Incessant idiopathic ventricular tachycardia in an infant : a case report

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ABSTRACT :

Incessant ventricular tachycardia is rare in childhood and usually difficult to treat. We report a case of incessant idiopathic ventricular tachycardia in a 9 month-old child complicated by tachycardia mediated cardiomyopathy. The arythmia was resistant to Vagal maneuvers, multiple doses of adenosine and also to electrical cardioversion. However, cardioversion was achieved with high dose of amiodaronne (800mg/m2sc/day). The heart has recovered a normal left ventricule function after 15 days of treatment. No recurrence of arrhythmia was observed during 4 years of follow up.

Keywords :

Ventricular tachycardia ; amiodaronne ; cardiomyopathy ; infant

INTRODUCTION :

Ventricular tachycardia (VT) is rare in infancy and childhood. Few small pediatric series focused on this arrhythmia have been reported in litterature. Roggen et al reported 27 cases of spontaneous VT in a pediatric population, detected among 252000 children aged <16 years, being a VT incidence of 1,1 episodes/100000 childhood years (1). Idiopathic VT is the most frequent form in the observed cases of pediatric VT and involving structurally normal hearts. Episodes of idiopathic VT are defined as sustained when lasting 30sec, otherwise, they are defined as nonsustained or incessant. The VT is usually paroxysmal but occasionally incessant in nature, and, thereby, may result in tachycardia mediated cardiomyopathy (TMC) (2); leading to ventricular dilation and systolic dysfunction with signs and symptoms of heart failure (3). Incessant idiopathic VT represents a severe form of VT which could result in collapsus and death and often difficult to manage in children.

We report an incessant idiopathic VT leading to TMC in a 9 month-old infant to highlight the difficulty of the diagnosis confirmation of this arythmia and of its management in pediatric population.

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OBSERVATION :

A nine month-old male baby admitted for acute dyspnea and poor feeding. He was a full term baby born by normal delivery with uneventful antenatal and perinatal periods; the birth body weight was 4300g. Upon admission, physical examination showed a normal grown child with a weight of 9Kg (+ 0,2 standard deviations (SD)) and length of 73 cm (+ 1 SD). The body temperature was at 37,5°C. The child was tachycardic (200/min), polypneic (50/min) with oxygen saturation in air at 96%, blood pressure at 100/75mmHg and no heart murmur. Abdominal palpation found an isolated hepatomegaly.

Chest X-ray revealed cardiomegaly (Cardio-thoracic ratio= 61%). Electro cardiogram (ECG) showed a wide QRS tachycardia (QRS width= 120ms) with right bundle branch block (RBBB) morphology (Figure 1) suggesting a diagnosis of VT rather than supraventricular tachycardia. Echocardiography revealed dilated left ventricle with severe depressed systolic function and mild mitral regurgitation. Because echocardiography was performed during tachycardia, calculation of an ejection fraction was difficult and thus didn't be done. The coronary arteries were normal, and no pericardial or pleural effusion was detected.

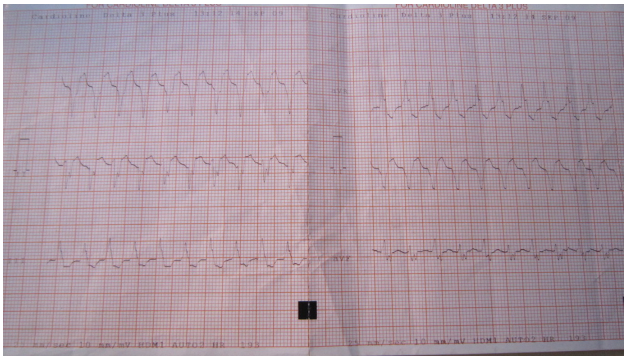

FIGURE 1 a :

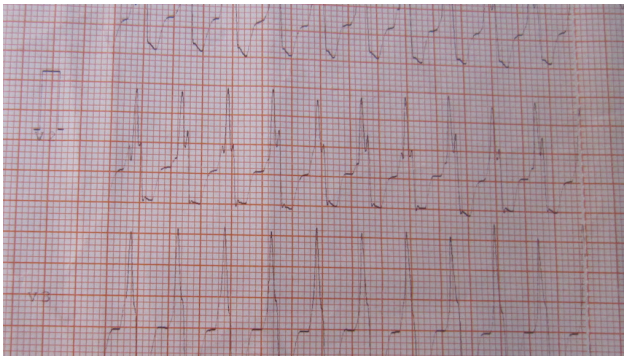

FIGURE 1 b :

FIGURE 1 :ECG showing wide complex tachycardia with QRS= 120ms, heart rate=200 beats per minute (figure 1a) and right bundle branch block morphology (figure 1b)

Biochemical analysis showed renal function with normal level: blood urea= 7mmol/L, Creatinine= 34 mol/L; ionogram with natremia= 136mmol/L and kaliema: 4,8mmol/L; Brain Nattriuretic peptid was increased at 2477pg/ml; Serum transaminases was at normal level (ASAT= 36/ ALAT= 43UI/L) and Creatine phosphokinase level was above 38UI/L. Troponin I was at 0,1g/L (normal < 0,04). The complete blood count showed severe anemia with hemoglobin=10,7g/dl, White blood cells= 7830/mm3, Platelets= 350000/mm3. Thyroid hormones were in normal levels with free thyroxine and thyroid stimulating hormone levels were 1,33 ng/dl (normal range: 0,70– 1,48ng/dl) and 1,9 UI/ml (normal range: 0,35– 4,94mU/l), respectively. The arythmia was resistant to Vagal maneuvers, multiple doses of adenosine and also to electrical cardioversion. We decided to treat the child with oral Amiodarone (500mg/m2sc/day). The dosage was then increased to 800mg/m2sc/day since there was no cardioversion of the tachycardia. With this new dosage we obtained cardioversion to a normal sinus rhythm after 7 days of treatment. ECG in sinus rhythm was normal with a corrected QT interval of 420 ms. Echocardiographic evaluation after 15th days of treatment showed complete reversal of left ventricular function with a left ventricle sizes and systolic function at normal ranges. No recurrence of arrhythmia was observed during 4 years of follow up.

DISCUSSION :

Our case illustrate a severe situation to manage. Firstble, it was difficult to differentiate between VT and supraventricular tachycardia, with aberrant conduction. In fact, VT is usually misdiagnosed as supraventricular tachycardia. Atrioventricular dissociation or sinus capture and fusion beats are characteristic of VT and could be usefull to differentiate between these twoo diagnosis. However, these signs occur in only 60% of cases (4). ECG assessment of VT can define the site of origin of the VT based on the bundle branch block morphology (5). Usually left bundle branch block morphology VT arises from the right ventricle and RBBB morphology VT arises from the left ventricle (5). In our patient, there was no atrioventricular dissociation on ECG, nor sinus capture and fusion beats. However, the QRS morphology in V1-V3 showed a RBBB pattern suggestive of VT arising from the left ventricule. Vagal maneuvers, adenosine, and cardioversion were attempted without success or slowing. This fact was a second argument for the VT diagnosis rather than supraventricular tachycardia.

Severals cardiac disease could be complicated by VT such as myocardial hamartomatous, myocarditis or a long QT interval (6). Dilated cardiomyopathy could be also cause VT as well as be the consequence of this arythmia. The distinction

between these twoo diagnosis is a challenge for the physician. The rapid improvement of left ventricular dysfunction after resolution of the arythmia confirmed the diagnosis of TMC in our patient. Several tachyarrhythmias have been reported to result in ventricular dysfunction especially supraventricular tachycardias. However, idiopathic incessant VT leading to a TMC is a very rare form of clinical presentation (7). Garson et al (6) reported 21 cases of incessant VT in infants. All of these children had no structural abnormalities found on the echocardiogram or angiocardiogram. Because of the continued tachycardia and the resistance to antiarrhythmic drugs, surgical treatment was performed for all patients. In 15 cases of them a tumor was found in biopsy: 13 myocardial hamartomas and 2 rhabdomyomas. Although, the myocardial biopsy could be normal, diagnosis of cardiac tumor could not be excluded definitely. The hamartomatous focus might be microscopic in size and missed by intra-operative biopsy. Thus, it seems that in infancy, incessant ventricular tachycardia may be due to a cardiac tumor, despite a normal echocardiogram (8) and in these situations, it will be recommended to complete investigations by either cardiac magnetic resonance imaging or positron emission tomography (6).

Incessant VTs are usually difficult to treat and refractory to conventional or investigational antiarrhythmic drugs (8). Early and agressive treatment is indicated ; otherwise it could result in cardiac arrest. In patients hemodynamically stable, like our patient, high dose of amiodarone (500 to 2000 mg/m2/24 hr) is the treatment of choice for VT in children, even by oral administration (9). Patiens with VT refractory to drugs could be treated by radiofrequency catheter ablation, or even invasive surgical procedures (8).

CONCLUSION :

Incessant idiopathic VT is a rare and severe arythmia which could be life-threatning in infancy. Its treatment is difficult. Our case showed that amiodaronne with high dose may have a good result. Patients presenting this arythmia should be followed up regularly in order to detect recurrence or apearence of cardiac tumor.

Conflicts of interest : none

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