Too Much For a Single Heart : Case Report

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**Introduction** : Accessory pathway AP is the second most common substrate on young population and are more likely to have bidirectional conduction properties, 90 or 95% of the cases the anterograde conduction is over AV node -his purkinje tissue and retrograde conduction over AP “orthodromic AVRT”. but, in 5 to 10 % the anterograde conduction is over AP and retrograde conduction over AV node - his purkinje tissue “antidromic AVRT”. Idiopathic Fascicular Left Ventricular Tachycardia represents 10 to 15% of cases of idiopathic ventricular tachycardia. It was first described by Zipes et al. in 1979 and described a typical RBBB pattern with left axis deviation with relatively narrow QRS complex (120 - 140 ms).

**Methods** : Sometimes when we go into a EP study with a patient with a documented tachycardia, we preconceive some hypothesis about what the rhythm is, and what the mechanism might be. But, unexpected things sometimes happens. We can find a different tachycardia mechanism or even more, another unexpected arrhythmia substrate and at this point the question is, which is the clinical tachycardia? Should we ablate the other unexpected substrate too?. We present an atypical case of 35 years old male patient with history of recurrent palpitations, with normal structural heart, also normal CXR. The 12 lead ECG showed a regular wide complex tachycardia and on sinus rhythm a patent preexcitation.

**Result** : In Electrophysiology Laboratory an orthodromic AVRT was induced with successful ablation of AP and then after ablation, another tachycardia was induced but not related to AP with clear AV dissociation RBBB pattern and left axis deviation making the diagnosis of Idiopathic Fascicular Left Ventricular Tachycardia (ILFVT).

**Conclusion** : • Even when we have a substrate on resting tracing an AP , as in this case, we have to be sure that there is no other hidden potential substrate. In this case we can point out that symptoms is not specific to make a differentiate between supraventricular or ventricular tachycardia because the ventricular tachycardia did not show any hemodynamic compromise therefore not any clinical sign. • In this case the clinical tachycardia was the antidromic AVRT which was successfully ablated and the patient was discharged and programmed for long tern follow up looking for recurrence of LAFVT. • Always expect another bystander tachyarrhythmia in the same patient which became obvious on giving extra-stimulus. • We did not find any report about the association of AP with left fascicular ventricular tachycardia as a common pathophysiological bases or random association between the substrates.