Gitelman’s Syndrome With Exercise-Induced Ventricular Tachycardia

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A 62-year-old female with palpitations was admitted to hospital where she recorded 12,299 monofocal ventricular premature contractions (VPCs) in 24 h and nonsustained ventricular tachycardia (VT) on exertion. She had hypokalemia with renal potassium wasting, a chloride-resistant metabolic alkalosis, elevated plasma renin, elevated plasma aldosterone (relative to the serum K concentration), hypomagnesemia with renal magnesium wasting, decreased urine calcium excretion, and normal blood pressure. The hypokalemia and hypomagnesemia were thought to have precipitated the VT. The coronary angiogram showed normal coronary arteries; however, the left ventriculogram revealed akinesis of the posterolateral wall. Because the VT could not be induced by programmed electrical stimulation either before or during intravenous administration of isoproterenol, the VPC with the same QRS morphology as the VT became the target of radiofrequency catheter ablation (RF-CA). Intracardiac mapping showed that the earliest activation site was situated in the asynergic area of the left ventricle (LV) and radiofrequency catheter ablation directed at the LV asynergy area completely eliminated the VPCs without any complications. During the follow-up period (6 months), she was free from palpitation and VT was not clinically documented. (Circ J 2004; 68: 509–511)

Key Words: Catheter ablation; Gitelman’s syndrome; Ventricular tachycardia

We present a case of Gitelman’s syndrome with exercise-induced ventricular tachycardia (VT) in which the VT was successfully cured by radiofrequency catheter ablation (RF-CA). Written informed consent for this study was obtained from the patient.

Case Report

A 62-year-old female presented with palpitations at the hospital of Osaka Medical College. She had been free from symptoms until 3 months before admission, when she first noticed palpitations on exertion. Her medical history was unremarkable and she had no history of laxative or diuretic abuse or vomiting, nor was she taking any medications. Her 65-year-old brother had a history of chronic hypokalemia and hypomagnesemia, and her 55-year-old sister had been also diagnosed with hypokalemia and hypomagnesemia, and her 55-year-old sister had been diagnosed with Gitelman’s syndrome with exercise-induced ventricular tachycardia. The hypokalemia and hypomagnesemia were considered to have precipitated the VT in the present case. After correcting the hypokalemia with potassium chloride (2,400 mg/day) and spironolactone (50 mg/day), and the hypomagnesemia with magnesium phosphate (2.75 g/day), the VT did not develop on exertion. However, the VPCs did not disappear and 1 month after admission, cardiac catheterization and an electrophysiological study were carried out. The coronary angiogram showed normal coronary arteries, but the left ventriculogram revealed akinesis of the posterolateral wall of the left ventricle (LV) (Fig 2). Induction of the VT was attempted with programmed pacing (single to triple) and burst pacing from the right ventricular apex and outflow tract, but because it could not be induced either before or during intravenous administration of isoproterenol, VPCs with the same QRS morpholo-
gy as the VT became the target of ablation. The ablation site was the earliest activation site of the VPC, which was the same location where a perfect pace map had been recorded (Fig 3) in the asynergic area of the LV (Fig 4). Radiofrequency delivery directed at the area of LV asynergy completely eliminated the VPCs, even during intravenous administration of isoproterenol. During the follow-up period (6 months), she was free from palpitations and the VT was not documented by ambulatory ECG monitoring or treadmill ECG; only 1 VPC was documented by treadmill ECG after RF-CA.

Discussion

The present patient had several electrolyte abnormalities: hypokalemia with renal potassium wasting, a chloride-resistant metabolic alkalosis, elevated plasma renin concentration, elevated plasma aldosterone (relative to the serum K concentration), hypomagnesemia with renal magnesium wasting, decreased urine calcium excretion, and normal blood pressure. These findings are compatible with either abuse of thiazide-like diuretics or Gitelman’s syndrome, and because of the patient’s family history and the negative diuretic screen, Gitelman’s syndrome was diagnosed.

This syndrome was first described in 1966 by Gitelman et al. and the metabolic abnormalities are similar to those of Bartter’s syndrome: a hypokalemic, chloride-resistant metabolic alkalosis, with elevated plasma renin levels and normal blood pressure. However, Gitelman’s syndrome is distinguished from the classic Bartter’s syndrome by the presence of hypocalciuria and hypomagnesemia. Because the electrolyte disturbances resemble the effects of chronic thiazide administration, Gitelman’s syndrome is postulated to be caused by a defect in the distal thiazide-sensitive sodium-chloride transport.

Hypokalemia and/or hypomagnesemia have been identified as precipitants for arrhythmia, but fatal cardiac arrhythmia is surprisingly infrequent in patients with Gitelman’s syndrome. We could not find any data regarding a relationship between Gitelman’s syndrome, frequency of arrhythmia or response to treatment, and there were no published case reports.

In the present case, intracardiac mapping showed that the VPCs and VT originated from the area identified as LV asynergy, although the etiology is unknown. A recent myocardial infarction because of thrombus and/or spasm or local myocarditis could not be ruled out. After correcting the hypokalemia and hypomagnesemia, VT did not develop on exertion, but the VPCs did not disappear even after correction of the electrolyte abnormalities. After ablating the area associated with asynergy in the LV, VT was not
clinically documented and the number of VPCs markedly decreased. These findings indicate that the LV asynergy was the principal cause of the VPCs and VT, and the hypokalemia and hypomagnesemia related to Gitelman’s syndrome were the precipitants for the arrhythmia.

**References**