The candlenut (Aleurites moluccana) is marketed as a natural weight-loss supplement, although relatively little is known about its mechanism of action. It is highly accessible due to its sale on common websites as a raw seed or in capsules. Its purported therapeutic effects include weight loss and cholesterol reduction, as well as management of alopecia, cellulite, and tobacco use disorder.1 Subrato et al2 analyzed the composition of candlenut oil and found a high content of oleic acid, linoleic acid, and linolenic acid, all of which have health benefits. However, Gonzalez-Stuart et al1 advised that no part of the seed be consumed because of bioactive ingredients that are cocarcinogenic and act as strong laxatives.

Candlenut use has resulted in multiple adverse symptoms, including headache, nausea, vomiting, diarrhea, fatigue, cardiac dysrhythmias, and 3 reported deaths.3 One case report4 described a woman whose electrocardiogram (ECG) revealed evidence of bradycardia and a first-degree atrioventricular (AV) heart block after candlenut consumption. These harmful effects have led to its ban in Argentina, Spain, Brazil, and other South American countries.1,3,5 We found no other reported cases of a second-degree, Mobitz type II AV block after candlenut consumption, which we describe here.
Report of Case
A 44-year-old woman with no significant prior medical history and no currently-prescribed medications presented to the emergency department (ED) with altered mental status; she was only responsive to sternal rub. Minutes after presentation, she was hyperventilating and responsive enough to report numbness in her face, tongue, and arms. Over the next several hours, her mental status returned to baseline. She reported having recently lost 46 pounds after being on a strict, self-monitored weight loss regimen for 2 months; the patient was consuming only lettuce, meat, and water. She discussed her weight loss with her family doctor, who suggested she take an over-the-counter Brazilian candle-nut weight loss supplement. She began to take “1 small piece” per day, 2 days prior to ED arrival. She immediately began experiencing abdominal pain, progressing to diarrhea on the second day. On the day of her presentation to the ED, she reported vomiting, generalized weakness, palpitations, chest discomfort, and shortness of breath. An ECG showed a second-degree AV heart block with characteristic shortened QT interval with ST-T changes consistent with digitalis effect (Figure 1). The patient’s vital signs and initial electrolytes, renal function, serum troponin, urine drug screen, and complete blood count were all unremarkable. No maneuvers (such as carotid massage) or pharmacologic interventions were made to correct the bradycardia.

The patient was admitted to the hospital for monitoring. During her admission, she received intravenous fluids and pantoprazole, oral sucralfate, and a clear liquid diet. The following morning, a repeat ECG was performed, which continued to show AV block with increased sinus rate (Figure 2). The AV block resolved on hospital day (HD) 2. There were no reported AV block variations and the abnormal rhythm corrected to normal sinus rhythm without any intervening rhythm disturbances. This finding was confirmed on telemetry, and the patient’s rhythm strip before discharge is shown (Figure 3). A digoxin concentration on HD 2 measured 0.3 ng/mL. This concentration was not repeated. The patient was discharged in good health on HD 2 and asked not to consume candlenut.

Discussion
In most reported cases, previously-reported symptoms of candlenut were either general constitutional or...
gastrointestinal in nature. Rarely, cases involving cardiac abnormalities have been reported, including descriptions of bradycardia and first-degree AV block. One previous report from Spain in 2017 described a very similar case of a patient with elevated digoxin concentration after candlenut ingestion.

AV heart block occurs when there is a disruption in normal impulse conduction through the heart. Normal electrical conduction will begin at the sinoatrial (SA) node, disseminate throughout the atria, collect at the AV node, travel down to the bundle of His and the bundle branches, then propagate along the Purkinje fibers in the ventricles. Conduction failure can occur both at the AV node and infranodally. However, most Mobitz type II blocks in humans occur from infranodal disruption. AV heart blocks can manifest from ischemia, drug toxicity, hyperkalemia, excessive vagal stimulation, cardiac valvular calcification, myocarditis, or infiltrative cardiomyopathy.

The mechanism by which the candlenut causes toxicity is largely unknown. However, phorbol esters are present in candlenuts, which can cause diarrhea, vomiting, and polyuria. Phorbol esters also have cocarcinogenic effects, mimicking the action of diacylglycerol in the body, which then promotes protein kinase C (PKC) activity. This increase of PKC activity in the presence of another carcinogen aids in promoting a tumor’s growth. PKC activation is also responsible for some of the inflammatory effects of candlenut. The etiology of heart blocks secondary to

Figure 2.
The patient’s electrocardiogram from the day after admission showed continued Mobitz Type II AV heart block with increased sinus rate.

Figure 3.
The patient’s rhythm strip before discharge from the hospital showed a return to normal sinus rhythm with continued ST-T changes consistent with digitalis effect.
candlenut ingestion, however, remains enigmatic. A digoxin concentration measured on HD 2 in our patient was detectable, but the patient denied any exogenous cardioactive steroid exposure. Moreover, there was no suspicion of endogenous digoxin-like immunoreactive substances in this otherwise healthy patient. Plant-based cardioactive steroids can cross-react with digoxin assay, which assists in making a presumptive diagnosis of nonpharmaceutical cardioactive steroid exposure.10-12 It is unknown whether candlenut exposure could, in fact, cause a falsely-elevated serum digoxin concentration. Potential adulterants in unregulated supplements could also cause the described adverse effects and toxicities. In this case, the presence of a detectable digoxin concentration may be indicative of a cardioactive steroid-based adulterant, contaminant, or component of the candlenut.

There are several limitations to a case study based on a patient’s subjective report of what she may have ingested, including but not limited to a lack of reliability for the timing of ingestion, recall of exactly what was ingested, and other exact details of her recent history. There may also be a component of social desirability bias, as the patient may have been taking another medication or supplement that she did not report. Furthermore, we had no previous records of this patient to know what her baseline psychiatric or cardiac history may have been. She also did not bring a sample of the actual product to the hospital and we were unable to obtain it afterward. We do not know how the candlenut product was processed before it was ingested. There was also no expanded or confirmatory drug screening done at the hospital for this patient. Although absolute confirmation of exposure was impossible, the temporal relationship of symptom development abruptly after exposure to candlenut with cessation quickly thereafter, without any other obvious etiology, lend credence to candlenut toxicity. Though we cannot definitively claim causality, there seems to be an association with this patient’s ingestion of candlenut supplement and her presentation.

Conclusion

Although the mechanism of action for this patient’s second-degree, Mobitz type II AV hear block after candlenut ingestion is unclear, this report serves not only as a warning about the risks unregulated supplements, but also as a reminder about the importance of taking a complete patient history, including inquiry about nonprescribed medications. Further study is required to elucidate the cardioactive components of the candlenut.

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Author Contributions

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