ISSN: 2476-230X

Review Article

Cardiology: Open Access

Ectopic Atrial Tachycardia in Children: A Literature Review

Bula Y Dhinsa*

Cardiac Care Unit, Children's Hospital of Philadelphia, US

***Corresponding Author**
Bula Y Dhinsa, Cardiac Care Unit, Children's Hospital of Philadelphia, US.

Submitted: 2023, May 15; **Accepted:** 2023, Dec 20; **Published:** 2024, Jan 05

Citation: Dhinsa, B. Y. (2024). Ectopic Atrial Tachycardia in Children: A Literature Review. *Cardio Open, 9*(1), 01-04.

Abstract

Introduction: *Ectopic atrial tachycardia (EAT) is an unusual and potentially risky arrhythmia that can result in left ventricular dysfunction if not properly managed. In adults, EAT is mainly caused by diseased atrial myocardium and responds poorly to antiarrhythmic drugs. The characteristics of EAT in children might be different from those in adults because of their immature myocardium and the different electrophysiologic characteristics of their conduction tissue.*

Purpose: *To present the data on the natural history and treatment of EAT in children.*

Material and method: *The study material consisted of reviewed articles on the topic found on the globally accepted electronic databases, PubMed, Medline and Google Scholar regarding the Ectopic atrial tachycardia in children and its treatment.*

Results: *The diagnosis of atrial ectopic tachycardia is based on the presence of a narrow complex tachycardia (in the absence of aberrancy or pre-existing bundle branch block) with visible P waves at an inappropriately rapid rate. The rates range from 120 to 300 beats per minute (bpm) and are typically higher than 200 bpm, although physiologic rates may be observed. EAT in children without underlying heart disease can be effectively treated using antiarrhythmic drugs. Spontaneous resolution of EAT after medication in children was frequent (75%).*

Conclusion: *EAT in children without underlying heart disease can be effectively treated using antiarrhythmic drugs. The results of studies suggest that a stepwise approach using digoxin, a beta-blocker, and a class I antiarrhythmic drug may be the most effective treatment for EAT.*

Keywords: Ectopic Atrial Tachycardia, Treatment

1. Introduction

Ectopic atrial tachycardia (EAT) is an unusual and potentially risky arrhythmia that can result in left ventricular dysfunction if not properly managed. In adults, EAT is mainly caused by diseased atrial myocardium and responds poorly to antiarrhythmic drugs. The characteristics of EAT in children might be different from those in adults because of their immature myocardium and the different electrophysiologic characteristics of their conduction tissue [1].

Atrial ectopic tachycardia is the most common form of incessant supraventricular tachycardia (SVT) in children. Atrial ectopic tachycardia is believed to be secondary to increased automaticity of a no sinus atrial focus or foci. This arrhythmia, which is also known as ectopic atrial tachycardia or automatic atrial tachycardia, has a high association with tachycardia-induced cardiomyopathy. Atrial ectopic tachycardia is often refractory to medical therapy and is not usually responsive to direct current (DC) cardioversion [2]. Ectopic atrial tachycardia occurs at a rate of 5%–20% among children with supraventricular tachycardia. Atrial appendages are the most frequent focus in children, differing from the situation in adults. However, ectopic atrial tachycardia originating from aneurysms of the right atrial appendage is very rare and in fact has not yet been clearly defined [3]. The causes and significance of postoperative ectopic atrial tachycardia (EAT) remain unknown [4].

Atrial ectopic tachycardia is predominantly observed in infants and children; this account for a peak of 11-16% of tachycardia is for which a mechanism is determined in young childhood [2]. The diagnosis of atrial ectopic tachycardia is based on the presence of a narrow complex tachycardia (in the absence of aberrancy or pre-existing bundle branch block) with visible P waves at an inappropriately rapid rate. The rates range from 120 to 300 beats per minute (bpm) and are typically higher than 200 bpm, although physiologic rates may be observed [2].

Atrial ectopic tachycardia is generally well tolerated. Syncope is unusual, and cardiac arrest is rare, except when encountered as a complication of treatment. Tachycardia-induced cardiomyopathy is the most significant sequela of atrial ectopic tachycardia and may be insidious. The time to development depends on the rate

and duration of the tachycardia; however, ventricular dilatation may be present on initial presentation. This can also be reversed with successful treatment of the arrhythmia [2]. EAT in children without underlying heart disease can be effectively treated using antiarrhythmic drugs. Spontaneous resolution of EAT after medication in children was frequent (75%) in this series [1]. The purpose of this paper is to present all the data on the natural history and treatment of Ectopic Atrial Tachycardia in children.

2. Materials and Methods

The study material consisted of reviewed articles on the topic found on the globally accepted electronic databases, PubMed, Medline, Google Scholar, regarding the Ectopic atrial tachycardia in children and its treatment. The key words used were ectopic atrial tachycardia in children and its treatment. The study material consisted of scientific books, reviews and research papers published online.

2.1. Diagnosis of Ectopic Atrial Tachycardia

Although atrial ectopic tachycardia (AET) is occasionally encountered in patients following surgery for congenital heart disease, most patients have structurally normal hearts and are symptomatic. Palpitations, chest pain, light-headedness, presyncope, and dyspnoea are the most common symptoms. Asymptomatic or preverbal patients may be noted to be tachycardia or dyspnoeic on routine evaluation. Difficulty feeding or diaphoresis may accompany the tachycardia in infants. Exercise intolerance and heart failure are late manifestations of secondary cardiac dysfunction. The history must be sufficiently broad to rule out causes of persistently elevated heart rates, such as hyperthyroidism, anaemia, or catecholamine-producing malignancy. The family history is rarely positive for atrial ectopic tachycardia [2].

2.2. Physical Examination

The heart rate is inappropriately elevated for the degree of activity. If second-degree atrioventricular (AV) block is present, the heart rate may be irregular. The patient may be tachypneic. In advanced cardiomyopathy, pulses and perfusion are poor, and evidence of cardiac enlargement is present. Hepatic and pulmonary congestion may be present [2]. Ectopic atrial tachycardia usually differs from the P-wave morphology in sinus rhythm although it can be difficult to differentiate atrial origins close to the sinus node. Adenosine can be diagnostic as it induces transient AV-block clearly showing the atrial tachycardia [5]. Atrioventricular block may be seen during EAT. EAT P wave morphology is distinctly different from the sinus P morphology and spontaneous tachycardia termination is usually with a QRS complex and not with AV block. EATs originating from the right atrial appendage or close to the sinus node may be indistinguishable from sinus tachycardia, particularly if there is no sinus P wave for comparison. In such cases pointed P waves with slight prolongation of the PR interval ought to raise suspicion for EAT. Distinctive from re-entrant SVT, vagal manoeuvres or adenosine do not permanently terminate EAT. Adenosine administration may be a useful diagnostic manoeuvre. By blocking the AV node, it may isolate the P waves from the QRS and T waves and it may slow down the EAT automaticity thereby slowing the ectopic atrial rate and on occasion slow enough to allow the normal sinus rate to appear with its P wave morphology. This may be useful for comparison purposes and diagnosis [6].

2.3. Etiology

Spontaneous depolarization is a phenomenon of automatic myocardium. The sinus node is usually the pacemaker of the heart because it has the most rapid spontaneous rate of firing. A small cluster of cells with abnormal automaticity is presumed to be responsible for atrial ectopic tachycardia. The conduction spreads from this cluster to the surrounding atrium and to the ventricles via the atrioventricular (AV) node. A conduction delay from atrium to ventricle often occurs, with most patients demonstrating first-degree AV block and some showing second-degree block. Because atrial ectopic tachycardia is often incessant, tachycardia-induced cardiomyopathy is commonly observed. Although the exact underlying mechanism of the development of cardiac dysfunction in the setting of chronic arrhythmias is unknown, numerous reports have documented improved cardiac function following ventricular rate control and treatment of the arrhythmia [2]. Atrial ectopic tachycardia is usually idiopathic. Occasionally, mycoplasma or viral infections, such as respiratory syncytial virus, may trigger this arrhythmia, although more complex atrial tachycardia's, such as chaotic atrial tachycardia, are more frequently found in this scenario. Atrial tumours have been reported to be associated with atrial ectopic tachycardia. Reports of familial cases with an autosomal dominant inheritance are present in the literature [7]. This arrhythmia is also observed in patients who have congenital heart disease and have undergone surgical treatment of this congenital heart disease [2]. The adult form of atrial ectopic tachycardia may have a different etiology and natural history than the paediatric form [2].

2.4. Prognosis

Atrial ectopic tachycardia is generally well tolerated. Syncope is unusual, and cardiac arrest is rare, except when encountered as a complication of treatment. Tachycardia-induced cardiomyopathy is the most significant sequela of atrial ectopic tachycardia and may be insidious. The time to development depends on the rate and duration of the tachycardia; however, ventricular dilatation may be present on initial presentation. This can also be reversed with successful treatment of the arrhythmia [2]. Several reports have documented the spontaneous remission of atrial ectopic tachycardia in the paediatric population and in young adults [8]. This may occur in as many as one third of patients following withdrawal of medication. A review from Texas Children's Hospital suggests that children younger than 3 years have a better response to medication and a higher rate of spontaneous resolution of the arrhythmia [2].

2.5. Treatment of EAT in Children

EAT in children without underlying heart disease can be effectively treated using antiarrhythmic drugs [1]. Younger children respond to antiarrhythmic therapy and have a high incidence of EAT resolution, thus warranting a trial of antiarrhythmic therapy [9].

2.5.1. Treatment of Acute AET

For patients who present in cardiac arrest or with hemodynamic compromise, establish the circulation, airway, and breathing, the CABs, as is standard; provide appropriate monitoring; make sure that a defibrillator is available; and attempt conversion with a defibrillator if necessary [2]. Patients with atrial ectopic tachycardia (AET) may present with circulatory collapse similar to patients with cardiomyopathy. Although these patients may benefit from afterload reduction and inotropy, primary therapy aimed at reversing their tachycardia is usually more successful. Immediate rate control is desired in the child who requires significant support, including intubation, in the intensive care unit (ICU). This can often be achieved without resorting to negatively inotropic antiarrhythmic agents. Digitalization and the use of intravenous (IV) amiodarone may quickly achieve rate control. An additional maneuver involves the use of atrial pacing (e.g., esophageal, transthoracic, transvenous) to overdrive the atrial tachycardia to a point of consistent 2:1 atrioventricular (AV) block, thus lowering the ventricular response rate. In the era of radiofrequency (RF) ablation, most patients who require this degree of support undergo an attempt at ablation of the focus, particularly if it is an incessant tachycardia. The use of inotropic agents such as epinephrine may increase the tachycardia rate and cause clinical deterioration [2].

2.5.2. Treatment of Chronic AET

Three options are available for treatment of patients with atrial ectopic tachycardia (AET), including medication to suppress the arrhythmia or control the ventricular response, surgery, or radiofrequency (RF) ablation [2]. Long-term oral medication is the mainstay of therapy in patients not undergoing RF ablation. Class IC and III antiarrhythmic agents are generally the most effective, and a staged approach is recommended. Medical therapy may be effective in as many as 75% of patients, but more than one medication is usually needed [2]. Radiofrequency (RF) ablation can be curative for atrial ectopic tachycardia and can be performed with a high degree of success, a low complication rate, and a low recurrence rate. Success rates range from 75-100%. The complication rates are similar to other RF ablation procedures, with a higher risk of recurrence. The encircling technique uses 2 catheters capable of delivering RF energy as mapping catheters, alternating the reference and roving catheters, until no site provides an earlier signal than the reference. This early reference catheter is then used to deliver ablation. Atrial angiography may occasionally be helpful as a roadmap during RF catheter ablation [2].

Noncontact mapping systems have gained an increasing role in the ablation of atrial ectopic tachycardia [10,11]. The ability to localize the focus, including a non-sustained focus, with accuracy is an advantage of this technique. A limitation in the paediatric population is the size of the equipment and duration of the procedures. Cummings et al reported better results using a 3-dimensional mapping system than with conventional mapping in a series of 16 patients who underwent ablation [12].

Drugs like beta-adrenoceptor blocking agents and amiodarone are effective in the control of ectopic atrial tachycardia in children [13]. In infants and young children, the anatomic substrate of EAT is different compared to older patients. It has been hypothesized that EAT in small individuals is based on abnormal embryonic cells with enhanced automaticity and is linked with a high risk for complications during catheter ablation due to the low body weight of these children. As spontaneous resolution of the tachycardia is more likely, initially medical treatment is usually preferred [5]. Spontaneous resolution of EAT after medication in children was frequent (75%) in this series [1].

In children ≥3 years, AET is unlikely to resolve spontaneously, and antiarrhythmic medications are frequently ineffective [9]. A combination therapy of two or more antiarrhythmic is needed in up to 50% of patients with beta-blocker, Amiodarone, Digoxin, Flecainide, and Sotalol being used. The combination of Ivabradine and beta-blocker was effective and did not cause bradycardia [5]. The combination of sotalol and propafenone performed well in controlling EAT in children [complete control in 35 (49.3%) of 71]. The mean time of conversion to sinus rhythm was 24 days, and the mean duration of therapy was 11 months in children with resolution [14]. Ivabradine, a novel heart rate–reducing agent, acts via selective inhibition of the funny current responsible for spontaneous depolarization of cardiac pacemaker cells. Ivabradine is currently FDA approved to reduce hospitalizations in adults with stable heart failure and is also commonly used for the treatment of inappropriate sinus tachycardia [15]. Ivabradine is a safe treatment option, but data on its use especially in younger children is very rare [5].

Surgical resection of an ectopic focus, cryoablation and surgical electrical ablation are recently described techniques in the management of ectopic atrial tachycardia. These invasive procedures appear to be an attractive alternative to long-term medical therapy. However, some problems with these surgical techniques need to be addressed. Before surgical ablation, invasive electrophysiologic mapping and intraoperative mapping are necessary to identify accurately the site of an arrhythmogenic focus. Automatic ectopic atrial tachycardia is sensitive to temperature changes and anaesthesia and unlike re-entrant tachycardia, may not be inducible electrically, making intraoperative mapping impossible [13].

2.6. Complications

Atrial ectopic tachycardia (AET) is one of the incessant tachycardia's, which may become associated with myocardial dysfunction if the average ventricular rate remains elevated over a long term [2].

2.7. Guidelines

The 2015 American College of Cardiology, American Heart Association, and the Heart Rhythm Society (ACC/AHA/HRS) joint guidelines for the management of supraventricular tachycardia note that while the guidelines are intended for adults (≥18 years of age) and offers no specific recommendations for paediatric patients, in some cases, the data from non-infant paediatric patients was reviewed and helped inform the guideline [16]. The guidelines state that pharmacological therapy is largely based on practice patterns because random controlled

trials (RCTs) of antiarrhythmic medications in children are lacking. Amiodarone, sotalol, propafenone, or flecainide can be used in infants. In older children, beta-blockers are used most often as the initial therapy. Flecainide is not used as a first-line medication in children because of the rare occurrence of adverse events. Catheter ablation can be successfully performed in children of all ages, with success rates comparable to those reported in adults [16].

3. Conclusion

EAT in children without underlying heart disease can be effectively treated using antiarrhythmic drugs. Spontaneous resolution of EAT after medication in children was frequent (75%) in this series. The results of this study suggest that a stepwise approach using digoxin, a beta-blocker, and a class I antiarrhythmic drug may be the most effective treatment for EAT [1]. Ectopic atrial tachycardia occurs at a rate of 5%–20% among children with supraventricular tachycardia and potentially risky arrhythmia that can result in left ventricular dysfunction if not properly managed [3].

Conflict of Interest

The author declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

References

1. Wang, J. N., Wu, J. M., Tsai, Y. C., & Lin, C. S. (2000). Ectopic atrial tachycardia in children. *Journal of the Formosan Medical Association, 99*(10), 766-770.
2. Shubhayan Sanatani, Pediatric Atrial Ectopic Tachycardia, *Medscape, Sept.*19, 2016
3. Kanaya, T., Nishigaki, K., Yoshida, Y., Nakamura, Y., Murakami, Y., & Suzuki, T. (2018). Ectopic atrial tachycardia originating from right atrial appendage aneurysms in children: three case reports. *HeartRhythm Case Reports, 4*(1), 2-5.
4. Rosales, A. M., Walsh, E. P., Wessel, D. L., & Triedman, J. K. (2001). Postoperative ectopic atrial tachycardia in children with congenital heart disease. *The American journal of cardiology, 88*(10), 1169-1172.
5. Michel, H., Heißenhuber, F., Wellmann, S., Melter, M., & Gerling, S. (2020). Ectopic atrial tachycardia in a 12-month-old girl treated with ivabradine and beta-blocker, a case report. *Frontiers in Pediatrics, 8*, 313.
6. Benatar, A., & Dewals, W. (2018). Incessant ectopic atrial tachycardia in the pediatric age: clinical presentation and therapeutic options. *Ann Cardiol Cardiovasc Med, 2*(2), 1016.
7. Dagres, N., Gutersohn, A., Wieneke, H., Sack, S., & Erbel, R. (2004). A new hereditary form of ectopic atrial tachycardia with autosomal dominant inheritance. *International journal of cardiology, 93*(2-3), 311-313.
8. Bauersfeld, U., Gow, R. M., Hamilton, R. M., & Izukawa, T. (1995). Treatment of atrial ectopic tachycardia in infants< 6 months old. *American heart journal, 129*(6), 1145-1148.
9. Salerno, J. C., Kertesz, N. J., Friedman, R. A., & Fenrich, A. L. (2004). Clinical course of atrial ectopic tachycardia is age-dependent: results and treatment in children< 3 or≥ 3 years of age. *Journal of the American College of Cardiology, 43*(3), 438-444.
10. Higa, S., Tai, C. T., Lin, Y. J., Liu, T. Y., Lee, P. C., Huang, J. L., ... & Chen, S. A. (2004). Focal atrial tachycardia: new insight from noncontact mapping and catheter ablation. *Circulation, 109*(1), 84-91.
11. Liew, R., Catanchin, A., Behr, E. R., & Ward, D. (2008). Use of non-contact mapping in the treatment of right atrial tachycardias in patients with and without congenital heart disease. *Europace, 10*(8), 972-981.
12. Cummings, R. M., Mahle, W. T., Strieper, M. J., Campbell, R. M., Costello, L., Balfour, V., ... & Frias, P. A. (2008). Outcomes following electroanatomic mapping and ablation for the treatment of ectopic atrial tachycardia in the pediatric population. *Pediatric Cardiology, 29*, 393-397.
13. Mehta, A. V., Sanchez, G. R., Sacks, E. J., Casta, A., Dunn, J. M., & Donner, R. M. (1988). Ectopic automatic atrial tachycardia in children: clinical characteristics, management and follow-up. *Journal of the American College of Cardiology, 11*(2), 379-385.
14. Ge, H., Li, X., Liu, H., & Jiang, H. (2017). Predictors of pharmacological therapy of ectopic atrial tachycardia in children. *Pediatric cardiology, 38,* 289-295.
15. Janson, C. M., Tan, R. B., Iyer, V. R., Vogel, R. L., Vetter, V. L., & Shah, M. J. (2019). Ivabradine for treatment of tachyarrhythmias in children and young adults. *HeartRhythm Case Reports, 5*(6), 333-337.
16. Page, R. L., Joglar, J. A., Caldwell, M. A., Calkins, H., Conti, J. B., Deal, B. J., ... & Al-Khatib, S. M. (2016). 2015 ACC/AHA/HRS guideline for the management of adult patients with supraventricular tachycardia: executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society. *Circulation, 133*(14), e471-e505.