Idiopathic Outflow Tract Tachycardias

Current Perspectives

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Abstract
Outflow tract ventricular tachycardias (OT-VTs) are the most common form of idiopathic VTs. In > 80–90% of cases OT-VT originates from the right ventricular outflow tract, however, other origins like the septum, the left ventricular outflow tract, the pulmonary artery, the aortic sinus of Valsalva, the area near the His bundle, and the epicardial surface of the ventricles have been described. OT-VT is a diagnosis by exclusion, i.e., the possible concomitant structural heart disease should be adequately explored and ruled out in all patients presenting with OT-VT. Ablation should be recommended for all patients who present with syncope and who also remain symptomatic despite optimal medical therapy with a well-tolerated drug.

Idiopathische Ausflusstrakttachykardien. Aktuelle Perspektiven

Zusammenfassung
Ventrikuläre Ausflusstrakttachykardien (OT-VTs) sind die häufigste Form der idiopathischen VTs. Obwohl sie normalerweise im rechten Ausflusstrakt entstehen, sind auch OT-VTs aus dem linken Ausflusstrakt, der Pulmonalarterie, den Aortenästen, der parahisären Region und dem epikardialen Bereich beschrieben worden. Der Ausschluss struktureller Herzerkrankungen, insbesondere einer arrhythmogenen rechtsventrikulären Kardiomyopathie, vor Diagnose einer idiopathischen OT-VT ist äußerst wichtig. Die individuelle Therapiestrategie beinhaltet die antiarrhythmische Pharmakotherapie und die Katheterablation. In dieser Übersicht werden die aktuellen pathophysiologischen Grundlagen, diagnostische und therapeutische Konzepte sowie Daten zur Prognose für die rechtsventrikuläre OT-VT zusammengefasst.

Key Words:
Idiopathic outflow tract tachycardia · Sudden cardiac death · Catheter ablation · Arrhythmogenic right ventricular cardiomyopathy

Introduction
Outflow tract ventricular tachycardias (OT-VTs) are the most common form of idiopathic VTs which accounts for nearly 10% of all patients referred for evaluation of VT [1–3]. In > 80–90% of cases OT-VT originates from the right ventricular outflow tract, however, other origins like the septum, the left ventricular outflow tract, the pulmonary artery, the aortic sinus of Valsalva, the area near the His bundle, and the epicardial surface of the ventricles have been described [4–12]. Although OT-VT usually happens in patients with no concomitant structural heart disease [13], recent studies have debated this issue [14–16]. This review intended to summarize the current available data on OT-VT and its management.

Clinical Presentation of OT-VT
OT-VT occurs more frequently in women [17]. Clinical presentation of OT-VT is variable and symptoms usually occur between the age of 20 and 50 years [2]. Two clinical manifestations of OT-VT are more frequent, exercise- (stress-)induced VT and repetitive monomorphic VT at rest, and both forms are characterized by adenosine sensitivity. Nonsustained VT which usually occurs as repetitive salvos of monomorphic VT is frequent, comprising 60–92% of reported series. Occasionally, VT is incessant. Less commonly, patients present with paroxysmal sustained tachycardia, separated by relatively long intervals of infrequent premature ventricular beats [18]. Episodes tend to increase in frequency and duration during exercise and emotional stress. In > 80% of patients, the origin of tachycardia is in the right outflow tract. Most of the patients with OT-VT show a benign course suggesting that this arrhythmia does not represent an occult cardiomyopathy and, in general, is not accompanied by hemodynamic deterioration. Right outflow tract is also a site of origin of ventricular fibrillation (VF) in patients with Brugada syndrome, catecholaminergic polymorphic VT and idiopathic VF [19–21]. Distinguishing these patients from patients with OT-VT is usually not difficult as the former present with syncope or cardiac arrest and have rare ventricular premature beats with a short coupling interval falling on the peak of the T wave, while
the latter present with palpitations and have frequent premature ventricular beats with a long coupling interval [21, 22]. Viskin et al. presented three patients with typical “benign right outflow tract ectopy” who went on to develop spontaneous VF or polymorphic VT [21]. However, the coupling interval of the premature ventricular beats offered clues to their malignant nature as it was shorter than that of truly benign monomorphic OT-VT [21, 22]. Noda et al. reported the largest series of patients with “malignant” polymorphic OT-VT [20]. The authors compared 16 patients with “malignant” OT-VT and 85 patients with monomorphic “benign” OT-VT [22]. In their series neither the number nor the coupling interval of the premature ventricular beats distinguished patients with malignant (polymorphic) from those with truly benign (monomorphic) right OT-VT [22]. Therefore, radiofrequency ablation should be strongly considered for the following patients with a potentially malignant form of OT-VT: (1) a history of syncope; (2) very fast VT (ventricular rates > 230 bpm, which can be associated with polymorphic VT); and (3) ventricular premature beats with short coupling interval, although the absence of short coupling intervals is no guarantee against polymorphic malignant OT-VT. Radiofrequency ablation applied to the site of arrhythmia origin in the right outflow tract was curative for most patients in both groups. Finally, the diagnosis of OT-VT is one of exclusion. In addition to the aforementioned diseases, the diagnosis of arrhythmogenic right ventricular dysplasia/cardiomyopathy should be strongly considered and ruled out in every patient with suspected OT-VT.

**Electrocardiographic Presentation**

The ECG during sinus rhythm is usually normal in patients with OT-VT; however, nearly 10% of patients with OT-VT show complete or incomplete right bundle branch block [23].

The QRS morphology during OT-VT is usually left bundle branch block configuration with an inferior (right or left) axis (Figure 1). The QRS duration is usually < 140 ms, if it originates from the right outflow tract septal region [23]. QRS morphology can vary slightly during tachycardia, which is usually associated with minor variations in the local electrograms near the site of origin of the tachycardia, however, multiple distinct VTs are very rare, and should raise the suspicion of occult underlying structural heart disease or other tachycardia mechanisms [24].

Often, the QRS morphology of the VT is the first clue to the possible site of successful ablation. There have been several reports on the application of twelve-lead ECG findings for localization of OT-VT [5, 7, 25–28]. Table 1 summarizes the findings of these studies.

An exercise test can induce clinical arrhythmia in 25–50% of the patients; in patients with repetitive monomorphic VT, however, the exercise test usually suppresses the arrhythmia. VT can happen either during the exercise or recovery phases of the stress test [23]. Limited studies on the heart rate variability in patients with OT-VT suggest that activation of sympathetic tone plays an important role in the occurrence of VT originating from the outflow tract [29, 30].

Finally, Ainsworth et al. have recently proposed an ECG algorithm for differentiating OT-VT from arrhythmogenic right ventricular cardiomyopathy. The authors showed that lead I QRS duration ≥ 120 ms had a sensitivity of 100%, a specificity of 46%, a positive predictive value of 61%, and a negative predictive value of 100% for arrhythmogenic right ventricular cardiomyopathy. The addition of R < S in lead III to the above criterion increased the specific-