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PRACA POGLĄDOWA
REVIEW

Sheehan syndrome – not a mystery anymore

Zespół Sheehana – coraz mniej tajemniczy

Dariusz Szczuraszek[1], Paulina Soja[1], Dariusz Kajdaniuk[2]

[1]Students' Scientific Club, Department of Pathophysiology, Faculty of Medical Sciences in Zabrze, Medical University of Silesia, Katowice, Poland

[2]Department of Pathophysiology, Faculty of Medical Sciences in Zabrze, Medical University of Silesia, Katowice, Poland

ABSTRACT

Sheehan syndrome is a condition of postpartum failure or complete necrosis of the pituitary gland in women, caused by a postpartum hemorrhage. It is most often chronic and reveals itself many years after delivery. Women develop pituitary insufficiency as a result of postpartum hemorrhage, leading to a deficiency of one or more pituitary hormones. Sheehan syndrome is rare, and its diagnosis and course are influenced by the quality of obstetric care provided. Frequent growth hormone deficiency causes metabolic complications, e.g., lipid profile abnormalities or insulin resistance. The disease also affects the mental health of patients, reducing quality of life. The basic form of treatment is pharmacological supplementation of the deficient hormones. Sheehan syndrome is an increasingly recognizable disease which – properly diagnosed and treated – will not significantly affect the patient's health. An essential adverse factor is late diagnosis, which reduces the effectiveness of treatment and risks the development of serious complications. Articles about Sheehan syndrome, postpartum hemorrhage and pituitary insufficiency were searched in the PubMed database using following keywords in English: "Sheehan syndrome," "treatment," "pituitary dysfunction," "hemorrhage," "postpartum hemorrhage," and "cardiovascular." Materials from the library collection of the Medical University of Silesia Faculty of Medical Sciences in Zabrze were also used. The purpose of this article is to summarize the current state of knowledge about Sheehan syndrome in terms of its epidemiology, etiology, treatment methods, and complications.

KEYWORDS

Sheehan syndrome, hormones, obstetric care, hemorrhage

STRESZCZENIE

Zespół Sheehana jest stanem poporodowej niewydolności lub całkowitej martwicy przysadki mózgowej u kobiet, wywołanej krwotokiem poporodowym. Najczęściej ma charakter przewlekły i ujawnia się wiele lat po porodzie. U kobiet w wyniku krwotoku poporodowego dochodzi do rozwoju niewydolności przysadki, skutkującej niedoborem jednego lub kilku hormonów przysadki. Zespół Sheehana występuje rzadko, a na jego rozpoznawalność i przebieg wpływa jakość świadczonej opieki położniczej. Częsty niedobór hormonu wzrostu przyczynia się do powikłań metabolicznych, np. zaburzeń profilu lipidowego lub insulinooporności. Choroba wpływa również na zdrowie psychiczne pacjentek, obniżając jakość ich życia. Podstawową formę leczenia stanowi farmakologiczne uzupełnienie

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Address for correspondence: Dariusz Szczuraszek, Studenckie Koło Naukowe, Zakład Patofizjologii, ul. Jordana 19, 41-800 Zabrze, tel. +48 32 275 59 30, e-mail: dszczuraszek@gmail.com

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niedoboru hormonów. Zespół Sheehana to coraz bardziej rozpoznawalna jednostka chorobowa, która – prawidłowo zdiagnozowana i leczona – nie wpłynie znacząco na stan zdrowia pacjentki. Istotnym czynnikiem rokowniczo niekorzystnym jest często zbyt późne postawienie diagnozy, co zmniejsza skuteczność leczenia i zwiększa ryzyko wystąpienia poważnych powikłań. Przeszukano bazę danych PubMed pod kątem artykułów na temat zespołu Sheehana, krwotoku poporodowego i niewydolności przysadki, używając słów kluczowych w języku angielskim: „Sheehan syndrome", „treatment" „pituitary dysfunction", „hemorrhage", „postpartum hemorrhage" i „cardiovascular". Wykorzystano także materiały ze zbioru biblioteki Śląskiego Uniwersytetu Medycznego − Wydziału Nauk Medycznych w Zabrzu. Celem pracy jest podsumowanie aktualnego stanu wiedzy na temat zespołu Sheehana w zakresie jego epidemiologii, etiologii, metod leczenia oraz powikłań.

SŁOWA KLUCZOWE

zespół Sheehana, hormony, opieka położnicza, krwotok

Introduction

Sheehan syndrome (or Sheehan's syndrome) is a condition of postpartum insufficiency of the anterior or both anterior and posterior lobes of the pituitary gland (panhypopituitarism) in women as a result of postpartum hemorrhage [1,2]. To this day, postpartum hemorrhage, which underlies Sheehan syndrome, remains one of the leading causes of maternal death [3]. Sheehan syndrome is often diagnosed many years after the hemorrhage occurs [4]. The most common symptoms include lactation problems, menstrual disorders, or even an absence of menstruation [5]. Progressive pituitary insufficiency results in symptoms characteristic of deficiency of individual pituitary hormones, leading primarily to dysfunction of the thyroid, adrenal glands, and gonads [6]. Treatment is mainly based on supplying the deficient hormones [2]. Sheehan syndrome is still not well-known, but better understood than in the past. There is a growing awareness in the medical community of the risks of diagnosing Sheehan syndrome too late and the health implications it can have on women.

Pathomechanism

The pituitary gland of pregnant women can almost double in size. There is an increase in both the volume of the entire gland and the number of pituitary cells [7]. The main cause of this process is hyperplasia of the primarily prolactin-producing cells (lactotrophs) and an increase in the number of other anterior lobe building cells. Hyperplastic cells show a higher demand for metabolites, but this does not entail an enhanced blood supply in the gland – that remains unchanged. The low--pressure circulatory system which supplies the anterior lobe with sufficient nutrients becomes inefficient during pregnancy, which promotes ischemia and subsequent necrosis of the gland. It is assumed that the more severe the postpartum bleeding, the more advanced the necrosis of the pituitary cells. Due to the high-pressure nature of the blood supply, the posterior part of the pituitary gland is not affected by shock or hypovolemia [8]. In cases of massive hemorrhage and increasing failure of the anterior lobe, the posterior pituitary lobe may fail [9].

Excessive hemorrhage during childbirth results in pituitary stroke, from which Sheehan syndrome develops. As a result of pituitary insufficiency, a number of endocrine glands become dysfunctional [10]. Sheehan syndrome can also develop during physiological blood loss while in labor. It is assumed that the disease may have an autoimmune etiology, which is suggested by the presence of antibodies against hypothalamic and pituitary cells in patients diagnosed with Sheehan syndrome [11,12].

Epidemiology

A population-based study conducted in Spain in 2001 showed that the prevalence of hypopituitarism was 45.5 per 1,000,000 and the incidence was 4.2 new cases per 1,000,000 per year [2]. Pituitary tumors are the most common causes of hypopituitarism; Sheehan syndrome accounts for only 6%–8% of all causes of hypopituitarism. The incidence of Sheehan syndrome among postpartum women over 19 years of age was 3.1%. On the other hand, in countries with better obstetric care, in 2009 only, the rate was 5.1 per 100,000 women [2]. It should be mentioned that Sheehan syndrome is a rare condition that consequently may be confused with other diseases, such as lymphocytic pituitary glanditis.

Due to better access to medical care, the disease is rare in developed countries. However, it still occurs at a higher prevalence in developing countries – India, for example – as a result of the high number of home births. Such labor conditions are associated with a higher risk of complications, including postpartum hemorrhage [13].

Clinical features

Sheehan syndrome is caused by developing pituitary insufficiency. It can affect the functioning of the anterior lobe only, resulting in an isolated deficiency of one or several hormones. It can also be total, when both lobes of the pituitary gland become inefficient, and the level of all pituitary hormones falls (panhypopi-tuitarism) [8]. The clinical feature of Sheehan syndrome is nonspecific, depending on the nature of the condition (acute or chronic) and includes symptoms of pituitary insufficiency and a number of general symptoms. Symptoms associated with anterior pituitary

lobe dysfunction include lactation insufficiency due to hypoprolactinemia, fatigue and weakness, menstrual cycle disorders, persistent irregular cycle, and thermoregulatory disorders. However, the most characteristic symptoms are lactation problems. In laboratory tests, hyponatremia is observed. If damage to the posterior lobe occurs, diabetes insipidus develops [13,14,15,16]. In the acute form, hypotension, postnatal hyponatremia, headache, tachycardia, and hypoglycemia are predominant. It is a life-threatening condition [14,15,16]. One of the most serious complications of acute Sheehan syndrome is adrenal crisis. If this occurs, it is necessary to compensate for adrenal hormone deficiencies as soon as possible [10].

Fortunately, the acute form of Sheehan syndrome is encountered much less frequently than the chronic form. In most patients, the average time between hemorrhage and the first symptoms is several months or even many years after delivery [16]. Any physician should be aware of the significant variability of the syndrome's symptoms. Thus, each case should be considered on an individual basis, taking into account the patient's general condition and possible comorbidities [16,17].

Predisposing factors

The development of Sheehan syndrome is known to be influenced by massive postpartum hemorrhage, resulting in subsequent hypopituitarism and pituitary necrosis. According to recent reports, a higher risk of postpartum hemorrhage was observed in a group of pregnant women taking medications with psychotropic effects, particularly selective serotonin reuptake inhibitors (SSRIs) and benzodiazepines [18]. In addition, the risk increases when several drugs are used in combination. However, this is the only report of such side effects of taking drugs with psychotropic effects during pregnancy.

Excessive coagulation

Excessive blood clotting after postpartum hemorrhage has not been shown to be directly associated with an increased risk of Sheehan syndrome. It is well known that the pituitary gland shows higher estrogen sensitivity during pregnancy [19]. One of estrogen's mechanisms is to increase the synthesis of thrombin − a prothrombotic factor in the clotting cascade. As coagulation factor IIa (formed from factor II − prothrombin), it causes the conversion of soluble fibrinogen into insoluble fibrin, which has a prothrombotic effect. However, studies have not shown significantly higher levels of this factor in women with Sheehan syndrome. The role of tumor necrosis factor alpha (TNF-α) was also considered. An association between elevated levels of this cytokine and increased risk of thrombosis has been detected, but, in the context of Sheehan syndrome, this hypothesis has not been confirmed [20].

Quality of post-obstetric care

To effectively diagnose and treat Sheehan syndrome, a multidisciplinary approach is required. It is necessary for internists, endocrinologists, gynecologists, and obstetricians to work together. Because of the chronic nature of the disease and the significant interval between the onset of postpartum hemorrhage and the first symptoms, a good, reliable patient history is the most important factor. Information about the course of pregnancy, possible complications during pregnancy or during labor, and the cause of postpartum hemorrhage can help the physician select effective treatment and avoid the serious complications of untreated or poorly treated Sheehan syndrome. According to a study conducted in Norway in 2020––2021, where 12 maternity wards were inspected, the quality of obstetric care plays a significant role [21]. The study showed that regardless of the size of the facility, the incidence of postpartum hemorrhage in patients is influenced by the quality of services provided by the obstetric staff. Comparing the facilities included in the study, a lower rate of failures per 1,000 deliveries was observed in facilities where the national recommendations for staff training and skill development were followed.

Sheehan syndrome from a gynecological perspective

Postpartum necrosis of the pituitary gland due to hemorrhage causes it to fail, resulting in a deficiency of all pituitary hormones. These include folliculotropic hormone (FSH) and luteinizing hormone (LH), gonadotropic hormones which regulate the secretory function of the ovaries. Disruption of the hypothalamic--pituitary-gonadal axis therefore requires external supplementation of deficient hormones to restore homeostasis [22]. Patients with Sheehan syndrome require hormone replacement therapy to correct hormonal deficiencies and prevent the further complications of hypoestrogenism: primarily excessive bone loss and impaired bone remodeling [23].

To effectively prevent Sheehan syndrome, its immediate cause – postpartum hemorrhage – must be prevented. After the onset of hemorrhage, in addition to determining the cause of bleeding, appropriate treatment should be implemented immediately and the volume of lost blood should be replenished. The patient's condition should also be constantly monitored. The complete procedure for treating a hemorrhage is described in the recommendations of the Polish Society of Gynecologists and Obstetricians [24].

Despite hormonal and gynecological abnormalities, patients with Sheehan syndrome can consider becoming pregnant. A factor that improves the prognosis is early detection of the disease and appropriate and prompt treatment [11]. Patients can also undergo ovulation induction. Cases of spontaneous

pregnancy in patients with Sheehan syndrome have also been described. This phenomenon is not completely explained, but most likely caused by improving pituitary function and partially inhibited gonadal function [16,25].

Quality of life of patients with Sheehan syndrome

Mental disorders may be the first symptom of Sheehan syndrome; they may also appear during treatment [26]. The incidence is about 25%–40%. Studies have shown that in patients with Sheehan syndrome, lower serum sodium levels are associated with a higher risk of mental disorders [26]. Depression, anxiety, fear, and hallucinations may occur. Particularly severe symptoms have been observed mainly in patients additionally diagnosed with hypothyroidism. Thyroid hormone supplementation led to improvement in the patients' condition, indicating a link between hormonal deficiencies and the mental state of patients with Sheehan syndrome. Hyponatremia may also play an important role, but in this case delirium is more common. Of lesser but nevertheless noticeable importance may be pituitary and gonadal insufficiency [27]. The greater the failure of the anterior pituitary lobe, the more severe the psychiatric symptoms. Stress factors, such as infection or tissue damage, lead to elevated levels of inflammatory factors (TNF-α and IL-6), which increases the inflammatory response in patients with psychiatric disorders [26].

A rapid increase in sodium level increases the risk of mental disorders, likely due to sudden changes in osmotic pressure in the brain. Therefore, sodium correction should be carried out gradually, strictly following the latest management protocols [26].

Patients with Sheehan syndrome often experience apathy, depressed mood, sexual dysfunction, difficulties in social relationships, problems with cognitive function, and reduced quality of life [28]. Endocrine disorders which accompany Sheehan syndrome can lead to acute neuropsychiatric symptoms. It remains unclear what their long-term effects may be, especially in the context of treatment noncompliance [29].

Metabolic disorders

Metabolic disorders in Sheehan syndrome are often associated with a deficiency of growth hormone (GH). Among other things, patients exhibit improper body composition and an abnormal lipid profile, such as higher total cholesterol and triglyceride counts and a higher low-density lipoprotein (LDL) fraction, along with a lower high-density lipoprotein (HDL) fraction [30]. It is also often accompanied by osteoporosis, which, in addition to GH deficiency, is exacerbated by hypoestrogenism [23,30]. Patients with Sheehan syndrome show elevated leptin levels; obese women have higher levels than women with normal body mass index (BMI) [30]. The problem of high BMI in women with Sheehan syndrome is increasingly common. It is likely due to abnormal eating habits and the overlapping multiple hormonal disorders present in patients with Sheehan syndrome.

Laway and Baba [31] noted a higher prevalence of metabolic-associated fatty liver disease in a group of patients with Sheehan syndrome compared to a group of healthy women of the same age and BMI. High BMI and GH deficiency are two major factors in the development of steatosis, both of which often accompany Sheehan syndrome. There are indications that insulin-like growth factor (IGF-1) is effective in improving liver function in non-alcoholic steatosis by reducing reactive oxygen species and decreasing triglyceride accumulation. This study was conducted on rats with GH deficiency [32].

Other common concomitant disorders are diabetes and abnormally high postprandial and fasting glucose levels. Compared to a control group, patients with Sheehan syndrome have been reported to have higher homeostatic model assessment for insulin resistance scores, which translates into a tendency toward insulin resistance [31].

The metabolic disorders described above entail a number of implications and complications. They negatively affect the overall well-being of women with Sheehan syndrome, increase the risk of osteoarticular dysfunction, and promote obesity and cardiovascular complications.

Differential diagnosis

When Sheehan syndrome is suspected, other diseases should be considered: postpartum autoimmune pituitary inflammation, pituitary tumor, cancer metastasis, lymphocytic pituitary inflammation, amyloidosis, sarcoidosis, chronic infections, human immunodeficiency virus, rheumatic diseases, and hemochromatosis [11].

It is very important to differentiate Sheehan syndrome from Addison's disease. Adrenal insufficiency develops in both of these conditions, but Sheehan syndrome and lymphocytic pituitary glanditis occur after childbirth, while Addison's disease manifests during pregnancy. Lymphocytic pituitary inflammation should also be excluded. Sheehan syndrome is characterized by a generalized deficiency of many pituitary hormones, while lymphocytic pituitary inflammation usually involves a deficiency of only a few pituitary hormones, especially adrenocorticotropic hormone (ACTH) [33]. Whenever Sheehan syndrome is diagnosed, empty sella syndrome should be kept in mind [34]; Sheehan syndrome is one of the causes of secondary empty sella syndrome [35].

Treatment

The basic treatment of Sheehan syndrome is hormone supplementation [2,8]. Most common are deficiencies of thyroid hormones, adrenal hormones, and hormones secreted by the gonads, so patients are given appropriate substitutes: levothyroxine, possibly liothyronine (thyroid hormone substitutes), hydrocortisone or prednisone (adrenal hormone substitutes), and

the sex hormones estrogen and progesterone [2,8, 22]. Treatment of the adrenal glands has a priority in the treatment of overall hormonal deficiencies. Glucocorticosteroids should be administered first, and only then should other hormones be supplemented [2,10]. If necessary, it is advisable to administer increased doses of glucocorticosteroids intravenously [22].

Hormone substitution shows the greatest benefit in the treatment of chronic Sheehan syndrome; treatment of the acute form poses more difficulties. Therefore, if possible, it is better to prevent the acute form of the disease. One of the causes of acute Sheehan syndrome is hypotension. Preventing the patient from developing hypotension and anemia can reduce the risk of acute Sheehan syndrome [17]. Sometimes blood transfusion is necessary if blood loss is significant [25].

In GH-deficient Sheehan syndrome, the supply of GH has also a positive effect. The dose should be based on the patient's condition, adjusted to her current needs [8,34]. The use of GH results in improved lipidogram parameters – higher HDL-C levels and lower fractions of total cholesterol, LDL-C, and triglycerides – and in expanded exercise capacity. Thus, GH supplementation is a strongly recommended therapy in Sheehan syndrome patients with GH deficiency and a high risk of cardiovascular disease [31,36]. Side effects can include elevated IGF-1 levels and decreased insulin sensitivity, which can cause fluctuations in blood glucose levels [37]. The aforementioned decreased insulin sensitivity may result not from taking GH per se, but from overusing it [31].

Sheehan syndrome is one of the most common causes of hypoprolactinemia and lactation problems are its main symptom [14,38,39]. Studies have shown the efficiency of recombinant prolactin in treating lactation insufficiency in women with both low and normal prolactin levels. Although this hormone is not the only factor affecting lactation, an improvement has been observed after the supply of a recombinant form of it [39].

Conclusions

Sheehan syndrome remains a diagnostic and therapeutic challenge today. The non-specificity of the symptoms and the delayed onset of the disease mean that it is often diagnosed when long-term complications occur. Prevention of the primary cause of Sheehan syndrome, postpartum hemorrhage, seems to be very important. Successful control of a hemorrhage promises to be beneficial by significantly reducing the risk of Sheehan syndrome in postpartum women. Given the etiology of the condition and the growing interest in home births among pregnant women, it is worth considering whether there is a correlation between home births and an increased risk of postpartum hemorrhage. However, if Sheehan syndrome develops, the priority should be to prevent adrenal crisis, which remains one of the most serious complications.

Authors' contribution

Study design – D. Szczuraszek, P. Soja, D. Kajdaniuk
Data collection – D. Szczuraszek, P. Soja
Manuscript preparation – D. Szczuraszek, P. Soja, D. Kajdaniuk
Literature research – D. Szczuraszek, P. Soja
Final approval of the version to be published – D. Kajdaniuk

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