Hypocalcemia in *Cleistanthus collinus* Poisoning

The leaves of *Cleistanthus collinus* (CC) known as *oduvanthazhai* in Tamil language is a commonly consumed plant poison in rural areas, usually with a suicidal intention. Distal renal tubular acidosis is known to occur with CC poisoning and hypokalemia is a risk factor for mortality [1,2]. However, hypocalcemia in children with CC poisoning has not been documented.

Two girls (12 years and 8 years of age) and their brother (9 years) were brought to our hospital with alleged history of consumption of 100-200 mL of boiled leaf extract of CC. Their mother had given them the poison before consuming herself due to a family dispute. They developed vomiting, drowsiness and breathing difficulty after an hour. Gastric lavage was done in a nearby hospital after 4 hours of consumption and brought to JIPMER after 48 hours of ingestion. The two girls had polyuria, altered sensorium and shock while the boy was hemodynamically stable. The eldest sibling had sinus bradycardia and prolonged QTc. Blood investigations revealed severe metabolic acidosis, hypokalemia, and hypocalcemia. However, the levels of urea, creatinine and albumin were normal. Despite, mechanical ventilation and supportive care including potassium replacement and inotropic support, the two girls had cardiac arrest and died on the third and fourth day of hospitalization.

The intake of boiled leaf extract preparation (increased concentration of plant glycosides) and the delay in gastric lavage probably contributed to the death of the 2 girls [3]. The boy probably survived owing to decreased amount ingested. Apart from metabolic acidosis and hypokalemia, all three children had persistent hypocalcemia. Hypercalciuria associated with distal renal tubular acidosis probably contributed to the hypocalcemia. However, urinary calcium excretion could not be documented. In addition to metabolic acidosis, hypocalcemia perhaps contributed to myocardial dysfunction and mortality. In a child with CC poisoning, it is prudent to watch for hypocalcemia and correct the same, if present.

C VENKATESH AND B ADHISIVAM
Department of Pediatrics,
Jawaharlal Institute of Post graduate Medical Education and Research,
Pondicherry 605 006, India.

REFERENCES

Iron and Zinc Deficiency in Children

The article on ‘Effect of Iron and Zinc deficiency on short term memory in children’ [1] is an eye opener, as micronutrient malnutrition and its ill effects are rampant in our country. Iron deficiency anemia is the most common nutritional disorder, even in the current era. Iron is essential for oxygen carrying, muscle functions, immune function and brain myelination, neurotransmission and cognitive functions [2]. Even mild to moderate anemia in infancy and early childhood are known to leave a permanent signature on the growing brain. Iron has effects on the neurotransmitters like dopamine and probably serotonin [2]. Iron deficiency reduces dopaminergic receptors and the reduction in dopaminergic receptors leads to increase in opiate receptors and resultant defective learning ability and cognition. The role of iron deficiency on aggravating breath holding spell, febrile seizure, and hypercyanotic blue spell are also being increasingly observed in clinical practice. Similarly, zinc is essential for enzyme function, metabolism, immune function, taste sensation, reproduction, cognition and retinal function [3]. The findings of the above study prove robust evidence in this context.

However, data regarding how many children had malnutrition, how many had anemia, the distribution according to hemoglobin level, severity of iron and zinc deficiency are lacking in the presentation [1]. Serum