
بسم الله الرحمن الرحيم

DIAGNOSIS OF
HEPATIC ENCEPHALOPATHY

BY

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DIAGNOSIS OF CHRONIC HEPATIC ENCEPHALOPATHY

- When patients, with and without known liver disease, present with neuropsychiatric symptoms or neurological signs, it is necessary to ask one of the following questions:

  (1) Does this patient have HE? or

  (2) Could this patient have HE?

  There are two components to making a diagnosis of HE:

  - one is to determine that minimal or overt encephalopathy is present, and

  - the other is to obtain information consistent with hepatocellular insufficiency and increased portal-systemic shunting.

LABORATORY TESTS:

- A patient with significant liver injury may have normal results.
- Elevated serum aminotransferases (AST)
- Hyperbilirubinemia, hypoalbuminemia, and hyperglobulinemia
- Elevated alkaline phosphatase
- Prolonged prothrombin time
- Hypomagnesemia, hypophosphatemia, and hypokalemia

LABORATORY TESTS:

- Primary respiratory alkalosis, due to centrally-mediated hyperventillation is the most common acid-base disturbance in patients with severe liver disease, especially with superimposed encephalopathy. The exact etiology is unclear but may be related to the hormonal imbalance associated with liver failure. Estrogen and progesterone have been implicated, a situation somewhat similar to that seen in pregnancy.

LABORATORY TESTS:

- Anemia (from folic acid and vitamin B12 deficiency), gastrointestinal blood loss, or toxic effects of alcohol on bone marrow.
- Plasma ammonia levels are not consistently raised in patients with HE; they correlate poorly with the stage of HE and they do not provide a reliable index of the efficacy of treatments for HE.

- Lumbar puncture is not done unless indicated by atypical clinical or laboratory findings. It carries increased risk because of the presence of coagulopathy and, if ICP is increased in FHF, the possibility of precipitating cerebral herniation.

PSYCHOMETRIC TESTS:

- Simple psychometric tests include:
- orientation to time, person, and place,
- recall of current events,
- subtraction of serial sevens,
- handwriting, and
- figure drawing.

The inability to draw a five-pointed star (constructional or ideational dyspraxia) has received special attention.

EUROPHYSIOLOGICAL TESTS:

e EEG abnormalities that occur in
E are non-specific, being found in
her metabolic encephalopathies.
e main EEG abnormalities in HE
e a progressive bilaterally
nchronous decrease in wave
quency and an increase in wave
mplitude. Preterminally there is a

common with other cases of
etabolic encephalopathies,
roxysmal triphasic waves may
cur, even in the early stages of
E, and are characteristically
sociated with a frontal to
cipital phase shift.

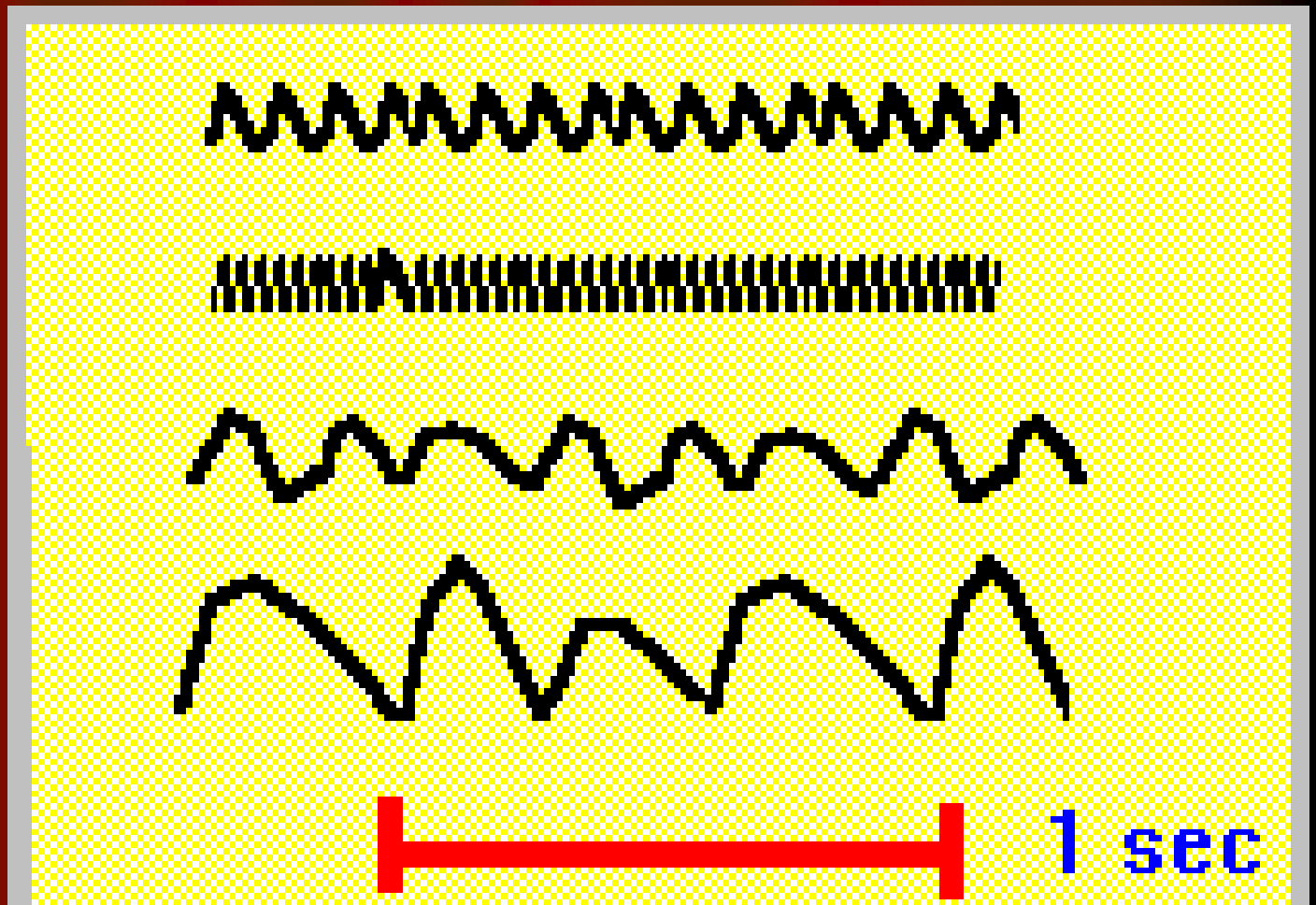
EG WAVES AS DEFINED BY FREQUENCY:

PHA

TA

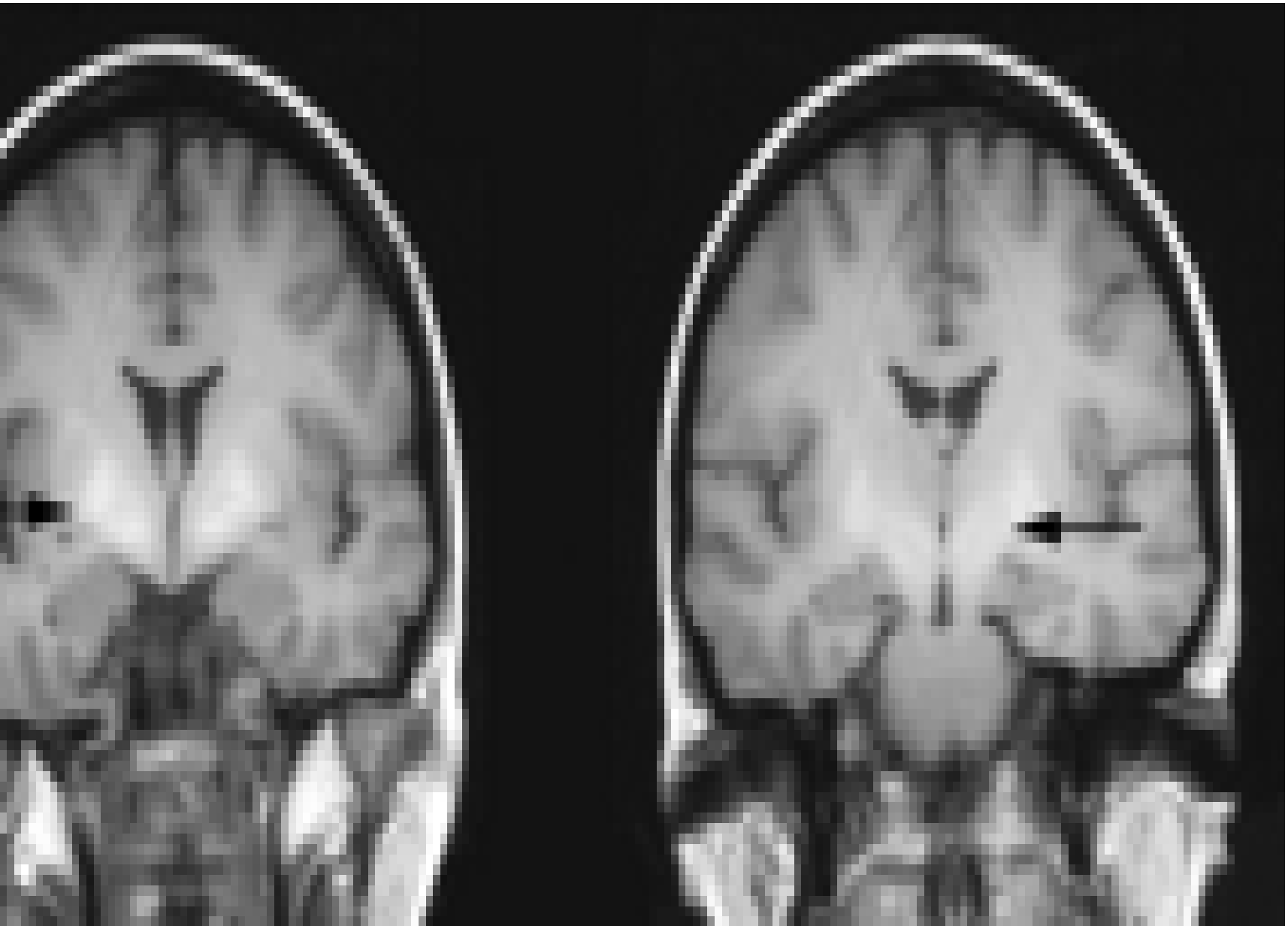
ETA

ELTA



MRI:

agnetic resonance imaging of the
ain proved an abnormally high
gnal on T1-weighted imaging in the
sal ganglia, particularly the globus
llidus. This high signal is now
lieved to be due to manganese
position, and post-mortem studies
ve shown levels up to seven times



RS, PET

ke MRI findings, in cirrhotic tients magnetic resonance ectroscopy and 18-fluoro-deoxy- ucose positron emission mography have also disclosed normal findings in the basal nglia. The relationship of these

REBRAL BLOOD FLOW:

rebral blood flow is low in patients
th liver cirrhosis and, among
rhotics, it is lower in alcoholic and
al cirrhosis than in cholestatic
er disease. In patients with
evious alcohol abuse, cerebral
ood flow is reduced in the frontal

UTOPSY STUDIES:

ructural changes in neurons In tients who die with cirrhosis and rtal-systemic shunts, proved an crease in the number and size of trocytes, particularly Alzheimer pe 2 astrocytes. Such changes may induced by raised concentrations ammonia, but they are not a ature of the brain in fulminant

IFFERENTIAL DIAGNOSIS
F HEPATIC ENCEPHALOPATHY

Other metabolic encephalopathies:

ypernatraemia, the manifestations of ypernatemia are those of yperosmolality. The symptoms range om lethergy to seizures, coma and eath.

yponatraemia, the manifestations are ainly attributable to CNS edema, which usually not seen until the serum odium falls to 120 meq/l or less. ymptoms range from mild lethergy to

ypoxic-ischemic encephalopathy:

lateral hippocampal damage causes rsakoff's amnesia. This is a memory order characterized by inability to retain w information (anterograde amnesia) and less severe defect of recall of old mories (retrograde amnesia).

ffuse cortical, thalamic, or combined uronal loss (with intact brainstem) ults in dementia or the persistent getative state (loss of cognitive functions d emotion with preservation of sleep-

perglycaemia or hypoglycaemia,

percapnia, the clinical manifestations
hypoventilation syndromes include
spiratory acidosis, usually warsened at
ht with subsequent morning headache
d daytime somnolence with eventual
ellectual impairment.

aemia, Uremic encephalopathy is an
ganic brain disorder. It develops in
tients with acute or chronic renal
ure, usually when creatinine clearance

nifestations of this syndrome vary from
d symptoms (eg, lassitude, fatigue) to
vere symptoms (eg, seizures, coma).
verity and progression depend on the
e of decline in renal function; thus,
mptoms are usually worse in patients
h acute renal failure. Prompt
ntification of uremia as the cause of
cephalopathy is essential because
mptoms are readily reversible following

Wilson's disease (Hepatolenticular
generation):

autosomal recessive disorder, typically
pearing in late adolescence. Copper is
reased to saturation levels in the liver
owed by accumulation in the brain,
rnea (Kayser-Fleisher ring round corneal
bus), and kidney. Metabolic defect
known, may be inability of bile duct to

ain: Degeneration of basal ganglia: coordination (especially involving e movements), clumsiness, owness of voluntary limb ovements and speech, tremor, sarthria, excessive salivation, axia, dysphagia, and mask-like cies

ntoxication with sedative/hypnotic
ugs (e.g. triazolam):

izziness - drawsiness

headache - nervousness

nausea - vomiting

coordination problems

Consequences of head trauma
ostconcusive syndrome):
elirium and wishing not to be moved.
evere memory loss.
cal deficit.
obal confusion.
epetitive vomiting and nystagmus.
owsiness.
abetes insipidus.
ositive findings on CT scan or EEG
uld be common in such cases.

Organic intracranial lesions: In ome cases the symptoms are elatively nonspecific and usually re characterized by an intermittent eadache accompanied by some egree of personality change, rowsiness, or confusion. This ondition is easily confused with rug intoxication, cerebral stroke,

Alcohol intoxication and withdrawal syndromes:

rnicke's encephalopathy: nystagmus, axia, and confusion often accompanied ophthalmoplegia. Cardiovascular olvement may be signaled by chycardia as an early manifestation of ripheral vasodilatation. Thiamine should administered promptly-preferably fore glucose is given-to any person in om subclinical thiamine deficiency is

rsakoff's syndrome: many of the
coholic patients who recover from
e acute encephalopathy will be left
th profound defect in memory and
arning known as *Korsakoff's*
ychosis.

Delirium tremens (DTs) may occur in patient with underlying alcoholic liver ease. It is important, therefore, to tinguish this syndrome from HE. In ntrast to asterixis associated with HE, ients with DTs have a rapid postural d action tremor. Furthermore, the nifestations of DTs, including delirium, ggest cortical excitation rather than the sumed cortical inhibition that seems to


تبارك الله أحسن الخالقين

DIAGNOSIS OF MINIMAL HEPATIC ENCEPHALOPATHY

e technique of critical flicker
quency might be effective in
entifying cases of MHE. This
chnique establishes the frequency
which a flashing light appears to
op flashing and becomes
ntinuous (fusion frequency). The
sion frequency dropped with

uropsychological tests: Can be
plied to detect and quantitate
normalities of mental function in
tients with liver diseases, who have
E or early prestupor stages of HE.
umber connection tests part A
CT-A); is a derivative of the trial making
lities that measures the cognitive
nction. Patients perform the test by
nnecting numbers printed on paper

Digit symbol test (DST); this is a
bset of the Wechsler Adult
elligence Scale and measures
otor speed and accuracy. The
tient is given a list of digits
sociated with symbols from 1-9
d is asked to fill in blanks with
mbols that correspond to each

europhysiological assessment:

e EEG was recorded by standardized
hniques. Patients were graded into the
ferent stages of HE according to their
an Dominant Frequency (MDF), and the
ative powers of delta and theta activity.
oked potentials testing is of greatest
ity in detecting subclinical spinal cord
d optic nerve lesions. However, it could

Neuroimaging techniques such as
agnetic resonance spectroscopy
RS) and positron emission
mography (PET) have been used in
e assessment of MHE, but at the
oment they are more useful in
search and in further establishing
e pathophysiology of the condition.


من إله غير الله
يأتيكم بضياء

DIAGNOSIS OF FULMINANT HEPATIC FAILURE

ute hepatic necrosis leading to
patic encephalopathy and
agulopathy develops secondary to
virus, toxin or immune mediated
ack. It is associated with failure
hepatic regeneration. The
ocesses leading to such profound
patic damage are unknown, but
e multifactorial and depend on the
e and susceptibility of the host

ab Studies:

ver function studies:

Levels of hepatic enzymes do not correlate well with the severity of the disease; they may be elevated, normal, or even decreased in patients with FHF.

Levels often are markedly elevated in patients with metabolic disorders.

With progressive necrosis of the liver,

rum bilirubin: Both direct and indirect
um bilirubin levels usually are elevated.
pically, conjugated hyperbilirubinemia is
esent.

chemistry: Glucose level is decreased,
pecially in infants. Hyponatremia,
perkalemia, respiratory alkalosis, or
tabolic acidosis also may be present.

agulation profile: Prothrombin time (PT)
prolonged. However, it does not respond

ral studies:

HAV, HBV, HCV, HDV, and hepatitis E viruses account for approximately 50% of cases. Many viruses other than hepatitis also are recognized causes of FHF in childhood.

HBV is the most common cause of FHF n endemic areas. Presence of IgM anti-HBcAg or HBsAg in serum supports the

HAV infection is a recognized cause of FHF in individuals of all ages. Diagnosis of HAV infection is made by the presence of anti-HAV IgM in the patient's serum.

HCV infection is diagnosed with detection of anti-HCV antibody or HCV RNA in the serum.

HDV is diagnosed by the presence of anti-HDV RNA in the serum.

Other causative viruses include Epstein-Barr virus, CMV, herpesviruses, and

ver biopsy:

Liver biopsy is usually an essential procedure to consider in the management of FHF. It contributes to the working diagnosis and subsequent therapy. However, samples should be examined with caution because results correlate poorly with prognosis. Liver biopsy mostly is required to further assist in reaching a likely diagnosis or in

view of the presence of agulopathy, weight the risk of liver opsy against its contribution to agnosis and management. ministration of vitamin K typically s not been found to result in a tisfactory drop in PT in FHF. ansvenous biopsy is not commonly used as a relatively fe route in such a clinical

Conclusion:

ere are no specific clinical features or tterns of laboratory test results that e diagnostic of HE. Accordingly, the gnosis of HE requires:

clinical judgment and

involves establishing the presence of pato-cellular insufficiency and


THANK YOU