Atrial Tachycardia from Enhanced Automaticity in Children: Diagnosis and Initial Management

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Abstract

Ten patients (aged 0-9 years) with the diagnosis of automatic atrial tachycardia (AAT) from August 1997 to August 2000 were reviewed. Three patients had paroxysmal (repetitive) AAT and the tachycardia was incessant in six (defined as presence of AAT for more than 90% of the time). The type of AAT in one patient was unknown. Four patients presented with congestive heart failure (CHF), one with pre-syncope, one with palpitation, and four were asymptomatic. Six patients (60%) had depressed left ventricular ejection fraction. All patients with CHF had incessant AAT with atrial rate > 220/min and ventricular rate > 200/min at admission. After treatment with antiarrhythmic medications, all patients had adequate control of the AAT (9 had complete elimination of AAT and 1 partial control). Amiodarone (alone, or in combination with digoxin) was effective in 5 of 6 cases (83%), although complete elimination of the AAT was usually delayed (median = 5 days, range 30 minutes to 17 days). Other effective medications were digoxin, digoxin + propranolol and atenolol (all in patients who did not have CHF on presentation). At the time of this report, 3 patients had no AAT off antiarrhythmic medication, 5 patients were still receiving treatment (with good control) and 2 patients died from sepsis during the same admission even though AAT was controlled. All surviving patients had normal ventricular ejection fraction on follow-up. AAT in children is rare, but when it occurs in persistent form at a fast rate, it is usually associated with CHF and is difficult to treat. Amiodarone (± digoxin) effectively controls the arrhythmia in the majority of cases, although full effect may take several days. With successful treatment, most patients do well and some can be taken off the medication(s) without recurrence of the arrhythmia.

Key word : Atrial Tachycardia, Ectopic Atrial Tachycardia, Automatic Atrial Tachycardia, Cardiac Arrhythmia, Children, Infants

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Atrial tachycardia (AT) is a relatively rare form of supraventricular tachycardia (SVT) in children. In one study, AT accounted for 14 per cent of children who presented with SVT[1]. AT can further be subdivided according to its mechanism into "reentry" and "non-reentry" types, the distinction of which is important in the management of these arrhythmias. Most non-reentry AT in children is probably caused by enhanced automaticity of atrial tissue[2]. Although AT from enhanced automaticity (automatic atrial tachycardia, AAT) is rare in children, it can be very difficult to control due to its refractoriness to treatment[2-7]. In this study, we reviewed our experience in the initial managements of AAT at Chulalongkorn Hospital.

Method

We collected information on all children (aged 0-14 years) seen at Chulalongkorn Hospital from August 1997 to August 2000 who had the diagnosis of automatic atrial tachycardia (AAT). AAT was defined by clinical criteria as previously described[3-6]. In short, the diagnosis was made by examining the 12-lead electrocardiogram (EKG) for the following characteristics: 1) narrow QRS tachycardia with distinctly identified P-wave with a rate inappropriate for age and activity, 2) "warming-up" and "cooling-down" phenomenon (gradual shortening or lengthening of tachycardia cycle length) in response to autonomic tone or activity, 3) variable rate and RR interval from minute to minute and hour to hour, 4) second-degree atrioventricular block (if present), and 5) unresponsiveness to appropriately done DC cardioversion (if performed). The demographic and clinical data of all patients were collected. The tachycardia was classified as persistent (Fig. 1A) if AAT was present continuously or more than 90 per cent of the time[1,3]. If AAT was present in less than 90 per cent but frequently recurred after interruption by sinus rhythm, it was classified as repetitive (Fig. 1B). Myocardial dysfunction was diagnosed when the ejection fraction, as determined by echocardiography, was less than 5 percentile for age. Congestive heart failure (CHF) from AAT was defined as the presence of pulmonary congestion and/or hepatomegaly associated with myocardial dysfunction. Treatment was considered fully effective if the AAT was suppressed completely. Partial effectiveness was defined when the treatment reduced the tachycardia rate, or caused enough atrioventricular block to result in improvement of symptoms and/or ventricular function. Data are expressed as mean ± SD unless noted otherwise.

RESULTS

Ten patients (6 male, 4 female, mean age 2.5 ± 3.2 years) were identified, 9 of whom were diagnosed for the first time during these 3 years. The other patient was diagnosed with "acute myocarditis" at the age of 8 months and subsequently had recurrent episodes of atrial tachycardia which recurred at the age of 6 years. The age of the patients ranged from intrapartum (28 weeks' gestation) to 9 years. At initial presentation, half of the patients had severe symptoms (congestive heart failure or presyncope). The others were either asymptomatic or had relatively mild palpitation. The presenting symptoms were associated with the types (incessant or repetitive) and rate of tachycardia. All patients who had severe symptoms had incessant tachycardia. Those with congestive heart failure were infants < 1 year old with incessant tachycardia with an atrial rate above 220/min, and ventricular rate above 200/min. Very fast atrial rates (400-500/min) were seen in two patients (patients #4 and #6) who had severe CHF and cardiogenic shock (Fig. 2). Patient #6 also had wide QRS tachycardia, which was probably ventricular tachycardia (Fig. 2, top strip). The patients who either had repetitive type of tachycardia or had a relatively slow (highest rate < 200/min) incessant

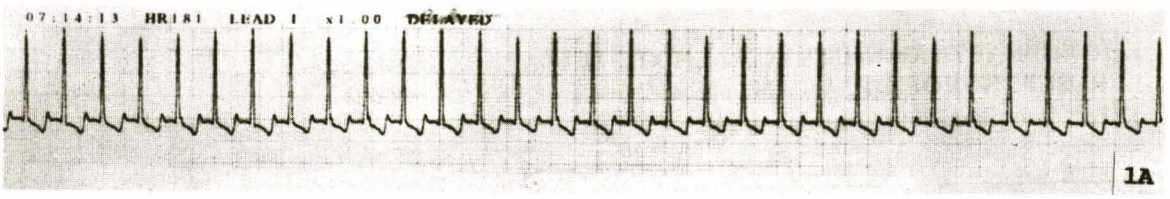

Fig. 1A. Incessant automatic atrial tachycardia in patient #7.

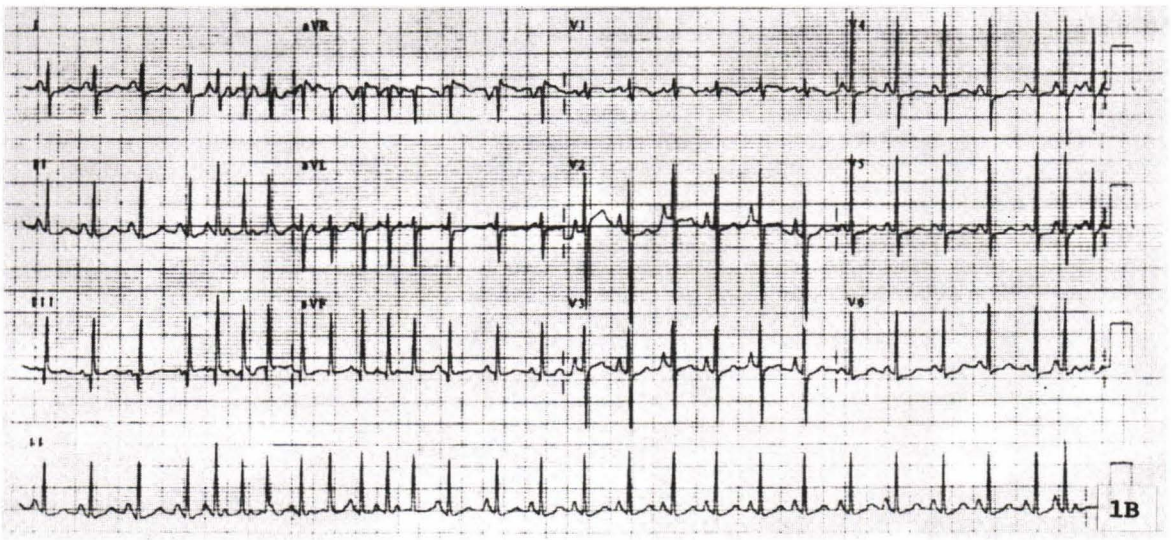

Fig. 1B. Repetitive bursts of automatic atrial tachycardia in patient #2.

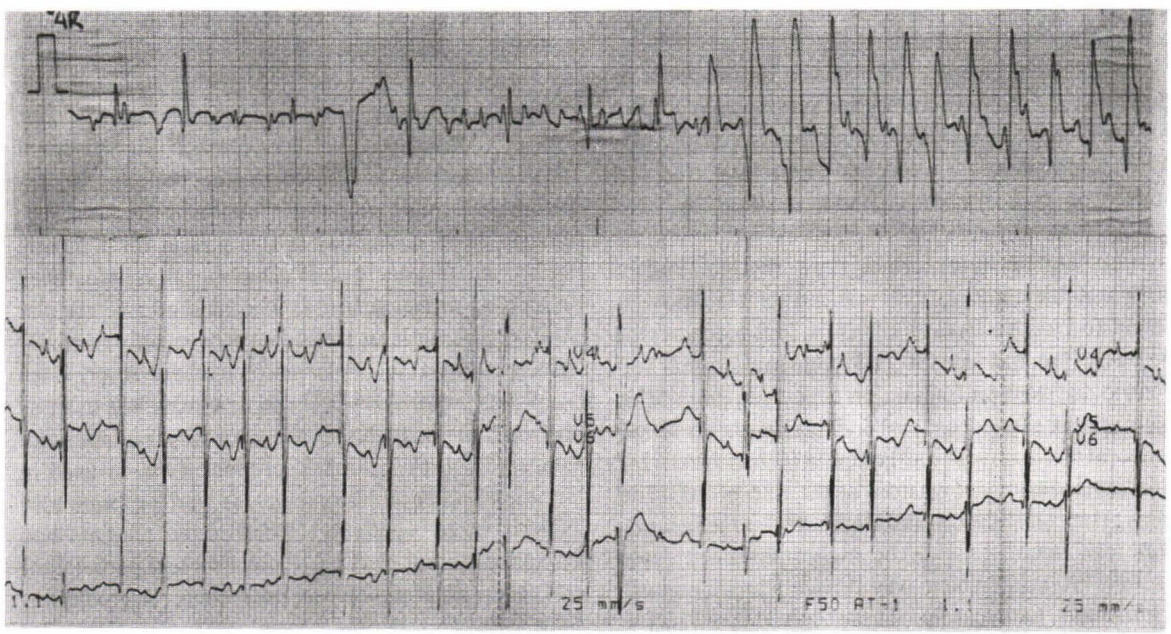

Fig. 2. Very fast (rate 400-500/min) automatic atrial tachycardia in patient #6 (top) and patient #4 (bottom).

atrial tachycardia did not have severe symptoms, although two of these patients (both had incessant tachycardia) had depressed or borderline-low ventricular function on echocardiogram. All patients had distinct P waves preceding QRS complex with PR interval less than RP (PR/RP<1). The P wave axes

Table 1. Clinical data of 10 patients with AAT.

Patients	Sex	Age	Mode of AAT	Presentation	EF %	Max rate Atr/Ven	P wave axis	Associated condition(s)
1.	F	*In utero*	inc	CHF/Hydrops	<30	300/300	+30	
2.	F	2 months	rep	asymptomatic	70	220/220	+30	
3.	M	3 years	rep	asymptomatic	N/A**	220/220	+30	PDA
4.	M	3 months	inc	CHF/shock	<30	450/200	-30	HLHS
5.	M	8 months	inc	CHF	N/A**	240/240	-30	Down/VSD
6.	F	6 months	inc	CHF/shock	<30	500/240	-30	CM, VT
7.	M	6 months	inc	asymptomatic	50	190/190	+30	VSD
8.	M	6 years	inc	pre-syncope	42	160/160	+60	
9.	F	9 years	N/A*	asymptomatic	55	160/160	+60	
10.	M	5 years	rep	palpitation	66	210/210	0-90***	

* = This patient was treated as an outpatient and Holter monitor was not done, so the type of AAT could not be classified.
** = echocardiogram before complete control of AAT was not available.
*** = P wave axis during AAT was similar to sinus rhythm on Holter monitor. AAT = automatic atrial tachycardia, Atr = atrial, CHF = congestive heart failure, CM = cardiomyopathy, Down = Down's syndrome, EF = ejection fraction, HLHS = hypoplastic left heart syndrome, inc = incessant, PDA = patent ductus arteriosus, rep = repetitive, Ven = ventricular, VSD = ventricular septal defect, VT = ventricular tachycardia

in 7 cases (70%) were between 0-90 degrees, suggesting high right atrial origins of the tachycardia. The other 3 patients had P wave axes of -30 degrees. The demographic and clinical data of these patients are summarized in Table 1.

Three patients were started on treatment as outpatients. One (patient#1) had paroxysmal attacks of AAT (Fig. 1B), which was observed at the age of 2 months during a well-child clinic visit. She was started on oral digoxin (without loading) and was followed as an outpatient. At 1-week follow-up, the rhythm had become normal sinus rhythm. Patient #9 was an asymptomatic 9 year-old female who was found to have tachycardia by a school teacher. She had AAT at the rate of 160 with low-normal left ventricular ejection fraction on echocardiogram. This patient was started on oral atenolol with no recurrence of the tachycardia. Patient #10 had brief (1-2 min) episodes of tachycardia at the rate of 200-210/min. After starting on oral digoxin, the tachycardia was less frequent, with the highest rate decreased to 180-190/min, which was difficult to distinguish from sinus tachycardia. These episodes of tachycardia were so infrequent (2-3/days) and brief (1-2 minutes each) that a decision was made not to add another antiarrhythmic medication to this patient.

Seven patients were admitted and treatments was started as in-patients: five patients who had severe symptoms (4 congestive heart failure, 1 pre-syncope), one asymptomatic 6-month-old infant who had mild ventricular dysfunction (ejection fraction = 50%), and a 3-year-old child who was already in the hospital after PDA ligation (this patient was asymptomatic). Amiodarone (alone, or in combination with digoxin) was the most common final antiarrhythmic drug chosen. It was completely effective in 5 of 6 times it was used (83%), although full effects could take several days (median 5 days, range 30 minutes - 17 days), and a high dose of amiodarone was necessary in one patient (#6) for loading (20 mg/kg/day) and maintenance (250 mg/m^2/day or 15 mg/kg/day in this patient for 1 month, then gradually decreased). Digoxin alone was tried initially in one patient (#7) who had mild ventricular dysfunction without CHF, but it was only partially effective. Because of its ineffectiveness in previous published reports[3,4,6,7], digoxin was not used as the sole antiarrhythmic agent in any patient with congestive heart failure. Beta blockers were used in 3 patients (atenolol alone in one and propranolol+digoxin in two patients). These three patients had complete control of AAT. Patient #8 had partial control when propranolol was used alone. Digoxin was later added in this patient resulting in complete control of the tachycardia. None of these three patients had congestive heart failure, although ventricular functions were depressed in two and low-normal in one. Thyroid function tests were done in 8 patients and they were all normal.

Overall effectiveness (complete + partial) of antiarrhythmic medication in acute treatment of automatic atrial tachycardia in our population was

Table 2. Results of management of patients with AAT.

Patients	Hosp	Medication		Results	Time to control	Current status
		Unsuccessful	Successful			
1.	Y	Dig*	Amio	Complete	5 days	Died from sepsis
2.	N		Dig	Complete	N/A**	NSR, off med (18 mo)
3.	Y		Amio	Complete	3 days	NSR, off med (8 mo)
4.	Y		Amio + Dig	Complete	17 days	NSR, off med (1 mo)
5.	Y		Amio	Complete	30 min	Died from sepsis
6.	Y		Amio + Dig	Complete	14 days	NSR, on med
7.	Y	Dig Dig + Amio	Dig + Prop	Complete	2 days	NSR, on med
8.	Y	Prop alone	Dig + Prop	Complete	4 days	NSR, on med
9.	N		Atenolol	Complete	N/A**	NSR, on med
10.	N		Dig	Partial	N/A**	NSR, on med

* = *In-utero* treatment. ** = Time to control not known because treatment was done as an outpatient.
AAT = automatic atrial tachycardia, Amio = amiodarone, Complete = complete control of AAT, Dig = digoxin, Hosp = hospitalization, NSR = normal sinus rhythm, on med = on medication(s), Partial = partial control of AAT, Prop = propranolol

100 per cent (all but one patient had complete elimination of their tachycardia). Despite complete control of atrial tachycardia, two patients died from sepsis. Both patients had fast AAT with congestive heart failure and required mechanical ventilation. At present, no patient has undergone electrophysiologic study or radiofrequency ablation. Three patients have had no medication for 1-18 months without recurrence of the tachycardia. Follow-up ventricular systolic functions were normal in all surviving patients. One patient (patient #6) had global ventricular hypertrophy, which was thought to represent a form of cardiomyopathy. The antiarrhythmic medication used for controlling these arrhythmias and the results of treatment are summarized in Table 2.

DISCUSSION

Although atrial tachycardia from enhanced automaticity (automatic atrial tachycardia, AAT; or ectopic atrial tachycardia) is rare in children, it is an arrhythmia that frequently causes morbidity and mortality. Two features make AAT a dangerous arrhythmia: 1) its tendency to be incessant, and 2) its refractoriness to treatment[2-7]. A fast AAT which occurs in persistent form often causes tachycardia-induced cardiomyopathy and congestive heart failure (CHF), especially in pre-verbal children who cannot communicate their symptoms to their parents. From other studies[2-7], CHF or left ventricular dysfunction was seen in 50-88 per cent of patients with AAT, which is similar to our findings. Diagnosis of AAT is sometimes difficult because the surface electrocardiogram often resembles sinus tachycardia, thus a high index of suspicion is needed to recognize AAT. Diagnosis can be done clinically by examining the 12-lead EKG, which can diagnose AAT in the majority of cases without resorting to intracardiac electrophysiologic study. Diagnosis is made on the basis of 1) atrial origin of tachycardia, 2) enhanced automaticity mechanism, and 3) ruling out sinus tachycardia (either by different P wave morphology, or by the inappropriately high atrial rate). In this and most previous studies[3,4,6], the P wave axis of AAT in children was between 0-90 degrees, the feature which makes it difficult to differentiate AAT from sinus tachycardia based on P wave morphology or axis alone. Because of its mechanism, AAT does not respond to DC cardioversion. Acute treatment of AAT usually starts with antiarrhythmic medication, followed by radiofrequency ablation if it is refractory to the treatment.

From other studies[2-4,6,7], medication controlled AAT in 25-100 per cent, with the median numbers of antiarrhythmic medication used = 3-3.7 [4,7]. The medications which were reported to be effective in these studies were amiodarone[3,4,6,7], class Ic agents[4,6,7], sotalol[7], i.v. propranolol[3] and amiodarone+class Ic agents[2]. In our experience, we were able to control AAT in all patients with medication, although multi-drug regimens were frequently used. Our management of AAT depended on the severity of the arrhythmia and its results on ventricular function. Because of its refractoriness

to treatment, all patients who had a persistent form of AAT, especially if associated with ventricular dysfunction and/or CHF were admitted to the hospital for treatment. In patients with congestive heart failure or severe myocardial dysfunction on echocardiogram, we usually started with i.v. amiodarone from the beginning because digoxin usually takes too much time for full effect and often is ineffective[3,4,6,7]. In our experience, i.v. amiodarone (alone or combined with digoxin) was fully effective in 83 per cent (5/6 patients) of patients, although full control of AAT could take several days and a high dosage might be needed (one of our patients required a loading dose of up to 20 mg/kg/day). Even though its full effect was delayed, amiodarone (± digoxin) could be beneficial from the first several hours if ventricular rate was reduced (by causing more AV block or slowing the atrial rate, or both). When used with amiodarone, digoxin dosage was reduced by 50 per cent because its clearance is reduced by amiodarone. One patient (patient #4) in our series who had severe myocardial dysfunction had a severe side effect from amiodarone (cardiac arrest) when the loading dose was inadvertently pushed intravenously (instead of intravenous drip). This patient was successfully resuscitated but nevertheless demonstrated a potential hazard when giving a bolus dose of amiodarone to a patient with severe ventricular dysfunction. Intravenous procainamide is another antiarrhythmic drug which can be used to treat children with AAT and severe CHF, but it is unavailable in most hospitals in Thailand at present.

In patients who had no or mild ventricular dysfunction without congestive heart failure, we usually started with digoxin. If no ventricular dysfunction was present, beta blockers such as atenolol or propranolol could also be used as first line agents. In this situation, digoxin or beta blocker alone or a combination of both was effective in majority of cases (4/5 cases had complete control, and 1/5 partial control in our series). From previous published reports, digoxin was often ineffective and the results of beta blockers (or its combination with digoxin) were variable[3,4,6,7]. However, we think this strategy is reasonable in patients who are stable, in order to avoid potential long term side effects of amiodarone or potential proarrhythmic side effects of class Ia, Ic, or other class III antiarrhythmic medications. We do not have any experience in using class IV agents (calcium channel blockers) in any of our patients.

Radiofrequency (RF) ablation has been successfully used in children and adults with automatic atrial tachycardia with good results[8-10]. We did not need RF ablation for acute control of AAT in any of our patients, although it will be a good alternative to medication in our older patients who may have recurrence of AAT after discontinuation of antiarrhythmic drugs. In infants and small children who are well-controlled on medication without serious side effects, we still recommend using antiarrhythmic agents over RF ablation because of potentially higher complications of RF ablation in small children[11,12], and a high incidence of spontaneous remission[5,7] in this age group.

SUMMARY

Automatic Atrial tachycardia (AAT) in children is rare, but when it occurs in persistent form at a fast rate, it is usually associated with CHF and is difficult to treat. Antiarrhythmic medication was used to control AAT in the majority of cases, although combinations of medication were frequently needed. In severe cases, amiodarone (± digoxin) effectively controlled the arrhythmia in the majority of cases it although complete control could take several days. With successful treatment, most patients did well and some could be taken off antiarrhythmic medication without recurrence of the arrhythmia.

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Atrial tachycardia จาก Enhance automaticity ในเด็ก: การวินิจฉัย และการรักษาเบื้องต้น

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ผู้รายงานได้รายงานผู้ป่วยเด็ก 10 ราย (อายุ 0 – 9 ปี) ซึ่งได้รับการวินิจฉัยเป็น automatic atrial tachycardia (AAT) ที่หน่วยโรคหัวใจเด็ก โรงพยาบาลจุฬาลงกรณ์ระหว่างสิงหาคม 2540 – สิงหาคม 2543 ผู้ป่วย 3 คนเป็น AAT แบบ repetitive, 6 คนเป็นแบบ persistent (มี rhythm เป็น AAT มากกว่า 90%) ส่วนชนิดของ AAT ในผู้ป่วยอีก 1 คนไม่ทราบแน่ชัด อาการของผู้ป่วยที่มาพบแพทย์ ได้แก่ หัวใจวาย (4 คน), pre-syncope (1 คน), ใจสั่น (1 คน) และไม่มีอาการ (4 คน) ผู้ป่วย 6 ราย (60%) มีการลดลงของ left ventricular ejection fraction ผู้ป่วยทุกคนที่มีอาการหัวใจวาย เป็น AAT ชนิด incessant ซึ่งมี atrial rate มากกว่า 220/min และ ventricular rate มากกว่า 200/min ผู้ป่วยทั้ง 10 คนได้รับการรักษาด้วยยาควบคุมการเต้นหัวใจผิดจังหวะ ซึ่งพบว่าได้ผลเป็นที่น่าพอใจ โดยสามารถหยุด AAT ได้ในผู้ป่วย 9 คน และควบคุม AAT ได้ดีในผู้ป่วยอีก 1 คน amiodarone (± digoxin) ใช้ได้ผลดีในผู้ป่วย 5 ใน 6 คน (83%) แต่อาจใช้เวลานานในการออกฤทธิ์ (median = 5 วัน, range 30 นาที – 17 วัน) ยาอื่นซึ่งได้ผลได้แก่ digoxin, digoxin ร่วมกับ propranolol และ atenolol (ยากลุ่ม beta blocker ใช้ในผู้ป่วยที่ไม่มีภาวะหัวใจวาย) ผู้ป่วย 3 คน สามารถหยุดยาโดยไม่มีการเป็นซ้ำ ผู้ป่วย 5 คน ยังคงได้รับยาควบคุมจังหวะการเต้นของหัวใจ และ ผู้ป่วยอีก 2 คนเสียชีวิตจากการติดเชื้อ แม้จังหวะการเต้นของหัวใจจะกลับเป็นปกติแล้วก็ตาม left ventricular ejection fraction ในผู้ป่วย 8 รายที่ไม่เสียชีวิต กลับเป็นปกติทุกรายภายหลังจากการรักษา

สรุป : AAT เป็นภาวะการเต้นผิดปกติของหัวใจที่พบไม่บ่อยในเด็ก แต่อาจทำให้เกิดภาวะหัวใจวาย และการควบคุมการเต้นของหัวใจให้กลับมาปกติอาจทำได้ยาก amiodarone เป็นยาซึ่งใช้ได้ผลดี แต่อาจใช้เวลานานในการออกฤทธิ์ ผู้ป่วยซึ่งการรักษาได้ผลดีจะมีการพยากรณ์โรคที่ดี และในบางรายอาจสามารถหยุดยาควบคุมการเต้นหัวใจผิดจังหวะได้ โดยไม่มีการกลับเป็นซ้ำของ AAT

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