

Case Report: Ketamine-Induced Nephropathy and Uropathy Authors / Investigators: DelaRosa, V, Davalath, D, Mathews.R AtlantiCare Regional Medical Center, Atlantic City, N.J., U.S.A.

Introduction

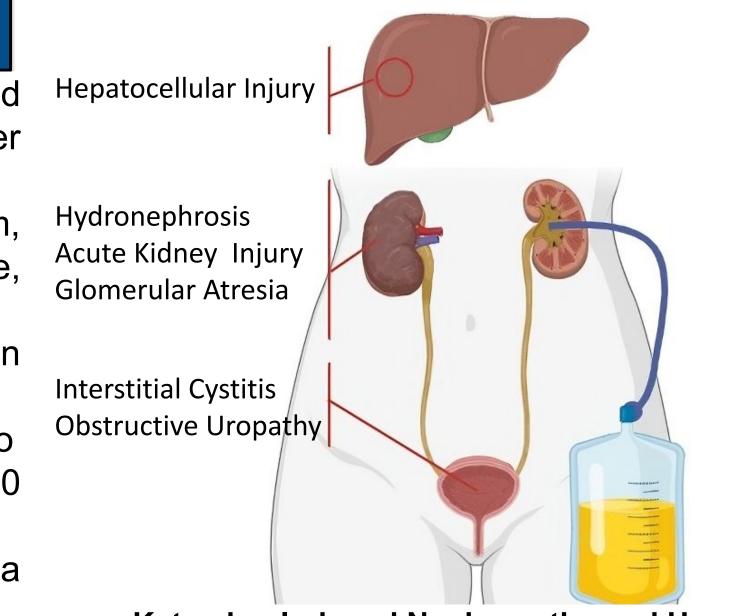
- Prolonged and excessive ketamine use can lead to Ketamine-Induced Nephropathy and Uropathy (KINU), with symptoms such as hydronephrosis, inflammatory cystitis, and liver function abnormalities.
- Medication-induced nephropathy is believed to stem from irritation of the urologic system, leading to symptoms such as urge incontinence, reduced bladder compliance and volume, detrusor overactivity, hematuria, and rarely, hydronephrosis or papillary necrosis.
- Recent data highlights an increase in ketamine misuse among individuals aged 16–24 in the United Kingdom, with usage increasing from 1.9% in 2008/2009 to 2.9% in 2018/2019.
- Ketamine usage is alarmingly high in Asian countries like China and Taiwan, with up to 40% of drug users resorting to this substance with Hong Kong reported over 2000 documented cases of ketamine abuse in 2014.
- This report focuses on a case of Ketamine-Induced Nephropathy and Uropathy (KINU) in a 24-year-old female patient

Discussion

- This case report exemplifies the intricate link between chronic ketamine abuse and the resulting kidney and bladder damage.
- The patient developed reversible hydronephrosis, a ureter blockage that can result in kidney failure, due to the precipitation of ketamine metabolites within the ureters causing obstructive renal failure.
- Ketamine abuse has also been associated with inflammatory cystitis, an inflammation of the bladder that can lead to low bladder volume
- Furthermore, the patient exhibited abnormal liver function tests, another potential complication of ketamine abuse.
- The exact process by which ketamine causes uropathy and hepatobiliary damage is not fully understood. However, possible explanations could include direct effects of ketamine or its metabolites, immunological reactions to impurities in street ketamine, or interactions with other abused substances.
- Clinical improvement after the insertion of a percutaneous nephrostomy tube is evident in our case with improvement of serum creatinine levels from 5.0 to 2.37 mg/dL and GFR from 12 to 29 mL/min/1.73 m² in a span of 3 days.

Conclusion

Ketamine-induced nephropathy and its associated complications are progressively emerging as a formidable healthcare challenge, especially among the younger demographic engaged in recreational ketamine abuse. Timely recognition of ketamine-induced uropathy is pivotal for instituting effective management strategies. Severe cases characterized by hydronephrosis may necessitate interventions such as percutaneous nephrostomy to safeguard the integrity of the upper urinary tract.



Ketamine-Induced Nephropathy and Uropathy s/p PCN placement



Image 1: CT Abdomen and Pelvis. Abnormal appearance of both kidneys. Bilateral Hydronephrosis. Both kidneys are heterogeneous with lobulated contours and some scattered internal low attenuation areas.

Case presentation

Our patient, a 24-year-old Asian female with a past medical history of renal failure due to ketamine abuse, presented with complaints of epigastric pain and bilateral flank discomfort. Her medical history disclosed a 1.5-year history of ketamine abuse as a means to self-manage mental depression. Her daily ketamine consumption ranged from 200-500mg, obtained illicitly, and notably, she had not been prescribed any antidepressant medications.

Approximately 1.7 years before her current admission, after a period of 2 to 3 months following antibiotic treatment for UTI, the patient experienced a recurrence of bilateral flank pain and frequent urination (8-10 times a day, with small volumes). The patient noted the absence of hematuria, fever, nausea, vomiting, abdominal pain, or suprapubic pain. One year before her current admission, the patient sought care at NYU. She underwent a month-long hospitalization, which included two weeks for the placement of a nephrostomy tube and two weeks in the psychiatric unit. During her psychiatric unit stay, she received medication (details of which she cannot recall) that partially alleviated her depressive symptoms. However, upon discharge, she continued to use ketamine once daily as a means of managing her depression. Three months before her current admission, the bilateral nephrostomy tube was removed at NYU. In the intervening period, the patient persisted in daily ketamine use for depression. One day before her current admission, the patient's tolerance for bilateral flank pain reached its limit, prompting her to seek admission for further evaluation and care.

The primary diagnosis upon admission was sepsis, secondary to acute pyelonephritis, with concomitant Pseudomonas bacteremia and AKI, attributed to bilateral hydronephrosis and obstructive uropathy. Imaging via CT of the abdomen without contrast revealed bilateral hydronephrosis, with both kidneys presenting heterogeneous and lobulated contours. Urinalysis displayed positive leukocyte esterase and a significant presence of non-lactose fermenting gram-negative rods, with a urine culture showing a bacterial load exceeding 100,000 CFU per mm. To address the sepsis, the patient was treated with cefepime, which was de-escalated from meropenem, and vancomycin. Notably, her overall renal function exhibited marked improvement following the replacement of the percutaneous nephrostomy tube.Her initial creatinine level was alarmingly elevated at 5.0, with an estimated glomerular filtration rate (eGFR) of 12. Sodium levels were recorded at 124, and transaminitis was also evident. Remarkably, following the placement of percutaneous nephrostomy tubes (PCN), her creatinine levels exhibited a remarkable improvement, dropping from 5.0 to 2.37 within just three days. Furthermore, her GFR showed a substantial increase from 12 to 29 in the same timeframe.

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