Hypertrophic cardiomyopathy

MAY 2018

TO THE EDITOR: We read with interest the article by Young et al on hypertrophic cardiomyopathy (HCM)[1] and would like to raise a few important points.

HCM has a complex phenotypic expression and doesn't necessarily involve left ventricular outflow obstruction. Midventricular obstruction is a unique subtype of HCM, with increased risk of left ventricular apical aneurysm (LVAA) formation. We reported that 25% of HCM patients with midventricular obstruction progress to LVAA compared with 0.3% of patients with other HCM subtypes.[2] Magnetic resonance imaging plays a pivotal role in assessing midventricular obstruction, owing to asymmetric geometry of the left ventricle and the shortcomings of echocardiography in assessing the apical aneurysm.[2]

Anticoagulation remains one of the cornerstones in treating midventricular obstruction with LVAA. We performed a systematic review and found a high prevalence of atrial arrhythmia, apical thrombus, and stroke, which necessitated anticoagulation in one-fifth of patients.[2]

Ventricular arrhythmias are prevalent in midventricular obstruction with LVAA, mainly from increased fibrosis formation at the apical rim.[3] In our review, 25.7% of patients with midventricular obstruction with LVAA and an implantable cardioverter-defibrillator (ICD) experienced appropriate shocks.[2] Our finding was in line with those of Rowin et al,[3] who showed appropriate ICD shocks in one-third of HCM patients with apical aneurysm. Apical aneurysm is currently considered an independent risk factor for sudden cardiac death in HCM, with an increased rate of sudden death of up to 5% every year.[3,4]

It is imperative to distinguish midventricular obstruction with LVAA as a unique disease imposing a higher risk of thromboembolism, ventricular arrhythmia, and progression to end-stage heart failure.[3] We suggest that those patients be evaluated early in the course of disease for anticoagulation, ICD implantation, and early surgical intervention.[2]

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Postsurgical hypoparathyroidism is not primary hypoparathyroidism

MARCH 2018

TO THE EDITOR: I read with interest the case of a 67-year-old woman with bilateral hand numbness, published in the March 2018 issue of the *Journal*, and I would like to suggest 2 important corrections to this article.[1]

The authors present a case of hypocalcemia secondary to postsurgical hypoparathyroidism but describe it as due to primary hypoparathyroidism. The patient had undergone thyroidectomy 10 years earlier and since then had hypocalcemia, secondary to postsurgical hypoparathyroidism, that was

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treated with calcium and vitamin D, until she stopped taking these agents. Postsurgical hypothyroidism is the most common cause of acquired or secondary hypoparathyroidism and is *not* primary hypoparathyroidism. I strongly feel that this requires an update or correction to the article. This patient may have associated malabsorption, as the authors alluded to, as the cause of her "normal" serum parathyroid hormone level.

The patient also had hypomagnesemia, which the authors state could have been due to furosemide use and "uncontrolled" diabetes mellitus. Diabetes doesn't need to be uncontrolled to cause hypomagnesemia. Hypomagnesemia is common in patients with type 2 diabetes mellitus, with a prevalence of 14% to 48% in patients with diabetes compared with 2.5% to 15% in the general population.[2] It is often multifactorial and may be secondary to one or more of the following factors: poor dietary intake, autonomic dysfunction, altered insulin resistance, glomerular hyperfiltration, osmotic diuresis (uncontrolled diabetes), recurrent metabolic acidosis, hypophosphatemia, hypokalemia, and therapy with drugs such as metformin and sulfonylureas.

Patients with type 2 diabetes and hypomagnesemia often enter a vicious cycle in which hypomagnesemia worsens insulin resistance and insulin resistance, by reducing the activity of renal magnesium channel transient receptor potential melastatin (TRPM) type 6, perpetuates hypomagnesemia.[3]

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IN REPLY: We thank Dr. Parmar and appreciate his important comments.

Regarding the difference between primary and secondary hypoparathyroidism, the definition varies among investigators. Some define primary hypoparathyroidism as a condition characterized by primary absence or deficiency of parathyroid hormone (PTH), which results in hypocalcemia and which can be congenital or acquired, including postsurgical hypoparathyroidism.[1–4] In principle, this is similar to the classification of disorders affecting other endocrine glands as primary and secondary. For example, primary hypothyroidism refers to a state of low thyroid hormones resulting from impairment or loss of function of the thyroid gland itself, such as in Hashimoto thyroiditis, radioactive iodine therapy, or thyroidectomy, among others.[5] We adopted this definition in our article. In contrast, secondary hypoparathyroidism is characterized by low PTH secretion in response to certain conditions that cause hypercalcemia. *Non-PTH-mediated hypercalcemia* is a more common term used to describe this state of secondary hypoparathyroidism.

Other investigators restrict the term "primary hypoparathyroidism" to nonacquired (congenital or hereditary) etiologies, while applying the term "secondary hypoparathyroidism" to acquired etiologies.[6]

Concerning the association between diabetes mellitus and hypomagnesemia, we agree that diabetes does not need to be uncontrolled to cause hypomagnesemia. However, the patient described in our article presented with severe hypomagnesemia (serum level 0.6 mg/dL), which is not commonly associated with diabetes. Most cases of hypomagnesemia in patients with type 2 diabetes mellitus are mild and asymptomatic, whereas severe manifestations including seizures, cardiac arrhythmias, and acute tetany are rarely encountered in clinical practice.[7] Furthermore, numerous studies have shown a negative correlation between serum magnesium level and glycemic control.[7–11] A recent study reported that plasma triglyceride and glucose levels are the main determinants of the plasma magnesium concentration in patients with type 2 diabetes.[12]

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Our patient's diabetes was uncontrolled, as evidenced by her hemoglobin A_{1c} level of 9.7% and her random serum glucose level of 224 mg/dL. Therefore, it is more likely that "uncontrolled diabetes mellitus" (in addition to diuretic use) was the cause of her symptomatic severe hypomagnesemia rather than controlled diabetes mellitus.

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