

**Retrospective study on clinical and laboratory profile of Cleistanthus collinus poisoning**

¹Dr.Kiran Thomas, Associate Professor, Department of General Medicine, Government Medical College Palakkad.

²Dr.Leena Philipose, Associate Professor, Department of Dentistry, Government Medical College Palakkad

Corresponding Author: Dr. Kiran Thomas, Associate Professor, Department of General Medicine Government Medical College, Palakkad.

How to citation this article: Dr. Kiran Thomas, Dr. Leena Philipose, “Retrospective study on clinical and laboratory profile of Cleistanthus collinus poisoning”, IJMACR– October– 2026, Volume – 9, Issue – 5, P. No. 623 – 629.

Open Access Article: © 2026 Dr. Kiran Thomas, et al. This is an open access journal and article distributed under the terms of the creative common’s attribution license (<http://creativecommons.org/licenses/by/4.0>). Which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

Type of Publication: Original Research Article

Conflicts of Interest: Nil

Abstract

Introduction: Cleistanthus collinus is one of the most common plant poisoning encountered in South India. Cleistanthus collinus (C.collinus), belongs to the family Euphorbiaceae and is commonly found in South India, Srilanka, Africa and Malaysia. All parts of the plant are poisonous but leaves are commonly used for poisoning. The toxin Cleistanthins A, B are considered to be responsible for major toxic effects causes uncoupling of oxidative phosphorylation and leads to increased ADP level. This is likely to be the mechanism causing distal renal tubular acidosis in humans and subsequent clinical features. Here we have done a retrospective study on clinical and laboratory profile of Cleistanthus collinus poisoning.

Materials and Methods: Adult patients with a diagnosis of Cleistanthus collinus poisoning, between January 2022 and December 2023 requiring admission to a medical ward, the Medical ICU, or high dependency unit (HDU) of the teaching hospital

of Government Medical College, Palakkad were included in the study. The patients were identified from the data extracted from inpatient medical records. Descriptive statistics were obtained for all variables in the study.

Results: The study comprised of 49 patients. 32 patients were males and 17 patients were females. The mean age of the patients was 34.66 ± 8.31 (mean $\pm$ SD). The average time between consumption of C.collinus and presentation to the hospital was 5.41 ± 2.61 hrs. Vomiting was the commonest symptom at presentation. 43 (87.75%) patients presented with vomiting. Hypokalemia was the commonest laboratory parameter abnormality and was observed in 39 (79.59%) patients and mean potassium level was 3.11 ± 0.43 mmol/L. Metabolic acidosis was observed in 28 (57.14%) patients and mean bicarbonate levels were 19.31 ± 2.7 . The overall mortality in our study was 14.28%. And death occurred due to Type 1 respiratory failure, refractory

hypotension, metabolic acidosis and multi organ failure.

Conclusion: Cleistanthus collinus poisoning commonly leads to renal tubular dysfunction and subsequent dyselectrolytemia and metabolic acidosis. It can also lead to Type 1 respiratory failure. Death occurs due to refractory hypotension, metabolic acidosis, renal tubular dysfunction and subsequent multi organ failure. Management is essentially supportive with close monitoring preferably in intensive care units especially in patients with features of organ failure. Overall mortality is around 15% in these patients.

Keywords: Cleistanthus Collinus, Dyselectrolytemia, Hypokalemia.

Introduction

Poisoning is a major public health problem world over. Acute deliberate self-poisoning is an important cause of admission to intensive care units (ICUs) in India. Cleistanthus collinus is one of the most common plant poisoning encountered in South India.¹ Cleistanthus collinus, belongs to the family Euphorbiaceae and is commonly found in South India, Sri Lanka, Africa and Malaysia. It is known by the following names in various Indian languages, Garari (Hindi), Vadisaaku (Telugu), Oduvanthalai (Tamil), and Odaku (Malayalam). All parts of plant are poisonous but it's the leaves that are most commonly used in poisoning. The leaves are consumed either by chewing or by making a decoction of the leaves. Its also used as cattle poison and abortifacient. Easy availability make it one of the most common method of plant poisoning in South India especially among women. Cleistanthus A, B are considered to be responsible for major toxic effects in humans. C. collinus causes

uncoupling of oxidative phosphorylation and leads to increased ADP level.² This is likely to be the mechanism causing distal renal tubular acidosis in humans and subsequent clinical and laboratory features.

Materials and Methods

Patients and settings

Adult patients with a diagnosis of Cleistanthus collinus poisoning, between January 2022 and December 2023 requiring admission to a medical ward, the Medical ICU, or high dependency unit (HDU) of a teaching hospital of GMC Palakkad are included in the study. The patients were identified from the data extracted from inpatient medical records.

A diagnosis of C. collinus poisoning was made when a patient presented with an alleged history of consumption of C. collinus and exhibited clinical symptoms of C. collinus toxicity.

A detailed history of the amount of poison consumed whether as leaves or paste or any other part of the tree, time of consumption, time of presentation to hospital, and any form of treatment given prior to presenting to the hospital were documented. Physical examination findings including vital signs like pulse, blood pressure and oxygen saturation were noted from the case records. The results of routine blood tests were noted including complete blood count, liver function tests, renal function tests, arterial blood gas analysis and electrolytes. Heart rate, ST-T changes, and rhythm abnormalities from the baseline electrocardiogram (ECG) and serial ECGs subsequently taken were also noted. Treatment outcomes were noted as cured, died, referred.

Patient Management and outcome parameters

All patients were managed with supportive therapy. This included fluid resuscitation, correction of dyselectrolytemia, hemodynamic support as and when needed, monitoring and management of cardiac rhythm abnormalities if any, respiratory support etc.

The outcomes of interest were duration of hospital stay, electrolyte abnormalities, arterial blood gas analysis variations and mortality.

Ethical approval and funding

This retrospective observational study protocol was approved by the Institutional Ethics Review Board, Government Medical College, Palakkad. The data for the study was collected from inpatients medical records and collected information was anonymized.

Statistical analysis

Descriptive statistics were employed for all the variables in the study. All continuous data were expressed as mean with standard deviation. Statistical analysis was done using Statistical Package for Social Sciences for windows.(SPSS Inc version 16)

Results

Baseline Characteristics

The study comprised of 49 patients. 32(65.3%) patients were males and 17(34.69%) patients were females. Male to female ratio was 1.88 :1. The mean age in years of the patients was 34.66 $\pm$ 8.31 (mean $\pm$ SD). The average time between consumption of Cleistanthus colinus and presentation to the hospital was 5.41 $\pm$ 2.16 hrs. Out of 49 patients included in the study 34 (69.38%) patients got admitted to the study centre within 6 hours of consumption of poison, 11 (22.44%) patients

presented to the study centre within 6-12 hours and 4(8.16%) presented beyond 12hrs.

The method of consumption of poison was as follows, 26(53.06%) patients consumed crushed leaves and its juice, 16 (32.6%) patients boiled extract/decoction, 4(8.16%) patients consumed raw leaves and 3(6.12%) patients consumed seeds and leaves. 38(77.5%) patients received treatment in a primary or secondary care centre before presenting to the study centre. All these 38 (100%) patients received gastric lavage at the centre where they initially presented for treatment. 14(28.5%) patients received IV fluids from the peripheral centres from where they got first line management. On presentation at the study centre 49 (100%) patients received gastric lavage. The commonest symptom at presentation was vomiting in 43(87.75 %) patients. Other symptoms included nausea in 19(38.77%), giddiness in 13 (26.5%) patients, dyspnoea in 8(16.33%) patients. Tachycardia was present in 19 (38.77%) patients, tachypnoea in 8(16.3%) patients, hypoxia in 6(12.24%) patients and hypotension in 4(8.16%) patients..

The laboratory parameters at presentation included serum sodium levels 136.32 $\pm$ 2.41 and potassium levels 3.11 $\pm$ 0.43 (mean $\pm$ SD) mmol/L. 39(79.59%) patients had hypokalemia. 33(67.34%) had hypokalemia on admission and 6(12.24%) developed during hospitalization. Mean duration taken to correct hypokalemia was 3.9 $\pm$ 1.6 (mean $\pm$ SD) days. 33 of these 39 patients survived. All patients were given intravenous potassium replacements. Hyponatremia was present in 16 (32.6%) of patients. Random blood sugar was 101.24 $\pm$ 38.64 (mean $\pm$ SD) mg/dL. The creatinine levels were 1.16 $\pm$ 0.29 (mean $\pm$ SD) mg/dL. 7

patients had acute renal failure. 28(57.14%) patients had metabolic acidosis at the time of admission. The bicarbonate values were 19.31 ± 2.7 (mean $\pm$ SD) mmol/L. Neutrophilic leucocytosis was recorded in 18(36.73%) of patients.

The demographic characteristics, laboratory parameters and symptoms at presentation are given in Table 1.

Table 1:

Demographic, symptoms and laboratory parameters in patients with C.colinus poisoning.(n=49)		
Characteristics	Number(n)	Value
Demographic Age, mean (SD) years	49	34.66(8.31)
Gender ratio (Male: Female)	49	1.88:1
Method of consumption		
Crushed leaves & juice	49	26(53.06%)
Boiled extract/decoction	49	16(32.6%)
Raw leaves	49	4 (8.16%)
Leaves & seeds	49	3(6.12%)
Symptoms		
Nausea	49	19(38.77%)
Vomiting	49	43(87.75%)
Giddiness	49	13(26.5%)
Dyspnoea	49	8(16.33%)
Laboratory Parameters		
Sodium, mean(SD) mmol/L	49	136.32 ± 2.41
Potassium mean (SD) mmol/L	49	3.11 ± 0.43
Random Blood Sugar mean(SD) mg/dL	49	101.24 ± 38.64
Creatinine mean(SD) mg/dL	49	1.16 ± 0.29
Bicarbonate mean(SD) mmol/L	49	19.31 ± 2.7
Neutrophilic leucocytosis	49	18(36.73%)

Discussion

Cleistanthus collinus is a common plant poison used for deliberate self harm in South India. Published studies are relatively few about C.collinus poisoning. In the present study more male patients

The overall mortality in our study was 7(14.28%). All these patients had hypoxia and Type 1 respiratory failure. They also had refractory hypotension, severe metabolic acidosis and acute kidney injury. 3 patients were intubated and given mechanical ventilation.

were present when compared to females and male to female ratio was 1.88 :1. While in some other studies a female preponderance was observed. The mean age in years of the patients was 32.66 ± 8.31

(mean $\pm$ SD), indicating a relatively younger age group of patients.

Hypokalemia(79.59%) was the most common feature in our patients. It may be due to renal tubular dysfunction. Vomiting also might have contributed to it. Metabolic acidosis was noted in 28(57.14%) of patients. Previous study from Vellore recorded metabolic acidosis in 100% of patients while another study from Puduchery noted metabolic acidosis in 21.7% of patients. Renal tubular dysfunction might contribute to hypokalemia and metabolic acidosis. Metabolic acidosis and hypokalemia which is commonly found in such patients point to the fact that renal injury is a significant component of *C. collinus* toxicity. Toxin mediated inhibition of the vacuolar H^+ ATPase activity in the renal brush border membrane appears to be the underlying mechanism of injury.^{4,5} Animal models have demonstrated that the toxins cause depletion or inhibition of thiol or thiol dependent enzymes leading to loss of ATPase activity.⁶ Distal tubular cells are commonly affected. Involvement of proximal tubular cells or global tubular dysfunction with reduced glomerular filtration rate (GFR) can occur in more severe forms.

Cardio toxicity has been described as a feature of *C. collinus* poisoning.⁸ No cardiac arrhythmias were recorded in our patients as was seen in some other studies. Hypokalemia, direct effect of toxin on myocardium and also cardio depressant effect of the plant toxin may contribute to development of arrhythmia. Study done in Vellore has described the use of temporary pacing in all such patients even in the absence of arrhythmias. But the beneficial effects of such an intervention in all patients is unknown.

Further studies with more selective criteria like bradyarrhythmia, Heart block or QT prolongation for use of temporary pacing is warranted.

The overall mortality in our study was 7(14.28%). All these patients had Type 1 respiratory failure, Acute Kidney injury, metabolic acidosis and persistent hypokalemia despite treatment and refractory hypotension. This also reflects the fact that *C. collinus* poisoning can affect multiple organs leading to multi organ failure and death.

Hypokalemia and metabolic acidosis are the most significant derangements that occur in patients with *C. collinus* poisoning, most likely due to tubular dysfunction. Serum potassium under-estimates the total potassium deficits especially in presence of metabolic acidosis. This reinforces the fact that pre-emptive supplementation of potassium even with low normal level is warranted in such patients. In our study this was practiced. Early replacement of potassium with frequent monitoring should be done in all patients. Metabolic acidosis should be aggressively managed with fluid correction and bicarbonate supplementation.

Limitations of our study include , the inherent limitations of retrospective observational studies and the complete panel of renal dysfunction was not done in any of the patients. Instead indirect renal tubular dysfunction indicators like metabolic acidosis, hypokalemia, low serum bicarbonate etc were used.

Conclusion

Cleistanthus collinus poisoning is common form of plant poisoning seen in South India. The toxin predominantly affects distal renal tubules leading to distal Renal Tubular Acidosis(dRTA) . Severe forms

can affect proximal tubules also and lead to global tubular dysfunction and decreased GFR. Metabolic acidosis and hypokalemia has to be aggressively managed in these patients. Mortality occurs due to Type 1 respiratory failure, refractory shock, hypokalemia, metabolic acidosis and renal failure indicating a multisystem involvement.

References

1. Srivastava A, Peshin SS, Kaleekal T, Gupta SK. An epidemiological study of poisoning cases reported to the National Poisons Information Centre, All India Institute of Medical Sciences, New Delhi. *Hum Exp Toxicol*. 2005;24(6):279–285. doi: 10.1191/0960327105ht527oa. DOI: [DOI] [PubMed] [Google Scholar]
2. Shankar V, Jose VM, Bangdiwala SI, Thomas K. Epidemiology of *Cleistanthus collinus* (oduvan) poisoning: clinical features and risk factors for mortality. *Int J Inj Contr Saf Promot*. 2009;16(4):223–230. doi: 10.1080/17457300903307094. DOI: [DOI] [PubMed] [Google Scholar]
3. Arun Raja V, Mohan A, Harikrishna J, Sarma KVS. Prospective study of predictors of outcome in 159 patients with acute poisoning admitted in medical intensive care unit. *J Assoc Physicians India*. 2015;63:88. [Google Scholar]
4. Sarathchandra G, Balakrishnamurthy P. Perturbations in glutathione and adenosine triphosphatase in acute oral toxicosis of *Cleistanthus collinus*: an indigenous toxic plant. *Indian J Pharmacol*. 1997;29:82–85. [Google Scholar]
5. Asolkar LV, Kakkar KK, Chakre OJ, editors. Second Supplement to Glossary of Indian Medicinal Plants with Active Principles Part-1(A-K) (1965-1981). New Delhi: National Institute of Science Communication, Council for Scientific and Industrial Research; 2000. p. 214. [Google Scholar]
6. Viswanathan N, Joshi BS. Toxic constituents of some Indian plants. *Curr Sci*. 1983;52(1):1–8. [Google Scholar]
7. Aleem MA, Paramasivam M. Spectrum of acute poisoning in villagers. *J Assoc Physicians India*. 1993;43:859. [Google Scholar]
8. Ramesh C, Ravindranath N, Ram TS, Das B. Arylnaphthalide lignans from *Cleistanthus collinus*. *Chem Pharm Bull*. 2003;51(11):1299–1300. doi: 10.1248/cpb.51.1299. DOI: [DOI] [PubMed] [Google Scholar]
9. Parasuraman S, Raveendran R, Madhavrao C. GC-MS analysis of leaf extracts of *Cleistanthus collinus* Roxb. (Euphorbiaceae). *Int J Ph Sci*. 2009;1:284–286. [Google Scholar]
10. Pradheepkumar CP, Panneerselvam N, Shanmugam G. *Cleistanthin A* causes DNA strand breaks and induces apoptosis in cultured cells. *Mutat Res*. 2000;464:185–193. doi: 10.1016/S1383-5718(99)00179-5. DOI: [DOI] [PubMed] [Google Scholar]
11. Kettimuthu KP, Kini A, Manickam AS, Lourthuraj AA, Venkatraman A, Subramani S, et al. *Cleistanthus collinus* poisoning affects mitochondrial respiration and induces oxidative stress in the rat kidney. *Toxicol Mech Methods*. 2019;29:561–568. doi: 10.1080/15376516.2019.1624905. DOI: [DOI] [PubMed] [Google Scholar]

12. Kumar M, Ramaswamy S, Jayanthi M, Raveendran R. Alphaadrenergic receptor blocking effect of *Cleistanthus collinus* (Roxb.) Benth. and Hook F. Leaf extract on guinea pig isolated smooth muscle preparations. *Indian J Exp Biol*. 2011;49:339–342. [PubMed] [Google Scholar]
13. Parasuraman S, Raveendran R. The effects of aqueous extract of *Cleistanthus collinus* (Roxb.) (Euphorbiaceae) leaves on rat blood pressure. *Pharmacognosy Res*. 2012;4(3):178–180. doi: 10.4103/0974-8490.99086. DOI: [DOI] [PMC free article] [PubMed] [Google Scholar]
14. Jose VM, Anand KN, Jeyaseelan L, Ernest K, Kuruvilla A. Effect of potassium channel modulators on toxicity of *Cleistanthus collinus*. *Indian J Exp Biol*. 2004;42(1):81–85. [PubMed] [Google Scholar]
15. Nandakumar NV, Vijayalakshmi KM. Experimental myasthenic crisis like neuromuscular impairment with *Cleistanthus collinus* leaf extract administration in rat. *Physiother Res*. 1996;10:121–126. [Google Scholar]
16. Damodaram P, Manohar IC, Kumar DP, Mohan A, Vengamma B, Rao MH. Myasthenic crisis-like syndrome due to *Cleistanthus collinus* poisoning. *Indian J Med Sci*. 2008;62(2):62–64. doi: 10.4103/0019-5359.39368. DOI: [DOI] [PubMed] [Google Scholar]
17. Mohan A, Naik GS, Harikrishna J, Kumar DP, Rao MH, Sarma K, et al. *Cleistanthus collinus* poisoning: experience at a medical intensive care unit in a tertiary care hospital in south India. *Indian J Med Res*. 2016;143:793–797. doi: 10.4103/0971-5916.192068. DOI: [DOI] [PMC free article] [PubMed] [Google Scholar]
18. Subrahmanyam DK, Mooney T, Raveendran R, Zachariah B. A clinical and laboratory profile of *Cleistanthus collinus* poisoning. *J Assoc Physicians India*. 2003;51:1052–1054. [PubMed] [Google Scholar]
19. Devaprabhu S, Manikumar S, David SS. Toxicology and prognostic profile of patients with *Cleistanthus collinus* poisoning. *Indian J Trauma Anaesth Crit Care*. 2007;8:642–646. [Google Scholar]
20. Bammigatti C, Suryanarayana BS, Harichandra Kumar KT, Ganesh Kumar S. Pattern and outcome of *Cleistanthus collinus* (Oduvanthalai) poisoning in a tertiary care teaching hospital in South India. *J Forensic Leg Med*. 2013;20:959–961. doi: 10.1016/j.jflm.2013.08.011. DOI: [DOI] [PubMed] [Google Scholar]
21. Benson BE, Hoppu K, Troutman WG, Bedry R, Erdman A, Höjer J, et al. American Academy of Clinical Toxicology; European Association of Poisons Centres and Clinical Toxicologists. Position paper update: gastric lavage for gastrointestinal decontamination. *Clin Toxicol*. 2013;51:140–146. doi: 10.3109/15563650.2013.770154. DOI: [DOI] [PubMed] [Google Scholar]
22. Raja G, Kumaran SS, Chandrasekaran VP. Outcome of *Cleistanthus collinus* poisoning with and without charcoal. *Acad Emerg Med*. 2007;14(5):e111. doi: 10.1197/j.aem.2007.02.010. DOI: [DOI] [Google Scholar]