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Letters to the editor

Rhabdomyolysis associated with omeprazole

To the Editor: There have been a few reports about muscle toxicity induced by omeprazole. We would like to report a case of rhabdomyolysis associated with the intravenous administration of omeprazole.

A 56-year-old Japanese man visited Tokyo Metropolitan Police Hospital with an episode of nausea and dizziness that had begun the previous evening. He had been diagnosed as suffering from arrhythmia at the age of 51, and was a heavy drinker. Physical examination was unremarkable. Clinical investigations revealed anemia (hemoglobin [Hb], 10.8 g/dl); leukocytosis (WBC, 11 900/μl); and hyperlipidemia, (triglyceride [TG], 169 mg/dl). Renal and liver functions were within normal limits.

After being admitted, he presented with massive hematemesis due to Mallory-Weiss syndrome. Esophageal and gastric hemorrhage was treated with endoscopic clipping and intravenous omeprazole, 20 mg twice a day.

The patient generally improved, but his serum creatine phosphokinase level gradually became elevated. On his fifth day in hospital, it rose to 3856 IU/l (normal range, 43–272 IU/l), while his serum myoglobin level was 467 ng/ml (normal range, ≦65 ng/ml). Physical and neurological examination results were unremarkable. Creatine kinase isoenzyme showed a 0.5% MB fraction, and serum myosin light chain I and cardiac troponin T levels were within normal limits. Electrocardiogram results and thyroid function were normal. After withdrawal of omeprazole, the laboratory data improved within 5 days. The patient was discharged on the fifteenth hospital day, and a follow-up examination showed that he was doing well 6 months later.

Although our patient's physical findings were negative for rhabdomyolysis, these laboratory findings usually indicate rhabdomyolysis due to destruction of skeletal muscle.[1] There are a few scattered reports of muscle toxicity with marked elevation of serum creatine phosphokinase[2,3] induced by omeprazole taken orally. The exact mechanism is not known, but we should be aware of possible side effects associated with the intravenous administration of omeprazole.

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Massive gastrointestinal bleeding in a patient with polyarteritis nodosa

To the Editor: Polyarteritis nodosa (PAN) is a type of systemic necrotizing vasculitis that involves the small and medium-sized vessels. As no test or clinical finding reliably indicates the presence or absence of PAN, diagnosis requires the integration of clinical, angiography, and biopsy findings. Gastrointestinal bleeding occurs less frequently, with approximately 6% of patients with PAN developing bleeding.[1] We report a case of massive gastrointestinal bleeding in a patient with PAN.

A 66-year-old man was admitted to our hospital with anemia. The laboratory tests showed severe anemia (RBC count, 2.5×10^6/μl; hemoglobin [Hb], 6.5 g/dl), and an elevated erythrocyte sedimentation rate (ESR) (124 mm/h) and C-reactive protein (CRP) level (12.97 mg/dl). Gastroduodenoscopy revealed an active open ulcer in the anterior wall of the duodenal bulb, without active bleeding. This ulcer healed in a month following omeprazole therapy. He soon became febrile (37°C–39°C), with polyarthralgia, finger swelling, skin eruption, and a painful sensation in the lower extremities. He started taking prednisolone (20 mg daily) on the 26th hospital day following the provisional diagnosis of collagen disease. His condition remained stable until the 69th hospital day, when he passed a considerable amount of tarry stools. On the 72nd hospital day, massive rectal bleeding developed into hemorrhagic shock. Emergency abdominal arteriography revealed extravasated contrast medium and several microaneurysms in the first jejunal branch of the superior mesenteric artery (Fig. 1). A partial jejunectomy was subsequently performed on the same day. Pathological examination revealed two pinpoint erosions of the jejunum (Fig. 2), but no obvious source of bleeding could be identified. As shown in Fig. 3a, histological examination revealed that the small and medium-sized arteries in the submucosa and muscular layers exhibited fibrinoid necrosis of the vessel wall with neutrophilic infiltration (necrotizing arteritis). An organizing thrombus, which had obliterated the lumen, was