Sir,

Given the volume of use of analgesic preparations with approved indications for acute pain, a number of drug interactions including less well-known and potentially clinically significant drug-drug interactions are increasingly being reported.[1] A 32-year-old male on treatment for cysticercosis was receiving Tab. albendazole, phenytoin, steroids and ranitidine. Meanwhile, the patient developed allergic rhinitis, fever, body and joint pain. His general and systemic examination was normal. He was started on cetrizine for allergy and tramadol for body and joint pain. There was no concomitant use of antidepressants. The patient continued to receive Tab. albendazole and oral steroid without any change in dosage. After 6 hours he developed violent behavior and delirium. There were no myoclonic twitches or muscular rigidity. On examination he had tachycardia and excessive sweating. Pupils were bilateral 3 mm in size and reacting to light. Bowel and bladder functions were normal. Blood investigations showed raised erythrocyte sedimentation rate, blood urea and serum creatinine were normal. Facilities for estimation were not available. The abnormal behavior could be controlled with haloperidol and both the drugs (tramadol and cetrizine) were discontinued. The Naranjo score[2] for adverse drug reaction was 9. There was no history of any significant illness prior to cysticercosis and he was not an alcoholic. He responded well and recovered completely. Although tramadol may represent a well-established, safe therapy for pain,[3-5] abuse and dependence on tramadol as well as tramadol-related deaths have been increasingly reported, either when the drug is ingested alone or taken in combination with other potentially interactive drugs.[5-7] Mild and transient central nervous system stimulation during therapy with tramadol has been reported in 7% of patients in clinical trials.[7] The debilitating reaction following a single oral dose of tramadol may include ataxia, dilation of the pupils, numbness, tremulousness, and dysphoria lasting for hours and disappearing after discontinuation of the therapy.[7] Rarely may it lead to fluctuating confusion and cognitive deficits (reversible after discontinuation of the drug).[8] The exact mechanism of the adverse response is not known; however, based on phenotyping results, it has been suspected that it may be related to high concentrations of the active O-desmethyl metabolite of tramadol.[7]
HYPOTHYROID MYOPATHY OR RHABDOMYOLYSIS

Sir,

The article “Hypothyroidism-associated rhabdomyolysis” was interesting.[1] The clinical description of the patient fits in with the diagnosis of hypothyroid myopathy. The serum creatinine value is practically normal (1.6 mg%), and the fact that the patient has a normal blood urea and a normal urine output indicates that the renal function is virtually normal. Rhabdomyolysis is a more severe illness which presents acutely with features of myalgias, hyperkalemia, renal dysfunction, metabolic acidosis and features of disseminated intravascular coagulation, apart from elevated creatinine kinase levels. Also, the serum creatinine kinase levels are about 5 times the normal, which is possible even in hypothyroid myopathy. Even levels up to 9000 have been reported.[2] All in all, the case appears to be more of a hypothyroid myopathy rather than a rhabdomyolysis.

The authors also mention that the patient has borderline first-degree heart block on EKG. In the setting of rhabdomyolysis, this should alert the clinician about the possibility of hyperkalemia secondary to muscle damage. The authors fail to mention the serum K+ levels and also whether the abnormality improved with treatment.

VISHAL SHARMA, ALKA SHARMA, SOURABH AGGARWAL
Department of Medicine, University College of Medical Sciences and GTB Hospital, Delhi, India

Correspondence:
Vishal Sharma, 19 Gobind Nagar Chheharta, Amritsar - 143 105, India.
E-mail: docvishalsharma@gmail.com

REFERENCES


DOI: 10.4103/0019-5359.49246