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Atypecal Case Presentation of Supraventricula Trachycardia in Recently Diagnosed Hyperthyroidism

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Abstract: In this case we have disused presentation of Supraventricular tachycardia in newly diagnosed case of 46 years male primary hyperthyroidism patient, to whom we treated successfully with adenosine and sent home treatment of underlying thyroid disorder leading to supraventricular tachycardia.
Keywords: Supraventricula, Trachycardia, Adenosine, Propanolol

INTRDODUCTION

Supraventricular tachycardia (SVT) is a common fatal cardiac arrhythmia; it usually presents with frequent episodes of tachycardia, which vary in frequency and severity. Many patients suffer recurrent symptoms that have a major affects quality of life. These episodes of tachycardia can cause considerable anxiety — many patients curtail their lifestyle as a result, and many prefer curative treatment. Sudden-onset, rapid, regular palpitations characterise SVT and, in most patients, a diagnosis can be made with a high degree of certainty from patient history alone. Repeated attempts at electrocardiographic documentation of the arrhythmia may be unnecessary. Treatment of SVT depends on severity and frequency. When episodes of tachycardia occur frequently, are prolonged or are associated with symptoms that affect quality of life, catheter ablation is the first choice of treatment; it is a low-risk procedure with a high success rate. Long-term preventive pharmacotherapy is an alternative approach in some patients.

CASE PRESENTATION

36 years old man came into the ER complaining of sudden onset of dizziness, palpitations, sweating and blurred vision. He had cold, clammy skin and pallor. Vital signs were HR = 188 bpm; BP =100/60 mm Hg; RR = 24 rpm. Full and equal pulses.He was conscious but anxious in CPR room.Vital signs were checked again and humidified oxygen was administered via nasal cannula and I.V. access was established and ECG monitor was attached and blood taken for routine investigations.An 12 leads ECG was done and shows the following presence of supraventricular tachycardia.

Investigations

A 12 leads ECG was done and shows the following presence of supraventricular tachycardia.

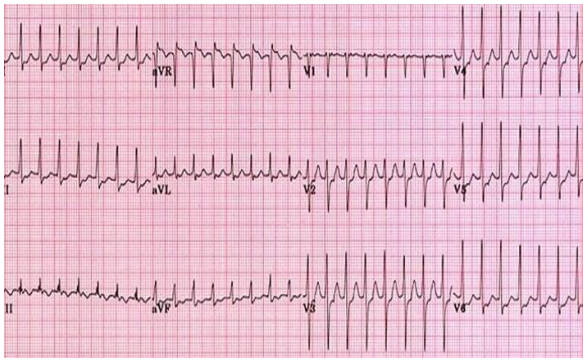

Fig-1: First presentation ECG

The 2decho was normal no structural abnormality found, but patient found to be having primary hyperthyroidism. Biochemistry report is characterised by high levels of serum thyroxine and triiodothyronine, and low levels of thyroid-stimulating hormone.

Treatment

Steps we had done

We immediately start by giving her fluid bolus 500 ml of normal saline and try to slowing heart rate using vagal manoeuvre (carotid massage) but no response. Then we decided to give adenosine 6 mg given as a rapid intravenous bolus over a 1-2 second period but Supraventricular tachycardia still present so we repeated adenosine 12 mg then patient started responding after 5 min. On monit or ECG showing reversal of rhythm to Sinus Tachycardia. Patient shifted to CCU after hemodynamic stabilization patient revaluated with biochemistry testing and 2dEcho .the 2decho was normal no structural abnormality found, but patient found to be having primary hyperthyroidism, we have started Beta-blocker and Carbimazole for threating his thyrotoxicosis.

Outcome and follow-up

In this case we have disused presentation of Supraventricular tachycardia in newly diagnosed case of 46 years male primary hyperthyroidism patient, to whom we treated successfully with adenosine and sent home treatment of underlying thyroid disorder leading to supraventricular tachycardia. Now patient is hemodynamically stable and taking treatment for primary hyperthyroidism.

DISCUSSION

36 years old man came into the ER complaining of sudden onset of dizziness, palpitations, sweating and blurred vision. He had cold, clammy skin and pallor. Vital signs were HR = 188 bpm; BP =100/60 mm Hg; RR = 24 rpm. Full and equal pulses.He was conscious but anxious in CPR room.Vital signs were checked again and humidified oxygen was administered via nasal cannula and I.V. access was established and ECG monitor was attached and blood taken for routine investigations.An 12 leads ECG was done and shows the following presence of supraventricular tachycardia. We immediately start by giving her fluid bolus 500 ml of normal saline and try to slowing heart rate using vagal manoeuvre (carotid massage) but no response. Then we decided to give adenosine 6 mg given as a rapid intravenous bolus over a 1-2 second period but Supraventricular tachycardia still present so we repeated adenosine 12 mg then patient started responding after 5 min. On monit or ECG showing reversal of rhythm to Sinus Tachycardia. Patient shifted to CCU after hemodynamic stabilization patient revaluated with biochemistry testing and 2dEcho .the 2decho was normal no structural abnormality found, but patient found to be having primary hyperthyroidism, we have started Beta-blocker and Carbimazole for threating his thyrotoxicosis.

Vagal Maneuvers

Vagal maneuvers are an appropriate first treatment option in patients with hemodynamically stable SVT [1-2]. The most commonly performed maneuvers are the Valsalva maneuver and carotid sinus massage. The increase in intrathoracic pressure resulting from the Valsalva maneuver stimulates aortic and carotid baroreceptors, causing an increase in vagal input into the atrioventricular node. Most recent studies advocate placing the patient in a supine position and attempting the maneuver for 15-20 seconds [1, 3, 4]. The Valsalva maneuver has generally been shown to be most effective in adults, having a superior effect on SVT termination compared to carotid sinus massage. Caution is advised when considering whether to attempt carotid sinus massage in older patients, as there is a risk of carotid atheroembolism and stroke even in the absence of an audible bruit

Pharmacologic Treatment

Pharmacologic therapy for acute termination of SVT is appropriate in patients when vagal maneuvers fail. The preferred initial agents are intravenous (IV) adenosine or a nondihydropyridine calcium channel blocker. Adenosine's effects are mediated by membrane hyperpolarization that typically occurs within 15-30 seconds after administration. Adenosine has a powerful effect on the atrioventricular node and is highly effective in causing temporary, complete atrioventricular nodal block. Transient sinus bradycardia or sinus arrests often occur but are short lived. The ECG should be continuously recorded during adenosine administration to document the effect of the drug on SVT and to monitor for the rare occurrence of pro arrhythmia [5, 6].

Direct-Current Cardio version

The use of direct-current cardioversion is generally limited to cases of hemodynamically unstable SVT, a rare phenomenon. When utilized, low energies (eg, 25-50 J) will suffice. Most research focuses on direct-current cardioversion in the setting of atrial fibrillation that is more commonly treated using this modality [4].

Expectant Management

The American College of Cardiology/American Heart Association/European Society of Cardiology (ACC/AHA/ESC) guidelines recommend expectant management for patients with infrequent episodes of hemodynamically stable SVT, normal left ventricular function, and a normal resting ECG [27]. Referral to an arrhythmia specialist is indicated for patients with poorly tolerated arrhythmias [7].

Pharmacologic Treatment

Chronic prophylactic drug therapy is an option for patients with SVT who cannot self-terminate the arrhythmia. Frequency of SVT, patient age, and symptom burden should all be taken into account before considering daily prophylactic drug therapy for a sometimes infrequent condition. Pharmacotherapy is associated with side effects and often does not result in complete freedom from arrhythmia. Both long-acting calcium channel blockers and beta blockers improve symptoms in 60%-80% of patients with SVT [5, 8].

Catheter Ablation

In general, guidelines recommend ablation for patients with recurrent SVT despite treatment or poorly tolerated SVT, although recommendations vary based on the category of SVT and known success rates [7, 9]. Most SVT ablation is performed under conscious sedation. Between 2-5 multipolar electrodes are positioned in the heart via the femoral (and sometimes jugular) veins under fluoroscopic guidance. The catheter electrodes have sensing and pacing functions that are used for induction and localization of the re-entrant circuit or ectopic focus. The mechanism of tissue destruction during radiofrequency ablation is thermal injury that occurs at tissue temperatures >50°C,

and tissue temperatures of 60°C-70°C are often targeted. At electrode temperatures >100°C, plasma proteins may denature to form a coagulum on the catheter tip, decreasing the density of the current delivered and increasing impedance, both of which can limit lesion growth [10].

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