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Usefulness of the Implantable Loop Recorder to Detect Syncope in a Patient With Coronary Spastic Angina

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An 80-year-old man was urgently transported because of unconsciousness. On admission, the chest electrocardiogram showed a negative T-wave; there was a high possibility of cardiac syncope. No disorder was detected on coronary angiography or on cardiac electrophysiological study. Although he was diagnosed as having coronary spastic angina, a loop recorder was implanted because syncope didn't occur during acetylcholine load test. Syncope occurred again, a loop recorder detected increasing ST elevation and atrioventricular junctional rhythm without high infrequent pulse or tachyarrhythmia. Decreased blood pressure and syncope due to Bezold-Jarisch reflex with coronary vasospasm was strongly suspected. With benidipine and nicorandil, he was discharged without recurrence of syncope for 3 years thereafter.

Key words: syncope, coronary spastic angina, Bezold-Jarisch reflex, implantable loop recorder, calcium channel blocker

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INTRODUCTION

An implantable loop recorder（ILR）is an electrocardiograph implanted under the skin for patients with syncope in whom a specific cause cannot be identified. It is a very effective measure for diagnosing the cause of syncope by capturing electrocardiographic observations that occur during syncope［1］. The ILR automatically records cardiac arrest, bradyarrhythmia, and tachyarrhythmia, but not changes in ST elevation. Electrocardiograms（ECG）can be stored manually for a few minutes as an event record when symptoms occur. We describe our experience with a case in which the ILR was effective for diagnosing the cause of syncope in a patient with coronary spastic angina（CSA）.

CASE REPORT

An 80-year-old man was urgently transported because of transient unconsciousness when sitting. His past medical history was prostatic cancer but it was cured, and he was not taking any internal medicine. He had a smoking history（10 cigarettes a day）, but no intake of alcohol. Transient loss of consciousness occurred without convulsion or craniocerebral trauma. He recovered completely after several minutes in the recumbent position; however, he had experienced syncope three times in the recumbent position within 2 years, so he was hospitalized for examination and additional treatment. Once was associated with chest discomfort, but the accessory symptom was not clear about other twice.

On admission, he was clear and his vital signs were as follows: blood pressure of 121/59 mmHg, pulse rate of 69 beats per minute, peripheral oxygen saturation of 99% on room air, and body temperature of 35.6°C. Head trauma and tongue biting were

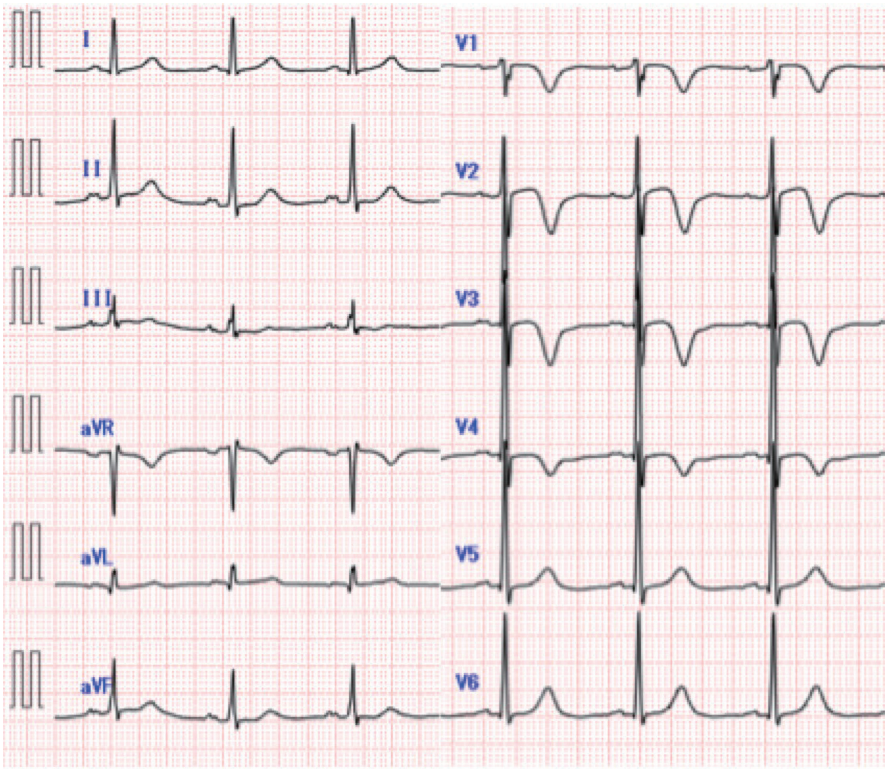

Fig. 1. An electrocardiogram revealed sinus rhythm and negative T-waves in lead $V_{1\text{-}4}$.

absent. No heart murmurs nor rales were audible. Neurologic examinations revealed no abnormalities. An electrocardiogram revealed sinus rhythm and negative T-waves in lead $V_{1\text{-}4}$ (Fig. 1). In chest X-ray, the cardiothoracic ratio was 50.1% and no pulmonary congestion was observed. Blood test results revealed normal levels of troponin I 0.03 ng/mL, creatine kinase 81 IU/L, and creatine kinase-myocardial band 10 IU/L. Liver function and renal function were normal, and anemia and an electrolyte abnormality were absent too. The transthoracic echocardiogram showed a left ventricular diastolic diameter of 38 mm, systolic diameter of 27 mm, and an ejection fraction of 60%. A wall motion abnormality, left ventricular hypertrophy and valvular disease were absent. We did not observe significant stricture in either carotid artery with carotid duplex ultrasonography.

He was considered to be at high risk for cardiogenic syncope due to his history of syncope in the recumbent position and new negative T-waves in the ECG, so he underwent coronary angiography and cardiac electrophysiological study on day 2. The coronary angiogram showed moderate stenosis in segment 7 of the left anterior descending artery and segment 3 of the right coronary artery, without severe stenosis, and hypoplastic circumflex artery (Fig. 2a, b). The acetylcholine load test (50 μg) indicated 90% stenosis in segment 4 of the right coronary artery. With 100 μg of acetylcholine in the left coronary artery, 99% stenosis was found in the anterior descending artery with chest tightness, and the ECG showed complete left bundle branch block (Fig. 2c, d). Transient unconsciousness did not occur during the examination. He was diagnosed as having CSA, and calcium channel blocker (4mg tablets of benidipine) administration was started. However, we could not confirm CSA as the cause of syncope. The cardiac electrophysiological study did not show abnormality in the sinus node or atrioventricular conduction, and tachyarrhythmia was not induced, too. Therefore, the cause of syncope was not clear. A loop recorder was implanted near the V_3 lead on day 3, and he was discharged from the hospital.

Transient unconsciousness with chest tightness occurred again in the sitting position during consulting the urologic outpatient on the day of hospital discharge. The ILR was reported by his wife who

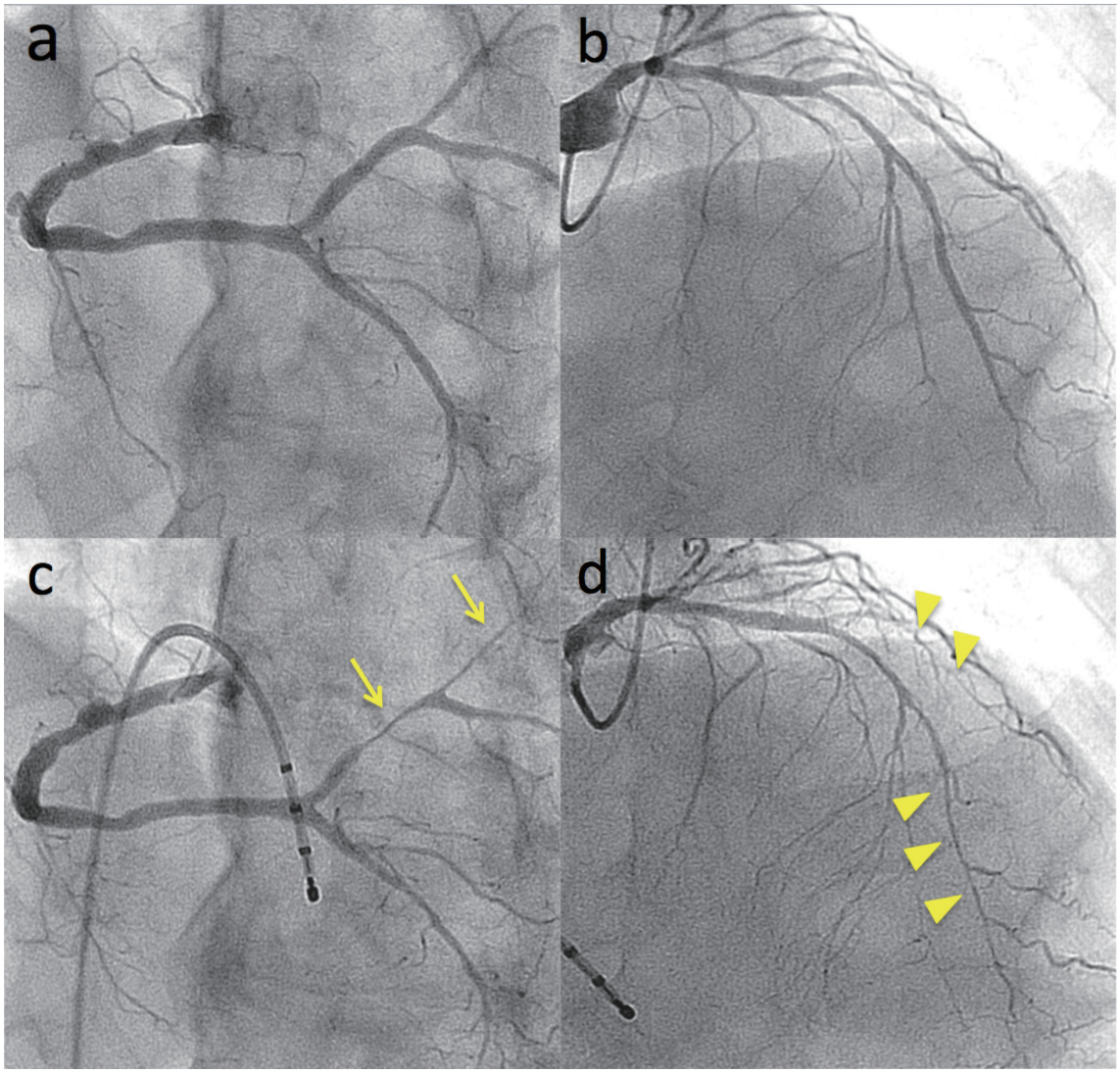

Fig. 2. The coronary angiogram
The coronary angiogram showing 50% stenosis of the distal segment of the right coronary artery with left cranial angulation (a) and 75% stenosis of the middle segment of the left descending artery with right cranial angulation (b). The right coronary angiogram shows 90% stenosis (arrow) of the atrioventricular node branch after acetylcholine load (50 μg) (c). The left coronary angiogram shows 99% stenosis with delay of the left descending artery, including the diagonal branch (arrow hedad), after acetylcholine load (100 μg) (d).

manually pressed the event button, and he recovered to consciousness after several minutes. His blood pressure decreased, with the systolic blood pressure being 66 mmHg during syncope. The event record of the loop recorder showed sinus arrest and junctional rhythm for 48 beats per minute, and increased ST elevation from about 80 seconds before the episode of unconsciousness (Fig. 3). This means that CSA occurred when he experienced syncope, although bradyarrhythmia or tachyarrhythmia did not manifest with syncope. There was also no elevation of the myocardial biomarker. He was strongly suspected as having syncope induced by CSA because hypotension was documented during syncope.

We recommended lifestyle changes, including quitting smoking, and avoiding stress, and prescribed three 5mg tablets of nicorandil. We confirmed no recurrence of syncope for three days and discharged him from the hospital. After verifying no events without recurrence of syncope for 3 months, the loop recorder was removed. No recurrence of syncope occurred for 3 years thereafter.

DISCUSSION

The occurrence rate of syncope is unknown;

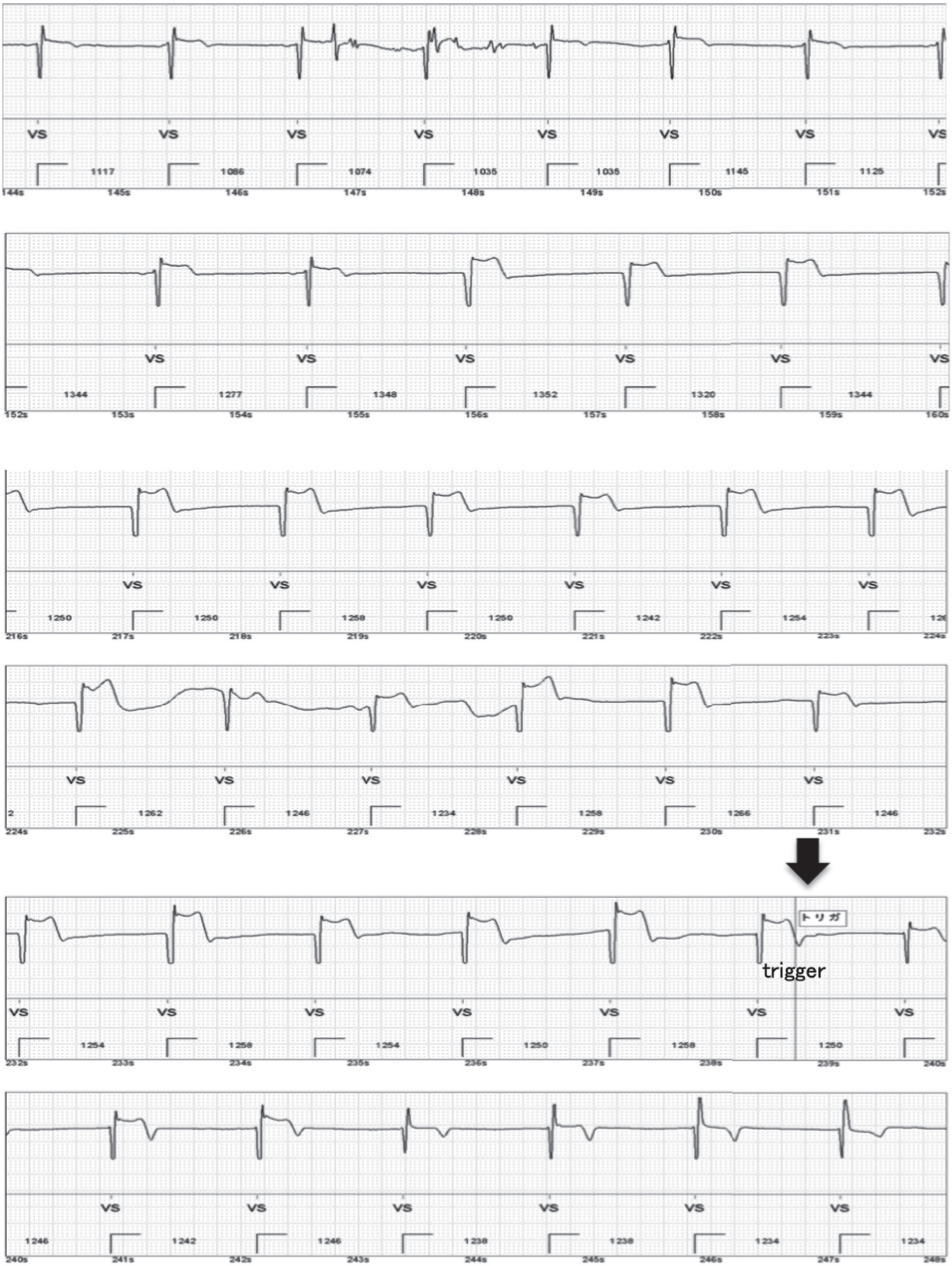

Fig. 3. Implantable loop recorder shows ST elevation and atrioventricular junction rhythm for approximately 80 seconds when his wife pushed the event button（arrow）.

however, the presumed occurrence rate from the Framingham study is 0.62% of the entire population in Japan, and patients with an unspecified cause of syncope after various examinations account for 24,000/year. Vasovagal syncope occurred most frequently in patients with syncope, and the rate of cardiogenic syncope was 9.5% [2]. The event recorder can be mounted for a longer time than a Holter ECG, but the event onset during monitoring is rare [3]. The battery life of an implantable loop recorder is about 3 years, and even after conducting various examinations, a diagnosis can be made for about 2/3 of patients with syncope of unknown cause [4]. Moreover, appropriate treatment can be selected based on the diagnostic result [5].

A patient with cardiogenic syncope has a poor prognosis than one with noncardiogenic syncope, and examination is required for patients with existing heart disease or those considered as high risk for syncope during work or in the recumbent position [6]. In the present patient, CSA was diagnosed; yet, syncope did not occur during the acetylcholine load test, it was therefore unknown whether CSA caused the syncope. He was not diagnosed based on findings from the cardiac electrophysiological study, but the loop recorder was implanted because of his history of syncope in the recumbent position, which put him at high risk for cardiogenic syncope. Because ST elevation was detected by using the loop recorder during syncope, it was shown that coronary spasm was involved.

In patients with CSA, most episodes of syncope are caused by the occurrence of arrhythmia with ischemia [7], sometimes with Bezold-Jarisch reflex (BJR) [8]. BJR is thought to result from some chemical stimulation within the activated unmyelinated sensory nerve fibers (C-fibers) of the heart that pass via the vagus nerve to the brainstem. This causes a vasodepressor response, the classical BJR [9]. Activation of BJR leads to decreased vasomotor output, bradycardia, and hypotension [10]. BJR is mainly found in inferior to posterior wall infarction due to differences in the distribution of the C-fiber afferent receptor, but it can also occur in anterior wall infarction. Gaideon et al. reported that BJR occurred in 2 of 28 (7.8%) patients with anterior wall acute myocardial infarction and in 10 of 24 (41.7%) patients with inferior wall infarction [11]. In the present case, increasing ST elevation was detected near the V3 lead by the implantable loop recorder; thus, complete occlusion of the left anterior descending artery was suggested to be the cause of syncope. Bradyarrhythmia or tachyarrhythmia did not occur with syncope, but bradycardia and hypotension did occur with syncope. His syncope was finally considered due to BJR caused by CSA.

Usually, the loop recorder does not record automatically by ST change, but events can be recorded by manually pressing a button. In the present case, the cardiac rate was 50 beats/minute during syncope; severe bradyarrhythmia was not recorded automatically, but because we provided sufficient guidance to the patient and his wife, the wife was able to press the event button and manually record the event.

Regarding pharmacotherapy for CSA, medications such as nitric acid, a calcium channel blocker, and nicorandil are effective. Calcium channel blockers are the first-line therapy for CSA. These agents prevent vasoconstriction and promote vasodilation in the coronary vasculature, thereby alleviating symptoms. One study demonstrated that the use of calcium channel blocker therapy was an independent predictor of myocardial infarct-free survival in CSA patients [12]. The long-acting nitrates are also effective in alleviating symptoms, but the occurrence of nitrate tolerance makes them a less desirable first-line approach.

CONCLUSION

For patients with syncope, risk stratification should be conducted when a high-risk symptom of cardiogenic syncope such as during recumbent position is observed; additionally, when the cause of syncope cannot be determined from a comprehensive evaluation or a specific treatment method cannot be decided, an implantable loop recorder should be considered. With the implantable loop recorder, sufficient guidance about the device should be provided to the patient, and in cases similar to ours, the possibility of syncope due to BJR caused by coronary vasospasm should be investigated.

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None.

CONFLICTS OF INTEREST

The authors declare that there is no conflict of interest.

REFERENCES

1) Krahn AD, Klein GJ, Norris C, Yee R. The etiology of syncope in patients with negative tilt table and electrophysiologic testing. *Circulation* 1995;92:1819-24.
2) Soteriades ES, Evans JC, Larson MG, *et al*. Incidence and prognosis of syncope. *N Engl J Med* 2002;347:878-85.
3) Bass EB, Curtiss EI, Arena VC, *et al*. The duration of holter monitoring in patients with syncope. *Arch Intern Med* 1990;150:1073-8.
4) Brignole M, Vardas P, Hoffman E, *et al*. Indications for the use of diagnostic implantable and external ECG loop recorders. *Europace* 2009;11:671-87.
5) Brignole M, Sutton R, Menozzi C, *et al*. Early application of an implantable loop recorder allows effective specific therapy in patients with recurrent suspected neurally mediated syncope. *Eur Heart J* 2006;27:1085-92.
6) Brignole M, Moya A, de Lange FJ, *et al*. 2018 ESC Guidelines for the diagnosis and management of syncope. *Eur Heart J* 2018;39:1883-948.
7) Myerburg RJ, Kessler KM, Mallon SM, *et al*. Life-threatening ventricular arrhythmias in patients with silent myocardial ischemia due to coronary-artery spasm. *N Engl J Med* 1992;326:1451-5.
8) Alboni P, Brignole M, Menozzi C, *et al*. Diagnostic value of history in patients with syncope with or without heart disease. *J Am Coll Cardiol* 2001;37:1921-8.
9) Grimm DR. Neurally mediated syncope: a review of cardiac and arterial receptors. *J Clin Neurophysiol* 1997;14:170-82.
10) Campagna JA, Carter C. Clinical relevance of the bezold-jarisch reflex. *Anesthesiology* 2003;98:1250-60.
11) Koren G, Weiss AT, Ben-David Y, Hasin Y, Luria MH, Gotsman MS. Bradycardia and hypotenstion following reperfusion with streptokinase (Bezold-Jarisch reflex): a sign of coronary thrombolysis and myocardial salvage. *Am Heart J* 1986;112:468-71.
12) Yasue H, Takizawa A, Nagano M, *et al*. Long-term prognosis for patients with variant angina and influential factors. *Circulatio*n 1988;78:1-9.