CASE PRESENTATION – DILATED CARDIOMYOPATHY MAY BE SECONDARY TO SCAR POSTABLATION FOR PAROXYSMAL SUPRAVENTRICULAR TACHYCARDIA

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Abstract

Adolescent known with paroxysmal supraventricular tachycardia (SVPT) of approximately 5 years (electrophysiological study benefits from a slow track ablation), converted to sinus rhytm and variable antiarrythmic treatment, is hospitalized with signs and symptoms significant for severe congestive heart failure. ECG on admission shows SVPT, transthoracic echocardiography shows severely depressed contractile function, dilatation of both ventricules, chest radiography shows a global increase of the heart mostely due to the left ventricle increase. The pacient is diagnosed with arrythmic dilated cardiomyopathy - ADCM (possibly by reentry mecanism) and severe congestive heart failure, it was frequently investigated, currently under treatment with Amiodarona, reserved for long- term prognosis.

Keywords: cardiomyopathy, supraventricular tachycardia, heart failure.

Introduction

Any type of rhytm disorder SVPT with high frequencies and long action causes impaired myocardial contractile function and installing of DCM. Recovery capacity of the myocardium is directly proportional to the period of action of arrhythmia, if arrhythmic pathology is resolved early can lead to restitutio ad integrum of myocardial function, whereas the prolongation of action will lead invariably to irreversible DCM, even trough arrhythmic pathology will be treated. In this combination SVPT plus DCM, causality is hard to establishe, witch disease generated the other one. Some DCM (eg toxic, ereditary) are easily diagnosed and excluded, remains in question the association between viral myocarditis and DCMP.

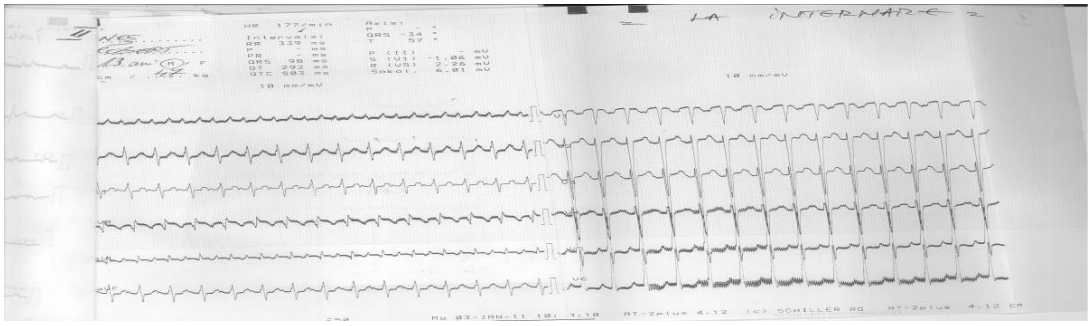
Case report

IR, 13,6 years, male admitted to emergency presenting extreme fatigue, palpitations, nausea/ vomiting - occured during the examination, moderate dyspnea, jugular turgidity- pulse jugular, hepatic-splenomegaly, pulse and weight subxifoidiene/ abdominal pain, stetacustic gallop rhytm, with the presence zg 3 and 4, systolic gr III/VI murmur- irradiated in axilla and subxifoidian, biological samples- normal. Family history is non significant for the underlying disease.

At age 8 the patient was diagnosed with SVPT (tachycardia type intranodal cleave to slow-fast) and frequency of 200-240bpm, with hemodynamic deterioration and acute heart failure. Electrophysiological study was performed and radiofrecquency ablation of the slow pathway, the patient is subsenquently converted to sinus rhytm and put under home observation in good clinical condition. Postablation the patient followed antiarrhytmic theraphy with amiodarone, propafenone, beta-blocker in variable dose. The last treatment was Rythmonorm, 3x 75mg (propafenona cp 150mg).

ECG on admission revealed sinus rhytm, regular, AV= 180bpm, QRS complex thin normal driving routes, supraventricular tachycardia (fig1).

Fig.1



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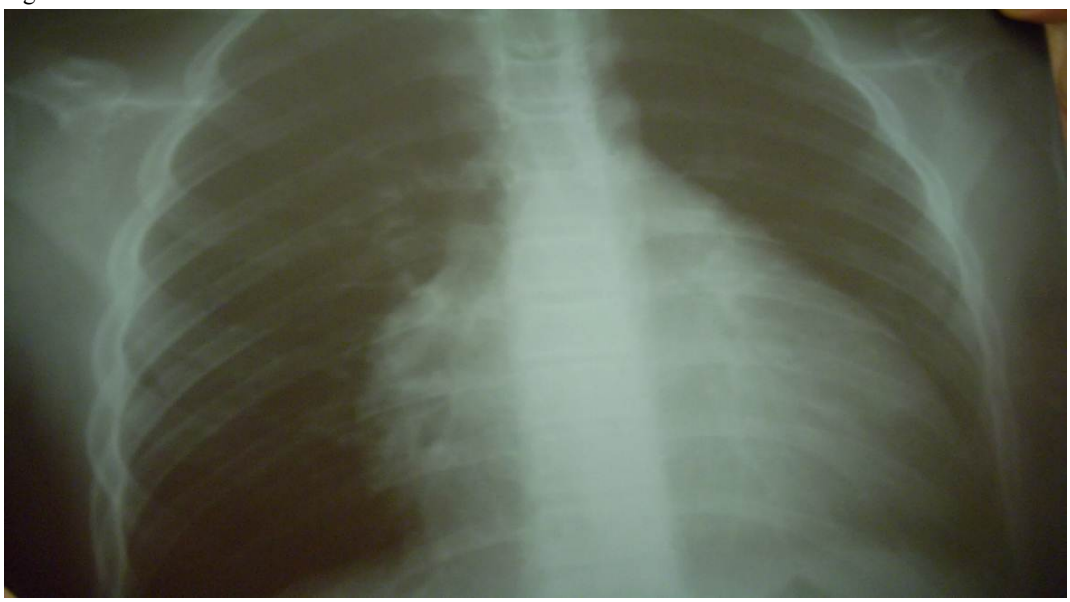
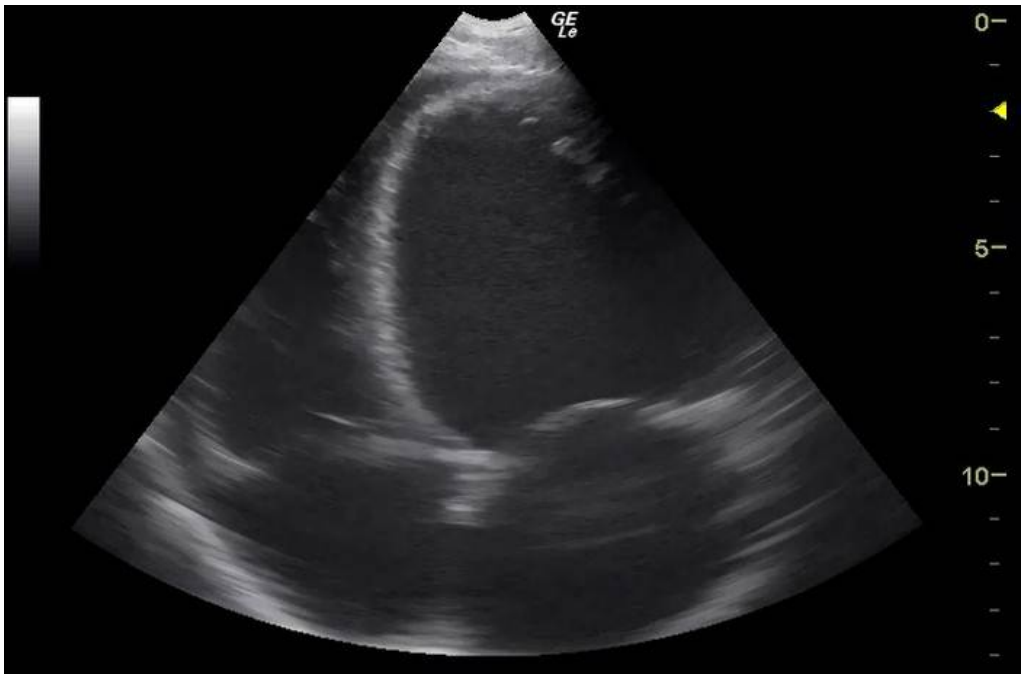
Chest X-ray bachground of asthenic chest, relatively normal lung, moderate congestive and increased overall hart dimensiones, ICT=0.7, in contrast with narrow vascular pedicle, cardiomegaly is global, but is predominantly due to left ventricular increase.(fig2)

Fig. 2



Trasthoracic echocardiography on admission revealed severely depressed contractile function aspect, marked dilatation of both ventricules; DTDvd=79mm, DTDvd=56mm, EF=35%, hipocontractilithy, septal akinesia. Cord arrhythmic pulse wave, aortic flow and pulmonary flow with small aspect, the blocking period, the contraction following the pause will slightly increase the flow extrasystole, Ao=29mm to the ring, Ap=27mm to the ring, I/II degree tricuspid insufficiency (E=0.72m/s, A=0.44m/s), I degree aortic insufficiency (Vmax=0,77m/s, Pmax=2,40mmHg), III degree miral insufficiency (E=0,74m/s, A=0.58m/s), wall motion - global hypokinesia (fig 3).

Fig. 3



Emergency clinical diagnosis: paroxysmal supraventricular tachycardia- possible reantry mecanism trough re-scaring on radio frequency ablation and severe congestive heart failure NYHA III/IV.

The recommended initial regimen was Rithmonorm 3x75mg/day with Carvedilol 12,5mg- then 18.75mg/day. Following the introduction beta-blocker decreased heart rate (160-140-90bpm)(fig. 4), then converting to sinus rhythm temporarily (fig.5).

Fig. 4

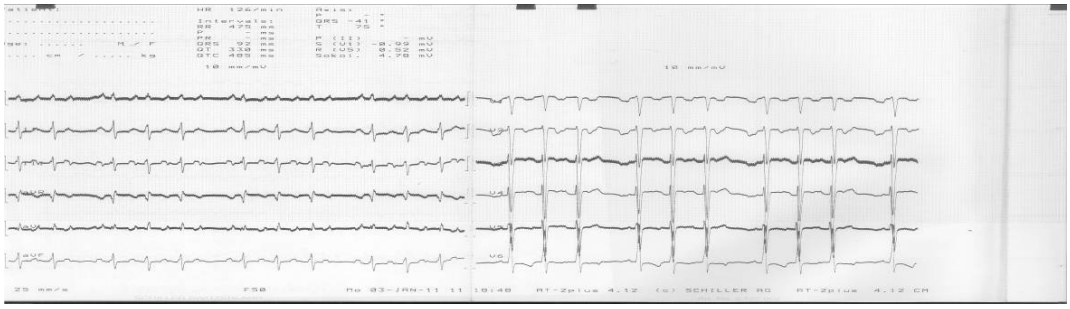
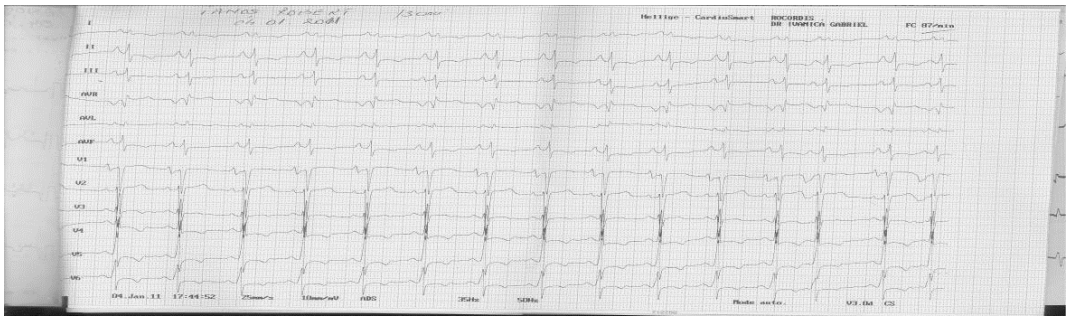


Fig. 5



Following specialist consultans at arrhythmology departement, Rithmonorm discontinue (because the negative inotropic effect) and introduce treatement with Cordarone (antiarrhythmic drugs with the lowest negative inotropic effect) in 15mg/kg/day (loading dose). After changing various antiarrhythmic drugs the patient has varaible arrhythmias: jonctional extrasystole (fig 6), 2 degree Mobitz 2 AV block, jonctional trigeminism, and good clinical condition.

Fig. 6

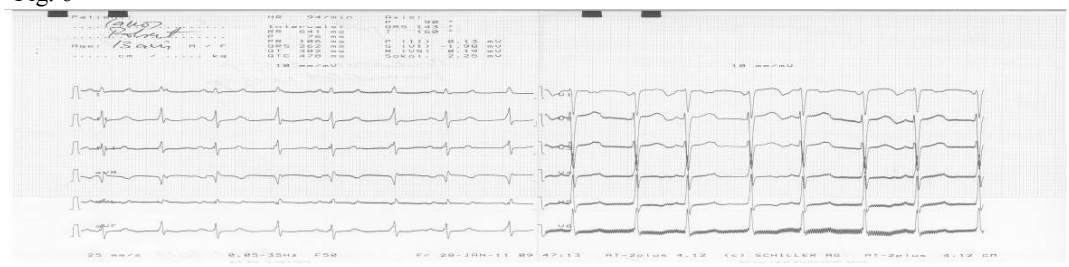
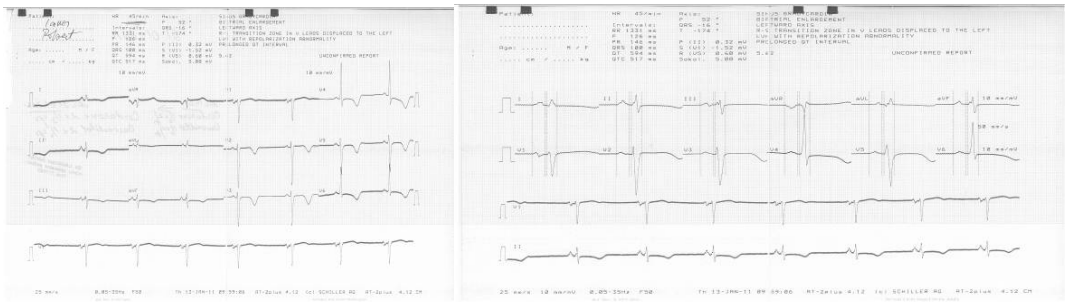


Fig. 7



The dose of Cordarone 3x1cp/day→AV= 45bpm (fig 7), it was decided to reduce the 2x1cp antiarrhythmic drugs Cordarone/day (Cordarone 1cp=200mg). The patient is currently treated with Cordarone 2x1/2cp/day and Carvedilol 2x1/4cp/day, is in sinus rhytm, AV=70-80bpm, with AV jonctional bigeminism, clinical status improved. Echocardiographic parameters improved, contractile function reversed, diastolic function improved, DTDvs decreased from 70mm to 59mm, EF increased from 35 to55%, mitral regurgitation decreased.

In this case the differential diagnosis involved other arrhythmias for TPSV and other DCM for ADCM. TPSV with other arrhythmias : atrial ectopic tachycardia, mutifocal atrial tachycardia, junctional ectopic tachycardia, atrial reentrant tachycardia, atrial fibrilation/atrial flutter, WPW, reentrant NAV tachycardia, reentrant accesory fascicle tachycardia, supraventricular ontodromic tachycardia, supraventricular antidromic tachycardia, permanent junctional tachycardia. The final diagnosis (witch diferentiates beteween diferent types of tachycardia) is made using the ECG and electrophysiological study. ADCM with other DCM : toxic DCM (chemotherapic agents, alcohol), viral infection DCM (CMV, HIV, richetioze), metabolic abnormalities DCM (selenium deficiency, thiamin deficiency, carnitine deficiency, hypothyroidism, thyrotoxicosis, diabetes, Cushing disease, hypocalcemia, hypophosphatemia), parasitic infections DCM (toxoplasmosis, trichinosis), inflammatory disease DCM – collagen disease, muscular diseases with DCM.

Evolution/ Aims

In this acute stage electrophysiological study was not indicated because of the fragility of the patient. The purpose of antiarrhythmic therapy in this situation is to improve hemodinamic performance before electrophysiological sudy is possible.

The electrophysiological study will identify the mecanism and will perform the ablation.

ADCM has an unpredictable evolution depending on the antiarrhythmic and cardiac insuficiecy treatment, possibly evolving towards gradual deterioration of contractile function and the need for cardiac transplantation.

The treatment of arrhythmia (in the electrophysiological study) consiste in the removal of scar tissue and the ablation of arrhythmia generating pathways. If, as a complication of extensive ablation, the pacient develope a total heart block, he will need permanent electrostimulation. If the myocardium has been compromised over time, with the installation of global heart failure, the patient may benefit resynchronisation therapy and electrophysiological study.

Drug treatment is long term, Cordarone is provisionally due to numerous side effects. After an electrophysiological study and ablation theraphy is performed, it is considered a prophylactic antiarrhythmic theraphy, the most likely drug used beeng Propafenona.

Prognosis reserved due to: the age on onset, the frequent TPSV, expansion and remodeling of the heart, postablation arrhythmia relapse, ADCM complications, congestive heart failure, mitral, tricuspidian and aortic insufficiency.

Along with other conditions that generate DCM, acute or cronic arrhythmias may cause compromised contractile function of the heart, leading to congestive heart failure, with progressive loss of contractile function and posible indication of cardiac transplantation.

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