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A Case of Sjögren's Syndrome-associated Fanconi Syndrome Presenting with Hypokalemic Cardiac Arrest and Complicated by Osmotic Demyelination Syndrome

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Objectives : Introduction: Sjögren's syndrome (SS) can manifest with severe renal tubulopathy, including Fanconi syndrome (FS) and distal renal tubular acidosis (dRTA). These conditions predispose patients to profound electrolyte disturbances that can be fatal. We report a rare case of cardiac arrest triggered by FS-associated hypokalemia, further complicated by osmotic demyelination syndrome (ODS) despite the absence of preceding hyponatremia.
Methods : .
Results : Case Presentation: A 45-year-old female with SS visited our emergency room following head trauma sustained during a fall caused by progressive generalized weakness, which rapidly progressed to cardiac arrest induced by profound hypokalemia. Initial laboratory findings (Table 1A,1B) confirmed SS-associated FS, presenting with severe hypokalemia, hypernatremia, extreme hypophosphatemia, and massive tubular proteinuria (UPCR 4.93 g/g and UACR 0.519 g/g). After resuscitation, serum sodium was corrected to 147 mEq/L within 48 hours, and potassium normalized by day 5. However, the clinical course was complicated by septic shock from pneumonia, requiring mechanical ventilation and vasopressor support. On day 7, shortly after discontinuing midazolam sedation, the patient experienced a 15-minute generalized seizure, at which time serum sodium had risen back to 153 mEq/L. Subsequent brain MRI (FLAIR) demonstrated diffuse expansile hyperintensity in the pons and middle cerebellar peduncles (MCP), consistent with ODS (Figure 1). After a period of challenging management due to persistent electrolyte fluctuations, serum sodium and phosphorus levels were eventually stabilized. Following this comprehensive stabilization, the patient's consciousness and motor functions gradually recovered.
Conclusions : Conclusion: This case highlights that Fanconi syndrome in Sjögren's syndrome can lead to life-threatening events. Furthermore, ODS can occur in the absence of hyponatremia, driven by rapid sodium fluctuations under a 'second-hit' mechanism involving severe hypokalemia, hypophosphatemia, and systemic inflammation. Achieving multi-electrolyte stability while minimizing sodium fluctuation is paramount to preventing potentially irreversible neurological injury in critically ill patients with complex renal tubulopathies.

Table 1A & 1B.png

Blood test	Results	Urine analysis	Results
Hemoglobin (g/dL)	11.4	Glucose	2+
WBC (*1000/μL)	31.96	Protein	1+
Platelet (*1000/μL)	314	Creatinine (mg/dL)	22.06
BUN (mg/dL)	25.1	Sodium (mEq/L)	105.9
Creatinine (mg/dL)	1.10	Potassium (mEq/L)	8.2
Sodium (mEq/L)	160.5	Chloride (mEq/L)	83.4
Potassium (mEq/L)	1.7	Uric acid (mg/dL)	6.2
Chloride (mEq/L)	122	Calcium (mg/dL)	7.8
Uric acid (mg/dL)	3.3	Phosphorus (mg/dL)	3.9
Ionized calcium (mg/dL)	4.5	UPCR (<0.15g/g)	4.93
Phosphorus (mg/dL)	0.7	UACR (<0.03g/g)	0.519

Table 1A & 1B.png