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RESEARCH ARTICLE

UNUSUAL PRESENTATION- MY BRAIN IS WET WITHOUT ALCOHOL: A CASE REPORT OF NON ALCOHOLIC WERNICKE'S ENCEPHALOPATHY

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Abstract

Case of 72 years male with Nonalcoholic Wernicke's encephalopathy (NWE) who was initially investigated managed as Stroke, Metabolic encephalopathy or Suspected Meningoencephalitis. Patient managed clinically, until MRI report and with clinical correlation patient was diagnosed with NWE. Thiamine deficiency is associated with chronic alcoholics which was not among the common differentials in this case and its emphasis in ED among the differentials to be ruled out. A male who is Non-alcoholic was brought to ED having Slurred Speech, confusion, weak memory and evaluated, investigated with multiple tests which turned out to be normal Until MRI showed a well defined area of hyper intensity on DWI in peri-ductal region possibility of WE. We recommend using criteria to diagnose and treat NWE as early as possible to avoid misdiagnosis and treatment delays. We should focus on patients with altered mental symptoms even in coma, administering parenteral thiamine.

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Introduction:-

Wernicke's encephalopathy is caused by thiamine deficiency and is most commonly associated with heavy alcohol intake paired with poor nutrition. Wernicke encephalopathy is an acute neurological condition characterized by a clinical triad of ophthalmoplegia, ataxia, and confusion. This disease is caused by thiamine deficiency, which primarily affects the peripheral and central nervous systems. This should be differentiated from Korsakoff syndrome, a neuropsychiatric disorder associated with confabulation and significant deficits in anterograde and retrograde memory. This activity reviews the pathophysiology and evaluation of Wernicke encephalopathy and highlights the role of the inter professional team in the management of patients with this illness.

Thiamine is important for carbohydrate metabolism because it is a crucial cofactor for α -ketoglutarate dehydrogenase, pyruvate dehydrogenase, and transketolase. Therefore, areas of the brain with high energy metabolism may be damaged because of excess accumulation of toxic intermediates, which is caused by thiamine deficiency

Wernicke's encephalopathy (Sarayu & Anil, 2021) is an acute neurological condition characterized by clinical triad of Confusion, Horizontal Nystagmus, ophthalmoplegia (ptosis of eyelids, atrophy of optic nerve) and Truncal ataxia. This is usually caused by thiamine deficiency, which primarily affects central and peripheral nervous system. The people at high risk of Wernicke's encephalopathy are chronic alcoholics¹. Chronic alcohol consumption impairs

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absorption of thiamine from intestine, thereby leading to thiamine deficiency (Natalie, Kimberley, & Clive). But there are several other causes that may lead to Wernicke's encephalopathy, such as malnutrition, hyperemesis, prolonged parenteral nutrition, malignancies, immunodeficiency syndromes, liver disease, hyperthyroidism and severe anorexia nervosa.

While working in ER, an emergency physician faces many cases of confusion and nystagmus. Wernicke's encephalopathy being more of a clinical diagnosis, it is necessary for an Emergency Physician to maintain a high level of suspicion while examining similar patients.

As discussed earlier, not every patient presenting with signs of Wernicke's encephalopathy may be a chronic alcoholic. Hence, an in-depth history taking may also help in reaching to the diagnosis earlier and may save the patient.

Case Study

A 72 years old man who had never consumed alcohol was brought into ED with complaints of slurring of speech, fever, and generalised body weakness for 3-4 days. Patient had no known comorbidities and he had surgical history of Right Tubeless PCNL with DJ stenting on 26/03/2022. No drug abuse or tobacco use was reported throughout his life. The patient has received primary treatment from another tertiary hospital before referring admitting hospital.

On Clinical examination patient was drowsy, dyspnoeic, with Oxygen saturation of 91% on 2 litres, BP 144/79, with the respiratory rate of 34 breaths per minute, baseline glucose level was 178mg/dl. He had crepitations bilaterally and there was nuchal rigidity present at the time of examination, sensory examination was normal, Motor examination was power 5/5 with slow mentation and slow limb movements. His eye movements were normal, deep tendon reflexes were intact. Ataxia could not be assessed reliably due to impaired level of mental status.

The patient managed as Stroke with differentials of Metabolic encephalopathy; Meningoencephalitis; Seizures; Nutritional deficiency.

The patient was investigated in form of the investigations listed on the next page and its relevancy in a tabulated form. The patient was managed in a multidisciplinary approach with Neurology critical care advice sought on.

Patient was investigated and the results were as follows:

Day 1: ABG, CBC, RFT, LFT, MRI BRAIN, CSF.

The patient on Day 1 still had symptoms on arrival and was admitted in ED until the following investigation reports were achieved.

ABG:	Normal findings	In view of metabolic causes
CBC:	ALL Parameters were in the normal range.	To assess the infective cause
LFT:	2 fold elevated AST, ALT	To assess for metabolic encephalopathy
RFT:	ALL parameters in normal range	To assess for metabolic encephalopathy
S. Calcium	Parameter in Normal Range	Rule out Hypocalcemia.
Urine & Blood Culture	Urine: trace albumin	Infective causes
MRI Brain:	Well defined area of hyper intensity on DWI in peri ductal region , possibility of Wernicke Encephalopathy.	Diagnose Brain pathology
ECG:	Normal sinus rhythm with sinus tachycardia	Rule out Arrhythmias , syncope.

CSF	Glucose was reduced(82 in CSF whereas 178 in blood glucose) RBCs 50, Lymphocytes 100	Rule out Meningoencephalitis.
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After the Only Positive Findings of MRI:

Well defined area of hyper intensity on DWI in peri ductal region, possibility of Wernicke Encephalopathy.

The patient was managed as Non alcoholic Wernicke's encephalopathy was put on fluids and thiamine.

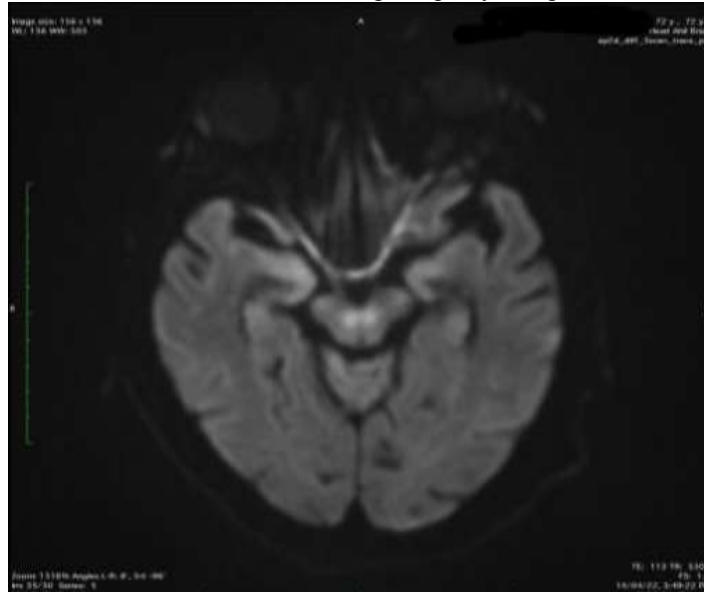


Figure 1:- MRI Image Showing the DWI image with hyper intensity in peri ductal region.

Day 2: Patient became oriented and he's conscious level improved markedly.

Day 3: Patient able to carry out his daily activities.

Discussion:-

We came across several case reports wherein the diagnosis of Wernicke's encephalopathy was either missed or diagnosed late due to absence of alcohol in patient's history.

A case reported by Stamatis Karakonstantis and others (Natalie, Kimberley, & Clive, 2011) showed 23 year old female who was on liquid only diet and had lost about 30kgs of weight, presented with clinical features of Wernicke's Encephalopathy. Even brain MRI showed typical WE findings. The patient improved after intravenous thiamine supplementation. This diagnosis was delayed to delay in detailed history taking.

In another case report by Won Jae Kim and Myung Mi Kim a 49 year old female patient who suffered from ischemic colitis and was on prolonged parenteral nutrition came to ED with sign of bilateral complete horizontal downward gaze palsy and mental confusion. (Liyang, Hatim, Joseph, Gregory P, & Kimberely) Even the MRI showed signs specific to Wernicke's encephalopathy.

A systemic review by Erik Oudman and others (Erik, Jan, Misha, & Mirjam) showed that all patients who had rapid loss in weight, or were suffering from eating disorder such as anorexia nervosa were vulnerable to Wernicke's encephalopathy. Hence, prophylactic thiamine checks were necessary in such patients.

Liyang Tang and others (Erik, Jan, Misha, & Mirjam) did a study wherein 25.7% of the patients who were operated for vertical sleeve gastrectomy had thiamine deficiency.

Conclusion:-

Non alcoholic Wernicke's encephalopathy should be a differential or thought about atleast in all the altered mental status or confused/Drowsy State.

In summary, we recommend using operational criteria to diagnose and treat nonalcoholic WE as early as possible to avoid misdiagnosis and treatment delays. Nonalcoholic WE remain a clinical diagnosis, and certain examinations are helpful for this diagnosis, such as measuring serum thiamine concentrations. We should focus on patients who present with abnormal mental symptoms, even those in a coma, and administer parenteral thiamine before any carbohydrate to reduce the high frequency of residual morbidity.

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