Rhabdomyolysis Caused by Distigmine Bromide

Key words: distigmine bromide, rhabdomyolysis

To the Editor: We report a 70-year-old woman suffering from urinary retention that was treated with distigmine bromide, and our investigations indicated that this treatment had caused rhabdomyolysis.

The patient took 10 mg of distigmine bromide from March 2001. On October 11, 2002, she was admitted for reduced urine volume, edema and ascitis. She was fully conscious and experiencing some thoracic and pelvic limb muscle weakness, although no muscle pain was present. Serum creatinine level was 0.7 mg/dl (normal range was from 0.3 to 0.8 mg/dl), potassium level was 2.1 mmol/l (normal range is from 3.6 to 4.8 mmol/l), lactate dehydrogenase (LDH) level was 600 IU/l (normal range is from 119 to 229 IU/l), and creatine kinase (CK) level was 4,069 IU/l (normal range is from 45 to 163 IU/l). At first, we suspected that the patient’s rhabdomyolysis had been caused by colestyramine and pravastatin sodium. Consequently, we discontinued these drugs at once; however, the LDH and CK level were still elevated. The maximum levels of LDH and CK were elevated to 655 IU/l, and 5,144 IU/l, and myoglobin and aldolase were elevated to 2,007 ng/ml (normal range is below 60 ng/ml) and 56.2 IU/l (normal range is from 1.7 to 5.7 IU/l), respectively, on October 13. We discontinued distigmine bromide on that day. On October 16, the CK level was lowered to 3,411 IU/l. The patient’s edema and ascitis disappeared on October 22, and the CK level returned to the normal range on October 24.

Distigmine bromide has been widely used for many years in the treatment of voiding difficulties experienced by patients after spinal injuries, prostatectomy, abdominal surgery and gynecological procedures (1-4). The major adverse effect of this drug has been reported to be cholinergic crisis (5). In the present patient, cholinesterase was presented at a low level of 54 IU/l (normal range is from 185 to 431 IU/l) on admission, and the typical symptoms of cholinergic crisis did not occur. It might be assumed that the rhabdomyolysis had developed under simultaneous intake of colestyramine and pravastatin sodium. However, the patient’s CK level did not decrease after discontinuing these drugs but did decrease after discontinuing distigmine bromide. This suggests that the rhabdomyolysis resulted from distigmine bromide. This adverse effect on her muscles might also have been accelerated by co-administrating colestyramine and pravastatin.

When administering distigmine bromide, it is important to carefully check the patient’s age and history of taking drugs for anti-hyperlipidemia, especially statins or fibrates. The present case also suggests that rhabdomyolysis is an important complication stemming from treatment with distigmine bromide.

Yutaka Tsutsumi, Junji Tanaka*, Takuya Miura,
Hiroaki YAMATO, Hiroe KANAMORI,
Takahito KAWAMURA, Shinji OBARA,
Masahiro ASAKA**, Masahiro IMAMURA* and Nobuo MASUZU

The Department of Internal Medicine, Hakodate Municipal Hospital, Hakodate, *the Departments of Hematology and Oncology and **Gastroenterology, Hokkaido University Graduate School of Medicine, Sapporo

Received for publication May 26, 2003; Accepted for publication August 7, 2003

Reprint requests should be addressed to Dr. Yutaka Tsutsumi, the Department of Internal Medicine, Hakodate Municipal Hospital, 1-10-1 Minato-cho, Hakodate 041-0821

References