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CASE REPORT

POST-OPERATIVE VENTRICULAR TACHYCARDIA LEADING TO CARDIAC ARREST IN AN ASAG- I PATIENT OF CHOLECYSTECTOMY: A CASE REPORT

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ABSTRACT: We here present our experience with ventricular tachycardia (VT) leading to cardiac arrest in a patient with American Society of Anesthesiologists grade-I (ASA-I) 11 hours after cholecystectomy. Excessive fluid overload and hypoxemia due to lung congestion may lead to cardiac arrest in this case. Immediate diagnosis and appropriate intervention saved the life of the patient.
KEYWORDS: Ventricular tachycardia (VT), cardiac arrest, excessive fluid overload, hypoxemia, lung congestion.

INTRODUCTION: Cardiac dysrhythmia that occur during the perioperative period can usually be explained on the basis of abnormalities of cardiac pulse conduction, re-entry or impulse formation, specific rhythm disturbances, arterial hypoxemia, electrolyte disturbances (Potassium, magnesium), Acid base disturbances, altered activity of autonomic nervous system, hypertension, intubation, myocardial ischemia, catecholamine's, volatile anesthetics, co-existing cardiac diseases can be associated with initiation of cardiac dysrhythmias.[1] Better understanding of pathophysiology of the specific arrhythmia is essential for the Surgical team, because evoking event of arrhythmia occurrence correction should be tried before starting anti arrhythmic drugs. Here we are presenting a case of cholecystectomy who developed ventricular tachycardia after 11hours of uneventful cholecystectomy, and ventricular tachycardia (VT) turned into cardiac arrest. Excessive fluid overload and hypoxemia due to lung congestion may lead to VT which caused cardiac arrest in this case.

CASE REPORT: A 45 year old, 48kg, female of ASAG-I posted for open cholecystectomy. During pre-operative check-up her vitals were: pulse rate 86/min, blood pressure 130/80 mm Hg, chest bilaterally clear, cardiovascular system revealed no abnormality. Routine blood investigations, ECG, chest X-Ray were normal. Night before operation alprazolam 0.25mg tablet and Ranitidine 150mg tablet orally was given. On the day of operation after peripheral cannulation ondansetron 8mg, Pantoprazole 40mg, midazolam 1 mg intravenous (i.v) given. Glycopyrollate 0.2mg and antibiotic (ceftriaxone 1gm) injected after sensitivity test. Induction was done with propofol, tramadol. Intubation was done with succinyl choline and maintenance of anesthesia was done with vecuronium, nitrous oxide, oxygen, sevoflurane. Intravenous fluid was given DNS/RL with the formula 4-2-1.

Surgical finding was empyema of gallbladder with single stone in the neck of gallbladder. Cholecystectomy was done. Surgical procedure was uncomplicated. Intraoperative vitals of the patient were stable. Operation time was 1 hr. Reversal of anesthesia done with neostigmine and glycopyrollate. Recovery was satisfactory and after 10 minutes patient shifted to Post-operative ward. Post-operative analgesia with given with diclofenac 75mg intramuscular 8hry and Pentazocine

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30mg intramuscular s.o.s. After 11 hours she developed shortness of breath, tachycardia, tachypnea, restlessness. On examination we found basal crepts, oxygen saturation was 80%, blood pressure 130/76 mmHg. Oxygen by face mask supplemented, patient positioned in propped up. Frusemide 20mg i. v was given, ECG was done which showed wide QRS complexes >120ms, rate 156/min, AV dissociation, QRS complexes monomorphic. Diagnosed as ventricular tachycardia.

After few seconds patient suffered cardiac arrest. Immediately Cardiopulmonary resuscitation started. Cardioversion was successful. Patient ventilated with 100% oxygen. Normal cardiac rhythm restored within minutes, pulse become regular, blood pressure returned to normal. Amiodarone drip continued as preventive measure. Then amiodarone tablet continued for next few days. After 7 days Patient discharged from the hospital with satisfactory condition.

DISCUSSION: Ventricular tachycardia, defined as a series of three or more ventricular complexes at a rate of 100-250 beats/min, ECG shows wide QRS complexes, usually > 120ms, ventricular tachycardia may be monomorphic which is defined as having nearly identical QRS morphology in all of the component beats. Polymorphic VT is characterized by QRS morphology that varies from beat to beat.

VT can develop as an early or late complication of a heart attack, Patients with cardiomyopathy, heart Surgery, myocarditis, valvular heart disease. VT can also occur without heart disease. VT can be caused by anti-arrhythmic medication, changes in blood chemistry, changes in pH, lack of oxygen. [2] Amiodarone has emerged as leading antiarrhythmic therapy for termination & prevention of ventricular arrhythmia in different clinical settings.[3] In our patient excessive i.v fluid overload leading to pulmonary edema and subsequently hypoxia may lead to ventricular tachycardia.

Patient weighing 48kg require 88ml fluid/hour (4-2-1 formula) as maintenance fluid. But by mistake of nursing staff patient received 2 litres of intravenous fluid in 10 hours in place of 880ml. So she developed excessive i.v fluid related pulmonary edema, which led to hypoxia. VT may have developed due to this hypoxia.

CONCLUSION: Excessive fluid overload in the immediate Post-Operative period caused pulmonary oedema in this patient. Subsequently patient suffered hypoxia which caused VT. Immediate interpretation and management saved the life of the patient. Proper Post-operative fluid management and monitoring is essential for the outcome of the patient, otherwise life threatening event can occur.

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Fig. 1: ECG showing ventricular tachycardia (VT), Atrio-ventricular (AV) dissociation, wide, monomorphic QRS.

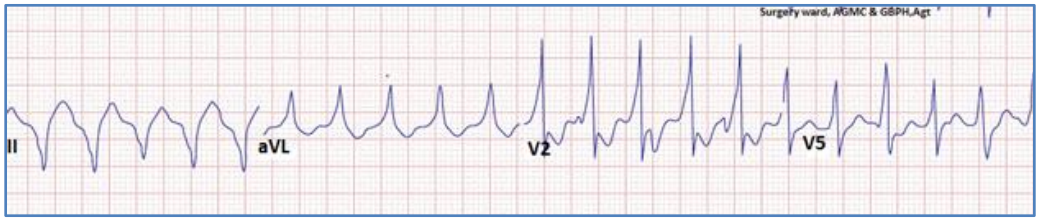

Figure 1

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