



Noncommunicating Hydrocephalus

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Noncommunicating hydrocephalus is often referred to as obstructive hydrocephalus and is by definition an intraventricular obstruction of cerebrospinal fluid flow. Patient symptoms depend on the rapidity of onset. Acute obstructive hydrocephalus causes sudden rise in the intracranial pressure, which may lead to death, whereas in chronic hydrocephalus there may not be any symptoms. Computed tomography and magnetic resonance imaging play important roles in the diagnosis and management of hydrocephalus. Advances in magnetic resonance imaging such as the 3D sequences and phase-contrast imaging have revolutionized the preoperative and postoperative assessment of noncommunicating hydrocephalus. We would be discussing the various causes of noncommunicating hydrocephalus and their imaging.
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Introduction

Noncommunicating hydrocephalus occurs when there is obstruction to cerebrospinal fluid (CSF) flow within the ventricular system. The CSF produced by the choroid plexus, ependymal lining of the ventricles, and brain parenchyma, courses through the lateral ventricles and exits through the foramen of Monro into the third ventricle. The CSF then flows from the third ventricle into the fourth ventricle through the cerebral aqueduct and finally exits the fourth ventricle into the subarachnoid space through the foramina of Luschka and Magendie. The causes of obstruction can be congenital or Toxoplasmosis, Other (syphilis, varicella-zoster, parvovirus B19), Rubella, Cytomegalovirus (CMV), and Herpes (TORCH) infections, especially at the foramen of Monro and cerebral aqueduct, webs and synechiae of prior hemorrhage, intraventricular lesions or cysts, and space-occupying lesions with significant size to obstruct the ventricular outlets (Table). In noncommunicating hydrocephalus, the CSF production continues, despite the obstruction, resulting in the elevation of the ventricular pressure. The ventricles enlarge and cause mass effect on the adjacent brain parenchyma. Noncommunicating hydrocephalus can be acute or chronic. Acute obstructive hydrocephalus can be life threatening if not immediately detected and treated, as this may lead to brain herniation and even death. Chronic noncommunicating hydrocephalus may not cause significant symptoms as the ventricular system

proximal to the level of obstruction gradually enlarges and contributes to atrophy of the surrounding parenchyma. Although computed tomography (CT) is useful in the detection of hydrocephalus and documenting the degree and cause of obstruction, the advances in magnetic resonance imaging (MRI) help in better characterization of the cause, surgical planning, and postoperative evaluation.

Diagnosis and Management

Clinical symptoms of noncommunicating hydrocephalus depend upon the nature of obstruction. Sudden acute obstruction (eg, occurring at the foramen of Monro from colloid cyst obstruction) can cause sudden rise in the intracranial pressure and may cause sudden death.¹ Symptoms of raised intracranial hypertension, depending upon the rapidity of onset to the obstruction of intraventricular CSF flow, include headache, nausea, vomiting, altered mental status, papilledema, blindness, coma, or even death.

CT is often the initial imaging modality to detect and characterize the type of hydrocephalus, degree and possible cause of obstruction, and complications from acute obstruction. The radiological features of hydrocephalus include dilatation of the ventricular system depending on the level of obstruction and can be compared with the prior imaging if available. The earliest feature of hydrocephalus is rounding of the frontal horns of the lateral ventricles and dilatation of the temporal horns. Bulging of the floor and the lateral walls of the third ventricle can be seen. When the fourth ventricle is also dilated, the differentiation had to be made from the communicating hydrocephalus. Confluent periventricular

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Table Classification of noncommunicating hydrocephalus based on level and cause of obstruction.

Level of Obstruction	Causes for Obstruction
Foramen of Monro	Congenital stenosis, TORCH infections, hemorrhage, intraventricular cysts such as colloid cyst, choroid plexus cyst, arachnoid cyst, dermoid, or epidermoid Neoplasms in the adjacent tissue causing external compression at this level such as glial tumors, ependymomas, or subependymomas
Aqueduct	Congenital stenosis, webs and membranes, cysticercosis, hemorrhage, periaqueductal infective lesions, pineal gland neoplasms, tectal and tegmental gliomas, and metastasis
Foramina of Luschka and Megendie	Intraventricular or cerebellar hemorrhage, cerebellar infarction, infections, extrinsically compressing posterior fossa neoplasms such as medulloblastoma, ependymoma, cerebellar astrocytoma, brain stem glioma, hemangioblastoma, cerebellopontine angle tumors, and metastasis

hypoattenuation from transependymal (interstitial) flow would be seen in acute hydrocephalus. Effacement of sulci is seen from surrounding mass effect. Elevation of intracranial pressure from acute obstruction can cause brain herniation with compression of the brain stem, which can be detected on CT. In chronic noncommunicating hydrocephalus, the ventricles proximal to the level of obstruction gradually dilate over a period of time. There would be thinning and elevation of corpus callosum, fenestration of corpus callosum, and depression of fornices.

Conventional MR images including axial and sagittal turbo spin echo T1-WI, axial, coronal turbo spin echo T2-WI, axial fluid-attenuated inversion recovery, and axial or sagittal magnetization prepared rapid acquisition gradient echo. Post-gadolinium T1-weighted images in various planes and additional T1 magnetization prepared rapid acquisition gradient echo images may be used based on the clinical indication. Conventional MR sequences are useful in further evaluating the cause of noncommunicating hydrocephalus and characterizing the lesions causing the obstruction. However, other causes, such as webs, membranes, and intraventricular neurocysticercosis, may not be clearly visible on the conventional MR sequences. Acute obstruction demonstrates periventricular T2-weighted and fluid-attenuated inversion recovery hyperintensities from transependymal flow. Diffusion imaging can be used to detect the improvement of interstitial edema posttreatment of acute hydrocephalus by calculating the average diffusion constants.²

3D sampling perfection with application optimized contrasts using different flip angle evolution (SPACE) and 3D constructive interference in steady state (CISS) are effective techniques for evaluation of intraventricular obstructive hydrocephalus, especially for evaluation of webs and membranes. Phase-contrast MRI (PC-MRI) is useful for quantitative and qualitative evaluation of communicating and noncommunicating hydrocephalus. These advances in the MR technique are very useful in the preoperative surgical planning, especially in endoscopic third ventriculostomy (ETV) and also in post-operative assessment. These techniques have almost replaced the invasive contrast-enhanced CT cisternography or contrast-enhanced MR cisternography (CE-MRC).

PC-MRI gives quantitative and qualitative information of CSF circulation. PC-MRI detects only the moving nuclei canceling the signal from stationary nuclei. For optimal signal, the selected velocity encoding (VENC) should be close to the

CCF flow velocity, and the mean VENC value is 5-8 cm/s. Low VENC value (2-4 cm/s) is required in the evaluation and discrimination of noncommunicating and communicating hydrocephalus.³ A total of 2 series of images are acquired, 1 series in axial plane perpendicular to cerebral aqueduct with through-plane velocity encoding in craniocaudal direction for quantitative assessment. The other series is acquired in sagittal plane with in-plane velocity encoding in craniocaudal direction for qualitative assessment. As the CSF flow is pulsatile and synchronous with the cardiac cycle, cardiac gating can be used to increase the sensitivity of imaging. Cardiac gating can be retrospective or prospective; however, retrospective gating provides more accurate results.⁴ Magnitude and phase images are obtained from this sequence for anatomical and flow information, respectively.

3D CISS (Siemens) and Fast Imaging Employing Steady State Acquisition with Phase Cycling (GE) combine 2 consecutive runs of 3D-balanced steady state free precession, the first run with alternating flip angle and the second with constant flip angle. Maximum intensity projection is acquired between the 2 acquired data sets.⁵ The 3D CISS images provide excellent morphologic evaluation.

T2-weighted 3D SPACE (Siemens) and Cube (GE) are acquired with isotropic voxel size ($0.6 \times 0.6 \times 0.6 \text{ mm}^3$) and variable flip angle. T2-weighted 3D SPACE with variable flip angle mode provides excellent physiological information in addition to morphologic information.^{6,7} The presence of flow-related artifacts on T2-weighted 3D SPACE suggests patency, especially in the evaluation of aqueductal stenosis and hence provides qualitative flow information comparable to PC-MRI.⁸

Contrast-enhanced MR cisternography (CE-MRC) is performed with intrathecal administration of gadolinium, which provides morphologic and physiological information especially in the evaluation of aqueducts stenosis, webs, and membranes at the ventricular outlets. Because of the invasive nature of the examination and potential risks of off-label intrathecal gadolinium use, T2-weighted 3D SPACE, in addition to 3D CISS and PC-MRI, have almost replaced the CE-MRC as these sequences provide combination of anatomical and physiological information.

Technique of CE-MRC: after the acquisition of 3D T1-weighted and 3D heavily T2-weighted images, 0.5 mL of gadolinium diethylene triamine pentaacetic acid is diluted 2-fold with CSF and injected into the subarachnoid space using 24-26 gauge spinal needle in the L4-L5 level or lateral portions

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