Ketamine-Associated Uropathy in Therapeutic Contexts: Reply to Abdelrahman

To the Editor: I thank Dr Abdelrahman for his interest in my article[1] and for his thoughtful comments[2] on it. I concur with his concerns about the risk of uropathy in recreational users of ketamine. However, I am less certain that "ketamine-associated uropathy poses a low but meaningful risk even in therapeutic settings"; the references that he cites[3,4] insufficiently support this assertion. I had provided a critical appraisal of the review by Kerr-Gaffney et al[3] in my article[1] and will not repeat my comments here. I agree that the case report by Chang et al[4] is more convincing; however, the patient described in the report was prescribed moderately high doses of oral ketamine, 240 mg per dosing occasion, at the high frequency of 4 times a week for an unspecified period, perhaps running into months or longer. Such dosing more closely approximates recreational use than therapeutic use. In usual therapeutic contexts, when treating depressed patients with a view to achieving sustained remission, it is more common to administer oral doses of 150–200 mg thrice a week for 2–3 weeks, to afterward taper the frequency to once weekly, and to then discontinue with the hope that the ongoing regime of conventional antidepressant treatment will maintain the improvement.

On the one hand, it is reassuring that ketamine-associated uropathy has so rarely been reported despite (presumably) hundreds of thousands of patients having received the treatment for depression during the past 1-2 decades. On the other hand, as Dr Abdelrahman correctly points out, the absence of evidence should not result in dismissal of concerns. Absence of evidence may be a result of unawareness, underassessment, and underreporting, which is why my article did offer recommendations for early detection among other risk mitigation strategies.

I take this opportunity to reiterate my suggestion about hyperhydration and frequent emptying of the urinary bladder in relation to especially oral ketamine sessions.[1] Doing so will reduce the intensity and duration of exposure of the bladder to ketamine and its metabolites, thereby potentially reducing the risk of cystitis. Whereas this suggestion is not evidence-based, it is intuitive. Taking these precautions literally costs nothing, may help, and should not be summarily dismissed.

Finally, the suggestion for multidisciplinary structured surveillance will burden the healthcare system if implemented routinely. It is better to first gather evidence to determine the true risk and to afterwards construct recommendations for more intensive monitoring of therapeutic ketamine by a multidisciplinary team. However, I do agree that when it is necessary to treat patients with high-dose, high-frequency ketamine for periods running into months to years, close urological monitoring would be wise.

References

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