NOTES
ENCEPHALOPATHY

GENERALLY, WHAT IS IT?

PATHOLOGY & CAUSES

- Abnormal brain structure/function
- Permanent/reversible brain injury due to direct injury/other illness

SIGNS & SYMPTOMS

- Altered mental status
  - Irritability, agitation, confusion, somnolence, stupor, coma, psychosis, delirium
- Seizure, myoclonus, asterixis, ataxia, tremor

DIAGNOSIS

DIAGNOSTIC IMAGING

Brain imaging (CT scan, MRI, etc.)

- Changes indicative of Wernicke–Korsakoff syndrome (e.g. shrunken mammillary bodies)

LAB RESULTS

Blood studies

- Complete blood count (CBC), comprehensive metabolic panel (CMP)
  - ↑ ammonia, ↑ transaminases, ↑ prothrombin time, hyper/hypoglycemia

Cerebrospinal fluid (CSF)

- Determine underlying cause, rule out other causes

OTHER DIAGNOSTICS

Electroencephalogram (EEG)

- High-amplitude low-frequency, triphasic waves

TREATMENT

MEDICATIONS

- Anticonvulsants
  - Individuals with seizures due to encephalopathy

OTHER INTERVENTIONS

- Careful monitoring, supportive measures (e.g. IV fluids, nutritional support)

BERIBERI

osms.it/beriberi

PATHOLOGY & CAUSES

- Thiamine (vitamin B1) deficiency
  - Decreased intake/inability to absorb thiamine

RISK FACTORS

- Common in individuals who are alcoholic, malnourished, elderly

COMPLICATIONS

- "Wet beriberi"
  - Cardiomegaly, cardiomyopathy, heart failure

SIGNS & SYMPTOMS

- Nystagmus, ataxia, ophthalmoplegia (triad of Wernicke–Korsakoff syndrome), confusion
- *Wet beriberi:* tachycardia, dyspnea, edema
- *Dry beriberi:* peripheral neuropathy, confusion, pain; AKA Wernicke–Korsakoff syndrome

DIAGNOSIS

DIAGNOSTIC IMAGING

CT scan/MRI

- Changes indicative of Wernicke–Korsakoff syndrome (e.g. shrunken mammillary bodies)

OTHER DIAGNOSTICS

- History
  - Alcoholism/low nutritional state

TREATMENT

MEDICATIONS

- IV thiamine supplementation
  - Avoid glucose before thiamine if alcoholic; can precipitate encephalopathy

HEPATIC ENCEPHALOPATHY

osms.it/hepatic-encephalopathy

PATHOLOGY & CAUSES

- Brain injury due to toxic metabolites; not removed by liver due to liver dysfunction
- Accumulation of toxic metabolites (e.g. ammonia), byproduct of nitrogen metabolism
- Ammonia detoxification in astrocytes → glutamine accumulation → osmotic stress → swelling
- Other injuries (e.g. alkalosis, metabolic abnormalities, medications, bleeding, infection) → hepatic encephalopathy

SIGNS & SYMPTOMS

- *Mental status:* confusion, poor concentration, stupor, coma
- *Neuromuscular:* asterixis, rigidity, hyperreflexia
- Graded by severity
  - *Grade I:* mild; short attention span; mood, sleep problems
  - *Grade II:* moderate; decreased energy, slurred speech, tremors
  - *Grade III:* severe; confusion, stupor, anxiety
  - *Grade IV:* coma

DIAGNOSIS

DIAGNOSTIC IMAGING

T1-weighted MRI

- Hyperintensity of globus pallidus

LAB RESULTS

- Blood tests
  - ↑ ammonia

OTHER DIAGNOSTICS

- Psychometric tests
  - Inhibitory control test (ICT); mental status changes
- History
  - Liver disease, altered mental status

EEG

- High-amplitude low-frequency, triphasic waves

TREATMENT

MEDICATIONS

- Lactulose
  - Decrease absorption of ammonia
- Rifaximin
  - Kill bowel flora that produce ammonia

OTHER INTERVENTIONS

- Nutritional support
  - Limit protein intake

REYE SYNDROME

osms.it/reye-syndrome

PATHOLOGY & CAUSES

- Encephalopathy, liver failure associated with salicylate use in children with viral illness
- Rare syndrome in children ages 4–12; associated with aspirin use during viral infection (e.g. varicella, influenza A/B)
- Uncoupling of oxidative phosphorylation reactions
- Oxidative phosphorylation in mitochondria fails → liver damage → nitrogen-containing toxins not removed from blood → ammonia accumulates in blood → crosses blood-brain barrier → swelling, oxidative damage to astrocytes → brain inflammation, edema → encephalopathy

SIGNS & SYMPTOMS

- Five stages
  1. Quiet, sleepy, vomiting
  2. Stupor, seizures, decorticate response, intact pupillary reflex
  3. Possible coma, decerebrate response, absence of pupillary reflex
  4. Coma, absence of deep tendon reflex
  5. Death

DIAGNOSIS

LAB RESULTS

- Blood studies
- ↑ ammonia, ↑ transaminases, ↑ prothrombin time, hyper/hypoglycemia

OTHER DIAGNOSTICS

- History
  - Viral illness, aspirin use

TREATMENT

MEDICATIONS

- Mannitol, glycerol
  - Manage cerebral edema

OTHER INTERVENTIONS

- Hyperventilation
  - Manage cerebral edema
- Careful monitoring, supportive measures (e.g. IV fluids)

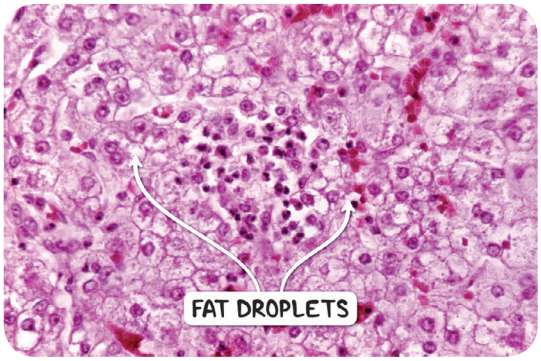

Figure 74.1 The histological appearance of the liver of a child who died from Reye syndrome. The hepatocytes have accumulated fat droplets which causes a pale appearance.