IN REPLY TO "THYROTOXIC PERIODIC PARALYSIS, β_2-ADRENERGIC BRONCHODILATOR, AND INSULIN—AN INTERESTING INTERPLAY"

To the editor: We fully appreciate the comments on this case of β_2-adrenergic bronchodilator–induced thyrotoxic periodic paralysis (TPP).[1,2] We agree that hyperinsulinemia plays an important role in the pathogenesis of acute hypokalemia in TPP patients. β_2-Agonist is known to stimulate insulin secretion and may accordingly predispose TPP patients to acute hypokalemia.[3] On the other hand, it should be noted that nearly 80% of TPP attacks cannot be triggered by iatrogenic hyperinsulinemia after carbohydrate load.[4] The pathogenesis of TPP is clearly multifactorial and includes environmental stimulations (e.g., vigorous exercise, carbohydrate meal, etc.), endogenous hormones (e.g., catecholamines, insulin, and thyroid hormone), and recently unveiled genetic variations in *KCNJ2* and *KCNJ18*.[5,6] These genetic variations were supposed to reduce potassium efflux via muscular potassium channels and lead to potassium accumulation in intracellular space.

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