Atrial Flutter: An Uncommon Pediatric Manifestation of Hyperthyroidism

ABSTRACT. Objective. Atrial flutter is an uncommon arrhythmia in the pediatric population except for in the immediate newborn period or following atrial repair of congenital heart disease. In children the diagnosis of atrial flutter may be difficult, attributable to rapid atrioventricular conduction and superimposition of flutter waves on QRS and T waves. Atrial flutter secondary to hyperthyroidism has been rarely reported in adults, but there are no reports of children presenting with atrial flutter as the initial manifestation of hyperthyroidism. 

Case Report. We report an interesting case of hyperthyroidism in a 3-year-old presenting with congestive heart failure and atrial flutter with 1:1 atrioventricular conduction. The responses to adenosine administration and to cardioversion were unusual and ultimately helpful in suggesting the diagnosis of hyperthyroidism.

Conclusion. When atrial flutter is encountered in a pediatric patient in whom there is 1:1 atrioventricular conduction, a lack of a response to adenosine, and persistent sinus tachycardia after cardioversion, the clinician should be alert to the possibility of thyrotoxicosis. 

PEDIATRICS 1997;100(2). URL: http://www.pediatrics.org/cgi/content/full/100/2/e11; atrial flutter, hyperthyroidism, pediatric, adenosine.

Atrial flutter is an uncommon arrhythmia in the pediatric population. It is principally encountered in the newborn period or after atrial repair of congenital heart disease. Although hyperthyroidism is clearly known to be a cause of atrial flutter in older adults, there are no reports of children presenting with atrial flutter as the initial manifestation of thyrotoxicosis.

When children present with atrial flutter, the diagnosis is often difficult, attributable to rapid atrioventricular conduction and superimposition of flutter waves on QRS and T waves. The presence of hyperthyroidism would be expected to further affect these relationships. We report here an interesting case of thyrotoxicosis in a 3-year-old presenting with atrial flutter in whom the response to adenosine and to cardioversion were unusual and ultimately helpful in suggesting the diagnosis of thyrotoxicosis.

CASE REPORT

A 3-year-old black girl presented with an increased heart rate noted for 9 hours and decreased activity. She had a similar episode of “fast heart rate” 2 days previously. She had no history of fever, heat intolerance, neck pain, significant weight loss or hoarseness, but was extremely hyperactive. Her past medical history was only remarkable for scarlet fever treated with amoxicillin 2 months before admission. The family history was remarkable for a maternal aunt with an overactive thyroid and another cousin with an overactive thyroid gland. All other viral and bacterial studies were negative.

Pathologic evaluation was remarkable for a diffusely enlarged thyroid gland weighing 6 g with histologic evidence of chronic lymphocytic thyroiditis and diffuse lymphocytic follicles, consistent with Hashimoto’s thyroiditis. Her postoperative course was complicated by transient hypocalcemia. Follow-up echocardiograms showed a left atrial diameter of 29 mm, atrial flutter may be difficult, attributable to rapid atrioventricular conduction, a lack of a response to adenosine, and persistent sinus tachycardia after cardioversion, the clinician should be alert to the possibility of thyrotoxicosis. Typically, 1:1 atrioventricular conduction is often difficult, attributable to rapid atrioventricular conduction and superimposition of flutter waves on QRS and T waves. Atrial flutter secondary to hyperthyroidism has been rarely reported in adults, but there are no reports of children presenting with atrial flutter as the initial manifestation of hyperthyroidism.

When children present with atrial flutter, the diagnosis is often difficult, attributable to rapid atrioventricular conduction and superimposition of flutter waves on QRS and T waves. The presence of hyperthyroidism would be expected to further affect these relationships. We report here an interesting case of thyrotoxicosis in a 3-year-old presenting with atrial flutter in whom the response to adenosine and to cardioversion were unusual and ultimately helpful in suggesting the diagnosis of thyrotoxicosis.

CASE REPORT

A 3-year-old black girl presented with an increased heart rate noted for 9 hours and decreased activity. She had a similar episode of “fast heart rate” 2 days previously. She had no history of fever, heat intolerance, neck pain, significant weight loss or hoarseness, but was extremely hyperactive. Her past medical history was only remarkable for scarlet fever treated with amoxicillin 2 months before admission. The family history was remarkable for a maternal aunt with an overactive thyroid and another cousin with an overactive thyroid gland. All other viral and bacterial studies were negative.

Pathologic evaluation was remarkable for a diffusely enlarged thyroid gland weighing 6 g with histologic evidence of chronic lymphocytic thyroiditis and diffuse lymphocytic follicles, consistent with Hashimoto’s thyroiditis. Her postoperative course was complicated by transient hypocalcemia. Follow-up echocardiograms showed a left atrial diameter of 29 mm, atrial flutter may be difficult, attributable to rapid atrioventricular conduction, a lack of a response to adenosine, and persistent sinus tachycardia after cardioversion, the clinician should be alert to the possibility of thyrotoxicosis. Typically, 1:1 atrioventricular conduction is often difficult, attributable to rapid atrioventricular conduction and superimposition of flutter waves on QRS and T waves. Atrial flutter secondary to hyperthyroidism has been rarely reported in adults, but there are no reports of children presenting with atrial flutter as the initial manifestation of hyperthyroidism.

When children present with atrial flutter, the diagnosis is often difficult, attributable to rapid atrioventricular conduction and superimposition of flutter waves on QRS and T waves. The presence of hyperthyroidism would be expected to further affect these relationships. We report here an interesting case of thyrotoxicosis in a 3-year-old presenting with atrial flutter in whom the response to adenosine and to cardioversion were unusual and ultimately helpful in suggesting the diagnosis of thyrotoxicosis.

CASE REPORT

A 3-year-old black girl presented with an increased heart rate noted for 9 hours and decreased activity. She had a similar episode of “fast heart rate” 2 days previously. She had no history of fever, heat intolerance, neck pain, significant weight loss or hoarseness, but was extremely hyperactive. Her past medical history was only remarkable for scarlet fever treated with amoxicillin 2 months before admission. The family history was remarkable for a maternal aunt with an overactive thyroid and another cousin with an overactive thyroid gland. All other viral and bacterial studies were negative.

Pathologic evaluation was remarkable for a diffusely enlarged thyroid gland weighing 6 g with histologic evidence of chronic lymphocytic thyroiditis and diffuse lymphocytic follicles, consistent with Hashimoto’s thyroiditis. Her postoperative course was complicated by transient hypocalcemia. Follow-up echocardiograms showed a left atrial diameter of 29 mm, atrial flutter may be difficult, attributable to rapid atrioventricular conduction, a lack of a response to adenosine, and persistent sinus tachycardia after cardioversion, the clinician should be alert to the possibility of thyrotoxicosis. Typically, 1:1 atrioventricular conduction is often difficult, attributable to rapid atrioventricular conduction and superimposition of flutter waves on QRS and T waves. Atrial flutter secondary to hyperthyroidism has been rarely reported in adults, but there are no reports of children presenting with atrial flutter as the initial manifestation of hyperthyroidism.

When children present with atrial flutter, the diagnosis is often difficult, attributable to rapid atrioventricular conduction and superimposition of flutter waves on QRS and T waves. The presence of hyperthyroidism would be expected to further affect these relationships. We report here an interesting case of thyrotoxicosis in a 3-year-old presenting with atrial flutter in whom the response to adenosine and to cardioversion were unusual and ultimately helpful in suggesting the diagnosis of thyrotoxicosis.

CASE REPORT

A 3-year-old black girl presented with an increased heart rate noted for 9 hours and decreased activity. She had a similar episode of “fast heart rate” 2 days previously. She had no history of fever, heat intolerance, neck pain, significant weight loss or hoarseness, but was extremely hyperactive. Her past medical history was only remarkable for scarlet fever treated with amoxicillin 2 months before admission. The family history was remarkable for a maternal aunt with an overactive thyroid and another cousin with an overactive thyroid gland. All other viral and bacterial studies were negative.

Pathologic evaluation was remarkable for a diffusely enlarged thyroid gland weighing 6 g with histologic evidence of chronic lymphocytic thyroiditis and diffuse lymphocytic follicles, consistent with Hashimoto’s thyroiditis. Her postoperative course was complicated by transient hypocalcemia. Follow-up echocardiograms showed a left atrial diameter of 29 mm, atrial flutter may be difficult, attributable to rapid atrioventricular conduction, a lack of a response to adenosine, and persistent sinus tachycardia after cardioversion, the clinician should be alert to the possibility of thyrotoxicosis. Typically, 1:1 atrioventricular conduction is often difficult, attributable to rapid atrioventricular conduction and superimposition of flutter waves on QRS and T waves. Atrial flutter secondary to hyperthyroidism has been rarely reported in adults, but there are no reports of children presenting with atrial flutter as the initial manifestation of hyperthyroidism.

When children present with atrial flutter, the diagnosis is often difficult, attributable to rapid atrioventricular conduction and superimposition of flutter waves on QRS and T waves. The presence of hyperthyroidism would be expected to further affect these relationships. We report here an interesting case of thyrotoxicosis in a 3-year-old presenting with atrial flutter in whom the response to adenosine and to cardioversion were unusual and ultimately helpful in suggesting the diagnosis of thyrotoxicosis.

CASE REPORT

A 3-year-old black girl presented with an increased heart rate noted for 9 hours and decreased activity. She had a similar episode of “fast heart rate” 2 days previously. She had no history of fever, heat intolerance, neck pain, significant weight loss or hoarseness, but was extremely hyperactive. Her past medical history was only remarkable for scarlet fever treated with amoxicillin 2 months before admission. The family history was remarkable for a maternal aunt with an overactive thyroid and another cousin with an overactive thyroid gland. All other viral and bacterial studies were negative.

Pathologic evaluation was remarkable for a diffusely enlarged thyroid gland weighing 6 g with histologic evidence of chronic lymphocytic thyroiditis and diffuse lymphocytic follicles, consistent with Hashimoto’s thyroiditis. Her postoperative course was complicated by transient hypocalcemia. Follow-up echocardiograms showed a left atrial diameter of 29 mm, atrial flutter may be difficult, attributable to rapid atrioventricular conduction, a lack of a response to adenosine, and persistent sinus tachycardia after cardioversion, the clinician should be alert to the possibility of thyrotoxicosis. Typically, 1:1 atrioventricular conduction is often difficult, attributable to rapid atrioventricular conduction and superimposition of flutter waves on QRS and T waves. Atrial flutter secondary to hyperthyroidism has been rarely reported in adults, but there are no reports of children presenting with atrial flutter as the initial manifestation of hyperthyroidism.

When children present with atrial flutter, the diagnosis is often difficult, attributable to rapid atrioventricular conduction and superimposition of flutter waves on QRS and T waves. The presence of hyperthyroidism would be expected to further affect these relationships. We report here an interesting case of thyrotoxicosis in a 3-year-old presenting with atrial flutter in whom the response to adenosine and to cardioversion were unusual and ultimately helpful in suggesting the diagnosis of thyrotoxicosis.

Received for publication Oct 31, 1996; accepted Feb 18, 1997.

Reprint requests to (W.A.S.) Pediatric Cardiology, RBC 380, Rainbow Babies and Children’s Hospital, 11100 Euclid Avenue, Cleveland, OH 44106–6011. PEDIATRICS (ISSN 0031–4005). Copyright © 1997 by the American Academy of Pediatrics.
ers the effect of hyperthyroidism on the heart and conducting tissue.

Atrial flutter as a manifestation of hyperthyroidism, to the best of our knowledge, has not been previously reported in a child. Previous studies evaluating children with hyperthyroidism have reported that sinus tachycardia, increased ECG voltages, left atrial hypertrophy, left ventricular hypertrophy, or combined ventricular hypertrophy are the most commonly seen electrocardiographic effects. Very rarely, atrial fibrillation, ventricular fibrillation, or complete atrioventricular block have also been documented. In the largest published study evaluating 380 children and young adults with atrial flutter, none were attributable to hyperthyroidism. From a different perspective, review of several series of thyrotoxicosis in childhood encompassing 279 patients failed to identify any with atrial flutter.

Serum thyroid hormone has direct and indirect effects on the heart. T3 and T4 both stimulate the Ca-ATPase of the myocardial sarcolemma resulting in decreased extracellular calcium, accelerated diastolic relaxation, and enhanced sinoatrial automaticity. T4 influences automaticity of the sinoatrial node and atrial tissue by increasing the rate of phase four depolarization resulting in a decreased action potential duration and increased frequency of sinoatrial node discharge. The latter effects when coupled with the enhanced catecholamine response also seen in thyrotoxicosis, leads to shortening of the atrial effective refractory period as well as decreasing the atrial stimulation threshold in late diastole. Shortening of atrial refractoriness may act to promote atrial flutter, by allowing areas of block to recover quickly enough to allow reentry.

Thyroid hormone has also been shown by Goel et al in 1972 to increase atrioventricular conduction in hyperthyroid dogs. In the latter study hyperthyroid denervated dog hearts were shown to have a decreased atrioventricular node functional refractory period and conduction time allowing for faster ventricular rates independent of the sympathetic nervous system. This may help to explain why patients with thyrotoxicosis and supraventricular tachycardia tend to have faster ventricular rates despite normal to subnormal levels of serum catecholamines. Others have shown that the increased catecholamine sensitivity may be due to either increased adrenergic receptor density or G-protein synthesis and not attributable to increased levels of serum catecholamines.

In normal doses, the endogenous nucleoside adenosine inhibits atrioventricular conduction and, when given to patients with atrial flutter, the brief interruption of atrioventricular conduction reveals the flutter waves. However, it failed to have any such effect in this patient despite very high doses. The lack of a response to adenosine was unusual, but explainable when one considers the known effects of thyroid hormone. Adenosine acts by competing for binding at the adenosine 1 receptor which seems to mediate the inhibition of atrioventricular node conduction. In this case however, it may be that thyroid hormone also competitively binds, induces increased inactiva-

Fig 1. Twelve-lead ECG at presentation demonstrating a narrow QRS tachycardia with 1:1 atrioventricular conduction. Negative flutter waves are best seen in lead V2, but are also well seen in V1, V3, and the inferior limb leads (II, III, and aVF).
tion, or negatively influences the interaction between adenosine and its receptor.

Finally, the significant sinus tachycardia seen well after cardioversion was also unusual for patients with isolated atrial flutter, but entirely consistent with a diagnosis of hyperthyroidism, and this provided a clue that the arrhythmia was not the primary problem. This was confirmed after the laboratory studies demonstrated a thyrotoxic profile. Although one might ascribe such tachycardia to the possible presence of tachycardia-induced cardiomyopathy, in this patient, the shortening fraction was in fact in the normal range immediately after cardioversion, and so a severe cardiomyopathy was unlikely. The question of whether this patient had an inherent predilection to develop atrial flutter which was brought out by hyperthyroidism, or whether the patient developed atrial flutter secondary to electrophysiologic changes precipitated by hyperthyroidism has not been resolved. If atrial flutter were to recur, an electrophysiologic study may be warranted.

In summary, when atrial flutter is encountered in a pediatric patient in whom there is 1:1 atrioventricular conduction, a lack of a response to adenosine, and persistent sinus tachycardia after cardioversion, the clinician should be alert to the possibility of thyrotoxicosis.

William A. Suarez, MD
George F. Van Hare, MD
Isaiah D. Wexler, MD
Divisions of Cardiology and Endocrinology
Department of Pediatrics

Case Western Reserve University School of Medicine
Cleveland, OH 44106

James E. Arnold, MD
Division of Pediatric Otolaryngology
Department of Otolaryngology-Head and Neck Surgery
Case Western Reserve University School of Medicine
Cleveland, OH 44106

REFERENCES
10. Saxena KM, Crawford JD, Talbot NB. Childhood thyrotoxicosis: a long-


Atrial Flutter: An Uncommon Pediatric Manifestation of Hyperthyroidism
William A. Suarez, George F. Van Hare, Isaiah D. Wexler and James E. Arnold

Pediatrics 1997;100:e11
DOI: 10.1542/peds.100.2.e11

The online version of this article, along with updated information and services, is located on the World Wide Web at:
/content/100/2/e11.full.html