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Plant poisoning – Cleistanthus collinus

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C collinus (Oduku, Oduvan) belongs to family Euphorbiaceae and is a small tree with elliptical leaves, greenish yellow flowers in clusters, and gooseberry like fruits. It is grown in various parts of India, SriLanka, Malaysia and Africa. Self-poisoning reports are documented mainly from south India and young females from rural area use this as suicidal agent due to easy access. Bark, leaves, fruits, and roots of the plant are poisonous. Cleistanthin A and B are the major toxins responsible for its toxicity; Cleistanthin C, D, Collinusin, Cleistanone are the other toxins identified. It causes neuromuscular blockade, arrest of cell cycle progression, increased apoptosis, Type 1 renal tubular acidosis, cardiac arrythmias, alpha blockade, and refractory hypotension in animal studies. Toxicity is mainly by oxidative stress induced by inhibition of thiol/thiol dependent enzymes. In renal brush border membrane of rat, C. collinus showed uncoupling of oxidative phosphorylation and increased ADP levels, which in turn inhibit the V-ATPase in the BBM of distal renal tubule. This is the likely mechanism causing distal RTA.

In a five year hospital based study of self-poisoning conducted in our institution, it was shown that 19.38% of self-poisoning and 9.1% of poisoning death was constituted by plant poisoning. Among plant poisons, most common was C collinus (8.9%) with case fatality of 1.5%. In a 20-year autopsy study in the same institution, 0.03% of poisoning death was identified as plant poisoning and commonest identified plant toxin was Cleistanthin (Oduvin) glycoside.

Common clinical features on self-poisoning are nausea, vomiting, abdominal pain, diarrhoea neurological weakness, dilated pupils, altered consciousness and dyspnoea. Patients can develop ARDS, renal failure, and

seizures. Hypokalemia, metabolic acidosis (type 1 RTA), ECG changes (tachycardia, ST depression, T inversion, QT prolongation), and leukocytosis are the common laboratory abnormalities with poisoning. Management includes risk assessment, symptomatic management, identifying poor prognostic signs, correction of hypokalemia, and ventilatory support. Amount of poison, altered sensorium, hypokalemia, metabolic acidosis, leukocytosis, QTc prolongation in ECG were found to be associated with poor outcome in our series.