Panic attacks and supraventricular tachycardias: the chicken or the egg?

G. Frommeyer · L. Eckardt · G. Breithardt

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Abstract Panic attacks occur in about 2 % of the population. Symptoms include a racing or pounding heart beat, chest pain, dizziness, light-headedness, nausea, difficulty in breathing, tingling or numbness in the hands, flushes or chills, dreamlike sensations or perceptual distortions. The symptoms of paroxysmal supraventricular tachycardia (PSVT) may be similar. A PSVT is often difficult to document on the ECG since it has often ceased before the patient comes to medical attention. Besides, a tachycardia may still be present and even be documented but interpreted as a phenomenon secondary to the panic attack. In addition, ECG abnormalities between episodes can often not be identified. The evidence that in some patients paroxysmal SVT is the cause, but not the consequence of a panic attack, is based on observations that catheter ablation was able to cure patients presenting with panic disorders. To better establish the prevalence of SVT as the underlying mechanism of a panic attack, there is a need for prospective studies and/or registries. Whereas gastric ulcer has in some patients changed from a psychosomatic disorder to an infectious disease, we may hypothesise that a certain proportion of panic disorders may mutate into an underlying arrhythmia rather than a primary psychiatric disorder.

Keywords Panic attacks · Panic disorder · Paroxysmal supraventricular tachycardia

Physicians are frequently confronted with patients who present with sudden onset of symptoms. The type of medical speciality will determine which of the various causes of such symptoms is considered first. The cardiologist will most probably first take a cardiac problem into consideration whereas the neurologist or psychiatrist will consider a psycho-pathological condition first. If symptoms were disease-specific, no major deception would occur. However, a simple but rarely done comparison of textbooks, specific publications and other sources, now including the internet, will reveal that the symptoms of two specific entities are quite similar and overlap [1, 2]. These are panic attacks and paroxysmal supraventricular tachycardia (PSVT) (Table 1).

Panic attacks occur in almost 2 % of the population, reduce quality of life and may elevate the risk of anxiety disorders and other psychiatric disease patterns [3]. Therefore, panic attacks are an important cause of morbidity and increased utilisation of medical care. Approximately half of all visits to primary health care providers are precipitated by symptoms that also frequently occur during a panic attack such as unspecific chest pain, tachycardia, dyspnoea, dizziness, or abdominal discomfort but may well be due to somatic diseases. Therefore, patients frequently undergo comprehensive and expensive diagnostic procedures to rule out relevant somatic diseases [4].

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G. Frommeyer · L. Eckardt · G. Breithardt
Division of Electrophysiology, Department of Cardiovascular Medicine, University of Muenster, Muenster, Germany

G. Frommeyer (✉)
Abteilung für Rhythmologie, Department für Kardiologie und Angiologie, Universitätsklinikum Münster,
Albert-Schweitzer Campus 1,
48149 Münster, Germany
e-mail: gerrit.frommeyer@ukmuenster.de

Symptoms and diagnosis of panic attacks versus PSVT

One of the characteristic signs of both entities is the rapid action of the heart. In PSVT, it is a primary arrhythmia due to reentry in the atrioventricular (AV) node or between atria and ventricles including an accessory AV pathway. The

Table 1 Symptoms are quite similar in paroxysmal supraventricular tachycardia and in panic disorders

Paroxysmal tachycardia	Panic disorders
<ul style="list-style-type: none"> • Sudden onset • Palpitations • Chest pain • Dyspnoea • Increased perspiration • Anxiety • Dizziness • Presyncope or syncope 	<ul style="list-style-type: none"> • Sudden onset • Palpitations, tachycardia • Chest pain or discomfort • Feeling of choking, smothering or shortness of breath sweating, chills, or hot flushes • Anxiety, panic attack • Nausea, ‘butterflies’, or abdominal distress • Dizziness, feeling light headed or fainting

ECG may be completely normal or nearly normal, presenting only minor abnormalities such as absence of Q waves or discrete delta waves (instead of full-blown, easily identifiable preexcitation). If an ECG is taken exactly during the attack, the diagnosis is simple since, besides the rapid heart action often at rates of 160 to 180 beats/min, there is no longer the normal activation of the heart with a P wave followed by the QRS complex but the P wave is mostly hidden within the QRS complex. Conversely, in a panic attack, the rapid heart action is due to an increased sympathetic drive which leads to an increase in sinus rate, with the sequence of the P and QRS being maintained.

One of the major problems occurs when PSVTs are short in duration and do not leave time to do an adequate ECG recording. Similarly, in a patient with a panic attack, it is difficult to obtain an ECG during the attack. Patients may then be sent to an internist or cardiologist to exclude a cardiac origin of symptoms but since the ECG outside an attack is normal (except for cases with Wolff-Parkinson-White syndrome which are normally easy to identify) and there are no other signs of a structural heart disease, these patients are labelled as having no underlying cardiac problem. In this situation, it is of eminent importance that a PSVT cannot be rejected as a cause.

Furthermore, especially female patients frequently face difficulties in being correctly diagnosed as having a PVST [5] since they more frequently have AV node reentrant SVT where there is no clue in the ECG outside an attack, whereas male patients more frequently have AV reentrant tachycardia which may manifest by signs of preexcitation during attacks (WPW syndrome). An improved documentation of the ECG during an attack can be achieved by long-term ECG recorders, patient-activated ECG recorders or implantable loop recorders.

A fundamental problem is the difference of the general approach to PSVT by different medical subspecialists. Cardiologists will most likely regard PSVT as the primary

diagnosis and attribute symptoms such as palpitations, chest pain, dyspnoea, anxiety, panic or dizziness as a response to the reentrant PSVT. The sympathetic arousal that may accompany an arrhythmia may be experienced by the patient as acute anxiety or panic rather than as a cardiac event. In contrast, in psychosomatic medicine or psychiatry, a panic attack may frequently be regarded as the primary event characterised by the described symptoms (Table 1) and the observed or reported tachycardia is regarded as the consequence of the panic attack. Electrocardiographic documentation due to the short nature of attacks is usually difficult. Especially in these cases attribution to other conditions is more likely.

So far, only few studies have addressed these issues. Several case reports have described a primary misdiagnosis of panic attacks which ceased after catheter ablation of PSVT [6–8]. Lessmeier et al. [1] investigated the potential for PSVT to simulate symptoms of a panic attack in a retrospective single-centre study in patients who underwent an electrophysiological study due to PSVT as a consequence of an AV nodal reentrant tachycardia (AVNRT) or atrioventricular reciprocating tachycardia using an accessory pathway (AVRT). Symptoms were correlated with the criteria of panic disorders as listed in the *Diagnostic Manual of Mental Disorders, Fourth Edition* (DSM-IV). Of the included patients, 67 % fulfilled the diagnostic criteria for a panic disorder. PSVT had been unrecognised after initial medical evaluation in 59 patients (55 %), including 13 of 32 patients (41 %) with ventricular preexcitation on the ECG (which should have alerted to the diagnosis of PSVT), and had remained unrecognised for a median of 3.3 years. Prior to eventual identification of PSVT, physicians (non-psychiatrists) attributed symptoms to panic, anxiety, or stress in 32 of the 59 patients (54 %). When PSVT was unrecognised, women were more likely than men to have symptoms ascribed to psychiatric origins (65 % vs. 32 %, respectively; $P < 0.04$). Most frequently, symptoms were attributed to panic, anxiety or stress, as well as mitral valve prolapse or caffeine consumption. Even 41 % of patients presenting with ventricular preexcitation remained misdiagnosed for a median of 3.3 years [1]. Paroxysmal SVT was detected in only 6 of 64 patients (9 %) using long-term ECG (Holter) monitoring versus 8 of 17 patients (47 %) using event recorders ($P < 0.001$). 81 % underwent catheter ablation leading to immediate therapeutic success in 86 % of these patients. Subsequently only 4 % of the successfully treated patients still presented diagnostic criteria for panic disorders despite a lack of evidence for PSVT recurrence at a median follow-up duration of 20 months [1]. The success of an intervention such as catheter ablation in removing the symptoms of panic attacks is a strong argument that in these patients, the PSVT was the primary mechanism. Of course, a placebo effect cannot be excluded but seems highly unlikely.

These findings are in keeping with a recent study where Arentz et al. [2] investigated the impact of a targeted history on success rates of catheter ablation of PSVT. The authors concluded that suitable patients for catheter ablation of PSVT can be identified by their history with a positive predictive value of 92 %. Sudden onset and cessation of tachycardia as well as regular pulse sensations were identified as most important factors and helped to distinguish between PSVT and paroxysmal atrial fibrillation. In addition, psychological consequences of PSVT such as emotional reactivity and limitations of daily life due to fear of new attacks are more important for quality of life than frequency and duration of PSVT attacks [2].

A systematic analysis of various studies on cardiac testing for the diagnosis of arrhythmias in patients with palpitations identified a known history of cardiac disease, having palpitations affected by sleeping or palpitations at work as factors which slightly increase the likelihood of an arrhythmia as the underlying source of palpitations. In addition, regular rapid-pounding sensations as well as visible neck pulsations increased the likelihood of AVNRT. Duration of palpitations of less than five minutes or a known history of panic disorder decreased the likelihood of veritable arrhythmias [9]. However, clinical examination on its own is not sufficient to precisely diagnose or to exclude an underlying tachycardia. Therefore, a correlation of symptoms with Holter monitoring is crucial for a precise diagnosis [9].

Psychopharmacotherapy and cardiovascular side effects

Some antipsychotic drugs are known to have various unfavourable side effects. Particularly tricyclic antidepressants (i.e. imipramine, desipramine, amitriptyline or clomipramine) as well as neuroleptics (i.e. haloperidol, droperidol, thioridazine or pimozide) interact with ion channels and can thereby enhance life-threatening arrhythmias [10]. Several new antipsychotic agents (i.e. risperidone, sertindole or aripiprazole) and selective serotonin reuptake inhibitor antidepressants (SSRIs, i.e. paroxetine, fluoxetine, citalopram or escitalopram) have recently been introduced and were assumed to have fewer and more benign side effects than long-established substances [11]. However, an increasing number of case reports demonstrate that employment of new antipsychotics may be associated with occurrence of severe arrhythmias, a prolonged QT interval, or orthostatic hypertension in patients without a history of cardiovascular disease. In agreement, several new compounds exhibited inhibitory effects on cardiac and vascular ion channels.

The results of experimental studies and reported cases suggest potential clinically important cardiovascular effects of the new generation of antidepressants and antipsychotics. As a consequence, clinicians ought to be aware of potential adverse reactions, especially in patients with a history of

cardiovascular events. Therefore, regular electrocardiographic controls are recommended [10].

In the context of the present topic, successful treatment of a patient with presumed panic attacks with such drugs leaves the possibility that the success is due to the antipsychotic effect of such drugs but may also represent an electrophysiological side effect on the reentrant circuit of an underlying PSVT. Successful treatment by antipsychotic drugs can therefore not be used as an argument in favour of a primary psychopathological mechanism of panic attacks in a given patient.

Panic disorder, psychological imbalance and links to cardiovascular disease

Apart from the described problem of separating panic disorders and PSVT from each other, chronic panic disorders are also associated with an increased occurrence of subsequent coronary heart disease and myocardial infarction [12]. In a large cohort study of patients with panic attacks or panic disorder, a significantly elevated incidence of myocardial infarction was found in patients under 50 years of age with recent onset of panic attacks and a higher incidence of coronary heart disease through all age groups as compared with people unexposed to panic attacks [12]. The authors speculate that this correlation may be based on an initial misdiagnosis of coronary heart disease as panic attacks since comparable characteristic symptoms can occur in both entities.

Due to the availability of continuous rhythm monitoring, patient cohorts with implantable cardioverter-defibrillators (ICD) are mostly examined in studies on correlation of psychological factors and arrhythmias. Further evidence that psychological disorders can be relevant for cardiovascular disease and mortality has been reported by Steinberg et al. [13]. They described that psychological status assessed by different quality-of-life instruments can predict mortality in patients with a history of life-threatening ventricular arrhythmias [13]. In accordance, anger has been identified as a precipitator in another cohort of patients with ICDs [14]. In the cited study, ventricular arrhythmias which occurred in the setting of emotional distress were more likely to be pause dependent and rather polymorphic than monomorphic. The authors speculate based on these observations that anger may lead to a more disorganised arrhythmogenic substrate as the underlying link of emotional distress and sudden cardiac death [14].

Conclusions

Panic attacks and PSVT frequently present similar clinical symptoms. Especially in cases without unambiguous electrocardiographic documentation, it can be difficult to

discriminate between these entities. Both entities most likely occur in young and otherwise healthy patients. Women are more frequently afflicted from panic disorders than men but also develop AV-node reentrant tachycardias more frequently. Preexcitation as a probable cause for tachycardias can mostly be easily recognised on the ECG (if done in such a patient) whereas the potential for AV-node reentrant tachycardias cannot be identified in the ECG during sinus rhythm.

Since repeated panic attacks can lead to chronic panic disorders, an adequate diagnosis and targeted therapy of primary PSVT is important to prevent chronic processes. In addition, psychological factors obviously contribute to prognosis of cardiovascular diseases.

In order to establish the prevalence of SVT as the underlying mechanism of a panic attack, there is a need for a prospective studies and/or registries of patients with panic attacks. Whereas gastric ulcer has at least in some patients changed from a psychosomatic disorder to an infectious disease, we may hypothesise that at least a certain proportion of panic disorders may mutate into an underlying arrhythmia rather than a primary psychiatric disorder upon more detailed evaluation.

References

1. Lessmeier TJ, Gamperling D, Johnson-Liddon V, et al. Unrecognized paroxysmal supraventricular tachycardia. Potential for misdiagnosis as panic disorder. *Arch Intern Med.* 1997;157(5):537–43.
2. Arentz T, Baumgartner P, Kalusche D. [Hochfrequenzablation supraventrikulärer Tachykardien: Gezielte Anamnese identifiziert geeignete Patienten—auch ohne Anfalls-EKG]. *Cardiovasc.* 2003;3(5):24.
3. Goodwin RD, Lieb R, Hoefler M, et al. Panic attack as a risk factor for severe psychopathology. *Am J Psychiatry.* 2004;161(12):2207–14.
4. Katon W. Panic disorder: relationship to high medical utilization, unexplained physical symptoms, and medical costs. *J Clin Psychiatr.* 1996;57 Suppl 10:11–8. discussion 9–22.
5. Wood KA, Wiener CL, Kayser-Jones J. Supraventricular tachycardia and the struggle to be believed. *Eur J Cardiovasc Nurs.* 2007;6(4):293–302.
6. McCrank E, Schurmans K, Lefcoe D. Paroxysmal supraventricular tachycardia misdiagnosed as panic disorder. *Arch Intern Med.* 1998;158(3):297.
7. Domschke K, Kirchhof P, Zwanzger P, et al. Coincidence of paroxysmal supraventricular tachycardia and panic disorder: two case reports. *Ann Gen Psychiatr.* 2010;9:13.
8. Laederach-Hofmann K, Glauser R. Paroxysmal tachycardia in a patient without panic disorder. *Arch Intern Med.* 1998;158(8):929.
9. Thavendiranathan P, Bagai A, Khoo C, et al. Does this patient with palpitations have a cardiac arrhythmia? *JAMA.* 2009;302(19):2135–43.
10. Pacher P, Kecskemeti V. Cardiovascular side effects of new antidepressants and antipsychotics: new drugs, old concerns? *Curr Pharm Des.* 2004;10(20):2463–75.
11. Eckardt L, Breithardt G, Haverkamp W. Electrophysiologic characterization of the antipsychotic drug sertindole in a rabbit heart model of torsade de pointes: low torsadogenic potential despite QT prolongation. *J Pharmacol Exp Ther.* 2002;300(1):64–71.
12. Walters K, Rait G, Petersen I, et al. Panic disorder and risk of new onset coronary heart disease, acute myocardial infarction, and cardiac mortality: cohort study using the general practice research database. *Eur Heart J.* 2008;29(24):2981–8.
13. Steinberg JS, Joshi S, Schron EB, et al. Psychosocial status predicts mortality in patients with life-threatening ventricular arrhythmias. *Heart Rhythm.* 2008;5(3):361–5.
14. Stopper M, Joska T, Burg MM, et al. Electrophysiologic characteristics of anger-triggered arrhythmias. *Heart Rhythm.* 2007;4(3):268–73.