

Neurological Disorders

Transient Ischemic Attack (TIA)

Brief period of neurological deficit – hemiparesis, slurred speech, visual loss – lasting maximum of 24 hours with full recovery. May be prelude to CVA. Patients who experience TIA's are at higher risk for having a stroke.

Cerebrovascular Accident (CVA) = Stroke “Acute Brain Attack”

Sudden loss of brain function resulting from disruption of blood flow to a part of the brain. Maybe the culmination of vascular disease over time.

Causes:

Cerebral Thrombosis – a blood clot within the vessels of the brain.

Embolism- a blockage originating from a site distant to the brain, like fat embolus from a

broken bone or DVT (Deep Vein Thrombosis) that embolizes into vascular system..

Ischemia- decrease of blood flow to the brain tissue as seen in arteriosclerosis and the build

of plaque in the vessels which narrows the lumen ↓ blood supply

Cerebral hemorrhage-Rupture of a blood vessel due to degenerative changes in the vessel,

tumor or some medications

Risk Factors: Hypertension, diabetes mellitus, genetic predisposition, prior TIA's and oral

contraceptives

Characteristics:

Sudden headache, dizziness, loss of reflexes, bladder dysfunction, and convulsion.

Loss of motor control - hemiplegia

Abnormal vital signs such as increase of BP with normal pulse rate, increase of temperature, change in respiration (faster –slower –uneven) or unequal pupils.

Aphasia, alteration in communication:

Receptive Aphasia (does not understand what is coming in)

Expressive Aphasia, (difficulty in expressing thought, written or spoken)

Global Aphasia (both) expressive and receptive

Altered levels of consciousness.

Dysphagia – difficulty swallowing

Homonymous hemianopsia – loss of a visual field of one side of each eye

Apraxia – loss of ability to perform skilled tasks

Dysphasia- difficulty speaking.

Spinal cord injuries

Pathophysiology:

Most common in males age 15 – 25; R/T accidents

Trauma to spinal cord → localized damage → hemorrhagic areas / edema → ischemia → necrosis

Spinal shock –Cord below damage site fails to function → hypotension; bradycardia; lack of thermoregulation below injury; loss of reflexes below level of injury.

Temporary condition that lasts about a week, but can last months, so don't know what function may return till this is complete.

Contusion, laceration or compression, which causes permanent damage to the cord. Need immediate management to immobilize the injured area at the scene to reduce damage to the underlying spinal cord.

Characteristics:

Flaccid paralysis occurs below the level of the injury

In later stages muscle may become hyperreflexic and spastic

Absence of reflexes below the level of the injury

Hypotension – P/T ↓ innervation of sympathetic nervous system (loss of sympathetic tone)

Hypotonia of bladder and bowel leads to distension causing **autonomic dysreflexia**: exaggerated autonomic responses → hypertension, headache, diaphoresis and flushed skin

Inability to sweat in the affected areas

Damage above C-4 respiration will be involved “Above C4 = breath no more”

Parkinson's disease

Progressive disorder affecting the brain center that is responsible for control and regulation of movement. A decrease of the neurotransmitter dopamine in the basal ganglion is the main problem. The cause is unknown but there may be contributing factors such as a virus, CVA, chemical or physical trauma. Also may be associated with a reduction of cerebral blood and dementia (15-20%) in late stage. The disease may progress slowly 15-20 years until patient is invalid. Clients usually die from complications of a fall due to the inability to maintain balance.

Characteristics:

Bradykinesia (slow movement),

Resting tremor

Muscle stiffness, and muscle rigidity,

Over all weakness and loss of postural reflexes.

Specific symptoms:

Mask like facial expression

Pill rolling hand movements

Propulsive or a shuffling gait

Impaired speech and difficulty writing impairs communication.

Drooling, dysphasia and a decrease in appetite may lead to weight loss.

Complications of the altered immobility

Increased risk of constipation, incontinence, UTI and pneumonia.

Death often occurs from falls and accidents resulting in a CVA.

Medication may cause hallucinations and night terrors.

Multiple Sclerosis

Chronic debilitating disease caused by the progressive demyelination of nervous tissue in the brainstem, cerebrum, cerebellum, and spinal cord. Cause of the demyelination is unknown; categorized as autoimmune. Myelin is a protective covering of the nerves which helps nerve conduction. (think of a coated electrical wire)

Demyelination leads to disruption in nerve impulse transmission.

Characteristics

- Onset early adulthood 20-40 years
- Higher incidence in females, in Caucasians and people in cold climates
- Periods of remission and exacerbation
- Fatigue, infection, physical trauma, heat, and stress increase symptoms
- Short term increases in body temperature and serum Ca⁺⁺ may also increase symptoms

Clinical Findings

- Dysphagia
- Tonic head turning
- Paresthesias (tingling sensation) lasting only moments
- Blurred and double vision, blindness in late stage
- Increase in deep tendon reflexes
- Spastic paralysis
- Impairment of bladder and bowel

Myasthenia Gravis

Chronic, progressive, neuromuscular autoimmune disorder due to the reduction of acetylcholine (Ach) receptor sites at the neuromuscular junction. The receptor sites for Ach are destroyed therefore the muscles do not receive the neuro transmission so they will no longer contract.

Characteristics

- Higher incidence in young females
- Associated with pathological changes in the thymus
- Periods of remission and exacerbation
- Affects swallowing and respirations

Avoid people with respiratory infection can lead to pneumonia

Clinical Findings

- Dysphagia
- Double vision
- Drooping eyelid
- Extreme muscle weakness often seen first in the muscle of the face and than moving down the muscle of the trunk

Alzheimer's Type Dementia

Progressive deterioration in memory & cognition that may last up to 20 years. No single cause, but may be immune dysfunction, viral infection, & genetic predisposition. Find beta-amyloid plaques in cerebral cortex; excessive loss of cholinergic neurons – especially in areas for memory & cognition.

Characteristics

- Insidious onset
- Flattening of affect
- Impaired cognition & memory
- May see nocturnal wandering
- Personality change to stubborn, belligerent, lacking social graces

Overall goals are:

- Maintain functional ability for as long as possible
- Maintain a safe environment

Meet personal care needs

Liver

Gross Anatomy

Four lobes each divided into several smaller lobules

Hepatic duct delivers bile to Common bile duct from the liver

Cystic duct delivers bile to the Common Bile Duct from the Gallbladder

Common hepatic bile duct which passes into duodenum to deliver the Bile

Histology

Hepatocytes produce and secrete bile, HCO₃

Kupffer cells these cells act as phagocytes engulfing foreign bacteria, these cells are also found in the spleen, lungs, and lymph nodes

Blood supply

Hepatic artery-oxygenated blood

Hepatic portal vein-deoxygenated blood rich with newly absorbed nutrients from small intestine, spleen, and stomach enters the liver on the inferior aspect

Hepatic vein-exits the liver on the superior aspect rejoining the Inferior Vena Cava

Blood flow in the liver

Both O₂ and DeO₂ blood enter the liver and form a common capillary bed called the sinusoids of the liver and passing by the hepatocyte

Blood flows from the sinusoids to the central vein to the hepatic vein and finally rejoins the Inferior Vena cava

Functions:

ONE: Metabolism of carbohydrate, lipids, and proteins to maintains normal glucose level in the blood

Metabolic processes

Glucogenesis-glucose from a carbohydrate source is turned into glycogen and stored

Glycogenolysis- glycogen turned back into glucose for cellular use

Gylconeogenesis -lipids or proteins converted into glucose

Protein Metabolism

The byproduct of the conversion of amino acids into glucose is Ammonia (NH₃) this is a toxin, Which is then converted into urea which is less toxic and is then removed from the body either by the kidneys or in the feces. This process is normal as long it is keep down to a minimum and protein is not being used as the main source of energy for the body. However if there becomes an abnormal amount of protein synthesis, the ammonia build up can damage the liver and cross the blood - brain barrier causing coma

The production of Plasma Proteins

Albumin- Lifespan @14 days

Alpha and beta globins

Transport proteins

Clotting Factor-with the aid of Vitamin K Lifespan 24-36 Hours

Lipoprotein- used to make the cell membrane

Lipid Metabolism

The by-product of the conversion of lipids into glucose are ketone bodies. Lipids are used for quick energy and fatty acids, which are used to produce cholesterol, hormones, and complex lipid.

TWO: Production of Bile

Substance used in the emulsification of large fat molecules into smaller particles to aid in the digestion of lipids

Produced by the Hepatocytes

Contains: Water, Na⁺, Ca, K, Cl⁻, HCO₃⁻, Fatty Acids, Cholesterol, bilirubin, and bile salts

Stored in the Gallbladder

THREE: Conjugation of Bilirubin

In the spleen old RBC are broke into three parts the **Globin** (protein), the **Ferritin** (iron) and **Heme** (pigment)

The globulin and the Ferritin are recycled.

The Heme however is transported to the liver where it under goes various changes. First it is unconjugated **bilirubin (lipid based and water insoluble)** and then it is converted to conjugated **bilirubin (protein based and water-soluble)** by the hepatocytes this allows the bilirubin to move in a water solution. The bilirubin is then secreted into the bile and move to the small intestine. A bacteria in the small intestine converts bilirubin to **urobilinogen** this substance can either be removed by the feces or reabsorbed and secreted by the kidney in the urine

If unconjugated bilirubin is released into the blood stream in high levels it can damage cell by diffusing into the cell through the lipid based membrane.

FOUR: Detoxifies the blood of heavy metals, drugs such as barbiturates and amphetamines, also breaks down hormones

FIVE: Storage of vitamins (A, B-12, D, E, and K), and minerals such as iron and copper

SIX: Phagocytosis of worn out white and red blood cells, some bacteria

SEVEN: Activation of vitamin D

Characteristics of Liver Disorders

ASCITES

Abnormal accumulation of fluid (over 500ml) in the abdominal region

Fluid consist of proteins and electrolytes

Fluid leaks from veins and lymphatic vessel in the abdominal cavity caused by the increased pressure in the hepatic portal system

Decrease of fluid in the blood as it leaks into abdomen causes a decrease in blood volume and finally leads to a decrease in urine output

Causes:

Increased portal hypertension

Impairment of the kidneys causes an increase of H₂O and Na⁺ in the blood

Reduction of plasma proteins production causing a decrease in the osmotic pressure in the blood

Increase of aldosterone due to the fact that the damaged liver can not metabolize and degrade the hormone causes an increase of H₂O and Na⁺ in the blood

ESOPHAGEAL VARIES*

Enlarging of the veins in the esophagus due to an increase of blood flow to the area (collateral circulation). The increased blood flow is due to diminished blood flow in the hepatic portal system (Increased portal hypertension)

Varies come rupture leading to intense bleeding, can also manifest in slow chronic bleeding.

* Varies may also be found in other areas of the GI tract such as the colon and stomach

JAUNDICE

The yellow or greenish-yellow coloration of the skin caused by the build up bilirubin in the blood

Hemolytic Jaundice

Increase in the destruction of the RBC Bilirubin can not be excreted fast enough by the normal liver this would be unconjugated bilirubin Example Sickle cell anemia

Hepatocellular Jaundice

Diseased liver is unable to clear the bilirubin from the blood. This would be both unconjugated bilirubin and conjugated bilirubin

Obstructive Jaundice

Obstruction of the bile duct causes the bile to back up into the liver. The bile maybe reabsorbed into blood to be excreted by the kidney. This would be conjugated bilirubin

Gallbladder

Function-store and concentrate bile until it is needed and is released into the small intestine by way of the Sphincter of Oddi

DISORDERS OF THE LIVER

Hepatitis A infectious hepatitis

Oral - fecal route of transmission, ingestion of contaminated food / water.

Incubation: 1-7 wk., average 4 wk., duration 4-8 wk. mild flu-like upper respiratory infection, fever low grade, anorexia, jaundice

Hepatitis B Serum. Transmitted through body fluids

Longer incubation period 2-5 months, insidious and variable

Symptoms include anorexia, abdominal pain, generalized aching, malaise, weakness, possible jaundice, and enlarged and tender liver.

Hepatitis C or Non A or B:

Blood borne carrier possible.

Causes:

Major cause: Blood transfusion. Related viral hepatitis, no jaundice,

Similar to hepatitis B may become chronic.

Cirrhosis: scarring of the liver Enlarged liver, loaded with fat-firm and sharp edge on palpation, may become nodular and healthy tissue is replaced with scar tissue.

Laennec's portal- alcoholic, nutritional, -most frequent

Postnecrotic -wide bands of scar tissue acute hepatitis

Biliary-chronic biliary obstruction and infection;

Rare insidious onset and protracted course (30 yrs. or more)

Hepatic Coma

Accumulation of ammonia and other toxins. The ammonia is released into the blood stream in its raw form instead of in the form of urea. The ammonia then enters the CNS causing coma and then death.