Sudden Death of a Diabetic Patient during Holter Monitoring

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The sudden death of a diabetic patient (NIDDM) during Holter monitoring is described. The patient was a 64-yr-old male with an 11-yr history of diabetic nephropathy, retinopathy, autonomic nervous dysfunction, and old myocardial infarction. In spite of all these complications, he remained asymptomatic and eventually resumed his normal activities, including a daily 1-h walk, until his sudden death. The cause of death was considered to be cardiac disease, but was not confirmed by autopsy. A detailed analysis of his Holter monitoring is given. (Internal Medicine 31: 91–93, 1992)

Key words: asymptomatic myocardial ischemia, sudden death, Holter monitoring, diabetes mellitus

Introduction

The incidence of myocardial infarction among diabetic patients is high, with a prognosis that is considered to be poor (1). Triple vessel coronary artery disease (2) is also more frequent in diabetic patients than in non-diabetic patients. Furthermore, autonomic nervous dysfunction, a known diabetic complication (2–5), can induce sudden death and/or respiratory arrest apart from cardiac disease. In our opinion, the increased number of documented sudden deaths revealed during long-term electrocardiographic recordings accompanied by Holter monitoring, is not particularly relevant to diabetic patients who have also undertaken Holter monitoring. We know of only one case where sudden death occurred in a diabetic patient during Holter monitoring. This patient and a complete analysis of his ambulatory electrocardiograms are the subject of the present study.

Case Report

The patient was a 64-yr-old male with a history of 11 yr of diabetes mellitus (NIDDM), and 6 yr of hypertension. He also was afflicted with nephropathy, retinopathy (Scott 3), neuropathy, and autonomic nervous dysfunction. He was stricken with a myocardial infarction on April 10, 1986, and completely recovered 1 month later. The patient underwent a gallstone resection on September 25 of that same year. His medications were tolbutamide, dipyridamol, β -blocker, and warfarin until

his death. Although he did not drink, he smoked 20–30 cigarettes a day. He visited our hospital on July 11, 1989 for a routine stomach examination, and underwent a yearly X-ray examination of his stomach after his gall bladder resection operation. It was during one of these examinations that abnormal changes in his stomach mucosa were noted. Stomach fibroscopy performed 3 days later revealed malignancy in the early stages. Laboratory and physiological tests, and Holter monitoring were done prior to gastric cancer surgery. Fasting venous blood was drawn and analyzed with an autoanalyzer (Hitachi 705, Hitachi, Japan). The following results were obtained: erythrocytes $468 \times 10^4/\text{mm}^3$; leucocytes $7,800/\text{mm}^3$; hemoglobin 14.6 g/dl; hematocrit 51.3%; platelets $31 \times 10^4/\text{mm}^3$ in venous blood; total protein 7.2 g/dl; uric acid 5.9 mg/dl; BUN 19.1 mg/dl; creatinine 0.92 g/dl; albumin 3.0 g/dl; A/G 0.71; GOT 10.1 IU/l; cholesterol 287 mg/dl; triglyceride 225 mg/dl; HDL cholesterol 25.6 mg/dl; Na 141 mEq/l; K 3.9 mEq/l; Cl 101 mEq/l; HbA_{1c} 9.1%; fasting blood glucose (morning) 109 mg/dl in blood serum; urine protein (+), and urine sugar (+). Electrocardiogram (ECG) disclosed right bundle branch block. Heart rate was 73/min and blood pressure 158/94 mmHg. Both patella tendon and achilles tendon reflex were negative. Chest X-ray visualized moderate enlargement of the heart (cardiothoracic ratio 60.7%). Coefficient of variation of R-R intervals was calculated to be 2.1% by R-R estimator (TM 50, Japan Electric Co., and Sanei, Japan) measuring 100 heart beats/min at bed rest. His ht was 167 cm, and his wt was 74 kg. Holter

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monitoring was performed using Oxford Medical Systems and Oxford Medilog 2 MR 14 recorder. Holter monitoring was started on August 10, 1989 at 9:23 AM.

The patient never complained of chest pains, and he continued to take his daily 1-h morning walk until the day of his death. Shortly after finishing his dinner at 18:15 he complained of nausea and epigastralgia, and went to the toilet. He made no mention of having a headache at this time. Five minutes later his wife found him lying unconscious on his back. His face was ashen and covered with a cold sweat. His wife called for an ambulance at 19:10 and he was admitted to our hospital at 19:27. His face was pale, and both pulse and respiration were absent. He appeared to have expired, and ECG was unable to record any heartbeat. We attempted electric shock, heart massage, and artificial respiration using an umbue bag. The patient remained unresponsive, and was declared dead at 19:40.

The ECG obtained on July 11, 1989 (Fig. 1) reveals a right bundle branch block, and an old myocardial infarction. His earlier medical records confirmed that he had been stricken with an inferior myocardial infarction. We analyzed the cause of death through his Holter monitoring record. Figure 2 shows the Holter electrocardiogram from 18:24 to 18:53. It shows a normal heart rate, but, as seen in other ECG tracings, a slight ST depression was persistently observed. Figure 3 shows the Holter electro-

cardiogram from 18:54 to 19:21, including the onset of this occurrence. Figure 4 shows the Holter ECG at the time of attempted resuscitation, and the patient's eventual death. The Holter monitor showed a normal heart rate until 18:54, and apparent bradycardia and atrial-ventricular (A-V) block at 18:55. The bradycardia with A-V block continued, and sinus arrest, ventricular extra beats, and escaped beats appeared at 18:55. Sinus brady-

Fig. 1. ECG recorded on July 14, 1989

Fig. 2. Holter cardiogram showing normal heart rate just before lethal arrhythmia.

Fig. 3. Holter ECG at the time of lethal arrhythmia.

Fig. 4. Holter ECG at the time of resuscitation and death.

cardia became more apparent, and ventricular rhythms appeared at 19:02. His heart stopped beating at 19:09. When he arrived at the hospital at 19:27, the heartbeat had completely stopped. Some deflections were recorded, but they were temporary due to heart massage. We declared him dead at 19:40. In summary, ST and T depression were present on the Holter monitoring record, but the patient remained asymptomatic. Bradycardia then appeared, followed by more severe bradyarrhythmias, ventricular fibrillation and, eventually, death.

Discussion

Sudden death is defined as unexpected death within 60 min to 24 h after the onset of symptoms (6). Lawrence and Minkel (7) reported that most cases of sudden death that occur within 60 min after the onset of symptoms are due to cardiac arrhythmias such as ventricular fibrillation. The patient under study had suffered from an inferior myocardial infarction 3 yr before, was obese, smoked, and was diabetic. Although the patient complained of nausea and vomiting, there was no trace of blood nor blood coagula in his vomitus, and no headache. We feel, therefore, that it is not likely that the patient died from cerebral hemorrhage, or rupture of the digestive tract. Since it is well known that nausea and vomiting often occur in patients with acute occlusion of the coronary arteries, we are convinced that the cause of death in this case was cardiac in origin and that acute occlusion of the right coronary artery and the eventual development of bradyarrhythmia should be considered as the cause of death.

The multiple risk factors that may result in heart failure and sudden death include: hypertension, diabetes mellitus, hypercholesterolemia, and a history of myocardial infarction (8, 9). William and Kannel (10) reported that the clinical risk factors leading to sudden death are: hypertension, hypercholesterolemia, diabetes mellitus,

left ventricular hypertrophy, tobacco, and obesity. Nikolic et al (8) reported six cases of sudden death during Holter monitoring, five of which developed ventricular fibrillation, and one who developed bradycardia. To our knowledge, there have been no reports in the literature describing the sudden death of a diabetic patient during Holter monitoring. The present patient could likely be considered as someone who could well die a sudden death. He was diabetic, a heavy smoker, hypertensive, developed diabetic nephropathy, retinopathy, neuropathy, had autonomic nervous dysfunction and a past history of myocardial infarction. He lived quietly and managed to include a daily 60-min walk up until the time of his death. His medications included warfarin, dipyridamole, tolbutamide, and β -blocker. It should be noted that he did not complain of any symptoms that would suggest ischemic heart disease.

There were no signs of dyspnea, chest pain, heart palpitations, edema, nor tachycardia. His only complaint was epigastralgia, which occurred just prior to the onset of this episode. Although most instances of sudden death do not show any characteristic warnings of any duration, the asymptomatic daily life of this patient deserves notice (9). The cause of his sudden death was apparently cardiac failure, and it is important to place consideration on the fact that he did not complain of any apparent symptoms before he died. There are many reports of asymptomatic diabetic myocardial infarction or angina, and it is critical to be especially alert when medically managing diabetic patients (1, 2, 9–11). The pathogenesis of asymptomatic myocardial infarction should be the subject of intensive study for further clarification.

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