vilazodone. Though DM was a potential confounding factor in the first case, there was no confounding factor in the second case. It is therefore suggested that there is an involvement of this drug in increasing plasma sugar level.

There is a need to have more studies to find out whether vilazodone-induced hyperglycemia is dose-dependent or idiosyncratic and also to examine the complex relationship and mediational pathways between vilazodone and insulin release. Close monitoring of patients on vilazodone for blood sugar levels is therefore warranted.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Intact Higher Mental Functions Despite High Serum Urea and Creatinine Levels in a Patient with Acute Kidney Injury: A Case Report

Sir,

About 34% of the population may suffer from acute kidney injury (AKI).[1] Sudden loss of renal functions seen in AKI leads to the accumulation of nitrogenous waste products such as urea and creatinine in the body. AKI is associated with cognitive dysfunction.[1,2] At urea levels above 40 mg/dl and creatinine levels above 2 mg/dl, progressive drowsiness, disorientation, and amnesia are seen. As the deficits increase further with an increase in urea and creatinine levels, we expect higher values of these to be associated with poorer mental functions.[3] However, we report an unusual case of AKI with intact higher mental functions despite having high serum urea and creatinine levels.

The patient is a 28-year-old female diagnosed with adjustment disorder with mild depressive symptoms for the past three weeks. This was in relation to frequent arguments and interpersonal issues with her husband, who was staying separately for a month. She had a mixed diet. There was no history related to renal dysfunction. She had pallor but no physical signs of renal injury or urinary disturbances. She had nonpervasive low mood, with occasional death wish. However, during the routine investigations, her serum urea level was 231.5 mg/dl and serum creatinine level was 15.9 mg/dl. Urine routine showed mildly increased protein, and her hemoglobin was 6.7 g/dl. These reports were subsequently re-examined and found to be correct. The estimated glomerular filtration rate (eGFR) came to 2.7 mL/min/1.73 m^2. Her heart rate was 80 beats/minutes and blood pressure was 130/80 mm of Hg. Random blood sugar, electrolytes, and other parameters of complete blood hemogram were within the normal range. Score on the Mini

Mental State Examination was 30/30. Our detailed mental status examination included evaluation of attention, language, memory and new learning ability, construction ability, clock drawing test, fund of information, calculation, abstraction, apraxia, psychomotor speed, right-left disorientation, astereognosis, topographical disorientation, neglect, and alternate hand sequences. We also did cranial nerve examination, motor and sensory nervous systems examination, and fundoscopy. All these domains were remarkably normal. X-ray of kidney, ureter and bladder showed no structural abnormalities. She was diagnosed to have AKI and was referred to a higher center for renal dialysis. The diagnosis of AKI was kept in consultation with the department of medicine, as there were no previous renal problems, diabetes, or hypertension; no rapid loss of kidney functions reported in the last three months, and absence of severe proteinuria. The cause of AKI remained elusive. We could not evaluate the renal function tests of her parents or siblings, to rule out hereditary factors. We also could not associate her depressive symptoms with AKI and did not start her on any medication.

Neurocognitive disturbances are common signs of uremia but were absent in our patient. Her urea level was about six times higher and creatinine was about eight times higher than the normal ranges. Unimpaired cognitive functions at these levels are unusual.[1,4] The cognitive deficits are mainly due to increased nitrogenous waste, changes in electrolytes, and inflammatory reaction impairing the blood–brain barrier integrity.[1,5] The last two factors were not present in our patient, as her electrolytes and white blood cell counts were within normal range. Although cross-sectionally higher urea and creatinine levels are associated with cognitive dysfunction, the rate of change of urea and creatinine may be more important in causing cognitive deficits. A previous case report documented the serial cognitive improvement in a patient on renal dialysis as the serum urea level of 1216 mg/dl and a creatinine level of 27 mg/dl gradually reduced.[2] Those authors found that a rapid correction of urea may lead to higher osmolality disturbances and worse cognitive outcomes. The rate of accumulation of urea and creatinine may be equally important for cognitive disturbances to occur. For this, we require prospective studies to determine the rate of change for these products. This unusual case also shows the need of routine baseline investigations in every patient we treat.

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