Monomorphic Ventricular Tachycardia (M-VT)

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M-VT-RVOT: (Monomorphic Ventricular Tachycardia arising from Right Ventricular Outflow Tract): VT with LBBB morphology because its origin is in the RV. If it is originated in the RVOT, the SÂQRS is generally deviated to the right between + 90° and +120 ° (QRS of the "qR" or "QS" type in DI).

M-VT-RVIT: (Monomorphic Ventricular Tachycardia arising from Right Ventricular Inflow Tract):

LBBB morphology and SAQRS to the left. The focus is located in the RVIT, the apex or the inferior wall of the RV. A VT with LBBB morphology and SÂQRS to the left, nearly always suggests structural heart disease.

The principal conditions that need to be differentiated from ARVC/D, is Idiopathic Monomorphic Ventricular Tachycardia arising from Right Ventricular Outflow Tract (IMVT-RVOT)

ARVC/D is a genetically determined and progressive heart muscle disease or sporadic associated with arrhythmia, and SCD. The entity with time may lead to more diffuse RV involvement and LV abnormalities and culminate in CHF.

The IMVT-VT can be exactly the same, but there is no structural abnormality of the heart, unlike the situation in ARVC/D where commonly there is dilation of the ventricle.

IMVT-RVOT is more common than ARVC/D; and both observed in young, otherwise healthy people.

In ARVC/D the VT arise from right side of the heart. The regions preferentially involved include the RVIT, the RVOT, and the RV apex. It usually occurs in the diaphragmatic, apical, and infundibular regions known as “the triangle of dysplasia”. However, the LV free wall may be involved in some cases. Involvement of the ventricular septum is rare. The areas involved are prone to aneurysm formation.

IMVT- RVOT is more frequently that ARVC/D, has excellent prognosis and always T wave upright from V_2- V_5.

In ARVC/D the incidence of SCD is approximately 2.5% a year and negative T waves of V_1 to V_2 or V_3 are very characteristic when present in children over 12 years old in the absence of RBBB. T-wave inversions in V_1 through V_3 were observed in 85% of ARVC/D patients in the absence of RBBB compared with none in RVOT and normal controls, respectively. (**Nasir K, Bomma C, Tandri H, et al. Electrocardiographic features of arrhythmogenic right ventricular dysplasia/cardiomyopathy according to disease severity: a need to broaden diagnostic criteria. Circulation. 2004; 110:1527-1534.**).

In ARVC/D epsilon wave is present in 30% of cases. The R' wave may be mistaken or an epsilon wave. They correspond to late potentials that can be translated into RV delayed activation. Epsilon wave was present in 56.3% of patients with M-VT and LBBB, in 4.8% of patients with P-VT and in none of the patients with M-VT and RBBB. **(Makarov LM, Gorlitskaia OV, Kuryleva TA, et al. Prevalence of electrocardiographical signs of right ventricular arrhythmogenic dysplasia Kardiologiia. 2004; 44:23-28.).**

In 10% of IMVT it may originate in the LV, in the region of the postero-inferior division of left bundle with RBBB morphology and extreme deviation of SÂQRS to the left.

Another morphology that suggests the focus of origin in the LV, is LBBB associated to early transition in the V_2 lead.

Dominant R in V_1 and inferior SÂQRS points the focus of superior origin in the LV.

Rarely, it could have an epicardial origin, characterized by positive concordance in precordial leads and negative complexes in DI and aVL.

Both entities ARVC/D and IMVT-RVOT being significantly different in prognosis and treatment, follow-up monitoring is essential sometime to establish the definitive diagnosis (**Kuhn A, Kottkamp H, Thiele H, Idiopathic right ventricular tachycardia or arrhythmogenic right ventricular tachycardia? Dtsch Med Wochenschr. 2000; 125:692-677.**).