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Case Report - Deliberate Self-Harm with Enalapril Poisoning: A Rare Case Successfully Managed in a Low - Resource Setting in Sri Lanka

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Case Report

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Abstract

Enalapril is a widely used angiotensin-converting enzyme inhibitor prescribed for hypertension. It is a prodrug converted to its active metabolite, enalaprilat, with pharmacokinetic properties that may require twice-daily dosing for optimal 24-hour blood pressure control. Despite its widespread availability in Sri Lanka, reports of acute overdose are exceedingly rare. We report the first documented case of adult enalapril overdose in Sri Lanka involving a 37-year-old male who presented after intentional ingestion of 100 mg. He developed severe symptomatic hypotension and transient stage 2 acute kidney injury (AKI), likely secondary to prerenal azotemia from systemic hypotension. Initial management included gastric decontamination with activated charcoal and aggressive intravenous fluid resuscitation. Due to persistent hemodynamic instability, vasopressor support with noradrenaline was required. Serial biochemical monitoring demonstrated subsequent clinical improvement, and renal function recovered without the need for hemodialysis, although extracorporeal removal remains a recognized option for enalaprilat clearance in severe cases.

Categories: Emergency Medicine, Pharmacology, Internal Medicine

Introduction

In Sri Lanka, hanging remains the most common method of deliberate self-harm, while pesticide-related suicides, although reduced through effective regulatory measures, remain the second most common method. An increase in firearm-related suicides, high-point jumping, and other emerging methods has also been reported [1]; however, Drug poisoning involving commonly available antihypertensive agents is infrequently reported.

Enalapril is a dicarboxylic acid monoester that is chemically described as ethyl 4-phenylbutanoate, in which a hydrogen alpha to the carboxy group is substituted by the amino group of L-alanyl-L-proline (S configuration). It has a role as a prodrug. It is also a dipeptide and is functionally related to enalaprilat anhydrous [2]. It belongs to the angiotensin - converting enzyme (ACE) inhibitor drug class that acts on the renin angiotensin -

aldosterone system, which regulates blood pressure and fluid and electrolyte homeostasis. It is an orally active, long-acting, non-sulfhydryl antihypertensive agent. [3]. As a prodrug, enalapril is rapidly converted to its active metabolite, enalaprilat, which is responsible for its pharmacological actions. Enalaprilat competitively inhibits ACE, thereby reducing the production of angiotensin II, a key component of the renin-angiotensin-aldosterone system that promotes vasoconstriction and renal sodium reabsorption in the kidneys. This results in decreased vasoconstriction and reduced renal sodium reabsorption, ultimately lowering blood pressure and circulating fluid volume. Long-term blockade of the renin-angiotensin system is generally well tolerated [4].

It was developed through targeted research using molecular modelling. Enalapril was approved by the FDA for the management of hypertension, heart failure, and asymptomatic left ventricular dysfunction,

as well as for its antiproteinuric effects [5]. It is also available in combination with hydrochlorothiazide for hypertension management [5]. The active metabolite, enalaprilat, is available in intravenous formulations for injection [6]. In Sri Lanka, there is limited information regarding acute enalapril overdose. We report a case of a patient who attempted self-harm by ingesting a large dose of enalapril.

Case Presentation

The patient was a 37-year-old male construction laborer with no known chronic illnesses who intentionally ingested 100mg of enalapril at once (5mg x 20 tablets) on an empty stomach (0 hours) in a suicide attempt following a quarrel with his wife. He was previously in good health, with a body weight of 68 kg. He did not smoke or consume alcohol. The tablets belonged to his mother, a known hypertensive patient living with him. Ingestion was confirmed by himself and corroborated by empty enalapril blister packs found in his bedroom. He denied co-ingestion of other medications, toxic substances, or alcohol.

Immediately after ingestion, he developed abdominal bloating and nausea. He was found by his wife approximately 30 minutes post-ingestion, at which time he was conscious and oriented, with only mild dizziness. He was admitted to Teaching Hospital, Jaffna, Sri Lanka approximately 1 hour after ingestion. On admission, his blood pressure was 124/80 mmHg, and systemic examination was unremarkable. His Glasgow Coma Scale (GCS) score was 15. Initial investigations-including blood count, serum creatinine, electrolytes, liver function tests, random blood glucose, and electrocardiogram were within normal limits. During initial resuscitation, two wide-bore intravenous cannulas were inserted, and 1 to 2L of 0.9% sodium chloride was administered intravenously as a rapid flush. This was followed by gastric lavage with 0.9% saline via a nasogastric tube and administration of activated charcoal (68 g; approximately 1 g/kg) orally to reduce further drug absorption. At 4 hours post-ingestion, the patient remained fully oriented (GCS 15) but complained of persistent dizziness and nausea. His vital signs showed hypotension compared with his baseline (blood pressure 100/68 mmHg) with a weak but palpable pulse at 122 beats per minute; respiratory rate was 22 breaths per minute, temperature 97.4°F, cold peripheries and oxygen saturation was 97% on room air.

Physical examination remained otherwise unremarkable. He was subsequently admitted to the High Dependency Unit (HDU) for close monitoring. Continuous monitoring of blood pressure and heart rate was undertaken. Measurement of serum enalaprilat concentration, plasma angiotensin II levels, and angiotensin-converting enzyme (ACE) activity was not performed due to unavailability in the state sector and patient refusal to undergo testing in private laboratories, because of higher cost in our setting.

Further management included aggressive intravenous fluid resuscitation with crystalloid, with cumulative administration reaching 7L over 48 hours guided by hemodynamic response including additional boluses as required. Due to persistent hypotension, noradrenaline infusion was initiated at 0.1 µg/kg/min at approximately 7 hours post ingestion when hemodynamic parameters did not improve despite repeated fluid challenges.

Over the next several hours, his blood pressure fluctuated between systolic blood pressure 70 to 90mmHg, with titration of noradrenaline (up to 0.4 - 0.6 mcg/kg/min), mean arterial pressure was maintained around 65-70 mmHg. After receiving a total of approximately 8000 mL of 0.9% sodium chloride, his blood pressure improved to 110/70 mmHg within 48 hours. With continuous monitoring of vital signs and biochemical parameters, we were able to alleviate the toxic effects of enalapril. **Table 01** summarizes the temporal evolution of hemodynamic parameters and interventions, demonstrating the development of hypotensive shock at 7 hours post-ingestion, requirement for vasopressor escalation, and gradual hemodynamic recovery over 48 hours.

At discharge, his blood pressure stabilized at 124/86 mmHg. Counselling was arranged to prevent future incidents. Laboratory investigations remained within normal limits throughout hospitalization, except for a transient acute kidney injury stage 2 (AKI), which subsequently resolved, with mildly swollen kidneys on ultrasound likely secondary to prerenal azotemia from hypotension in the context of ACE inhibitor poisoning. Other hematological, biochemical, cardiac, and imaging findings were largely unremarkable. Detailed investigation results are summarized in **Table 2.**

Time (hours)	Blood Pressure (mmHg)	Pulse (/min)	SpO_2 (%)	Intervention
1	124/80	110	99	**Gastric lavage, activated charcoal 68 g, 2000 mL crystalloid infusion**
2	116/72	118	98	500ml crystalloid infusion
3	130/90	124	99	200ml/h crystalloid infusion
4	100/68	122	97	150ml/h crystalloid infusion
5	90/60	130	95	1000ml bolus crystalloid infusion
7	70/40	138	94	Oxygen 2**L/min,** Lactate 4.4 mmol/L, 150ml/h crystalloid infusion, Noradrenaline 0.1/mcg/kg/min
9	96/66	124	98	Oxygen 2**L/min,** 120ml/h crystalloid infusion, Noradrenaline 0.2/mcg/kg/min, IVC assessment within normal range
14	100/60	120	98	Oxygen 0.5**L/min,** 100ml/h crystalloid infusion, Noradrenaline 0.2/mcg/kg/min
20	110/76	110	97	100ml/h crystalloid, Lactate 1.4 mmol/L, Noradrenaline 0.4/mcg/kg/min
30	124/88	100	96	100ml/h crystalloid infusion, Noradrenaline 0.6/mcg/kg/min
38	130/90	110	97	100ml/h crystalloid infusion, Noradrenaline 0.4/mcg/kg/min
48	120/80	106	98	100ml/h crystalloid infusion

(Note: Limited lactate analysis was performed due to low availability of blood gas analysis reagents; Inferior Ven cava - IVC.)

Table 01: Timeline of important patient parameters and interventions

Laboratory parameters	Values	**Normal range**
White blood cell count (WBC)	7.36 x 10^9 /L	4.0 - 11.0 x 10^9 /L
Hemoglobin (Hb)	15.3 g/dL	12 - 17 g/dL
Red blood cell count (RBC)	4.91 x 10^{12} /L	4.0 - 5.6 x 10^{12} /L
Mean corpuscular volume (MCV)	91fL	86.0 - 100.0 fL
Platelet count (PLT)	215 x 10^9 /L	150 - 450 x 10^9 /L
Serum Sodium	139 - 135 mmol/L	136 - 145 mmol/L
Serum Potassium	4.1 - 4.6 mmol/L	3.5 - 5.1 mmol/L
Serum Creatinine	52 to 120 → 90 → 70 µmol/L	62- 115 µmol/L
AST	13 U/L	16 -63 U/L
ALT	49 U/L	15 - 37 U/L
ALP	102 U/L	46 - 116 U/L
Total protein	72 g/L	64 - 82 g/L
Albumin	38 g/L	34 - 50 g/L
Total bilirubin	22.8 µmol/L	0 - 17 µmol/L
C-reactive protein (CRP)	3.4 mg/L → 9.0 mg/L	0 - 3 mg/L
APTT	24.2 seconds	20 - 28 seconds
INR	1.23	< 1.4
Urine analysis	Clear appearance, Trace protein, WBC 1-2, and nil RBC	
Serum Magnesium	0.9 mmol/L	0.7 - 1.0 mmol/L
Serum Phosphate	1.2 mmol/L	0.8 - 1.45 mmol/L
Corrected Calcium	2.12 mmol/L	2.1 - 2.54 mmol/L

Thyroid stimulating hormone (TSH)	1.5 mIU/L	0.45 - 4.1 mIU/L
Troponin I	0.020 ng/mL	< 0.120 ng/mL
Chest radiograph	Normal lung fields.	
Electrocardiogram	Demonstrated T-wave inversions in V1 with sinus rhythm	
Ultrasound abdomen	revealed mildly swollen kidneys suggestive of acute kidney injury.	
Two-dimensional echocardiography	Normal with an ejection fraction of 60%.	

TABLE 2: Detailed investigation and results

Discussion

Enalapril is a safe drug at normal therapeutic doses [7]. It is a long-acting angiotensin-converting enzyme (ACE) inhibitor that is hydrolyzed in vivo to its active metabolite, enalaprilat [8]. Its pharmacokinetics are not significantly affected by circadian rhythm, and the administration of enalapril does not markedly alter the time course of ACE inhibition [9]. Poisoning due to enalapril ingestion can result in significant hypotension through blockade of the renin-angiotensin system [10]. Patients with ACE inhibitor overdose are usually managed effectively with fluid resuscitation, with or without the addition of vasopressor, and in some cases, angiotensin II infusion has been used [11]. Although angiotensin II is not freely available in our setting, its use has shown efficacy in antihypertensive overdose and may be particularly beneficial in ACE inhibitor toxicity. However, further studies are required to confirm its appropriate indications.

In this case report, the patient ingested a potentially toxic dose of enalapril and developed life-threatening hypotension shortly after ingestion. Clinical evidence, including a history of ingestion and an empty blister pack, supported the diagnosis. This was likely due to high concentrations of the active metabolite, enalaprilat, sufficient to inhibit ACE activity.

The patient remained hypotensive despite aggressive intravenous fluid resuscitation and vasopressor therapy. Measurement of plasma converting enzyme activity can serve as an excellent indicator of sufficient drug exposure and may act as a useful “bioassay” in monitoring poisoning. Additionally, plasma ACE activity has been shown to remain stable for several weeks when stored at − 20°C [8]. However, in our resource-limited setting, we were unable to measure serum concentrations of enalapril and enalaprilat, plasma renin activity, or levels of angiotensin II and aldosterone following ingestion. Pharmacokinetic data on enalapril overdose remain limited among the Sri Lankan population.

The patient developed stage 2 acute kidney injury (AKI), which did not necessitate hemodialysis. There is limited evidence in the literature regarding clear indications for renal replacement therapy (hemodialysis) in acute enalapril overdose. However, enalaprilat is one of the few toxins that is amenable to dialysis due to its water solubility, small volume of distribution, and minimal protein binding. Therefore, hemodialysis may be an appropriate method for removal [10], although it can be challenging in hemodynamically unstable patients.

Additional benefits of hemodialysis may include correction of metabolic acidosis and normalization of electrolyte disturbances such as hypokalemia. It may be particularly useful in patients who do not respond to standard treatment, especially those with renal impairment, which can increase sensitivity to the drug and delay its clearance [10].

This patient was a previously healthy adult male with no underlying disease. A large volume of intravenous fluids was administered to maintain blood pressure. However, this approach should be used cautiously in patients with chronic kidney disease, heart failure, or in the elderly, as it may increase the burden on the kidneys and heart, potentially leading to organ failure and pulmonary edema. These patients may develop edema and effusion, which may have been related to enalapril toxicity itself, as well as to the administration of high doses of vasopressor therapy and rapid fluid infusion [10].

Conclusions

Acute intentional overdose with enalapril is uncommon but may lead to profound vasodilatory shock and transient acute kidney injury, even in previously healthy individuals. This case demonstrates that severe hypotension can persist despite aggressive crystalloid resuscitation and may require prolonged vasopressor support. Early recognition, prompt gastric decontamination, close hemodynamic monitoring, and timely escalation of supportive therapy were crucial in achieving a favorable outcome in our patient.

This report also highlights the challenges of managing ACE inhibitor toxicity in a resource-limited setting, where advanced toxicological assays and therapies such as angiotensin II infusion may not be readily available. Although our patient recovered with conservative management alone, hemodialysis remains a potential adjunctive therapeutic option in selected severe cases, particularly in patients with refractory shock or significant renal impairment, owing to the dialyzable nature of enalaprilat. Increased awareness of the clinical manifestations, complications, and management strategies of enalapril poisoning may help improve patient outcomes and contribute to the development of future management protocols in similar healthcare settings.

Declarations

Ethics and Consent to Participate

Ethical approval

was not required for this case report according to institutional policy.

Consent for Publication

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Patient Perspective:

The patient expressed gratitude for the prompt treatment received and reported complete recovery following discharge. He appreciated the counselling provided and stated that the experience reinforced the importance of seeking help during emotional distress.

Competing Interests

The authors declare that they have no competing interests.

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Author Contribution

AS contributed substantially to the clinical management of the patient, conception and design of the work, acquisition, analysis, and interpretation of data, and drafting and revision of the manuscript.NJ contributed substantially to the clinical management of the patient, conception and design of the work, acquisition, analysis, and interpretation of data, and drafting and revision of the manuscript.PT contributed substantially to the clinical management of the patient, conception and design of the work, acquisition, analysis, and interpretation of data, and drafting and revision of the manuscript.All authors read and approved the final manuscript and agree to be accountable for all aspects of the work.

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Availability of Data and Materials

The information used in preparing this case report was obtained from the patient's bed head ticket at Teaching Hospital Jaffna, Sri Lanka. All relevant data generated or analyzed during this study are included in this published article. This report does not contain any patient-identifiable information that could compromise privacy. Additional data may be made available by the corresponding author upon reasonable request, subject to ethical, institutional, and confidentiality requirements.

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