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Repetitive Ventricular Fibrillation
Preceded by Both ST Segment Depression and Elevation during
Acute Myocardial Ischemia*

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A patient had repetitive ventricular fibrillation preceded by alternating ST segment depression and elevation. The ECG changes were confined to the precordial leads only, reflecting subendocardial and transmural ischemia, respectively. It is speculated that the patient exhibited consecutive episodes of subtotal and total coronary occlusion, both episodes being critical enough to induce lethal arrhythmias.

Both partial, as well as total, occlusion of a major coronary artery may lower the threshold of ventricular vulnerability, and the greater the ischemia of the myocardium, the more it is primed to develop fatal ventricular arrhythmias. Ischemic ST segment elevation and ST segment depression may represent either a different distribution of ischemia in the ventricle (transmural vs subendocardial), or a different spatial relationship between the boundary of the ischemic segment and the electrocardiographic leads used. It is the purpose of this manuscript to describe a patient with recurrent anterior wall myocardial ischemia, in whom both ST segment depression and elevation were found to precede repetitive ventricular fibrillation (VF) attacks.

CASE REPORT

A 75-year-old man with no previous history of cardiac disease or cardiac risk factors, was admitted because of recurrent chest pain. On admission, blood pressure was 100/70 mm Hg and pulse rate was 68 bpm. Physical examination was unremarkable. The baseline electrocardiogram (ECG) including QT interval, showed normal findings. Serum electrolytes and cardiac enzyme levels were also normal. Nitrate therapy was instituted for treatment of angina and the patient remained asymptomatic until ten hours later, when he complained again of retrosternal pain. Repeated examination revealed blood pressure of 120/80 mm Hg and a pulse rate of 84 bpm. An ECG showed ST segment depression of up to 3 mm in the precordial leads (Fig 1A). During the episode of ischemic pain, bedside ECG was constantly recorded. After three minutes, the ECG disclosed further increase of heart rate with a short run of ventricular tachycardia degenerating into VF (Fig 1B). Direct current shock was immediately applied and sinus rhythm was restored. The ST segment returned to the preischemic baseline pattern with no subsequent electrocardiographic evidence of infarction. At this stage, a bolus injection of lidocaine (1 mg/kg) was given and a continuous infusion was started together with intravenous administration of heparin. Four hours later, still under lidocaine infusion, the patient complained again of severe sharp precordial pain. Blood pressure was unchanged. Cardiac monitoring demonstrated sinus rhythm with frequent premature ventricular contractions. At this time, the ST segment was markedly elevated.

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FIGURE 2A. Electrocardiogram taken at 10:15 PST during chest pain, showing marked ST segment elevation in precordial leads and occasional ventricular ectopic beat in V₂. B. 17 minutes later, continuous monitoring demonstrating sinus tachycardia with persistent ST segment elevation, interrupted by consecutive R on T ventricular extra-systoles leading to ventricular fibrillation. C. 13 hours later, ECG disclosed transmural anterior wall myocardial infarction.

in the anterior chest leads (Fig 2A). Unifocal premature ventricular beats initially seemed to respond well to an additional bolus of lidocaine. Continuous monitoring revealed gradual increase of sinus rate with persistent ST segment elevation until sinus rhythm was interrupted by VF (Fig 2B) initiated by ventricular complex encroaching on the T wave. Defibrillation was accomplished with restoration of sinus rhythm. Serial ECGs and cardiac enzyme assays were compatible with AMI (Fig 2C). The patient was discharged after a three-week period of uneventful recovery.

DISCUSSION

ST segment deviation is accepted as the most reliable ECG marker of myocardial ischemia. ST depression may represent either diffuse narrowing of a major coronary artery, or subtotal spastic occlusion superimposed on pre-existing atherosclerotic lesion. This pattern coupled with coronary spasm, can also be found in the absence of underlying atherosclerotic disease. Total occlusion of a large epicardial coronary artery may be caused by coronary spasm or thrombosis. It is usually associated with ST elevation, suggesting more severe myocardial ischemia than depression. The prevalence of arrhythmias during coronary occlusion is well correlated with the magnitude of the ST shift which may well reflect the intensity of coronary flow abnormality and the severity of ischemia. In variant angina, ventricular arrhythmia and ectopy occur more frequently with transmural (ST elevation) rather than with subendocardial (ST depression) ischemia.

In a series of studies investigating the VF during AMI and its mode of onset, there is, to the best of our knowledge, no reported evidence of different electrocardiographic ischemic patterns leading to VF in the same patient. In the majority of cases published, ST segment elevation was the predominant feature preceding the occurrence of VF. Downsloping ST segment depressions were antecedent manifestations in several cases of sudden death documented by Holter monitoring. However, precordial ST segment depression seen in ambulatory monitoring might be misleading and does not necessarily reflect anterior wall subendocardial ischemia. One might attribute these findings to benign electrical phenomena reflecting “concealed” transmural ischemia affecting the inferior wall. It is an important question whether this ischemic pattern constitutes an adverse risk factor in predicting the appearance of ventricular arrhythmias, and thus, increases the likelihood of sudden death. In spite of apparently favorable prognosis of ischemic ST segment depression concerning further evolution of cardiac arrhythmias, previous studies have also provided substantial evidence that ST segment depression is caused by rapid heart rates even in the presence of normal coronary arteries. In our patient, it is unlikely that increase of demand could account for a nontransmural reduction of coronary flow as heart rate reached 84 bpm once the ST segment configuration was already apparent (Fig 1) with no remarkable changes of blood pressure levels. In the absence of opponent changes in the diaphragmatic leads, it most probably represents subendocardial ischemia affecting the adjacent anterior wall.
In both instances of VF, heart rate tended to increase gradually prior to the onset of the event. They differed in antecedent ST segment deviation and ectopic activity (ventricular tachycardia versus short cycle ectopic beats), the median time between onset of chest pain and interruption of sinus rhythm, and the propensity for evolving infarction. Necrosis in our patient occurred only after the second ischemic episode.

A common denominator of both episodes of VF seemed to be active myocardial ischemia which can undoubtedly decrease the threshold of ventricular vulnerability. We may speculate that our patient exhibited recurrent spastic occlusion of the left anterior descending coronary artery leading to consecutive episodes of subtotal and total occlusion of the artery, episodes which were both critical enough to induce lethal ischemic arrhythmias.

REFERENCES

Noninvasive Nasal Mask-Assisted Ventilation in Respiratory Failure of Duchenne Muscular Dystrophy*

David Segall, M.B., B.S., F.C.C.P.

The effects of noninvasive nasal mask-assisted ventilation were studied in two patients with chronic respiratory failure due to Duchenne’s muscular dystrophy. Observations were made with continuous recordings of transcutaneous CO2 and O2 and ear oximetry. In one case, the mean tcPco2 fell from 72 mm Hg to 43 mm Hg. The tcPo2 increased from 38 mm Hg to 62 mm Hg without supplementary oxygen. In the second case, hypercapnia associated with supplementary oxygen was corrected, and at five months follow-up, hypoxemia was corrected without supplementary oxygen. Prolonged therapy during sleep has resulted in sustained clinical improvement for more than 18 months.

Successful mask intermittent positive pressure ventilation (NIPPV) has recently been reported in respiratory failure due to neuromuscular diseases by several authors.14 We wish to report two cases of Duchenne’s muscular dystrophy (DMD) treated with NIPPV during the hours of sleep. In both cases, there has been sustained clinical and physiologic improvement for more than 18 months. Both patients reported an increased sense of well being, absence of previously reported daytime hypersomnolence, and less daytime dyspnea. The effectiveness of this technique is graphically demonstrated.

CASE REPORTS

CASE 1

A 29-year-old white man had DMD diagnosed at age five years. At age 28 years, he complained of dyspnea at night, daytime hypersomnolence, headaches, and palpitations. On physical examination, he was unable to maintain a sitting position without support. There was severe generalized muscle wasting with contractures of the legs. There was deformity of the chest with pectus carinatum and kyphoscoliosis. The pulse was 140 beats per minute, respiration, 30 per minute, and shallow. The FVC was 460 ml or 10 percent predicted. An arterial blood gas (ABG) analysis awake on room air yielded the following results: pH, 7.37; PaCO2, 60 mm Hg; and PaO2, 55 mm Hg. He was admitted to the hospital and placed on NIPPV.

Two months following discharge, using NIPPV at night at home, he was much improved. He was less short of breath during the day with no daytime hypersomnolence. The FVC was 520 ml. An ABG analysis awake on room air yielded the following results: pH, 7.36; PaCO2, 50; PaO2, 69.

Figure 1 shows results of continuous transcutaneous recordings of PC02 and PO2 on room air and subsequently on NIPPV at a rate of 15 breaths per minute, and Vr, 400 ml on room air. The tcPC02 fell from a mean of 72.3 mm Hg to 43.2 mm Hg. The tcPO2 increased from a mean of 37.6 mm Hg to 61.8 mm Hg. Ear oximetry findings ranged from a low of 71 percent O2 saturation without support to a high of 96 percent.

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