central sleep apnea, or the conversion of treated obstructive sleep apnea to central sleep apnea, should be considered."[1,p63] We agree that the combination of opioids and benzodiazepines should be avoided whenever possible. We also agree that nonpharmacological approaches should always be tried in RLS; regrettably, these are rarely effective in patients with refractory disease and many of these relaxation techniques actually worsen RLS. Rotation of nonopioids has not been found to be an effective strategy in clinical practice in most patients with RLS, but there have not been adequate studies of this approach. Random pill counting is considered highly intrusive by most patients, and we do not feel that it adds sufficiently to abuse prevention to recommend it on a routine basis.

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Electrocardiographic Finding in Zika Virus Infection

To the Editor: We read the article by Villamil-Gómez et al[1] with great interest. Villamil-Gómez et al reported 2 interesting cases of Zika virus–infected patients with abnormal electrocardiographic (ECG) findings and mentioned that "there is a lack of literature about cardiovascular manifestations in adult patients with ZIKV infection."[1,p393] In fact, Zika virus infection might cause cardiac involvement and it is a possibly forgotten clinical presentation.[2] Nevertheless, the effect on cardiac rhythm is extremely rare. In the present report from an endemic area in tropical Asia, there is no problem of cardiac arrhythmia among the infected cases.[3] Focusing on the present report by Villamil-Gómez et al, the ECG problem is reversible, which might imply that there might be a reversible cardiac abnormality due to Zika virus infection. The exact underlying pathophysiology of abnormal ECG changes in the reported cases is unknown and might not relate to the Zika virus–induced cellular pathology, which is generally not reversible.

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In Reply—Electrocardiographic Finding in Zika Virus Infection

We thank Yasri and Wiwanitkit for their comments on our work,[1] which focus on the fact that, as we also stated, there is a lack of literature about cardiovascular manifestations in adult patients with Zika virus (ZIKV) infection. To date, even after a significant number of cases have been reported, particularly in the Americas, during recent outbreaks in countries such as Brazil and Colombia, no observational studies have been conducted to establish that the relative frequency of cardiac compromise in patients infected with ZIKV is "extremely low." Probably, silent asymptomatic or most likely oligosymptomatic cases would present electrocardiographic alterations as found in our study, leading to a lack of reports of such compromise. Moreover, we do not know yet if there would be long-term cardiovascular consequences associated with

ZIKV infection, but it certainly deserves further detailed clinical research. Reports such as those from Brazil by Schwartzmann et al[2] and Cavalcanti et al[3] as well as ours from Colombia[1] suggest the potential relevance of cardiac compromise, which deserves more analyses. These reports reinforce the possibility of frequent subclinical cardiovascular involvement in patients with ZIKV infection, especially in immunocompromised patients.[4]

As we stated, prospective systematic electrocardiographic assessments in patients with ZIKV infection are required.[1] Patients suspected of ZIKV infection would benefit from cardiovascular assessment, and electrocardiography should be routinely performed, especially in pregnant women. Serum cardiac enzymes and markers, as well as echocardiographic studies, should also be considered in these patients. In vivo studies with endomyocardial biopsies would be interesting to demonstrate ZIKV. These procedures are invasive and difficult to perform, but more sensitive and specific. In addition, necropsies analyzing tissues from heart are useful to describe if changes could also be part of a systemic inflammation response,[4] or due to a direct virus aggression with or without a local inflammatory cellular and/or humoral response. As has been demonstrated in other studies and tissues, organs, and fluids, it is possible that there would be a long viral persistence in myocardium and other heart tissues.[5] In addition, in patients with cardiovascular disease, it is not clear how ZIKV infection would make decompensation of "controlled" conditions, as reported in other arboviral diseases such as dengue and chikungunya, especially during the acute phase, but may be also after that. Nevertheless, in general, there is not yet a published study showing that cardiovascular and electrocardiographic alterations are not reverted after acute ZIKV infection.

Initially, clinical suspicion is of utmost importance, supported by cardiovascular tests, to potentially confirm and detect alterations, which is recently being reported increasingly in the scientific literature and should be considered and studied further.

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An Outbreak of Brodifacoum Coagulopathy Due to Synthetic Marijuana in Central Illinois

To the Editor: Brodifacoum (BDF) is an anticoagulant found in rodent bait and is commonly used as a household rodenticide. It is known as a superwarfarin, with up to 100 times the potency of warfarin and a half-life of at least 16 days compared with 40 hours in warfarin.[1] It has been used in various hazardous situations including suicide, homicide, accidental and intentional ingestions, and more recently, in the lacing of recreational drugs. As of April 27, 2018, the Illinois Department of Public Health (IDPH) has received reports of 156 cases, including 4 deaths, due to BDF coagulopathy linked to K2.[2] We report a case of a 38-year-old white man with hematuria and hemoptysis due to a BDF coagulopathy from synthetic K2 marijuana use.

CASE REPORT

A 38-year-old white man presented to the hospital after coughing up blood and noticing dark urine for 2 days. He denied a history of anticoagulation use, bleeding disorders, and exposure to rat poisoning. He did, however, admit to smoking K2 synthetic marijuana 2 weeks previously. He reported smoking K2 regularly for at least 3 to 4 years, but these symptoms had never occurred in the past. Although his hemoglobin was normal at 15 g/dL, he had an international normalized ratio greater than 10, a prothrombin time greater than 150 seconds, and a partial thromboplastin time of 54 seconds. An anticoagulant poisoning panel was positive for BDF in his blood.

The patient's case was reported to the IDPH and was discussed with poison control. The patient was initially given intravenous vitamin K and was transitioned to oral vitamin K (phytonadione) the following day. Because of the long half-life of BDF, poison control recommended that the patient be discharged on phytonadione twice a day for at least 1 month.