ORIGINAL RESEARCH PAPER

Volume-7 | Issue-2 | February-2018 | PRINT ISSN No 2277 - 8179

INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

HAEMODYNAMIC INSTABILITY : AN INDICATOR OF POOR OUTCOME IN PATIENTS OF SUSTAINED VENTRICULAR TACHYCARDIA (VT) – A TERTIARY CARE CENTER EXPERIENCE

Cardiology

Dr. Rahul Sudan* Senior Resident, Department Of General Medicine Shri Guru Ram Rai Institute Of Medical And Health Sciences, Shri Mahant Indiresh Hospital (SGRRIMHS, SMIH) Dehradun, Uttrakhand, India, 248001 *Corresponding Author

Dr. Gunjan Gupta Senior Resident, Department Of Dermatology, Shri Guru Ram Rai Institute Of Medical And Health Sciences, Shri Mahant Indiresh Hospital (SGRRIMHS, SMIH) Dehradun, Uttrakhand , India , 248001

Dr. (Prof.) Khursheed Aslam Professor, Department Of Cardiology, Sher I Kashmir Institute Of Medical Sciences (SKIMS), Soura, Srinagar, Jammu And Kasmir, India, 190001

Dr. Irfan Yaqoob Post DM Cardiology Scholar, Department Of Cardiology, Sher I Kashmir Institute Of Medical Sciences (SKIMS), Soura, Srinagar, Jammu And Kasmir, India, 190001

Dr. Mehroz Ahmed Senior Resident, Department Of General Medicine Shri Guru Ram Rai Institute Of Medical And Health Sciences, Shri Mahant Indiresh Hospital (SGRRIMHS , SMIH) Dehradun, Uttrakhand , India , 248001

ABSTRACT

BACKGROUND : Sustained Ventricular Tachycardia (VT) is one of the most life threatening arrhythmias presenting in emergency. Haemodynamic instability in such patients further increases the morbidity and mortality.

AIMS AND OBJECTIVES : To study the effect of haemodynamic instability on the final outcome of sustained VT patients presenting in emergency and to study the clinicoaetiological profile of such patients..

METHODS : A total of 63 patients of sustained ventricular tachycardia (VT) presenting in emergency were enrolled. These patients were divided in to haemodynamically stable and unstable groups and were observed for their clinicoaetiological profile and outcome.

RESULTS : Haemodynamic instability at presentation was present in 22 patients (55 %) of sustained VT, presenting in emergency. Majority of these patients were males (68 %). Polymorphic VT was present in 10 patients ; out of these 10 patients , 9 patients (90%) were haemodynamically unstable. coronary artery disease (CAD) was the most common aetiology of haemodynamic instability in 9 patients (41 %). Decreased Left Ventricular ejection Fraction (LVEF) was seen in 19 patients(86 %) of haemodynamic instability.

CONCLUSION : Haemodynamic instability in sustained VT increases the mortality in such patients. Decreased LVEF and polymorphic pattern of VT are associated with increased chances of haemodynamic instability. Coronary artery disease (CAD) is most common aetiology of haemodynamic instability in patients of sustained VT.

KEYWORDS

Haemodynamic instability , Sustained ventricular tachycardia (VT) , Polymorphic VT , Left Ventricular Ejection Fraction (LVEF)

INTRODUCTION

The most common cause of wide complex tachycardia is ventricular tachycardia (VT).[1,2] VT that persists longer than 30seconds or that requires immediate termination is known as sustained VT and that lasts shorter than 30 seconds of duration is known as nonsustained VT. It may present with a wide variety of clinical and electrocardiographic (ECG) features. [3] The spectrum of haemodynamic consequences of sustained VT ranges from completely asymptomatic to overt symptoms of cerebral hypoperfusion ,cardiogenic shock or loss of consciousness. On the basis of the haemodynamic status of the patient , sustained VT can be classified in to haemodynamically stable and haemodynamically unstable. Haemodynamically unstable patients have signs or symptoms of insufficient oxygen delivery to the vital organs as a result of tachycardia. Such manifestations may include hypotension , shock ,altered level of consciousness or loss of consciousness and congestive cardiac failure. Haemodynamically stable patients have adequate vital end organ perfusion and thus do not experience signs or symptoms of haemodynamic compromise. The underlying substrate for VT varies. Ischemic heart disease and non ischemic cardiomyopathies are among the most common causes.[4] Sustained VT is an important cause of sudden cardiac death (SCD). ECG recordings at the time of SCD have shown that ,in approximately sixty percent of SCD victims , an episode of VT was identified as the initial event.[5-7]. The presence of haemodynamic instability in VT patients is an indication for immediate intervention in the form of DC cardioversion.

Haemodynamic status of VT patient at the time of presentation plays an important role in determining the final outcome. We tried to evaluate the effect of haemodynamic instability at presentation on the final outcome of such patients and to know about the profile of haemodynamically unstable patients of sustained VT.

METHODS

Aims : To study the effect of haemodynamic instability on the final outcome of sustained VT patients presenting in emergency department and to study the clinicoaetiological profile of such patients.

Study design : This observational study was conducted at Sher I Kashmir Institute of Medical Sciences (SKIMS), a tertiary care center in Srinagar , Jammu and Kashmir , India , between August 2013 to May 2015.

Study population :

Inclusion criteria : All the cases of sustained VT presenting in the emergency department were enrolled in the study. Sustained (VT) was diagnosed using The " Brugada algorithm[8]" and/or The "Avr Vereckei algorithm[9]"

Exclusion criteria : The following patients were excluded from the study.

1. Patients who were already admitted in hospital and developed sustained VT during the course of their hospital stay were not included in this study.
2. Patients with nonsustained VT.
3. Patients who did not give consent for further evaluation.

Consent : An informed consent was taken from all the patients

Initial evaluation : Every patient of sustained VT presenting in emergency , was placed on noninvasive monitor for continuous ECG , blood pressure and pulse oximetry monitoring . Patients presenting

with pulseless sustained VT were initially treated as per ACLS guidelines till the time DC cardioversion was given. After aborting acute attack of sustained VT , all the patients were subjected to detailed history taking,and physical examination. Routine baseline investigations including 12 lead ECG and chest radiography were done in all the patients.

On the basis of haemodynamic status of the patient at the time of presentation in emergency department , patients were divided in to two groups: Haemodynamically unstable and Haemodynamically stable. ***HAEMODYNAMIC INSTABILITY*** was defined as a need for immediate intervention (DC cardioversion) due to the clinical state of the patient. This group included patients , presenting with hypotension, shock , pulseless VT, loss of consciousness or congestive heart failure**.** ***HAEMODYNAMIC STABILITY*** : All other patients were categorized as haemodynamically stable.

Patients in both the groups were then subjected to detailed investigations to look for their aetiological profile .Echocardiography was done in all the patients. Coronary angiography(CAG) was done in the patients with definitive or suspected myocardial infarction , patients with high risk of coronary artery disease (CAD) derived from Framingham risk calculator and in patients who were having global hypokinesia and low ejection fraction on echocardiography to prove a diagnosis of non ischemic dilated cardiomyopathy (DCMP). Cardiac MRI was done in patients where no specific aetiology was clear and a diagnosis of a specific cardiomyopathy like that of Arrhythmogenic Right Ventricular Cardiomyopathy /Dysplasia (ARVC/D) was suspected . The diagnosis of ARVC/D was done on the basis of Modified Task Force Criteria .All the patients in haemodynamically unstable group were given D/C cardioversion whereas patients in haemodynamically stable group received intravenous drugs initially and then D/C cardioversion in cases which did not respond to drugs alone.

Patients in both the groups were observed for their epidemiology , clinical features , final outcome to evaluate the effect of haemodynamic instability on the final outcome.

RESULTS

In our study, a total of 63 patients of sustained VT were enrolled. Out of which, 43 patients (65 %) were males and remaining 22 patients (35 %) were females with a male to female ratio of 1.8 : 1. The range of the age of the patients was in between 19 years to 88 years and the mean age at presentation was 52 years. We classified the patients in to haemodynamically stable and unstable groups. A total of 22 patients presented in emergency in a state of haemodynamic instability. Among these 22 haemodynamically unstable patients , 15 patients (68 %) were males and remaining 7 patients (32 %) were females with a male to female ratio of 2.1 :1. The mean age at presentation in haemodynamically unstable group was slightly higher (53 years) than the mean age at presentation in the haemodynamically stable group (51 years). The most common presentation of haemodynamic instability was congestive cardiac failure in 13 patients (59 %) followed by patients who presented with a combination of pulseless VT , shock and loss of consciousness (LOC) in 7 patients (32 %)

A total of 53 patient (84 %) had monomorphic pattern of sustained VT while the remaining 10 patients (16 %) showed polymorphic pattern of sustained VT . Echocardiography was done in all the patients. Left ventricular ejection fraction (LVEF) was found normal (LVEF > 50 %) in 31 patients (49 %) and decreased (LVEF < 50 %) in 32 (51 %) patients. Coronary angiography (CAG) was done in 29 patients (46 %) , out of these , 17 patients were diagnosed as coronary artery disease (CAD) patients and in remaining 12 patients , it was done as echocardiography was showing global hypokinesia and decreased LVEF suggesting a diagnosis of non ischemic dilated cardiomyopathy (DCMP). MRI was done in 10 patients only , where rest of investigations pointed towards a possible diagnosis of arrhythmogenic right ventricular cardiomyopathy/ dysplasia (ARVC/D).

In our study , the most common aetiology resulting in haemodynamic instability was CAD in 9 patients (41 %), this was followed by non ischemic dilated cardiomyopathy (DCMP) in 6 patients (27 %). .All the patients who presented with haemodynamic instability (22 patients) were initially given DC cardioversion. Among the patients presenting in a state of haemodynamic stability (41 patients) ,all the patients were initially managed with intravenous drugs . Among these patients who were initially given intravenous drugs , 31 patients (76 %) successfully reverted to sinus rhythm whereas the remaining 10 patients required the additional use of DC cardioversion as could not be reverted with drugs alone. Among the haemodynamically unstable group , all the patients were successfully reverted by DC cardioversion initially.

Mortality was seen in a total of 19 patients (30 %) . Analyzing the characteristics of these patients ,the following important results were observed.

Out of 41 patients presenting in a haemodynamically stable state , 8 patients died (20 %) whereas 11 patients (50 %) , out of a total of 22 patients in unstable group died . This result was statistically significant ***(p < 0.05)*** . Thus haemodynamic instability at presentation increases the mortality in VT patients. Morphology of the VT also influenced the final outcome. In our study 53 patients had monomorphic VT and 10 patients had polymorphic VT. Among 53 patients presenting with monomorphic VT, 13 patients (25%) were haemodynamically unstable whereas among 10 patients presenting with polymorphic VT, 9 patients (90 %) were haemodynamically unstable. This result was statistically significant ***(p <0.05)*** .Among 53 patients with monomorphic VT , mortality was seen in 11 patients (21%) whereas among 10 patients presenting with polymorphic VT , mortality was seen in 8 patients (80 %) ,this was statistically significant too ***(p < 0.05).*** It was therefore , concluded in our study that polymorphic VT at presentation was a poor prognostic factor leading to haemodynamic instability and increased the overall mortality in sustained VT patients. . Among 32 patients with decreased LVEF, haemodynamic instability was present in 19 patients (59 %) whereas among 31 patients with normal LVE F, haemodynamic instability was present in 3 patients (10 %).This result was statistically significant ***(p < 0.05)*** and we concluded decreased LVEF is associated with haemodynamic instability and thus a poor outcome.

TABLE NO. 1

SEX	HAEMODYNAMIC STATUS		TOTAL
	STABLE	UNSTABLE	
MALE	26	15	41
FEMALE	15	7	22
TOTAL	41	22	63

Table (1) showing sex distribution of haemodynamically stable and unstable patients

TABLE NO. 2

HAEMODYNAMIC STATUS	OUTCOME		TOTAL
	DISCHARGED	DIED	
STABLE	33	8	41
UNSTABLE	11	11	22
TOTAL	44	19	63

Table (2) showing final outcome with respect to haemodynamic status at presentation

TABLE NO .3

VT MORPHOLOGY	HAEMODYNAMIC STATUS		TOTAL
	STABLE	UNSTABLE	
MONOMORPHIC	40	13	53
POLYMORPHIC	1	9	10
TOTAL	41	22	63

Table (3) showing haemodynamic status with respect to morphology of sustained VT

TABLE NO. 4

VT MORPHOLOGY	OUTCOME		TOTAL
	DISCHARGED	DIED	
MONOMORPHIC	42	11	53
POLYMORPHIC	2	8	10
TOTAL	44	19	63

Table (4) showing final outcome with respect to morphology of sustained VT

TABLE NO. 5

(LVEF)	HAEMODYNAMIC STATUS		TOTAL
	STABLE	UNSTABLE	
< 50 %	13	19	32
> 50 %	28	3	31
TOTAL	41	22	63

Table (5) showing final outcome with respect to left ventricular ejection fraction (LVEF)

TABLE NO. 6

TREATMENT	OUTCOME		TOTAL
	DISCHARGED	DIED	
DRUGS	31	0	31
SHOCK	11	11	22
BOTH*	2	8	10
TOTAL	44	19	63

Table (6) showing outcome with respect to treatment modalities *Both means patients who received both drugs and DC cardioversion

TABLE NO.7

AETIOLOGY	HAEMODYNAMIC STATUS		TOTAL
	STABLE	UNSTABLE	
CAD	16	9	25
DCMP	6	6	12
RCMP	2	0	2
PPCMP	0	2	2
ARVC/D	6	4	10
IDIOPATHIC	5	1	6
MISCELLANEOUS	6	0	6
TOTAL	41	22	63

Table (7) showing haemodynamic status with respect to aetiology of sustained VT.

CAD = coronary artery disease ***,DCMP*** = non ischaemic dilated cardiomyopathy ***,RCMP*** = restrictive cardiomyopathy ,***PPCMP*** = peripartum cardiomyopathy, ***ARVC/D*** = aahythmogenic right ventricular cardiomyopathy /dysplasia ***,IDIOPATHIC*** = Those cases where no specific aetiology could be found. This group included 2 patients with suspected long QT syndrome ,2 patients had morphology suggestive of outflow tract tachycardia and one patient each of fascicular VT and Brugada syndrome. Only 1 patient (Long QT syndrome) presented with haemodynamic instability from this group. ***MISCELLANEOUS*** =This group comprised of those cases where VT was secondary to either electrolyte abnormality , medication , or some extra myocardial disease. , 4 patients were having hypokalemia , 2 had constrictive pericarditis.

FIGURE NO. 1

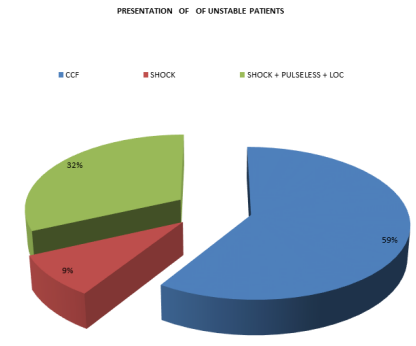

Figure (1) showing presentation of haemodynamically unstable patients (LOC = Loss Of Conciousness)

DISCUSSION

VT is one of the most life threatening arrhythmias presenting in the emergency department. There are several associated factors which make VT often difficult to treat and hence increase the overall mortality. Haemodynamic instability at presentation is one such factor. The main observations of our study are as follows.

1. Haemodynamic instability at the time of presentation is associated with an overall increased mortality in patients of sustained VT.
2. Ischemic heart disease is the most common cause of haemodynamic instability among sustained VT patients.
3. Higher mean age and low ejection fraction are more commonly associated with haemodynamic instability.

A total of 63 patients of sustained VT presenting in emergency were enrolled in our study. Out of them, 22 patients (35%) were haemodynamically unstable at the time of presentation . The fact that 41 patients (65 %) of sustained VT were haemodynamically stable at presentation shows that it is misleading to believe that VT always causes severe haemodynamic disturbance.[10] The mean age of sustained VT patients in our study was 52 years. We classified the patients in to haemodynamically stable and unstable groups. The mean age at presentation in stable group was 51 years whereas it was 53 years in the unstable group. Haemodynamic instability was more common among the males (68 %). Thus elderly males more commonly presented with haemodynamic instability in our study. This may be related to the fact that in other reports [11] , ischemic heart disease was present in majority of the patients of sustained VT. Lemery et al. reported that patients with idiopathic VT are younger and often haemodynamically stable than the ischaemic VT patients.[12]

Most common aetiology of haemodynamic instability in our study was coronary artery disease which was followed by non ischemic dilated cardiomyopathy , both of which are well known causes of sustained VT. Other forms of cardiomyopathy like ARVC/D and peripartrum cardiomyopathy were also seen as a cause of haemodynamic instability in sustained VT patients. Two patients of peripartum cardiomyopathy (PPCMP) presented with sustained VT in a haemodynamically unstable state .Both these patients developed VT after few days of child birth and were having grossly decreased LVEF. One patient from idiopathic group also presented with haemodynamic instability.This patient was a suspected case of long QT syndrome.

Among 53 patients presenting with monomorphic VT , haemodynamic instability was present in 13 patients (25 %) whwereas among 10 opatients presenting with polymorphic VT , haemodynamic instability was present in 9 patients (90 %). Moreover mortality was more in the patients presenting with polymorphic VT (80%). Both these results were statistically significant ($p < 0.05$). Thus polymorphic pattern of sustained VT is an independent risk factor for both haemodynamic instability and overall mortality. Domanovits et al. studied clinical profile of VT presenting in the emergency department and showed that only ECG characteristic that correlated with haemodynamic instability was polymorphic pattern.

Haemodynamic instability was present more commonly (58 %) in patients presenting with decreased LVEF . Mortality was also higher among such patients. This is consistent with several studies in the past which showed that overall mortality is more in patients of sustained VT with low LVEF.[13,14]

Intravenous drugs in the form of amiodarone , sotalol , lignocaine and i/v beta blockers were initially given to all the haemodynamically stable (41) patients which reverted VT to sinus rhythm in 31 patients (75 %).The safety and efficacy of these drugs for acute management has been reported.[15,16]

Mortality was 50 % among patients presenting with haemodynamic instability as compared to 20 % among patients with haemodynamic stability. Haemodynamic instability in sustained VT is a poor prognostic indicator.

REFERENCES

1. Akhtar M, Shenasa M, Jazayeri M, Caceres J, Tchou T. Wide QRS complex tachycardia. Reappraisal of a common clinical problem. Ann Intern Med 1988;109:905–12.
2. Steinman RT, Herrera C, Schuger CD, Lehmann MH. Wide QRS tachycardia in the conscious adult. Ventricular tachycardia is the most common cause. J Am Med Assoc 1989;261:1013–6.
3. Miller JM. The many manifestations of ventricular tachycardia. J Cardiovasc Electrophysiol 1992;3:88–107.
4. Arnow WS , Ahn C, Mercando AD et al.Prevalance and association of ventricular tachycardia and complex ventricular arrhythmias with new coronary events in older men and women with and without cardiovascular disease. J Gerontol A Biol Sci Med Sci 2002 ;57 march (3) :MI 78-80
5. Bayes-de-luna A, Coumei P ,Leclerc QJF. Ambulatory sudden cardiac death : Mechanisms of production of fatal arrhythmias on the basis of data from 157 cases. Am Heart J. 1989 : 117 :151-159
6. Do Manovits H , Paulis M , Nikafardjam M , Sustained ventricular tachycardia in the emergency department. Resuscitation : 1999 sept : 42 (1) : 19-75

7. Stevenson WG : Ventricular tachycardia : Trans Am Clin Climatol Assoc. 2009 : 120: 403-412
8. Brugada P , Brugada J , Mont L et al. A new diagnostic approach to the differential diagnosis of a regular tachycardia with wide QRS complex. Circulation : 1991 ;83:1649.
9. Vereckei A , Duray G , Szenasi G et al. Application of a new algorithm in the differential diagnosis of wide QRS complex tachycardia. Eur Heart J. 2007 ;28:"589-600
10. Morady F, Baerman JM, DiCarlo LA, De Buitleir M, Krol RB, Wehr DW. A prevalent misconception regarding wide complex tachycardias. J Am Med Assoc 1985;254:2790–2.
11. Trappe HJ, Brugada P. Prognosis of patients with ventricular tachycardia and ventricular fibrillation: role of the underlying etiology. J Am Coll Cardiol 1988;12:166.
12. Lemery R, Brugada P, Della Bella P, Dugernier T, Dool A, Wellens HJ. Nonischemic ventricular tachycardia: clinical course and long term follow-up in patients without clinically overt heart disease. Circulation 1989;79:990–9.
13. Moss AJ, Zareba W, Hall WJ, et al. Multicenter Automatic Defibrillator Implantation Trial II Investigators Prophylactic implantation of a defibrillator in patients with myocardial infarction and reduced ejection fraction. N Engl J Med. 2002March21;346(12):877-883 Epub 2002 Mar 19
14. Bardy GH, Lee KL, Mark DB, et al. Sudden Cardiac Death in Heart Failure Trial (SCD-HeFT) Investigators Amiodarone or an implantable cardioverter-defibrillator for congestive heart failure [published correction appears in N Engl J Med. 2005;352(20):2146] N Engl J Med. 2005;352(3):225-237
15. GriffithMJ, Linker NJ , Garrat CJ et al. Relative efficacy and safety of intravenous drugs for termination of sustained ventricular tachycardia . Lancet. 1990 ;336 : 670-673
16. Roden DM , Risks and benefits of antiarrhythmic therapy. New Engl J Med.1994-331 ;785-791