Could you say that was an atrial flutter or not?

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Muscle-tremor artefact is a potential cause of misdiagnosis of atrial arrhythmias on electrocardiography (ECG) monitoring. Such errors may lead to inappropriate and potentially dangerous therapies in some patients. We present a case of a patient with uncontrolled seizures whose bedside electrocardiogram monitor analysis appeared to demonstrate atrial flutter with 4:1 conduction through the AV node. The ECG monitor and ECG rhythm strip additionally showed a regular ventricular rate of 94 bpm with an underlying regular ‘saw-tooth’ baseline. We applied cardioversion to convert to sinus rhythm. Amiodarone was loaded and added to the patients therapy who had atrial flutter after cardioversion. Echocardiogram was performed by a pediatric cardiologist and they noted that the atrial rate and ventricular rate were equal. After this, we began to suspect this situation might be a pseudo-flutter due to his muscle contractions. We applied rocuronium to the patient to understand whether this was a pseudo-flutter or not. We saw that the ECG returned to normal sinus rhythm.

Physicians especially working in intensive care units should be aware of artifact to avoid unnecessary therapeutic procedures. As Hippocrates said centuries ago “First, do no harm.”

Key words: atrial flutter, electrocardiogram, cardioversion.

Electrocardiographic (ECG) artefacts may closely imitate both supraventricular and ventricular tachycardias.¹⁻⁵ Muscle-tremor artefact is a potential cause of misdiagnosis of atrial arrhythmias on ECG monitoring.⁶ Such errors may lead to inappropriate and potentially dangerous therapies in some patients.⁷ This is particularly the case if a single lead, especially a limb lead, is relied on for making a diagnosis of the underlying rhythm. We present a case of a patient with uncontrolled seizures whose bedside electrocardiogram monitor analysis appeared to demonstrate atrial flutter with 4:1 conduction through the AV node.

Case Report

A 9-month-old boy with a history of motor delay was admitted to the intensive care unit because of uncontrolled seizures. Despite the use of five anti-epileptic drugs, his convulsions could not be stopped. His medications on admission included fenitoin (5 mg/kg), levatirasetam (30 mg/kg), vigabatrin (30 mg/kg), fenobarbital (5 mg/kg), clobazam (1 mg/kg) daily. On physical examination, he appeared microcephalic and had a regular heart rhythm at 140 beats per minute (bpm) and blood pressure of 91/47 mm Hg. Laboratory testing revealed hemoglobin level of 10.2 g/dL, sodium 137 mEq/L, potassium 4.8 mEq/L, chloride 97 mEq/L, calcium 9.7 mg/dL, serum urea nitrogen 5 mg/dL, creatinine 0.6 mg/dL, and glucose 145 mg/dL. The electrocardiogram showed sinus rhythm at 136 bpm. We added midazolam infusion (0.1 mg/kg/h) to treatment because of the ongoing convulsions. For his airway safety, the patient was intubated.
On the third day we noticed an abnormal rhythm on his bedside ECG monitor. ECG rhythm strip showed a regular ventricular rate of 94 bpm with an underlying regular ‘saw-tooth’ baseline. Consultation was sought regarding antiarrhythmic therapy for what was thought to be atrial flutter with 4:1 AV conduction (ventricular rate of 94 bpm). (Figs 1 and 2) We consulted the pediatric cardiology department and they interpreted the ECG as atrial flutter. They recommended cardioversion to convert to sinus rhythm. Amiodarone was loaded to the patient and added to his therapy who had atrial flutter after cardioversion. Echocardiogram was performed by pediatric cardiologist and they noted that the atrial rate and ventricular rate were equal. After this, we began to suspect this situation might be a pseudo-flutter due to his muscle contractions. We applied rocuronium to the patient to understand whether this was a pseudo-flutter or not. We saw that the ECG returned to normal sinus rhythm (Fig. 3). We stopped his antiarrhythmic therapy. We took informed consent from the parent of the patient.

Discussion

Typical atrial flutter has a characteristic ECG appearance, classically described as an undulating ‘saw-tooth’ baseline without isoelectric intervals between flutter waves. The atrial rate is typically around 250-350 per minute (cycle frequency of 5 Hz). With 4:1 conduction through the AV node (i.e., every fourth atrial impulse conducted), this would lead to a ventricular rate of 65-85 bpm. The ECG findings in our patient mimicked atrial fibrillation both in frequency and amplitude. Despite the presence of atrial flutter on ECG, his hemodynamics was stable, his pulse was rhythmic, and his blood pressure was normal. However, the consistent ECG findings
with flutter waves and the resistant nature of the arrhythmia distracted us. These findings directed us to inappropriate and potentially dangerous therapies.

There is limited information in the literature regarding the clinical implications of misdiagnosing an ECG artefact and this is the first case in an infant in the literature. All cases are in adult age group or neonatal period. The most likely reported causes of ECG artefacts that mimic both supraventricular and ventricular tachycardia are body movements, muscular fasciculations or contractions, tremor in patients with Parkinson’s disease, ECMO applications, dialysis treatments.2-7

This case shows that the misdiagnosis of electrocardiographic artifact as atrial flutter may lead to unnecessary medication and interventions such as cardioversion and amiodarone. We think that physicians especially those working in intensive care units should be aware of artifacts to avoid unnecessary therapeutic procedures. As Hippocrates said centuries ago “Primum non nocere.”

REFERENCES


Fig. 3. Normal sinus rhythm after rocuronium.