

Metoclopramide-Induced Hyperglycemia and Leukocytosis in a Patient with Type 2 Diabetes and Septic Arthritis: Letter to the Editor

Tip 2 Diyabet ve Septik Artriti Olan Bir Hastada Metoklopramide Bağlı Hiperglisemi ve Lökositoz

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Metoclopramide, which has dopamine 2 receptor antagonistic properties, is widely used in patients with emesis, especially among those hospitalized.¹ Sedation is the most common side effect and other adverse effects such as akathisia and dystonic reactions may develop.

Neuroleptic malignant syndrome (NMS) is a rare adverse effect of neuroleptic agents and dopamine receptor antagonists such as metoclopramide.² Elevated white blood cell (WBC) count is among the characteristics of the syndrome and rarely may associate with deterioration of glycemia.^{2,3}

A 33-year-old male patient with type 2 diabetes presented with swelling and pain of the left knee. He had an associated hyperglycemic hyperosmolar syndrome (HHS). Physical examination revealed dry oral mucosa, swelling and hyperemia on the left knee. He had leukocytosis on complete blood count. Sedimentation rate and C-reactive protein (CRP) levels were high. Ultrasonography of the left knee revealed that, the articular space was widened with a minimal fluid collection. No material was aspirated from the articular space. Standard therapy for HHS and antibiotherapy were started. Persistent elevation of CRP and WBC count necessitated surgical management of the left knee. On the postoperative follow-up, inflammatory markers and glycemic regulation improved and parenteral metoclopramide 3 x 10 mg/day was initiated for emesis. WBC count dramatically increased without any sign of infection on the following days, but creatinine phosphokinase (CPK) remained normal. The patient had weakness and glycemic dysregulation that had started after metoclopramide therapy. He became immobilized on the following days but did not show any other symptoms of NMS.

Retrospective review of the medications indicated that the patient's ill-state, glycemic dysregulation and leukocytosis had co-occurred with the initiation of metoclopramide therapy and it was stopped on the 4th day. The patient's complaints, leukocytosis and glycemic regulation improved after cessation of metoclopramide (Table 1).

TABLE 1: The course of leukocyte count and fasting glucose before and during metoclopramide therapy and after the cessation of the metoclopramide therapy.

	Before metoclopramide therapy		After metoclopramide therapy		After cessation of metoclopramide therapy	
WBC count (cells/mm ³)	3.42	4.8	26.0	30.0	12.6	11.4
FPG (mg/dL)	170	145	340	380	145	130

Leukocytosis, glycemic dysregulation and generalized weakness are the characteristics of NMS, one of the unexpected and rare complications of metoclopramide therapy.² Three-day course of metoclopramide therapy led to generalized weakness, progressive immobilization, glycemic deterioration and leukocytosis in our patient. Although these symptoms may be related to NMS induced by metoclopramide, laboratory findings other than leukocytosis and hyperglycemia did not suggest a full NMS. Lack of elevation in CPK may be related to the early recognition of metoclopramide-indu-

ced NMS and early cessation of the therapy. Hyperglycemia in this patient may be due to the induction of acute phase reaction, which was reported in NMS previously.⁴ Such adverse effects may be attributed to the patient's primary disease, but important clinical and laboratory clues may help the clinicians to diagnose this rare condition.

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