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A CASE OF VENTRICULAR TACHYCARDIA WITH COMORBIDITIES

General Medicine

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ABSTRACT

Ventricular tachycardia (V-tach or VT) is a type of regular and fast heart rate that arises from improper electrical activity in the ventricles of the heart. Although a few seconds may not result in problems, longer periods are dangerous. Short periods may occur without symptoms or present with lightheadedness, palpitations, or chest pain. Ventricular tachycardia may result in cardiac arrest and turn into ventricular fibrillation. Ventricular tachycardia is found initially in about 7% of people in cardiac arrest. Ventricular tachycardia can occur due to coronary heart disease, aortic stenosis, cardiomyopathy, electrolyte problems, or a heart attack. Diagnosis is by an electrocardiogram (ECG) showing a rate of greater than 120 bpm and at least three wide QRS complexes in a row. It is classified as non-sustained versus sustained based on whether or not it lasts less than or more than 30 seconds. The term "ventricular tachycardias" refers to the group of irregular heartbeats that includes ventricular tachycardia, ventricular fibrillation, and torsades de pointes. Here I present a case of a patient having monomorphic ventricular tachycardia who was treated and discharged in a satisfactory condition. The patient had a few comorbidities also like COPD, Diabetes Mellitus Type2, coronary artery disease, thyrotoxicosis.

KEYWORDS

Introduction:-

The morphology of the tachycardia depends on its cause and the origin of the re-entry electrical circuit in the heart. In monomorphic ventricular tachycardia, the shape of each heart beat on the ECG looks the same because the impulse is either being generated from increased automaticity of a single point in either the left or the right ventricle, or due to a reentry circuit within the ventricle. The most common cause of monomorphic ventricular tachycardia is scarring of the heart muscle from a previous myocardial infarction (heart attack). This scar cannot conduct electrical activity, so there is a potential circuit around the scar that results in the tachycardia. This is similar to the re-entrant circuits that are the cause of atrial flutter and the re-entrant forms of supraventricular tachycardia. Other rarer congenital causes of monomorphic VT include right ventricular dysplasia, and right and left ventricular outflow tract VT. Polymorphic ventricular tachycardia, on the other hand, is most commonly caused by abnormalities of ventricular muscle repolarization. The predisposition to this problem usually manifests on the ECG as a prolongation of the QT interval. QT prolongation may be congenital or acquired. Congenital problems include long QT syndrome and catecholaminergic polymorphic ventricular tachycardia. Acquired problems are usually related to drug toxicity or electrolyte abnormalities, but can occur as a result of myocardial ischemia. Class III anti-arrhythmic drugs such as sotalol and amiodarone prolong the QT interval and may in some circumstances be pro-arrhythmic. Other relatively common drugs including some antibiotics and antihistamines may also be a danger, in particular in combination with one another. Problems with blood levels of potassium, magnesium and calcium may also contribute. High-dose magnesium is often used as an antidote in cardiac arrest protocols1.

Case Report:-

The patient is a female aged 56 years and is a housewife by occupation. She is a right handed person. The chief complaints were

Cough for the past 2 days
Vomiting for the past 1 day
Fever for the past 1 day
Altered sensorium since that day morning

The patient was alright 2 days earlier when she started having cough which was productive in nature. It was yellowish in colour and was about 20 ml/day. There was no diurnal variation. Ther was no h/o haemoptysis, no history of chest pain. History of dyspnoea was present. Patient also had vomiting for the past 1 day which was non-projectile in nature. It contained the food particles that the patient had eaten earlier. There was no history of haematemesis, no history of pain abdomen. Patient had fever for the past 1 day which was of high grade and continuous in nature. There was no history of diurnal variation of fever. Patient developed altered sensorium on the morning of the day she presented to this hospital. She was not able to speak and was not giving any meaningful response to her relatives calls. There was no history of bladder or bowel incontinence, no history of fall or trauma, no history of headache, no history of involuntary movements, no history of any preferential weakness. Past history showed that- Patient was an old case of thyrotoxicosis and was on Tab Neomercazole. Patient was a known case of DM, but was on irregular treatment. Patient was an old treated ccase of Pulmonary TB. She was also an operated case of Ca Breast (left sided). Patient had cough off and on in the past 3 years . Family history had nothing significant. Personal history showed that the patient was a vegetarian, habits normal, no addictions. In menstrual history, patient had attained menopause at 45 years of age. On GPE, patient was stuporose, lying supine on the bed, moderately built and nourished. Patient was afebrile, PR-160/min, regular, good volume, no special character, equal, no radiofemoral delay, all peripheral pulses well felt, condition of vessel wall normal. RR- 30/min, thoracoabdominal, BP- 130/80 mm Hg in the right arm, supine position,

Pallor present, JVP not raised SpO2 90% without oxygen.
CNS examination showed

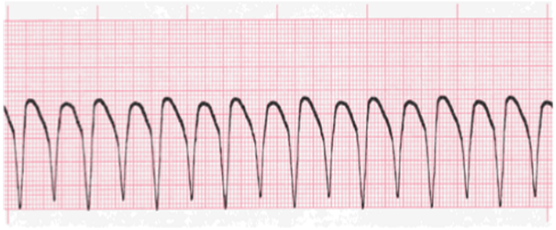
Patient stuporose
Responding to DPS by moving all 4 limbs
Pupils NSRTL
DTRs normal
Plantar reflex b/l flexor response
GCS – E3 V3 M3 = 9
CVS- S1 S2 normal
Tachycardia present

RS- Chest emphysematous
Left breast old mastectomy done
Chest expansion diminished
Trachea shifted to the right side
Impaired note present in right ICA
Dull note present in right ISA
B/l expiration prolonged, b/l ronchi present
Crepitations present right ISA

P/A-
No organomegaly
Soft
BS present

INVESTIGATIONS-
CBC
Hb 12.3 gm%
TLC 19,500/ cu.mm
P-85%
Platelet count 2.85 lac/cu.mm

LFT, RFT- WNL
Sr Na 144 mEq/lt
Sr K 3.0 mEq/lt
Urine R,M – Protein 1+
Sugar 3+
Urine for ketones- Positive
RBS – 398 mg%
CPK-MB 18
Troponin-I Negative
Viral markers- Negative
FLP- WNL
ABG-
Ph 6.9
Pco2 19
Po2 102
HCO3 10
TFT- WNL
CXR (PA) e/o NHO rt LZ
Reticulonodular shadows rt UZ
Trachea towards rt.
ECG – monomorphic sustained ventricular tachycardia.



Echo
EF 35%
LV WMA present
Diagnosis was -
Old treated Pul TB
Rt UL fibrosis
COPD
LRTI
Septicaemia
Old operated Ca Breast (left)
Old thyrotoxicosis
Diabetic ketoacidosis
CAD
Sustained monomorphic ventricular tachycardia
Patient was started on treatment with

IV fluids
Antibiotics
Insulin infusion
Potassium replacement
Amiodarone with a loading dose and furthur infusion
Ecosprin
Atorvastatin
Bronchodilators and
Neomercazole was continued
Course of the patient in hospital was-
The patient improved with the treatment.
VT was terminated and sinus rhythm restored.
Septicaemia subsided.
Potassium levels came up.
Patient was discharged in good condition.

Discussion:-
Ventricular tachycardia can be classified based on its morphology: Monomorphic ventricular tachycardia means that the appearance of all the beats match each other in each lead of a surface ECG. Scar-related monomorphic ventricular tachycardia is the most common type and a frequent cause of death in patients having survived a heart attack or myocardial infarction , especially if they have weak heart muscle. RVOT tachycardia is a type of monomorphic ventricular tachycardia originating in the RVOT. RVOT morphology refers to the characteristic pattern of this type of tachycardia on an ECG. The source of the re-entry circuit can be identified by evaluating the morphology of the QRS complex in the V1 lead of a surface ECG. If the R wave is dominant (consistent with a RBBB morphology), this indicates the origin of the VT is the left ventricle. Conversely, if the S wave is dominant (consistent with a LBBB morphology, this is consistent with VT originating from the right ventricle or interventricular septum.

Polymorphic ventricular tachycardia, on the other hand, has beat-to-beat variations in morphology. This may appear as a cyclical progressive change in cardiac axis, previously referred to by its French name torsades de pointes ("twisting of the spikes"). However, at the current time, the term torsades de pointes is reserved for polymorphic VT occurring in the context of a prolonged resting QT interval.

Another way to classify ventricular tachycardias is the duration of the episodes: Three or more beats in a row on an ECG that originate from the ventricle at a rate of more than 100 beats per minute constitute a ventricular tachycardia. If the fast rhythm self-terminates within 30 seconds, it is considered a non-sustained ventricular tachycardia. If the rhythm lasts more than 30 seconds, it is known as a sustained ventricular tachycardia (even if it terminates on its own after 30 seconds). A third way to classify ventricular tachycardia is on the basis of its symptoms: Pulseless VT is associated with no effective cardiac output, hence, no effective pulse, and is a cause of cardiac arrest. In this circumstance, it is best treated the same way as ventricular fibrillation (VF), and is recognized as one of the shockable rhythms on the cardiac arrest protocol. Some VT is associated with reasonable cardiac output and may even be asymptomatic. The heart usually tolerates this rhythm poorly in the medium to long term, and patients may certainly deteriorate to pulseless VT or to VF. Less common is ventricular tachycardia that occurs in individuals with structurally normal hearts. This is known as idiopathic ventricular tachycardia and in the monomorphic form coincides with little or no increased risk of sudden cardiac death. In general, idiopathic ventricular tachycardia occurs in younger individuals diagnosed with VT. While the causes of idiopathic VT are not known, in general it is presumed to be congenital, and can be brought on by any number of diverse factors2.

Treatment:
Therapy may be directed either at terminating an episode of the abnormal heart rhythm or at reducing the risk of another VT episode. The treatment for stable VT is tailored to the specific person, with regard to how well the individual tolerates episodes of ventricular tachycardia, how frequently episodes occur, their comorbidities, and their wishes. Individuals suffering from pulseless VT or unstable VT are hemodynamically compromised and require immediate electric cardioversion to shock them out of the VT rhythm.

Cardioversion
If a person still has a pulse, it is usually possible to terminate the episode using electric cardioversion This should be synchronized to the heartbeat if the waveform is monomorphic if possible, in order to avoid degeneration of the rhythm to ventricular fibrillation. An initial energy of 100J is recommended. If the waveform is polymorphic, then higher energies and an unsynchronized shock should be provided (also known as defibrillation).

Defibrillation
A person with pulseless VT is treated the same as ventricular fibrillation with high-energy (360J with a monophasic defibrillator, or 200J with a biphasic defibrillator) unsynchronised cardioversion (defibrillation). They will be unconscious.

The shock may be delivered to the outside of the chest using the two pads of an external defibrillator, or internally to the heart by an implantable cardioverter-defibrillator (ICD) if one has previously been inserted.

An ICD may also be set to attempt to overdrive pace the ventricle. Pacing the ventricle at a rate faster than the underlying tachycardia can sometimes be effective in terminating the rhythm. If this fails after a short trial, the ICD will usually stop pacing, charge up and deliver a defibrillation grade shock.

Medication
For those who are stable with a monomorphic waveform the medications procainamide or sotalol may be used and are better than lidocaine. Evidence does show that amiodarone is better than procainamide. As a low magnesium level in the blood is a common cause of VT, magnesium sulfate can be given for torsades de pointes or if a low blood magnesium level is found/suspected.Long-term anti-arrhythmic therapy may be indicated to prevent recurrence of VT. Beta-blockers and a number of class III anti-arrhythmics are

commonly used, such as the beta-blockers carvedilol, metoprolol, and bisoprolol, and the Potassium-Channel-Blockers amiodarone, dronedarone,bretylium, sotalol, ibutilide, and dofetilide. Angiotensin-converting-eynsyme (ACE) inhibitors and aldostrone antatagonists are also sometimes used in this setting.

Surgery

An implantable ICD is more effective than drug therapy for prevention of sudden cardiac death due to VT and VF, but may be constrained by cost issues, as well as patient co-morbidities and patient preference. Catheter ablation is a possible treatment for those with recurrent VT. Remote magnetic navigation is one effective method to do the procedure.There was consensus among the task force members that catheter ablation for VT should be considered early in the treatment of patients with recurrent VT. In the past, ablation was often not considered until pharmacological options had been exhausted, often after the patient had suffered substantial morbidity from recurrent episodes of VT and ICD shocks. Antiarrhythmic medications can reduce the frequency of ICD therapies, but have disappointing efficacy and side effects. Advances in technology and understanding of VT substrates now allow ablation of multiple and unstable VTs with acceptable safety and efficacy, even in patients with advanced heart disease3.

Conclusion:-

Ventricular tachycardia is a rare entity to be encountered in the daily practice. The patient is at risk of sudden cardiac death when it occurs. One has to recognize it early to treat it in a timely fashion. VT can degenerate into more dangerous rhythms like VF. Early defibrillation or antiarrhythmic drugs like Amiodarone have to be given emergently to treat the arrhythmia. The comorbid conditions of the patient should also be treated as the underlying disease is usually the precipitating factor in most of the cases.

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References:-

1. Baldzizhar, A; Manuylova, E; Marchenko, R; Kryvalap, Y; Carey, MG (September 2016). "Ventricular Tachycardias: Characteristics and Management". Critical care nursing clinics of North America. 28 (3): 317–29.
2. Stewart RB, Bardy GH, Greene HL (June 1986). "Wide complex tachycardia: misdiagnosis and outcome after emergent therapy". Annals of Internal Medicine. 104 (6): 766–71.
3. deSouza, IS; Martindale, JL; Sinert, R (February 2015). "Antidysrhythmic drug therapy for the termination of stable, monomorphic ventricular tachycardia: a systematic review". Emergency medicine journal : EMJ. 32 (2): 161–167.