Diagnostic challenges in supraventricular tachycardia: anticipating value of natriuretic peptides

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It is important not to overlook supraventricular tachycardia (SVT) in patients complaining of palpitation or tachycardia-related symptoms but with normal ECG and heart rate in emergency department or outpatient clinics. The severity and presentation of symptoms is highly variable and depends on features including heart rate, duration of tachycardia, underlying heart disease, and individual patient perception. Early measurement of natriuretic peptides in patients presenting with palpitation and/or tachycardia-related symptoms would guide the clinician to rule out tachycardia and/or SVT. High levels of natriuretic peptides within 30 min of postattack period would certainly increase the likelihood of SVT. *Cardiovasc Endocrinol Metab* 7:34–36

Introduction

Supraventricular tachycardia (SVT) refers to regular, narrow QRS complex, rhythms having an electrophysiologic substrate arising above the bundle of His and causing heart rates typically in excess of 150 bpm. Although atrial flutter and atrial fibrillation are the classes of SVT by definition, the mechanism is slightly different than those of typical SVTs. The prevalence of SVT in literature has been supplied by Orejarena *et al.* [1]. They have reported the prevalence of SVT in general population as 225 per 1000 persons with an incidence of 35 per 100 000 person-years based on ECG documented records. Accordingly, real-world prevalence might be expected to be higher than the reported prevalence owing to cases of nondocumented but having undiagnosed SVTs.

The most common symptom during SVT is palpitation or a sensation that the heart is beating rapidly, fluttering, or racing. Other common symptoms include shortness of breath, hyperventilation, syncope, sweating, light-headedness, dizziness, chest pain, loss of consciousness, and anxiety [1,2]. Occasionally they may have no symptoms. Serious presentations such as overt heart failure, myocardial infarction, and syncope are uncommon [3]. Moreover, psychological symptoms such as anxiety and depressed mood are also common in SVT [4,5]. Sudden and unexpected nature of tachycardia may subsequently lead to or trigger anxiety disorders in patients during the clinical course of disease. Anxiety disorder has been found to be present in ~25% of patients with SVT [4].

Hypothesis

It is important not to overlook SVT in patients complaining of palpitation or tachycardia-related symptoms but with normal ECG and heart rate in emergency department or

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outpatient clinics. Therefore, it is essential for general practitioners, cardiologist, and electrophysiologists to be aware of this symptomatically complex electrophysiologic entity to diagnose the SVT as an underlying etiology. Natriuretic peptides, namely atrial (ANP), brain (BNP), and C-type natriuretic peptides (CNP) have shown to be increased during and immediately after the SVT. Accordingly, it might be hypothesized that measurement of natriuretic peptides early in postattack periods would help to ascertain the presence of SVT in emergency department in selected cases.

Diagnostic challenges in supraventricular tachycardia

The main contributing factors complicating the diagnosis of SVT in clinical practice are summarized in Table 1. The severity and presentation of symptoms is highly variable and depends on features including heart rate, duration of tachycardia, underlying heart disease, and individual patient perception. One might feel high-rate SVT as a slight chest discomfort; one might experience a sudden syncope without feeling any palpitation. Moreover, unusual presentations of SVT such as tinnitus, burping, asthma-like attacks, and absence seizure-like attacks have been reported recently [6–9]. Symptoms of SVT have been reported to be owing to not only heart

Table 1 Diagnostic challenges in supraventricular tachycardia

- 1 Short duration and paradoxical nature of index tachycardia
- 2 Individual differences in perception of index tachycardia
- 3 Tachycardia documented approaches of electrophysiologists
- 4 Absence of index tachycardia at hospital admittance or emergency department
- 5 Unusual presentation of index tachycardia; noncardiovascular symptoms, misdiagnosis as an acute coronary syndrome

rate but also to cross-talk between cranial nerves, cervical ganglions, and cardiac nerves [10].

Tachycardia induces chest pain and troponin positivity mimicking acute coronary syndrome. It has been reported that patients experiencing paroxysmal SVT had elevated troponin levels. Coronary angiography is often performed in some patients presenting with PSVT because they have chest pain and high troponin levels. Ben Yedder *et al.* [11] have reported ~30% troponin positivity in their series of 73 patients who required hospitalization for SVT. Even in the absence of documented tachycardia, slow pathway ablation successfully terminated paradoxical chest pain attacks in a patient who underwent coronary angiography several times [12].

In terms of clinical diagnosis, duration of SVT is one of the major challenges. Short duration of tachycardia or tachycardia-related symptoms may not leave time to make ECG recording and clinical diagnosis. A significant proportion of patients in clinical practice may not have ECG recordings of the index tachycardia (nondocumented SVT) or only have recordings of limited diagnostic value. It is not always easy to obtain a diagnostic ECG, and in case of sporadic symptoms, the continuous ECG record for 24 h (Holter) might not be efficient [13].

Documentation of index tachycardia has pivotal role to make a clinical correlation with symptoms and to make a correct diagnosis for guiding therapy. Although common symptom during SVT is palpitations or a sensation that the heart is beating rapidly, fluttering, or racing, this is not the case in clinical practice. Despite the highly specified symptomatic and ECG diagnostic criteria, diagnosis treatment and referral for electrophysiologic studies are not always straightforward.

Even in the presence of documented SVT, it is not always possible to induce arrhythmia in the electrophysiology laboratory. A recent report has shown that a substantial number of patients with suspected paroxysmal tachycardia, but without ECG documentation, have inducible SVTs and obtains a clear clinical benefit from electrophysiological study [14]. A subtle inference of this study is that prevalence of SVT might be higher than the ECG-based reported prevalence in literature [1].

Individual approaches with a high suspicion index and concerning the paroxysmal nature and postattack symptoms would help us to identify patients experiencing SVT in complex cases. It is important to question the presence of postattack symptoms, such as polyuria, fatigue, or dizziness. Fatigue and diuresis have also been reported to occur after an episode of SVT [6–10]. Although the mechanism is not known yet, patients describe fatigue lasting several hours to days after an episode of SVT [15]. A diuresis has been reported to occur in 20–50% of episodes of paroxysmal SVT. Typically, this response occurs with heart rates greater

than 120 bpm after a duration of at least 10–30 min [16–18]. In addition to SVT, polyuric response has also been noted after the episodes of atrial fibrillation, atrial flutter, and ventricular tachycardia. The characteristics of the diuresis during SVT include an increase in urine flow and volume, free water clearance, and sodium excretion and a decrease in urine osmolality [16–19].

Natriuretic peptides and supraventricular tachycardia

ANP, BNP, and CNP are a family of structurally related peptides that participate in the integrated control of renal and cardiovascular function. Although it shares structural and physiological properties with ANP and BNP, CNP is predominantly secreted from the vascular endothelium [20]. ANP is a 28-amino acid peptide secreted from the atrium tissue and possesses natriuretic, vasoactive, and rennin-inhibiting actions [21,22]. Increased ANP and decreased vasopressin release have been shown to be the responsible mechanism of polyuria after SVT episodes [23]. Oliver *et al.* [23] have reported that plasma concentrations of ANP may rise early after the onset of paroxysmal SVT and remain raised for prolonged periods of tachycardia, independent of clinical indices of left ventricular failure. In experiencing an episode of atrioventricular nodal tachycardia, plasma and urine vasopressin were also suppressed [23]. Left atrial stretch receptors and vagal afferent pathways have been implicated in the mediation of vasopressin inhibition. During left atrial distention, a decrease in plasma vasopressin concentration has been demonstrated to occur [24]. In contrast, Kaye *et al.* [25] have reported that the mechanisms of diuresis do not appear to involve arginine-vasopressin level alone.

Increase of ANP secretion has been shown to occur during SVT and sustain for 30 min [26,27]. Infusion of ANP has been demonstrated to produce an increase in urine, sodium, potassium, and chloride excretion; an increase in glomerular filtration rate and urine flow; an inhibition of aldosterone secretion; a relaxation of vascular smooth muscle; and a systemic hypotensive effect [21,28,29]. Infusion of synthetic ANP into normal human volunteers has also produced significant natriuresis, kaliuresis, and diuresis [30,31]. Although it has never been assessed in literature, diuresis, kaliuresis, natriuresis, and subsequent hypotension are the likely underlying reasons of postattack fatigue and dizziness.

BNP secreted from the myocardium and CNP secreted predominantly from the vascular endothelium have also shown to increase after SVT. Kuo *et al.* [32] have assessed the secretion of natriuretic peptide by measuring levels of natriuretic peptides in the coronary sinus and femoral artery and shown that ANP, BNP, and CNP levels in the coronary sinus at baseline and each time point after paroxysmal SVT were significantly higher than those in the femoral artery in individuals with paroxysmal SVT and normal left ventricular systolic function, indicating that,

in the body, the heart is a major source of natriuretic peptides, including ANP, BNP, and CNP, at rest and after SVT. In addition, sustained SVT for 30 min has been shown to enhance the heart to release ANP, BNP, and CNP. The increase of ANP to paroxysmal SVT is to a greater degree but decreases faster, compared with BNP and CNP [32].

Early measurement of ANP, BNP, CNP in patients presenting with palpitation and/or tachycardia-related symptoms would guide the clinician to rule out tachycardia and/or SVT. High levels of natriuretic peptides within 30 min of postattack period would certainly increase the likelihood of SVT. Measurement of natriuretic peptides as early as possible at emergency department in those patients might be of clinical relevance.

Plasma elimination half-lives of ANP, BNP and CNP are relatively short, ranging from 2 to 5 min in human beings [33,34]. Despite the short half-life, higher plasma concentrations of ANP, BNP, and CNP have been reported up to 30 min after termination of paroxysmal SVT [26,27,32]. Although ANP has the highest plasma levels after induction of paroxysmal SVT, it returns the normal limits within 30 min after termination of paroxysmal SVT. In contrast, BNP and CNP levels have remained raised at 30 min after termination of tachycardia. In clinical practice, prehospital duration and rate of paroxysmal SVT might influence the plasma levels and elimination time of natriuretic peptides by modulating production and release of natriuretic peptides. Besides, precursors of BNP and CNP, namely, NT-proBNP and pro-CNP, which have longer elimination half-life [33,34], lengthen the time interval between the termination of SVT and blood sampling to detect possible increased levels of natriuretic peptides.

Conclusion

Measurement of natriuretic peptides in patients presenting with palpitation or tachycardia-related symptoms and normal ECG at emergency department may lead to identify the patients experiencing SVT, who are otherwise overlooked.

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Conflicts of interest

There are no conflicts of interest.

References

- Orejarena LA, Vidaillet H Jr, deStefano F, Nordstrom DL, Vierkant RA, Smith PN, Hayes JJ. Paroxysmal supraventricular tachycardia in the general population. *J Am Coll Cardiol* 1998; **31**:150–157.
- Wang PJ, Estes NAM III. Supraventricular tachycardia. *Circulation* 2002; **106**:e206–e208.
- Ganz LI, Friedman PL. Supraventricular tachycardia. *N Engl J Med* 1995; **332**:162–173.
- Lessmeier TJ, Gampelring D, Johnson-Liddon V, Fromm BS, Steinman RT, Meissner MD, Lehmann MH. Unrecognized paroxysmal supraventricular tachycardia. Potential for misdiagnosis as panic disorder. *Arch Intern Med* 1997; **157**:537–543.
- Frommeyer G, Eckardt L, Breithardt G. Panic attacks and supraventricular tachycardias: the chicken or the egg? *Neth Heart J* 2013; **21**:74–77.
- Yetkin E. An extremely rare presentation of supraventricular tachycardia: burping. *Int J Cardiol* 2015; **184**:369–370.
- Yetkin E, Kaleagzi FC. Recovery of absence seizure-like symptoms in a patient after slow pathway radiofrequency ablation. *Int J Cardiol* 2015; **182**:44–45.
- Yetkin E, Tandogan I. Tinnitus preceding tachycardia and syncope. *Int J Cardiol* 2015; **198**:93–94.
- Ozturk S, Turhan H, Yetkin E. Asthma-like attacks terminated by slow pathway ablation. *Ann Thorac Med* 2017; **12**:127–128.
- Yetkin E. Symptoms in supraventricular tachycardia: is it simply a manifestation of increased heart rate? *Med Hypotheses* 2016; **91**:42–43.
- Ben Yedder N, Roux JF, Paredes FA. Troponin elevation in supraventricular tachycardia: primary dependence on heart rate. *Can J Cardiol* 2011; **27**:105–109.
- Yetkin E. Documented but non-induced supraventricular tachycardia and vice versa. *Int J Cardiol* 2015; **182**:438–439.
- Rockx MA, Hoch JS, Klein GJ, Yee R, Skanes AC, Gula LJ, et al. Is ambulatory monitoring for 'community-acquired' syncope economically attractive? A cost-effectiveness analysis of a randomized trial of external loop recorders versus Holter monitoring. *Am Heart J* 2005; **150**:1065.
- Lauschke J, Schneider J, Schneider R, Nesselmann C, Tischler T, Glass A, Bansch D. Electrophysiological studies in patients with paroxysmal supraventricular tachycardias but no electrocardiogram documentation: findings from a prospective registry. *Europace* 2015; **17**:801–806.
- Wood KA, Wiener CL, Kayser-Jones J. Supraventricular tachycardia and the struggle to be believed. *Eur J Cardiovasc Nurs* 2007; **6**:293–302.
- Wood P. Polyuria in paroxysmal tachycardia and paroxysmal atrial flutter and fibrillation. *Br Heart J* 1963; **25**:273–282.
- Luria MH, Adelson EI, Lochaya S. Paroxysmal tachycardia with polyuria. *Ann Intern Med* 1966; **65**:466–470.
- Nicklas JM, diCarlo LA, Koller PT, Morady F, Diltz EA, Shenker Y, Grekin RJ. Plasma levels of immunoreactive atrial natriuretic factor increase during supraventricular tachycardia. *Am Heart J* 1986; **112**:923–928.
- Ghose RR, Jockes AM, Kyriachou EH. Renal response to paroxysmal tachycardia. *Br Heart J* 1965; **27**:684.
- Suga S, Nakao K, Itoh H, Komatsu Y, Ogawa Y, Hama N, Imura H. Endothelial production of C-type natriuretic peptide and its marked augmentation by transforming growth factor-beta. Possible existence of 'vascular natriuretic peptide system'. *J Clin Invest* 1992; **90**:1145–1149.
- Burnett JC Jr, Granger JP, Oppenorth TJ. Effects of synthetic atrial natriuretic factor on renal function and renin release. *Am J Physiol* 1984; **247**:F863–F866.
- De Bold AJ, Borenstein HB, Veress AT, Sonnenberg H. A rapid and potent natriuretic response to intravenous injection of atrial myocardial extracts in rats. *Life Sci* 1981; **28**:89–94.
- Oliver JR, Twidale N, Lakin C, Cain M, Tonkin AM. Plasma atrial natriuretic polypeptide concentrations during and after reversion of paroxysmal supraventricular tachycardias. *Br Heart J* 1988; **59**:458–462.
- Ledsome JR, Wilson N, Ngsee J. Time course of changes in plasma vasopressin during atrial distention. *Can J Physiol Pharmacol* 1982; **60**:1210–1218.
- Kaye GC, Bayliss P, Lowry PJ, Drury PL, Camm AJ. Effect of induced supraventricular tachycardias on changes in urine output and plasma hormone levels in man. *Clin Sci (Lond)* 1992; **82**:33–37.
- Tsai RC, Yamaji T, Ishibashi M, Takaku F, Pang SC, Yeh SJ, et al. Atrial natriuretic peptide during supraventricular tachycardia and relation to hemodynamic changes and renal function. *Am J Cardiol* 1988; **16**:1260–1264.
- Kojima S, Fujii T, Ohe T, Karakawa S, Lida T, Hirata Y, et al. Physiologic changes during supraventricular tachycardia and release of atrial natriuretic peptide. *Am J Cardiol* 1988; **62**:576–579.
- Maack T, Marion DN, Camargo MJF, Leinert HD, Laragh JH, Vaughan ED, Atlas SA. Effects of auricularin (atrial natriuretic factor) on blood pressure, renal function and the renin-aldosterone system in dogs. *Am J Med* 1984; **77**:1069–1075.
- Atarashi K, Murlow PF, France-Saenz R, Rapp J. Inhibition of aldosterone production by an atrial extracts. *Science* 1984; **224**:992–994.
- Richards AM, Ikram H, Yandle TG, Nicholls MG, Webster MWJ, Espiner EA. Renal hemodynamic and hormonal effects of human alpha atrial natriuretic peptide in healthy volunteers. *Lancet* 1985; **1**:545–549.
- Tikkanen I, Fyhrquist F, Metsarinne K, Leidenius R. Plasma atrial natriuretic peptide in cardiac disease and during infusion in healthy volunteers. *Lancet* 1981; **1**:66–69.
- Kuo JY, Wang AM, Chang SH, Hung CL, Chen CY, Shih BF, Yeh HI. Responses of cardiac natriuretic peptides after paroxysmal supraventricular tachycardia: ANP surges faster than BNP and CNP. *Am J Physiol Heart Circ Physiol* 2016; **310**:H725–H731.
- Del Ry S, Cabiati M, Clerico A. Natriuretic peptide system and the heart. *Front Horm Res* 2014; **43**:134–143.
- Potter LR. Natriuretic peptide metabolism, clearance and degradation. *FEBS J* 2011; **278**:1808–1817.