Metabolic alkalosis-an adverse effect of baking soda misuse: A case report and literature review

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Abstract. Baking soda overdose is rarely reported. However, several cases have been previously documented, as baking soda has gained popularity as an over-the-counter remedy. The present study reported the case of a 69-year-old male patient hospitalized with metabolic alkalosis (pH 7.61; bicarbonate levels, 53.2 mEq/l), hypokalemia (K^+ 2.6 mEq/l), acute kidney injury (serum creatinine level 4.02 mg/dl) and hepatic toxicity (alanine transaminase, 955 U/l; aspartate transaminase, 1,091 U/l) in the context of baking soda misuse as an alternative treatment for gout. The patient's past medical history included chronic uric acid nephropathy, gout, arterial hypertension and permanent atrial fibrillation. Under corrective treatment for the hydro-electrolyte and acid-base imbalances, the hepatic injury and inflammation markers were within normal limits; uric acid and creatinine serum levels also decreased.

Introduction

To date, the potential side-effects of alternative medicine remedies have remained largely elusive. Sodium bicarbonate (baking soda) is a low-cost, accessible, over-the-counter substance, widely recognized for its multiple uses. Most frequently, it is consumed as an antacid, despite the availability of proton pump inhibitors and H2 blockers (1). The undesired effects of baking soda misuse may present as different clinical scenarios, which have not been systematized until now. While baking soda is generally considered safe, various cases who developed metabolic alkalosis and electrolyte imbalances due to baking soda overdose have been documented (1). The present study reported on a case of metabolic alkalosis caused by baking soda misuse and provided a review of similar cases published to date. To the best of our knowledge, the patient of the present study is the first reported case of baking soda toxicity related to its oral ingestion as an alternative remedy for gout.

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Case report

Case presentation. A 69-year-old male patient was referred to the neurology emergency department of 'N. Oblu' Emergency Hospital (Iasi, Romania) in October 2021, by his family due to altered mental state, including confusion, which started 2 days prior to admission, with progressive worsening. The neurological exam did not reveal any focal clinical signs and craniocerebral CT excluded the possibility of an acute neurological event. A brief biological investigation indicated elevated serum creatinine levels (4.02 mg/dl; normal range, 0.6-1.2 mg/dl), metabolic alkalosis (pH 7.61; normal range, 7.31-7.41) bicarbonate levels in blood plasma of 53.2 mmol/l (normal range, 22-29 mmol/l) and hypokalemia (serum potassium K^+, 2.6 mEq/l; normal range, 3.5-5.1 mEq/l). On the next day, the patient was consequently referred to the Nephrology Department of the Clinical Hospital 'Dr C.I. Parhon' (Iasi, Romania) for further investigation.

Medical history. The patient's medical history included gout diagnosed in 1974, without any rheumatology consultation, for which the patient received intermittent treatment with colchicine 1 mg once daily (od) (3-6 months/year), ketoprofen 100 mg twice daily (bid) and allopurinol 100 mg/day od. The patient's cardiovascular antecedents included third-degree arterial hypertension and permanent atrial fibrillation since 2015, for which he was chronically anticoagulated with a direct oral anticoagulant (DOAC) (apixaban 5 mg bid). The patient's home medication also included a β-blocker (carvedilol 6.25 mg bid), an angiotensin II receptor blocker (candesartan 8 mg bid), a diuretic (indapamide 1.5 mg od)

and a statin (atorvastatin 20 mg od). The patient had recently undergone surgical interventions for a perianal fistula, and on this occasion, examination revealed elevated creatinine levels, interpreted in the context of chronic uric acid nephropathy (1.9 mg/dl in April 2021). The perianal fistula was interpreted in the context of prolonged sitting due to the patient's occupation as a bus driver.

Physical examination upon admission. The patient had an altered mental state, namely he was confused and agitated, without fever (temperature of 36.5˚C), with a heart rate of 100 beats/min, blood pressure of 110/80 mmHg, respiratory rate of 13 breaths/min and oxygen saturation of 98% on room air. Close examination revealed multiple tophi on the extremities and celsian signs on the extremities (redness, swelling, heat and pain) (Fig. 1). According to the 2015 ACR-EULAR Gout Classification Criteria, the score calculated for our patient is 20 (out of 23) (2). Cardiac examination revealed an irregular heart rate and rhythm with no murmur or extra heart sounds. The lungs were clear of auscultation bilaterally. No peripheral edema was noted.

The electrocardiogram on admission revealed atrial fibrillation with a ventricular rate of 104 beats/min, a -15˚ QRS axis and left anterior fascicular block.

The blood tests indicated elevated serum creatinine and urea levels [creatinine, 4.02 mg/dl; urea, 148 mg/dl (normal range, 20-49 mg/dl)]; hypochloremic metabolic alkalosis (pH 7.61); bicarbonate, 53.2 mmol/l; pCO_2, 53 mmHg; Cl, 78 mEq/l (normal range, 94-110 mEq/l in blood plasma), hypokalemia (K^+, 2.6 mEq/l), elevated hepatic cytolysis enzymes, including alanine transaminase (955 U/l; normal range, 4-33 U/l); aspartate transaminase (1,091 U/l; normal range, 8-35 U/l), hyperuricemia [serum uric acid (SUA), 10.3 mg/dl; normal range, 3.5-7 mg/dl], inflammatory syndrome [white blood cells, 10,920/mm^3; normal range, 4,000-10,000/mm^3; C-reactive protein, 440 mg/l; normal range, <5 mg/l; erythrocyte sedimentation rate (ESR) >140 mm/h; normal range, 1-10 mm/h]; and normocytic normochromic anemia [red blood cells, 3,49 million/mm^3 (normal range, 4.6-6.0x10^6/mm^3); hemoglobin, 10.5 g/dl, (normal range, 13-16 g/dl); hematocrit, 35.2% (normal range, 40-50%). The secretion culture from the perianal fistula was positive for *Staphylococcus aureus* and *Proteus mirabilis*.

Treatment of the patient. Given the patient's altered mental state, a thorough anamnesis was only possible on the second day of hospitalization at the nephrology department, revealing that the patient had consumed 20 g of baking soda dissolved in 2 liters of water per day as an alternative treatment for 'dissolving the tophi' in the week prior to admission.

Intravenous (iv) hydration with physiological serum 0.9% 1,000 ml, iv KCl 60 mEq/l/day was initiated along with potassium correction, iv dexamethasone 6 mg and *per os* colchicine 0.5 mg (1 tablet twice per day) for gout attack and antibiotic therapy with ceftriaxone 1 g twice per day in accordance with the antibiogram for 7 days. The in-hospital response of the patient was favorable, with remission of the hydro-electrolyte and acid-base imbalances. At discharge, the hepatic injury and inflammation markers were within normal limits and the creatinine serum level was 1.9 mg/dl. Angiotensin II receptor blocker treatment was ceased, with normal in-hospital blood pressure control. Given the atrial fibrillation, heart rate, CHA_2D_2-VASc score (a score used to assess the risk of stroke in atrial fibrillation patients defined by the Congestive heart failure, Hypertension, Age ≥75 years, Diabetes mellitus, Stroke, Vascular disease, Age 65-74 years, Sex category-female) of 3 points and HAS-BLED score of 3 points (a score to predict bleeding risk based on the following risk factors: Hypertension, Abnormal renal/liver function, Stroke, Bleeding history or predisposition, Labile INR, Elderly - >65 years, drugs/alcohol; a score over 3 indicates high bleeding risk), the patient was discharged with instructions to continue his DOAC and β-blocker therapy (3). In addition, febuxostat therapy with a low starting dose (40 mg od) was prescribed.

Treatment outcomes and follow-up. The 1-month follow-up revealed a euvolemic, hemodynamically stable patient. Post-discharge creatinine levels were stationary, while SUA levels improved (SUA, 6.5 mg/dl). At-home blood pressure control was adequate without angiotensin II receptor blocker. However, the patient complained about the acute gout arthritis of his upper left limb (Fig. 2). At 1 day prior to his follow-up, the patient consulted a rheumatologist who recommended short-term colchicine administration and an increase of the dose of febuxostat to 80 mg od.

Discussion

A literature search was performed in the PubMed/MEDLINE and ScienceDirect/Elsevier electronic databases for reported cases of sodium bicarbonate toxicity in adult patients. Search terms included 'baking soda misuse', 'baking soda toxicity', 'baking soda overdose', 'sodium bicarbonate misuse', 'sodium bicarbonate toxicity' and 'sodium bicarbonate overdose'. References from the included articles were also scanned to identify possible additional publications.

EndNote X7 (Thomson Reuters) bibliography manager was used to check the title and abstracts of the retrieved and screened articles. Duplicate articles were electronically and manually removed (in the case of differences in the citation style of the different journals). After screening all the relevant articles found, a total of 21 clinical cases published between 1986 and 2020 were included. Basic characteristics of the studies were summarized in Table I (name of the first author, year of publication, medical history, baking soda exposure, methods of diagnosis, laboratory values, reference test). Baking soda is a commonly used remedy to counteract high acidity symptoms, such as heartburn. However, other uses include the treatment of hyperkalemia, metabolic acidosis, prevention of contrast-induced nephropathy and urine alkalinization (4). According to popular beliefs, sodium bicarbonate, as a strong base, neutralizes the acidic state in which uric acid precipitates. Although no scientific evidence supports these effects, baking soda has gained popularity as an alternative treatment for gout. As an over-the-counter antacid, baking soda is considered safe by the Food and Drug Administration at a maximum daily dosage of 200 mEq sodium bicarbonate in young individuals and 100 mEq sodium bicarbonate in those aged >60 years (5). One teaspoon (5 g) of baking soda contains ~59 mEq of sodium bicarbonate (6). The patient of the present study declared the

Figure 1. Multiple tophi and celsian signs on the hands.

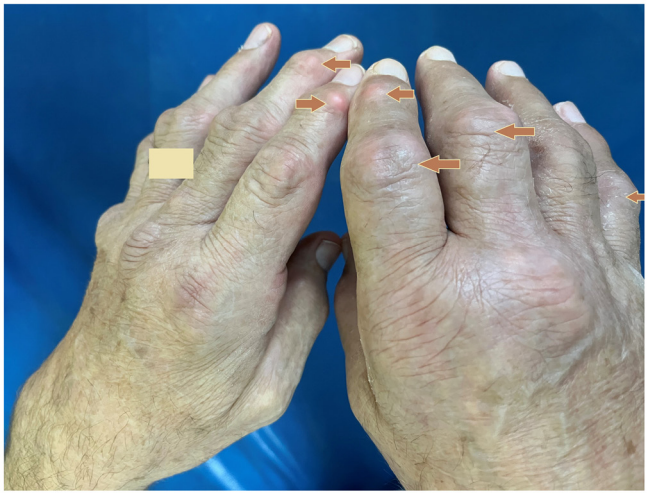

Figure 2. Gout on the hands at 1 month after discharge from the hospital.

intake of ~20 g of sodium bicarbonate, which translates into ~238 mEq sodium bicarbonate, exceeding the recommended safe dose.

The increase in renal tubular pH by baking soda alters the excretion of anti-inflammatory medications, increasing their serum levels (7). In the specific case of the present study, this may explain the onset of acute kidney injury and liver toxicity as a result of high intake of baking soda combined with ketoprofen. Caution should be advised when prescribing sodium bicarbonate, particularly when combined with non-steroidal anti-inflammatory drugs or other anti-inflammatory drugs.

Chronic interstitial nephritis that is associated with hyperuricemia may be considered when SUA levels are disproportionately high in contrast with the patient's renal impairment (4). Patients usually consult the medical unit with hypertension and mildly impaired renal function. Minor proteinuria and tubular dysfunction may be present (4). Given the patient's poor response to allopurinol over the years, the choice of chronic treatment recommendation for the present case was febuxostat, a selective xanthine oxidase inhibitor, with a low starting dose (40 mg od). In contrast to allopurinol, febuxostat does not require dose adjustments at a lower estimated glomerular filtration rate and is associated with a smaller incidence of hypersensitivity or nephrotoxicity (4). At the 1-month follow-up, the patient's condition was under good control with febuxostat treatment, with normal SUA levels.

Metabolic alkalosis due to baking soda oral ingestion to treat dyspepsia has been previously reported. A number of cases of adverse toxic effects of topically applied baking soda have been reported, which was used as an alternative to chemotherapy for breast (5) and colon cancer (6), for the treatment of leg ulcer (7) and as a toothpaste additive (8). A review indicated that the most common reasons for bicarbonate exposure were antacid misuse (60.4%), urinary drug testing alterations (11.5%), treatment of urinary tract infections (4.7%) and as a means for body detoxification (4.7%) (1). A case of misuse as a remedy for gout was also identified (1).

The most common presentation of baking soda toxicity is metabolic alkalosis and electrolyte changes. Usually, the kidney responds by increasing bicarbonate excretion, which may become impaired in the setting of renal insufficiency or volume contraction. Metabolic acidosis is a common finding in chronic kidney disease (4). Though less common in patients with end-stage renal disease undergoing hemodialysis, there were 2 reported cases of metabolic alkalosis attributed to oral baking soda intake (9,10).

In spite of a history of third-degree arterial hypertension, the patient of the present study had controlled blood tension values without the use of any hypotensive agent both during hospitalization and at the 1-month follow-up. However, further monitoring is necessary to evaluate whether the effect is temporary and investigate its cause. Previously, 2 cases who developed hypotension and required vasopressors for hemodynamic support were reported (11,12). One of them also exhibited a decrease in anesthetic demand (12). On the other hand, transient daytime hypertension has been observed, but was interpreted in the context of sleep apnea (13).

The altered mental state of the patient of the present study delayed the accurate identification of the underlying cause of metabolic alkalosis. Mental state alterations are common in patients with severe metabolic alkalosis. Agitation and confusion (14-16), dizziness with or without loss of consciousness (17,18), stuttering (15), obnubilation (11) and coma (19,20) have been reported. For 1 case, hypernatremic hemorrhagic encephalopathy due to baking soda ingestion was reported, with multiple areas of intracranial and subarachnoid hemorrhage observed on the cranial computed tomography (15). Neuromuscular symptoms are commonly described in patients with severe metabolic alkalosis. Paresthesia and carpopedal spasm without a positive Chvostek's sign (20), involuntary facial twitching (15), increased motor tone with flexion of elbows and wrists (20), myoclonus (13) and epileptiform convulsions (21) have also been described. Furthermore, 2 patients with sleep apnea were referred, which may occur in the metabolic alkalosis setting due to central ventilatory drive depression (9,13).

Patients with a history of alcohol abuse may have an increased susceptibility to developing metabolic alkalosis due to high baking soda intake (17,18,20-22). Chronic alcoholics are more inclined to ingest antacids for dyspepsia relief, while their dehydration status may promote and aggravate metabolic alterations. Rhabdomyolysis has been

Table I. List of reported cases of baking soda toxicity in adult patients.

First author, year	Presentation	Medical history	Baking soda exposure	Laboratory values	(Refs.)
Al-Abri and Olson, 2013	Metabolic alkalosis Ventricular tachycardia	HTN DM Zollinger-Ellison syndrome	Antacid replacement: 1 teaspoon of baking soda on an as needed basis for years	pH 7.5 HCO_3 46 mmol/l K^+ 2.0 mEq/l Na^+ 122 mEq/l Cl^- 59 mEq/l Cr 0.9 mg/dl	(6)
Cervantes *et al*, 2020	Metabolic alkalosis	CKD G3bA1 HTN AF Hyperlipidemia GERD	Topical use three times/day for tooth hygiene for the previous 6 months	pH 7.5 HCO_3 44 mmol/l K^+ 3.6 mEq/l Na^+ 146 mEq/l Cl^- 95 mEq/l Cr 1.37 mg/dl	(8)
Solak *et al*, 2009	Metabolic alkalosis Sleep apnea Volume overload Uncontrolled HTN	ESRD CHF DM type 2 Hypothyroidism Chronic gastritis	Antacid abuse: 4-5 packs of baking soda a day over the last month	pH 7 637 HCO_3 45 mmol/l K^+ 3.3 mEq/l Na^+ 141 mEq/l Cl^- 83 mEq/l	(9)
Sahani *et al*, 2001	Metabolic alkalosis	ESRD	Hiccup relief: Consumption of ¾ bottle of Bromo-Seltzer (containing 89 g sodium bicarbonate)	pH 7.53 HCO_3 40 mmol/l K^+ 3.5 mEq/l Na^+ 143 mEq/l Cl^- 89 mEq/l Cr 1.0 mg/dl	(10)
Galinko *et al*, 2017	Metabolic alkalosis Acute kidney injury Acute hypoxic respiratory failure Lactic acidosis Altered mental state Ventricular tachycardia	HTN Meningioma (surgical resection, ventriculoperitoneal shunt and radiation therapy) Breast cancer	Alternative topical treatment for breast cancer -113 g of baking soda applied topically every 4 days for several weeks	pH 7.66 HCO_3 >45 mmol/l K^+ 2.6 mEq/l Na^+ 158 mEq/l Cl^- 92 mEq/l Cr 1.9 mg/dl	(11)
Soliz *et al*, 2014	Metabolic alkalosis	Colon cancer Sinonasal melanoma	Alternative treatment for colon cancer: 1 liter of pH 7.8-8 water obtained from bottled water, key lime juice a quarter of tablespoon baking soda	pH 7.65 HCO_3 39 mmol/l K^+ 2.6 mEq/l Na^+ 123 mEq/l	(12)
Okada *et al*, 1996	Metabolic alkalosis and myoclonus	HTN Cerebral infarction Gastrectomy (gastric ulcer)	Antacid (625 mg sodium bicarbonate) for a 6-month period	pH 7.481 HCO_3 33.9 mmol/l K^+ 2.7 mEq/l	(13)
John *et al*, 2012	Metabolic alkalosis Respiratory alkalosis High-anion gap metabolic acidosis	Foot ulcers for 2 years	Ingestion and application of baking soda to leg ulcers for 1.5 years	pH 7.69 HCO_3 54 mmol/l K^+ 1.8 mEq/l Na^+ 148 mEq/l Cl^- 73 mEq/l Cr 3.4 mg/dl	(14)

Table I. Continued.

First author, year	Presentation	Medical history	Baking soda exposure	Laboratory values	(Refs.)
Hughes *et al*, 2016	Metabolic alkalosis Hypernatremia Hemorrhagic encephalopathy Altered mental state	Schizophrenia Polysubstance abuse	Patient was unable to explain cause of ingestion- 1 box of baking soda (454 g)	pH 7.53 HCO_3 50 mEq/l K^+ 2.5 mEq/l Na^+ 172 mEq/l Cl^- 98 mEq/l Glucose 433 mg/dl Cr 1.85 mg/dl	(15)
Ajbani *et al*, 2011	Metabolic alkalosis	HTN COPD	Antacid replacement: Unknown quantity of baking soda over several weeks	pH 7.59 HCO_3 56 mmol/l K^+ 1.7 mEq/l Na^+ 121 mEq/l Cl^- 53 mEq/l Cr 3.3 mg/dl	(16)
Fitzgibbons and Snoey, 1999 (Case 1)	Metabolic alkalosis Ventricular tachydysrhythmia	Peptic ulcer disease with perforation	Antacid replacement: Several tablespoons of baking soda	pH 7.56 HCO_3 58 mmol/l K^+ 1.8 mEq/l Na^+ 129 mEq/l Cl^- 55 mEq/l Cr 2.8 mg/dl	(17)
Fitzgibbons and Snoey, 1999 (Case 2)	Metabolic alkalosis Altered mental state	HTN Herpes zoster infection Hepatitis	Antacid replacement: One box of baking soda	pH 7.49 HCO_3 41 mmol/l K^+ 2.8 mEq/l Na^+ 146 mEq/l Cl^- 90 mEq/l Cr 0.9 mg/dl	(17)
Thomas and Stone, 1994	Metabolic alkalosis	Alcoholic esophagitis and gastritis	Antacid misuse: 10-12 oz of baking soda	pH 7.55 HCO_3 44.5 mmol/l Na^+ 136 mEq/l K^+ 2.5 mEq/l Cl^- 77 mEq/l Cr 2.4 mg/dl	(18)
Gawarammana *et al*, 2007	Metabolic alkalosis Coma (GCS 3/15)	N/A	Antacid: 2 liters of Gaviscon in the prior 48 h	pH 7.54 HCO_3 50.6 mmol/l K^+ 1.6 mEq/l Na^+ 127 mEq/l Cl^- 66 mEq/l	(19)
Mennen and Slovis, 1988	Metabolic alkalosis Cardiopulmonary arrest Death	HTN Peptic ulcer disease Alcohol abuse Seizure disorder	Antacid misuse: Amount unknown	pH 7.73 HCO_3 >40 mmol/l Na^+ 154 mEq/l K^+ 3.2 mEq/l Cl^- 53 mEq/l Cr 1.4 mg/dl	(20)
Forslund *et al*, 2008	Metabolic alkalosis Epileptiformic convulsions Subdural hemorrhage Rhabdomyolysis	Alcohol abuse	Antacid abuse: 40 years history of baking soda abuse; 10-15 g daily initially, slowly increasing up to 50 g daily during last year	pH 7.57 HCO_3 85 mmol/l K^+ 2.3 mEq/l Na^+ 147 mEq/l Cl^- 46 mEq/l	(21)

Table I. Continued.

First author, year	Presentation	Medical history	Baking soda exposure	Laboratory values	(Refs.)
Yi *et al*, 2012	Metabolic alkalosis	Alcohol abuse	Antacid replacement: 3-5 tablespoons daily	pH 7.6 HCO_3 53 mmol/l K^+ 1.6 mEq/l Na^+ 131 mEq/l Cl^- 65 mEq/l Cr 3.8 mg/dl	(22)
Scolari Childress and Myles, 2013	Rhabdomyolysis Peripartum cardiomyopathy	Pregnant, at 37 weeks of gestation with history of hyperemesis and iron-deficiency anemia	Hiccup remedy: A box of baking soda (454 g) every day for several years	Hb 8.2 g/dl Ht 26.3% K^+ 2.1 mmol/l AST 134 U/l ALT 60 U/l	(23)
Lazebnik *et al*, 1986	Stomach rupture	N/A	Antacid misuse: One tablespoon of sodium bicarbonate	N/A	(24)
Linford and James, 1986	Metabolic alkalosis	N/A	Bicarbonate abuse: 50-150 g daily	HCO_3 >40 mmol/l K^+ 1.8 mEq/l	(25)
Okada *et al*, 1999	Metabolic alkalosis Sleep apnea Hypertension	ESRD	Antacid replacement: 4-5 g baking soda daily	pH 7.47 HCO_3 40.1 mmol/l	(26)

Normal ranges: Cr, 0.6-1.2 mg/dl; HCO_3^- in blood plasma, 22-29 mmol/l; pH in blood plasma, 7.31-7.41; serum K^+, 3.5-5.1 mEq/l; serum Na^+, 135-145 mEq/l; serum Cl^-, 94-110 mEq/l; ALT, 4-33 U/L; AST, 8-35 U/l; Hb, 13-16 g/dl; Ht, 40-50%. AF, atrial fibrillation; CHF, congestive heart failure; CKD, chronic kidney disease; COPD, chronic obstructive pulmonary disease; DM, diabetes mellitus; GCS, Glasgow Coma Scale; GERD, gastroesophageal reflux disease; HTN, hypertension; ESRD, end-stage renal disease; AST, aspartate transaminase; ALT, alanine transaminase; N/A, not applicable; Cr, serum creatinine; ALT, alanine transaminase; AST, aspartate transaminase; Hb, hemoglobin; Ht, hematocrit.

reported in an alcoholic patient (21) and in pregnant patients with pica leading to baking soda intake (23). Other systemic complications, such as spontaneous gastric rupture, have also been described (24).

In conclusion, the present study reported a case of baking soda toxicity resulting in metabolic alkalosis, toxic acute hepatitis and acute kidney injury. The case described herein provided a challenge in establishing a diagnosis of metabolic alkalosis with unknown etiology until thorough anamnesis was possible. The present case report and literature review highlights the importance of closely evaluating all the alternative therapies that patients resort to. Exposure to these substances may not be easily disclosed, as their implications are frequently underrated. Physicians should be aware of the potential practices and associated side effects in order to ensure prompt diagnosis and adequate treatment.

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Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Authors' contributions

All authors substantially contributed to this paper. AD and LF designed the study, searched the literature and wrote the first draft of the manuscript. CEV, IF and RA collected and interpreted the relevant data. AD, LF and IF confirm the authenticity of all the raw data. CEV and LF supervised the literature review and revised the manuscript for important intellectual content. All authors read and approved the final manuscript.

Ethics approval and consent to participate

Not applicable.

Patient consent for publication

The patient provided written informed consent for the publication of this case report, including medical information and the accompanying images.

Competing interests

The authors declare that they have no competing interests.

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