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AN INTERESTING CASE OF NORMONATREMIC EXTRAPONTINE MYELINOLYSIS IN AN ALCOHOLIC

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ABSTRACT

The clinical presentation of Osmotic Demyelination Syndrome (ODS), bifurcated into Central Pontine Myelinolysis (CPM) and Extrapontine Myelinolysis (EPM), has long been tethered to the dogma of rapid hyponatremia correction. However, contemporary neurology increasingly recognizes a phenotype of the disorder that defies this established etiology, occurring in normonatremic patients particularly within the context of chronic alcohol use disorder.[1] This report presents the comprehensive case analysis of a 35-year-old male with a significant history of alcohol dependence, who presented with acute encephalopathy and a constellation of laboratory findings—severe bicytopenia, massive lactate dehydrogenase elevation, and hypocomplementemia—that strongly mimicked a catastrophic autoimmune event such as Evans Syndrome or Systemic Lupus Erythematosus. Through rigorous exclusion of autoimmune pathologies via specific serological profiling and the subsequent identification of characteristic lesions in the basal ganglia and subcortical white matter on magnetic resonance imaging, a diagnosis of Extrapontine Myelinolysis was established. Crucially, this occurred in the absence of documented hyponatremia, challenging the conventional osmotic gradient hypothesis. This paper provides an exhaustive examination of the betraying cues in alcoholic EPM, dissecting the pathophysiology of alcohol-induced blood-brain barrier dysfunction, the mechanisms of hematological and immunological mimicry in alcoholic liver disease, and the prognostic implications of EPM when detached from its classic precipitant. The favorable response to corticosteroid therapy in this case supports emerging theories regarding the predominance of vasogenic over cytotoxic edema in alcohol-associated myelinolysis, suggesting a wider therapeutic window than previously assumed.

INTRODUCTION

The entity of Central Pontine Myelinolysis (CPM) was first introduced as a disease characterized by a non-inflammatory demyelination centered within the basis pontis. For nearly two decades, the etiology remained obscure, attributed vaguely to nutritional deficiencies or direct toxins. It was not until the mid-1970s and early 1980s that the link between CPM and the rapid correction of hyponatremia was elucidated, fundamentally shifting the understanding of the disease toward an iatrogenic "osmotic" etiology. This paradigm posited that the rapid elevation of serum tonicity in a brain adapted to hypotonicity leads to intracellular dehydration, oligodendrocyte apoptosis, and myelin destruction. The concept was expanded to include Extrapontine Myelinolysis (EPM) when lesions were identified in the basal ganglia, thalamus, and subcortical white matter, often sparing the pons or occurring in conjunction with pontine lesions.[4] While the osmotic stress hypothesis remains the bedrock of ODS pathophysiology, a subset of cases continues to challenge the universality of the hyponatremic trigger. Reports of EPM occurring in patients with normal serum sodium levels—termed "normonatremic ODS"—have accumulated, particularly among chronic alcoholics and

liver transplant recipients as alcohol acts as a potent neurotoxin and metabolic disruptor.[4]

CASE REPORT

A 35-year-old male, was admitted to TVMCH following an acute deterioration in neurological status. The patient's social history was dominated by chronic alcohol dependence of 15 years' duration. His consumption pattern had historically been moderate, quantified at approximately 270 ml of brandy once weekly until April 2025. However, the three months preceding his admission marked a significant escalation in usage, driven by unspecified family issues. During this period, his intake increased to 180 ml consumed five days per week. This chronic escalation culminated in a binge phase in the five days immediately prior to the onset of symptoms, where consumption doubled to approximately 360 ml per day. Crucially, this binge was accompanied by a near-total cessation of solid food intake, a phenomenon common in late-stage alcoholism where caloric needs are met solely by ethanol ("alcohol anorexia"), setting the stage for profound nutritional depletion. The patient presented with a history of altered sensorium persisting for four days. Family members described a progressive confusion, unawareness of surroundings, and eventual unresponsiveness to verbal commands. By the time of admission, he was bedridden and in a state of metabolic encephalopathy. The initial differential diagnosis was broad, necessitated by the patient's obtunded state and history.

Considerations included

- **Wernicke's Encephalopathy:** Given the nutritional history and alcoholism.
- **Infectious Meningoencephalitis:** Considering potential exposure to leptospirosis or scrub typhus common in the region.
- **Autoimmune Encephalitis/Vasculitis:** Triggered by the initial laboratory findings of cytopenias.
- **Metabolic Encephalopathy:** Hepatic or uremic origin.

Laboratory investigations- The initial laboratory evaluation presented a picture that strongly diverted the diagnostic focus toward a systemic autoimmune or hematological malignancy. A Complete Blood Count (CBC) and peripheral smear analysis revealed significant bicytopenia. The smear showed anisocytosis, with a mix of macrocytes, macro-ovalocytes, schistocytes and tear drop cells. The presence of schistocytes raised immediate concern for microangiopathic hemolytic anemia, often seen in Thrombotic Thrombocytopenic Purpura (TTP) or Disseminated Intravascular Coagulation (DIC). The patient exhibited profound thrombocytopenia with a count of **29,000/µL**. Based on these further detailed evaluation was done and the diagnostic considerations are summarised in the following table.

Differential Diagnosis	Supportive Cues ("The Betrayal")	Exclusion Criteria (Why it was rejected)	Pathophysiological Reality in Case
Systemic Lupus Erythematosus (SLE)	Low C3 (52.5 mg/dL), Bicytopenia, Encephalopathy.	**Negative ANA Profile** (15 antigens negative). Normal/Low-Normal C4 (10.1 mg/dL) suggests lack of classical pathway consumption.	**Acquired C3 Deficiency:** Impaired hepatic synthesis due to alcoholic liver disease.
Evans Syndrome (AIHA + ITP)	Thrombocytopenia (29k), Anemia, High LDH (>13k), "Response to Steroids".	Absence of Coombs positivity (implied). Smear showed "giant platelets" (dysplasia) rather than purely destruction.	**Alcohol Marrow Toxicity:** Direct suppression of megakaryocytes + Hypersplenism. **High LDH:** Rhabdomyolysis + Shock Liver.
TTP / HUS	Schistocytes on smear, Low Platelets, Confusion.	Normal renal function (implied by context of other diagnoses). No mention of fever/severe renal failure triad.	**Zieve-like Syndrome:** Hemolysis due to altered RBC membrane lipids in liver disease + Alcohol toxicity.
Wernicke's Encephalopathy	Alcoholism, Malnutrition, Confusion.	MRI showed lesions in **Basal Ganglia**, not typical mammillary bodies/periaqueductal gray.	**Extrapontine Myelinolysis (EPM):** Distinct radiological entity, though likely co-occurring with thiamine deficiency.
Sepsis (Leptospirosis/Scrub)	Leukocytosis, Organ failure profile.	**Negative Serology** for Lepto/Scrub. **Blood Culture Negative**.	**Leukemoid Reaction:** Stress response to acute alcoholic hepatitis or tissue necrosis (high LDH).

BONE MARROW ANALYSIS

The patient's bone marrow analysis images are shown in figures 1 and 2

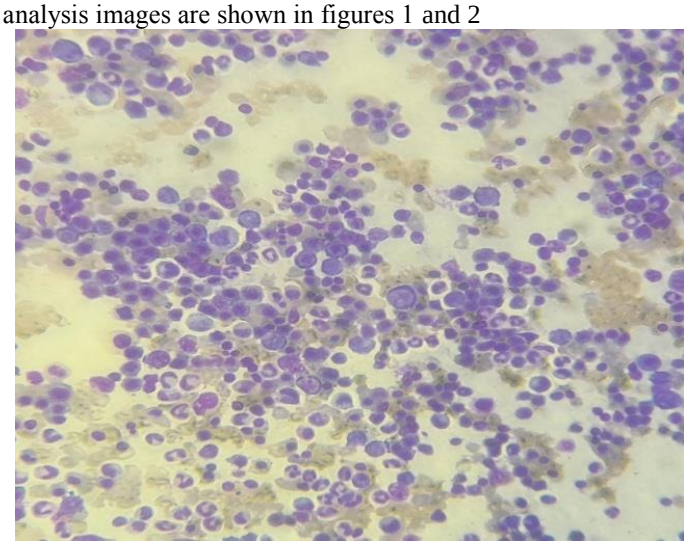

Figure 1.

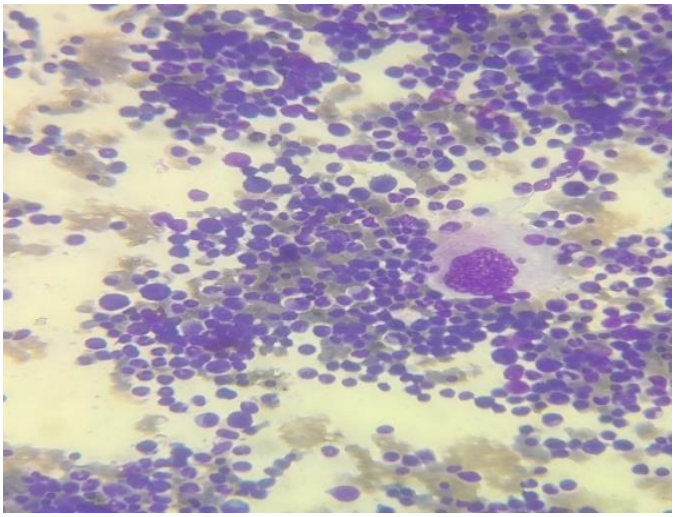

Figure 2.

ANALYSIS OF THE BONE MARROW FINDINGS

Finding in Report	Connection to Alcohol Toxicity
Thrombocytopenia (Reduced Platelets)	Alcohol has a direct toxic effect on megakaryocytes. This is often the first and most common sign of alcohol-induced marrow suppression.
Megaloblastic Maturation	Alcohol interferes with Folate metabolism leading to defective DNA synthesis. This causes cells to grow large but fail to divide.
Micronormoblastic Maturation	Alcohol damages mitochondria in red blood cells, preventing iron from being incorporated into hemoglobin. This causes **Acquired Sideroblastic Anemia**, leading to small (microcytic/micronormoblastic) cells.
Erythroid Hyperplasia	The marrow is hyperplastic to make red blood cells, but the process is "ineffective" (cells get lysed in the marrow), so the patient remains anemic.

Further evaluation was done with MRI of the brain to interrogate the encephalopathy directly. The imaging revealed characteristic signal alterations involving the **Basal Ganglia (BG) and Subcortical (SC) white matter**.[1] These findings—bilateral, symmetrical hyperintensities in the extrapontine gray and white matter—are the radiological hallmark of **Extrapontine Myelinolysis (EPM)**. The diagnosis was established as EPM in a chronic alcoholic, occurring in the absence of documented rapid correction of hyponatremia.[1,4]

MRI SHOWED - Symmetric intensities present in the bilateral globus pallidus, genu and posterior limb of the internal capsule.

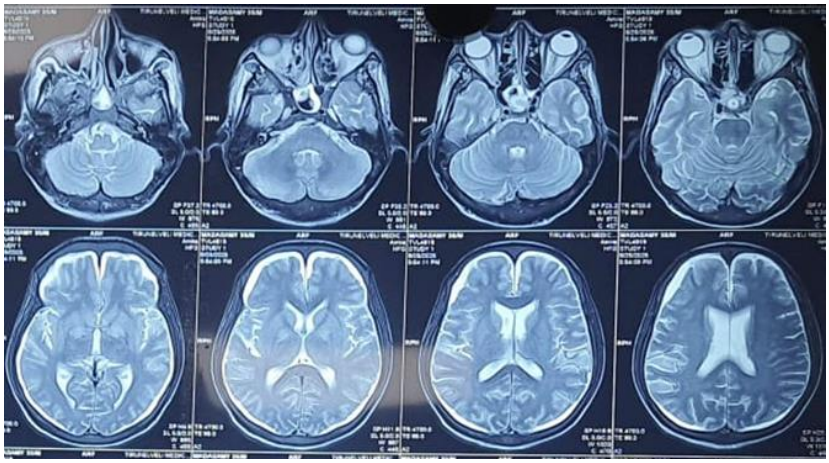

Figure 1.

DISCUSSION

In the complex alcoholic patient, the presentation of encephalopathy is rarely isolated. It is frequently accompanied by hematological derangements such as thrombocytopenia and anemia, as well as immunological abnormalities like hypocomplementemia. To the clinician, this triad (encephalopathy, cytopenia, low complement) forms a compelling pattern recognition for autoimmune vasculitis or connective tissue diseases like Systemic Lupus Erythematosus (SLE). This report underscores the danger of this pattern recognition when applied to the alcoholic patient, where marrow toxicity mimics autoimmune cytopenia and hepatic synthetic failure mimics immune complement consumption. This paper details the case to illustrate the rigorous clinical reasoning required to dismantle these decoys. By systematically analyzing the discordant lab values—such as the massive LDH elevation and the specific pattern of complement depression—we elucidate how the diagnosis of EPM was reached via exclusion and imaging. Furthermore, we have explore the distinct prognostic profile of alcohol-associated EPM, which is better than hyponatremic counterpart.[2] While managing the patient, though there are devastating outcomes of locked-in syndrome associated with pontine myelinolysis, the normonatremic alcoholic phenotype appears to carry a distinct, and potentially more favorable, prognostic profile as the critical distinction in alcoholic EPM is the predominance of vasogenic edema over cytotoxic edema.[2] This explains the response to steroids observed in the case. Based on these evidences the patient was managed with thiamine suppliments, steroids and adequate care was taken to avoid inadequate shifts in serum osmolality.

CONCLUSION

This case reinforces the existence of Normonatremic EPM as a distinct clinical entity driven by osmolyte depletion and blood-brain barrier dysfunction rather than iatrogenic sodium shifts. Most importantly, it offers a hopeful prognostic perspective: unlike the irreversible cellular death of classical ODS, the vasogenic nature of alcoholic EPM[4] renders it potentially responsive to corticosteroids. By recognizing the betrayal of the cues and understanding the nuance of alcohol pathophysiology, clinicians can identify this reversible condition before permanent neurological devastation occurs. This patient was discharged with nil residual neurological deficits at the time of discharge.

REFERENCES AND TABULAR COLUMN REPRESENTING THE INVESTIGATIONS

Parameter	17/08	18/8	21/8	22/8	25/8	2/9
TC	6400	5000	7000	6200	16800	12300
DC (N/L/E)	(58/32)	(84/32)	(74/4.1)	-	-	-
RBC	0.61	1.05	1.54	1.05	1.53	2.17
Hb	2.3	3.3	5.0	3.5	4.5	6.4
PCV	38	40.1	43.2	10.6	14.5	24
MCV	118	101	117	101	94.1	110
Platelet ($x10^3$)	24000	6000	16000	27000	34000	1.02 LAKH

Parameter	17/08	18/08	19/08	26/08	28/08	2/9
Total Bilirubin	6.5	6.2	4.5	2.09	2.5	1.1
Direct Bilirubin	3.4	3.7	2.7	1.2	1.4	0.5
Indirect Bilirubin	3.1	2.5	1.7	0.9	1.1	0.6
SGOT	323	191	159	27.1	81	28
SGPT	360	326	321	111	105	116
ALP	45	34	49	47	53	100

Albumin	3.5	3.7	3.7	3.0	2.0	3.3
Globulin	2.4	2.0	2.1	2.0	1.0	2.0
Total Protein	5.9	5.7	5.3	5.0	3.0	5.3

Parameter	**17/08**	**18/8**	**22/08**	**23/08**	**24/8**	**25/8**	**26/8**	**29/8**	**02/9**
Urea	168	145	247	109	43	34	35	39	40
Creatinine	1.22	1.1	1.5	1.4	1.2	1.1	1.0	0.9	1.1
Sodium	147	140	140	147	148	152	146	149	149
Potassium	4.5	3.7	4.4	3.5	4.3	3.6	3.4	4.1	3.9
RBS	168	234	247	246	106	102	110	126	102

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