

Osmotic Demyelination Syndrome

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Introduction

- ❑ Osmotic demyelination syndrome (ODS) is an acute demyelinating CNS disorder that results from rapid changes in serum osmolality
- ❑ Encompasses Central Pontine Myelinolysis (CPM) and Extrapontine Myelinolysis (EPM)

Historical Aspect

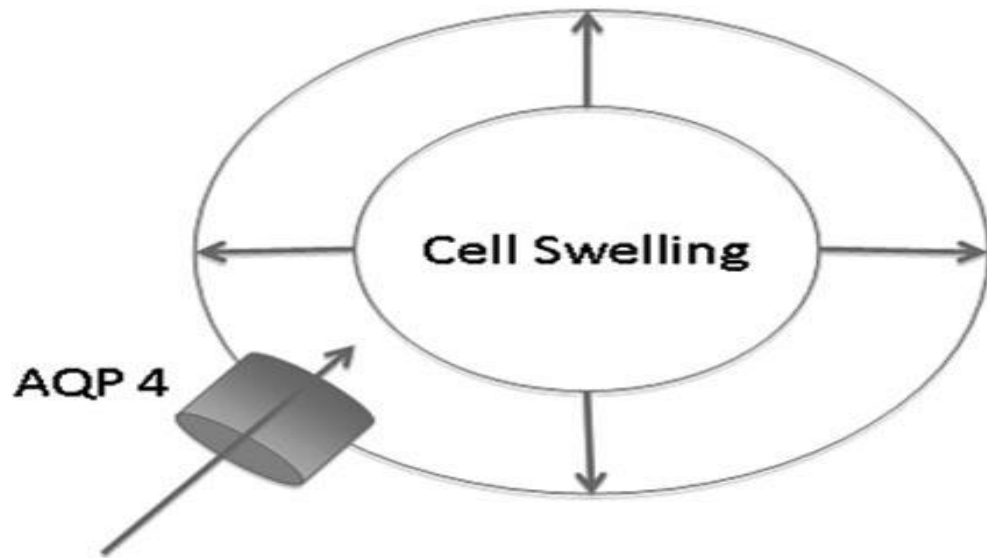
- Victor and Adams first described CPM in 1959
- From 1962 EPM was recognized
- Almost all patients had chronic medical conditions
- A link was established in the mid-1970s between the rapid correction of hyponatremia and CPM
- Rapid change in Osmolality without change in serum sodium has been reported to cause ODS

Epidemiology

- The largest autopsy series have found a prevalence of 0.25% to 0.5% in a general population
- Higher rates of ODS in alcoholics and liver transplant patients
- ODS has a peak incidence in adults aged 30 to 60 years and a male preponderance
- ODS has been reported at a rate of 0.4-0.56% for patients admitted to neurology services and 0.05% of all admitted in a general hospital

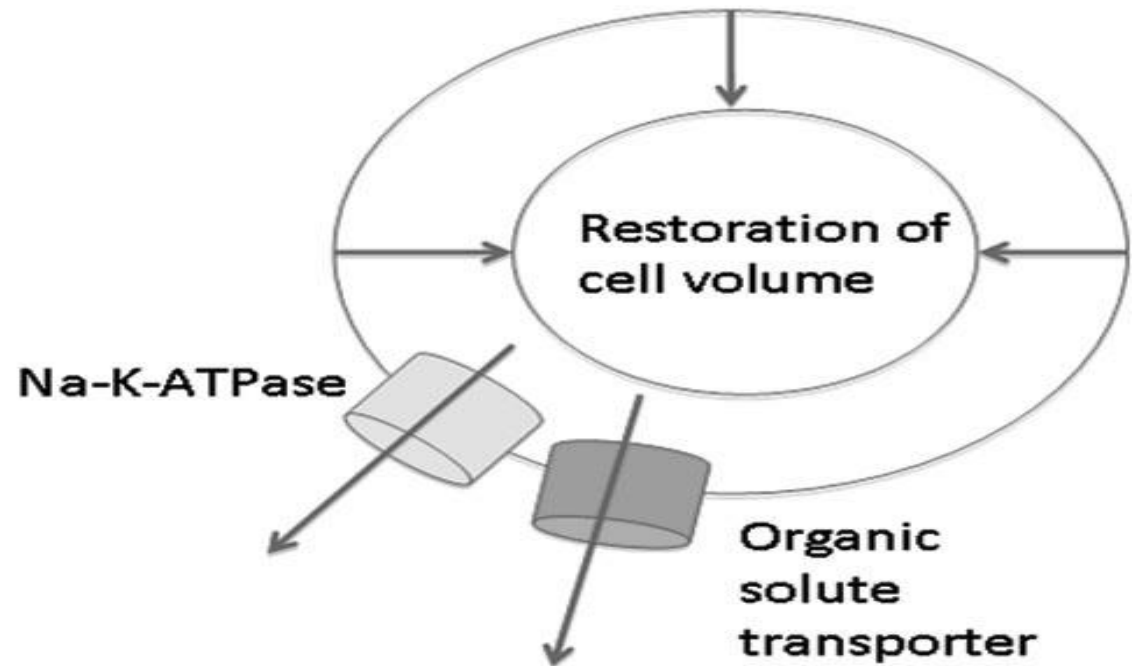
Pathophysiology

Acute Hyponatremia



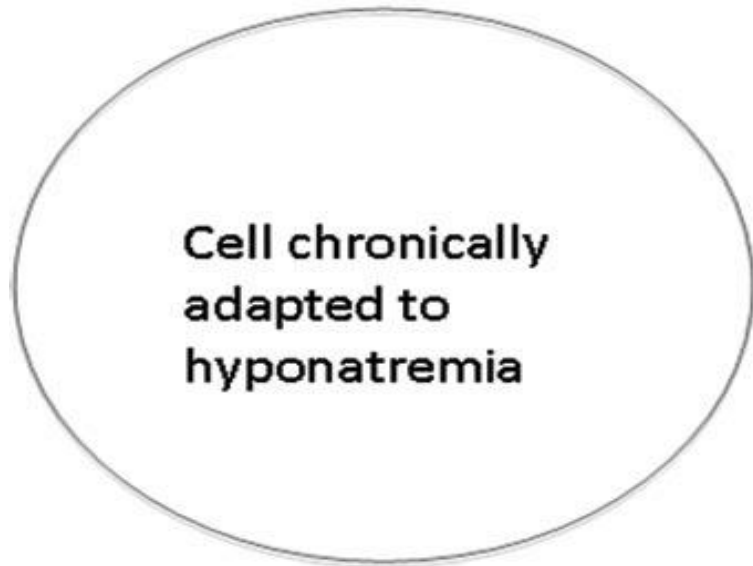
Water entry from area of low osmolality to intracellular area of higher osmolality

Chronic Hyponatremia



Removal of inorganic and organic solutes and water from cell

Cont...



Rapid correction of hyponatremia



1. Breakdown of blood-brain barrier
2. Exposure of glial cells to cytokines, complement
3. Axonal shear injury
4. Cellular ATP depletion
5. Induction of apoptosis



Osmotic Demyelination

Conditions Associated with ODS

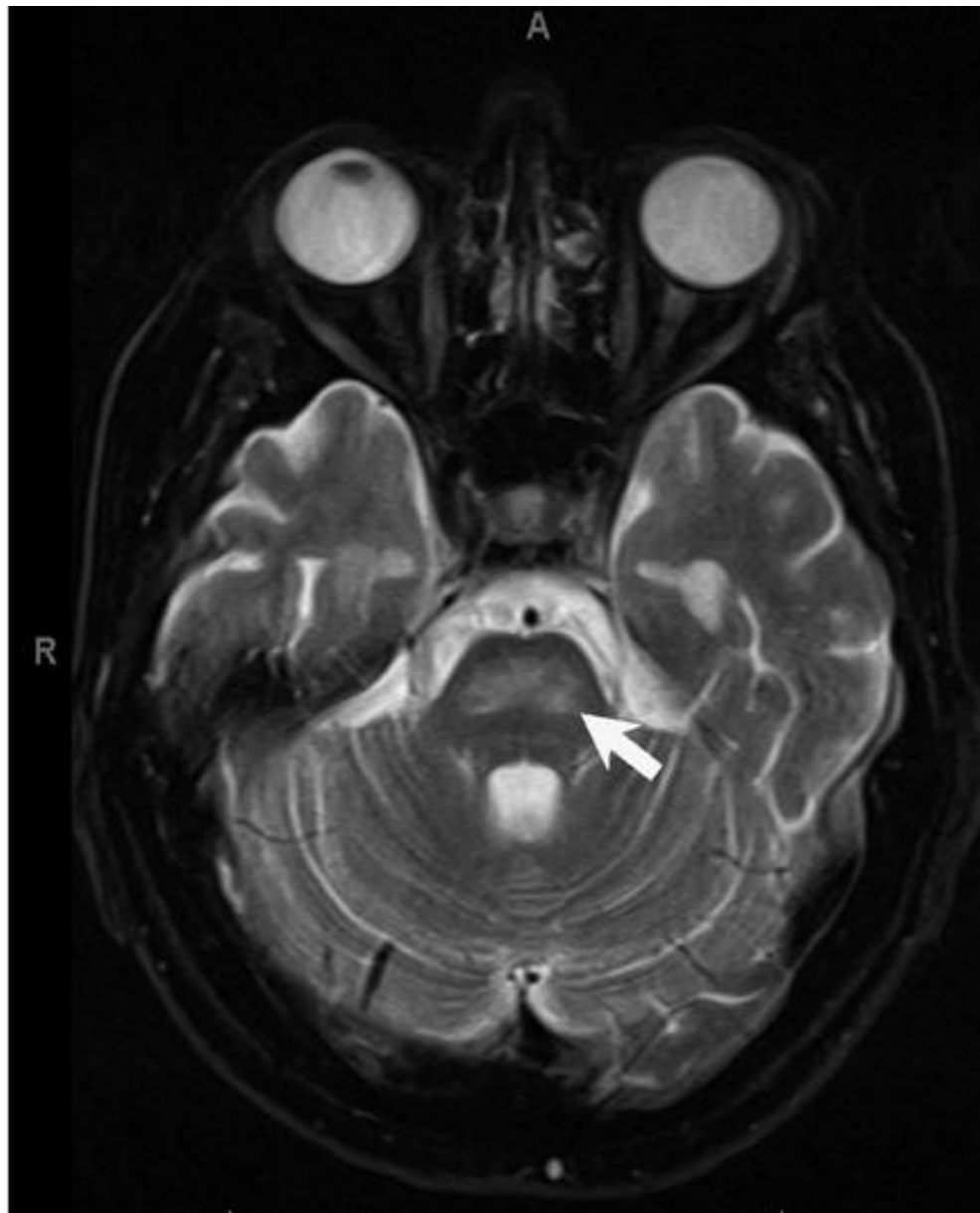
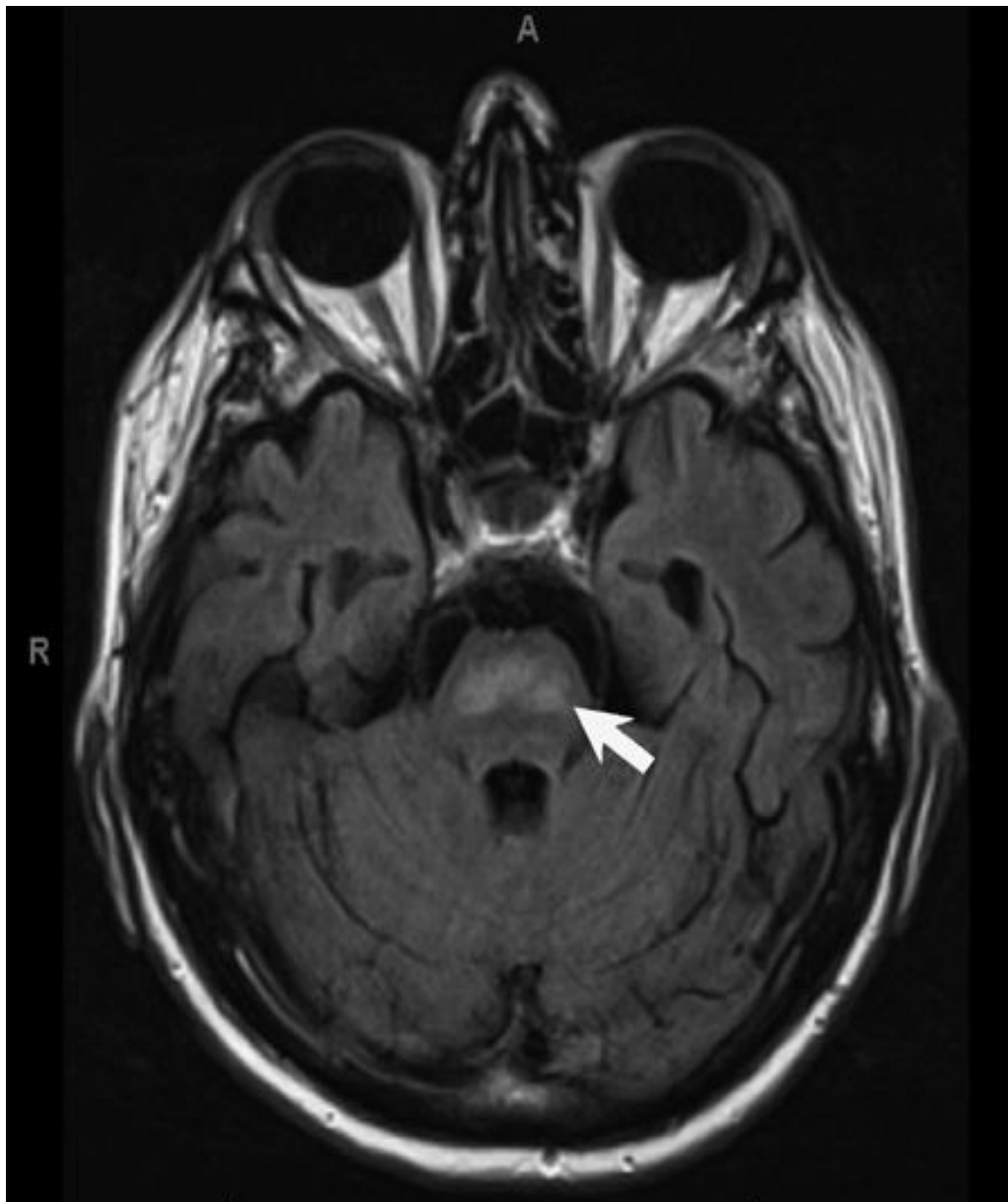
- Alcoholism
- Malnutrition
- After prolonged diuretic use
- Psychogenic polydipsia
- Burns
- Post-liver transplant
- Post-pituitary surgery
- Post-urological surgery/gynaecological surgery

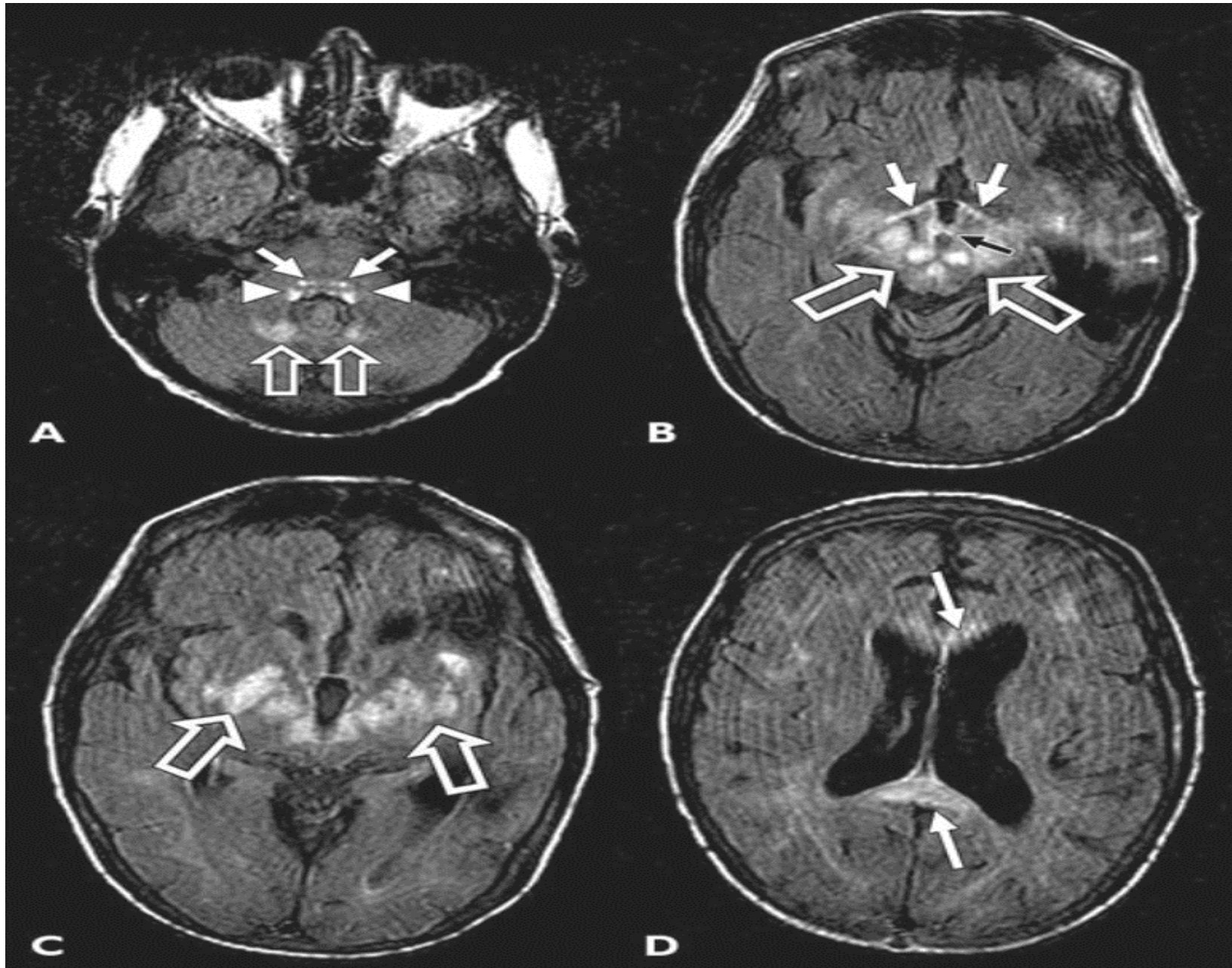
Clinical Manifestation

- Paresis
- Dysarthria
- Dysphagia
- Ataxia
- Mutism
- Agitated delirium
- Catatonia
- Parkinsonism
- Dystonia
- Encephalopathy
- Tremor
- “Locked-in” syndrome
- Coma
- Seizures
- Impairment in short-term memory
- Deficits in attention span

Diagnosis

- Diagnosis is principally made through correlation of clinical findings with radiologic studies
- MRI of brain is more sensitive than CT scan
- Hyperintense lesion on T2 weighted & FLAIR
- Hypointense lesion on T1 weighted
- DWI might have capability of detecting lesion undetectable on T2
- MRI changes may be delayed





Management

- ❑ Supportive treatment is only recommended with certainty
- ❑ Other potential options-
 - Reintroduction of hyponatremia
 - Corticosteroids
 - Administration of myoinositol
 - Plasmapheresis
 - Intravenous immunoglobulin

Prevention

- Treatment of acute symptomatic hyponatremia should include hypertonic 3% saline to acutely increase plasma Na⁺ conc. by 1–2 mmol/h to a total of 4–6 mmol
- Plasma Na⁺ concentration should be monitored every 2–4 h during treatment
- The rate of correction in chronic hyponatremia is slow (<8–10 mmol in the first 24 h and <18 mmol in the first 48 h)

Prognosis

The outcome may be death, disability, or recovery to a virtually normal level of function

Key points

- ODS to be considered in a patient who-
 - has failed to recover as expected after a severe illness requiring I/V fluids
 - manifesting “psychiatric” symptoms after such an illness, even if imaging is negative
- There may be no “safe” limit for the rate of Na^+ rise but should not exceed the recommended limit in chronic condition

Cont...

- MRI changes may be delayed
- MRI severity is not prognostic
- Prognosis is not uniformly poor
- There is no established recommendations for treatment

Conclusion

- ✓ Till now, there is no specific treatment of established ODS, so prevention is the main aspect
- ✓ An appropriate rate of hyponatremia correction, careful change in osmolality and treatment of comorbid conditions are essential to reduce the risk of ODS
- ✓ Suspected and diagnosed cases should be reported & discussed with different medical professionals
- ✓ Research should be continued to find out the safest way to prevent & effectively treat ODS

Thank You