Kratom Ingestion and Emergency Care: Summary and a Case Report

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Contribution to Emergency Nursing Practice

- Kratom is a plant native to southeast Asia with psychoactive properties commonly ingested for recreational intent, pain relief, or self-treatment of opioid withdrawal symptoms. Kratom is easily obtainable through purchase over the internet.
- This article provides a single-patient report of respiratory failure and shock after kratom ingestion and a brief overview of kratom's clinical features and pharmacologic properties.
- Key implications for emergency nursing practice found in this article include understanding a less-familiar psychoactive substance and unique patient presentation that will help identify and treat patients who present to the emergency department after kratom ingestion.

Abstract

Kratom ingestion for its psychotropic effect or to self-treat opioid withdrawal symptoms has increased over the last 10 years in the United States. Although mild adverse effects have been observed in users, reports of respiratory failure and shock after kratom consumption remain rare. In this case, a 35-year-old man initially presented to the emergency department with profound circulatory shock, metabolic acidosis, hypoxia, and symptoms of autonomic nervous system dysfunction. The patient required vasopressor support, multiregimen sedation and rapid sequence intubation, mechanical ventilation, and emergent hemodialysis. Within 72 hours, the patient's condition stabilized, and he was extubated. The patient reported regular consumption of large quantities of kratom as well as injection of heroin and cocaine. In this report, a rare clinical presentation after kratom ingestion is described.

Key words: Kratom; Critical care; Case report; Emergency nursing

Background

Kratom, also known as *Mitragyna speciosa,* is a tropical tree native to southern Thailand. The leaves of kratom can be chewed, brewed as a drink, smoked, or ingested orally.[1-3] Kratom is sold over the internet as well as in tea shops, bars, and convenience stores. The substance can be found in a variety of forms, including loose, chopped leaves; capsules; compressed tablets; and concentrated extracts under the name kratom, ketum, and *M speciosa*. As previously reported,[4] kratom use has increased in the United States and Europe over the last 5 years, and consumers generally believe that there is little or no risk with using.[5,6] Kratom is currently illegal in Alabama, Arkansas, Indiana, Tennessee, Vermont, Rhode Island, and Wisconsin, as well as in Thailand and Malaysia.[3] However, there are minimal data regarding the pharmacokinetics and toxicity of kratom in humans. Clinical presentations of the adverse effects of kratom use vary, and critical illness or fatality has been rarely reported.[7-9] In this case report, an unusual clinical presentation with critical illness after kratom ingestion is reported.

Case Report

A 35-year-old man was presented by emergency medical services to the emergency department for a suspected drug overdose. Paramedics had found the patient unresponsive

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and apneic with a respiratory rate of 6 breaths per minute and a heart rate of 120 beats per minute. The patient was given 2 mg intravenous (IV) naloxone before ED arrival, with improvement in mentation and respiratory status. A bystander reported to the paramedics that the patient had a known illicit drug use history, but the drug(s) of choice were unknown. In the emergency department, the patient was alert, anxious, and severely agitated but cooperative, with a Glasgow coma score of 14, heart rate of 118 beats per minute, respiratory rate of 28 breaths per minute, blood pressure 60/33 mm Hg, temperature 34.5 °C (94.1 °F), and oxygen saturation was 95% on a nonrebreather mask. The physical examination was notable for 3-mm pupils, normal breath sounds, and bilateral lower extremity fixed plantar flexion with mild clonus on passive ankle dorsiflexion. The patient reported a primary complaint of being unable to hear (ie, subjective sensorineural hearing loss); however, the patient responded to clinician communication after the clinician spoke loudly and repeated questions. The patient endorsed taking pills but could not provide the names or class. Other past medical history was unattainable. Approximately 10 minutes after arrival, the patient developed central cyanosis, peripheral mottling, and signs of profound mixed cardiogenic and distributive shock with impending cardiorespiratory arrest. Differential diagnoses or etiologies included psychotropic drug overdose, other toxic ingestion, blunt traumatic injury, hypovolemia, metabolic derangement, environmental exposure, neuroleptic malignant syndrome, serotonin syndrome, infection, intracranial injury or lesion, aortic dissection, and pulmonary embolism.

INVESTIGATIONS

Initial laboratory investigations included arterial blood gas; serum kidney function, electrolyte, and liver function panel; cardiac enzymes; total creatine kinase; lactic acid; complete blood count with differential; coagulation studies; urinalysis; urine drug screen (UDS); and serum toxicology panel. Additional studies included electrocardiogram, chest x-ray, noncontrast head computed tomography, and computed tomography angiography of the chest, abdomen, and pelvis. The Table details all relevant abnormal laboratory results of the initially performed tests. The remaining ED laboratory investigations were normal. No intracranial, thoracic, or abdominal abnormalities were seen on imaging. The 5-panel UDS was positive for cocaine (enzyme immunoassay) only, and the serum toxicology panel was negative (ie, alcohol, acetaminophen, and salicylates).

TABLE
Relevant abnormal blood and urine test results obtained in the emergency department

Laboratory test	Normal values	Results
pH	7.35-7.45	7.01
pCO_2 (mm Hg)	35-45	8.8
pO_2 (mm Hg)	75-100	20.0
HCO_3 (mEq/L)	22-26	2.2
BE (mEq)	–2 to +2	–25.0
White blood cells (K/mcL)	4-11	27.6
Hemoglobin (g/dL)	12-17	12.9
Glucose (mg/dL)	70-130	94
BUN (mg/dL)	7-20	21
Creatinine (mg/dL)	0.6-1.2	2.8
Sodium (mEq/L)	135-145	140
Potassium (mEq/L)	3.5-5.0	6.4
Chloride (mEq/L)	95-105	102
Anion gap (mEq/L)	3-10	18
Calcium (mg/dL)	8.5-10.5	7.8
Lactic acid (mmol/L)	<2	5.9
Urine drug screen	Negative	Positive for cocaine, negative for opioids

BE, base excess; BUN, blood urea nitrogen; HCO_3, bicarbonate.

INTERVENTIONS

Immediately on arrival to the emergency department, the patient was continued on supplemental oxygen through a nonrebreather mask, triaged as an Emergency Severity Index level 1 acuity, and the ED physician attending was called to the bedside owing to the need for immediate lifesaving interventions. Two large-bore peripheral IV catheters were placed in the antecubital fossa bilaterally, and a 2000-mL sodium chloride 0.9% bolus was administered using pressure bags. Because of a reported improvement in patient condition by Emergency Medical Services, continued agitation, and hemodynamic instability, an additional dose of naloxone (0.4 mg IV) was administered without effect. A subdissociative[10] IV bolus of ketamine (50 mg or 0.50 mg/kg) was administered, which improved agitation but had no effect on improving hemodynamics. Therefore, a peripheral IV norepinephrine infusion was initiated for cardiovascular support. The ED physician performed a bedside ultrasonography that showed no free fluid in the abdomen, no tamponade, good cardiac squeeze, and no pulmonary edema. The norepinephrine infusion was rapidly increased to more than 0.5 mcg/kg/min (50 mcg/min) with no improvement in blood pressure. Therefore, an EPINEPHrine infusion was initiated that resulted in an increased systolic blood pressure of more than 90 mm Hg. The patient was intubated for severe hypoxia and respiratory distress in the setting of severe anion gap metabolic acidosis (Table).

Controlled rapid sequence intubation was performed using 20 mg IV of etomidate and 100 mg IV of rocuronium. A rapid and unexpected sedation and induction reaction occurred. Immediately after the administration of etomidate, the patient exhibited extreme agitation and seemed to be in severe pain (ie, loud screaming, grimacing, and combativeness). This was followed by full-body muscle rigidity. Rocuronium was administered and resulted in total contraction of the masseter muscle, preventing direct laryngoscopy despite multiple attempts over a 4- to 5-minute onset period, which prohibited intubation. Continuous bag-valve-mask ventilation was maintained, and an IV bolus of 100 mg of ketamine was rapidly administered that subsequently relaxed the masseter muscle and facilitated endotracheal intubation through direct laryngoscopy. The patient was initiated on propofol and dexmedetomidine infusions for sedation and placed on assist-control mechanical ventilation at a rate of 28 breaths per minute, tidal volume 450 mL, fraction of inspired oxygen 1.0, and positive end-expiratory pressure of 5 cm of water. A central venous catheter was placed for continued administration of vasopressors. Hyperkalemia protocol was administered: 10 units IV insulin, 25 g IV dextrose 50%, 1 g IV calcium chloride, and 100 mEq sodium bicarbonate. A sodium bicarbonate infusion was prescribed at 100 mL/h. Hemodynamics improved with norepinephrine and EPINEPHrine infusions. The regional poison control center was consulted, and poison control personnel recommended continued supportive care.

OUTCOME

The patient was admitted to the intensive care unit, a trialysis catheter was placed, and 1 session of emergent hemodialysis was performed. Passive warming and other supportive measures were provided per usual care. The remainder of the intensive care unit course was unremarkable, and the patient was extubated within 72 hours. On interview, the patient endorsed regular kratom use, which was primarily ingested for heroin self-detoxication and to prevent withdrawal symptoms. The patient also reported cocaine use; however, he stated that the last cocaine use was more than 24 hours before admission. On hospital day 4, the patient was discharged to inpatient rehabilitation with good cognitive function.

Discussion

Reporting of toxicity from kratom use is rare.[9] Clinical presentations vary significantly in the literature. The reported adverse effects of kratom have included tachycardia, hypertension, hypotension, agitation, nausea, vomiting, confusion, tremor, diaphoresis, seizures, coma, and death.[6,9,11-14] Between 2011 and 2017, 6 cases of respiratory arrest and 5 cases of cardiac arrest were reported to US poison control centers.[9] However, the Centers for Disease Control and Prevention reported 90 deaths where kratom was found on postmortem toxicology analysis between July 2016 and December 2017.[15] This suggests that kratom toxicity may be more widespread, and clinicians may be unaware. Of note, most clinical laboratories are not capable of confirmatory or quantitative analysis; therefore, clinical evaluation and history are the initial methods of diagnosis.

The pharmacokinetics of kratom in humans remains unclear.[16] The leaves of *M speciosa* contain more than 20 biologically active alkaloids of kratom.[3-16] The 2 most important alkaloids are mitragynine and 7-hydroxymitragynine, with 66% and 2% total alkaloid content, respectively.[3-17] These compounds bind the opioid mu receptors and delta receptors, whereas mitragynine is also an agonist of the alpha-2 adrenergic, adenosine, dopamine, and serotonin

receptors.[3,17-19] On the basis of the stimulation of alpha-2 adrenergic receptors, kratom can attenuate the symptoms of opioid withdrawal.[3,18] It was also found that 7-hydroxymitragynine is 46-fold more potent than mitragynine and 13-fold stronger than morphine.[3] Kratom's major alkaloids (eg, mitragynine and ciliante) are cytochrome P450 (CYP) inhibitors, with high inhibition of the 2D6 enzyme that is responsible for xenobiotic metabolism.[20] When combined with other CYP substrates such as codeine, HYDROcodone, fentaNYL, methadone, oxyCODONE, and traMADol, their effect is increased.[3] It has been reported that other substances that are used in combination with kratom include caffeine, codeine, and dextromethorphan (CYP substrates), as well as diphenhydrAMINE (CYP inhibitor), leading to an increased drug effect.[3] Mitragynine is a lipophilic alkaloid that is poorly soluble in water but is soluble in alcohol, chloroform, and acetic acid.[1,3] The pharmacokinetics in humans is not well known; however, in animal models, the oral and IV duration of action demonstrated an elimination half-life of 3.9 to 9.4 hours and a very high distribution of 37.9 to 89.5 L/kg.[3,21]

Aggarwal et al[1] recently described a case of the death of a patient who presented to the emergency department in cardiopulmonary arrest after an unknown quantity ingestion of kratom 24 hours earlier. Taking advantage of the lipophilic nature of mitragynine, the authors report the use of an IV intralipid bolus in attempts to counteract the effects of kratom. The authors described a 30% reduction in vasopressor needs as well as improved oxygenation and ventilation; however, the effects were short-lived. The optimal dosage for intralipid infusion therapy has not been established for kratom, but the toxicology-recommended dosing includes a bolus of 1.5 mL/kg followed by 0.25 mL/kg/min over 30 to 60 minutes.[1,3]

In this case report, despite the lack of confirmatory laboratory test results, it was determined that kratom was the primary contributor to the observed symptoms. However, the patient's self-reported polysubstance ingestion was a potential confounding factor. Although the UDS was positive for cocaine, the patient denied acute use before admission, and endorsed cocaine use in the 1 to 2 days before admission. In addition, it is unlikely that acute heroin use contributed to the clinical presentation because the initial UDS was negative for opiates, and the ED administration of naloxone had no effect. Because quantitative analyses of kratom, cocaine, and heroin were not obtainable, the ED clinical presentation was initially assumed to be of a mixed toxic ingestion. The initial improvement in respiratory and mental status after administration of naloxone is consistent with mu-opioid antagonism, which has been seen in at least 1 other case report where concurrent opioid use was not present.[22] Very few cases of shock or severe acidosis have been reported after kratom ingestion before a cardiopulmonary arrest.[1,7,8] In this case, despite multiple investigations, other etiologies of severe acidosis or shock were not identified.

The limitations in this report were the unknown concurrent illicit substance use, chronic impacts of kratom use with or without other polysubstance abuse, and other unknown medical conditions exacerbating toxicity. Of note, the dose and duration of kratom use were not known. Acute heroin and cocaine use was ultimately denied by the patient; however, owing to the lack of more sensitive testing and quantitative analysis, concurrent ingestion could not be definitively ruled out. Furthermore, the literature on kratom's interactions with concurrent heroin and cocaine remains minimal. The patient reported purchasing kratom over the internet, but no further sourcing information was available.

Implications for Emergency Nursing

Illicit drug ingestion, especially involving synthetic or herbal substances, requires rapid assessment to determine the need for aggressive airway, cardiovascular, and respiratory support.[23] Hypotension refractory to crystalloids may require early vasopressor support. Administration of peripheral venous vasopressor agents has been considered safe, with low incidence of complications.[24-26] Emergency nurses should administer peripheral vasopressors through large-gauge catheters at the most proximally available location (ie, antecubital fossa). When patients present with symptoms associated with neuroleptic malignant syndrome or serotonin syndrome, antipsychotics (eg, haloperidol and metoclopramide) and serotonergic drugs should be avoided.[23,27] Gordon and Schmelzer[28] previously described the pathophysiology and clinical practice when caring for a patient with excited delirium, and benzodiazepines are generally the first-line agent for the control of agitation, neuromuscular dysfunction, tachycardia, and hypertension.[23] However, when patients are hemodynamically unstable or concern for potentiating respiratory depression exists, benzodiazepines are less ideal, and ketamine may be considered.[29,30] Ketamine produces anesthesia and analgesia by antagonizing the N-methyl-D-aspartate receptor while preserving cardiopulmonary status and potentially increasing cardiac output.[29] In many presentations, intubation and mechanical ventilation may be required, and emergency nurses should be prepared to assist with emergent airway support (eg, suction, bag-valve-mask ventilation, oxygen administration, and rapid sequence intubation; see Supplementary Infographic). Depolarizing

neuromuscular blockers (ie, succinylcholine) should not be administered owing to the risk of increasing serum potassium levels or worsening rhabdomyolysis.[23,27]

Conclusion

Although illegal in some regions, kratom is widely available to the public and frequently purchased over the internet.[5,6] Toxic effects of kratom consumption are rare; however, cases describing critical illness with varying clinical presentations have been reported.[1,9,11,13,31] Health care providers who evaluate patients with acute toxidromes should consider kratom consumption as a causal agent for unusual presentations when the substances are not known. Further research is needed to understand acute kratom toxicity and differentiate from other toxidromes in the emergency department.

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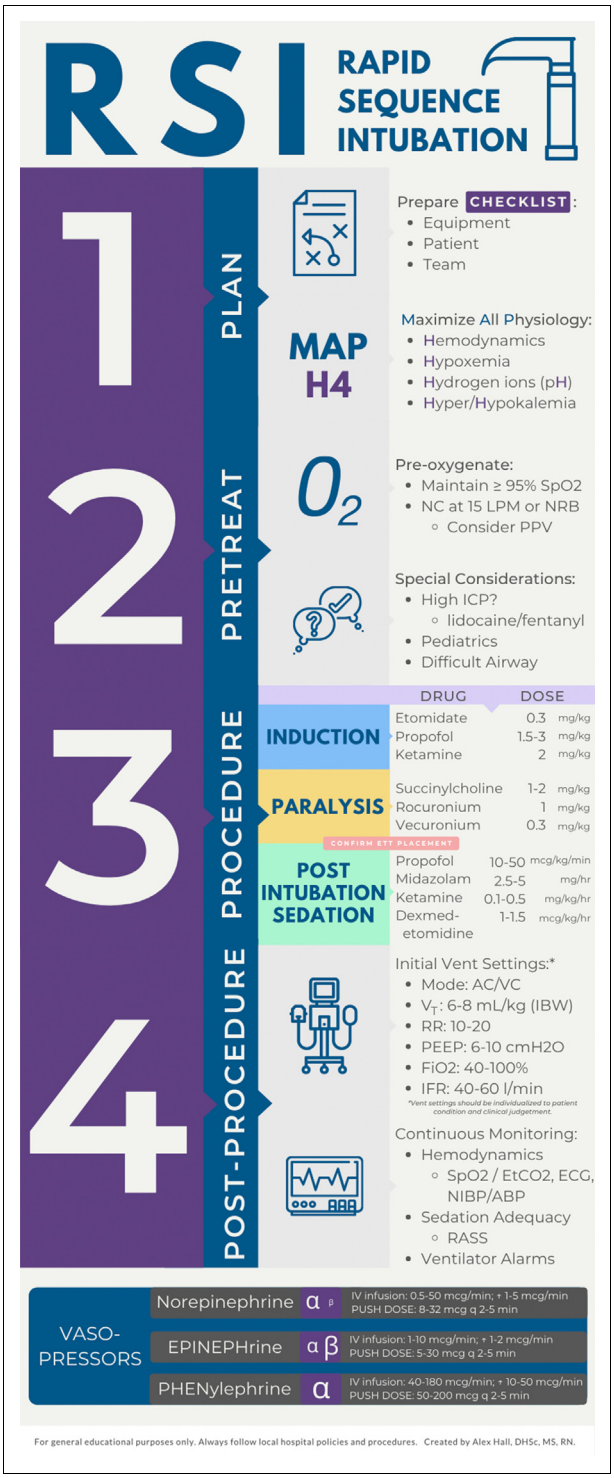
RSI
RAPID SEQUENCE INTUBATION
1 PLAN
2 PRETREAT
3 PROCEDURE
4 POST-PROCEDURE
Prepare CHECKLIST:
• Equipment
• Patient
• Team
MAP H4
Maximize All Physiology:
• Hemodynamics
• Hypoxemia
• Hydrogen ions (pH)
• Hyper/Hypokalemia
O2
Pre-oxygenate:
• Maintain ≥ 95% SpO2
• NC at 15 LPM or NRB
◦ Consider PPV
Special Considerations:
• High ICP?
◦ lidocaine/fentanyl
• Pediatrics
• Difficult Airway
DRUG DOSE
INDUCTION
Etomidate 0.3 mg/kg
Propofol 1.5-3 mg/kg
Ketamine 2 mg/kg
PARALYSIS
Succinylcholine 1-2 mg/kg
Rocuronium 1 mg/kg
Vecuronium 0.3 mg/kg
CONFIRM ETT PLACEMENT
POST INTUBATION SEDATION
Propofol 10-50 mcg/kg/min
Midazolam 2.5-5 mg/hr
Ketamine 0.1-0.5 mg/kg/hr
Dexmedetomidine 1-1.5 mcg/kg/hr
Initial Vent Settings:*
• Mode: AC/VC
• VT: 6-8 mL/kg (IBW)
• RR: 10-20
• PEEP: 6-10 cmH2O
• FiO2: 40-100%
• IFR: 40-60 l/min
*Vent settings should be individualized to patient condition and clinical judgement.
Continuous Monitoring:
• Hemodynamics
◦ SpO2 / EtCO2, ECG, NIBP/ABP
• Sedation Adequacy
◦ RASS
• Ventilator Alarms
VASO-PRESSORS
Norepinephrine α β IV infusion: 0.5-50 mcg/min; + 1-5 mcg/min PUSH DOSE: 8-32 mcg q 2-5 min
EPINEPHrine α β IV infusion: 1-10 mcg/min; + 1-2 mcg/min PUSH DOSE: 5-30 mcg q 2-5 min
PHENylephrine α IV infusion: 40-180 mcg/min; + 10-50 mcg/min PUSH DOSE: 50-200 mcg q 2-5 min
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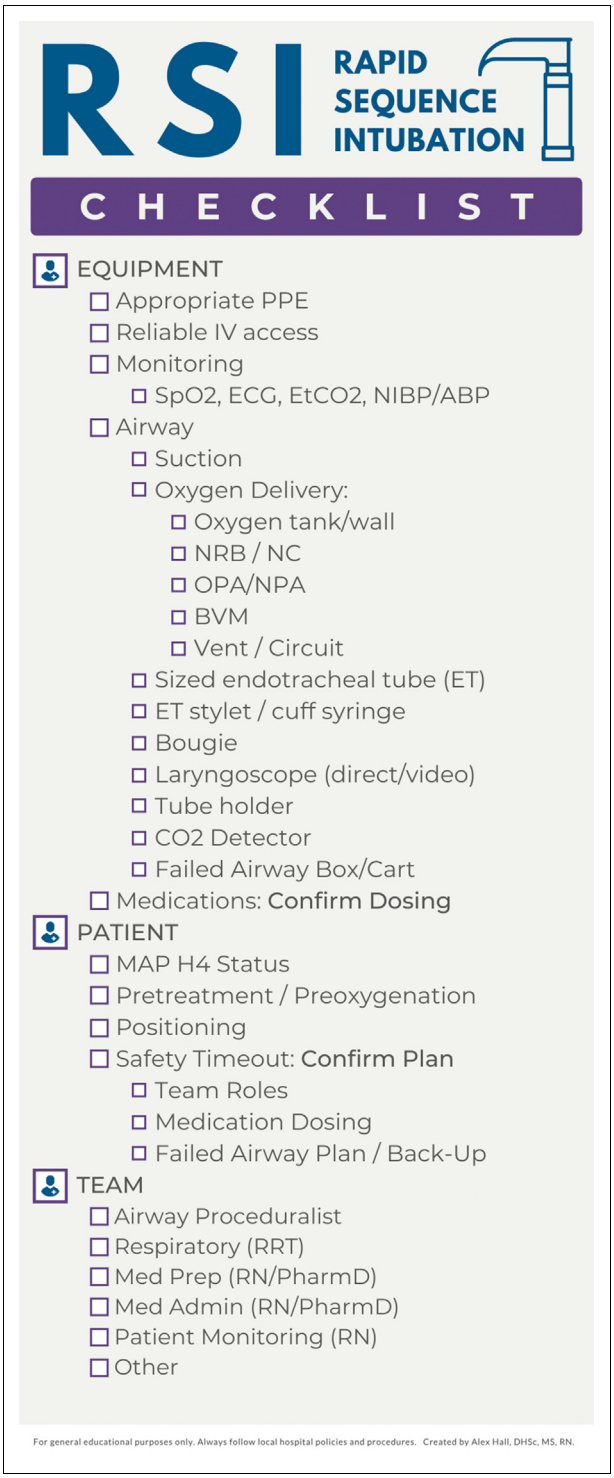
RSI
RAPID SEQUENCE INTUBATION
CHECKLIST
EQUIPMENT
☐ Appropriate PPE
☐ Reliable IV access
☐ Monitoring
☐ SpO2, ECG, EtCO2, NIBP/ABP
☐ Airway
☐ Suction
☐ Oxygen Delivery:
☐ Oxygen tank/wall
☐ NRB / NC
☐ OPA/NPA
☐ BVM
☐ Vent / Circuit
☐ Sized endotracheal tube (ET)
☐ ET stylet / cuff syringe
☐ Bougie
☐ Laryngoscope (direct/video)
☐ Tube holder
☐ CO2 Detector
☐ Failed Airway Box/Cart
☐ Medications: Confirm Dosing
PATIENT
☐ MAP H4 Status
☐ Pretreatment / Preoxygenation
☐ Positioning
☐ Safety Timeout: Confirm Plan
☐ Team Roles
☐ Medication Dosing
☐ Failed Airway Plan / Back-Up
TEAM
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