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Gitelman's Not-So-Benign Syndrome

TO THE EDITOR: Gitelman's syndrome, or congenital hypokalemic hypomagnesemic hypocalciuria with metabolic alkalosis, is widely described as a benign¹ or milder² variant of Bartter's syndrome. Despite symptoms including presyncope, vertigo, ataxia, and blurred vision, few data have been accumulated regarding formal cardiac evaluation for patients with Gitelman's syndrome.³

We report a case of Gitelman's syndrome in a patient presenting with presyncope coincident with long runs of ventricular tachycardia at 230 beats per minute that was decidedly malignant. An otherwise well 39-year-old woman taking no medications (not even thiazides) presented with recurrent presyncope. An echocardiogram and the results of a stress test were normal. A computed tomographic (CT) scan of the abdomen showed no mass lesion, and CT angiography of the chest showed normal coronary-artery anatomy with no stenoses. Holter monitoring revealed recurrent 2-to-8-second runs of monomorphic ventricular tachycardia with a cycle length of 260 msec coincident with presyncope.

On admission, the patient's serum electrolyte levels were as follows: potassium, 2.9 mmol per liter; magnesium, 0.9 mg per deciliter; sodium, 144 mmol per liter; chloride, 102 mmol per liter; and carbon dioxide, 34 mmol per liter. Urine electrolyte levels were as follows: sodium, 80 mmol per liter; chlorine, 128 mmol per liter; potassium, 83 mmol per liter; and magnesium, 10 mg per deciliter. Hypokalemic hypomagnesemic hypercalciuria persisted for several days in the hospital. An electrocardiogram showed sinus rhythm with a PR interval of 148 msec, a QRS interval of 94 msec, a QT interval of 424 msec, and a prolonged corrected QT interval (QTc) of 470 msec. Blood urea nitrogen and serum creatinine levels were normal. Plasma renin and aldosterone levels were 63 U per milliliter and 24.3 ng per deciliter, respectively, while the patient was in

a recumbent position. An electrophysiological study was negative for inducible, sustained ventricular tachycardia; however, the patient continued to have ventricular tachycardia with presyncope despite aggressive potassium and magnesium supplementation and treatment with amiloride (an aldosterone antagonist), captopril (an angiotensin-converting-enzyme inhibitor), and lidocaine.

Amiodarone was initiated, in place of lidocaine, to control symptomatic runs of rapid, nonsustained ventricular tachycardia, and a defibrillator was implanted to guard against a potential breakthrough of sustained ventricular arrhythmia. The arrhythmia subsided and the patient was discharged to her home in good condition.

Gitelman's syndrome is an autosomal recessive disorder with an inactivating mutation of the thiazide-sensitive sodium-chloride cotransporter in the distal renal tubule. Scant literature exists regarding cardiac evaluation^{3,4} and suggests that about half the patients with Gitelman's syndrome have QTc prolongation. Although Gitelman's syndrome is described as an asymptomatic or benign disorder with characteristic electrolyte abnormalities, most reports of clinical series document presyncope, vertigo, ataxia, and blurred vision. The present case suggests that more diligent investigation into ventricular arrhythmia, particularly in patients with prolonged QTc, may be warranted.

Roman T. Pachulski, M.D.

South Texas Heartbeat Associates
San Antonio, TX 78229
rtpach@mac.com

Fernando Lopez, M.D.

Alamo Heart Associates
San Antonio, TX 78229

Rashid Sharaf, M.D.

South Texas Nephrology
San Antonio, TX 78229

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Localization of a Corticotropin-Secreting Tumor by Thoracoscopic Pulmonary Venous Sampling

TO THE EDITOR: Corticotropin-secreting pulmonary tumors are an uncommon cause of Cushing's syndrome.¹ Precise localization of ectopic (nonpituitary) sources of corticotropin can be difficult: radiologic studies are often nonspecific, and surgical exploration by standard thoracotomy is associated with morbidity. Video-assisted thoracic surgery (VATS) is gaining popularity for anatomical and nonanatomical pulmonary resections. We describe a patient with Cushing's syndrome due to ectopic secretion from a single pulmonary carcinoid; percutaneous sampling of blood from the pulmonary veins by VATS to determine corticotropin levels permitted precise localization of the tumor and confirmed the completeness of the resection.

A 25-year-old man was examined because of weight gain, hypertension, hirsutism, and purple striae of the abdomen and thighs. The initial results of biochemical evaluation included an elevated 24-hour urinary cortisol level (237 μg ; normal range, <105) and an elevated corticotropin level (53.6 ng per liter; normal range, 6 to 48). The high cortisol levels could not be suppressed by dexamethasone administration. Extensive workup for a pituitary adenoma was negative.

Computed tomographic scanning of the chest revealed two lesions larger than 1.0 cm — one in the superior segment of the right lower lobe and one in the lingula. Both lesions had moderately increased uptake on octreotide scintigraphy.

The right inferior and left superior pulmonary veins were punctured to allow measurement of corticotropin levels by the VATS technique. Sampling from the pulmonary veins was done before and after segmentectomy of the pulmonary nodule on the right side and wedge resection of the lingular nodule. The right side was approached first, followed

by the left. The lesion in the right lower lobe was later determined to be a carcinoid tumor and that in the lingula a cryptococcal lesion.

The corticotropin level had been markedly elevated in the right lower pulmonary vein but decreased by 85 percent after the right superior segmentectomy (Fig. 1). Wedge resection of the nodule in the lingula did not result in any change in the corticotropin level. The patient had a biochemical cure, with corticotropin and cortisol levels of 9 ng per liter and 2 μg per deciliter by postoperative day 1. The cutaneous, hemodynamic, and psychiatric symptoms related to the hypercortisolism resolved after surgery and have not recurred during six months of follow-up.

Pulmonary ectopic sources of corticotropin are well recognized and include small-cell cancer of the lung and bronchial adenoma; however, nonspe-

