Journal of Anesthesia and Surgery

ISSN:2377-1364
Case Report

OMMEGA Publishers
Answering Intelligence

OPEN ACCESS
DOI: 10.15436/2377-1364.23.6048

Aberrant Supraventricular Tachyarrhythmia during Transoperative and Management

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Abstract

Supraventricular tachycardias are all those tachyarrhythmias that originate in the His bundle tissue or above it. They have a prevalence of 2.25 per 1,000 people and an incidence of 35 per 100,000 people per year. For the correct diagnosis of these pre-excitatory rhythms, a 12-lead ECG, a functional echocardiogram and, if necessary, a Holter study. According to the American College of Cardiology and the American Heart Association, acute management consists of vagal maneuvers, use of IV adenosine, possible cardioversion and pharmacological treatment. Subsequently, the patient should be protocolized to evaluate a definitive treatment that may consist of pharmacological treatment with beta-blockers or calcium channel blockers, catheter ablation or placement of an implantable cardioverter defibrillator. In this article we present a patient with an on-set supraventricular tachycardia during the transoperative period in which it is decided to start antiarrhythmic treatment, suspend surgery and start complementary studies for the protocolization of the tachyarrhythmia as well as its subsequent definitive management.

Introduction

Transoperative tachyarrhythmias can be challenging for any anesthesiologist, which is why tachyarrhythmia should be defined as an abnormal heart rhythm with a ventricular rate above 100 beats per minute (bpm), which can be classified depending on its origin as Supraventricular Tachycardia or Ventricular Tachycardia[1]. There are three pathophysiological mechanisms described: Reentrant arrhythmias, anomalous automaticity arrhythmias and trigger dysrhythmias[2].

In supraventricular tachycardia (SVT) the arrhythmia originates in or above the His bundle tissue including: atrial fibrillation, atrial flutter, atrial tachycardia, premature atrial contraction, atrioventricular nodal reentrant tachycardia, atrioventricular reentrant tachycardia and atrioventricular junctional extrasystoles[1-3].

In the general population, the prevalence of SVT is 2.25 per 1,000 persons with an incidence of 35 per 100,000 persons per year; before the age of 65 years, the female sex presents a higher risk and after this age, a higher incidence is observed in the male sex[3].

Clinical Case

The following is the case of a 71-year-old male patient diagnosed with small cell lung carcinoma who was admitted for port catheter placement. Among his important background information are breast cancer (mastectomy with 4 cycles of chemotherapy), nasal basal cell adenocarcinoma, coronary ischemic attack, ischemic heart disease (coronary angioplasty to a circumflex artery of anomalous origin with successful implantation), atrial fibrillation in and out with successful persistence to sinus rhythm, COPD GOLD II, right carotid endarterectomy, pulmonary nodule resection, surgically resolved tension pneumothorax, positive smoking IT 165 (3 packs per day for 55 years).

On physical examination, the male patient was older than chronological age, conscious and oriented with no evidence of respiratory distress or cardiovascular compromise. On admission to the operating room he presented vital signs prior to induction HR 103 bpm, RR 14 bpm, BP 155/84 mmHg, TAM 106 mmHg, saturation 92%. It was decided to perform balanced general anesthesia, by induction with Propofol 100 mg, fentanyl 200 mcg, lidocaine 60 mg, Rocuronium 40 mg. Pharmacological hysteresis was given and a laryngeal mask type supreme no. 5

Received: August 28, 2023 **Accepted: September 7, 2023** **Publication: September 9, 2023**

Citation: Sara Fernanda, P.O., et al. Aberrant Supraventricular Tachyarrhythmia during Transoperative and Management. (2023) J Anesth Surg 9(1): 1-4.

was placed at the first attempt without incident. A 5 type laryngeal mask was placed at the first attempt without incidents. Prior to surgery the patient presented HR 133 bpm, BP 156/86 mmHg, TAM 109 mmHg, Sat 100%. The electrocardiogram showed tachyarrhythmia with stable narrow QRS of 150 beats per minute, so it was decided to perform vagal maneuvers without response and pharmacological management with boluses of 10mg of esmolol on 3 occasions, progressively decreasing the heart rate to 120 bpm, initiating amiodarone 150 mg impregnation; an infusion dose of 300 mg of amiodarone was administered postoperatively. On-set right bundle branch block was observed, so it was decided to perform transoperative ischemia protocol, performing cardiac enzymes, 12-lead electrocardiogram. Emersion was performed by pharmacological lysis with sugammadex and laryngeal mask was removed prior to aspiration of secretions and verification of airway protective reflexes, with hemodynamically stable patient, transferred to intensive care unit with Aldrete 10/10, Ramsay 2, RASS 0.

During his stay at the intensive care unit the following studies were performed: Transthoracic Echocardiogram preserved systolic function with LVEF of 62%, grade I diastolic dysfunction, calculated PSAP of 29 mmHg. Cardiac enzymes were reported with values CPK 82.6 U/L, CK-MB 1.4 ng/mL, Ultrasensitive Troponin I 3.0 pg/mL. Finding parameters within normal values, the patient was discharged from the hospital with continuous Holter monitoring of hours by the cardiology department, where a diagnosis was made of sinus rhythm base conduction alternating with aberrant atrial fibrillation (Figure 1), first degree AV block (Figure 2), as well as sustained ventricular tachycardia events (Figure 3).

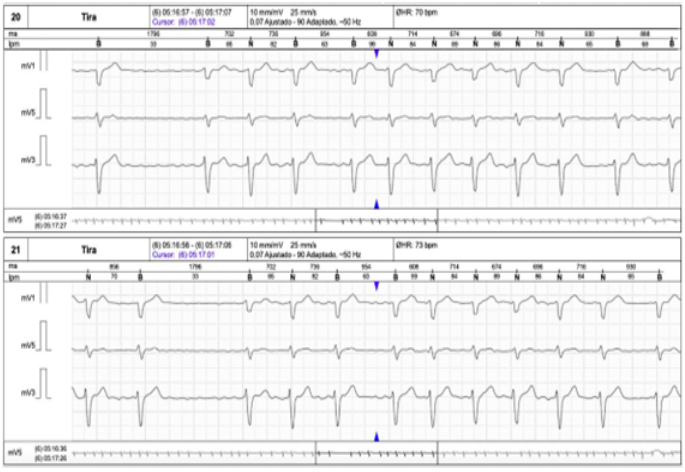

Figure 1: Aberrant Paroxysmal Atrial Fibrillation.

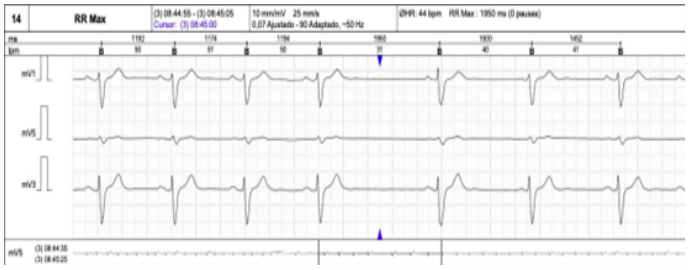

Figure 2: First Degree AV Block

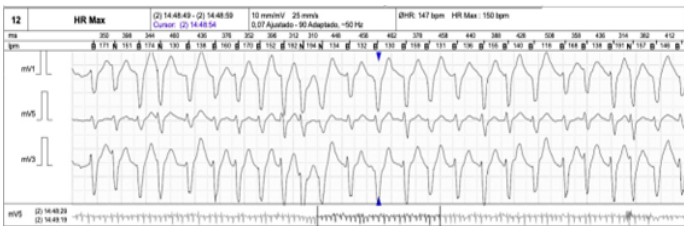

Figure 3: Ventricular Tachycardia

After diagnosis, a cardiac pacemaker placement was performed with a Biotronic Articor 7 DR-T-HM bicameral cardiac pacemaker defibrillator (Figure 4).

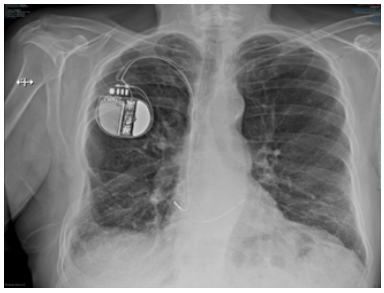

Figure 4: Control X-ray with pacemaker.

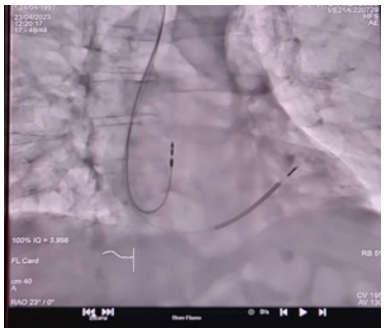

Figure 5: Invasive hemodynamic monitoring during pacemaker placement.

Discussion

The initial evaluation of a patient with suspected preexcitation rhythm begins suggestively with a 12-lead ECG at rest and a baseline echocardiogram; frequently a 24-hour ECG is performed; however, since this is an arrhythmia with infrequent episodes, a holter can be useful, although it is a study that is rarely requested. In patients with angina or risk factors for coronary artery disease, it is essential to perform myocardial ischemia tests[3,4].

In patients with SVT, the first step is to determine whether it is a narrow QRS complex (<120 ms) or wide QRS (> 120 ms); if it is a narrow complex, to establish whether it is a regular or irregular rhythm. That is why it is indicated to perform a 12-lead electrocardiogram at rest, a blood biometry, blood chemistry, thyroid function tests, a transesophageal echocardiogram together with anamnesis, and physical examination may provide a timely diagnosis and management[3,5].

According to the new 2019 ESC guidelines on the treatment of supraventricular tachycardias. The following points were stated as indications

- Use of IV Ibutilide and dofetilide for conversion of atrial flutter, in addition to cardiac pacing in case of presenting pacemaker or implantable defibrillator.
- Use of catheter ablation for asymptomatic patients whose electrophysiological study demonstrates high-risk characteristics despite having isoprenaline.
  - SPERRI <250ms
  - Refractory period of accessory pathways <250ms.
  - Multiple accessory pathways
  - Inducible tachycardia involving accessory pathways.
- If tachycardia secondary to tachycardiomyopathy cannot be controlled with drugs or ablation is not indicated, AV node ablation with biventricular or His bundle posterior cardiac pacing is recommended.

On the other hand, the ACC and the AHA gave the following recommendations for the management of supraventricular tachycardia. For acute treatment, the following was suggested

- Initiate with vagal maneuvers
- Administration of IV adenosine with regular rhythms.
- Synchronized cardioversion in hemodynamically stable patients who do not respond to pharmacological treatment.

IV Diltiazem or verapamil, as well as B-blockers in patients with stable SVT. For supportive treatment, these 2 statutes were established:

- Use of B-Blockers, diltiazem, or verapamil orally in patients with symptomatic SVT and who do not present ventricular preexcitation during sinus rhythm.
- Electrophysiological study with the option of catheter ablation is useful for diagnosis and treatment.

Radiofrequency catheter ablation referral

- AV nodal reentrant tachycardia
- Accessory pathway: AV reentrant tachycardia and pre-excited atrial fibrillation.
- Focal atrial tachycardia.
- Of the cavotricuspid isthmus in patients with atrial flutter that is symptomatic or refractory to pharmacological treatment[6].

Atrial fibrillation (AF) is defined as an arrhythmia with uncoordinated atrial activation resulting in ineffective atrial contraction. Its electrocardiographic features are characterized by 1) Irregular atrial activity 2) Absence of P waves and 3) Irregular R-R intervals. Ashman's phenomenon is an electrophysiological event caused by aberrancy of the supraventricular impulse secondary to a change in the length of the QRS complex, was described in 1947 by Gouaux and Ashman and is characterized by aberrant conduction of a wide QRS complex that occurs after a short R-R interval preceded by a long R-R interval, which can be mistaken for a premature ventricular contraction and usually shows a right bundle branch block pattern. Although it is more frequent during atrial fibrillation, it may also occur during ventricular tachycardia or after supraventricular extra systoles, this phenomenon does not produce any symptoms and does not require treatment, however, it is the disturbance of the rhythm that may produce symptoms and require medical treatment[1,7].

Atrial fibrillation is the most common type of sustained supraventricular tachyarrhythmia of a narrow complex with irregular rhythm. The most common risk factors are arterial hypertension and poorly controlled diabetes mellitus, followed by obesity, heart disease, obstructive sleep apnea, as well as smoking and alcoholism[5]. Its prevalence is estimated to be between 2-4%, with a presentation mostly in men. AF has been associated with 20-30% of all ischemic strokes, of which 10% are cryptogenic related to vascular atheroma; 20-30% develop LV dysfunction or HF secondary to excessive ventricular rate or irregular ventricular contractions; all this increases mortality by 1.5- 3.5 times and the annual hospitalization rate by 10-40%, hence a worse quality of life by > 60%[5].

According to the new ESC recommendations for the diagnosis of AF, a 12-lead ECG is essential for its diagnosis, as well as the presence of repeated, indistinguishable P waves and irregular RR interval. An important point to evaluate is the risk of stroke and bleeding to establish the steps to follow in the treatment.

On the treatment side, pharmacological cardioversion is indicated only in patients who are hemodynamically stable and who have undergone a previous evaluation of thromboembolic risk. If catheter ablation is considered, the risks of the procedure should be taken into consideration, as well as the risks of recurrence after the procedure. In addition to a strict control of risk factors that the patient presents and patient education for adherence to treatment with NOAC after having undergone cardioversion[5]. Since the first automatic defibrillator in 1980, the annual rate in the United States is estimated to be 143 thousand in 2005[8]. However, the indications for this device have been extended and specified for use in different clinical scenarios in guidelines and trials to facilitate rapid critical appraisal[8].

The indications for primary prevention are focused on LVEF without this being determined by a single cut-off figure; it is even described that there is an inter observer echocardiographic variability of 8% that complicates and relativizes the certainty of a standard figure[8]. It is recommended that these devices be placed in patients with post-infarction LV dysfunction and LVEF < 30-40% with the exception of those with LVEF < 35% and functional class I[8,9]. Similarly, implant placement is recommended in ischemic patients with LVEF < 30% and NYHA functional classes ≤ III with QRS ≥120 s and even patients with hypertrophic cardiomyopathy with a major risk factor[8]. On the other hand, placement is considered appropriate in those patients with LVEF ≥ 50% with previous AMI or non-ischemic dilated cardiomyopathy if ablation is not performed or probably appropriate in case it is performed satisfactorily[9] as well as in post-AMI patients (>40 days) without recent revascularization with NYHA I, II, III and LVEF ≤35%, NYHA III, IV with LVEF 36-40% and asymptomatic NSVT and induction of VT/VF in electrophysiological studies[9].

Otherwise, the indications for secondary prevention amount to 60.5% in patients resuscitated for sudden death, ventricular fibrillation, documented hemodynamically unstable sustained ventricular tachycardia or syncope taking into account that it is less than 5% and 15% respectively. It has been described that the reduction in overall mortality is between 25-28% and cardiac mortality is up to 50% compared to conventional pharmacological antiarrhythmic treatment[8,10].

However, those who present sustained VF or polymorphic VT in the context of coronary artery disease older than 40

days and without revascularization for less than 3 months benefit from the device regardless of LVEF when no reversible cause of cardiac arrest is identified without the need for revascularization or when coronary lesions are not favorable or when LVEF is ≤ 49%. Complementarily, the appearance of VF/VT on a stress test is considered appropriate regardless of LVEF in the presence of anatomy not favorable for revascularization[9].

Conclusion

Tachyarrhythmias are events that can occur during the transoperative period, either as isolated events or as an underlying pathology in the patient. The management of a de novo tachyarrhythmia in any scenario should investigate any physiological cause triggering these conditions and establish the first instance management and continue with multidisciplinary management for patient safety.

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