Do we need sick-day guidelines for hypoparathyroidism?

Sir,

In general, as well as in endocrine clinical practice, ‘sick-day’ guidelines form an integral part of the overall management of various diseases. Sick-day guidelines are designed to prevent exacerbation of an underlying chronic disorder during periods of stress so that appropriate adjustments in the drug doses are made. This will ensure that the patient is better equipped to counter any potential adverse effects of the stress and strain of daily living and reduce morbidity and even mortality. The success of ‘sick-day’ guidelines depends upon the dexterity of the clinician and compliance of the patient, and often reinforcement is required to achieve optimal benefit. Several guidelines already exist for various endocrine disorders such as diabetes mellitus, panhypopituitarism, and adrenal insufficiency. However, no such strategy is mentioned in the literature with regard to ‘sick-day’ guidelines for patients with hypoparathyroidism. The following cases will illustrate the same.

A 30-year-old female, with an established diagnosis of idiopathic hypoparathyroidism presented with perioral...
paresthesias, spontaneous carpopedal spasm, and impending laryngeal spasm, following two days of fever, associated with upper respiratory tract infection. She was on optimal therapy with calcitriol 0.25 µg twice / day and oral calcium 500 mg four times / day, with good compliance, as reflected in the stable serum calcium levels over time. Following the fever episode, she missed both the above-mentioned medicines for a period of two days. On examination, she had spontaneous carpopedal spasm and laryngeal stridor. She had albumin–adjusted serum Ca of 5.3 mg / dl (reference range 9 – 11 mg / dl), phosphate 6 mg / dl (reference range 3 – 5 mg / dl), serum creatinine (0.5 – 1.5 mg / dl), and alkaline phosphatase 12 KAU (reference range 3 – 13 KAU). A chest X-ray was normal, but a 12-lead electrocardiogram showed a prolonged QTc interval of 0.59 seconds (Reference value ≤ 0.42).

She was treated with intravenous calcium gluconate infusion and appropriate antibiotics. She improved quickly and the calcitriol dose was increased to 0.5 µg twice / day and oral calcium 500 mg every four hours, before discontinuing calcium infusion.

An 18-year-old boy was diagnosed to have hypoparathyroidism when he was investigated for carpal spasm. He was rendered asymptomatic with standard doses of calcium (500 mg elemental calcium qid) and active vitamin D (0.25 µg bid). He had gastroenteritis (diarrhea and vomiting) and missed three doses of scheduled calcium and calcitriol. During hospitalization he developed generalized tonic–clonic seizures, with carpal spasm. His albumin–adjusted serum Ca was 4.9 mg / dl (reference range 9 – 11 mg / dl), phosphate 7 mg / dl (reference range 3 – 5 mg / dl), and alkaline phosphatase was 15 KAU (reference range 3 – 13 KAU). The remaining routine biochemistry was normal, including serum creatinine, magnesium, and potassium.

He received intravenous fluids, appropriate antibiotics for gastroenteritis and phenytoin (1 mg / kg intravenously) loading for seizures. His gastroenteritis settled, but the seizures were not controlled, in addition he developed phenytoin induced cerebellar signs. The phenytoin was stopped in view of the cerebellar signs and the patient was treated with intravenous calcium gluconate infusion, with oral calcitriol, considering the possibility of a hypocalcemia-induced seizure. He became seizure-free and in the next few days the cerebellar signs also improved. He was shifted back to the previous regimen of oral calcium and oral calcitriol.

These cases illustrate that patients with hypoparathyroidism on stable maintenance doses of calcium and vitamin D therapies can still develop significant and serious hypocalcemia, even with seemingly minor infections or concurrent illnesses. The most plausible explanations include: (1) decreased intestinal absorption of calcium due to inter-current illnesses associated with vomiting and diarrhea, (2) initiation or dose escalation of antiepileptic medications, particularly phenytoin without the necessary adjustments in oral calcium and / or vitamin D doses. Several antiepileptic drugs might worsen hypocalcemia by accelerating catabolism of 25-hydroxyvitamin D to its inactive metabolites, as well as decreasing intestinal absorption of calcium from the gut. (3) Hyperventilation during upper respiratory tract disease or during an anxiety and stressful situation, resulting in carbon dioxide washout and alkalosis, both of which reduce ionized serum Ca.

Based on our experience we propose the following guidelines for patients with hypoparathyroidism on stable doses of oral calcium and active vitamin D (calcitriol).

During periods of stressful situations they must not stop taking their usual doses of calcium and vitamin D. In fact, they may have to double the daily doses for a few days. Their doctor must always be notified immediately about this change, as continued use of high-dose vitamin D metabolites may cause serious problems, including kidney failure.

If the patients are unable to take any oral medications they will require administration of calcium through a vein to avoid tetany and other serious problems associated with a sudden drop in their blood calcium. Therefore, they will have to call their doctor immediately or go to the nearest Emergency Room for appropriate care and advice.

Choice of fluids must be dextrose rather than 0.9% saline, as sodium increases calcium excretion. Use of the antiepileptic-like phenytoin may require adjustments in their calcium and vitamin D doses. Phenytoin must be avoided in patients with hypoparathyroidism, in view of its epileptogenic nature. The patient’s doctor has to be consulted immediately if this happens, although many physicians are aware of this problem. Nevertheless, it is a good idea for the patient to mention his / her hypoparathyroid condition to the treating physician.

As the patients’ condition is frequently associated with high blood phosphate levels, they must avoid foods and drinks rich in phosphate; these include milk (despite being a good source of Ca), other dairy products, and canned foods.

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