Bradycardia due to Methylprednisolone Therapy

To the Editor:

We describe 2 patients with asymptomatic marked sinus bradycardia due to methylprednisolone (MP) therapy, which resolved spontaneously. First one 25-year-old male had neuro-Behçet’s disease. MP pulse therapy was initiated; three 1000-mg doses were administered over 1 hour every consecutive day. On the third day of treatment, sinus bradycardia occurred. The patient had no clinical symptoms, and there were no changes in electrolyte levels. Because the patient’s clinical condition was ameliorated, treatment was continued as 1 mg/kg/day after the fourth day. By the eighth day, the patient’s pulse rate returned to pretreatment values and ECG revealed normal sinus rhythm. The second case was a 24-year-old woman. She was diagnosed with lupus nephritis. Sixty milligrams daily MP was initiated in 2 divided intravenous doses. On the fourth day of treatment, asymptomatic sinus bradycardia developed. In Holter monitoring, the lowest heart rate was 34 beats/min. There were no changes electrolyte levels. Since treatment resulted in a gradual improvement according to her clinical symptoms and laboratory parameters, we continued MP treatment. On the 11th day of treatment, the patient’s pulse rate returned to normal.

Cardiovascular complications have been reported due to corticosteroids. Mechanisms by which high-dose corticosteroids may produce important cardiovascular effects have been proposed. Some of authors suggest that MP may have a direct effect on the integrity of cardiac muscles, causing alterations in calcium and potassium flux across cell membranes. Although it had been postulated that large amounts of phosphate present in dexamethasone and MP preparations may be responsible for chelation of serum calcium,1 there has been no evidence of acute electrolyte abnormalities in our patients.

The delayed onset of bradycardia cannot be explained by the pharmacokinetics of MP. We administered MP pulse treatment to our first patient over 60 minutes and MP treatment to the second patient as 60 mg/day in 2 divided doses. Despite a slow rate of infusion and use of a low dose, the MP therapy may not be completely safe. Significant sinus bradycardia due to MP minipulse (125 mg daily) and oral MP treatment was also reported.2,3

Although we continued MP treatment after the development of bradycardia, the bradycardia did not deteriorate. The general health status of our patients improved according to laboratory tests. We believe that bradycardia is not an indication to stop potentially beneficial treatment. Tvede et al4 reported that sinus bradycardia in 4 patients received high-dose MP therapy was most pronounced on the fourth day. By the seventh day, pulse rates had normalized to pretreatment values in all patients.

Our experience suggests that cardiac monitoring of patients who are treated with glucocorticoids is very useful not only during high doses but also during standard doses. We consider that if a patient’s hemodynamics are not deteriorating, treatment may be continued.

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REFERENCES