Right Bundle Branch Block and Coved-Type ST-Segment Elevation Mimicked by Acute Cholecystitis

Masato Furuhashi, MD; Kikuya Uno, MD; Shin-ichiro Satoh, MD*; Kohki Hoshikawa, MD*; Eiji Sakai, MD*; Kazufumi Tsuchihashi, MD; Kazuaki Shimamoto, MD

A 69-year-old woman had acute cholecystitis that mimicked right bundle branch block with coved-type ST-segment elevation in the precordial electrocardiogram leads (Brugada-type ST shift). The patient did not have obvious heart disease, syncope, or a family history of sudden death. The coved-type ST-segment elevation disappeared as the acute inflammation subsided. Intravenous administration of pilsicainide, a pure sodium channel blocker, could reproduce the Brugada-type ST shift. This is the first report of the Brugada-type ST shift occurring in association with acute cholecystitis.

Key Words: Acute cholecystitis; Antidepressant; Brugada syndrome; Pilsicainide; Signal-averaged electrocardiogram

A characteristic electrocardiogram (ECG) consisting of right bundle branch block and coved (or saddle-back) type ST-segment elevation in the precordial leads (Brugada-type ST shift) has been associated with aborted cardiac sudden death without structural heart diseases. Because of the increased awareness of this ECG evidence, the Brugada-type ST shift is being discovered incidentally in more and more asymptomatic subjects; we previously reported a rate of 0.14% in the general Japanese population.

ECG changes consistent with myocardial ischemia have been associated with inflammatory conditions, such as acute cholecystitis, pancreatitis and myocarditis; usually in the form of T-wave inversion, ST-segment depression, but rarely ST-segment elevation regardless of the presence of coronary artery disease. However, Brugada-type ST shift has not been reported in such conditions. We describe a patient with asymptomatic Brugada-type ST shift mimicked by acute cholecystitis.

Case Report

A 69-year-old woman was referred to hospital because of abdominal discomfort and fever. She did not have a history of syncope or a family history of sudden cardiac death. She had previously been diagnosed with psychotic depression and treated with low-dose antidepressants, including a tetracyclic antidepressant (mianserin, 30mg/day), a tricyclic antidepressant (amoxapine, 10mg/day), and a serotonin–noradrenalin reuptake inhibitor (trazodone, 50mg/day). The patient continued to take these antidepressants after admission. Her blood pressure was 116/70 mmHg, pulse rate was 84 beats/min and regular, and temperature was 38.7°C. There were no abnormalities in heart sounds or respiratory sounds. Abdominal examination revealed mild tenderness without rebound in the upper abdomen. The results of hematological tests on admission showed elevated white blood cell count (11,400/µl), C-reactive protein (15.1mg/dl), aspartate aminotransferase (197IU/L), alanine aminotransferase (173IU/L), alkaline phosphatase (334IU/L), and ß-glutamyl transpeptidase (64IU/L). Serum bilirubin, amylase, electrolytes, and thyroid function were normal. The patient showed a negative reaction to a troponin T rapid test on admission. Serial changes of creatine kinase were normal with the MB fraction less than 5%. Chest X-ray revealed no pulmonary edema or cardiac enlargement. Abdominal ultrasound and computed tomography showed the appearance of gallbladder stones in a thickened gallbladder with pericholecystic fluid but without ductal dilation. These findings led to the diagnosis of acute cholecystitis.

The 12-lead ECG on admission showed sinus rhythm with normal PQ, QT, and corrected QT intervals, a heart rate of 82 beats/min, incomplete right bundle branch block, and ST-segment elevation in V1 and V2 (Fig 1A). The ECG abnormality was compatible with a Brugada-type ST shift consisting of a coved-type elevation in V1 and a saddle-back type in V2. The patient was treated with intravenous drips and antibiotic therapy (cefotiam, CTM). During the 7 days after hospitalization, the Brugada-type ST shift gradually disappeared concomitant with an improvement in the acute inflammation and a lessening of the fever (Fig 1B). Echocardiographic examination showed no obvious heart diseases, including those of the right ventricle. Cardiac ischemia and ventricular arrhythmias were not induced during a treadmill exercise test. Holter monitoring revealed 67 isolated premature atrial contractions and only one isolated premature ventricular contraction during a 24h period. Flocculation of the ST-segment elevation during 24-h ST-segment trend recordings was not detected. The late potential was negative on the signal-averaged ECG: RMS (root mean square voltage of the terminal 40ms) was 34µV, LAS (low amplitude signal below 40µV of the...
terminal QRS complex) was 34 ms, and f-QRS (the filtered QRS duration) was 90 ms. Intravenous administration of pilsicainide, a pure sodium channel blocker, (50 mg over a 5 min period) induced the same Brugada-type ST shift as shown by ECG on admission (Fig 2). Coronary angiography did not reveal any stenotic lesions. Coronary spasm was not induced by intracoronary injection of acetylcholine. Left and right ventriculograms showed no structural abnormalities. The His-ventricular (HV) interval was 40 ms during sinus rhythm in the control state. Programmed extra stimulation to document ventricular fibrillation was not performed at the patient’s request. Serial ECG recordings did not show the ST-segment elevation during a follow-up period of 3 months.

**Discussion**

The Brugada-type ST shift has been identified in various cardiac and non-cardiac diseases, such as myocardial ischemia, arrhythmogenic right ventricular cardiomyopathy and accidental hypothermia. Despite marked ECG abnormalities, myocardial cell damage, coronary hypoperfusion, coronary artery spasm, myocarditis, pericarditis or cardiomyopathy were not evident in the present case. Interestingly, slow regression of the coved-type ST-segment elevation was observed as there was improvement in the acute inflammation and fever. The mechanism of ST-segment elevation in acute cholecystitis is unknown, but some investigators have postulated the existence of a biliary – cardiac reflex: traction on the gallbladder may stimulate the vagus nerve and induce ECG changes. The patient’s heart rate on admission was relatively low despite the acute inflammation and fever, which suggests stimulation of the vagus nerve.

Sodium channel blockers, such as pilsicainide, flecainide, and ajmaline, enhance ST-segment elevation, unmask the Brugada syndrome and enable identification of patients at risk of sudden death. Antiarrhythmic agents are recommended as a diagnostic test in patients in whom Brugada syndrome is suspected and pilsicainide reproduced ST-segment elevation in the present patient. Implantation of a loop recorder is proposed for asymptomatic individuals with Brugada-type ST shift, but the patient refused any interventions.

There is a need for correct definition of the risk in asymptomatic subjects with Brugada-type ST shift and progress is being made with this problem. Some markers of risk stratification have been proposed: a late potential, a history of syncope, a family history of sudden death and a prolonged HV interval. The present case did not have any of these and in addition, she was atypical for Brugada syndrome in terms of age and sex. She may have had a relatively benign clinical course, but careful clinical follow up will be necessary.

It was recently reported that Brugada-type ST shift was induced by some psychotropic drugs, although most of the cases were observed during massive overdoses. The mechanism remains unknown, but blockade of the sodium channels, shortening of the action potential duration, and inducement of the intramyocardial electrical gradient have been postulated. The present patient had received relatively low doses of antidepressants and was still taking the medication during the treatment of the acute cholecystitis. It is possible that the antidepressants enhanced the ST-segment elevation induced by the acute cholecystitis and the unmasking of the Brugada-type ST shift by pilsicainide loading. Although QT interval prolongation on ECG was not found, we cannot deny the possibility of antidepressant overdose as a result of impaired liver metabolism caused by the cholecystitis.

The present case is unique in that an asymptomatic Brugada-type ST shift was mimicked by acute cholecystitis. To our knowledge, no previous reports have described this. Although their coexistence may be coincidental and rare, we need to pay attention to inflammatory conditions, other than acute cholecystitis.

**References**
