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CASE IMAGE

Primary adrenal insufficiency

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Abstract

Primary adrenal insufficiency is a potentially life-threatening condition. We report a case of a 49-year-old female patient who presented to the hospital for evaluation of dizziness, nausea, and vomiting. Darkening of the palmar creases and tongue was noted. The adrenocorticotropic hormone stimulation test confirmed the diagnosis of adrenal insufficiency.

1 | CASE REPORT

A 49-year-old female patient with a history of Hashimoto thyroiditis presented to the hospital for the evaluation of dizziness, nausea, and vomiting. The patient was hypotensive. Darkening of the palmar creases (Figure 1) and tongue (Figure 2) was noted. Laboratory data: serum so-dium 110 mEq/L, potassium 5.1 meq/L, serum osmolality 239 mosm/kg, urinary osmolarity 381 mosm/kg, and urinary sodium 94 meq/L.

What's the most likely diagnosis?

A Dehydration

B Syndrome of inappropriate antidiuretic hormone secretion

- C Primary adrenal insufficiency
- D Primary polydipsia

Correct answer: C. Primary adrenal insufficiency.

2 | DISCUSSION

The combination of nausea, vomiting, hypotension, hyponatremia, darkening of the palmar creases, and tongue merits high suspicion for primary adrenal insufficiency due to Addison's disease (choice C). While dehydration (choice A) is a common cause of hyponatremia, associated urinary sodium is frequently <25 mEq/L. Syndrome of inappropriate antidiuretic hormone secretion is a diagnosis

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FIGURE 1 Dark palmar creases



FIGURE 2 Dark tongue

of exclusion (choice B). The patient did not have a history of excessive water intake (choice D). The adrenocorticotropic hormone stimulation test was performed, and the diagnosis of adrenal insufficiency was confirmed (Table 1).¹ Hyperpigmentation of the skin and tongue is caused by excessive adrenocorticotropin hormone binding

Laboratory test	05:30 h	06:00 h	06:30 h	Units and reference range
Cortisol, 0' before cosyntropin	0.2			µg/dl
Cortisol, 30' post cosyntropin		0.2		hg/dl
Cortisol, 60' post cosyntropin			0.2	μg/dl
Aldosterone, 60' post cosyntropin			<1	lb/gn
Plasma renin activity	1.98			0.25-5.82 ng/mL/hour
Adrenocorticotropin hormone	2599			6–50 pg/ml
Adrenal antibodies	Positive			Negative
ote: A normal response to the 250 mcg ACTH stimulation test is a ive been identified for specific monoclonal antibody immunoassay	rise in serum cortisol after either ys or liquid chromatography with	30 or 60min to a peak of ≥18 tandem mass spectrometry (l	ncg/dl (500–550 nmol/L). Lc .C–MS/MS). A low aldosterc	wer thresholds for a normal response (14–15µg/dl) me value after ACTH administration denotes abnormal

mineralocorticoid function of the adrenal cortex, which is consistent with the Addison's disease. Elevated adrenocorticotropin hormone levels are binding to the melanocortin 1 receptor on melanocytes to produce melanin. Antibodies directed at the adrenal cortex are frequently seen in patients with primary Addison's disease. ž ha

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to the melanocortin 1 receptor on melanocytes to produce melanin.²

AUTHOR CONTRIBUTIONS

MZ wrote the case report. APD suggested improvements and revised the manuscript.

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CONFLICT OF INTEREST

The authors have nothing to disclose.

DATA AVAILABILITY STATEMENT

Data sharing is not applicable to this article as no new data were created or analyzed in this study.

ETHICAL APPROVAL

No approval from the Institutional Review Board was required.

CONSENT

Written informed consent for publication of their details was obtained from the patient.

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REFERENCES

- Bornstein SR, Allolio B, Arlt W, et al. Diagnosis and treatment of primary Adrenal insufficiency: an Endocrine Society clinical practice guideline. *J Clin Endocrinol Metab.* 2016;101(2):364-389. doi:10.1210/jc.2015-1710
- 2. Oelkers W. Adrenal insufficiency. *N Engl J Med.* 1996;335(16):1206-1212. doi:10.1056/NEJM199610173351607

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