] [41]. In children may be observed difficulty concentrating and a decline in school performance [39]. Psychiatric symptoms may appear even before the full clinical manifestation of Cushing's syndrome (during the prodromal phase), may occur during the course of the disease, and may also persist after successful treatment of CS, contributing to a significant reduction in patients' quality of life [38] [39] [40] [4]. After the successful cure of Cushing's syndrome, a significant recovery of neuropsychiatric function is possible [41]. However, in some cases, despite surgical intervention and remission of Cushing's syndrome, mood and behavior may remain unstable for months or even years [39] [40] [38]. This underscores the need for long-term follow-up, screening for psychiatric disorders, and assessment of suicide risk even after the completion of treatment.

Importantly, suicide accounts for a significant proportion of mortality in patients with Cushing's syndrome [17]. Thus, inadequate diagnosis and suboptimal treatment of psychiatric symptoms may contribute to the overall disease-related mortality and adversely affect the prognosis. Therefore, early recognition of these symptoms and timely initiation of appropriate treatment are essential in patients with Cushing's syndrome. Effective management of CS requires not only surgical and pharmacological interventions, but also the inclusion of psychological support as an integral component of care. Psychological interventions may be required, among other purposes, to treat depression associated with Cushing syndrome and to improve long-term prognosis, subjective well-being, and quality of life [42].

2.3. Clinical differences and characteristics of Cushing syndrome in children and the elderly

Diagnosing Cushing syndrome in children poses a significant challenge, primarily due to the insidious onset of hypercortisolism, the frequent lack of specific early clinical indicators, and the overall rarity of the condition during childhood [2] [43].

In pediatric patients, excessive weight gain is typically the earliest and most prevalent manifestation of Cushing's syndrome. When it occurs in conjunction with growth suppression, it is considered a hallmark feature of the disease [44] [43] [2]. It should be noted, however, that growth impairment is not always reported [45] [2] [46]. This is due to the fact that although reduced growth velocity or growth arrest is a typical manifestation resulting from the inhibitory effect of glucocorticoids on the growth plate, in individuals with coexisting hyperandrogenism, growth may be normal or even increased.

Disruptions of sexual maturation and gonadal function are frequently observed in pediatric and adolescent patients. These may include pseudo-precocious puberty, pubertal delay or arrest, as well as dermatological signs such as hirsutism and acne [44] [45] [23] [46]. In female patients, gynecological abnormalities may be observed, including absence of menarche, irregular menstrual cycles, or secondary amenorrhea [47].

Other typical clinical features commonly observed in pediatric and adolescent populations include plethora, moon face, striae rubrae, acanthosis nigricans, dorsal cervical or supraclavicular fat pads, headaches and osteopenia [2] [47] [23] [46]. Interestingly, striae may differ from those described in adults. Instead of being wide and purple color, they may appear thin and light pink in color (with a similarly lighter appearance on dark skin) [47]. Prepubertal children with Cushing Syndrome typically have also fine downy facial lanugo hair and may have temporal scalp hair regression [23].

Some symptoms may overlap with those observed in older patients. It is important to note that serious metabolic complications and cardiovascular conditions (such as arterial hypertension, hyperglycemia, dyslipidemia, and elevated liver enzyme levels) affect not only adults but also pediatric patients and may require pharmacological intervention despite the young age [47] [43].

Beyond the physical manifestations, children frequently exhibit neuropsychiatric symptoms such as fatigue, diminished energy, cognitive decrement, memory and concentration impairment, decreased attention

span, forgetfulness, and mood disturbances including emotional lability, depression and anxiety [47] [43] [46]. More severe manifestations may include suicidal ideation or psychosis [43] [46].

The clinical presentation of Cushing syndrome in elderly individuals differs significantly from that in younger patients, posing a considerable diagnostic challenge. In older adults, typical features of hypercortisolism, such as skin thinning, easy bruising, striae rubrae and hirsutism, are often absent [12] [48] [49]. Their body mass index or waist circumference tends to be lower compared to younger patients, and they are also less frequently obese [12] [48] [49].

Elderly with Cushing syndrome more frequently experience muscle wasting and weakness, osteoporosis and bone fractures than their younger counterparts [36] [48] [12] [49]. It should be noted that myopathy and potential fractures may significantly impair functional capacity in older adults, deteriorate their quality of life, and lead to increased dependency on others.

In comparison to younger patients, they demonstrate a higher prevalence of type 2 diabetes, hypertension, cardiovascular disease and venous thromboembolism [18] [12] [48] [49].

3. Conclusions

There are numerous sources available in the literature describing the clinical aspects of Cushing's syndrome. This condition can lead to both somatic and psychiatric symptoms, as well as a wide range of systemic complications. The clinical presentation of Cushing's syndrome is directly influenced by the effects of cortisol on the human body. Symptoms may not only result from excess cortisol but also from concomitant adrenal androgen overproduction and dysregulation of other endocrine axes, such as the hypothalamic–pituitary–thyroid axis.

Recognizing the signs and symptoms of the disease is the first step toward initiating comprehensive diagnostic evaluation and appropriate treatment. Therefore, a thorough understanding of the clinical course of the disease is essential. The timing of diagnosis and prompt therapeutic intervention are crucial, as they significantly influence the prognosis, improve outcomes and quality of life. Further research is needed to better delineate the natural history of Cushing's syndrome and its potential long-term consequences.

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