Polymorphic Ventricular Tachycardia in a Patient With Adrenal Insufficiency and Hypothyroidism

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We describe a 60-year-old patient with adrenal insufficiency and hypothyroidism who experienced syncope as a result of polymorphic ventricular tachycardia associated with long QT intervals. The deep inverted T waves and QT intervals were normalized about 8 weeks after starting steroid replacement therapy. Although there have been some reports on electrocardiographic abnormality or polymorphic ventricular tachycardia in patients with adrenal insufficiency, the pathogenesis remains unknown. Hormonal disorders should be considered as a cause of polymorphic ventricular tachycardia associated with long QT intervals, even if plasma electrolyte levels are normal, because life-threatening arrhythmia is treatable by supplementation of the hormone that is lacking. (Jpn Circ J 1998; 62: 543–545)

Key Words: Polymorphic ventricular tachycardia; Long QT interval; Adrenal insufficiency; Hypothyroidism

Because transient loss of consciousness developed in association with long episodes of polymorphic ventricular tachycardia in the emergency room, we inserted a temporary pacemaker in the right ventricle, which gave temporary ventricular pacing at a rate of 80beats/min; ventricular tachycardia then disappeared and the blood pressure was 130/80 mmHg. On the 3rd hospital day, spontaneous heart beat developed at a rate of 70beats/min without ventricular tachycardia, and deep inverted T waves and prolonged QT intervals improved to some degree.

Endocrine examination of basal plasma hormone levels was performed and revealed a cortisol level of less than 1.0 µg/100 ml (normal 5–15 µg/100 ml). The basal plasma levels showed of T4 was less than 2.0 µg/100 ml (normal 5.7–13.0 µg/100 ml), of free T4 0.19 ng/100 ml (normal 0.97–1.97 ng/100 ml), and of T3 72 ng/100 ml (normal 80–180 ng/100 ml). The basal plasma level of corticotropin (ACTH) was 20.4 pg/ml (normal 8–29 pg/ml) and of thyrotropin (TSH) 27.9 µU/ml (normal, 0.4–5.0 µU/ml); other anterior pituitary hormones were normal. ACTH increased normally but cortisol did not increase after stimulation with corticotropin-releasing hormone. A thyrotropin-releasing hormone stimulation test revealed a normal prolactin response and an accentuated TSH response. A growth hormone (GH)-releasing hormone test showed a normal GH response. Plasma epinephrine was 37 pg/ml (normal < 100 pg/ml) and noradrenaline was 671 pg/ml (normal < 400 pg/ml), and urinary catecholamine levels were 1 µg/day (normal < 12 µg/day) for epinephrine and 18 µg/day (normal 10–80 µg/day) for noradrenaline. Steroid replacement therapy was started on the 5th hospital day and thyroid replacement therapy on the 11th hospital day. Three weeks after starting steroid replacement therapy, the levels of cortisol and thyroid hormone were normalized. Two-dimensional echocardiography showed normal left ventricular wall motion. A computed tomographic scan and magnetic resonance imaging scan of the brain were normal. Computed tomography revealed of the abdomen no adrenal tumor.

A left ventricular endomyocardial biopsy performed 4 weeks after starting steroid replacement therapy revealed...
no inflammatory cells, and coronary angiography showed that the coronary arteries were normal. Titers of virus-neutralizing antibody were not increased. The deep inverted T waves and long QT intervals were normalized about 8 weeks after starting steroid replacement therapy (Fig 2).

Discussion

We described a case of adrenal insufficiency and hypothyroidism associated with QT prolongation and polymorphic ventricular tachycardia without plasma electrolyte abnormality.

Acute adrenal crisis has been reported to cause cardiovascular complications. A previous report described 2 patients in whom adrenal crisis with reversible left ventricular dysfunction developed in association with deep negative T waves; in one case, a short run of polymorphic ventricular tachycardia occurred. Another report described a case of Sheehan’s syndrome with hypomagnesemia and polymorphic ventricular tachycardia.

Hypopituitarism has been reported to be commonly associated with electrocardiographic abnormalities; ST-segment depression, inverted T waves, and prolonged QT intervals; however, the pathogenesis remains unknown. Ig et al suggested that catecholamine release induced by hypoglycemia might cause arrhythmia or abnormal wall motion of the left ventricle in patients with adrenal insufficiency. In our patient, however, polymorphic ventricular tachycardia occurred in the absence of hypoglycemia, high levels of plasma catecholamine, or plasma electrolyte abnormalities. Some previous reports have suggested that hypomagnesemia induced by adrenal insufficiency might cause myocytic intracellular-extracellular electrolytic imbalance, resulting in shortening of the effective refractory period and prolongation of the relative refractory period. Another report demonstrated that Kv1.5 K⁺ channel gene expression in rat ventricle is up-regulated by glucocorticoids. Furthermore, it is well known that inherited

Fig 1. An electrocardiogram on admission showing short episodes of polymorphic ventricular tachycardia associated with long QT intervals and deep inverted T waves.

Fig 2. Cortisol replacement therapy was started on 16 August and thyroid hormone replacement therapy on 22 August. The deep inverted T waves and long QT intervals were normalized on 16 October associated with normalization of plasma levels of these hormones. ACTH, corticotropin; TSH, thyrotropin.
long QT syndromes are caused by mutation of certain sodium or potassium channel genes.\textsuperscript{8,9} Thus, the deep inverted T waves and prolonged QT intervals seen in our patient might be mediated by a hormonal modulation of ion channels of cardiac cells, which could contribute to polymorphic ventricular tachycardia before plasma electrolyte abnormality. An electrocardiogram recorded 2 years before admission already revealed non-specific ST changes and prolonged QT intervals (Fig 2). The electrocardiogram on admission showed deep inverted T waves with markedly prolonged QT intervals, which improved to some extent before starting cortisol supplement therapy. This suggests that subclinical adrenal insufficiency developed into an acute adrenal crisis as a result of severe stress and was ameliorated to some extent by a period of rest. Myocarditis was unlikely because left ventricular endomyocardial biopsy revealed no inflammatory cells.

In conclusion, hormonal disorders must be examined as a cause of polymorphic ventricular tachycardia associated with long QT intervals even if plasma electrolyte levels are normal, because supplementation of insufficient hormone may permanently cure life-threatening arrhythmia.

References