Edelweiss Applied Science and Technology
ISSN: 2576-8484
Vol. 9, No. 1, 920-927
2025
Publisher: Learning Gate
DOI: 10.55214/25768484.v9i1.4271

Successful recognition of emergency conditions of primary adrenal insufficiency in a nine-month-old girl: A case report from a tertiary hospital in Indonesia

Karina Pharamita Dewi[1,2], Rayi Kurnia Perwitasari[1,2], Yuni Hisbiyah[1,2], Muhammad Faizi[1,2], Nur Rochmah[1,2*]
[1]Department of Child Health, Faculty of Medicine, Universitas Airlangga, Surabaya, Indonesia; kadedewi1117@gmail.com (K.P.D.) rayi.kurnia.p@gmail.com (R.K.P.) yuni.hisbiyah@gmail.com (Y.H.) muhammad.faizi@fk.unair.ac.id (M.F.)
[2]Department of Child Health, Dr. Soetomo General Academic Hospital, Surabaya, Indonesia; nur-r@fk.unair.ac.id (N.R.).

Abstract: Diagnosis of Adrenal Insufficiency (AI) in children is often challenging due to the relatively low incidence and non-specific presentation, so misdiagnosis often occurs, especially in emergency settings. Prompt recognition and management are crucial to reducing the morbidity and mortality linked to this potentially fatal illness, emphasizing the importance of heightened awareness among healthcare professionals. A 9-month-old girl was brought to the ER with a chief complaint of excessive vomiting. The girl presented with hyperpigmentation, leading to a suspicion of primary adrenal insufficiency secondary to Addison's disease. Laboratory findings revealed electrolyte imbalances (hyperkalemia and hyponatremia) and metabolic acidosis due to acute kidney injury secondary to fluid loss, prompting intravenous fluid therapy and hydrocortisone administration. Following hospitalization and comprehensive evaluation, including tuberculosis screening and abdominal ultrasound, the patient showed clinical improvement and was discharged with hydrocortisone and fludrocortisone as maintenance therapy. Regular follow-ups in the endocrinology clinic ensured continued management and adjustment of medication doses to maintain stability. Adrenal insufficiency may present with a spectrum of symptoms, including gastrointestinal complaints, and its diagnosis hinges on identifying specific hormone deficiencies. Primary adrenal insufficiency (PAI) often associated with mineralocorticoid deficiency, manifests with signs such as hypotension and electrolyte abnormalities, necessitating urgent evaluation and treatment.

Keywords: *Addison's disease, Children, Management, Primary adrenal insufficiency.*

1. Introduction

Adrenal insufficiency (AI) is a subtle diagnosis that might be confused with other dangerous endocrine illnesses, such as sepsis, metabolic problems, or cardiovascular disease. Pediatricians face a unique problem when dealing with subclinical adrenal insufficiency, which is the early stage of acute AI [1]. AI can result from various congenital or acquired diseases involving the hypothalamus, pituitary, or adrenal cortex. Primary Adrenal Insufficiency (PAI) results from the adrenal cortex's loss or failure, whereas secondary AI is caused by pituitary or hypothalamic illness. AI must be diagnosed and treated promptly to reduce morbidity and mortality [2].

Adrenal insufficiency can be either primary or secondary. PAI is a disorder caused by decreased steroid synthesis, adrenal breakdown, or aberrant gland growth in the adrenal cortex [3]. Addison Disease is the name given to acquired primary artificial intelligence. Central Adrenal Insufficiency (CAI) is caused by an inability of adrenocorticotropic hormone (ACTH) to produce or release. It can be caused by a pituitary condition (secondary AI) or by reduced hypothalamic corticotropin-releasing hormone

History: Received: 6 December 2024; Revised: 2 January 2025; Accepted: 10 January 2025; Published: 15 January 2025
* Correspondence: nur-r@fk.unair.ac.id

(CRH) release (tertiary adrenal insufficiency). Every incidence of adrenal insufficiency (AI) during the neonatal period or the first few months of life should be explored for an underlying hereditary etiology. However, AI is uncommon at this age (1:5000-10.000) [1].

Primary adrenal insufficiency is a rare illness, incidence of about 93-140 per 1,000,000 people [4, 5]. Congenital Adrenal Hyperplasia (CAH) is the leading cause of primary AI in children for 70% of cases, followed by autoimmune adrenalitis (Addison's disease) at 15%. The most common cause of CAH is a 21-hydroxylase deficiency for approximately 90% of all CAH cases and occurs in one out of every 14,000 live births [6]. Addison's disease is a rare, lifelong chronic endocrine condition with an incidence of 39 per million in Western countries. It is the leading cause of PAI in adolescents and adults. However, it is uncommon in children, with a prevalence of one in 10,000 [7]. The most prevalent cause of this illness is autoimmune adrenal cortex destruction, which causes a drop in glucocorticoids, mineralocorticoids, and adrenal androgen levels. Other reasons include TB, adrenal hemorrhage, and metastatic cancer. This condition has been linked to a family history and is more prevalent in women [8].

Addison's disease often begins with glucocorticoid deficit and progresses to mineralocorticoid deficiency, but it can also appear acutely, particularly during concomitant diseases. The appearance of adrenal insufficiency differs depending on the rate and severity of adrenal function impairment [9] [10]. Despite its rarity, Addison's disease can have serious effects if not detected and treated properly. The condition's insidious nature frequently delays identification until an acute adrenal crisis occurs, emphasizing the need for enhanced awareness within interprofessional healthcare teams to improve patient outcomes [2].

To increase our knowledge and awareness in diagnosing PAI, we report the case of a 9-months-18 days old girl who presented to the emergency room with recurrent vomiting, general weakness, and hyperpigmentation, which after a series of clinical and laboratory workups was found to be caused by PAI, with high suspicion towards Addison's Disease.

2. Case Illustration

AA, a 9-month-old girl, was admitted to the ER of Dr. Soetomo General Academic Hospital with a chief complaint of continuous vomiting since the morning prior; she was brought to the ER by her parents. Continuous vomiting persists every time she is given food or drink, leading to weakness, accompanied by twice-daily diarrhea starting two days before admission. Additionally, her parents noted progressive skin darkening with no concurrent fever, cough, or runny nose. The parents revealed that this condition had recurred several times since birth and has led to frequent hospitalizations in her place of origin due to unexplained vomiting, sometimes accompanied by diarrhea.

She was born by spontaneous labor, a term, and cried spontaneously, with a birth weight of 3,400 g and 50 cm in length, no cyanosis nor jaundice, and prominent dark skin. Still, her parents didn't initially realize this characteristic. She is the second child of two siblings, both alive and healthy, and her mother had a history of good antenatal care by a midwife. She has a complete immunization history, and a well-formed BCG scar was formed. The patient was exclusively breastfed until three months of age, was given additional formula milk from three months until six months, and began consuming additional food at seven months. The recorded growth and developmental milestones were appropriate for her age.

Physical examination revealed blood pressure of 80/32 mmHg (systolic Blood Pressure $<50^{th}$ percentile, diastolic Blood Pressure $<50^{th}$ percentile), heart rate was 120 bpm, palpable, regular, strong pulse, with warm extremities, capillary refill time was less than 2 seconds, spontaneous respirations with a frequency of 30 times per minute, temperature 36.9°C and saturation was 99% with 2 lpm of oxygen through a nasal cannula. There was generalized hyperpigmentation. Other physical and neurological examinations remain normal. From the endocrinology physical examination, we found Tanner 1, with no ambiguous genitalia in this patient. Her weight measured 6.6 kg, indicating a standard weight-for-age Z-score (WAZ), and her body length was 69 cm, with a standard length-for-age Z-score (LAZ). For the weight-for-length plotting, her nutritional status was classified as wasted. The body surface area was 0.356 m^2.

Edelweiss Applied Science and Technology
ISSN: 2576-8484
Vol. 9, No. 1: 920-927, 2025
DOI: 10.55214/25768484.v9i1.4271

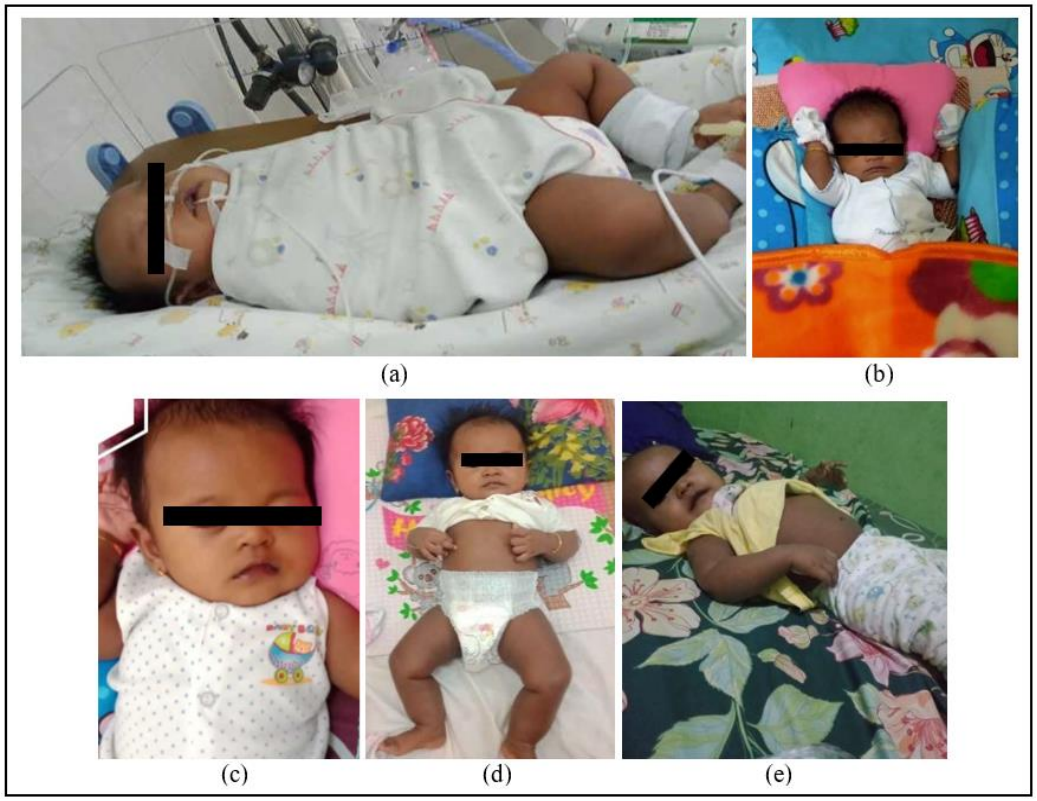

Figure 1.
(a). The patient was born with darker skin, (b). The patient's clinical feature in four months old, (c). The patient is two months old, (d). The patient in three months old, (e). The patient is six months old.

In the ER, the patient underwent laboratory examinations, including a complete blood count test, which revealed Hb 10,6 gr/dl, HCT 32%, WBC 20.340 u/L, PLT 257.000 u/L. Serum electrolyte was K^+ 6,5 mmol/L, Na^+ 124 mmol/L, Cl^- 88 mmol/L, and Ca^{2+} 8,9 mg/dL. Renal and liver function tests were also ordered: BUN 53 mg/dL, Cr 2,18 mg/dL (GFR 16.9), AST 61 U/L, ALT 41 U/L. Random blood glucose was 248 gr/dl. Additionally, arterial blood gas analysis revealed pH 7,27, pCO2 24, pO2 135, BE 15,9, HCO3 11, 17-OH Progesterone test <0.23 ng/ml (normal range ≤ 1.7 ng/ml), ruling out the diagnosis of Congenital Adrenal Hyperplasia. Additionally, a bone age radiology examination showed developmental equivalence to that of a 0 – 6 month-old girl.

These findings indicated leukocytosis, hyperkalemia, hyponatremia, metabolic acidosis, and acute kidney injury, which was attributed to excessive vomiting. To address the condition, the patient received rehydration and continued with maintenance intravenous fluid therapy (IVFD) and a loading dose of 50 mg hydrocortisone administered in the ER. Before this current admission, the patient had a history of recurrent hospital visits due to similar symptoms. According to information provided by the parents, previous examinations of serum electrolytes indicated the presence of hyponatremia, hyperkalemia, and episodes of hypoglycemia, mirroring the current clinical presentation.

Edelweiss Applied Science and Technology
ISSN: 2576-8484
Vol. 9, No. 1: 920-927, 2025
DOI: 10.55214/25768484.v9i1.4271

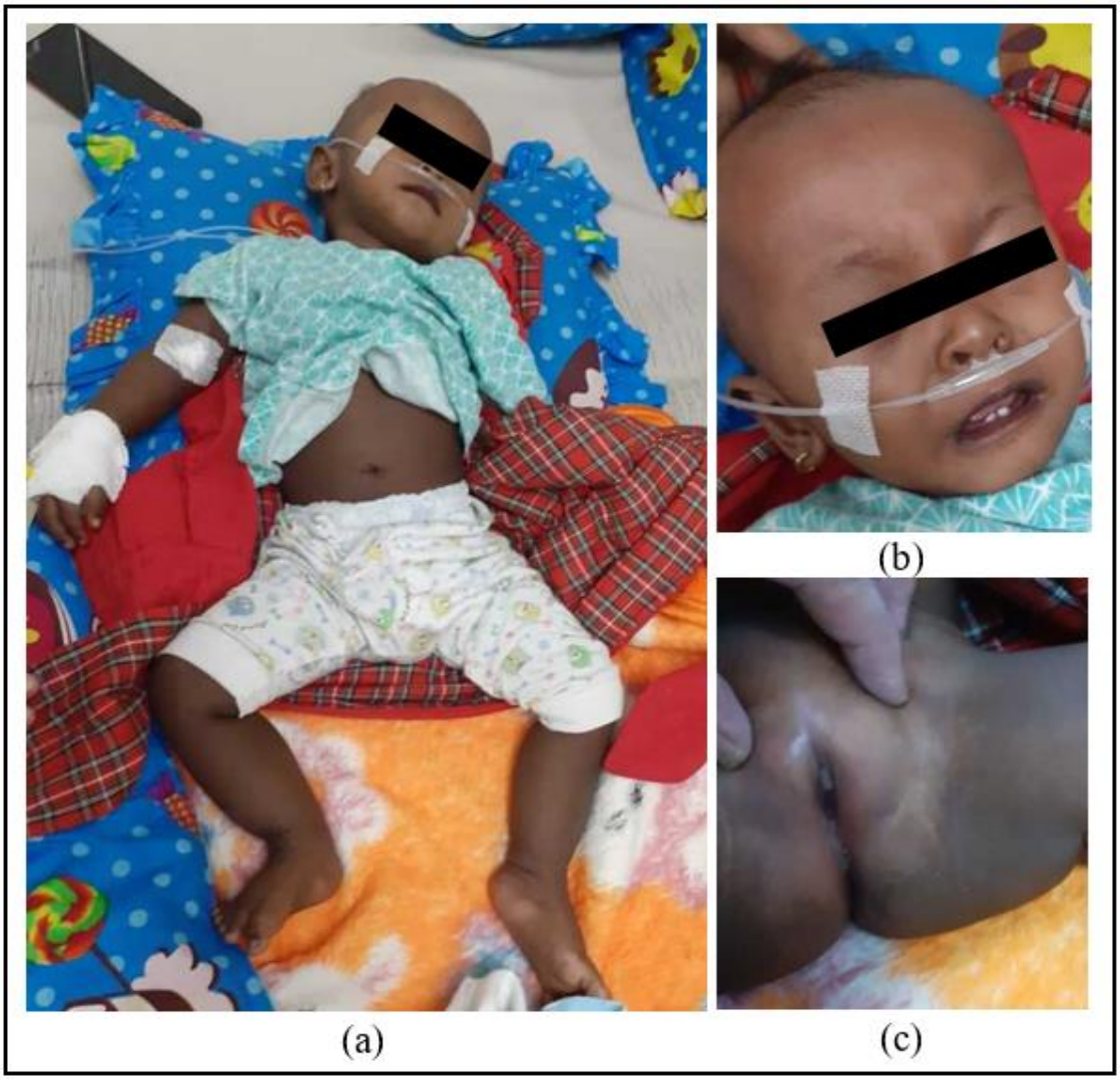

Figure 2.
(a). Patient's clinical features during presentation to the Emergency Department of Dr. Soetomo General Academic Hospital; (b) Patient has dark buccal mucosa and gums; (c). There were no ambiguous genitalia.

After stabilizing, the patient was transferred to low care and was given definitive therapy hydrocortisone injections with a gradually tapering dose until the third day of treatment, followed by maintenance hydrocortisone orally at 5 mg every 8 hours. Additional tests were conducted upon admission to the ward, including morning cortisol plasma, thyroid function tests, tuberculosis workup examination, and ultrasound to rule out any underlying causes of injury or bleeding.

Additionally, we collected a blood sample when the patient's blood glucose was low, registering at 37 mg/dL at 06:30 a.m., with a cortisol level of 25 nmol/L. Blood examination revealed hemoglobin at 9.8, hematocrit at 27.1, and white blood cell count at 10,240. Further tests included thyroid function (FT4 1.23; TSH 5.855) and electrolyte levels, and renal function was improved. A urine examination was also revealed normal result.

We conducted a Mantoux test for tuberculosis screening, which showed no induration, and performed a gene expert test on gastric aspirate, yielding a *Mycobacterium tuberculosis* (MTB) not

Edelweiss Applied Science and Technology
ISSN: 2576-8484
Vol. 9, No. 1: 920-927, 2025
DOI: 10.55214/25768484.v9i1.4271

detected. Abdominal ultrasound findings were normal, showing no inflammation or bleeding in the adrenal gland. However, due to the patient's condition, we have not yet conducted autoimmune testing.

After a five-day treatment at the ward, the patient gradually improved. Upon discharge, the patient was prescribed hydrocortisone at 3 mg every 8 hours, fludrocortisone at 0.15 mg every 8 hours, and salt tablets. Following discharge, the patient regularly visited the endocrinology outpatient clinic. As the condition improved, the dosage of hydrocortisone was gradually reduced to 1.5 mg every 8 hours and fludrocortisone to 0.1 mg every 8 hours. Currently, the patient's condition has gradually improved, with no recurrent admissions.

3. Discussion

Primary adrenal insufficiency is a diagnostic difficulty for doctors because of its uncommon occurrence and nonspecific clinical symptoms. It may develop slowly over time and cause delays in diagnosis, especially in emergency settings with sudden life-threatening illnesses [11]. Patients with chronic primary adrenal insufficiency may exhibit hyperpigmentation, weakness and exhaustion, loss of body weight, loss of appetite, gastrointestinal issues, hypotension, salt seeking, and postural changes. General hyperpigmentation of the skin and mucous membranes is an early and classic sign of Addison's disease caused by elevated ACTH levels. As a result, it does not appear in secondary AI. Hypotension occurs in 90% of cases and is followed by orthostatic symptoms, which can progress to shock in severe chronic or acute illnesses [2].

In this case, the patient was born with darker skin, initially causing some concern as it differed from her sibling's skin tone at birth. However, the parents did not recognize the significance of this difference at the time. It was only as the skin continued to darken over time and was accompanied by recurrent weakness and difficulty gaining weight that they began to suspect a more serious underlying condition, which prompted further specialty consultation. Along with preceding skin and mucosal hyperpigmentation symptoms present since birth, the array of recurrent hospitalizations with the same symptoms raises suspicion of primary adrenal insufficiency.

The clinical manifestations of adrenal insufficiency are contingent upon the specific hormones affected and the extent of the deficiency. ACTH encompasses glucocorticoids (e.g., cortisol), mineralocorticoids (e.g., aldosterone), and adrenal androgens (e.g., androstenedione, dehydroepiandrosterone). One or more of these hormones in PAI may have deficiencies. Conversely, individuals with secondary or CAI typically do not exhibit mineralocorticoid deficiencies or experience hyperpigmentation related to ACTH [12].

The presentation of PAI in children typically involves nonspecific symptoms such as fatigue, nausea, and vomiting, with additional manifestations specific to the affected hormones. Signs of mineralocorticoid deficiency, such as hypotension, dehydration, and electrolyte imbalances, are commonly observed [12]. The underlying cause of AI determines the specific type and severity of the deficiency. In our case, the diagnosis of PAI was confirmed through a comprehensive evaluation, including a history review, physical examination, and additional laboratory and radiological assessments.

Acute adrenal crisis is characterized by life-threatening, rapid clinical deterioration in patients with AI and hypotension. Other clinical symptoms that may occur include fever, dehydration, nausea and vomiting, anorexia, weakness, low glucose level, and a decrease in consciousness [13]. The appearance of general skin and mucosal hyperpigmentation in patients with unexplained vascular collapse supports PAI. In our situation, dehydration may have contributed to the beginning of the adrenal crisis. With no temperature, infection was ruled out as the underlying cause. In addition, the patient's normal urine examination and complete blood count levels helped to rule out other possible explanations.

PAI clinical symptoms are caused by glucocorticoid and mineralocorticoid deficiencies. Signs of glucocorticoid insufficiency include weakness, anorexia, and weight loss. Hypoglycemia with normal or low insulin levels is common and often severe in the pediatric population. Mineralocorticoid deficiency

Edelweiss Applied Science and Technology
ISSN: 2576-8484
Vol. 9, No. 1: 920-927, 2025
DOI: 10.55214/25768484.v9i1.4271

can cause tachycardia, hypotension, hyperkalemia, hyponatremia, acidosis, and salt cravings. The higher ACTH levels are due to a lack of glucocorticoid-negative feedback [14].

PAI is diagnosed based on a low morning (basal) cortisol hormone level (either serum or plasma), verified by low cortisol levels measured using corticotropin stimulation assays. PAI is suspected when basal cortisol levels are less than 140 nmol/L (5 μg/dL) and plasma ACTH concentrations are twice the upper limit of normal. The corticotropin stimulation test, often known as the cosyntropin or ACTH test, is considered the gold standard for diagnosing PAI [15]. This test should confirm the diagnosis of PAI in patients with clinical signs and symptoms unless the basal cortisol levels show AI. Unfortunately, doing this test in Indonesia is difficult since drugs are scarce. As a result, it is recommended that therapy be initiated in patients experiencing severe AI or adrenal crisis without waiting for the results of a confirmatory test. There is currently no test marker available to confirm an adrenal crisis. Thus, the diagnosis is entirely dependent on the patient's clinical presentation [16].

In our case, PAI was diagnosed based on a physical examination, recurrent hyponatremia, hyperkalemia, hypoglycemia, normal 17-OHP, a very low morning cortisol level in a hypoglycemia condition (25 nmol/L), and the presence of hyperpigmentation on the skin and mucosa from birth, which is a characteristic clinical sign of PAI produced by high ACTH levels. This condition is very pathognomonic, and one may imagine an AI having a strong suspicion of Addison's disease.

Adrenal insufficiency might manifest as non-specific clinical symptoms. As a result, a single cortisol measurement is frequently included in the biochemical work-up of a sick child [17]. Although a confirmatory evaluation with a corticotropin stimulation test was not possible in our case due to availability constraints, this did not delay the start of glucocorticoid administration as the treatment for our patient. The rapid administration of intravenous hydrocortisone resulted in significant clinical improvement for our patient, confirming the diagnosis of PAI. Other laboratory or radiological investigations have a limited impact on confirming the diagnosis of AI, but they play a more crucial role in defining the cause. After verifying primary adrenal insufficiency based on hormonal levels, the next step is to identify the underlying problems [18].

Glucocorticoid treatment is indicated for all patients suffering from PAI. The recommended regimen is 15-25 mg hydrocortisone or 20-35 mg cortisone acetate in two or three divided oral dosages per day. Prednisolone (3-5 mg/day) is an alternate regimen that is delivered orally in one or two doses [19].

Dexamethasone should not be administered in primary adrenal insufficiency due to the potential of a cushingoid effect caused by difficulty in titrating dexamethasone dosages. Adjusting the hydrocortisone dose to body surface area (5.5 mg/m2) or body weight (0.12 mg/kg) may result in higher physiological cortisol levels in primary adrenal insufficiency patients compared to fixed-dose regimens [20]. Monitoring therapy and adjusting an oral dose are also dependent on clinical presentations. In our scenario, the patient has an adrenal crisis, and the initial treatment consists of providing 50 mg of parenteral hydrocortisone alongside fluid resuscitation with isotonic saline. When the patient's condition stabilizes on the ward, the dosage of parenteral hydrocortisone is maintained intravenously at 15 mg every 8 hours. On the third day of hospitalization, the dosage is gradually reduced to 7.5 mg every 8 hours. The transition to oral medication depends on the patient's clinical progress, with oral hydrocortisone starting on the fourth day of hospitalization at a dosage of 7.5 mg every 8 hours.

Upon discharge, the patient was prescribed hydrocortisone at 3 mg every 8 hours, fludrocortisone at 0.15 mg every 8 hours, and salt tablets. Following a one-week evaluation, the patient returned to the Endocrinology outpatient clinic for a follow-up, where the doses were adjusted in response to her clinical improvement. The hydrocortisone dose was reduced to 1.5 mg every 8 hours, and the fludrocortisone dose was reduced to 0.1 mg every 8 hours.

Education for caregivers and parents is crucial to prevent adrenal crisis. Parents should be prompted to recognize their signs and symptoms and equipped with a steroid emergency card outlining sick day rules. Prescribing physicians should also ensure caregivers receive adequate training in

Edelweiss Applied Science and Technology
ISSN: 2576-8484
Vol. 9, No. 1: 920-927, 2025
DOI: 10.55214/25768484.v9i1.4271

administering hydrocortisone emergency self-injections and provide additional oral glucocorticoids as necessary [21].

4. Conclusion

This case report discusses the diagnostic challenges of primary adrenal insufficiency in pediatric patients. Clinical recognition and early intervention, especially in emergencies, are very important to prevent poor outcomes because misdiagnosis often occurs. Immediate management, including fluid resuscitation and glucocorticoid therapy, is essential to overcome severe electrolyte imbalance and prevent acute adrenal crisis. Treatment and regular monitoring are essential to optimize long-term outcomes in patients with adrenal insufficiency.

Funding:

There was no specific funding for this case report.

Author's Contributions:

The authors contributed to all study processes, including data collection and final manuscript preparation.

Conflict of Interest:

The authors declare no conflicts of interest regarding the article's publication.

Transparency:

The authors confirm that the manuscript is an honest, accurate, and transparent account of the study; that no vital features of the study have been omitted; and that any discrepancies from the study as planned have been explained. This study followed all ethical practices during writing.

Acknowledgments:

The authors would like to thank Dr. Soetomo General Academic Hospital, Universitas Airlangga, for supporting this work and their generous offer to publish this article.

References

[1] D. Nisticò, B. Bossini, S. Benvenuto, M. C. Pellegrin, and G. Tornese, "Pediatric adrenal insufficiency: Challenges and solutions," *Therapeutics and Clinical Risk Management*, pp. 47-60, 2022. https://doi.org/10.2147/tcrm.s294065

[2] S. A. Bowden and R. Henry, "Pediatric adrenal insufficiency: Diagnosis, management, and new therapies," *International Journal of Pediatrics*, vol. 2018, no. 1, p. 1739831, 2018. https://doi.org/10.1155/2018/1739831

[3] E. Charmandari, N. C. Nicolaides, and G. P. Chrousos, "Adrenal Insufficiency," *The Lancet*, vol. 383, no. 9935, pp. 2152-2167, 2014.

[4] K. Løvås and E. S. Husebye, "High prevalence and increasing incidence of Addison's disease in western Norway," *Clinical Endocrinology*, vol. 56, no. 6, pp. 787-791, 2002. https://doi.org/10.1046/j.1365-2265.2002.t01-1-01552.x

[5] R. Perry, O. Kecha, J. Paquette, C. Huot, G. Van Vliet, and C. Deal, "Primary adrenal insufficiency in children: Twenty years experience at the Sainte-Justine Hospital, Montreal," *The Journal of Clinical Endocrinology & Metabolism*, vol. 90, no. 6, pp. 3243-3250, 2005. https://doi.org/10.1210/jc.2004-0016

[6] P. Speiser, "Congenital adrenal hyperplasia due to steroid 21-hy-droxylase deficiency: an Endocrine Society clinical practice guideline," *Journal Clin Endocrinol Metab*, vol. 95, no. 9, pp. 4133-4160, 2010.

[7] T. M. Laghari, M. N. Ibrahim, Z. Khoso, M. I. Hanif, N. Meher-Un, and J. Raza, "Spectrum of addison's disease in children," *Journal Coll Physicians Surg Pak*, vol. 30, pp. 1086-1099, 2020.

[8] G. Meyer, K. Neumann, K. Badenhoop, and R. Linder, "Increasing prevalence of Addison's disease in German females: Health insurance data 2008–2012," *European Journal of Endocrinology*, vol. 170, no. 3, pp. 367-373, 2014. https://doi.org/10.1530/eje-13-0756

[9] T. Yamamoto, "Latent adrenal insufficiency: Concept, clues to detection, and diagnosis," *Endocrine Practice*, vol. 24, no. 8, pp. 746-755, 2018. https://doi.org/10.4158/ep-2018-0114

[10] S. Choudhury and K. Meeran, "Glucocorticoid replacement in Addison disease," *Nature Reviews Endocrinology*, vol. 14, no. 9, pp. 562-562, 2018. https://doi.org/10.1038/s41574-018-0049-6

[11] G. Patti *et al.*, "Central adrenal insufficiency in children and adolescents," *Best practice & Research Clinical Endocrinology & Metabolism*, vol. 32, no. 4, pp. 425-444, 2018.

[12] S. M. Mimi and M. Kim, "Clinical manifestations and diagnosis of adrenal insufficiency in children," *Wolters Keluwer*, vol. 67, no. 2, pp. 28–33, 2023.

[13] J. Park, M. Didi, and J. Blair, "The diagnosis and treatment of adrenal insufficiency during childhood and adolescence," *Archives of Disease in Childhood*, vol. 101, no. 9, pp. 860-865, 2016. https://doi.org/10.1136/archdischild-2015-308799

[14] C. Betterle, F. Presotto, and J. Furmaniak, "Epidemiology, pathogenesis, and diagnosis of Addison's disease in adults," *Journal of Endocrinological Investigation*, vol. 42, pp. 1407-1433, 2019. https://doi.org/10.1007/s40618-019-01079-6

[15] S. Cortez, A. M. Arbeláez, M. Wallendorf, and K. McNerney, "Peak serum cortisol cutoffs to diagnose adrenal insufficiency across different cortisol assays in children," *Journal of Clinical Research in Pediatric Endocrinology*, vol. 15, no. 4, pp. 375-379, 2023. https://doi.org/10.4274/jcrpe.galenos.2023.2023-2-3

[16] A. C. Willis and F. P. Vince, "The prevalence of Addison's disease in Coventry, UK," *Postgraduate Medical Journal*, vol. 73, no. 859, pp. 286-288, 1997.

[17] M. Shaunak, J. C. Blair, and J. H. Davies, "How to interpret a single cortisol measurement," *Archives of Disease in Childhood-Education and Practice*, vol. 105, no. 6, pp. 347-351, 2020. https://doi.org/10.1136/archdischild-2019-318431

[18] N. W. Wina Dharmesti, M. R. Saraswati, K. Suastika, W. Gotera, and I. M. P. Dwipayana, "Challenging diagnosis of Addison's disease presenting with adrenal crisis," *Case Reports in Endocrinology*, vol. 2021, no. 1, p. 7137950, 2021. https://doi.org/10.1155/2021/7137950

[19] S. R. Bornstein *et al.*, "Diagnosis and treatment of primary adrenal insufficiency: an endocrine society clinical practice guideline," *The Journal of Clinical Endocrinology & Metabolism*, vol. 101, no. 2, pp. 364-389, 2016. https://doi.org/10.1210/jc.2015-1710

[20] P. Peterson, J. Pitkänen, N. Sillanpää, and K. Krohn, "Autoimmune polyendocrinopathy candidiasis ectodermal dystrophy (APECED): A model disease to study molecular aspects of endocrine autoimmunity," *Clinical & Experimental Immunology*, vol. 135, no. 3, pp. 348-357, 2004. https://doi.org/10.1111/j.1365-2249.2004.02384.x

[21] H. Simpson, J. Tomlinson, J. Wass, J. Dean, and W. Arlt, "Guidance for the prevention and emergency management of adult patients with adrenal insufficiency," *Clinical Medicine*, vol. 20, no. 4, pp. 371-378, 2020. https://doi.org/10.7861/clinmed.2019-0324

Edelweiss Applied Science and Technology
ISSN: 2576-8484
Vol. 9, No. 1: 920-927, 2025
DOI: 10.55214/25768484.v9i1.4271