Ventricular tachycardia during basilar-type migraine attack

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Abstract: Autonomic dysfunction is a characteristic of migraine attacks, rarely, even cardiac repolarization abnormalities have been associated with migraine. We report a case of documented ventricular tachycardia during basilar-type migraine attack. The therapeutic implications of such a co-occurrence as well as a possible relationship between ventricular tachycardia and the underlying biology of basilar-type migraine are discussed.

Keywords: autonomic dysfunction, basilar-type migraine, cardiac arrhythmia, ventricular tachycardia

Introduction

Migraine is a primary, chronic intermittent neurovascular disorder characterized by episodic headache, vegetative symptoms, and in a minority of patients, by other neurological signs such as aura [Headache Classification Subcommittee of the International Headache Society, 2004]. Basilar-type migraine has been defined in the international classification of headache disorders as migraine with aura symptoms clearly originating from the brainstem and/or both hemispheres simultaneously [Headache Classification Subcommittee of the International Headache Society, 2004]. It differs from hemiplegic migraine by the absence of motor deficit. Aura consists of at least two of the following fully reversible symptoms: dysarthria, vertigo, tinnitus, hyperakusis, diplopia, visual symptoms occurring simultaneously in both the temporal and nasal fields of both eyes, ataxia, decreased level of consciousness, and simultaneous bilateral paresthesias. These symptoms can develop gradually over ≥ 5 min and/or different aura symptoms can occur in succession over ≥ 5 min or each aura symptom can last ≥ 5 and ≤ 60 min. Headache fulfilling criteria for migraine with aura begins during the aura or follows the aura within 60 min and the event is not attributed to another disorder [Headache Classification Subcommittee of the International Headache Society, 2004]. Despite these differences, aura reflects the dysfunction of the

brainstem. Basilar-type migraine represents 10% of migraine with aura with a male:female ratio of 1:3.8 [Kirchmann *et al.* 2006].

Dysfunction of the autonomic nervous system (ANS) is a primary characteristic of migraine [Melek *et al.* 2007; Sanya *et al.* 2005]. Therefore, patients with migraine have a variety of symptoms, such as vasodilatation, nausea, vomiting, diarrhoea, cutaneous vasoconstriction and sinus-type symptoms such as nasal congestion and rhinorrhoea. The electrocardiographic (ECG) changes seen during a migraine attack compared with the pain-free period could be secondary to a reversible disequilibrium of autonomic innervation of the heart [Melek *et al.* 2007]. Dysfunction of the ANS may therefore also affect atrial and ventricular repolarization [Aygun *et al.* 2003] (Table 1). Electrocardiographic analyses have provided more details in terms of the detection of abnormalities in atrial and ventricular repolarization, which potentially may result in arrhythmias in migraineurs [Aygun *et al.* 2003].

Case report

A 22-year-old white male patient was admitted to our department because of two acute episodes of vertigo, ataxia and excessive perspiration, each episode lasted about 40 min. The time interval between the two attacks amounted to 10 days.

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Table 1. Observed cardiac disturbances during migraine attacks.

Type of cardiac disturbance	References
Sinus bradycardia	Jhee <i>et al.</i> [1998]* Aygün <i>et al.</i> [2003]§
P-wave dispersion	Duru <i>et al.</i> [2006]‡
ST-segment abnormalities	Aygün <i>et al.</i> [2003]§
Nonspecific ST-T changes	Jhee <i>et al.</i> 1998*
Atrial fibrillation	Turhan <i>et al.</i> 2004
Atrial premature contraction	Aygün <i>et al.</i> 2003§
Ventricular premature contraction	Jhee <i>et al.</i> 1998* Aygün <i>et al.</i> 2003§
Coronary vasospasm	Lafitte <i>et al.</i> 1996
Increased QTc interval	Duru <i>et al.</i> 2006‡ Aygün <i>et al.</i> 2003§
Altered heart rate variability during autonomic tests	Pogacnik <i>et al.</i> 1993§
Right bundle branch block	Jhee <i>et al.</i> 1998*
Prolonged PR-intervals	Aygün <i>et al.</i> 2003§
[Adapted from Melek <i>et al.</i> [2007].]	
*A total of 67 patients studied.	
§A total of 30 patients studied.	
‡A total of 55 patients studied.	
§Autonomic function in 62 migraine patients compared with 45 healthy controls.	
Single case report.	

Both episodes were followed by hypaesthesia of both hands and arms. A bilateral occipital headache of a pulsatile character and a severity of 7 in the comparative pain scale manifested directly after the aura symptoms and lasted for about 4–6 h. The headache was accompanied by excessive nausea without phono- or photophobia. During monitoring in the emergency room, a short episode of a ventricular tachycardia was documented during the headache phase (Figure 1). At that time the patient had a stable blood pressure of 150/90 mmHg.

Except for a hypaesthesia of both upper extremities, a normal neurological status was documented in the clinical examination. Past medical history was normal with no family history of ventricular tachycardia or cardiac arrhythmias, and no known history of allergic reactions. The patient did not take any regular medication.

The complementary diagnostic examinations, including cerebral magnetic resonance imaging (MRI), MR-angiography, an extracranial and intracranial doppler and duplex sonography, transthoracic and transoesophageal echocardiography, stress-ECG and a 24-h ECG, electroencephalography, visual and acoustic evoked potentials, and lumbar puncture as well as screenings for

sympathomimetic intoxications, were without any pathological findings.

The patient was discharged with the diagnosis of a first diagnosis of a basilar-type migraine and with a prophylactic therapy of metoprolol, also following the recommendation of the cardiologists.

Discussion

In this case report we described a first typical basilar-type migraine of a 22-year-old male where an episode of ventricular tachycardia was documented. This led us to pursue an extensive cardiological diagnostic regime to exclude a possible cardiac cause of the arrhythmia.

The pathophysiology of migraine is not completely understood but it is assumed that migraine is a neurovascular disorder [Tfelt-Hansen and Koehler, 2011]. The hypotheses for the generation of migraine include cortical spreading depression (depolarization of neurons), vasodilatation and vasoconstriction leading to neurotransmitter dysregulation (serotonin reduction, substance P release), neurogenic inflammation and pain. Both vascular and neural influences cause migraine [Dreier, 2011; D'Andrea and Leon, 2010]. However,

Figure 1. ECG monitoring in the emergency room. After 3 beats with normal sinus rhythm (small QRS complex), polymorphic broad QRS complex tachycardia occurred, followed by a monomorphic ventricular tachycardia (broad QRS complex tachycardia with 16 beats), with spontaneous termination; thereafter normofrequent sinus rhythm was recorded.

the definite pathogenesis of basilar-type migraine remains uncertain [Kirchmann *et al.* 2006]. Recent hypotheses implicate the role of brainstem antinociceptive structures like the periaqueductal grey matter, the locus coeruleus and the mesencephalic raphe nucleus. The ‘central sensitization’ hypothesis is based on the altered processing of sensory inputs in the brainstem, principally in the trigeminal nucleus [Mishra *et al.* 2008].

It is known that migraine is commonly associated with a variety of autonomic accompaniments such as nausea, vomiting, pallor, flushing, pilo-erection and diaphoresis [Campbell, 1990]. This may be explained by varied autonomic dysregulation, perhaps the result of an imbalance between the sympathetic and parasympathetic systems [Havanka-Kanniainen *et al.* 1988]. Therefore, involvement of the ANS may be responsible for many of the clinical features.

Pathophysiological mechanisms of cardiac disturbances in migraine

The pathophysiology of the autonomic dysfunction in migraine was initially attributed to the central noradrenergic system and its cortical connections [Welch, 1987], however it has also been discussed whether the ANS dysfunction is the primary problem or secondary to diseases of the central nervous system and the peripheral nervous system. It has been suggested that the sympathetic dysfunction in migraine relates to an imbalance of sympathetic cotransmitters [Peroutka, 2004].

The similarity of migraine to pure autonomic failure is based upon reduced supine plasma norepinephrine levels, peripheral adrenergic receptor supersensitivity and clinical symptomatology directly related to sympathetic nervous system dysfunction [Martinez *et al.* 1993]. Specifically, it is suggested that a migraine attack is characterized by

a relative depletion of sympathetic norepinephrine stores in conjunction with an increase in the release of other sympathetic cotransmitters such as dopamine, prostaglandins, adenosine triphosphate and adenosine [Peroutka, 2004]. On the other hand, migraine differs from both pure autonomic failure and multiple system atrophy in that migraineurs retain the ability, although suboptimal, to increase plasma norepinephrine levels following physiological stressors [Martinez *et al.* 1993].

Regarding cardiac repolarization abnormalities, the rich autonomic innervation of the heart plays an important role in modifying cardiovascular function. Sympathetic hyperfunction and hypofunction are described in migraine case-control studies [Appel *et al.* 1992; Pogacnik *et al.* 1993; Jhee *et al.* 1998]. Dysfunction of the ANS may affect atrial and ventricular repolarization. Therefore disrupted autonomic innervation of the heart and coronary arteries in patients with migraine may result in possible ECG abnormalities during headache. An increased frequency of cardiac arrhythmias using single-lead ECG monitoring during migraine compared with a pain-free period has been reported [Aygun *et al.* 2003]. A prolonged QT interval and P-wave dispersion in the pain-free period can be predictors of atrial and ventricular arrhythmias [Schouten *et al.* 1991; Duru *et al.* 2006]. These findings may reflect atrial and ventricular repolarization abnormalities that are affected by a disturbed ANS. The results of these studies suggest that patients with migraine during attacks may be under risk for atrial and ventricular arrhythmias. Despite the results of these studies, an increased incidence of ventricular arrhythmias and sudden death in patients with migraine does not appear [Melek *et al.* 2007; Havanka-Kanninen *et al.* 1988] (Table 1). This could be explained by the relatively younger age of patients with active migraine who are free from cardiovascular risk factors [Turhan *et al.* 2004].

Study limitations

As ventricular tachycardia can occur rarely and spontaneously with an incidence of 2–3% in young people without overt heart disease [Alexander, 2001], it should be considered that the ventricular tachycardia could have been unrelated to the migraine. The simultaneous occurrence of two rare situations makes this scenario unlikely, but not impossible. On the other hand, there are reports of stress- or exercise-induced occurrence of ventricular tachycardia in a young population

[Tan and Scheinman, 2008]. Furthermore, a migraine attack could be considered to be a stressful event. In this case, the ventricular tachycardia would have been related to the stress of the migraine attack itself, rather than any relation to the specific pathophysiology of migraine.

Therapeutic implications

In the present case, metoprolol was given to prevent further episodes of ventricular tachycardia. Moreover, beta-blockers are commonly used in the prevention of migraine attacks. However, beta-blockers are known to decrease cerebral blood flow, cerebral glucose metabolism as well as cerebral oxygen consumption, and increase cerebral vascular resistance [Bardwell and Trott, 1987]. In this context, there is anecdotal evidence that propranolol may lead to cerebral ischaemia in patients with migraine attacks [Bardwell and Trott, 1987].

Thus, we discussed verapamil as an effective alternative drug in preventing ventricular tachycardia, especially since it appears to be effective for patients with basilar migraine [Evans and Linder, 2002]. On the other hand, verapamil does not affect ventricular tachycardia caused by re-entry and catecholamine-sensitive automaticity, but is only effective in suppressing ventricular tachycardia caused by triggered activity related to delayed afterdepolarizations [Sung *et al.* 1983].

Conclusion

To the best of our knowledge, we report the first case of documented ventricular tachycardia during basilar-type migraine attack. The mechanisms of this phenomenon could be the sympathetic dysregulation during a migraine attack, which has been extensively analysed in the literature. On the other hand, the unique type of basilar-migraine attacks involving the brainstem and especially the pons with the noradrenaline-producing locus coeruleus, the serotonin-producing dorsal raphe nucleus, point more to a direct dysfunction of these structures during migraine attacks. More research is needed to define the exact pathogenesis of cardiac arrhythmias in basilar migraine.

Our observation indicates that cardiac monitoring of further patients with basilar-type migraine could reveal interesting accompanying features of this type of migraine such as ventricular

tachycardia. Although the risk of sudden death does not appear to be increased in people with migraine [Melek *et al.* 2007], it could well be that in basilar-type migraine, ictal cardiac dysfunction is more severe and should thus be studied in more detail. Furthermore, diagnosis of basilar migraine and the risk of cardiac arrhythmias undoubtedly have therapeutic consequences in the choice of indicated therapeutic substances. For example, concerns are raised regarding the use of tricyclic antidepressants in basilar migraine, which due to their antimuscarinic properties can induce cardiac arrhythmias, and should theoretically be avoided as prophylactic medications in basilar-type migraine.

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Conflict of interest statement

The authors declare no conflicts of interest in preparing this article.

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