Hydrocephalus after aSAH can be classified based on **timing** and **pathophysiology**:

This condition can present in acute, subacute, or chronic forms:

Acute hydrocephalus (within 72 hours of hemorrhage) results from obstruction of CSF pathways by blood clots or impaired absorption at the arachnoid granulations.

Subacute hydrocephalus (occurs within days to weeks) is often due to persistent inflammation and clot organization.

Chronic hydrocephalus (develops weeks to months later) is usually a communicating hydrocephalus caused by scarring and fibrosis affecting CSF resorption.

1. **Acute Hydrocephalus (within 3 days of aSAH)**

1. Caused by obstruction of CSF flow due to **blood clots in the basal cisterns or ventricles**.
2. Can be **communicating** (impaired absorption at the arachnoid granulations) or **non-communicating** (obstructed CSF pathways, e.g., at the Sylvian aqueduct or fourth ventricle outlets).
3. Often manifests as **altered consciousness, headache, vomiting, and pupillary abnormalities**.
4. Treatment: External ventricular drainage (EVD) to relieve pressure and allow for CSF diversion.

2. **Subacute Hydrocephalus (4–14 days post-aSAH)**

1. Thought to result from ongoing inflammation and dysfunction of CSF absorption.
2. Patients may develop **progressive confusion, gait disturbances, and urinary incontinence**.
3. Treatment: Persistent hydrocephalus after EVD removal may require **lumbar drainage or shunt placement**.

3. **Chronic Hydrocephalus (>14 days post-aSAH)**

1. Typically **communicating hydrocephalus**, caused by scarring and fibrosis at the arachnoid granulations.
2. Presents insidiously with **cognitive decline, gait ataxia, and urinary urgency/incontinence**, resembling **normal pressure hydrocephalus (NPH)**.
3. Treatment: **Permanent CSF diversion** with a ventriculoperitoneal (VP) or ventriculoatrial (VA) shunt.

Risk Factors for Hydrocephalus After aSAH Several factors increase the likelihood of hydrocephalus after aSAH:

- **Higher Fisher grade (thick cisternal blood and IVH)** → greater inflammatory response and CSF obstruction. - **Intraventricular hemorrhage (IVH)** → direct obstruction of CSF flow. - **Older age** → reduced CSF resorption capacity. - **Female sex** → associated with a higher risk in some studies. - **Poor clinical grade (Hunt-Hess or WFNS score)** → higher initial brain injury burden. - **Aneurysm location (e.g., anterior communicating artery aneurysm)** → increased risk of basal cistern blood clotting.

Diagnosis - **CT Scan (Initial Modality)**: Enlarged **temporal horns**, **third ventricle**, and **sylvian fissures** with periventricular lucency (suggesting transependymal CSF flow). - **MRI with FLAIR and Cine Flow Studies**: Useful in chronic cases. - **Lumbar Puncture (Caution Required)**: Can confirm communicating hydrocephalus but should be avoided in **high-pressure states** to prevent herniation.

Prognosis and Outcomes - Hydrocephalus after aSAH is associated with **worse functional outcomes**. - Persistent hydrocephalus requiring shunting occurs in **~10-20% of cases**. - **Early recognition and intervention improve functional recovery**.

Conclusion Hydrocephalus is a significant complication of aSAH, requiring **timely diagnosis and appropriate management**. Understanding the risk factors and clinical course is essential to improving **neurological outcomes and quality of life** for these patients.
