Case Report

Iatrogenic Hypernatremia; Still Not Uncommon

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ABSTRACT

Background: FWe are aware that hemodialysis is a routine outpatient treatment for millions of patients with a proven safety record, accidents and errors do occur from time to time. Hypernatremia in CKD 5D is although rare and is often caused by technical failure.

Case presentation: We report the case of 50 year old male with symptomatic hypernatremia which manifested as altered level of consciousness immediately after dialysis. The hypernatremia was caused by technical error conductivity reading. Patient improved without any neurological deficit after hemodialysis treatment of the patient.

Conclusions: We should never ignore conductivity alarm and there should be urgent examination of the dialysate if the patient develops rapid onset altered sensorium.

Keywords: Hemodialysis; Iatrogenic hypernatremia; Sodium; Dialysis; Chronic kidney disease

Introduction

Improvements in hemodialysis machine technology, dialyzers, and consumables have allowed hemodialysis to evolve from a treatment limited to a minority of patients with acute kidney failure in the 1950s to a life-sustaining routine outpatient treatment to millions of patients with chronic kidney disease worldwide [1]. Hypernatremia in chronic kidney disease Stage 5 on dialysis (CKD 5D) is rare now a days and is often caused by technical failure which may lead to asymptomatic cases to patients with severe neurological symptoms [2]. In hemodialysis dependent patients, improper selection of dialysis concentrate, wrong labelling of container, failure of dialysis machine calibration, manufacturing error or manual error of overriding of conductivity alarms are the common causes [1]. The dialysis machines in use now-a-days mostly have online conductivity monitor which allow better adjustments and control of serum sodium [3]. The mortality relationship between serum sodium and high dialysate sodium is equivocal [4]. There is U shaped correlation observed between serum sodium and mortality among hemodialysis dependent patients [5]. We report a case of acute hypernatremia leading to altered sensorium during dialysis and recovered fully without any neurological damage.

Case Report

A 50 years old male, who was a known case of Type 2 Diabetes Mellitus (T2DM) for last 20 years, hypertension for last 10 years and was on dialysis for last 3 years. Before this admission, he went to outside dialysis center for routine hemodialysis sessions with no new complaints. His vital parameters were Pulse 88/min and BP 160/88 mmHg as per record. He was average built with weight of 68kg. During the current session of hemodialysis which was started in morning at 10am, he developed sudden onset of altered sensorium after 2 hours of hemodialysis. The initial possibilities kept for this clinical

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deterioration were hypotension, hypoglycaemia, dyselectrolytemia, intracranial bleed, ischemic stroke and less likely dialysis disequilibrium syndrome. The examination revealed vital parameters of pulse 96/min and BP of 152/84. Blood sugar at that time was 99 mg/dl. The former two possibilities were ruled out with clinical examination and bedside blood glucose testing. He was presented in this stage at our hospital. On central nervous system examination, he was in semicomatosed state with a GCS score of 12, with no focal deficits. His pupils were reacting to light and having equal light reflex. There was no hypoglycaemia, hypoxemia or introduction of any new drug. His laboratory investigations were immediately sent for ruling out other possibilities. His investigation reports revealed Hb 9.8 g/dl, Total Leucocyte Count (TLC) 8100 cu mm, platelets 108000/microL, , Blood Urea Nitrogen (BUN) 91 mg/dl, serum creatinine 14.9 mg/dl, serum potassium >6 mEq/L, serum sodium of 173 meq/l, serum calcium 9.2 mg/dl, serum phosphorus 4.4 mg/dl, Aspartate Aminotransferase (AST) 12 IU/L , Alanine Aminotransferase (ALT) 16 IU/L, Alkaline Phosphatase (ALP) 107 IU/L, total serum protein 5.8 g/dl, serum albumin 3.2 g/dl, prothrombin time 14.2 sec and International Normalized Ratio (INR) 1.04. He underwent Non Contrast Computed Tomograhy (NCCT) scan of head to rule out intracranial bleed which was reported as normal. His blood gas analysis revealed ph 7.28, po2 51.0, pco2 31.7, hco3 15.5, sodium 172 mEq/L, potassium 5.19. So, hypernatremia with a serum sodium of 173 mEq/L was considered as the most probable reason for his altered sensorium. His free water deficit was calculated and found to be 9.7 Litres. His urine output was 700 ml/day. However, this large volume of fluid cannot be given to the patient because of the kidney failure and dialysis dependency. Hence, he was managed with IV fluids along with dialysis to prevent fluid overload as well as for CKD. The patient was initially managed with intravenous Dextrose and half Normal Saline one litre per day through intravenous and ryle,s tube. The hemodialysis sessions were modified to short duration of 2 hours and technique of cocurrent dialysis. The sodium concentration in dialysis fluid was kept as 148 meq/L. The sensorium of patient was gradually improved over a period of 2 days along with normalization of serum sodium values. He required a total of 4 sessions of hemodialysis.

The possible causes of hypernatremia in this case could be due to iatrogenic, improper dialysis concentrate, overriding of conductivity alarm or the underlying disease. Improper dialysis concentrate along with overriding of conductivity alarm was found to be the most appropriate cause of hypernatremia [6,7].

Discussion and Conclusions

There is report in 2013 from United Kingdom Product Regulatory Authority on adverse incidents in dialysis noted that 22% were due to failure of dialysis equipment or disposable while 19% were due to failure of dialysis machine. As our case was of acute severe hypernatremia and CKD 5D which cannot be corrected by the conventional treatment for hypernatremia, in these patients well defined methods or guidelines have not been defined. Usual conventions are that one should avoid rapid correction. For treatment of hypernatremia, there are case reports of correction of severe hypernatremia with Hemodialysis and 3% normal saline6. In our patient we decreased the flow of dialysate and duration of dialysis and done concurrent dialysis along with fluid replacement. So there is slow correction and full recovery of the patient. We should never ignore conductivity alarm and there should be urgent examination of the dialysate if the patient develops rapid onset altered sensorium. There is a study by Navarro et al stating that during bicarbonate dialysis, there are chances that an inappropriate acidic dialysate could replace the normal dialysate and no alarms are triggered. Timely information of these changes in patients to the treating Nephrologist can prevent new avoidable complications. Although most renal physicians rely on renal technologists for support, they should be aware of the potential problems associated with dialysate errors, so that prompt action can be taken to rectify the situation.

References

1. Davenport A. Complications of hemodialysis treatments due to dialysate contamination and composition errors. Hemodial Int 19, S30-S33 (2015).
2. Obialo CI, John S, Bashir K. Iatrogenic hypernatremia in hemodialysis patients: A result of erroneous online conductivity monitor and conductivity meter reading. Hemodial Int 21(4), E73-E5 (2017).
3. Levin A, Goldstein M. The benefits and side effects of ramped hypertonic sodium dialysis. J Am Soc Nephrol 7(2), 242-246 (1996).
4. Hecking M, Karaboyas A, Saran R, et al. Predialysis serum sodium level, dialysate sodium, and mortality in maintenance hemodialysis patients: The Dialysis Outcomes and Practice Patterns Study (DOPPS). Am J Kidney Dis 59(2), 238-248 (2012).
5. Rhee CM, Ravel VA, Ayus JC, et al. Predialysis serum sodium and mortality in a national incident hemodialysis cohort. Nephrol Dial Transplant 31(6), 992-1001 (2016).
6. Marcano KD, Burry S, Aleman J, et al. Hemodialysis With 3% Normal Saline: An Uncommon Treatment to Reduce Risk of Cerebral Edema in Severe Hypernatremia Secondary to Parathyroid Carcinoma. Chest 152(4), A308 (2017).
7. Navarro JF, Mora-Fernandez C, Garci J. Errors in the selection of dialysate concentrates cause severe metabolic acidosis during bicarbonate hemodialysis. Artificial Organs 21(9), 966-968 (1997).