Epilepsy

EPILEPSY AND CONGENITAL LONG QT SYNDROMA: MISDIAGNOSIS OR COMORBIDITY

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A 34-year-old woman has been treated for epilepsy with generalized seizures since the age of 14. Electroencephalography demonstrated paroxysmal slow waves in response to hyperventilation. At the age of 18 a pituitary macroadenoma with hyperprolactinemia was revealed, transnasally surgically removed, followed by gamma-knife radiosurgical treatment and at the age of 22 Crohn`s disease. Because of repeated epileptic seizures, antiepileptic therapy (AET) was multiplied corrected. Four years ago, due to a clinical overlap between seizures and syncope, she was referred to a cardiologist who revealed bradycardia (heart rate 59 bpm) and QT prolongation (QTc 495 ms). In 2019 she gave birth to a female child who had also QT prolongation. Long QT syndrome (LQTS) genetic testing was performed for the child and the diagnosis of LQT2 was confirmed by the identification of a mutation in KCNH2. No genetic analysis was performed for the mother. Her holter electrocardiography performed three months after delivery recorded a symptomatic self-interrupting ventricular tachycardia and a cardioverter-defibrillator (ICD) was urgently installed. In 2021 she gave birth to a male child also with QT prolongation. The ICD was once activated, but seizure did not repeat. AET was not interrupted. The LQT2 subtype of congenital LQTS is a primary arrhythmic disorder caused by changes in potassium channel activation because of a mutation in KCNH2 gene. The differential diagnosis of epilepsy versus LQTS is challenging. The heart and brain expression of this mutated gene can cause, in addition to cardiac disorders, a tendency to recurrent epileptic activity.