New Onset Atrial Flutter Due to Carcinoid Heart Disease

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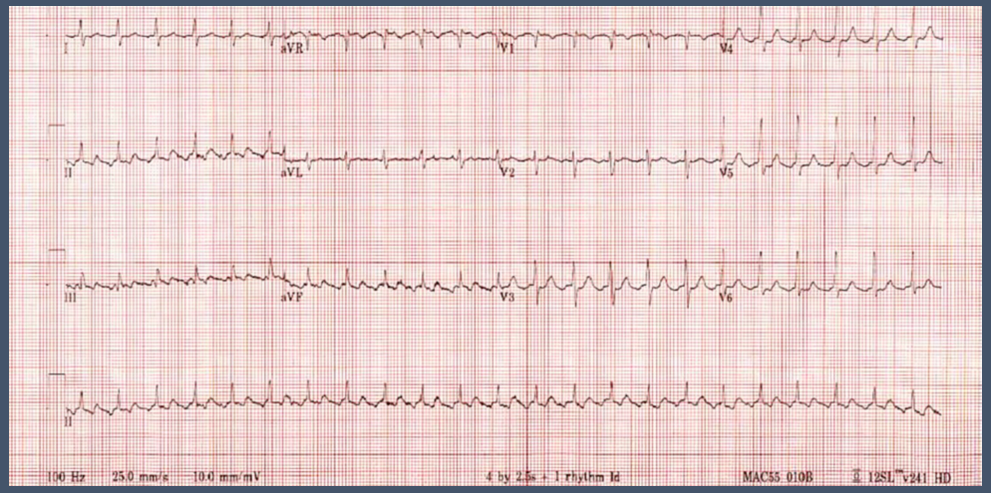
Introduction

Carcinoid syndrome (CS) is a collection of clinical symptoms that usually presents with episodes of flushing, diarrhea, and dizziness. Roughly 20-50% of patients with carcinoid syndrome will develop Carcinoid Heart Disease [1]. Fibrosis of the right-sided valves leading to Tricuspid and Pulmonic stenosis is a well-known complication of carcinoid syndrome [2]. However, CHD is not a well-known cause of cardiac arrythmias such as atrial flutter or atrial fibrillation. We here present a rare case of new onset atrial flutter as the first presenting symptom of carcinoid syndrome. To our knowledge, there have only been few sporadic reports of atrial arrhythmias, and only one other reported case of atrial flutter as the initial presentation of carcinoid syndrome [2] [3][4][5].

Case Presentation

A 66-year-old male with a medical history of hyperlipidemia and gout, presented with palpitations associated with dizziness. He denied having chest pain, shortness of breath, orthopnea, or syncope. His vital signs showed a blood pressure of 150/114 mmHg, heart rate of 152 bpm, Temperature of 98.2F and a SpO2 of 99%. On physical examination, he was tachycardic with irregular rhythm, normal S1 & S2 and no cardiac murmurs.
An electrocardiography (ECG) revealed atrial flutter with 2:1 A-V conduction and ventricular rate of 140 bpm. Both transthoracic (TTE) and transesophageal (TEE) echocardiography revealed low normal LV systolic function, ejection fraction 50-55%, normal diastolic function, normal left and right atrial size, and evidence of trace tricuspid regurgitation. There was no evidence of intracardiac thrombus, therefore a direct current cardioversion was performed with successful conversion to normal sinus rhythm. He was placed on apixaban and metoprolol succinate.
On further questioning, the patient reported multiple episodes of flushing and watery diarrhea. His thyroid stimulating hormone was within normal limits. Carcinoid syndrome was suspected therefore 5-hydroxyindoleaceticacid (5-HIAA) and Chromogranin A levels were ordered and found to be elevated confirming the diagnosis. He was started on somatostatin analogs. At his 3-month follow up appointment, his symptoms significantly improved. EKG showed normal sinus rhythm and TTE showed improved EF 60-65%.

Figure 1: ECG showing atrial flutter with 2:1 A-V conduction and ventricular rate of 140 bpm.



Discussion

Approximately 20-50% of patients with CS will also go on to develop CHD [1]. High levels of serotonin lead to the stimulation of 5-HT_{2B}, stimulating cardiomyocytes and fibroblasts to release proinflammatory cytokines to release transforming growth factor-beta (TGF-β) [1][2]. The release of cytokines and TGF-β lead to plaque-like deposits on the endocardial surface, leading to CHD [1][2]. Typically, patients develop right sided-valvular stenosis with or without regurgitation leading to right sided heart failure. Unlike the traditional CHD, our patient presented with new atrial flutter as the initial symptom of CS without major valvular abnormalities. We faced challenges with his management since there are no clear guidelines or literature to determine the best rhythm control strategy for this group of patients. It is unknown whether ablation therapy or anti-arrhythmic drugs would be more effective in maintaining sinus rhythm longer. If the latter is preferred, which group of anti-arrhythmic drug is more effective? Can we apply CHA2DS2-VASc to those patients? If so, are direct oral anticoagulant agents as effective as vitamin K-antagonists? If serotonin is involved in the pathophysiology of CS induced atrial arrythmias, would serotonin antagonists be enough to prevent future atrial flutter recurrence? These questions are currently unanswered.
While more information is needed to optimize treatment for atrial arrythmias in patients with CHD, rate control and preventing LV dysfunction should be of upmost importance when deciding therapy. For this, we elected to use metoprolol tartrate for rate control and Apixaban for thromboembolic prophylaxis. We acknowledge the need to investigate optimal therapy for atrial flutter in patients with carcinoid heart disease.

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