Accelerated Dialysis Disequilibrium Syndrome: A Fatal Case

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ABSTRACT

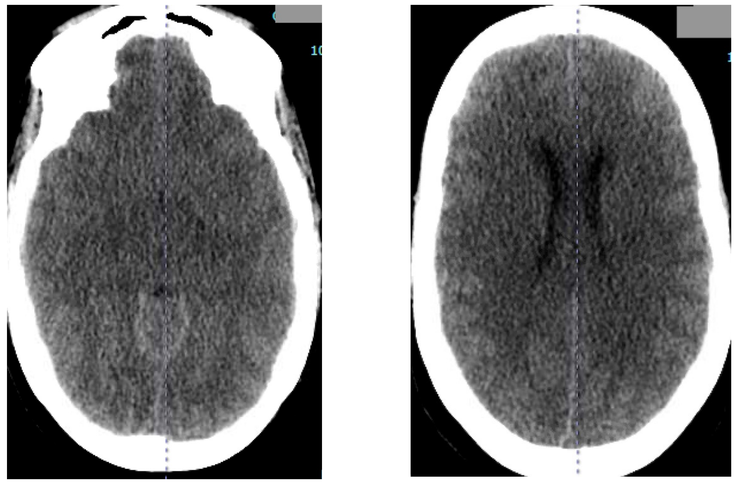
Hemodialysis complications are a common reason for rapid response in hospitals. Dialysis disequilibrium syndrome (DDS) is a rare complication that leads to cerebral swelling and traditionally precipitated by the reverse osmotic shift effect of severe azotemia. Most cases reported in literature related to fatal outcomes in DDS have the common denominator of chronic azotemia greater than 150mg/dL in addition to severe acidosis. Paradoxical brain acidosis generates an osmolar gradient that leads to cell edema and has been proven in animal models. Even though a consensus to target a low urea reduction ratio (URR) with a slow-short dialysis session for the prevention of DDS exists for chronic dialysis patients, the role of acidosis correction remains unknown. Predictive strategies to minimize fatal DDS events are scarce and acute rescue interventions are mostly anecdotal in the critical care community. Hereby we highlight the temporal relation between severe azotemia, the paradoxical brain acidosis effect and fatal DDS.

CASE SUMMARY

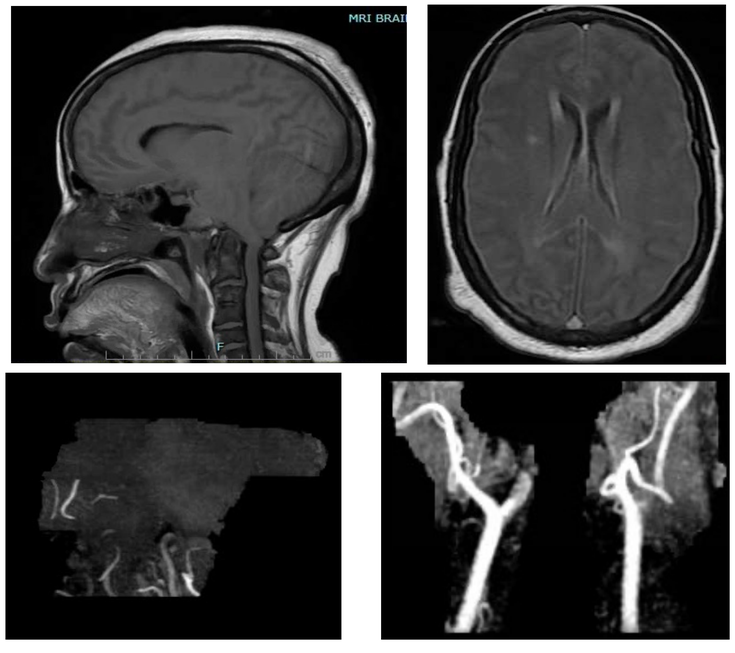
A 57-year-old African American female with chronic kidney disease stage four.

- Chief complaint: shortness of breath and anuria.
- On arrival with stable hemodynamics but tachypneic at 28rpm.
- Initial blood work: WBC 17k/uL, Na 138mEq/L, K 2.6mEq/dL, NaHCO3 <5mEq/L, BUN 116 mg/dL (baseline 40-50 mg/dL), Cr 8.56mg/dL, Mg 1.3mg/dL, Albumin 2.4g/dL. Osmolarity 328mOsm/kg.
- Underwent hemodialysis at a blood flow rate (BFR) of 250ml/min with total therapy time of 110 minutes and no ultrafiltration.
- Near to completion of the dialysis session, the patient became hypoxic and hypotensive, requiring endotracheal intubation and ICU level of care.
- While in ICU patient was noted with loss of spontaneous movement and brain stem reflexes.
- The patients deteriorating clinical condition was refractory to salvage hyperosmolar treatment and was transitioned to palliative care and organ procurement.
- The patient total hospital length of stay was 11 hours.

ICU IMAGING



(Top)Brain-CT-Scan: generalized diffuse edema



(Top)Brain-MRI-Scan: whole brain infarction with mild trans-tentorial and cerebellar tonsillar herniation.
(Bottom)Brain-MRA-Scan: limited-flow within the proximal intracranial cervical and vertebral vertebral/basilar system. No definite intracranial flow throughout the distal anterior and posterior circulation. Little flow shortly after the bilateral internal carotid artery bifurcations

ESTABLISHED DDS RISK FACTORS

- First dialysis session.
- Children and elderly patients.
- BUN >150mg/dL.
- Hypernatremia and hyperglycemia.
- Metabolic acidosis.
- Pre-existing neurological disorder, cerebral edema and any condition that increases blood-brain barrier permeability.

DISCUSSION

- The most recent preventive guidelines for DDS recommend a short-slow dialysis session of less than two hours with blood flow rates of 150-250 mL/min and a target urea reduction ratio of less than 45%.
- Our case had a URR of 57% at a slow-rate dose with a bicarbonate correction of more than 10 points.
- Hence, we theorize that the degree of acidosis confers a stronger predictor for DDS and accelerates the process of cerebral swelling leading to fatal outcomes.
- Although interventions such as mannitol infusion and sodium modeling have been used to mitigate large changes in osmolality, none exist for bicarbonate correction.
- A predictive point scale tool that could help identify patients at a higher risk for cerebral edema has never been established or tested and mainly due to the rarity of the disease and low recognition.
- A registry of patients with fatal DDS is necessary to develop predictive analysis.
- Lastly, we consider that patients with combined severe azotemia and acidosis should have stabilization of bicarbonate levels prior to initiating dialysis.

REFERENCES

1. Mistry K: Dialysis disequilibrium syndrome prevention and management. Int J Nephrol Renovasc Dis. 2019, 12:69-77. 10.2147/IJNRD.S165925
2. DiFresco V, Landman M, Jaber B, White A. Dialysis disequilibrium syndrome: an unusual cause of respiratory failure in the medical intensive care unit. Intensive Care Medicine. 2000;26:628–630. doi: 10.1007/s001340051214.
3. Anna Curtis, Christian Lamb, Hussain Rao, Andrew Williams, Amit Patel, "Dialysis Disequilibrium Syndrome and Cerebellar Herniation with Successful Reversal Using Mannitol", Case Reports in Nephrology, vol. 2020, Article ID 8850850, 4 pages, 2020. https://doi.org/10.1155/2020/8850850.
4. Bagshaw, S.M., Peets, A.D., Hameed, M. et al. Dialysis Disequilibrium Syndrome: Brain death following hemodialysis for metabolic acidosis and acute renal failure – A case report. BMC Nephrol 5, 9 (2004). https://doi.org/10.1186/1471-2369-5-9.