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Case Report

Temporal lobe epilepsy and postural orthostatic tachycardia syndrome (POTS)

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Abstract

We describe a 20-year-old woman suffering from right temporal epilepsy, behavioral disorder, and a complaint of paroxysmal palpitations accompanied by anxiety. Detailed cardiac evaluation revealed that the palpitations were due to episodes of marked sinus tachycardia secondary to a concomitant postural orthostatic tachycardia syndrome (POTS) and not of psychogenic origin as initially thought. Treatment with a beta-blocker resulted in the disappearance of palpitations and the associated anxiety. This is the first report of the coexistence of partial epilepsy and POTS. The recognition of such a syndrome in epileptic patients is important in order to offer appropriate therapy. © 2002 Elsevier Science (USA). All rights reserved.

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1. Introduction

Sinus tachycardia is the usual cardiac arrhythmia associated with complex partial and generalized epileptic seizures [1], although other arrhythmias such as severe sinus bradycardia, prolonged sinus arrest, and complete heart block during either the ictal or postictal phase have also been noted [2–4]. In this report we describe a young female patient who was initially diagnosed to have a co-occurrence of epileptic and psychogenic seizures and, in addition, paroxysmal supraventricular tachycardias that sometimes accompanied the seizures. Detailed neurologic and cardiac evaluation revealed that the patient had temporal lobe epilepsy and coexisting postural orthostatic tachycardia syndrome (POTS) with episodic, very rapid sinus tachycardias, one of the manifestations of POTS, and not an independent arrhythmia. To our knowledge, this is the first case in which both conditions are simultaneously present in the same patient.

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2. Case report

The patient is a 20-year-old right-handed woman, of African origin, with epileptic seizures since the age of 9 who underwent an initial evaluation shortly after arriving in Switzerland at the age of 10. There was no family history of epilepsy or of psychiatric disorders. It was also learned that she had convulsions during a febrile illness at the age of 3, secondary to cerebral malaria. Her seizure semiology (experiential phenomena followed by complex partial seizures) suggested a focal onset, although several electroencephalograms were normal. Carbamazepine was subsequently prescribed but the patient was non-complaint and lost to follow-up. She was reevaluated at the age of 18 after presenting to the emergency room with several hours of prolonged complex visual and auditory hallucinations as well as cardiac palpitations. Her interim history was noteworthy for several suicide attempts as well as of child abuse by a close family member. The patient was thought to be experiencing either nonconvulsive partial status epilepticus or psychotic decompensation in the context of a posttraumatic stress syndrome. MRI showed right hippocampal sclerosis, confirmed by volumetric measurements. EEG revealed bitemporal spike and sharp slow

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wave discharges. A diagnosis of temporal lobe epilepsy, with possible previous febrile convulsions, and post-traumatic stress syndrome was established. Carbamazepine, at a lower titration rate, was again prescribed and appeared to be effective but was discontinued because of neutropenia. Several other drugs, alone and in combination, were subsequently tried on an outpatient basis but either were ineffective or caused unacceptable side effects.

The patient was therefore hospitalized again for determination of the suitability of surgical treatment of her seizure disorder and underwent additional studies, including PET, neuropsychological testing, and long-term video-EEG monitoring. Two types of episodes were observed. The first were episodes labeled nonepileptic psychogenic in origin during which she was unresponsive, displayed profound breathing and arching, with eyes closed, and was resistant to passive eye opening. These events lasted up to 1 h, with no changes in the EEG. Of concern during these episodes was the occurrence of an assumed atrial tachycardia at rates up to 200/min lasting up to 10 min as noted on monitored single-lead ECG. The second type of episodes consisted of complex partial seizures with right anterior temporal onset in the EEG. Ictal EEG was concordant with the side of the hippocampal sclerosis and with the presence of a right temporal hypometabolism on PET, but interictal EEG showed mainly left temporal spikes or sharp waves (90%). Moreover, postictal neuropsychological testing revealed both verbal and visuospatial memory loss in comparison to her interictal performance. The diagnosis of right temporal epilepsy with significant left temporal dysfunction was thus retained. A comprehensive psychiatric evaluation was also performed and an additional diagnosis of Dissociative Disorder (300.15 DMS-IV, American Psychiatric Association, 1994) was made. On the basis of these findings, the decision was made to pursue drug treatment rather than surgery.

During the follow-up visits the patient began to complain increasingly of palpitations associated with significant anxiety and sometimes lightheadedness. She had first noticed it 5 years earlier but the frequency had recently increased. A 24 h-ambulatory ECG recording showed sinus rhythm with numerous periods of what seemed to be an atrial tachycardia at rates up to 200/min, with clear atrial activity associated with and driving each QRS complex in a 1:1 relationship. A stress test was subsequently performed, during which she displayed upward eye deviation with preserved consciousness, and was normal with a peak (sinus rhythm) heart rate of 179 min. Transthoracic echocardiography was normal. A beta-blocker, metoprolol, 50 mg/day was prescribed for presumed atrial tachycardia. Invasive investigations with foramen ovale and scalp recordings were also performed in parallel with the cardiac investigations because of the failure of drug therapy to fully control the seizure disorder. No EEG changes in the intracranial electrodes were observed during several supraventricular tachycardia episodes. Consistent with the phase 1 results, the left temporal focus predominated on interictal EEG. No actual seizures were recorded, however, despite sleep and prolonged drug withdrawal.

After hospital discharge, the patient, complaining of an ill-defined malaise that she associated with the beta-blocker, stopped taking the medication and returned with a complaint of recurrence of multiple self-terminating and intolerable episodes of palpitations and anxiety. She was therefore admitted for a diagnostic cardiac electrophysiology study (EPS), for the purpose of confirming the presence of an atrial tachycardia, with radiofrequency ablation of the arrhythmia to follow. No supraventricular arrhythmias could be induced during EPS but periodic sudden spontaneous accelerations of the sinus rate from 100 to 140 min, within 5 s, were observed while the patient was supine on the examination table, in her baseline state. A head-up tilt-table study was scheduled after it was also noted that the patient experienced a similarly rapid increase in sinus rate, as well as her usual anxiety and palpitation symptoms when getting up from the supine position. The head-up tilt-table study revealed a striking acceleration of sinus rhythm immediately after tilting from the horizontal to the 60° vertical position (Fig. 1). The sinus rate increased from 98 min supine to 190 min within 30 s. The blood pressure actually increased during this time from 123/83 supine to 156/98 at the end of 60 s. There was also complete reproduction of the patient's symptom complex of palpitations and lightheadedness. The sinus rate also decreased abruptly at the end of the study (3 min), from 170 to 112 min within 10 s of returning to the horizontal position. The early rapid and marked increase in heart rate with a blood pressure that remained at the baseline level or higher during the study along with the accompanying symptoms made it evident

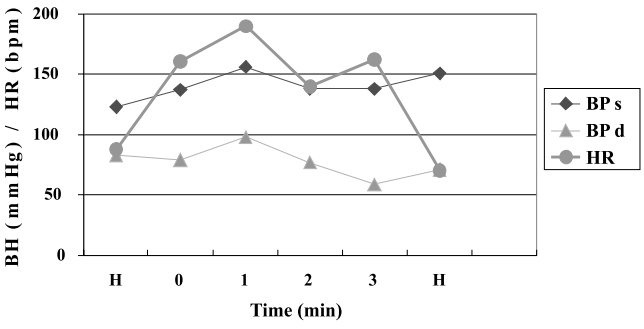

Fig. 1. Graph showing heart rate (HR) and blood pressure (BP) changes during head-up tilt-table testing. The table is tilted up to 60° immediately after Time 0 and brought back to horizontal position immediately after the 3-min reading. H, horizontal position, BP s, systolic blood pressure (diamonds), BP d, diastolic blood pressure (triangles); HR, heart rate (circles).

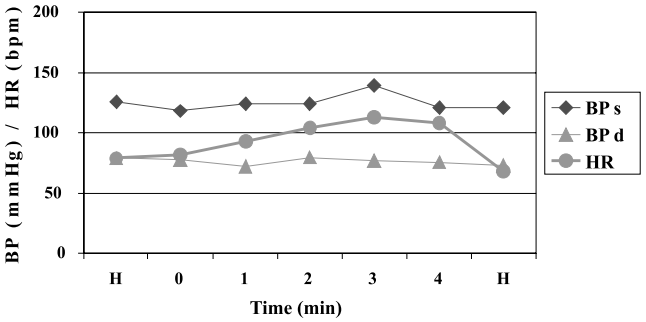

Fig. 2. Graph showing the variation in heart rate (HR) and blood pressure (BP) during head-up tilt-table testing after 10 weeks of bisoprolol treatment. The table is tilted up to 60° immediately after Time 0 and brought back to horizontal position immediately after the 4-min reading. For abbreviations, see Fig. 1.

that this patient had POTS. The patient was placed on another beta-blocker, bisoprolol, 5 mg/day, which she could tolerate, and she has not experienced any recurrences of palpitations since. In keeping with the clinical improvement, a repeat head-up tilt-table study 10 weeks later, on therapy, revealed a sinus rate at 75 min supine with a maximum of 113 min during the fourth minute (Fig. 2). There was no significant change in the blood pressure, which was 132/76 at baseline and 139/77 at the fourth minute. The patient remained asymptomatic throughout the study as well. In retrospect, based on the dynamics of the sinus node behavior noted on subsequent ECG recordings and comparison of "p"-wave morphologies among the different ECG tracings, it also became clear that the initial "atrial tachycardias" noted in this patient were actually episodes of sinus tachycardia (as part of POTS).

3. Discussion

We report the first case of coexistence of temporal lobe epilepsy and postural orthostatic tachycardia syndrome. POTS is a type of orthostatic intolerance characterized most frequently by the occurrence of orthostatic-type symptoms such as palpitations, light headedness, and near-syncope without a concomitant drop in blood pressure. Our patient manifested many of the typical clinical features of POTS patients, as described by Sandroni et al. [5]. She was young and female, consistent with the reported presentation age of 28.9 ± 9.5 years and a female:male ratio of 4:1. In addition, duration of symptoms was less than 5 years, increasingly frequent, as is the case with most patients with POTS. Her two biggest complaints—palpitations and light headedness—were noted in 89 and 95%, respectively, of the study group [5].

Head-up tilt-table testing is the method of choice to identify patients with POTS. The diagnostic criteria are: heart rate increase = 30 bpm within 5 min of standing or upright tilt, heart rate of 120 min within 5 min of standing or upright tilt, development of orthostatic symptoms without orthostatic hypotension, and absence of a known cause of autonomic neuropathy [6,7]. The present patient met these criteria; i.e., her rate increase was dramatic and even greater than the reported increase (from 79 ± 13 to 112 ± 28; mean $\pm$ SD bpm) observed by Sandroni et al. [5] The same group also noted the likely benefit of beta-blockers which were introduced successfully in our patient.

As mentioned earlier, our patient was diagnosed to have a dissociative disorder and her complaints of palpitations were thought to be of psychiatric origin. In reality, the episodes labeled as psychogenic seizures actually proved to be her behavioral reaction to the sinus tachycardia episodes as part of POTS. It is in fact not uncommon for POTS patients to be thought to have an anxiety or panic disorder [8]. Moreover, some of the symptoms, such as lightheadness and palpitations, are also experienced during epileptic auras. Given the similarity of symptoms in psychiatric, cardiologic, and epileptic disorders, the existence of an additional etiology in the cardiovascular domain needs to be considered in patients presenting with an unusual symptomatology to offer optimal treatment.

While the diagnosis and therapeutic management of POTS have become increasingly well defined, the neurophysiologic origin of the disease is not so clear. Suggested mechanisms of POTS include partial sympathetic denervation in the legs, excessive venous pooling, beta-receptor hypersensitivity, alpha-receptor hyper- or hyposensitivity, and altered sympathetic–parasympathetic balance [6]. Brainstem dysfunction has also been suspected [6], but so far, consistent with our findings, no evidence of a cortical dysfunction has emerged. No EEG changes in scalp and foramen electrode recordings were noted during the tachycardias in this patient despite signs of bitemporal damage, thus making a cerebral origin unlikely.

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