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ORIGINAL ARTICLE

A STUDY OF PROFILE IN PATIENTS WITH *CLEISTANTUS COLLINUS* IN A TERTIARY HOSPITAL

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ABSTRACT

Cleistanthus collinus belongs to family Phyllanthaceae, grows wild in dry hills of various parts of India from Himachal Pradesh to Bihar, and southwards into Peninsular India. The present study shows *Cleistanthus collinus* Concludes that In this study, various clinical and laboratory profile of cleistantuscollinus poisoning in patients presenting to a tertiary care hospital is studied.

Keywords: *Catla Cleistanthus collinus,* Tertiary Hospital

1.INTRODUCTION

Cleistanthus collinus belongs to family Phyllanthaceae, grows wild in dry hills of various parts of India from Himachal Pradesh to Bihar, and southwards into Peninsular India. Commonly called as Oduvanthalai in Tamil Nadu.[1] All parts of the plant are poisonous. Extract of the variousplant parts yielded a multitude of compounds of which the glycosides, arylnaphthalene lignan lactones are toxic.[2-7]These lignan lactones include cleistanthin A and B, collinusinand diphyllin, which in the past were known collectively as oduvin.[8] In this study, various clinical and laboratory profile of cleistantuscollinus poisoning in patients presenting to a tertiary care hospital is studied.

2.STUDY DESIGN

Forty-six patients admitted in Government medical college, Tiruvannamalai in department of medicine between April 2017 to October 2017with history of consumption of *Cleistanthus collinus* plant parts were studied, prospectively. History of time of consumption to presentation in casualty and symptoms was noted. ECG, urea creatinine and ABG was done daily at time of admission till day of recovery or death.

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3.RESULTS

The age of the patients ranged from 13-65 years, the majority (37/46 cases) being below 30yrs (80%) with 25 cases in their 3rd decade (54%). Fifteen patients expired. The overall female to male ratio was 28:18, while 11:4 in the expired group. The poison was ingested in one of the following ways swallowing the hand crushed plant parts especially the leaves, chewing the leaves and swallowing them, making a decoction by boiling the leaves in water and drinking it and making a paste of the plant parts and leaves and swallowing it.

Fifteen of the 46 cases studied died resulting in 32.6% mortality. Clinical features in those who died included vomiting, pain in abdomen especially epigastric,

breathlesness, visual disturbances like clouding/blurring/ coloured vision, giddiness, drowsiness, fever, tachycardia and terminally hypotension and or respiratory arrest. Survivors were asymptomatic or transiently symptomatic with abdominal pain, visual symptoms and giddiness.

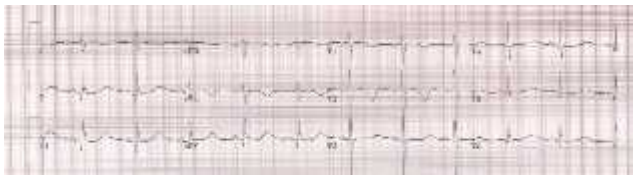
SYMPTOMS	***EXPIRED***	***SURVIVOR***
Persistent epigastric	13(87)	9(29)
Dyspnea after 24 hrs.	15 (100)	2 (7)
Eye symptoms after 24 hrs.	14 (93)	8 (26)
Giddiness after 24 hrs.	15 (100)	2 (7)
Pulse > 120 / min	12 (80) 2 (7)	2 (7)
Fever > 39 deg C	5(33) 2(7)	2 (7)
Pulse < 60/min	4 (27)	0
Hypotension SBP < 90 mm Hg	8 (53)	1 (3)
Respiratory rate > 30/min	15 (100)	3 (10)

NO. PARAMETERS	***EXPIRED***	***SURVIVOR***
1.ST depression	12 (80)	2 (7)
2.Prolonged QTc	4 (27)	4 (13)
3.Potassium < 3.1mEq/lit	6 (40)	2 (7)
4.Sodium ion <130mEq/lit	4(27)	1 (3)
5. Elevated total Biliriubin	3 (20)	0
6.Elevated AST	13 (87)	25 (93)
7. Elevated ALT	2 (14)	2 (7)
8. Elevated AlkP	1 (7)	2 (7)
9. Mean pH	7.28 ± 0.11	7.37 ± 0.05
10.Mean PaO2	86.61± 54	114.35 ± 18
11. Mean PaCO2	27.24 ± 8.36	31.47 ± 5.23
12.Mean HCO3	13.64 ± 5.76	21.67

4.DISCUSSION

Cases of suicide accomplished with parts of the Oduvan plant have been on the increase in recent times, in several parts of India, especially Tamil Nadu and Andhra Pradesh. The age, gender, mode of ingestion and mortality trends were similar to those observed in the previous studies. The number of cases (22%) peaked in the month of September. The plant part ingested was the leaf and when taken in decoction form was more fatal as observed in previous studies.[9-11] In addition, in this study, the ingestion of more than two handfuls (> 60) of leaves in any form was found to be harmful. Most of the clinical features described were also noticed in the previous studies except for the visual symptoms and giddiness.

ECG changes were mainly in the form of non-specific ST-T changes. QTc prolongation was not a prominent feature. These findings were in contrast to the study by Kurien et al,[9] where QTc prolongation was a prominent feature but similar to the observations made by Das et al.[11] The ST-T changes were significantly more in those who died.



A patient who had QTc prolongation in ECG

Hypokalaemia was observed at admission in both the survivors and those who died but in later it was more severe. A cut off value of 3.1mmol/l could be prognostic as well as measure of the severity of poisoning. Hypokalaemia per se could not be the cause of death as was suggested by Kurienet al[11] since it was corrected in nine of the 15 patients who died. Hyponatraemia of < 130 mmol/l though seen significantly more in the mortality group was less common, a finding also observed by Kurien et al. The enzymes AST, LDH, CPK, CPK-MB were elevated in both the survivors and those who died and those who died had significantly higher CPK-MB levels as compared to those who survived (80 ± 43 IU/L vs 58 ± 21 IU/L,'p' value 0.039) suggesting a possible cardiotoxic effect as also observed by Kurien et al.[11] The ALT and alkaline phosphatase levels were normal indicating absence of any liver injury. In animal studies a decrease in the activity of these enzymes has been observed.[12,13] ABG showed a picture of metabolic acidosis and, additionally in those with respiratory failure, hypoxia with widened A-aO2 gradient. This could indicate that it might also be causing lung injury. Pulmonary toxicity as a cause of mortality has also been documented by Das et al but not by Kurien et al who found that death was due to its cardiotoxicity.

5.CONCLUSION

Though oduvanthalai poisoning is very common and mortality is on the higher side studies have not been undertaken and no definite treatment protocols are being published or adopted. Nacetyl cysteine is being tried in many centers with loading dose of 150 mg/kg over 1 hour, followed by 50mg/kg over 4 hours, and 100mg/kg over the next 16 hours. As more and more studies like ours are directed towards clinical features, more studies are required to strategize a treatment protocol.

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