# Osmotic Demyelination Syndrome: A New Mime in the Circus of Neurology

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A 34-year-old man with end-stage kidney disease presented with new onset blood pressure lability, acute gait difficulties, bilateral leg weakness, hypersomnia, fecal incontinence, and urinary retention within 2 weeks of starting dialysis. His maximum urea was 40 mmol/L, and he was gently dialyzed with three short runs, leading to a 50% reduction in his urea over 3 days. His initial mild hyponatremia (132 mmol/L) was slowly corrected.

Examination revealed proximal hip flexor weakness (4/5), mild vibration loss distally, 1 + Achilles and patellar reflexes, and equivocal plantar reflexes with no saddle anesthesia. Upper extremities and coordination were normal. A computed tomography (CT) of the brain was unremarkable (Figure 1A). Full spinal magnetic resonance imaging (MRI) was performed, revealing mild radicular involvement in L4–L5 (Figure 1B), inconsistent with cauda equina syndrome (CES).

Given his proximal-predominant weakness, blood pressure lability, and urinary/bowel involvement, differential diagnosis included dysautonomia, myopathy, and polyneuropathy (including ganglionopathy). Electromyography revealed normal nerve conduction but a mild myopathic pattern in the iliopsoas muscles. Uremic-related myopathy was considered, given his history of kidney disease, but this did not explain his bowel/bladder symptoms. A kidney biopsy found thrombotic microangiopathic (TM) changes related to severe hypertension, but no features of immune or amyloid pathology to explain the systemic dysautonomia. An MRI of the brain was ordered, revealing a T2-hyperintense splenial lesion showing DWI restriction (Figure 1C-D), which was felt to be consistent with osmotic demyelination syndrome (ODS).

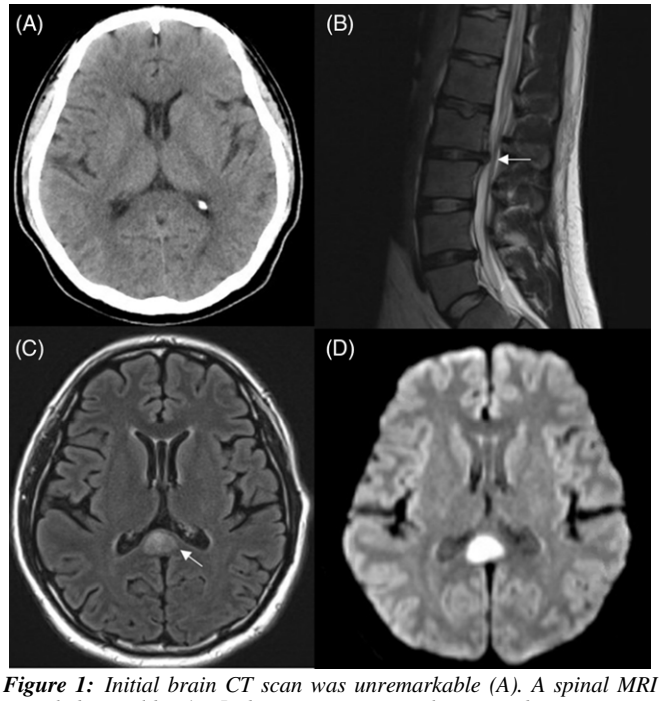


***Figure 1:*** *Initial brain CT scan was unremarkable (A). A spinal MRI revealed a mild L4–L5 disc protrusion, with no cord impingement (B). An MRI revealed a T2 hyperintense lesion in the splenium of the corpus callosum (C), which also showed diffusion restriction on DWI (D). No bleeding was observed in the lesion (not shown).*

ODS is a well-recognized complication of rapid correction of hyponatremia but associations with acute dialysis have also been demonstrated.[1,2] ODS has only rarely been found with dialysis in normonatremic contexts.[3] Typical lesions appear in the brain stem, but extrapontine myelinolysis is not infrequent, and may involve the centrum semiovale or the corpus callosum. The usual presentation is a brain stem syndrome with or without encephalopathy and four-extremity weakness. Urinary retention and bowel incontinence have also been reported but always in the context of pontine myelinolysis along with other classical features.[4] The splenium of the corpus callosum connects the posterior cortices, including parietal and temporal areas and primary and secondary visual areas from the occipital cortex.[5] While splenial lesions typically result in language, visuospatial, or cognitive deficits, urinary symptoms have also been described.[6]

Thrombotic thrombocytopenic purpura/hemolytic uremic syndrome was considered, as these may present with splenial lesions[7,8] but ADAMST13 levels were normal; and no purpura, prior infection, or diarrhea was observed. Systemic autoantibody panels and complement levels were negative. Furthermore, cerebral nervous system manifestations of TM are encephalopathic, and imaging reveals diffuse micro-hemorrhages,[9] while ODS consists of noninflammatory demyelination and selective white matter involvement.[1] Endothelial alteration may contribute to ODS[10] but further investigation is required.

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Clinical neurology is full of imitators and mimics, ranging from infectious to inflammatory neoplastic and metabolic. Here we present an atypical presentation of ODS, yet another condition with myriad presentations. Acute urinary/bowel symptoms and leg weakness were initially concerning for CES, and no findings initially localized to the brain. An MRI of the brain was performed only after excluding the alternative causes.

## Conflict of Interest

Dr. Camara-Lemarroy reports personal fees from EMD SERONO, outside the submitted work. The other authors have no conflicts of interest to declare.

## Statement of Authorship

KM, SW, RKC, ME, and TA gathered the clinical information, performed chart review, and revised the manuscript. KM, SW, RKC, and TA provided images and edited the content of the manuscript. CRCL wrote the manuscript and approved the final version.

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