MEDICINE & PUBLIC HEALTH
e-ISSN 2956-9443

2025, Vol. 3, No. 2
DOI: 10.31749/MandPH-AFMKU-25-2-003
Received: 2025-11-11
Accepted: 2026-01-16

CASE REPORT

Chronic adrenal insufficiency (Addison's disease) – diagnostic obstacles, problems of the patient and his or her family

Anna Lidia Krzentowska[1,A-C,E-F], *Patryk Hebda*[2,D]

[1] Department of Endocrinology and Internal Medicine, Medical College, Andrzej Frycz Modrzewski Krakow University, Poland

[2] Students' Scientific Interest Group, Department of Endocrinology and Internal Medicine, Medical College, Andrzej Frycz Modrzewski Krakow University, Poland

A – Research concept and design, B – Collection and/or assembly of data, C – Data analysis and interpretation, D – Writing the article, E – Critical revision of the article, F – Final approval of the article

Abstract

Addison's disease (AD) is a relatively uncommon illness presented by decreased adrenal production of adrenocortical hormones - mainly glucocorticoids. We present a case of a patient who had symptoms of chronic fatigue, depression that made it difficult for her to carry out everyday household activities. In basic laboratory tests, they mainly noted low sodium (Na), increased potassium (K) and low fasting glucose concentration. The patient's informed consent was obtained for the presentation of anonymised laboratory, clinical and imaging data. On the basis of the clinical picture and hormonal tests, Addison's disease was confirmed. Significant clinical improvement was observed following the addition of hydrocortisone to the treatment regimen. Advances in optimizing treatment for patients with Addison's disease have enabled them to lead normal lives. However, it is important to continuously educate patients and healthcare professionals about the ever-present threat of adrenal crisis.

Keywords: Addison's disease, primary adrenal insufficiency, hyperkalemia, hyponatremia, adrenal crisis

Introduction

Addison's disease (AD) was first described in 1855 by Thomas Addison as a syndrome characterized by wasting and hyperpigmentation occurring with adrenal damage [1]. However, it was not until 1949 that Edward Calvin Kendall developed a method to synthesize cortisone, a therapeutic breakthrough in the treatment of the disorder. Nowadays, this relatively rare disease, an uncontrolled course of which threatens the life of the patient, still causes many challenges. AD is a syndrome of clinical symptoms caused by primary adrenal insufficiency resulting in a deficiency of adrenal cortex hormones, particularly cortisol. The most common cause of the

✉ Anna Lidia Krzentowska, e-mail: akrzentowska@uafm.edu.pl

disease is autoimmune-mediated atrophy of the adrenal cortex due to the presence of anti-adrenal antibodies (often with coexisting autoimmunity to thyroid cells), and less commonly adrenal tuberculosis and other infectious diseases, destruction of the adrenal cortex by tumours or tumour metastases, amyloidosis, a condition after adrenal haemorrhage, bilateral adrenalectomy or drug-induced inhibition of glucocorticosteroid synthesis [2]. AD is manifested by nonspecific symptoms. The main symptoms are hyponatremia and hyperkalaemia in laboratory tests, a tendency towards hypoglycaemia (especially post-workout), low blood pressure, and characteristic darkening of the skin [3]. The main complaint reported by patients is excessive fatigue, even with low-level exertion, constant weakness and periodic fainting with orthostatic etiopathogenesis, weight loss, lack of appetite, salt craving, loose stools, muscle and joint pain [4,5]. The symptoms may initially resemble depression. Hormonal tests show low cortisol values and high levels of adrenocorticotropic hormone (ACTH) resulting from the lack of inhibition by cortisol (negative cortisol feedback mechanism on the pituitary gland and hypothalamus) [2]. To determine the secretory reserve of the adrenal cortex, a test with a synthetic derivative ACTH (Synacthen) is used.

Treatment of the disease consists in supplementing the missing hormones, primarily hydrocortisone.

Case presentation

For several years a 40-year-old female, the mother of three children, had experienced chronic fatigue, lethargy, extreme drowsiness and palpitations on slight exertion, e.g. when making breakfast for the children. She was tearful, had problems with climbing up to the first floor caused by significant fatigue, she had hyperpigmentation of the skin with reduced prominence of the hand creases (figures no. 1 and 2), light sensitivity, skin that burnt easily due to exposure to sunlight, and repeated convulsions lasting a few minutes. The symptoms worsened after the birth of her third child (five years ago). In addition, she experienced weight loss (more than 10 kg in six months), pain in the lower limbs after exertion, memory and concentration difficulties, attention problems, depression, anxiety, insomnia, and an excessive desire to eat salty food; the family reported that food prepared by her was oversalted. The patient was negatively perceived by the family and community due to her lack of strength to do any work at home. The patient felt an improvement in her well-being after drinking electrolyte preparations.

Figure 1. Author's own photographs – comparison of the hand of a patient with Addison's disease with the hand of a doctor – note the dark complexion of the patient's skin

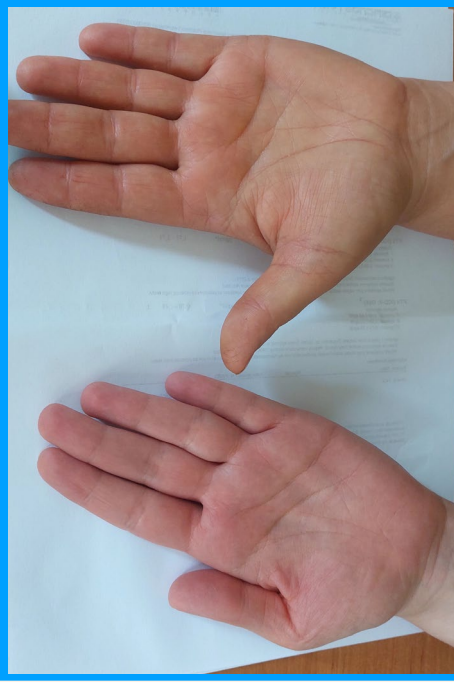

Figure 2. Author's own photographs – comparison of the hand of a patient with Addison's disease with the hand of a doctor – note the dark complexion of the patient's skin

In the physical examination, the following symptoms were noteworthy: hyperpigmentation of the skin, less prominent finger lines and creases on the hands, dimming of the nipples, low blood pressure of 90/60 mmHg, and a heart rate of 70 per minute.

After the birth of her third child, the patient experienced symptoms of very high fatigue and depression. A baseline blood count was ordered, but the results showed no abnormalities. Repeatedly low blood sodium and elevated potassium concentrations were also present in laboratory tests. The patient began to report multiple depressive disorders, balance problems, anxiety, memory and concentration difficulties. She was referred by a general practitioner to a psychiatrist, who prescribed multiple antidepressants. Treatment with antidepressants did not bring clinical improvement.

She was admitted to the Hospital Emergency Department in July 2018 due to dyspnoea occurring with very slight exertion, and limited tolerance to any exertion. Laboratory tests were notable for hyponatremia (Table 1).

Table 1. Laboratory tests after admission to Emergency Department in 2018

Laboratory tests (reference range)	Laboratory results
Na (136–145 mmol/l)	129.0 mmol/l
K (3.5–5.1 mmol/l)	4.84 mmol/l
Cl (98–107 mmol/l)	98.0 mmol/l
Ca (2.15–2.50 mmol/l)	2.51 mmol/l
CRP (0–5 mg/l)	<1.0 mg/l
GFR (>60 ml/min/1.73m^2)	70.0 ml/min/1.73m^2
Creatinine (0.50–0.90 mg/dl)	0.94 mg/dl
Glucose (70.0–99.0 mg/dl)	112 mg/dl
D-Dimers (0–499 ng/ml)	254 ng/ml

Legend: Na – sodium; K – potassium; Cl – chloride; Ca – calcium; CRP – C-reactive proteine; GFR – glomerular filtration rate.

In 2018 and 2019, there were two more hospitalizations due to a sudden deterioration in health status. The laboratory tests are shown in Table 2.

Table 2. Laboratory tests after admission to Hospital in 2019

Laboratory tests (reference range)	Laboratory results
Na (136–145 mmol/l)	137.0 mmol/l
K (3.50–5.10 mmol/l)	5.12 mmol/l
Ca (2.15–2.50 mmol/l)	2.35 mmol/l
Fe (5.83–34.50 mmol/l)	2.92 mmol/l
Glucose (3.9–5.5 mmol/l)	3,75 mmol/l
Thyroid hormonal tests	TSH – 7.49 uIU/ml (ref. range: 0.27–4.2) fT4 – 15.42 pmol/l (ref. range: 12.0–22.0) fT3 – 4.48 pmol/l (ref. range: 3.1–6.8) anti TPO – 546.0 IU/ml (ref. range <34) anti TG – 98.0 IU/ml (ref. range <115).
Cortisol (2.3–19.4 µg/dl)	at 8 a.m. 1.02 µg/dl
Diurnal cortisol profile	at 6 a.m. cortisol – 0.88 ug/dl at 8 a.m. cortisol – 0.52 ug/dl at 12 a.m. cortisol – 1.45 ug/dl
ACTH (7.2–63.0 pg/ml)	> 2000.0 pg/ml
DHEAS (60–337ug/dl)	5.76 µg/dl
Anti-adrenal antibodies	positive ++

Legend: ACTH – adrenocorticotropic hormone; DHEAS – dehydroepiandrosterone sulphate; TSH – thyroid-stimulating hormone; fT4 – thyroxine; fT3 – triiodothyronine; Anti-TPO – Antithyroid peroxidase antibodies; Anti-TG – thyroglobulin antibodies; Fe – ferrous.

The patient received the following treatment: hydrocortisone (pills) at a dose of 10–10–0 mg/day; fludrocortisone at a dose of 0.1 mg – ½ pill/day in the morning; prasterone in pills at a dose of 10 mg/day. Then L-thyroxine treatment was added, which resulted in improved well-being, complete resolution of excessive fatigue, lethargy, brightening of the skin, no seizures, no memory impairment, no dizziness, and resolution of depression and anxiety. The psychiatrist decided to discontinue all antidepressants and anti-anxiety medication. The patient reported no psychiatric symptoms. There was a significant improvement in blood pressure, which was oscillating around the reference range of 120/80 mmHg. Currently the patient is feeling very well and has stopped fainting. Blood laboratory tests

after treatment revealed an improvement in the sodium level in the serum and increased levels of glucose.

An abdominal CT scan performed in 2019 showed small narrow adrenal glands without focal changes. Thyroid ultrasonography revealed inhomogeneous and decreased echogenicity. The thyroid volume was 7.7 ml (left lobe 2.6 ml; right lobe 5.1 ml). The patient was trained to increase the dose of hydrocortisone in special situations, i.e. in the case of fever, illness, vomiting, nausea, minor and major procedures. The patient's family was also trained on how to manage this disease.

Discussion

This case represented non-specific initial symptoms, which included chronic excessive fatigue and fainting. The patient was treated psychiatrically due to the significant presence of negative symptoms. The ineffectiveness of the diagnosis contributed to the lack of improvement in health status and only attempted to alleviate psychiatric symptoms. Over time, some symptoms became more acute, and hospitalization in emergency departments was necessary. The case teaches us that when a patient complains of chronic fatigue and excessive fatigue despite little effort, chronic adrenal insufficiency should also be considered in a differential diagnosis. It is necessary to determine the concentrations of electrolytes (Na, K) and glucose, the concentrations of which may suggest the development of the disease. Clinical assessment of the patient (low blood pressure, darkening of the skin, hypersensitivity to the sun) is important. Establishing the cortisol and ACTH concentrations allows AD to be diagnosed. Elevated ACTH was caused by the lack of inhibition due to insufficient cortisol concentrations (the pituitary-adrenal axis is regulated according to negative feedback) [6–8]. The lack of cortisol was the cause of hyponatremia and hyperkalaemia. Undiagnosed or inadequately treated AD leads to the risk of the development of an adrenal crisis, which is a directly life-threatening condition. The main symptoms are severe weakness, impaired consciousness, vomiting, loose stools, low blood pressure accompanied by tachycardia and shock. Treatment consists of the rapid administration of hydrocortisone: 100 mg iv as a bolus, followed by a 100 mg iv infusion every six hours and the supply of fluids and glucose [9]. Patients diagnosed with AD should be informed about the risk of adrenal crisis and its prevention. According to a multicentre study conducted in the UK, Canada, Australia, and New Zealand, about 8% of patients diagnosed with adrenal insufficiency are hospitalized for adrenal crisis annually [10]. It is necessary to educate patients about the need to increase the dose of hydrocortisone in the event of diseases that, according

to retrospective studies, contribute the most to the occurrence of adrenal crisis (gastroenteritis (35–45%) and fever (17–24%), trauma, surgery, dental procedures and situations of severe mental stress) [6,11]. A doctor noticing a low concentration of sodium, a high concentration of potassium and a low concentration of fasting glucose in a patient with symptoms of chronic fatigue should pay particular attention to skin pigmentation and low blood pressure values and, suspecting AD, should refer the patient to an endocrinologist for an appropriate diagnosis. When diagnosed, it is necessary instruct the patient and his or her family about the procedure in the case of fainting, sport activity and the "sick days" rules.

Conclusions

Advances in optimizing treatment for patients with Addison's disease have enabled them to lead normal lives. However, it is important to continuously educate patients and healthcare professionals about the ever-present threat of adrenal crisis.

Declaration of interest

The authors declare that there is no conflict of interest that could be perceived as prejudicing the impartiality of the research reported.

The patient's informed written consent to publish the data was obtained.

References

1. Arlt W, Allolio B. Adrenal insufficiency. *Lancet*. 2003;361(9372):1881–1893, https://doi.org/10.1016/S0140-6736(03)13492-7.
2. Hellesen A, Bratland E, Husebye ES. Autoimmune Addison's disease – An update on pathogenesis. *Ann Endocrinol* (Paris). 2018 Jun;79(3):157–163, https://doi.org/10.1016/j.ando.2018.03.008.
3. Mosca AM, Barbosa M, Araújo R, Santos MJ. Addison's Disease: A Diagnosis Easy to Overlook. *Cureus*. 2021;13(2):e13364, https://doi.org/10.7759/cureus.13364.
4. Bornstein SR, Allolio B, Arlt W, Barthel A, Don-Wauchope A, et al. Diagnosis and Treatment of Primary Adrenal Insufficiency: An Endocrine Society Clinical Practice Guideline. *J Clin Endocrinol Metab*. 2016;101(2):364–389, https://doi.org/10.1210/jc.2015-1710.
5. Esposito D, Bobbio E, Di Fraia R, Mone P, Accardo G, et al. Patients with adrenal insufficiency have cardiovascular features associated with hypovolemia. *Endocrine*. 2020;70(2):412–420, https://doi.org/10.1007/s12020-020-02458-3.

6. Dineen R, Thompson CJ, Sherlock M. Adrenal crisis: prevention and management in adult patients. *Ther Adv Endocrinol Metab*. 2019;10, https://doi.org/10.1177/2042018819848218.
7. Saverino S, Falorni A. Autoimmune Addison's disease. *Best Pract Res Clin Endocrinol Metab*. 2020;34(1):101379, https://doi.org/10.1016/j.beem.2020.101379.
8. Martin-Grace J, Dineen R, Sherlock M, Thompson CJ. Adrenal insufficiency: Physiology, clinical presentation and diagnostic challenges. *Clin Chim Acta*. 2020;505:78–91, https://doi.org/10.1016/j.cca.2020.01.029.
9. Rushworth RL, Torpy DJ, Falhammar H. Adrenal Crisis. *N Engl J Med.* 2019 Aug 29;381(9):852–861, https://doi.org/10.1056/NEJMra1807486.
10. White K, Arlt W. Adrenal crisis in treated Addison's disease: a predictable but under-managed event. *Eur J Endocrinol*. 2010;162(1):115–120, https://doi.org/10.1530/EJE-09-0559.
11. Betterle C, Presotto F, Furmaniak J. Epidemiology, pathogenesis, and diagnosis of Addison's disease in adults. *J Endocrinol Invest*. 2019;42(12):1407–1433, https://doi.org/10.1007/s40618-019-01079-6.