


Case Report

Transient total blindness associated with alcoholic ketoacidosis

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Background: Methanol poisoning is often suspected in patients with high anion gap metabolic acidosis and visual deficits. Although alcoholic ketoacidosis can cause high anion gap metabolic acidosis, reports on vision loss are limited. We report the case of a patient with alcoholic ketoacidosis with transient total blindness.

Case Presentation: A 56-year-old man complaining of total blindness was transferred to our hospital. Physical examination revealed a clear consciousness and mydriasis with an absence of pupillary light reflex. Blood analysis revealed high anion gap metabolic acidosis with a high ketone body concentration. Alcoholic ketoacidosis was diagnosed because the patient had a chronic alcohol abuse history and denied methanol intake. As acidemia improved because of fluid infusion and glucose and vitamin B1 supplementation, his visual acuity recovered. He was discharged after 44 days without visual deficits.

Conclusion: Patients with alcoholic ketoacidosis may present with acute vision loss, which recovers along with treatment.

Key words: Alcohol abuse, alcoholic ketoacidosis, toxicology/poisoning, transient blindness, vision loss

INTRODUCTION

ALCOHOLIC KETOACIDOSIS (AKA) usually occurs when patients with chronic alcoholism become malnourished and dehydrated.¹ In AKA, a β -hydroxybutyric acid-dominated ketone body elevation causes metabolic acidosis with a high anion gap. The symptoms are generally nausea, vomiting, abdominal pain, consciousness disorder, and shock. The treatment consists of fluid infusion and glucose and vitamin B1 supplementation.

Methanol poisoning is often suspected in patients with high anion gap metabolic acidosis and a visual deficit.² Formic acid, the methanol metabolite, causes various symptoms, including visual deficits. However, studies on AKA-associated visual deficits are limited. Herein, we report the case of a patient with AKA with transient total blindness and discuss its pathophysiology and treatment.

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CASE REPORT

A 56-YEAR-OLD MAN with a history of chronic alcohol abuse and type 1 diabetes, but without ophthalmic disease, was transferred to our hospital complaining of a gradual loss of bilateral eyesight that began 3 h before his emergency call. Additionally, he reported an inability to drink and eat because of fatigue during the previous day.

On admission, his vital signs were as follows: Glasgow Coma Scale score, 15 (E4V5M6); body temperature, 35.4°C; respiratory rate, 34 breaths/min; heart rate, 104 b.p.m.; blood pressure, 145/86 mmHg; and oxygen saturation, 100% without oxygen. He was completely blind with bilateral pupil dilation (7 mm in diameter, bilaterally) and no pupillary light reflex. Physical examination revealed no limb paralysis or extraocular movement disorders. Blood analysis revealed renal failure and high anion gap metabolic acidosis (Table 1). A fundus examination and head computed tomography showed no abnormal findings.

Initially, methanol poisoning was suspected because of the patient's visual deficit and high anion gap metabolic acidosis, although he denied methanol intake. Similarly, we considered diabetic ketoacidosis a possible diagnosis. However, because blood analysis revealed a β -hydroxybutyric

Table 1. Blood examination on admission of a 56-year-old man with alcoholic ketoacidosis and transient total blindness

Arterial blood gas analysis		Biochemical examination	
pH	6.724	AST	276 IU/L
PaO ₂	65.7 mmHg	ALT	69 IU/L
PaCO ₂	27.2 mmHg	LDH	711 IU/L
HCO ₃ ⁻	3.4 mmol/L	Total bilirubin	1.4 mg/dL
Anion gap	48.4 mmol/L	BUN	30.7 mg/dL
Lactate	23 mmol/L	Cre	2.05 mg/dL
Base excess	-28.2 mmol/L	Na	139 mEq/L
<i>Complete blood count</i>		K	5.8 mEq/L
WBC	28,600/ μ L	Cl	90 mEq/L
RBC	424 \times 10 ⁴ / μ L	Ethanol	65.2 mg/dL
Ht	44.7%	AcAc	976 μ mol/L
Hb	14.3 g/dL	3-OHBA	7,281 μ mol/L
Platelet count	18.4 \times 10 ⁴ / μ L	Glucose	307 mg/dL
		Vitamin B1	34 ng/mL
		Vitamin B12	681 pg/mL
		Folic acid	2.9 ng/mL

Vitamin B12 was measured after infusion of the vitamin B group (vitamins B1, B6, and B12). The normal ranges of vitamin B1, vitamin B12, and folic acid are 21–82 ng/mL, 233–914 pg/mL, and 3.6–12.9 ng/mL, respectively.

3-OHBA, 3-hydroxybutyric acid; AcAc, acetoacetic acid; ALT, alanine aminotransferase; AST, aspartate aminotransferase; BUN, blood urea nitrogen; Hb, hemoglobin; Ht, hydroxytryptamine; LDH, lactic acid dehydrogenase; RBC, red blood cells; WBC, white blood cells.

acid-dominated ketone body elevation superior to acetoacetic acid, AKA was diagnosed as the main cause.

After admission, fluid infusion, insulin injections, and supplementary glucose and vitamin B1 were initiated. As the patient's circulation and consciousness were stable on admission, we did not use bicarbonate. Moreover, as acidosis improved, he gradually began to see light and hand motions. At 4 h after admission, when the pH increased to 6.974, his visual acuity improved to counting fingers at 30 cm, and the light reflex had been recovered. However, at 5 h after admission, he experienced a sudden cardiac arrest with pulseless electrical activity, probably caused by prolonged severe metabolic acidosis after an acute symptomatic seizure. Thus, he was resuscitated by tracheal intubation and the treatment with adrenaline and bicarbonate. At 17 h after admission, the pH had normalized. He was extubated, and his visual acuity recovered completely on day 5. Following his eyesight evaluation, the visual acuity with correction of

each eye was 20/20 (fractional visual acuity), similar to prior-episode levels. He was discharged on day 44 without any neurological sequelae or visual deficits.

DISCUSSION

THIS CASE SUGGESTED that AKA can be associated with visual deficits in the form of transient total blindness. We speculated that vision loss associated with AKA is caused by a complex combination of acidemia, malnutrition, and ethanol toxicity.

Feeney *et al.* and Yanagawa *et al.* reported that severe acidemia could be the cause of acute vision loss in AKA based on animal experiments that suggest a possible association between the response of retinal horizontal cells to light and pH.^{3,4} We undertook a search of previously published reports on the PubMed electronic database and found 12 cases of metabolic acidosis with acute vision loss (Table 2),^{3–13} including five, three, and four cases of AKA, diabetic ketoacidosis, and metformin-associated lactic acidosis, respectively. All patients presented with acute bilateral blindness, and their visual acuity recovered without visual sequelae as acidemia improved. Additionally, visual acuity improved in some cases after administering sodium bicarbonate; in many cases, methanol poisoning was initially suspected.

Furthermore, nutritional and optic neuropathies should be considered as possible causes of visual deficits with AKA. Most patients with chronic alcohol abuse suffer from vitamin deficiency, including thiamine, cyanocobalamin, and folic acid, which can induce nutritional optic neuropathies. Additionally, ethanol is associated with tobacco–alcohol amblyopia, which was more frequently described prior to World War II in patients with severe tobacco and ethanol consumption history.¹⁴ Although these optic neuropathies generally present with subacute or chronic vision loss, Wilczyński *et al.* reported a case of alcohol-induced optic neuropathy with acute vision loss.¹⁵ Therefore, we believe that AKA-associated vision loss is caused by a complex combination of acidemia, malnutrition, and ethanol toxicity. Its treatment is based on treating the underlying conditions by improving metabolic acidosis and supplementation of vitamins. Although the infusion of sodium bicarbonate could improve visual acuity more immediately, it can similarly lead to intracellular acidosis and respiratory acidosis.¹⁶

However, when diagnosing patients with visual loss associated with AKA, intracranial, ophthalmologic, and psychiatric causes should be excluded by undertaking a head image and ophthalmoscopic examination. Particularly, posterior reversible encephalopathy syndrome, anterior

Table 2. Summary of reported cases of vision loss associated with acidemia

First author	Year	Age (years)	Sex	Cause of acidemia	pH on admission	Pupil dilation	Light reflex	MRI
Feeney	1998	49	F	AKA	6.79	–	+	–
Yanagawa	2004	44	M	AKA	6.70	+	–	–
Yanagawa	2009	53	M	AKA	6.49	+	–	–
Yanagawa	2009	63	M	AKA	6.61	–	+	–
Yanagawa	2009	66	M	AKA	6.85	–	–	–
Deutsch	1981	45	M	DKA	6.89	+	–	–
Oun	2018	45	F	DKA	6.80	+	–	+
Bockus	2019	59	M	DKA	6.95	+	–	+
Kreshak	2010	67	F	MALA	6.65	–	+	–
Cigarrán	2012	54	M	MALA	6.96	–	+	–
Jeon	2019	68	M	MALA	6.78	N/A	+	+
Ryu	2019	68	M	MALA	6.78	–	–	+

AKA, alcoholic ketoacidosis; DKA, diabetic ketoacidosis; F, female; M, male; MALA, metformin-associated lactic acidosis; MRI, magnetic resonance imaging; N/A, not applicable.

ischemic optic neuropathy, and posterior ischemic optic neuropathy should be considered differential diagnoses. Although we did not carry out magnetic resonance imaging, we ruled out posterior reversible encephalopathy syndrome because of the absence of headache, disorientation, and pupillary light reflex. Vision loss commonly progresses over the first few days in patients with anterior ischemic optic neuropathy, and the typical presentation of idiopathic posterior ischemic optic neuropathy is monocular vision loss, which differed from this case. Moreover, toxic optic neuropathies, including methanol poisoning, often present with severe metabolic acidosis that is similar to AKA, highlighting the importance of the patient's medical history. When the medical history is unreliable, physicians can resort to measuring the plasmatic osmolar gap and concentration of methanol as an effective way to establish the diagnosis.²

Our study had some limitations. First, as the number of cases was small and most emergency departments cannot undertake a more detailed ophthalmic examination (i.e., optical coherence tomography or visual evoked potentials) while visual symptoms persist, it was difficult to ascertain the precise cause of vision loss in patients with AKA. Similarly, it might be impractical to carry out magnetic resonance imaging because, in general, the patients' vital signs are often unstable during severe metabolic acidosis. In the future, more cases should be analyzed to achieve robust conclusions.

CONCLUSION

PATIENTS WITH AKA could present with acute vision loss, which recovers by treating the underlying

condition. In cases of severe metabolic acidosis with a visual deficit, methanol poisoning and AKA should be considered as a possible diagnosis.

DISCLOSURES

Approval of the research protocol: N/A.

Informed consent: Informed consent was obtained from the patient.

Registry and registration no. of the study/trial: N/A.

Animal studies: N/A.

Data sharing and accessibility: N/A.

Conflict of interest: None.

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