OF VENTRICULAR ARRHYTHMIAS IN PATIENTS ADMITTED TO A TERTIARY CARE HOSPITAL

Shah Zeb1, Mohammad Adil2, Junaid Zeb3, Rifaq Zeb4, Hikmatullah Jan5, Mohammad Irfan6

^{1,2,5,6} Department of Cardiology, Lady Reading Hospital, Peshawar–Pakistan.

- ³ Ayub Teaching Hospital, Abbottabad–Pakistan.
- ⁴ Khyber Teaching Hospital, Peshawar–Pakistan.

Address for Correspondence: Dr. Mohammad Adil

Assistant Professor, Department of Cardiology, Lady Reading Hospital, Peshawar–Pakistan.

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ABSTRACT

Objective: To determine the frequency of various types of ventricular arrhythmias, its causes, treatment, in-hospital course and short term outcome of ventricular arrhythmias in patients admitted to a tertiary care hospital.

Methodology: This observational study was conducted in Cardiology Unit of Lady Reading Hospital, Peshawar from Ist September 2014 to 31st March 2015. All patients with ventricular arrhythmias admitted during study duration were included after fulfilling the inclusion and exclusion criteria. Their baseline, clinical, echocardiographic characteristics, treatment and hospital outcome were recorded in a specially designed proforma. SPSS version 19 was used for statistical analysis.

Results: A total of 127 patients were included in the study. Males were 93 (73.22%). Mean age was 43.42 ±10.7 years. Causes of ventricular arrhythmias were acute coronary syndrome 51 (40.15%), ischemic cardiomyopathy 27 (21.25%) and idiopathic dilated cardiomyopathy 13 (10.23%). Successful pharmacological cardioversion was achieved in 29 (22.83%) patients with amiodarone and 3(2.36%) patients with lignocaine. The rest of patients were cardioverted with electrical cardioversion. A total of 8 (6.3%) patients expired.

Conclusion: Most common cause of ventricular arrhythmias was coronary artery disease followed by ischemic and dilated cardiomyopathies. Commonly used technique of cardioversion was pharmacological for hemodynamically stable patients and electrical for hemodynamically unstable patients.

Key words: Ventricular arrhythmias, Ischemic cardiomyopathy, Electrical cardioversion, Pharmacological cardioversion

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INTRODUCTION

Ventricular tachycardias (VT) arise from ventricular foci and are classified as broad complex tachycardias morphologically. It is the commonest form among all types of ventricular tachyarrythmias. It is either sustained i.e. lasting for more than 30 seconds or non-sustained, which lasts for <30 seconds. Fascicular tachycardia is relatively uncommon which arises from left bundle branch and of short duration i.e. 110-140 msec. Polymorphic ventricular tachycardia and torsades de pointes are morphologically the same but the former is associated with normal QT during normal sinus rhthym. The ECG trace of polymorphic VT have more similarity with AF plus pre excitation but the latter is far less common than torsades de pointes1. It can occur in acute myocardial infarction and may deteriorate into ventricular fibrillation (VF). Torsades de pointes is often associated with prolonged QT, which is either acquired or congenital.^{2,3} It may also occurs in patients with electrolytes abnormalities usually hypomagnesaemia. In structurally normal heart, VT can be associated with an increased risk of sudden death. Ventricular fibrillation usually occur because of coronary ischemia while monomorphic VT is often because of scar. More benign forms of idiopathic VT can also occur in structurally normal heart.

Treatment of VT involves both emergent management and prevention of recurrence with medical and device therapy. Some patients may need implantable cardioverter defibrillator (ICD). The left ventricular ejection fraction is used for risk stratification for future events in case of ischemic and non-ischemic cardiomyopathy and need for ICD. Catheter ablation may be used as a treatment option in selected cases. The aim of our study was to determine the frequency of various

causes of VT in patients admitting to our hospital and their in-hospital course and outcome. It will help us to know the real burden of the ventricular arrhythmias in our local population and help in strategic planning for allocation of healthcare resources to overcome it.

METHODOLOGY

This observational study was conducted in Cardiology Unit, Lady reading Hospital, Peshawar. The study was approved by hospital ethical committee and detailed informed consent was taken from study participants. All the consecutive patients of both gender and age more than 18 years admitted in Cardiology Unit, Lady reading Hospital, Peshawar with diagnosis of ventricular arrhythmias, from Ist September 2014 to 31st March 2015 were included in the study. Only patients admitted in CCU with primary diagnosis of ventricular arrhythmias were included in the study. Those patients who were already admitted with some other diagnosis and meanwhile developed ventricular arrhythmias in hospital stay were excluded. Also patients who have already ICD/ cardiac resynchronization therapy ICD, implanted were excluded. Sample size was calculated by using WHO sample size formula for qualitative variable and the mortality rate of around 9% of ventricular arrhythmias in various studies, with precision/absolute error of 5% and type 1 error of 5%, 127 patients were included.

Ventricular arrhythmias were diagnosed on 12 leads ECG as sustained ventricular arrhythmias or ventricular fibrillation by 02 cardiologists. All these patients were put on continuous telemetry for constant observation unless the arrhythmias were terminated and if any cause was present, itwas treated accordingly. Hemodynamically unstable VTs (with hypotension, altered mentation, pulselesness, chest pain, signs of heart failure) were cardioverted electrically. While Hemodynamically stable VTs were treated by pharmacological cardioversion. They were managed as per guideline directed medical therapy. The anti-arrhythmic drugs used were amiodarone, lignocaine and magnesium sulphate or calcium channel blockers. After cardio version to sinus

rhythm they remained admitted in CCU for 24 hours.

All the baseline investigations i.e., urea, creatinine, liver functions tests, serum electrolytes were done. Thyroid function tests and serum magnesium level were also done in indicated patients. Some of the patients in which we suspected the bradycardia induce ventricular tachycardia, they were subjected to 24 to 48 hours holter ECG to know about the pauses and bradycardia episodes; while some with suspected single irritable foci were send for electrophysiology study for possible ablation if feasible. The ECGs after successful cardioversion were then studied in detail with long lead II to specifically look for QT interval, Brugada pattern, WPW, epsilon waves, Osborn waves or hyperkalemic ECG changes. Transthoracic echocardiography was performed in all the patients by expert echo-cardiographer. All baseline clinical characteristics (diabetes, hypertension, smoking behavior, history of CAD), ECG findings, echocardiographic characteristics, treatment offered and hospital outcome were recorded in a specially designed profor-

Ventricular tachycardia (VT) was operationally defined as ≥ 3 ventricular extra-systoles which occur in succession rate of ≥ 120 beats per minute (bpm). Monomorphic VT was defined as typically regular rhythm originating from a single focus with identical or similar QRS complexes while Polymorphic VT was diagnosed if there was beat to beat variation in QRS complexes and irregular pattern.

Statistical analyses were done using SPSS version 19.0. Continuous variables were expressed as mean ± SD. Categorical variables were expressed as percentages. Data were presented in the form of tables.

RESULTS

A total of 127 patients were included in the study. Males were 93 (73.22%) and females were 34 (26.77%). Mean age was 43.42 \pm 10.7 years. Diabetes was present in 30 (23.6%), hypertension 39 (30.7%) and dyslipidemia was present in 18 (14.17%) patients. Baseline characteristics of patients are shown in table 1.

Table 1: Baseline characteristics of patients with ventricular arrhythmias

Variables	Frequency	Percentages
Males	93	73.22
Females	34	26.77
Diabetes	30	23.6
Hypertension	39	30.7
Smokers	27	21.25
Family History of CAD	42	33.07
Past History of CAD	37	29.13
Dyslipedemia	18	14.17

Table 2: Etiology of ventricular arrhythmias

Variables	Frequency	Percentages
Acute Coronary Syndrome	51	40.15
Ischemic Cardiomyopathy	27	21.25
Dilated Cardiomyopathy	13	10.23
Creatinine >5 mg/dl	13	10.23
Hyperkalemia (>6meq/l)	3	2.36
Hypomagnesaemia	4	3.15
Long QT Interval	5	3.93
Wheat Pills Poisoning	6	4.72%
Arrythmogenic Right Ventricular Dysplasia	1	0.7%
Brugada Syndrome	2	1.57%
Severe Bradycardia Induced Ventricular Arrhythmias	6	4.7%
Severe Mitral Regurgitation	6	4.72%
Severe Aortic Regurgitation	4	3.15%
Tetrology of Fallot	3	2.36%
Ebstein Anomaly	2	1.57%
Severe Pulmonary Hypertension	7	5.5%
No Pathology Identified	38	29.92%

Table 3: Treatment given to patients with ventricular arrhythmias

Variables	Number	Percentages
Spontaneously Reverted	5	3.93%
Amiodarone	29	22.83%
Lignocaine	3	2.36%
Amiodarone & DC Cardioversion/Defibrillation	16	12.6%
DC Cardioversion/Defibrillation Only	64	50.4%
Pacemaker Implantation for Bradycardia Induce Arrhythmia	6	4.7%

Monomorphic VT occur in 83 (65.35%), polymorphic VT occurred in 19 (14.96%), torsade depointes in 9 (7.08%) and ventricular fibrillation in 16 (12.6%) patients. Hemodynamically stable patients with monomorphic VT were 48 (37.8%), while hemodynamically unstable with low blood pressure were 18 (14.17%) and hemodynamically unstable with acute heart failur were 17(13.38%) patients.

Specific etiology for ventricular arrhythmia was acute coronary syndrome in 51 (40.15%) patients, ischemic cardiomyopathy in 27 (21.25%) patients and idiopathic dilated cardiomyopathy in 13 (10.23%) patients. Some patients with VT have multiple abnormalities. Relative frequencies of other causes are shown in table 2.

Amiodarone was the sole agent used for reverion to sinus rhythm in 29 (22.83%) patients, while 16 (12.6%) patients were given amiodarone followed by DC cardio-

version because of either failure to cardiovertion with drugs or because of hemodynamic instability. Lignocaine was given to 3(2.36%) patients. Other treatment modalities for VT are shown in table 3. In-hospital mortality was 8 (6.3%).

DISCUSSION

In this article, we have documented the etiologies and management of ventricular arrhythmias in patients presenting to our hospital. Ali et al⁴ showed mean age of 57.1 years supporting our study. Male to female ratio was 1.38:1. Major cardiovascular risk factors were encountered in 58% of the patients. Tachyarrhythmia's occurred in 42% patients without any associated risk factor supporting our data. Only 28% of patients with VT were managed with drugs alone. In their study defibrillation was required in all the patients with VF again

supporting our data. All the patients with pulseless VT (33%) were defibrillated but the stable VT with pulse were treated with anti-arrhythmic drugs (28%) and unstable VT with pulse (72%) received synchronized cardioversion. Amiodarone and combination of drugs was used in the same frequency as in our study^{4,5}.

The frequency of various types of ventricular arrhythmias in our study is supported by international data⁵⁻⁷. These international studies demonstrated that monomorphic VT was more common than polymorphic VT and most of the patients were hemodynamically stable. Frequency of ischemic etiology for most cases of ventricular arrythmia is supported by Terranova et al⁸, Wright et al⁹ and other international studies¹⁰⁻¹². Very few patients in our study were hemodynamically unstable and needed CPR. The CPR in our study population was as effective as the international Data¹³⁻¹⁵. Volpi et al¹⁶ observed in their study that ischemia was the main trigger for ventricular fibrillation.

In our study, 3.15% patients were having severe hypomagnesaemia and all of these patients have torsade de pointes as the presenting arrhythmia suggesting that severe hypomagnesaemia is an important cause of torsade de pointes. This observation is supported by Banai et al¹⁷, which showed that magnesium replacement is the best strategy to treat torsade de piontes. Intravenous magnesium is an effective and safe therapy for torsade de pointes and is now regarded as the treatment of choice for this arrhythmia.

About 10% of patients had deranged renal function tests with mean creatinine more than 5mg/dl and hyperkalemia was documented in 2.36% of them. Chronic kidney disease (CKD) patients are more susceptible to the occurrence of various ventricular arrhythmias; a leading cause of death in these subset of patients. There are various pathophysiological mechanism involve for arrhythmias in these patients which is complex and seems to be related to structural and functional cardiac abnormalities caused by CKD, associated with several triggers, such as water and electrolyte disorders, disturbances in acid base balance, hormonal disturbances, arrhythmogenic drugs and the dialysis procedure itself. Data is limited about the clinical outcomes in CKD patients with asymptomatic ventricular arrhythmias¹⁸. The frequency of these metabolic derangement as the cause for ventricular arrhythmias were also similar as with other national and international data.

Wheat pills poisoning was the cause of VT in 06 (4.72%) patients, of which 04 (3.15%) were expired and 02(1.57%) were reverted to sinus rhythm. Wheat pills contain aluminum phosphide which is cardiotoxic. These patients usually have severe metabolic abnormalities which leads to polymorphic VTs. The treatment offered to these patients was conservative and correc-

tion of metabolic abnormalities. Also the data regarding the VT associated with wheat pills poisoning is lacking in international guidelines. About 14% patients were referred for EP studies/VT ablation/ICD insersion to electro-physiologist. Ebstein anomaly (1.57%) and tetrology of Fallot (2.36%) were found in our study. Both of these are associated with ventricular arrhythmias in various studies^{19,20}. Severe pulmonary hypertension was present in 5.5% patients. All of them were having ventricular fibrillation. Hoeper et al²¹ reported the outcome of CPR in patients with pulmonary artery hypertension. He reported that about 16% of his study population experienced cardiac and respiratory arrest over 3 years follow up. The initial rhythm in ECG at time of arrest was VF in 8% and VT in 4% of cases. It also support our findings.

A total of 6.3% patients expired. Mortality in our study was lower than a similar study performed in Pakistan⁴, which may be because of more monomorphic VTs and hemodynamically stable patients and also a significant number of our study population had reversible causes.

CONCLUSION

Most common cause of ventricular arrhythmias was coronary artery disease followed by ischemic and dilated cardiomyopathies. Commonly used technique of cardioversion was pharmacological for hemodynamically stable patients and electrical for hemodynamically unstable patients.

REFERENCES

- Edhouse J, Morris F. ABC of clinical electrocardiography: Broad complex tachycardia-Part II. Br Med J 2002; 324:776-9.
- Kaye AD, Volpi-Abadie J, Bensler JM, Kaye AM, Diaz JH. QT interval abnormalities: risk factors and perioperative management in long QT syndromes and Torsades de Pointes. J Anesth 2013; 27:575-87.
- Trinkley KE, Page RL, Lien H, Yamanouye K, Tisdale JE. QT interval prolongation and the risk of torsades de pointes: essentials for clinicians. Curr Med Res Opin 2013; 29:1719-26.
- 4. Ali Z, Khokhar MM, Shahid S, Mahmood S, Tufail S. Frequency and Outcomes of Arrhythmias. J Rawal Med Coll 2013; 17:7-10.
- Galante A, Pitroiusti A, Cavazzini C, Magrini A, Bergamaschi A, Sciarra L et al. Incidence and risk factors associated with cardiac arrhythmias after coronary artery bypass surgery. Arch Phys Med Rehabil 2000; 81:947-52.
- Volpi A, Cavalli A, Santoro L, Negri E. Incidence and prognosis of early primary ventricular fibrillation in acute myocardial infarction. Am J Cardiol 1998; 82:265-71.
- 7. Reinelt P, Karth GD, Geppert A, Heinz G. Incidence and

- type of cardiac arrhythmias in critically ill patients. Intensive Care Med 2001; 27:1466-73
- Terranova P, Valli P, Severgnini B, Dell'Orto S, Maria GE. Early Outcomes of Out-of Hospital cardiac arrest after early defibrillation: a 24 months retrospective analysis. Indian Pacing Electrophysiol J 2006; 6:194-201.
- Wright D, Bannister J, Ryder M, Mackintosh AF. Resuscitation of patients with cardiac arrest by ambulance staff. Br Med J 1990; 301:600-2.
- Kaul TK, Fields BL, Riggin LS, Wyatt DA, Jones CR. Ventricular arrhythmia followingsuccessful myocardial revascularization. Eur J Cardiothorac Surg 1998; 13:629-36.
- Sayer JW, Archbold RA, Wilkinson P, Ranjadayalan K, Timmis AD. Prognostic implications of ventricular fibrillation in acute myocardial infarction: new strategies required for further mortality reduction. Heart 2000; 84:258-61.
- Abella BS, Alvarado JP, Myklebust H, Edelson DP, Barry A, O'Hearn N et al. Quality of Cardiopulmonary Resuscitation During In-Hospital Cardiac Arrest. J Am Med Assoc 2005; 293:305-10.
- 13. Sanders AB, Ewy GA. Cardiopulmonary resuscitation in the real world: When Will the Guidelines Get the Message? J Am Med Assoc 2005; 293:363-5.
- Bang A, Aune S, Ekström L, Lundström G, Holmberg S. Characteristics and outcome among patients suffering in-hospital cardiac arrest in monitored and non-monitored areas. Resuscitation 2002; 53:21-7.
- 15. Andreasson AC, Herlitz J, Bång A, Aune S, Ekström L, Lindgvist J et al. Characteristics and outcome among patients with a suspected in-hospital cardiac arrest. Resuscitation 2006; 69:191-7.

- Volpi A, Cavalli A, Santoro L, Negri E. Incidence and prognosis of early primary ventricular fibrillation in acute myocardial infarction--results of the Gruppo Italiano per lo Studio della Sopravvivenza nell'Infarto Miocardico (GIS-SI-2) database. Am J Cardiol 1998; 82:265-71.
- 17. Banai S1, Tzivoni D. Drug therapy for torsade de pointes. J Cardiovasc Electrophysiol 1993; 4:206-10.
- 18. Bonato FOB, Canziani MEF. Ventricular arrhythmia in chronic kidney disease patients. J Bras Nefrol 2017; 39:186-95.
- Sabate Rotes A, Connolly HM, Warnes CA, Ammash NM, Phillips SD, Dearani JA et al. Ventricular arrhythmia risk stratification in patients with tetralogy of Fallot at the time of pulmonary valve replacement. Circ Arrhythm Electrophysiol 2015; 8:110-6.
- 20. Obioha-Ngwu O, Milliez P, Richardson A, Pittaro M, Josephson ME. Ventricular Tachycardia in Ebstein's Anomaly. Circulation 2001; 104:E92-4.
- Hoeper MM, Galié N, Murali S, Olschewski H, Rubenfire M, Robbin IM et al. Outcome after cardiopulmonary resuscitation in patients with pulmonary arterial hypertension. Am J Respir Crit Care Med 2002; 165:341–4.

CONTRIBUTORS

SZ conceived and designed the format of the study and collected the data. MA, JZ and RZ performed the literature search and participated significantly in the analysis and interpretation of the results. HJ and MI supervised the study. All the authors participated in writing the manuscript. All authors contributed significantly to the submitted manuscript.