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Case Report

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Hyponatremia induced disequilibrium syndrome in regular hemodialysis: a case report and review of literature

Abstract

Hemodialysis Disequilibrium Syndrome (HDS) is characterized by rapid change of the osmotic gradient between brain and the plasma during hemodialysis leading to neurological manifestations. It is usually attributed to rapid and aggressive hemodialysis. HDS can have various neurological manifestations starting with headache, nausea, vomiting, muscle cramps, tremors up to disturbed consciousness and convulsions. In severe cases, patients can die from advanced cerebral edema. Blood urea play a major role in disequilibrium. In our case, we focus on the serum electrolytes mainly sodium as a contributor for HDS.

Keywords: hyponatremia, cerebral oedema, acute renal failure, chronic renal failure

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Ahmed Akl,[1,2,3] Abdulaziz Osailan,[2] Hanan Faruqui[2]

[1]DR. Soliman Fakeeh Hospital, Jeddah, KSA
[2]Fakeeh College of medical sciences, Jeddah, KSA
[3]Urology & Nephrology Center, Mansoura University, Egypt

Correspondence: Ahmed AKL, MD, FACP, FASN, DR. Soliman Fakeeh Hospital, Jeddah, KSA, Urology & Nephrology Center, Mansoura University, Egypt, Email aiak2001@yahoo.com

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Introduction

Hemodialysis Disequilibrium Syndrome (HDS), known since 1962.[1] It is well-known complication associated mainly with the first sessions of hemodialysis, which may present with mild symptoms like headache, nausea, vomiting or moderate like visual acuity transient instabilities , tremor, muscle cramps or severe forms as acute awkwardness, perplexity, convulsions or even coma.[2,3] Various risk factors been defined to be linked with disequilibrium syndrome, including the initial session of hemodialysis, extreme uremia, age, previous neurological disorders, and severe metabolic acidosis.[4] Blood urea being one of the main contributors of the blood osmosis, the rapid clearance of blood urea by hemodialysis may precipitate brain oedema as the sudden drop of blood urea is not associated with same changes in the interstial brain osmosis leading to influx of water and cellular oedema.[5]

Case report

51-year-old male, previously healthy with no known medical illness, obstructive acute kidney failure was discovered two months ago secondary to bilateral obstructing vesico-ureteric stones. He received urgent hemodialysis and followed by endoscopic removal of the vescio-ureteric stones and insertion of bilateral double J stents. No significant drop of serum creatinine was observed for more than three weeks and was diagnosed having advanced stage of CKD stage 5. He was grunted schedule for regular hemodialysis. He did not follow his schedule and was drinking plenty of tape water to improve the function of his kidney. In one week time he presented to our hospital with generalized fatigue and anorexia. His Laboratory work revealed arterial blood gases: PH 7.36, HCO3 14.5 mmol/l, PCO2 26.2 mmHg, Blood urea nitrogen (BUN) 111 mg/dl, serum creatinine 9.69 mg/dl, Serum potassium 3.6 mmol/l and severe hyponatremia with serum sodium 108 mmol/l. Renal ultrasound showed both kidneys have increase parenchymal echogenicity with mild to moderate hydronephrosis and bilateral renal double J stent.

Immediate start of intravenous 3% sodium chloride 10 meq/hour for consecutive 4 hours aiming to reach the target of serum sodium 115 mmol/l in the first 24 hours to avoid rapid correction with serum sodium monitoring every 6 hours. Serum sodium reached 115 mmol/l then in the following 6 hours it was 119 mmol/l. Next day, patient suffer uremic manifestations of gastropathy in the form of nausea, vomiting and fluid overload in the form of shortness of breath with increase demand of oxygen. Urgent hemodialysis session was initiated. After hemodialysis session Laboratory investigations showed arterial blood gases: PH 7.4, HCO3 27.1 mmol/L, PCO2 37.9, BUN 48.2 mg/dl, serum creatinine 4.66 mg/dl , low serum potassium 3.0 mmol/l and serum sodium reached 133 mmol/l. Data representing the serial monitoring of sodium and BUN levels are shown in Figure 1. In the third day post hemodialysis, patient suffer sudden weakness of both legs associated with slurred speech, followed by disturbed conscious level. He was transferred to intensive care unit (ICU) because of the aggressive changes in conscious level and agitations.

In ICU, he suffered an attack of tonic-clonic grand-mal fits, diagnosed as metabolic encephalopathy versus central pontine myelinolysis by our neurologist. Brain magnetic resonance (MRI) and computed tomography (CT) were done and revealed normal grey/white matter differentiation of both cerebral hemispheres, no mass effect or midline shift. No evidence of pontine myelinolysis was found. Based on this information disequilibrium syndrome was confirmed in a regular hemodialysis patient. Reviewing the data of serum Osmolality = (2 x (Na + K)) + (BUN / 2.8) + (glucose / 18), the sudden rise of serum sodium was associated with aggressive clearance of serum BUN that is why daily monitoring of osmolality failed to show major change to precipitate HDS. After 3 weeks of incremental hemodialysis sessions patient conscious level returned back to normal level with no attacks of seizure and normal cognitive functions.

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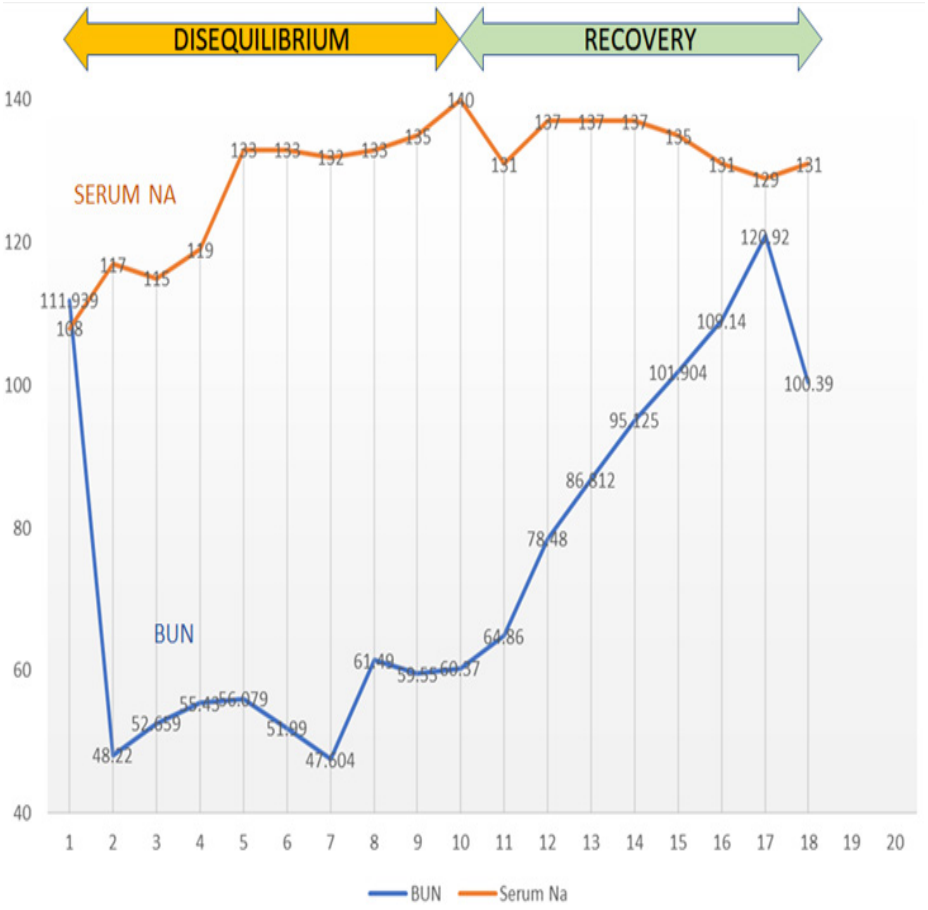

Figure 1 Daily levels of serum bun and sodium.

Discussion

Diagnosis of hemodialysis disequilibrium syndrome based on exclusion of other similar disorders of increased intracranial pressure (ICP) secondary to subdural hematoma, acute stroke, nonketotic hyperosmolar coma, dialysis dementia, severe hypoglycemia or severe hyponatremia.[6] The advances in neuro-monitoring and monitoring of intracranial pressure represent the mainstay of care and guide clinicians to target the therapeutic intervention.[7] Although post hemodialysis cerebral edema has been reported to be considered among the main reasons of acute deteriorated neurological condition, the direct evidence of ICP changes in HDS has been rarely reported.[3,8,9] First hemodialysis has been thought to increase the risk of HDS. In our case, no reported HDS were shown in the first four times of hemodialysis. We thought that associated hyponatremia might offer additional risk factor to precipitate HDS in our case. Therefore, the HDS didn't occur in our patient during the first-time hemodialysis as patient had normal serum sodium at that time. However, HDS was reported to be associated with cerebral edema and increased ICP,[10] in our case no evidence of brain oedema could be found.

In the prevention of HDS during hemodialysis, a slowly gentle start of hemodialysis, increasing dialysate sodium levels, and administration of osmotically active substances have been described.[4,6] In our case we could not have increase of sodium dialysate levels to avoid over correction of serum sodium levels as we are obliged to have only 8-10 meql/l change in 24hours.[11] It is recommended to replace hemodialysis in such situation with continuous veno-venous hemofiltration or sustained low-efficiency dialysis to allow gradual osmotic changes between blood and cerebrospinal fluid to reduce the risk of HDS especially in patients with hyponatremia.[4,9] If the patients developed symptoms suggestive of HDS, slowing or stopping dialysis to raise the plasma osmolality may be required and effective as we did in our case. Series studies including the CT scan or MRI of brain should be performed to exclude the other possible causes as mentioned above.

Conclusion

HDS is a complication that can occur at any time during regular dialysis if precipitating conditions were fulfilled. Sodium as a contributor in serum osmolality played a major role in triggering the HDS in our patient. Early and accurately diagnosis of HDS secures the optimal management and patient survival.

Acknowledgments

None.

Conflicts of interest

The author declares there is no conflict of interest.

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