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Letters to the Editor

An unusual case of cardiac glycoside toxicity

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Cardiac glycosides are naturally occurring toxins that reversibly inhibit the sodium–potassium adenosine triphosphatase (Na–K–ATPase) exchanger in myocardial cells. They are found in various plant species, such as foxglove (*Digitalis purpurea*, *Digitalis lanata*), ouabain (*Strophanthus gratus*), common oleander (*Nerium oleander*), yellow oleander (*Thevetia peruviana*), and sea mango (*Cerbera manghas*) [1]. Self-poisoning with the seeds or fruits of these plants is a major clinical problem in parts of the developing world causing a significant number of deaths each year [2]. In particular though, the seeds from the *Cerbera odollam* tree (also known as the "Suicide tree" or "Pong-pong tree") are excessively toxic, containing cerberin as the main active cardiac glycoside, and often used for suicide or homicide in certain rural areas of South Asia [3]. In countries far from the natural habitat of these extremely toxic plants, poisonings are relatively unknown to Western physicians and rarely reported. We present an unusual case of acute cardiac glycoside toxicity due to suicidal ingestion of "Pong-pong" seeds purchased from an online website.

A 51-year-old woman with a history of depression and prior suicide attempts with various drug overdoses presented to the Emergency Department complaining of nausea, vomiting, diarrhea, and chest tightness since awakening that morning. On physical examination, she was lethargic and profoundly bradycardic with an irregular pulse. Her heart rate was 30 beats per minute (bpm) and blood pressure 90/60 mm Hg. An electrocardiogram (ECG) demonstrated atrial flutter(AFl) with variable atrioventricular (AV) block and slow ventricular response, diffuse ST-segment depressions, shortened QT interval, and peaked T-waves (Fig. 1A). Laboratory studies were significant for a serum potassium level of 7.5 mmol/L (normal: 3.5–5.1), calcium of 10.9 mg/dL (normal 8.6–10.2), and creatinine of 2.6 mg/dL (normal: 0.7–1.2). Cardiac enzymes were mildly elevated with a troponin T level of 0.07 ng/mL (normal: <0.03). Comprehensive serum and urine toxicology screens were unremarkable. A digoxin concentration level was undetectable (<0.3 ng/mL).

The patient was administered atropine for her symptomatic bradycardia, which improved with a heart rate of 91 bpm. Calcium gluconate, sodium bicarbonate, glucose, and insulin therapy were given for severe hyperkalemia in the setting of an acute kidney injury. A repeat ECG showed continued AFl with variable AV block, shortened QT interval, and deeper, downsloping ST-segment depressions (Fig. 1B). A repeat serum potassium level was 5.5 mmol/L, correlating with resolution of the peaked T-waves on ECG. A repeat serum calcium level was 10.3 mg/dL, though the QT interval remained shortened on ECG. She was transferred to the Intensive Care Unit (ICU) for continued supportive therapy.

Upon further questioning in the ICU, the patient admitted to the intentional ingestion of an unknown quantity of seeds obtained from a "Pong-pong" tree she purchased several weeks prior from an online website. The Poison Control Center was immediately called, and the patient subsequently received 10 vials of empiric digoxin immune Fab. The following ECG revealed a return to normal sinus rhythm, continued QT interval shortening, and persistent ST-segment depressions with biphasic T waves, though improved compared to the prior ECG with resolution of dysrhythmias (Fig. 1C). Further evaluation with an echocardiogram and cardiac catheterization was normal. She was administered another 10 vials of digoxin immune Fab with ultimate normalization of the QT interval and resolution of ST-segment depressions within 24 h (Fig. 1D). The patient recovered uneventfully and later discharged in stable condition.

To the best of our knowledge, this is the first reported case in the medical literature of *Cerbera odollam* poisoning in the United States. Although common in certain parts of South Asia, non-pharmacologic cardiac glycoside toxicity is rare in the United States. Of more than 2.3 million human exposures reported in the 2011 National Poison Data System, only 569 calls were received for plant cardiac glycosides (excluding drugs) [4].

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The diagnosis and treatment in such cases present a clinical challenge. Different toxins have variable degrees of cross-reactivity with commercially available digoxin assays. While digoxin assays may be positive in certain non-pharmacologic cardiac glycoside poisonings, they do not accurately reflect the degree of toxicity [5,6]. Furthermore, *Cerbera odollam* has not been shown to be detected by digoxin assays. At present, detection is limited to thin-layer chromatography and liquid chromatography coupled with tandem mass spectrometry [3,7].

A 2005 Cochrane Review reported that anti-digoxin Fab antitoxin was effective at reducing cardiac toxicity following yellow oleander poisoning. However, the utility of this data in the setting of exposure

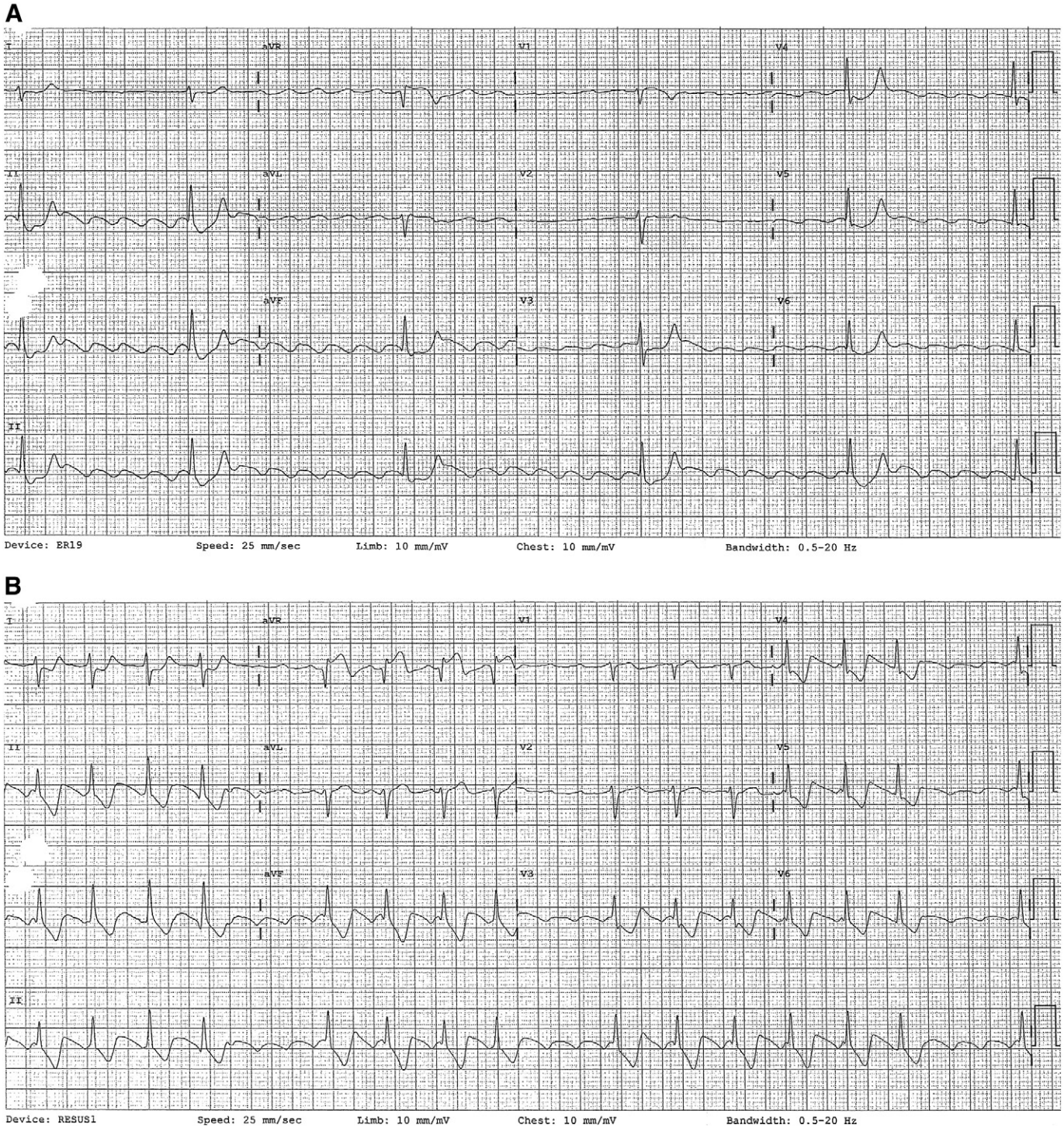

Fig. 1. The initial ECG demonstrated AFl with variable AV block and slow ventricular response, shortened QT interval, diffuse ST-segment depressions, and peaked T-waves (atrial rate of 220 bpm; QTc interval of 360 ms) (A). Repeat ECG following medical therapy revealed continued AFl with variable AV block, shortened QT interval, and deeper, downsloping ST-segment depressions (B). After the administration of digoxin immune Fab, the ECG showed a return to normal sinus rhythm, continued QT interval shortening, and persistent ST-segment depressions with biphasic T waves (C). Repeat administration of digoxin immune Fab showed normalization of ECG abnormalities (QTc of 460 ms) (D).

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