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## **Interesting Case of Headache with “Can’t Mix Food & Can’t Swallow”**

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### CLINICAL HISTORY

A 63-year-old male patient, hailing from Mandya district, a farmer by occupation who is a known case of interstitial nephritis, improved AKI and seizure disorder on Tab EPTOIN 100mg 1-0-2 presented with headache since 10 days, on the right side, moderate to severe in grade. 5 days following the headache, he developed slurring of speech and difficulty in consuming food. He faced difficulty in consuming food as he could not mix his food with the tongue. 2 days later, he developed hoarseness in his voice.

He had no history of fever, nausea, vomiting, or blurring of vision.

He had no history of loss of smell sensation, no visual disturbance, no loss of sensation on the face, no facial asymmetry, no hearing difficulty or giddiness, no difficulty in neck movements.

He had no history of weakness in any limbs, no muscle incoordination, no loss of consciousness, or alteration in behavior.

### EXAMINATION AND INVESTIGATIONS

An old aged man, moderately built and nourished, well oriented to time, place and person is conscious, cooperative, and alert.

Vitals:

PR- 80 bpm

BP- 130/70mm Hg

RR- 18cpm

Afebrile

No pallor/icterus/cyanosis/clubbing / pedal edema/ lymphadenopathy.

*Cranial Nervous System:*

Higher Mental Functions: conscious and oriented

Normal appearance, well dressed.

Cranial nerves:

- Glossopharyngeal and Vagus nerve: hoarseness of voice +, uvula deviated to left, gag reflex absent on right side, taste sensations lost on posterior 2/3<sup>rd</sup> of the tongue.
- Hypoglossal nerve: tongue deviated to the right.
- Other cranial nerves are intact.

Motor system: normal bulk, tone, power 5/5 in all 4 limbs;

Deep tendon reflexes 2+ in all 4 limbs

The sensory system: B/L sensations intact on all dermatomes

Autonomic system – normal

Cerebellar signs absent

Meningeal signs absent



**Deviation of uvula to left**

Hb- 7.8mg/dl

Total Leucocyte Count – 6800 Cells/cumm

Serum Urea – 36mg/dl

Serum Creatinine – 2.32 mg/dl

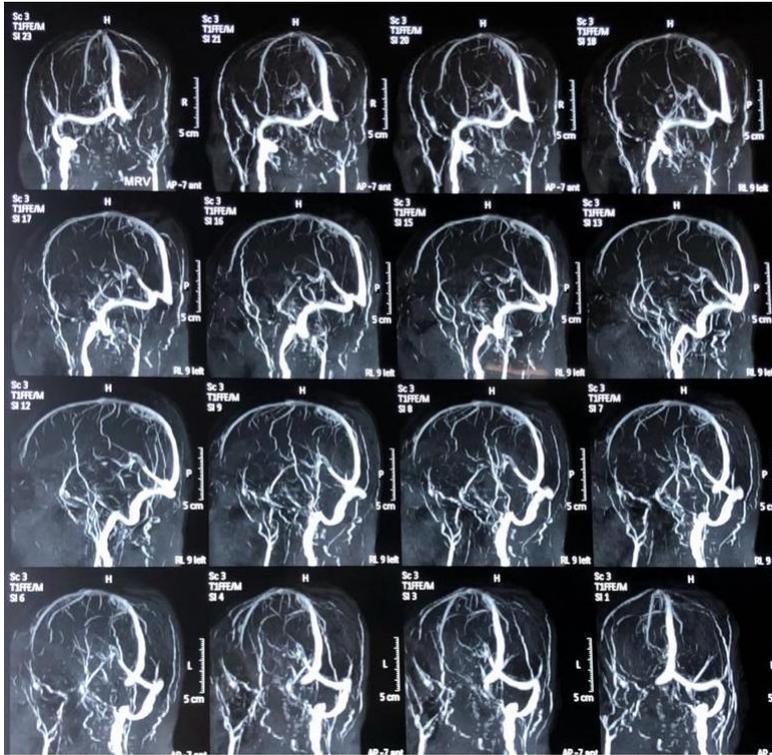
Random Blood Sugar- 118mg/dl

Serum electrolytes-:

Sodium -135meq/L; Potassium- 4.4meq/L; Chloride-95 meq/L

USG ABD & PELVIS: Bilateral small kidneys with mild renal disease.

MULTIPLANNER MR Venography OF BRAIN – Cerebral venous thrombosis of Right Transverse sinus, Sigmoid sinus, and Internal Jugular Vein



MRV showing thrombosis of

right transverse sinus, sigmoid sinus, IJV

### DIAGNOSIS

Bulbar palsy (involving IX, X, XII cranial nerve) secondary to Cerebral Venous Thrombosis of Right Transverse, Sigmoid sinus, and Internal Jugular Vein.

### DISCUSSION

The differential diagnosis for Lower Cranial Nerve palsy: [3]

They can be classified as:

1. Nuclear: parenchymal lesions, stroke, hemorrhage, trauma, demyelination
2. Intranuclear: fascicular, cisternal, canalicular, extra canalicular

Causes concerning site:

-within brainstem: infarction, syringobulbia, motor neuron disease

– around skull: nasopharyngeal carcinoma, glomus tumor, neurofibroma, jugular venous thrombosis, meningitis

– neck and nasopharynx: nasopharyngeal carcinoma, metastases, trauma, carotid artery dissection.

BULBAR PALSY is the lower motor neuron weakness of muscles whose cranial nerve nuclei lie in the medulla (bulb). [3]

Cranial nerve palsies are related only in 12% of cases with CVT, most commonly involving CN II, IV, and VI. Bulbar palsies related to CVT are even lesser in occurrence. [2]

CVT is a rare condition affecting the 0.6-7/100,000 population. It is defined as superficial or deep venous thrombosis in the brain. [1]

Thrombosis of the cerebral veins is much less common than arterial thrombosis. Superior sagittal sinus is most commonly involved. Thrombus may extend to transverse and sigmoid sinus. Deep venous system thrombosis (internal cerebral veins, the vein of Galen, or straight sinus) is much less common.

Causes: Local causes like trauma or infection to the sinuses, otitis media, mastoiditis, sinusitis, invasion, or compression of sinuses by neoplastic processes; Systemic causes like hypercoagulable states, dehydration.

Clinical features:

– features due to increased ICP like headache, nausea, vomiting, seizures, papilledema

– focal/motor deficits

– if spread to jugular foramen, it can involve cranial nerves 9,10,11,12 causing palsy.

Investigations:

1. Hematology: complete hemogram, CRP, coagulation studies
2. CSF examination to rule out meningitis/ SAH
3. Imaging: CT or MRI to rule out parenchymal lesions, CT or MR Venography: filling defect in the affected vessel

Treatment: Anticoagulants are the mainstay treatment. The patient can be started on heparin, followed by warfarin. Mechanical thrombectomy/ intradural thrombolysis in resistant cases.

Supportive measures like antibiotics, anticonvulsants, anti-edema measures, and analgesics are added.

ACKNOWLEDGEMENTS: None

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