DOI: 10.5958/2319-5886.2015.00137.X

International Journal of Medical Research & Health Sciences

www.ijmrhs.com Volume 4 Issue 3 Coden: IJMRHS Copyright @2015 ISSN: 2319-5886

Received: 16th Mar 2015 Revised: 20th Apr 2015 Accepted: 7th May 2015

Case report

PERSISTENT ARRHYTHMIAS AFTER ELECTROCUTION IN A PATIENT SCHEDULED FOR GRAFT SURGERY

RekhiBalwinder K.[1], RekhiHarnam S[2], *Singh Praneet[3], Singh Harcharan[4]

[1]Professor,[3]Resident, Dept. of Anaesthesia, [2]Professor, Dept. of Surgery, [4]Professor, Dept. of Cardiology Government Medical College and Hospital, Patiala, Punjab, India

***Corresponding author e**mail:singhpraneet87@gmail.com

ABSTRACT

PVC may occur due to numerous etiologies. Manifestations may range from an asymptomatic presentation to ventricular fibrillation and death. The incidence of PVC in healthy population may range from 0.5% in those aged below 20 years old to 2.2% in those older than 50. Here we present a case of persistent arrhythmias after six months of electrocution. Patient was admitted in our hospital for graft surgery post burns.

Keywords: Arrhythmias, Electrocution, PVC (Premature Ventricular Contractions), Holter Monitoring.

INTRODUCTION

Heart is most susceptible organ to electric injury with wide range of abnormalities. Cardiac arrhythmias can occur due to various etiological factors. Manifestations may range from an asymptomatic presentation to ventricular fibrillations and death. 3-4% of admissions in the burn units are accounted by electrical burn injuries.[1]Approximately 20% of all electrical injuries occur in children (with a bimodal peak incidence in toddlers and adolescents), usually via cable extensions or wall outlets.[2]Death most often occurs in young males (male:female = 9:1).[2]

CASE REPORT

We present a case of high voltage electrocution 0f 11,000kV ac and supraventricular and ventricular arrhythmias after six month of incidence. A 23 year old male patient electrician by occupation weighing 50 kg was scheduled for post burn graft surgery lower limb. About 5 months back patient got electrocuted with 11,000kV tension wire while doing some repair work. Baseline investigation of the patient were Hb 11.0 g% ,BT 2'5",CT 6'10", blood urea 34 mg%, serum creatinine 1.2 mg%, serum Na 140 meq/l ,serum K 4.2meq/l , ECG attached, breath holding more than 25 seconds.

In the operation theatre patient was found to be having irregular pulse, ECG showed multiple PVC, more than 13-14/min. without any symptom. Surgery was deferred and patient was sent to cardiology for expert opinion. Echocardiography was done, no major abnormality was observed. No regional wall motion abnormality was seen. Ejection fraction was 50%. After getting fitness patient was again scheduled for graft surgery. Irregular pulse persisted with heart rate 98/min BP 124/80 mm hg n right upper limb. On auscultation lung fields were clear. 12 lead ECG (Fig. 1) showed 12-15 PVC/min. Lignocaine 1.5% was given to abort ongoing arrhythmias, but there was no response. Defibrillator and anti-arrhythmic drugs were kept ready in case of progression of multiple PVC to ventricular fibrillation and unstable hemodynamic

Since the patient was hemodynamically stable subarachnoid block was performed at L3-4 level

using bupivacaine 10mg plus 25µg fentanyl. Patient remained hemodynamically stable throughout the procedure and shifted to recovery in satisfactory conditions.

Postoperatively Holter ECG study (Fig. 2) was done where the ventricular, supraventricular arrhythmia was observed. The report showed ventricular ectopics totaled 1254 averaging 59/hr. with 1247 single and four paired and here trigemni and zero R on T. Supraventricular ectopics totaled 713 averaging 33.6/hr- 663 single and 18 paired beats. Supraventricular tachycardia occurred four times.

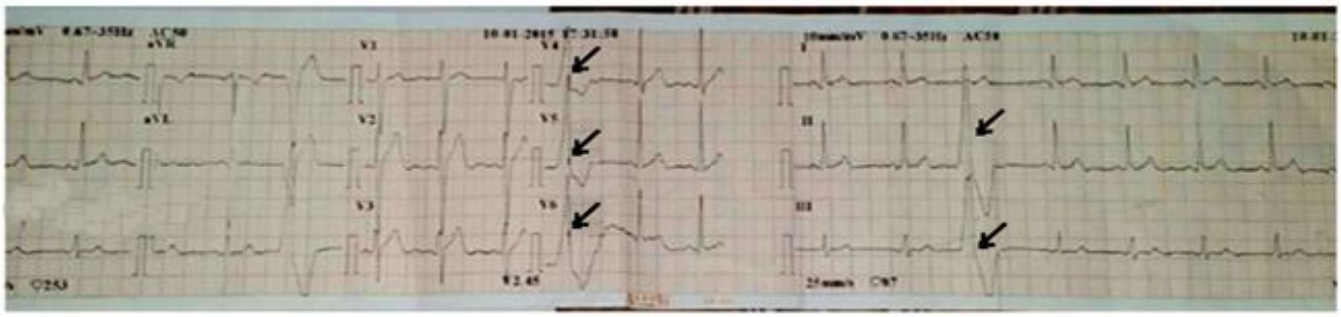

Fig 1: 12 Lead E.C.G.

Medication:
Indication:

Report Summary

BASIC RHYTHM

Rhythm:	
Total Beats:	103382
Avg Rate:	82
Min Rate:	50 bpm at 03:54D2
Max Rate:	169 bpm at 22:28D1
Brunch Block beats	absent
AFib percent	0.00
Paced beats	absent

VENTRICULAR ECTOPY

Total beats:	1254	Isolated Beats:	1247
Avg/Hour:	59.1	Paired beats:	4
Avg/1000:	11.9	R on T	0
		Trigeminy Beats:	3
Bigeminy Runs:	0	Bigeminy Beats:	0
VT Runs:	0	VT Beats:	0

ST ANALYSIS

Type	Max	CH	Lgth	Time
UP	4.1	3	492.0 m	09:24D2
HOR	2.3	3	6.0 m	10:12D2
DN	9.1	2	960.6 m	15:47D1

Median CH1:	-0.65 mm
Median CH2:	-1.12 mm
Median CH3:	-0.78 mm

SUPRAVENTRICULAR ECTOPY

Total beats:	713	Isolated SVE:	663
Avg/Hour:	33.6	Paired:	18
Avg/1000:	6.8		
SVT runs:	4	SVT beats:	32
Longest SVT run:		16 beats at 101 bpm at 15:47D1	
Fastest SVT run:		16 beats at 101 bpm at 15:47D1	

HEART RATE VARIABILITY

SDNN:	131	SDNN5:	78	PNN50:	22
TRIA:	620	SDANN:	98	RMSSD:	96
Night Heart Rate: (23:00-07:00)					73

Pauses

PAUSES >2.0 sec: absent

Interpretation:

The average heart rate, excluding ectopy, was 82 BPM with a minimum of 50 BPM at 03:54D2 and a maximum of 169 BPM at 22:28D1. Heart beats, including ectopy, totaled 105349 beats.
VENTRICULAR ECTOPICS totaled 1254 averaging 59.1 per hour with 1247 single, 4 paired, 3 trigeminy and 0 R on T.
SUPRAVENTRICULAR ECTOPICS totaled 713 averaging 33.6 per hour ,with 663 single and 18 paired beats.
SUPRAVENTRICULAR TACHYCARDIA occurred 4 times.
The fastest run was at 101 BPM and occurred at 15:47D1 with 16 beats.
The longest run was 16 beats at 15:47D1 at a rate of 101 BPM.

Fig 2: Post-operative Holter Study

DISCUSSION

Cardiac arrhythmias are usually explained on the basis of abnormalities of cardiac impulse conduction (reentry) or impulse formation (automaticity). Reentry excitation is the mechanism for most cardiac arrhythmias and reflex excitation of tissue by return of the same cardiac impulse in circuitous fashion. In automaticity cardiac impulses are generated each time to excite the heart. Enhanced automaticity leads to cardiac arrhythmia by facilitating repetitive firing from single focus. [3] The various events that may be associated with cardiac arrhythmias encountered by an anesthetist are

1) Use of Volatile anesthetics [4]
2) Arterial hypoxemia
3) Hypercarbia
4) Systemic hypertension
5) Endogenous or exogenous catecholamines
6) Electrolyte imbalance
7) Coexisting cardiac diseases.

In our patient no other disease was present so the changes observed could be exclusively connected with the electric accident. The mechanism of electrical induced arrhythmias is not much clear. Due to the differences in electrical resistance, current travels more preferentially along blood vessels and nerves, that makes the heart as an organ more susceptible to injury by electrical currents.[5]Patchy necrosis may be seen in heart muscle biopsy specimens after injury by electrical currents, the fibrotic tissue can act as a potential chronic arrythmogenic focus.[6] Increased cardiac sodium/potassium pump activities and an increase in potassium concentration have also been described as causes.[7] Cardiac arrhythmias may occur at the time of electrical shock or later, but mostly within the first day after an injury.[7,8]Arrowsmith et al.[9]retrospectively evaluated 145 patients with electrical injury in the same centre over a five year period; 128 (88%) had suffered low voltage injury and 17 (12%) had suffered high voltage injury (>1000 V). The frequency of cardiac complications was 3% (four patients). Atrial Fibrillation was detected in only one patient in their study, in this case normal sinus rhythm was restored after i.v. digoxin infusion. Purdue et al. [10] recommended cardiac monitoring of patients with electrical injury if there was loss of consciousness, recorded arrhythmia in the field, abnormal ECG on admission and rhythm disturbance during monitoring in the emergency room.

CONCLUSION

Since in our study no other concomitant disease history was present, so the PVC's observed could be exclusively related to the electrical incident that happened approximately 5 months ago.

ACKNOWLEDGMENT

We are thankful to our colleagues in the Department of Anaesthesia & Intensive Care and the Department Of General Medicine & Cardiology of Govt. Medical College, Patiala and associated Rajindra Hospital for their support and interest.

Conflict of Interest: Nil

REFERENCES

1. Cameron P,Jelinek G, Kelly A-M, Murray L, Brown AFT, Heyworth J. Textbook of Adult Emergency Medicine (2nd ed.), chapter 27.6: Electric shock and lightning injury, 2004. Churchill Livingstone; Postgraduate textbook.
2. Hettiaratchy S, Dziewulski P; ABC of burns: Pathophysiology and types of burns. BMJ 2004; 328: 1427-29.
3. Stoelting RK, Dierdorf Sf. Anesthesia and coexisting disease , 5th edition. Philadelphia: Churchill His in dogs. Anesthesiology 1972; 336:112-8
4. Akhtar M. Management of tachyarrythmias JAMA 1982; 247:671-4
5. Atlee JL, Rusy BF. Halothane depression of A-V conduction studied by electrograms of Bundle of His in dogs. Anesthesiology 1972; 336:112-8
6. Johnson RR, Eger EI, Wilson C. A comparative interaction of epinephrine with enflurane, isoflurane and halothane in man. AnesthAnalg 1976; 55:709-12.
7. Jensen PJ, Thompson PE, Bagger JP, Norgaard A, Baandrup U. Electrical injury causing ventricular arrhythmias. Br Heart J. 1987 Mar; 57(3):279-83
8. Ku CS, Lin SL, Hsu TL, Wang SP, Chang MS. Myocardial damage associated with electrical injury. Am Heart J. 1989 Sep; 118(3):621-24.
9. ArrowsmithJ ,Usgaocar RP, Dickson WA. Electrical injury and the frequency of cardiac complications.Burns.1997; 23:576–8.
10. Purdue GF, Hunt JL. Electrocardiographic monitoring after electrical injury: necessity or luxury? JTrauma. 1986; 26:166–7.