Moxifloxacin relieves the persistent symptoms of lower urinary tract after cessation of ketamine abuse

To the Editor—We read with great interest the recent article by Cheung et al., in which they described the relationship of urinary symptoms and quality of life after cessation of ketamine in female abusers. Herein we describe a patient with persistent lower urinary symptoms after cessation of ketamine. Confusingly, his symptoms improved significantly following treatment with moxifloxacin.

A 24-year-old man was forced to stay in a drug rehabilitation centre for 3 months from May to July 2011, where he accepted cessation of ketamine therapy, as he had a 2-year history of severe lower urinary tract symptoms (LUTS) after abusing the drug for almost 3 years. There was no improvement in symptoms for up to 2 months after cessation of ketamine. Two urine cultures grew no bacteria, mycoplasma, chlamydia, or acid-fast bacilli in the specimens. Nevertheless, treatment with moxifloxacin 400 mg daily was commenced for 1 week when he accepted this additional treatment for the first time. Surprisingly, the patient’s LUTS and quality-of-life score decreased significantly. In particular, his suprapubic pain and dysuria improved markedly from the second day of moxifloxacin therapy, and 1 day after discontinuing the drug they gradually recurred and finally returned to the level present prior to starting the medication. He was deemed to require moxifloxacin 400 mg daily, because without such treatment his symptoms recurred and when it was restarted they resolved. He still comes to our hospital intermittently for moxifloxacin to relieve his LUTS. Others have reported no such benefits from antibiotic therapy under these circumstances, and so the apparent response to treatment with moxifloxacin was surprising and difficult to explain. Notably, the patient also mentioned experienced increasing haematuria for a short period after moxifloxacin treatment was initiated, and that he still has some dysuria.

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Declaration
No conflicts of interest were declared by the authors.

References

Management of mercury exposure in Hong Kong Poison Centre

To the Editor—In their article on the assessment and clinical management of patients with mercury poisoning, Fan et al.1 studied individuals who were non-occupationally exposed to mercury. The authors presented the results of a retrospective analysis (41 persons) who were referred to the Hong Kong Poison Information Centre and described their experience clearly.1 We were pleased to see that the conclusions are fair and in line with those suggested previously.2 However, two points in the report by Fan et al need clarification. First, a sentence of the Results in the Abstract states that “Individuals with abnormal tissue mercury levels were uncommon.”1 This statement incorrectly suggests that both blood and urine mercury concentrations may reflect the ‘tissue’ body burden of mercury. Instead, both whole blood as well as urine mercury levels are not able to identify chronic mercury poisoning in exposed individuals.3,4 Several reported cases suggest that inconsistencies may occur between the patient’s urinary mercury levels and symptoms of severe mercury poisoning, even in accidental exposure.4 Second, the authors also claim that “Removal of existing amalgams without a good dental indication is also not advised, as this would...
temporarily raise blood Hg levels through inhaling more vapour. We would like to add that since 2003 we have developed a new technique for the removal of amalgam. This procedure makes it possible to control the release of mercury vapour during amalgam-replacement therapy because the entire mercury filling is removed en bloc, and mercury levels in saliva, blood, and urine did not oscillate from baseline levels.

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References

Authors’ reply

To the Editor—We agree to Guzzi and Pigatto’s comment that the blood and urine level of mercury may not reflect the body tissue burden of mercury. For mercury-poisoned patients, the best marker for body tissue burden should be the concentration in the effector organs, ie the brain and kidneys. However, in most scenarios, these concentrations are not measurable unless at autopsy. It is not practical to rely on these measurements to diagnose mercury poisoning. Therefore, in the report, we have stressed the importance of obtaining a detailed exposure history, evaluating the clinical signs and symptoms and measuring the blood and urine mercury levels. As for Guzzi’s technique of amalgam removal, we have no further comments.

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Malpractice claims: corrections

To the Editor—The authors in the Commentary “Medical malpractice: prevention is often a better strategy” in the October 2011 issue1 would like to make two corrections.

First, the Bolam case was quoted under the wrong name of Bolam v Chelsea and Kensington Hospital Management Committee [1968] 1 QB 428. It should have been Bolam v Frien Barnet Hospital Management Committee [1957] 2 All ER 118. In Bolam, McNair J enunciated the legal requirement for the standard of care in medical negligence, which is that in accordance with a practice accepted as proper by a responsible body of medical men skilled in the particular art. In another case, Barnett v Chelsea and Kensington Hospital Management Committee [1968] 1 QB 428, a watchman arrived in the Accident and Emergency nauseated after a cup of tea. The casualty doctor simply advised that he should see his general practitioner. The man died 5 hours later from arsenic poisoning. The court found that the man would have died anyway because it was too late to save him and it was not the casualty doctor’s negligence that had caused his death. The claimant thus failed to establish causation. This