Abstract

Objective: To present a rare cardiovascular manifestation of hyperthyroidism, its course, management and outcome.

Type of Study: Case report

Summary: We present a 39 year old woman with 2-year history of Graves' disease, who suddenly developed dizziness and syncope, one month after discontinuing her anti-thyroid medications. The patient manifested with a third degree AV-block necessitating temporary pacemaker insertion. With control of hyperthyroidism, the rhythm reverted to normal and the pacemaker was eventually removed.

Significance: Arrhythmias are common presentation of hyperthyroidism of which tachycardia and atrial fibrillation are common. Conduction abnormalities, however are rare and only one report has been published in local literature regarding these abnormalities in patients with hyperthyroidism.

Recommendations: Patients with hyperthyroidism should be observed for abnormal cardiac rhythms which may lead to life threatening arrhythmias. Immediate medical treatment may be all that is needed to address, and prevent such complications.

Introduction

It is not unusual to develop cardiovascular involvement in hyperthyroidism since excess adrenergic activity is part of the pathophysiology of hyperthyroidism. The most common arrhythmia in hyperthyroidism is tachycardia, while supraventricular tachyarrhythmias are more commonly seen in the elderly. AV block however are less frequently seen in hyperthyroidism, and high grade AV block are even rarer. These arrhythmias, however, more often than not, resolves with adequate treatment of hyperthyroidism, thus the emphasis is on good and complete history and clinical picture.

Case

P.M. 39 year old female from Sta Ana, Manila was admitted at our institution due dizziness. The patient has been diagnosed to have Graves’ disease 2 years ago and was maintained previously on Propylthiouracil and Propranolol with poor compliance.

About 4 months prior to admission she was seen at the Outpatient Department of the Department of Internal Medicine. She sought consult for hyperthyroid symptoms. The patient was eventually referred to the Section of Endocrinology for further management. Her propylthiouracil was shifted to Methimazole but her propranolol was continued. She was again lost to follow-up for 1 month and was not able to comply with the medications for the said duration.

About 4 days prior to admission, the patient developed fever and diarrhea. She also noted intermittent dizziness and near syncope. Palpitations were reported to have increased. No consult was done at that time.

Few hours prior to admission, she experienced worsening of dizziness and syncope. This prompted consult at the Emergency Department of our institution.

The patient was seen at the emergency room agitated and confused. She had a palpable blood pressure of 80mmg/Hg systolic. She was tachycardic and sweating profusely. The palpebrae were pale and the mouth and tongue were dry. The patient was also complaining of dyspnea. The patient had exophthalmos and further examination of the neck revealed thyromegaly of 4x6 cm in size. There was no palpable cervical lymphadenopathy.

On auscultation of the lungs, she had clear breath sounds, no crackles nor wheezing. The heart beat was faint and irregular. The pulses were weak. She was hyperreflexive with note of fine finger tremors.

Figure 1. Showing diffusely enlarged goiter

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The patient was hooked to a cardiac monitor which showed 3rd degree AV block. An IV access was inserted; the airway was secured and was immediately referred to the Cardiology service. A temporary pacemaker was inserted and the patient’s vital signs eventually became stable. The patient was eventually admitted at the Intensive Care Unit of the Department of Internal Medicine. She was started on Methimazole 40 mg tablet once a day. Thyroid function test done were as follows:

<table>
<thead>
<tr>
<th>Test</th>
<th>Normal Range</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>FT4</td>
<td>11-24 pmol/L</td>
<td>51.9 pmol/L</td>
</tr>
<tr>
<td>FT3</td>
<td>2.2-6.8 pmol/L</td>
<td>38.6 pmol/L</td>
</tr>
</tbody>
</table>

The patient was maintained on temporary pacemaker for 2 weeks. Her cardiac rhythm was monitored daily. She converted to a stable primary AV block after 1 week and eventually to normal sinus rhythm after 2 weeks.

The patient was extubated and was eventually discharged improved. On subsequent outpatient follow-up, she underwent ablative therapy for her Graves’ disease. Fortunately she did not developed hypothyroidism after ablative therapy for her Graves’ disease.

Discussion
Cases of high grade AV block complicating hyperthyroidism have been reported as early as 1970. A review of literature by Miller et al in 1980 revealed a total of 26 cases of varying degrees of AV block complicating hyperthyroidism, of which 16 cases are high grade AV block similar to our patient. At present there are a total of 30 reported cases of high grade AV block associated with hyperthyroidism published in the international literature. The clinical manifestations of these patients are similar presenting with history and clinical picture of thyrotoxicosis which may or may not be preceded by an antecedent primary or secondary AV block. The management in all the cases are the same with early recognition of hyperthyroidism and adequate control of thyrotoxicosis as the mainstay of therapy. Supportive management includes temporary pacemaker insertion to prevent cardiovascular collapse which was reported in only 3 patients. The outcome is usually favourable with death occurring in only 3 of the 30 cases. Sinus rhythm is almost always achieved upon control of hyperthyroidism.

In the local literature, this condition is highly underreported. At present there is only one published report on high grade AV block, that of a 26 year old female with similar presentation as our case. Temporary pacemaker was inserted to arrest cardiovascular collapse and was eventually removed after the patient’s thyrotoxicosis was controlled.

An unpublished article by Torres et al reviewed charts of patients with thyrotoxic heart disease in UP-PGH from 1981-1997. Of the 40 patients included in the study, only 34 had irregular rhythm. Atrial fibrillation is the most common abnormality of rhythm accounting for 85% (29 of 34) of the cases and only one report of 3rd degree AV block.

The possible etiology of AV block in hyperthyroidism is still controversial. However several authors agree that excessive thyroid hormone has a direct effect on the human heart. This was exemplified by the report of Eraker et al in 1978 of a patient who developed 3rd degree AV block after excessive treatment with thyroid hormone for hypothyroidism.

Another case by Miller et al showed a patient with only hyperthyroidism as the only risk factor for the development of cardiac dysrythmia.

Studies in rats show that excessive thyroid hormone increases the automaticity of the AV node which is independent of the autonomic nervous system. These findings were also noted in human studies which showed the direct effect of thyroid hormone in accelerating the heart rate independent of adrenergic and cholinergic stimulation.

Other authors however attribute the development of AV block to infection. A review of 6 cases of patients with high grade AV block in 1980 revealed that three of the six patients had a preceding tonsillitis and one patient had scarlet fever prior to the development of arrhythmia. In the one fatal case of the series, necropsy done revealed inflammation on the bundle of His which may have caused the AV block. The authors hypothesized that hyperthyroidism may predispose the patient to infection and with combined effect of excessive thyroid hormone may lead to myocardial inflammation and possible AV block.

Myocarditis in association with thyroiditis is also hypothesized to cause AV conduction abnormalities. Inflammatory cells may infiltrate the myocardium and conduction pathway, couple that with excessive thyroid hormone which increases the workload and oxygen requirement of the heart and the result is necrosis and fibrosis resulting to AV conduction abnormalities. The most common etiology is probably viral.

Our patient was apparently well with no history of previous cardiovascular conditions. Her condition only worsened when she became poorly compliant to her antithyroid medications. The probable cause of AV conduction

![Figure 1. Showing AV dissociation on ECG. (red arrow showing p-wave pacing independent of the QRS complex)](image-url)
abnormality in our patient may very well be the excessive thyroid hormone resulting from poor compliance.

Regardless of the cause, patients with hyperthyroidism should be observed for abnormal cardiac rhythms which may lead to life threatening arrhythmias. As with several cases reported, management of this subgroup of patients require primarily careful attention to cardiovascular stability and immediate treatment of hyperthyroidism. Because of hemodynamic instability, our patient underwent temporary pacemaking. As the hyperthyroidism is controlled, pacemaker can safely be removed once the patient reverts to lower grade AV block or sinus rhythm. The experience describe here suggest that rendering the patient euthyroid can convert the block to sinus rhythm and later prevent the recurrence of AV block.

References