Cureus
Part of SPRINGER NATURE

Open Access Case Report

Massive Clenbuterol Overdose: A Case Report and Literature Review

Ritvik Kumar [1], Jay Patel [1], Abhishek Sharma [1]

1. Emergency Medicine, Shalby Hospitals, Ahmedabad, IND

Corresponding author: Ritvik Kumar, ritvik.kumar151@gmail.com

Review began 07/04/2026
Review ended 11/10/2026
Published 11/13/2026

© Copyright 2026
Kumar et al. This is an open access article distributed under the terms of the Creative Commons Attribution License CC-BY 4.0., which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

DOI: 10.7759/cureus.119641

Abstract

Clenbuterol, a long-acting selective beta-2 adrenoceptor agonist, is licensed as a bronchodilator in some countries but is increasingly misused for its lipolytic and anabolic effects. Acute overdose can produce significant sympathomimetic toxicity even in patients who appear well at initial presentation.

A 35-year-old female presented to the emergency department approximately 2 hours after the deliberate ingestion of an estimated 30-40 tablets of clenbuterol 40 mcg (approximate total dose 1,200-1,600 mcg) as an act of impulsive self-harm. Despite a "well" general appearance and a Glasgow Coma Scale of 15, the patient had marked sinus tachycardia (heart rate 148 bpm) with a normal blood pressure (115/85 mmHg). Investigations demonstrated mild hypokalemia (potassium 3.3 mEq/L), mild hyperglycemia (glucose 6.8 mmol/L), and a markedly elevated lactate (6.49 mmol/L); a venous blood gas showed no significant acidosis (pH 7.34, bicarbonate 24 mmol/L, anion gap 10 mmol/L), and the corrected QT interval was normal. In view of the large estimated dose, the short interval since ingestion, an intact airway, and medication packaging confirming the agent, gastric lavage was performed with recovery of tablet fragments, followed by 50 g of activated charcoal. The patient was admitted to the intensive care unit for continuous cardiac monitoring, serial electrolyte assessment, and supportive care. Tachycardia resolved spontaneously without beta-blockade (heart rate 82 bpm by 36 hours), electrolyte and metabolic abnormalities corrected, and the patient was discharged at 72 hours following psychiatric assessment and arrangement of outpatient follow-up.

This case demonstrates that clenbuterol overdose may present with a deceptively benign appearance while producing significant tachycardia and metabolic derangement that can evolve over the ensuing 24-48 hours, consistent with the drug's prolonged half-life. Supportive care with continuous cardiac monitoring and careful electrolyte management is the mainstay of treatment; beta-blockade should be reserved for selected patients rather than used routinely, and gastric decontamination should be individualized. Psychiatric assessment and follow-up are essential following deliberate self-poisoning.

Categories: Substance Use and Addiction, Emergency Medicine
Keywords: beta-2 agonist, clenbuterol, drug overdose, emergency medicine, gastric lavage, intensive care, tachycardia, toxicology

Introduction

Clenbuterol (4-amino-α-[(tert-butylamino)methyl]-3,5-dichlorobenzyl alcohol hydrochloride) is a potent, long-acting selective β2-adrenoceptor agonist. Like other β2-agonists, it relaxes bronchial smooth muscle to produce bronchodilation; it was originally developed for the treatment of reversible airway obstruction. Its distinguishing pharmacological features are a high oral bioavailability, a comparatively slow onset, and a markedly prolonged elimination half-life of approximately 25-39 hours: considerably longer than that of short-acting agents such as salbutamol [1]. This long duration of action, combined with β1-adrenoceptor cross-reactivity that emerges at supratherapeutic doses, is central both to the drug's appeal as an agent of misuse and to its capacity to produce prolonged, evolving toxicity.

Clenbuterol occupies an unusual regulatory position that differs markedly between jurisdictions. It is licensed for human use as a bronchodilator in several countries, including parts of Europe, China, and several countries in Central and South America, where it is marketed for asthma and other obstructive airway diseases [2]. In many other jurisdictions, including the United States, it has never been approved for human use and is available only for veterinary indications such as the management of airway obstruction in horses [2]. Legitimate human therapeutic use has consequently remained narrow and, in many settings, has been further constrained by the availability of safer short-acting bronchodilators and by anti-doping restrictions; as a result, a large proportion of human exposures now occur entirely outside any licensed therapeutic context. Beyond these narrow legitimate indications, clenbuterol has acquired a substantial illicit following. Its β2-mediated lipolytic and skeletal-muscle anabolic effects have made it popular as a weight-loss and performance-enhancing agent among bodybuilders, athletes, and individuals seeking rapid fat loss, with much of this supply obtained through unregulated online channels [3].

This pattern of misuse has been accompanied by a documented rise in reported toxic exposures. A

retrospective review by the New South Wales Poisons Information Centre recorded 63 clenbuterol exposures between 2004 and 2012, with annual cases increasing from 3 in 2008 to 27 in 2012, and the majority of affected patients requiring hospitalization, including one young adult who sustained a cardiac arrest [4]. It is against this background of expanding availability and misuse that acute clenbuterol poisoning represents an increasingly relevant presentation for emergency clinicians.

Acute clenbuterol toxicity produces a characteristic sympathomimetic toxidrome. The cardinal feature is sinus tachycardia, frequently accompanied by tremor, palpitations, and anxiety, together with a distinctive constellation of metabolic derangements: hypokalemia, hyperglycemia, hyperlactatemia, and hypophosphatemia, which arise from β-adrenoceptor-mediated intracellular ion shifts and altered intermediary metabolism [3,5]. In severe cases, myocardial ischemia, ventricular arrhythmias, and rhabdomyolysis have been reported [3,4]. A clinically important and potentially deceptive feature is that patients may appear well on initial assessment despite significant biochemical and hemodynamic derangement; moreover, because of the drug's prolonged half-life, toxicity may continue to evolve over the 24-48 hours following ingestion rather than declaring itself fully at presentation.

Despite these recognized hazards, the published literature on massive clenbuterol overdose remains confined to isolated case reports and small case series, and there is limited consensus on several practical management questions, including the role of gastric decontamination, the indications for and selection of β-blockade, and the intensity of monitoring warranted for a patient who appears well. We report a case of acute clenbuterol toxicity following the deliberate ingestion of an estimated 1,200-1,600 mcg, and describe the clinical course, the rationale underlying key management decisions, and the outcome. In doing so, we situate our experience within the existing literature to help guide clinicians who encounter this uncommon but increasingly recognized presentation.

Case Presentation

Patient history and presentation

A 35-year-old female patient presented to the emergency department approximately 2 hours after deliberate ingestion of an estimated 30-40 tablets of clenbuterol (40 mcg per tablet), yielding a total estimated dose of 1,200-1,600 mcg. The ingestion appeared purposeful; the precise context was documented in the clinical record and appropriate psychiatric liaison was initiated.

The patient arrived ambulant and communicative, denying chest pain, palpitations, or dyspnoea at the time of triage. There was no history of prior cardiac disease, thyroid disease, use of sympathomimetic agents, respiratory illness, or psychiatric comorbidities. Current medications, excluding the ingested substance, were nil. No co-ingestion of alcohol, opioids, benzodiazepines, or other substances was initially reported, and this was confirmed in the urine toxicology screen.

Physical examination

On initial assessment, the patient's general appearance was well; they were alert and orientated to time, place, and person, with no overt signs of respiratory distress, diaphoresis, or altered mentation. The key abnormality identified on examination was marked sinus tachycardia.

Parameter	Value on Arrival	Reference Range
Heart Rate (bpm)	148 (sinus tachycardia)	60–100
Blood Pressure (mmHg)	115/85	90–140 / 60–90
Respiratory Rate (/min)	17-18	12–20
SpO2 (%)	≥ 98% on room air	≥ 95%
Temperature (°C)	Afebrile	36.1–37.2
GCS	15/15 (E4 V5 M6)	15

TABLE 1: Vital signs

Cardiac auscultation revealed a rapid but regular rhythm with no added sounds. The chest was clear to auscultation bilaterally. The abdomen was soft and non-tender. Neurological examination, including cranial nerves, power, coordination, and reflexes, was intact. Peripheral tremor of the upper limbs was noted on closer inspection but was not distressing to the patient.

Investigations

A 12-lead electrocardiogram (ECG) performed on arrival demonstrated sinus tachycardia at approximately 148 bpm with a normal QRS axis, no ST-segment changes, and a corrected QT interval (QTc) of 400 ms. Continuous cardiac monitoring was initiated.

Laboratory investigations were drawn immediately, and a venous blood gas was obtained. Significant findings included mild hypokalemia with a serum potassium of 3.3 mEq/L (reference 3.5-5.0 mEq/L), mild hyperglycemia with a blood glucose of 6.8 mmol/L (reference 3.9-6.1 mmol/L fasting), and markedly elevated serum lactate at 6.49 mmol/L (reference < 2.0 mmol/L). Renal and hepatic function tests were within normal limits, the full blood count revealed no significant abnormality, and urine toxicology screening returned negative for all other substances. Investigations are summarized in Table *2*.

Investigation	Result	Reference	Interpretation
Serum Potassium	3.3 mEq/L	3.5–5.0 mEq/L	Mildly Low ↓
Blood Glucose	6.8 mmol/L	3.9–6.1 mmol/L	High ↑
Serum Lactate	6.49 mmol/L	< 2.0 mmol/L	High ↑
QTc Interval	400 ms	< 440 ms	Normal
Renal Function	Normal	—	Normal
Liver Function	Normal	—	Normal

TABLE 2: Investigations

Emergency department management

Given the large estimated ingested dose and the relatively short interval since ingestion (approximately 2 hours), a decision was made to proceed with gastric lavage following a careful airway assessment and informed consent. A nasogastric tube (16 Fr) was inserted, and the stomach was irrigated with sequential aliquots of warm normal saline (200-300 mL per cycle) until the return was clear. Activated charcoal was administered following lavage to adsorb any remaining drug in the gastrointestinal tract. Tablet fragments were recovered. Intravenous access was secured with two large-bore peripheral cannulae, the patient was placed on continuous cardiac monitoring, and intravenous fluids were commenced for hydration and renal protection.

Selective beta-1 blockade with intravenous metoprolol (2.5 mg slow IV push, titrated to effect) was considered for rate control; however, given the patient's otherwise stable hemodynamic status, the attending team elected for cautious observation and deferred pharmacological rate control pending ICU consultation, to avoid precipitating hypotension or bronchospasm.

ICU admission and course

The patient was admitted to the intensive care unit for continuous monitoring and symptomatic management. Continuous cardiac monitoring demonstrated a gradual decline in heart rate over the initial 12-24 hours as the drug’s effects waned, consistent with clenbuterol’s prolonged elimination half-life. Serial serum potassium levels were measured every 4-6 hours; hypomagnesemia was also identified and corrected with appropriate supplementation. Hyperglycemia proved transient and self-limiting, resolving without the need for insulin intervention, and strict fluid balance monitoring was maintained throughout, with urine output remaining adequate.

Serial creatine kinase (CK) measurements were obtained for rhabdomyolysis surveillance and demonstrated a mild, transient elevation without clinical myoglobinuria; levels trended downward without the need for specific intervention. A formal psychiatric assessment was conducted during the ICU stay, with appropriate liaison and outpatient follow-up arrangements subsequently made. The patient remained neurologically intact throughout the admission, with no seizure activity documented.

No vasopressors, mechanical ventilation, or advanced hemodynamic support were required at any point. The patient’s tachycardia resolved progressively without pharmacological intervention; at 36 hours post-ingestion, the heart rate had normalized to 82 bpm.

Outcome and discharge

The patient was stepped down from the ICU to a general medical ward at 48 hours post-admission, at which point all vital signs, electrolytes, renal function, and cardiac monitoring parameters had normalized. Repeat ECG at 48 hours showed normal sinus rhythm with no repolarization abnormalities. The patient was medically fit for discharge at 72 hours following full clinical recovery and completion of the psychiatric assessment with arranged outpatient follow-up.

Discussion

Pharmacology and mechanism of toxicity

Clenbuterol exerts its primary effects through agonism of beta-2 adrenoceptors located on bronchial smooth muscle, skeletal muscle, adipose tissue, and cardiac muscle [6]. At therapeutic doses, it produces bronchodilatation with minimal cardiovascular side effects [6]. However, at supratherapeutic doses, as in this case, beta-1 adrenoceptor cross-reactivity becomes clinically significant, resulting in positive chronotropic and inotropic effects manifested as tachycardia and, potentially, arrhythmias [3].

The drug's pharmacokinetic profile compounds the clinical challenge: clenbuterol has an oral bioavailability of approximately 70-80%, reaches peak plasma concentration at 2-3 hours post-ingestion, and has a prolonged elimination half-life of 25-39 hours [1,6]. This means that even when a patient appears well at initial presentation, as occurred in this case, significant toxicity may still be evolving, and effects may persist for 48 hours or more [1]. The hallmark metabolic derangements of clenbuterol toxicity--hypokalemia, hyperglycemia, and hyperlactatemia--are consequences of beta-2 receptor-mediated intracellular potassium shifts (via Na+/K+-ATPase activation), stimulation of glycogenolysis and gluconeogenesis, and increased skeletal muscle lactate production, respectively [1,6]. These metabolic effects can potentiate the risk of ventricular arrhythmias, particularly in the context of concurrent hypokalemia [3].

Clinical presentation and the "well-appearing" paradox

A clinically important feature of this case, and one corroborated by the existing literature, is the discordance between the patient's benign general appearance and the underlying severity of sympathomimetic toxicity. This phenomenon has been described in prior case reports of clenbuterol poisoning and may lead to underestimation of clinical risk by emergency physicians unfamiliar with the drug's toxidrome [3]. The predominant presenting feature in clenbuterol overdose is sinus tachycardia, which was the cardinal finding in this patient [3]. Other reported features in the literature include tremor, palpitations, headache, nausea, hypertension, chest tightness, hypokalemia-related muscle weakness, and, in severe cases, ventricular tachycardia, myocardial ischemia, and acute kidney injury secondary to rhabdomyolysis [3,7]. The absence of these more severe features in our patient likely reflects both the adequacy of early decontamination and the absence of co-ingestants.

Gastric decontamination

Contemporary toxicological guidance does not support the routine use of gastric lavage. The position paper of the American Academy of Clinical Toxicology and the European Association of Poisons Centres and Clinical Toxicologists concludes that gastric lavage should not be undertaken routinely, if at all, in poisoned patients; that the evidence for benefit even in selected situations is weak; and that it should be considered only when a patient has ingested a potentially life-threatening quantity of a poison and the procedure can be performed soon after ingestion, generally within 60 minutes, in a patient able to protect their airway [8]. The procedure carries recognized hazards, including aspiration pneumonitis, hypoxia, esophageal or gastric injury, and arrhythmia, which must be weighed against any anticipated benefit [8]. Measured against this standard, the decision to perform lavage in the present case was not fully guideline-concordant, principally with respect to timing: the procedure was undertaken approximately two hours after ingestion, beyond the window in which benefit is considered most likely. We nonetheless judged lavage to be justifiable based on several converging factors. First, the estimated ingested dose (1,200-1,600 mcg, equivalent to 30-40 tablets of 40 mcg) was of the order of 10- to 20-fold the maximum therapeutic dose and carried a realistic potential for life-threatening cardiovascular toxicity. Second, the patient was fully alert with intact airway reflexes, was cooperative, and was formally assessed to be at low risk of aspiration, mitigating the principal hazard of the procedure. Third, the medication packaging brought by the patient corroborated both the identity of the agent and the plausibility of a large ingestion. Finally, clenbuterol is absorbed relatively slowly and has a prolonged pharmacokinetic profile, raising the possibility that unabsorbed drug remained in the stomach beyond the conventional one-hour window. The recovery of visible tablet fragments during lavage supported this reasoning and confirmed that the drug was still present in the stomach at the time of the procedure.

Activated charcoal (50 g) was administered following lavage. Clenbuterol is adsorbed by activated charcoal, and single-dose activated charcoal is a reasonable adjunct after recent ingestion of a substantial dose in a patient with a protected airway [9]. There is no specific antidote for clenbuterol toxicity, and management is otherwise supportive [1].

Pharmacological management considerations

Beta-adrenoceptor antagonists, particularly non-selective agents, such as propranolol, have been reported to reverse tachycardia, hypokalemia, and tremor in clenbuterol poisoning by competitive receptor antagonism [3,5]. Some authors advocate for selective beta-1 blockers (e.g., metoprolol, atenolol) to minimize the theoretical risk of unopposed alpha-adrenoceptor activity or bronchospasm, though this risk is generally considered low in this context [5,6]. In the present case, spontaneous resolution of tachycardia without pharmacological rate control was achieved, which is a reasonable approach in hemodynamically stable patients with mild-to-moderate toxicity. Electrolyte replacement, particularly potassium, is critical. However, caution must be exercised: the observed hypokalemia in clenbuterol toxicity reflects redistribution rather than true total body depletion [6]. Aggressive potassium replacement carries a risk of rebound hyperkalemia as drug effects wane [6]. Serial electrolyte monitoring is therefore mandatory.

Literature context

Clenbuterol poisoning, while historically uncommon, has been increasingly reported in association with its misuse in sport, bodybuilding, and weight loss [7,10]. High-dose tablet formulations, such as the 40 mcg tablets described in this case, have been particularly implicated in severe toxicity, as these represent doses 10- to 20-fold higher than the maximum therapeutic dose [7]. Published case series have documented cases ranging from mild tachycardia and tremor to life-threatening ventricular arrhythmias and myocardial infarction [3-5,11]. Fatalities, while rare, have been reported, particularly in cases involving very large doses, co-ingestants, or underlying cardiac disease [7,11]. The absence of fatality in our case, despite a very large estimated dose, may be attributable to prompt ED presentation, early gastric decontamination, and the absence of complicating factors.

Limitations

Several limitations should be borne in mind when interpreting this report. First, as a single, uncontrolled case, it can describe an approach and an outcome but cannot establish causation or the relative efficacy of any individual intervention; the observations reported here should not be generalized into management protocols. Second, the ingested dose was estimated from the patient's verbal account and the medication packaging rather than confirmed analytically: the patient was unable to recall the exact number of tablets taken, and no quantitative serum clenbuterol concentration was obtained, so the stated dose of 1,200-1,600 mcg represents an approximation. Third, standard urine toxicology does not detect clenbuterol and screens only for a defined panel of substances; although co-ingestion was not clinically apparent, agents outside the tested panel cannot be entirely excluded. Fourth, a clinical toxicologist or poisons information center was not consulted beforehand and, in retrospect, early consultation would have been preferable; it is recommended in comparable presentations. Furthermore, as an uncontrolled single case, this report cannot establish that lavage altered the clinical course, and it is not possible to determine whether decontamination contributed to the favourable outcome or whether supportive care alone would have sufficed. Finally, it is not possible to determine from this case whether gastric decontamination influenced the clinical trajectory, or whether an equally favourable outcome would have been achieved with supportive care alone.

We therefore present our experience descriptively rather than as an endorsement of gastric lavage in clenbuterol overdose, and we caution against extrapolating from it to routine practice.

Conclusions

This case illustrates that acute clenbuterol toxicity following massive ingestion can present with a deceptively benign initial appearance. Clinicians should not be reassured by a "well" general appearance alone: sinus tachycardia, electrolyte disturbance, and metabolic derangement may be the principal manifestations, and, because of the drug's prolonged elimination half-life, toxicity may evolve over the hours following presentation rather than being maximal at triage. Continuous cardiac monitoring and serial biochemical assessment over an adequate observation period are therefore prudent even in a patient who initially appears well. Supportive care remains the mainstay of management. In this patient, tachycardia resolved spontaneously and no specific pharmacological rate control was required; beta-adrenoceptor blockade is best reserved for selected patients with significant or sustained cardiovascular compromise rather than applied routinely. Electrolyte abnormalities warrant careful monitoring with judicious replacement, recognizing that the hypokalemia of β2-agonist toxicity is largely redistributive and that over-replacement risks rebound hyperkalemia as drug effect wanes. Where gastric decontamination is considered, the decision should be individualized and guided by current toxicological recommendations and, ideally, early poisons-service advice, rather than undertaken routinely. Psychiatric assessment and follow-up should not be overlooked in cases of deliberate self-poisoning.

Given the increasing availability and misuse of high-dose clenbuterol formulations, emergency physicians, intensivists, and toxicologists should maintain an appropriate index of suspicion for this uncommon but potentially serious presentation. Larger case series and prospective registry data would help better define its epidemiology and optimal management.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Ritvik Kumar, Jay Patel

Acquisition, analysis, or interpretation of data: Ritvik Kumar, Jay Patel, Abhishek Sharma

Drafting of the manuscript: Ritvik Kumar, Jay Patel, Abhishek Sharma

Critical review of the manuscript for important intellectual content: Ritvik Kumar, Jay Patel, Abhishek Sharma

Supervision: Jay Patel, Abhishek Sharma

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

References

1. Daubert GP, Mabasa VH, Leung VW, Aaron C: Acute clenbuterol overdose resulting in supraventricular tachycardia and atrial fibrillation. J Med Toxicol. 2007, 3:56-60. 10.1007/BF03160909
2. Kataveni S, Gourishetty RP, Mundada SM, Avvaru MP, Kollipara VS, Gottimukkala SP: Clenbuterol abuse in bodybuilding and athletics: unsupervised use, psychological motivations, and health consequences. Cureus. 2025, 17:e84904. 10.7759/cureus.84904
3. Spiller HA, James KJ, Scholzen S, Borys DJ: A descriptive study of adverse events from clenbuterol misuse and abuse for weight loss and bodybuilding. Subst Abus. 2013, 34:306-12. 10.1080/08897077.2013.772083
4. Brett J, Dawson AH, Brown JA: Clenbuterol toxicity: a NSW poisons information centre experience . Med J Aust. 2014, 200:219-21. 10.5694/mja13.10982
5. Hoffman RJ, Hoffman RS, Freyberg CL, Poppenga RH, Nelson LS: Clenbuterol ingestion causing prolonged tachycardia, hypokalemia, and hypophosphatemia with confirmation by quantitative levels. J Toxicol Clin Toxicol. 2001, 39:339-44. 10.1081/clt-100105152
6. Perry PJ, Lund BC, Deninger MJ, Kutscher EC, Schneider J: Anabolic steroid use in weightlifters and bodybuilders: an internet survey of drug utilization. Clin J Sport Med. 2005, 15:326-30. 10.1097/01.jsm.0000180872.22426.bb
7. Witkowska-Piłaszewicz O, Pingwara R, Szczepaniak J, Winnicka A: The effect of the clenbuterol-β2-adrenergic receptor agonist on the peripheral blood mononuclear cells proliferation, phenotype, functions, and reactive oxygen species production in race horses in vitro. Cells. 2021, 10:936. 10.3390/cells10040936
8. Benson BE, Hoppu K, Troutman WG, et al.: Position paper update: gastric lavage for gastrointestinal decontamination. Clin Toxicol (Phila). 2013, 51:140-6. 10.3109/15563650.2013.770154
9. American Academy of Clinical Toxicology; European Association of Poisons Centres and Clinical Toxicologists: Position statement and practice guidelines on the use of multi-dose activated charcoal in the treatment of acute poisoning. J Toxicol Clin Toxicol. 1999, 37:731-51. 10.1081/clt-100102451
10. Bonney CF, Hatten B, Wang GS: Toxicity from unintentional pediatric ingestion of a performance-enhancing drug: a case report with review of clenbuterol toxicity and treatment. J Emerg Med. 2019, 57:e105-8. 10.1016/j.jemermed.2019.06.016
11. Lan K, Saheba A, Mathew P: Low dose clenbuterol toxicity: case report and review of literature . HCA Healthc J Med. 2020, 1:201-4. 10.36518/2689-0216.1086