**HYDROCEPHALUS (HCP)**

**for 5th class: medical college: Al Mustansiriyah University**

**by Dr Mohamed Al Tamimi**

****

**DEFINITION**:

increased CSF volume: normal CSF volume = 100 - 150 mL (50 in ventricles, 25 around brain,75 around spinal cord)

CSF production is constant at 35 cc/hr (500 - 750 cc/day)

**MECHANISMS**

increased production e.g. choroid plexus papilloma (0.4-1% of intracranial tumours)

decreased absorption (see below)

**CLASSIFICATION**

**Non-Communicating (Obstructive) Hydrocephalus**

absorption is blocked within ventricular system - no escape of CSF into subarachnoid space

*causes/location of block*

 • intraventricular hemorrhage

 • ventricular tumours (e.g. 3rd ventricle colloid cyst)

 • supratentorial mass causing tentorial herniation and aqueduct compression

 • infratentorial mass causing 4th ventricle obstruction or aqueduct kinking

 • congenital e.g. aqueductal stenosis

*CT findings*

 • lateral and 3rd ventricles dilated

 • normal 4th ventricle (e.g. aqueduct stenosis) or deviated/absent 4th ventricle (e.g. posterior fossa mass)

**Communicating (Non-Obstructive) Hydrocephalus**

absorption is blocked at some part of extraventricular pathway, such as arachnoid granulations

*causes*

 • meningitis

 • SAH

 • trauma

*CT findings*

 all ventricles are dilated

**Normal Pressure Hydrocephalus (NPH)**

gradual onset of classic triad ( gait apraxia, incontinence, dementia)

CSF pressure often within clinically “normal” range

usually communicating in nature

**Hydrocephalus Ex Vacuo**

enlargement of ventricles (and sulci) secondary to diffuse brain atrophy

usually a function of normal aging

not true hydrocephalus

**CLINICAL FEATURES OF HYDROCEPHALUS**

**Acute HCP**

signs and symptoms of acute raised ICP

usually non-communicating type

**Chronic HCP**

similar to NPH

**INVESTIGATIONS**

**1. CT**

ventricular enlargement, may see prominent temporal horns

periventricular lucency (CSF forced into extracellular space)

narrow/absent sulci, +/– 4th ventricular enlargement

**2. Ultrasound (through anterior fontanelle in infants)**

ventricular enlargement

**MANAGEMENT**

1. spinal taps (for transient, communicating HCP)

2. remove obstruction (if possible)

3. choroid plexectomy (for choroid plexus papilloma)

4. third ventriculostomy (for obstructive HCP)

5. shunts

• ventriculoperitoneal (VP) = ventricle to peritoneum

• ventriculo-atrial (VA) = ventricle to right atrium

• lumboperitoneal = lumbar spine to peritoneum (for communicating HCP)

**Shunt Complications**

1. obstruction

• etiology: infection, obstruction by choroid plexus, buildup of proteinaceous accretions, blood, cells (inflammatory or tumor)

• signs and symptoms of acute HCP or increased ICP

• radiographic evaluation: “shunt series” (plain x-rays which only show disconnection of tube system), CT

2. infection (3-4%)

• etiology: *S. epidermidis, S. aureus*, gram-negative bacilli

• presentation: fever, nausea and vomiting, anorexia, irritability; signs and symptoms of shunt obstruction; shunt nephritis (antibodies generated against bacteria in shunt leads to kidney damage)

• investigation: CBC, blood culture, shunt tap (LP usually NOT recommended)

3. overshunting

• slit ventricle syndrome (collapse of ventricles leading to shunt catheter occlusion by ependymal lining)

• subdural effusion

• secondary craniosynostosis (children)

• low pressure headache

4. seizures

5. problems related to distal catheter (blockage)