EP CASE REPORT

An unusual case of oleandrin poisoning suggesting its possible antiarrhythmic activity

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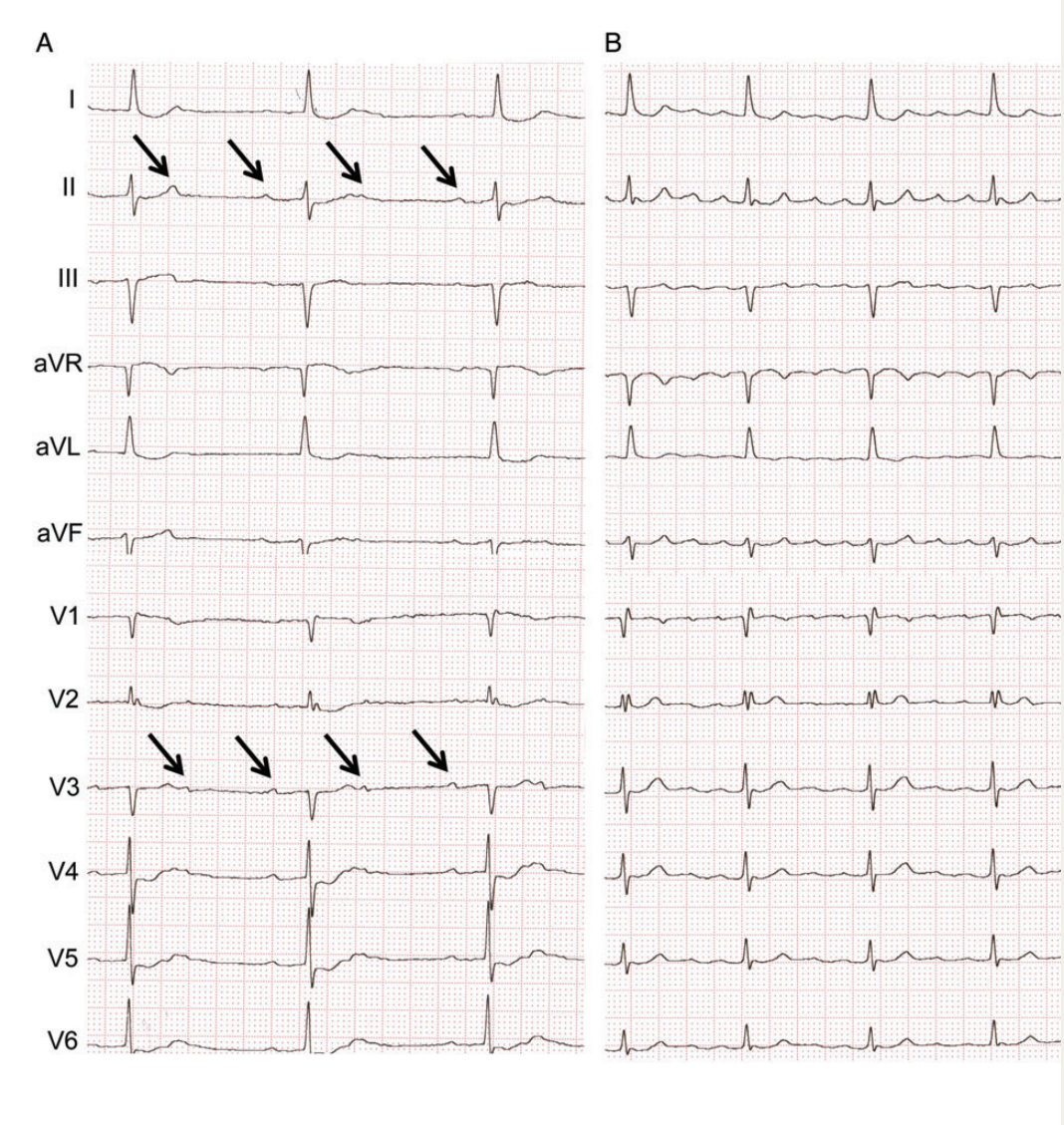
A 77-year-old woman with permanent atrial fibrillation for over 5 years was admitted for oleandrin poisoning after the ingestion of snails. The ECG at admission surprisingly revealed sinus rhythm with 2:1 atrioventricular block. In the following days, with the reduction of oleandrin serum concentration, the ECG showed the transition from sinus rhythm to atrial flutter, and finally to atrial fibrillation.

About 6 h after a meal which included an undefined amount of snails (of the species *Helix aperta*) collected under a plant of Nerium oleander, a 77-year-old hypertensive and hypercholesterolaemic woman with permanent atrial fibrillation (AF) for over 5 years (as confirmed by routine, semiannual ECG assessments), presented with a syndrome characterized by cardiovascular (bradycardia),



gastrointestinal (nausea, vomiting, and diarrhoea), and neurological (aphasia and space-time disorientation) symptoms similar to those observed in digoxin intoxication. A CT brain scan excluded structural brain lesions. The ECG surprisingly showed sinus rhythm (SR) at 96 bpm with 2:1 atrioventricular block (ventricular rate of 48 bpm), and repolarization abnormalities suggestive of digitalis effect (a diffuse sagging of the ST segment with upward concavity) (*Panel A*). The echocardiogram showed a hypertensive cardiopathy with preserved left ventricular systolic function and a moderate-to-severe left atrial dilatation (anteroposterior diameter 46 mm). Laboratory findings including electrolytes, liver enzymes, creatinine, and blood count were normal with the exception of serum digoxin level (quantified by immunoassay method) that was 7.8 ng/mL (therapeutic range: 0.5–2.0 ng/mL), although the patient had never taken digoxin. The patient was promptly hospitalized and the vital parameters continuously monitored. No specific treatment was performed. In the second day the patient showed marked improvement of symptoms. The serum digoxin concentration decreased to 1.2 ng/mL. The ECG showed an atypical atrial flutter with ventricular rate of 68 bpm and partially resolved repolarization abnormalities (*Panel B*). On the third day the patient showed a complete resolution of symptoms, the serum digoxin concentration was undetectable, and the ECG showed AF with ventricular rate of 70 bpm and normal repolarization. The patient was discharged in the seventh day.

Nerium oleander is an ornamental plant that contains high levels of oleandrin, a toxic cardiac-glycoside-like. Oleandrin positioning produces potentially fatal clinical and cardiovascular manifestations, similar to those observed in cardiac glycoside intoxication. Because of its structural similarity, oleandrin cross-reacts with the immunoassays used for quantification of serum concentration of digoxin. The cardiac toxic effects include cardiac arrhythmias (atrial and ventricular ectopic arrhythmias) and excito-conduction disturbances (sinus bradycardia, sinoatrial and atrioventricular node blocks).[1] In literature it is described another case of accidental oleandrin poisoning after the ingestion of snails that had eaten oleander leaves.[2] To our knowledge, this is the first report of oleandrin poisoning associated with cardioversion of a very long-lasting AF. In patients with asymptomatic long-lasting AF, restoration and maintenance of SR offer no clinical benefit compared with rate control. In AF, digoxin slows down atrioventricular conduction but has no effect on the restoration of SR.[3] The oleandrin effects in AF patients have never been described. The possible antiarrhythmic activity of oleandrin that we observed appeared to be dose-dependent, as suggested by the finding that with the progressive reduction of its serum concentration there was a gradual desynchronization of the atrial electrical activity (transition from sinus rhythm to atypical atrial flutter, and finally to AF).

The antiarrhythmic effect of oleandrin may be ascribed to its action on homeostasis of intracellular calcium (via Na^+, K^+ – ATPase inhibition, and dysfunction of sarcolemmal calcium release channels) and to the modulation of subcellular pathways, not reported with classical cardiac glycosides. However, their precise mechanism is unknown, and the findings of this report should stimulate further studies on electrophysiological properties of oleandrin for its potential therapeutic use in AF.

References

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