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Original Research Paper

Internal Medicine

BUNEMIA: A SEVERE CASE OF UREMIA COMPLICATED BY CHRONIC KIDNEY DISEASE

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ABSTRACT

Uremia is characterized by elevated nitrogen compounds in the blood and often arises from renal impairment. It can lead to severe complications like metabolic encephalopathy and platelet dysfunction. Supportive care with intravenous fluids can improve renal clearance. Severe sequela may necessitate dialysis. We describe a 56-year-old male with stage five chronic kidney disease who presented with reduced fluid intake, pan-colitis, and diarrhea. CT of the abdomen and pelvis without contrast reported non-specific pan-colitis with wall thickening. Blood urea nitrogen on presentation was 260 mg/dL and serum creatinine 17.09 mg/dL. Despite initial improvement in renal function with intravenous fluids and bicarbonate therapy. The patient subsequently developed stroke and hypertensive urgency. Dialysis was started for possible uremic encephalitis complicating right occipito-parietal ischemic stroke. After a challenging clinical course, discussions with the family led to the withdrawal of care, and the patient ultimately passed away. This case underscores the efficacy of hemodialysis in managing severe uremia, even when complicating factors such as chronic kidney disease are present. It also highlights the importance of considering intravenous fluids as an initial approach when dialysis is not immediately available. There is conflicting evidence of reduced mortality at 30 days with early initiation of hemodialysis. The impact of chronic uremia on mental status remains an area of uncertainty, warranting further investigation for prognostic and management considerations in similar cases.

KEYWORDS :

INTRODUCTION:

Uremia, or rising BUN (blood urea nitrogen), is generally associated with worsening renal function in the setting of end-stage kidney disease due to primary or secondary causes. BUN is predominantly eliminated via glomerular filtration, and the plasma level typically varies inversely with GFR. The production of urea varies with protein intake, liver function, and catabolic rate.

Uremic symptoms appear when creatinine clearance drops below 10 ml/min. It causes decreased platelet number and adhesion and increases their turnover, resulting in increased susceptibility to bleeding. Moreover, it decreases hydrogen ions and organic acid secretion buildup, which leads to increased anion-gap metabolic acidosis and hyperkalemia. It can also cause pericarditis, worsening cardiac valve abnormalities, and reproductive hormonal dysfunction, leading to infertility in both males and females.

Case:

A 56 year old non-hispanic non-smoker male with a past medical history of multiple CVA (cerebro-vascular accidents), insulin dependent type 2 diabetes mellitus, stage 5 chronic kidney disease, and hypertension presented with diarrhea for 1-2 weeks but denied melena or hematochezia.

Vitals and blood tests and during hospitalization are denoted in Table 1 and 2 respectively. Urinalysis revealed pyuria, hematuria, and 2+ bacteria. A stool test revealed positive occult blood. CT of the abdomen and pelvis reported non-specific pan-colitis with wall thickening and fat stranding involving the appendix.

On interviewing, I was alert, and my mental status was baseline. Physical examination was significant for right sided weakness and ronchi on lung auscultation.

He was started on vancomycin, cefepime, and flagyl for sepsis, and zosyn for suspected infectious pancolitis. Nephrology recommended 2 liters of bolus fluids and 150 ml/h bicarbonate drip with no urgent indication for dialysis. On day 2, the patient was diagnosed with oliguric stage-3 acute kidney injury, and nephrology increased bicarbonate infusion to 200 ml/hr and 1 liter of ringer lactate. Blood investigations and vital signs was as per Table 1 and 2.

On day 4, the patient reported worsening altered mental status and renal function, which nephrology attributed to uremic symptoms, switched fluids to 100 ml/hr normal saline, and recommended dialysis.

On day 6, the patient was hypertensive (199/78), remains confused and agitated, and plans a second session of dialysis.

On day 12, after multiple sessions of dialysis, the patient was normotensive but still had altered mentation. Over time, the patient's condition worsened, and the family decided to opt for comfort care. The patient was declared on day 17.

Table: 1

Vitals	On admission	Day 2	Day 4	Day 6	Day 12	Day 17
Temperature in Celcius	36.3	36.8	37.3	37.4	37.2	38.6
Blood Pressure mm /Hg	90/43	118/54	176/77	199/78	175/91	130/64

Table: 2

Lab Investigations	On admission	Day 2	Day 4	Day 6	Day 12	Day 15
BUN (mg/dL)	260	229	187	70	15	23
Creatinine (mg/dL)	17.09	14.68	13.18	4.98	2.88	4.62
eGFR(estimated glomerular filtration rate) (ml/min/BSA)	3	4	4	9	25	14
Anion Gap (mmol/L)	28	22	22	16	11	11
WBC (103/μl)	40.7	37.2	25.2	8.9	8.2	5.5
Hemoglobin (g/dl)	8.5	7.4	7.9	7.7	7.4	6.6

Platelets (103/μl)	451	468	274	280	332	409
Lactic acid (mmol/L)	0.7	0.7	-	0.9	-	-

DISCUSSION:

In general, increased BUN suggests impaired renal function. However, BUN levels are increased in congestive heart failure, burns, severe liver disease, and malnutrition[1].

In the past, studies have shown mixed data, of which Liu et al. reported improved survival of patients who started dialysis at BUN levels <75mg/dL, however, a study in the Netherlands reported no mortality benefit in early dialysis with high BUN. Elevated BUN levels are strongly associated with adverse outcomes in acute heart failure patients and are considered a prognostic factor.[1]

Symptoms associated with uremia include vomiting, fatigue, anorexia, weight loss, muscle cramps, pruritus, and changes in mental status. Hypertension, atherosclerosis, valvular stenosis and insufficiency, chronic heart failure, and angina may also develop as a result of a buildup of uremic toxins. Additionally, occult gastrointestinal bleeding is also reported in some cases due to platelet dysfunction.[2,3]

In the past, only two studies reported BUN of more than 200, Persuad et al.[1] reported one 23 year old male with BUN of 244 mg/dl and Raj et al.[2] reported BUN of 213 mg/dL, both were African-american in ethnicity. Both the patients were discharged after dialysis and the child was recommended for renal transplant.[1-3]

Our patient presented with the highest reported BUN in the literature. Except for symptoms of diarrhea and neurological deficits, he was stable and had no other complaints. It is unusual for a BUN level of 260 mg/dL to demonstrate no emergent or worsening signs and symptoms. Over the time during hospital course, he demonstrated neurological symptoms, however, there was significant improvement in uremia.

Regarding the treatment, a high BUN level does not change the management strategy. Dialysis is always the first line of treatment and should be gentle regardless of BUN levels to avoid disequilibrium syndrome. Other options include renal transplantation and peritoneal dialysis in appropriate patients. It is important to avoid protein restriction and malnutrition along with iron, calcium, and vitamin D supplements. Nephrotoxic medications should be avoided, and dosing should be titrated for renally excreted medications.[3,4,5]

Uremia is associated with high morbidity and mortality, hence, treatment should be started early in the disease course and should not be influenced by symptom onset of extreme high levels at the time of presentation.[4]

CONCLUSION:

This case demonstrates the effectiveness of hemodialysis in managing severe uremia. For patients without dialysis access, intravenous fluids might be a viable initial approach. The impact of chronic uremia on mentation remains unclear but may serve as a negative prognostic factor

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