Complications of SAH:

I. **Vasospasm:** insidious onset of delayed focal or diffuse narrowing of large capacitance arteries at the base of the brain.

- 60% angiographic, 30% clinical, 15% permanent neurological deficit or death despite maximum therapy.
- Presentations: early features(increasing headaches, low grade T, leucocytosis), decrease in the level of consciousness due to global decrease in CBF or vasospasm of the perforators supplying the reticular formation and focal deficit due to reduction in regional blood flow. Usually starts at 3-d day, peak 10-14 days, lasts 3-4 weeks.
- Diagnosis: A. Transcranial Doppler ultrasound (MCA velocity above 120cm/s is highly suggestive, MCA velocity > 150 cm/s – 80-100% sensitivity and 60-70% specificity in detecting vasospasm, increase in velocity > 50 cm/s is a strong indicator and using the MCA /ICA velocity index or carotid index which can differentiate the increase of MCA velocity due to vasospasm from that due to hyperdynamic state.

B. Angiography: the most accurate way, but invasive and carries risk of complications .It is indicated when the spasm is not responsive to usual medical therapy and provides opportunity to give intraarterial papaverine, verapamil and nimodipine (chemical angioplasty) or to perform balloon angioplasty.

C. PET, SPECT and xenon CT scan can give information about the cerebral blood flow but rarely done in clinical practice.

- Pathogenesis: there is strong correlation between the amount of SA blood and the rate vasospasm (Fisher et al: 4 grades 1 –no haemorrhage, 2-<1cm thick clot, 3->1 cm thick clot, 4-IVH, ICH. The risk of vasospasm is increased in grade 3). The SAH releases **oxyhaemoglobin** which is thought to play a major role in the development of vasospasm (by producing **superoxide radical** when it converts to **methaemoglobi**n). Other substances produced in the CSF that may play role in vasospasm include **endothelin (**strong vasoconstrictor produced by endothelium), eicosanoids (prostaglandins and leucotriens) and other cytokines.
- Treatment: Poiseuille's equation , r-radius,Δ P-pressure gradient, V- velocity, L-length of the tube

$$\text{Flow} = \frac{\lambda \, p \, X \, r^4}{L \, XV}$$

1. Triple H therapy: A. Hypervolemia aiming at CVP > 12 and PCWP-15 in ICU setting increases cardiac output and potentially reverses delayed neurological deficit due to spasm in some studies B. Hypertension using inotropes potentially improves the blood flow in ischemic areas where the autoregulation is not effective due to maximal dilatation of the precapillary arterioles. C. Hemodilution: improves the rheology of the blood. No controlled studies. Class 2-3 **evidence 2/3 of patients with ischemic neurological deficit will improve with triple H therapy (**Awad et al).

2. Calcium channel blockers(nimodipine, nicardipine): theoretically prevent vasospasm induced cerebral ischemia by blocking the entry of calcium into the smooth muscle cells, a common pathway for smooth muscle contraction , and blocking the entry of calcium into ischemic neurons, a common pathway mediating cell death. In reality the effect of nimodipine on angiographic vasospasm is minimal if any, however **2 controlled placebo** studies showed significant reduction in cerebral infarction and poor outcome in patients given nimodipine (650 patients. W& R). Side effects include hypotension, psudoobstruction and headaches.
3. If the patient with delayed cerebral ischemia secondary to vasospasm does not improve in **2-3 hours**, the next step is angiography and chemical or and balloon angioplasty (**60-80%** improvement in few min in one study, 5% risk of haemorrhage from unsecured aneurysm or from ruptured of parent artery, small risk of thrombosis, dissection).
4. Other lines of treatment that has not been widely used because of inconsistent results include free radical scavengers (nicotinamide, vitamin E), high dose of steroids (methyl prednisone, dexamethasone) inhibitors of lipid perioxidation and cyclooxegenase, Ticlopidine (leucotriens antagonist), surgical excision of the clot and fibrinolytic therapy intrathecally (t-PA). Magnesium (blocks NMAD receptor).

II. Hydrocephalus :

- Develops in **20-30%** of patients with SAH (communicating)
- 78% of patients with acute hydrocephalus improved with EVD
- **<10%** develop chronic hydrocephalus that require shunting.

III. **Hyponatremia**: **5-10%** of all patients and **33%** of those with A-con aneurysm develop hyponatremia (vasospasm of hypothalamic perforators-increased production of ANP leading to loss of Na and water "cerebral wasting syndrome" rarely due to SIADH.

IV. **Seizures:** early seizures develop in **10-20%** of patients, late seizures in **8%.** Seizures in the first 12 hours do not increase the risk of developing epilepsy. Antiepileptics decrease the incidence of early seizures, but has no effect on the development of late epilepsy. **Higher incidence of epilepsy in patients with MCA artery aneurysm, ICH, insular cortex retraction and gyrus rectus resection**

V. **Pulmonary complications:** 1**4%** of patients dying from SAH has neurogenic pulmonary oedema due to increased sympathetic discharge and left ventricular dysfunction

VI. **Cardiac complications**: **50%** of patients develop ST segment or T wave changes or arrhythmias secondary to subendocardial ischemia (increased circulating catecholamines).