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White Cerebellum Sign- A Case Report

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Introduction

White cerebellum sign is an uncommonly described radiological sign. This sign is mostly described in pediatric age group due to child abuse, head injury& hypoxia[1]. It is uncommon in adults but can also be seen following head injury & in postpartum period[2]. Here we describe an adult case with history of chronic alcohol consumption.

Case Report

A 40 year old Hindu male, chronic alcoholic admitted with the complaints of 1 day fever, altered sensorium, repeated attacks of generalised tonic clonic seizure, having no past history of head injury, similar illness, without any significant family history.

General physical examination revealed tachycardia, fever, stuporous condition, low SpO2 (88%).Central nervous system examination revealed GCS 4/15(E2VIM2), pupil bilateral equal size normal reaction to light, with intact corneal relex and brisk deep tendon reflexes. Bilateral non responsive plantar, meningeal signs were absent, skull & spine normal.

Cerebellar functions could not be examined. Other Systemic examination revealed no abnormality. With this, 3 differential diagnosis were made clinically:

1. Cerebral malaria
2. Alcoholic intoxication
3. Wernickes encephalopathy

Patient was treated with antimalarials, antibiotics, antiepileptics, injection dexamethasone, injection thiamine & oxygen inhalation. Lab invetigations showed neutrophilic leukocytosis, microcytic hypochromic anaemia, absence of malaria parasite, mild elevation of liver enzymes.

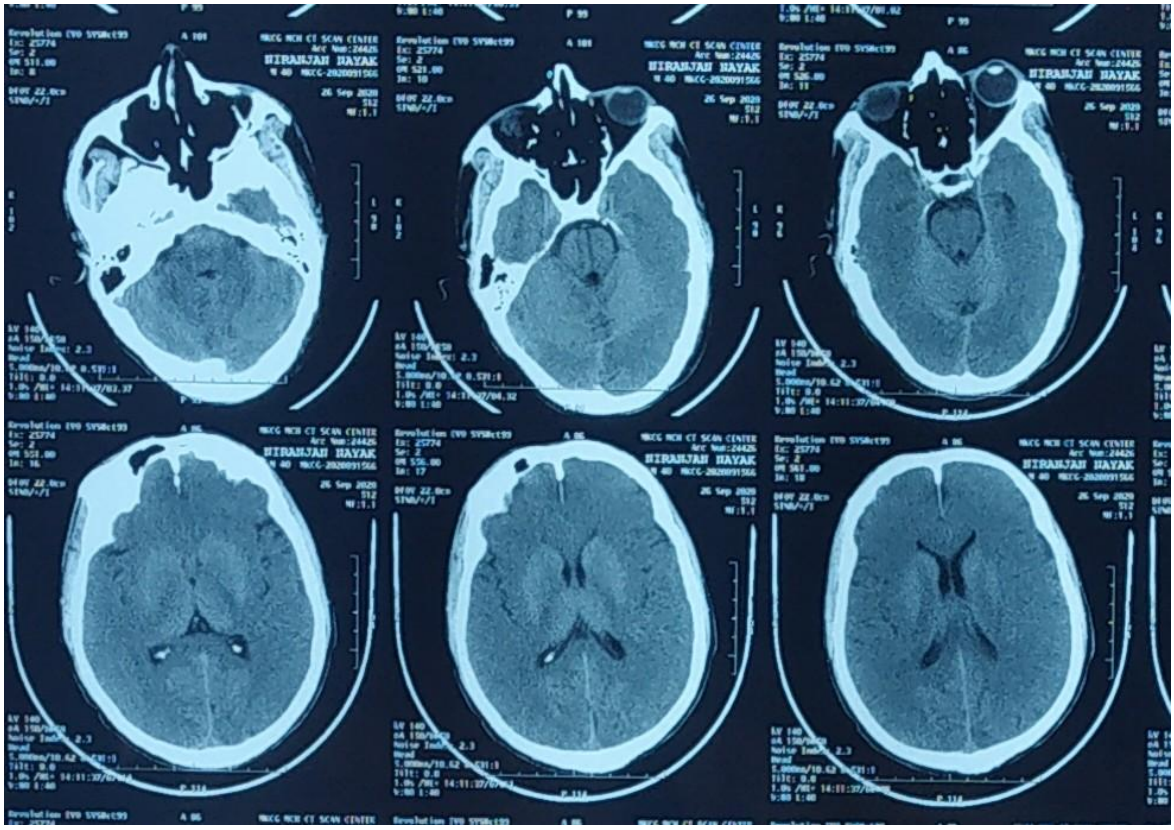
NCCT of brain showed diffuse hypodensity of bilateral cerebral hemispheres with sparing of basal ganglia, thalamus and cerebellum.(WHITE CEREBELLUM SIGN)-irreversible brain pathology.

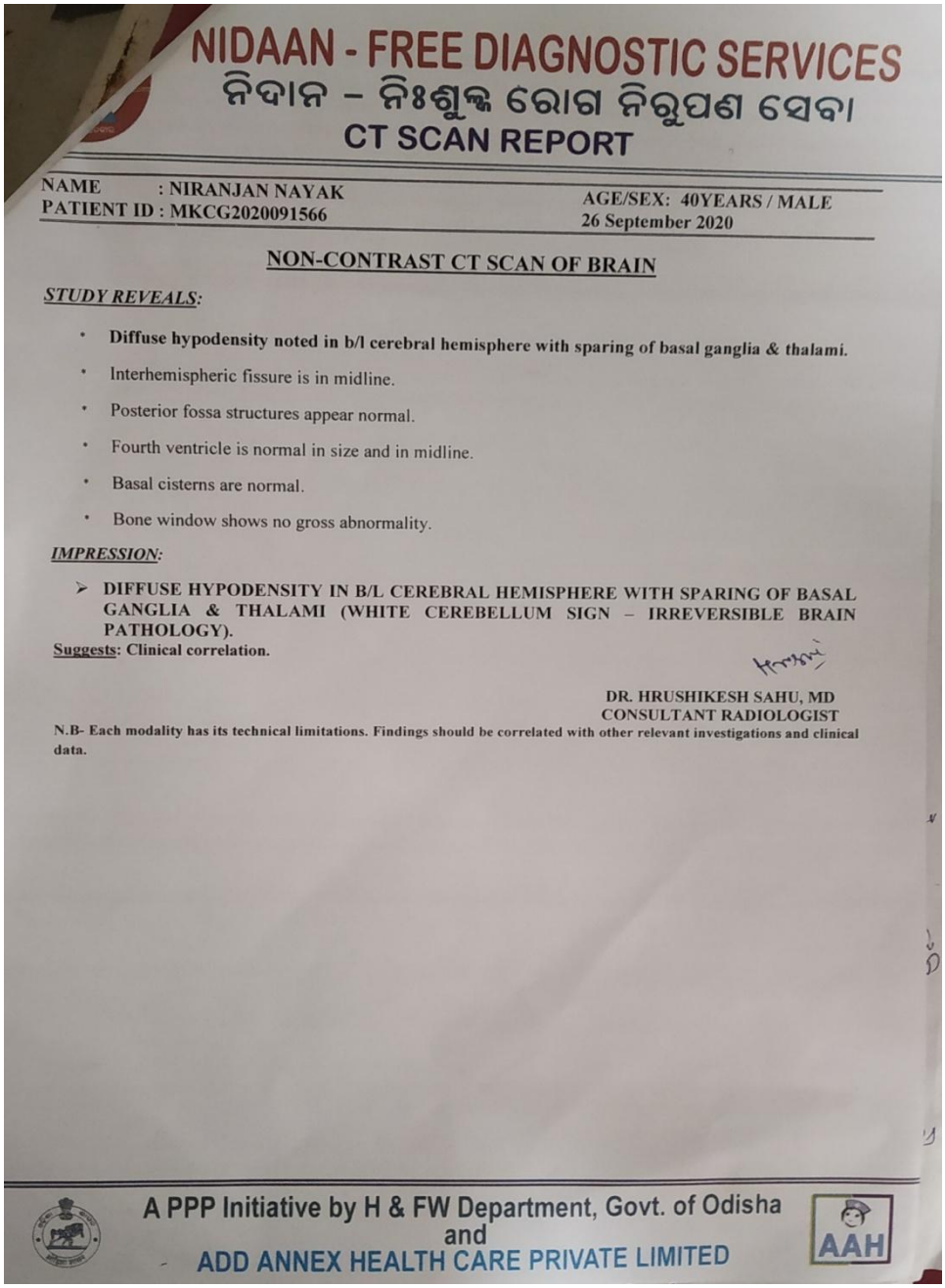
MRI report showed hypointense cortical & subcortical areas of both cerebral hemisphere on T1 & hyperintense on T2 & flair image. Basal gangia & bilateral cerebellar hemispheres were normal.

After imaging study, differential diagnosis of

1. Cerebral edema due to encephalitis
2. Wernickes encephalopathy (Alcoholic)
3. Hypoxic ischemic encephalopathy are made

After 10 days, GCS improved to E4V1M4 (9/15) & discharged on request with advice of Ryle's tube feeding, oral antibiotics, antiepileptics and tab thiamine.





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CT SCAN REPORT

NAME : NIRANJAN NAYAK
PATIENT ID : MKCG2020091566
AGE/SEX: 40YEARS / MALE
26 September 2020

NON-CONTRAST CT SCAN OF BRAIN

STUDY REVEALS:

- **Diffuse hypodensity noted in b/l cerebral hemisphere with sparing of basal ganglia & thalami.**
- Interhemispheric fissure is in midline.
- Posterior fossa structures appear normal.
- Fourth ventricle is normal in size and in midline.
- Basal cisterns are normal.
- Bone window shows no gross abnormality.

IMPRESSION:

- **DIFFUSE HYPODENSITY IN B/L CEREBRAL HEMISPHERE WITH SPARING OF BASAL GANGLIA & THALAMI (WHITE CEREBELLUM SIGN – IRREVERSIBLE BRAIN PATHOLOGY).**

Suggests: Clinical correlation.

DR. HRUSHIKESH SAHU, MD
CONSULTANT RADIOLOGIST

N.B- Each modality has its technical limitations. Findings should be correlated with other relevant investigations and clinical data.

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Discussion

White cerebellum sign in CT & MRI denotes a relative hypodensity of both cerebral hemisphere & hyperdensity of cerebellum, basal ganglia & thalamus. It has been documented in severe head trauma, birth hypoxia, child abuse, drowning, status epilepticus, bilateral meningitis, encephalitis, severe cardiac hypoxia. This sign suggest an Irreversible brain injury, mostly extra cerebral cause of hypoxia, like smothering seen in child abuse, it can be the only evidence of strangulation ,non accidental head injury. One third of these patients die while remaining population suffers Irreversible brain damage with subsequent vegetative state

Pathogenesis of white cerebellum sign is not fully understood. There are several theories:

i) Hypoxia decreases ATP production, thereby disturbing sodium pump leading to cytotoxic edema followed by increased intra cellular and extra cellular water accumulation (vasogenic edema).

ii) It could be due to raised ICP with partial venous obstruction leading to deep medullary vein distention.

iii) Preferential preservation of blood flow in the posterior circulation in comparison to anterior circulation.

iv) Partial relief of raised ICP due to trans-tentorial herniation leading to increased perfusion of central structures.

v) Hypoxia followed by ischemia causes hyperglycemic damage to cerebral cortex.

It could be due to raised ICP with partial venous obstruction leading to deep medullary vein distension or due to preferential preservation of blood flow in posterior circulation in comparision to anterior circulation.

Conclusion

This poor prognostic sign is mostly seen in children but this sign has been rarely reported in adult which may be due to diffuse cerebral edema, severe anoxia, in alcoholics, wernicke's encephalopathy. This patient recovered slowly with treatment with thiamine and other supportive treatment suggesting possibilities of wernicke's encephalopathy. There is few other case reports which supports that white cerebellum sign may not always be very ominously. Recognition of this sign is important for management especially for counselling of the patients and relatives.

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