Sudden Death Syndrome: A Case Study

*JAWED H. SIDDIQUI, M.D.**

SUMMARY:

In this case study a patient has been described who sustained several episodes of sudden cardiac death, most likely secondary to ventricular fibrillation. Luckilly the patient was successfully resuscitated several times, but the patient was still high risk for having future spells of sudden cardiac death. The patient's cardiac arrhythmia persisted inspite of successful coronary bypass surgery and his episodes of sudden cardiac death also persisted. An electrophysiological study was done and a proper antiarrhythmic regmine was selected. The patient responded to this therapy extremely well. Up to the time of this writing the patient is symptom free and fully active. A brief discussion of the importence of cardiac arrhythmia and it's relationship to sudden cardiac death was also reviewed.

Sudden cardiac death is a challenging problem in clinical practice. A large number of people die suddenly throughout the world. A case with sudden death syndrome is presented with a brief discussion about this very important problem.

CASE REPORT:

This 49 year old white male with insulin dependent diabetes mellitus has known history of coronary artery disease with evidence of inferior mypass graft in June of 1983. He has history of multiple episodes of cardiac arrest in June of 1983. He has history of multiple episodes of cardiac arrest probably secondary to ventricular arrhythmia. In April of 1983 the patient had syncopal episode with loss of consciousness lasting approximately one minute. The patient did not have any prodromal symptoms and no cerebral activity and he was not incontinent at that time. The patient spontaneously regained consciousness and felt perfectly normal. He did well until May 29, 1983 when he began feeling tired while climbing a flight of stairs which he thought at that time was possibly secondary to hypoglycemia. He decided to lie down and was found unresponsive by his wife. She resuscitated him with CPR and called an ambulance. The paramedics had to again resuscitate him with CPR, and he was admitted to the hospital where he was told he had a myocardial infarction. It was during that hospitalization that he became unresponsive and was found to be in ventricular fibrillation and was resuscitated with precordial thumps and CPR. A cardiac catheterization during that hospitalization showed severe two vessel disease and a coronary bypass graft was performed in June of 1983. After discharge, the patient was doing well and walking up to one mile a day. On the weekend before admission he had an episode of chills and an episode of heat intolerance, and later while eating he had some chest heaviness. He then again had a syncopal episode from which he spontaneously recovered. He was admitted to the hospital again for futher work-up. The patient had several electrocardiograms. They were all within normal range except a prolonged QT interval. A neurological work-up including CT scan of the brain was within normal limits. The blood tests did not reveal any evidence of hypoglycemia. He was monitored in the hospital in the intensive Care Unit for five days, but did not show any cardiac arrhythmias. He was transferred to Jewish Hospital on July 19, 1983 for evaluation and treatment of his cardiac arrhythmia. The patient had a strong family history of myocardial infarction and diabetes. The patient is married and has five children. They are all alive and healthy. The patient has two jobs. He worked for the Police Department which mainly involved writing parking tickets. He also worked in a shopping center as a part-time security guard. The

*St. Louis, Miss., U.S.A.

patient smoles two packs of cigarettes a day since he was a teenager, but he stopped smoking two years ago. There is no history of dirinking or any allergies. The patient had a coronary bypass surgery a month ago. In 1970 he had surgery done for a herniated disc over the lumbosacral spine area.

Physical examination revealed a well developed, well nourished, white male in no acute distress. Blood pressure was 155/75 mm Hg. Heart rate was 90/min and regular. Respirations were 16/min. The patient is afebrile. Examination of the neurological system did not reveal any localized or lateralized neurological findings. There is no jugular venous distention. There was no corotid bruit. Carotid pulses are normal bilaterally. Examination of the lungs was within normal limits. Examination of the cardiovascular system revealed 1st and 2nd heart sounds to be within normal limits. There was no murmur, gallop, or pericardial rub. The abdomen is soft and nontender. Liver and spleen were not palpable. No bruit was heard over the abdomen. Examination of the extremities did not reveal any abnormal findings.

Stool was negative for occult blood. Hemoglobin was 12.4 gm, hematocrit 36.8% with MCV 79.3, white count 7,100/cmm with 60% Segs, 7% Stabs, 3% Eosiniphils, 2% basophils, 25% lymphocytes, and 3% monocytes. PT and PTT were within normal limits. Serum electrolytes and blood urea nitrogen were within normal limits. Blood glucose was 273 mg/dl. Chest X-ray showed no acute lesion Electrocardiogram showed an inferior myocardial infarction of indeterminate age with a lateral nonspecific ST, T-wave abnormality. QT interval on admission was .34 sec. The QT interval on several other occasions was slightly prolonged. Please refer to copy of the electrocardiogram where the QT interval was .44 seconds (Fig. 1).

The patient was admitted to the telemetry floor to be followed by the arrhythmia service on July 21, 1983 and electrophysiological study was done involving programmed ventricular stimulation, including ventricular tachycardia wiich was initially sustained at a rate of 270 beats per minute. Following infusion of 1500 mgs of IV Procainaide, sustained ventricular tachycardia at a rate of 190 beats per minute was induced and was accompanied by hypertension, but no loss of consciousness. It was decided to start the patient on protocol drugs of Amioderone and Lorcanide. Prior to starting these medications and eye examination was done which was entirely normal and PFT's were done which were remarkable for FEVI and FVC, both slightly decreased at 2.48 litre and 3.73 litre respectively, with an F 25—75 of only 42 % of normal, consistent with early chronic obstructive lung disease. The patient was started on Amioderone and Lorcanide which he tolerated well except for minimal nausea, not requiring medical intervention. He had no episode of synocope or arrhythmia during his hospitalization except for those willfully induced during his electrophysiological studies. Hepatitis panel was done during the patient's hospitalization which showed nonreactive hepatitis-B surface antigen but reactive HBS-antibody and HB·Coreantibody consistent with a history of hepatitis-B infection with no active disease. A follow-up electrocardiogram done on July 29, 1983 was without change except for prolongation of the QT interval to .42. The patient underwent another electrophysiological study on August 5, 1983 after having been treated with protocol drugs for 2 weeks. Multiple short runs of nonsustained ventricular tachycardia and a few short runs of sustained ventricular tachycardia at a rate of 340-360/min were inducible, but induction of the ventricular tachycardia required more provocation than during his previous study. During these episodes, the patient's blood pressure was maintained at approximately 60 mm Hg and he had no symptoms. It was felt that the patient was improved on his present regime and would be continued on Amioderone and Lorcanide. He was discharged on August 6, 1983. At the time of discharge the patient's diabetes was under fairly good control. The patient was discharged

TABLE – I

LOWN GRADING OF PREMATURE VENTRICULAR CONTRACTIONS

GRADE	
0	No PVC's
1	Occasional PVC < 30/hr
2	Frequent PVC > 30/hr
3	Multifocal PVC's
4a	Repetitive couplets
4b	Repetitive salvos
5	Early; i.e. failing on T-wave

on Amioderone 400 mgs po t.i.d., Lorcanide 150 mgs b.i.d., Digoxin .25 mgs once a day, and Insulin 16 units regular plus 36 units NPH subcutaneous in the morning and aspirin 6 grains once a day. He was instructed to contine an 1800 calorie ADA diet and resume his normal activities.

Cardiac Electrophysiology Study Report:

I. DIAGNOSES :

The patient has a history of "sudden death: episodes. The patient has a diagnosis of coronary artery disease. The patient is status post cardiac surgery.

II. DEFINITIONS :

S_1 – basic driving stimulus, spontaneous or paced S_2, S_3, S_4 – 1st, 2nd, and 3rd premature stimuli following S_1.

ERP – effective refractory period

Repetitive Response – one or more spontaneous complexes initiated by premature stimuli

Nonsustained Ventricular Tachycardia – 6 or more spontaneous ventricular complexes at a rate of greater than 120/min., self-terminating within 30 seconds

Sustained Ventricular Tachycardia – ventricular tachycardia lasting more than 30 seconds or requiring immediate artificial interruption

STUDY DESCRIPTION

III. CATHETERS

No. 7 french USCI quadripolar	right ventricle
No. 5 french Hanafy	femoral arteries

IV. STIMULATION TECHNIQUES

Right ventricular apex stimulation was performed using incremental pacing from a cycle length of 590 msec. to 300 msec.

Right ventricular apex stimulation was performed using burst pacing from a cycle length of 340 msec, to a cycle length of 290 msec.

Right ventricular apex stimulation was performed using single, double, and triple extrastimuli from a cycle length of 750 msec. to 400 msec.

VII PHARMACOLOGIC INTERVENTIONS

The patient had been pretreated with medication prior to the study. Amiodrone was administered as a oral pretreatment in a daily dose of 1200 mg. Lorcainide was administered as an oral pretreatment in a daily dose of 300 mg.

STUDY RESULTS

VIII. REFRACTORY PERIODS

Site	rhythm	cycle lenght	E.R.P.	Drug
RV apex	sinus	750	350	Amiodarone & Lorcainide
RV apex	paced	600	340	Amiodarone & Lorcainide
RV apex	paced	500	330	Amiodarone & Lorcainide
RV apex	Paced	400	290	Amiodarone & Lorcainide

XIV. DYSRHYTHMIAS

Nonsustained ventricular tachycardia at a cycle length of 360 msec. was induced by triple premature ventricular stimuli at a basic cycle length of 400 msec. at the right ventricular apex and was terminated spontaneously with the patient experiencing no symptoms.

Nonsustained ventricular tachycardia at a cycle length of 360 msec. was induced by rapid ventricular pacing at a basic cycle length of 370 msec. at the right ventricular apex and was terminated spontaneously with the patient experiencing no symptoms.

Sustained ventricular tachycardia at a cycle length of 360 msec. was induced by triple premature ventricular stimuli at a basic cycle length of 500 msec. at the right ventricular apex and was terminated spontaneously with the patient experiencing no symptoms.

XVI. COMPLICATIONS

The patient ecountered no complications during the procedure.

XVII. SUMMARY AND RECOMMENDATIONS

On combination amiodarone-lorcainide therapy, but sustained and nonsustained monomorphic VT were inducible. However, these were well tolerated hemodynamically, with a mean blood pressure of 65 mm hg. and were totally asymptomatic. Additionally, they were more difficult to induce than during the baseline study and significantly slower. These findings especially in view of amiodarone therapy, suggest that the patient has a high likelihood of being protacted from reoccurences of his life-treatening ventricular tachyarrhythmias. The present regimen should be continued.

Discussion :

Sudden cardiac death remains a major challenge for Internists and Cardiologist. More than 400,000 persons die suddenly in the United States every year (nearly one death each minute) [1,2] The experiences gained in the Emergency Rooms, Coronary Care Units, and mobile life-saving units indicates that ventricular fibrillation is the usual basis of primary electrical failure

of the heart and that fibrillation occurs most commonly at the inception of an acute coronary event [2,3,4]. Another observation by mobile units and coronary care unit was that ventricular arrhythmia usually preceeded ventricular fibrillation. Because a majority of the sudden cardiac deaths are due to ventricular arrhythmia, it is reasonable to assume that prevention of sudden cardiac arrhythmias may decrease the incidence of sudden cardiac deaths. Several studies have indicated that the victims of sudden cardiac death do exhibit prolonged periods of electrical instability before the terminal event [4,5].

There has been enough epidemiologic evidence to indicate that premature ventricular contractions are a predictor of increased risk for sudden cardiac death [6]. Rubberman and co-workers studied 1700 men with myocardial infarction and followed them for 24 months. Each person had 2 hours of ECG monitoring. Analysis of survival data showed that people who have complex forms of premature ventricular contractions have a high incidence of sudden death (three times compared to those men who did not have complex premature ventricular contractions) [7]. Complex ventricular premature beats have been defined as multiform, bigeminal, repetitive, or early-cycle ectopic beats. This study also indicates that just the presence of premature ventricular beats does not increase the risk for sudden death. This data could only be extrapolated to the population with premature ventricular contractions who also had myocardial infarction [7,8].

Lown suggested a grading system of PVC's. This grading suggests that Grade I to VI. See Table—I. Lown and his colleagues studied 123 patients who also underwent 24 hour ambulatory holter monitoring. In this study a high grade of PVC's were found among the patients who had multi-vessel coronary artery disease, ventricular wall motion abnormalities, and elevated left ventricular end diastolic pressure [9]. Several studies have suggested that patients with severe left ventricular dysfunction frequently exhibit high grades of ventricular arrhythmia. It has been suggested that the majority of the individuals who showed ventricular premature beats do not require therapy [8,9]. Though it is difficult to make a therapeutic decision about which patients who should be treated with antiarrhythmic medications, certain guidelines could be suggested. Treatment is mandatory for individuals experiencing primary ventricular fibrillation [9,10] (ventricular fibrillation not occurring as a consequence of acute myocardial infarction). This is probably the most common cause of sudden cardiac death outside the hospital [8]. Patients with coronary artery disease who also have anginapectoris, if these patients are found to have Grade IV to V PVC's should be treated [6,8,10]. Those patients who have a prolonged QT interval and also show presence of high grade ventricular arrhythmia or a history of syncope should be treatment with antiarrhythmic medications [11]. Patient's with mitral valve prolaps should be treated with antiarrhythmic mecications unless they have symptomatic ventricular arrhythmia or if there is a family hsitory of sudden death or there is a concommitant QT interval prolongation.

At the present time ambulatory ECG monitoring is the most effective method for disclosing arrhythmia [3,6]. On occasional cases it is necessary to prolong this 24 hour period to 48 or 72 hour period. In this patient who show extreme variability of cardiac arrhythmia, exercise testing could also be useful in individuals who are at high risk. Coronary artery disease is found to be the most important cause of exercise provoked ventricular arrhythmia [5,7]. Electrophysiological studies are an extremely useful test both to expose arrhythmia and to determine a suitable antiarrhythmic therapy [12,13]. At the present time electrophysiological studies are suggested to only a selective group of patients who have shown life-threatening cardiac arrhythmias and who are difficult to manage with a standard antiarrhthmic therapy. Patients who exhibit high frequency of PVC's, serial drug testing should be carried out to select an ideal drug therapy. If a drug succeeds to suppress Grade IV PVC's, salvos, and ventricular tachycardia, the patient could be started on the selected antiarrhthmic drug. A 50 % reduction of PVC's are also considerable to be a therapeutic success. A total elimination of PVC's is usually not possible.

In the presented patient it was only possible after electrophysiologic studies to select the appropriate antiarrhythmic therapy for the patient because he had an extremely resistant kind of ventricular arrhythmia. Without electrophysiological studies, if would not have been possible to select the present antiarrhythmic regime.

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