Cefotaxime-Induced Encephalopathy in an Uremic Patient

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Dear Sir,

In a recent paper, Geyer et al. [1] reviewed their experience concerning cephalosporin-induced encephalopathy in uremic patients who had received either cefazolin or ceftazidime at high doses. We agree with this authors advising reduction of dosage for cefazolin and ceftazidime in uremic patients, as we have witnessed several cases of neurotoxicity related to ceftazidime. They assume that cefotaxime seems to be well tolerated at high doses by these patients because of its metabolism. We would like to present our observations regarding similar neurotoxic effects of cefotaxime in a patient with renal failure, an event probably not reported to date.

A 68-year-old male patient with moderate renal insufficiency of arteriosclerotic origin underwent surgical replacement of aneurismatic abdominal aorta. 24 h later, severe renal failure developed. Examination revealed a well-oriented individual with normal neurological findings.

Hemodialysis (HD) was performed on a daily basis, and nitrogen waste products as well as serum electrolytes were under control. Several days later low-grade fever of a probable abdominal source was noted. Intravenous cefotaxime 2 g t.i.d. and 80 mg tobramycin at the end of each HD were prescribed. An abdominal abscess was drained and HD stopped for 3 days, during which cefotaxime continued being administered. The patient developed lethargy, disorientation, asterixis, multifocal myoclonus and uncontrolled hand tremor. At that time he was afebrile and blood urea nitrogen was 76 mg/dl. Cefotaxime was withdrawn, having raised the suspicion of toxicity. HD was performed during 4 h. The patient's clinical status improved and asterixis and multifocal myoclonus disappeared within 24 h. Cefotaxime plasma level was 64 µg/ml (the sample was taken 36 h after the last injection). Thereafter, cefotaxime was reintroduced at a dose of 1 g every 24 h without any toxicity.

Toxic encephalopathy secondary to high-dose cephalosporins is not an uncommon event and we have to expect it when used in patients with severe renal failure [1, 2]. Though several authors have not advised reduction in dosage of cefotaxime in uremic patients [3], an accumulation of this cephalosporin may induce neurotoxicity. Hence, in case of severe renal failure it is necessary to decrease the usual dosage. According to pharmacokinetic studies [4] we suggest that an intravenous dose of 1 g cefotaxime every 24 h would yield enough concentrations to inhibit most aerobic bacteria species without toxicity. Probably, an individual variability in elimination rates of the drug exists and plasma concentration should be monitored in uremic patients to avoid excessive drug accumulation.

References

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