

"WORST HEADACHE OF MY LIFE" (Slides CC14-1)**CASE 3: Answers:**

A classic sign of *subarachnoid hemorrhage* (SAH) is sudden "thunderclap- like" WHOL that patient's can often pinpoint in time. In the vast majority (~85%) of cases, spontaneous SAH occurs due to rupture of an *aneurysm*. Much less often (~5%), SAH is due to a bleeding arterio-venous malformation (AVM) and in about 10% of cases a precipitating cause is not found. SAH can also occur with head trauma, but this will not be discussed here. Risk factors for intracranial aneurysm include atherosclerotic disease, congenital anomalies in cerebral blood vessels, polycystic kidney disease and connective tissue disorders such as Marfan's syndrome. In descending order, the most common locations for aneurysms are on the ACOM, PCOM, MCA, basilar and vertebral arteries. Risk factors for SAH include hypertension, cigarette smoking, alcohol and other situations causing sudden elevations in blood pressure.

The clinical effects of SAH can range from headache, often WHOL, and meningeal irritation causing nuchal rigidity and photophobia, to cranial nerve and other focal neurologic deficits, to impaired consciousness, coma and death. The overall mortality of SAH is about 50%, however, the prognosis is better in milder cases. In SAH due to ruptured aneurysm the risk of rebleeding is about 4% on the first day, and 20% in the first two weeks. Therefore, many neurosurgeons believe that in cases of mild SAH it is advisable to operate immediately and place a clip across the neck of the aneurysm to prevent a second potentially cataclysmic event.

CT Scan and Angiogram:

Following an initial assessment it was felt that the patient was stable enough to go for an urgent head CT to check for intracranial bleeding. Recall that the best study in this situation is a *non-contrast head CT*. Extravascular blood appears bright on head CT probably due to clot formation. The reason that contrast should not be used is that blood vessels will then appear bright and may, therefore, obscure bright areas due to SAH. MRI is not useful in suspected acute SAH since on MRI fresh blood will have the same appearance as CSF. The head CT in our patient (see slide) shows axial images progressing from more caudal to more rostral levels. Identify: frontal lobes, temporal lobes, cerebellum, occipital lobes, parietal lobes, temporal horns, frontal horns, third ventricle, caudate, lentiform nucleus, thalamus, and internal capsule. Note the presence of bright areas signifying SAH in the interhemispheric fissure, basal or interpeduncular cistern, cisterna ambiens (quadragemina) outlining the midbrain and extending back along the tentorium cerebelli.

The patient next underwent urgent cerebral angiography (see slide). Antero-posterior (left) and lateral (right) views are shown following injection of the left common carotid artery. Identify: internal carotid artery, middle cerebral artery, left anterior cerebral artery, and right anterior cerebral artery filling via the anterior communicating artery. Identify also the branches of the anterior communicating artery, the pericallosal and callosomarginal arteries. Note that in the region of the carotid siphon the carotid appears irregular and narrowed due to atherosclerotic disease. A saccular aneurysm (~6x8 mm) can be seen extending from the anterior communicating artery.

Clinical Course:

The patient was taken to the operating room where a large aneurysm was found extending from the postero-superior surface of the anterior communicating artery. A clip was carefully

placed across the neck of the aneurysm without compromising blood flow to the anterior communicating or anterior cerebral arteries. Post-operatively the patient did well, and went home after two weeks with no neurologic deficits or further bleeding.