subarachnoid haemorrhage [created by Paul Young 11/10/07]

general

subarachnoid haemorrhage is blood in the subarachnoid space from a pathological process
- most common medical use refers to non traumatic types of haemorrhage (usually from rupture of a berry aneurysm or AVM)

pathophysiology

primary SAH may result from:
- saccular aneurysm (account for 2/3rds of non-traumatic SAH)
- AVM
- mycotic aneurysmal rupture
- angioma
- neoplasm
- cortical thrombosis

SAH may reflect a secondary dissection of blood from an intraparenchymal haematoma (eg bleeding from hypertension or neoplasm)

aneuryms are associated with Ehlers-Danlos, Marfans, aortic coarctation & PCKD

- the most common sites of ruptured aneurysms are the internal carotid artery, including the posterior communicating artery junction (41%); anterior communicating artery-anterior cerebral artery (34%), middle cerebral artery (20%) and vertebrobasilar arteries (4%); about 20% of patients have multiple aneurysms

risk factors

- risk factors for SAH include:
(i) smoking
(ii) oral contraceptive use
(iii) alcohol abuse
(iv) hypertension
- the risk of SAH increases with age peaking at 55-60 years of age peaking at 55 to 60 years of age
- there is a slight male preponderance in younger patients and a slight female preponderance in older patients
- genetic factors may play a role in aneurysm formation and current recommendations are that individuals with two first degree relatives should undergo diagnostic evaluation for an aneurysm

presentation

- the most common initial symptom of SAH is sudden severe headache; less severe 'sentinal headaches' may precede the presenting event in as many as half the patients
- in 45% of patients , transient or persistent loss of consciousness accompanies the headache
- vomiting can be prominent in awake patients
- seizure activity may be reported but in some cases it is unclear whether this represents true epileptiform activity or reflex posturing related to transient ICP rise at the onset of bleeding
- after a few hours a stiff neck can develop reflecting the sterile meningeal inflammation induced by subarachnoid blood

ophthalmological signs:
- subhyaloid retinal haemorrhage (small round haemorrhage which may have a visible meniscus)
- other retinal haemorrhage
- papilledema

syndromes of mass effect from aneurysms:
(i) posterior communicating artery / internal carotid
- focal, progressive retro-orbital headaches & oculomotor nerve palsy
(ii) middle cerebral artery
- contralateral face or hand paresis, aphasia (left side), contralateral visual neglect (right side)
(iii) anterior communicating artery
- bilateral leg paresis and bilateral Babinski sign
(iv) basilar artery tip
- vertical gaze, paresis & coma
(v) intracranial vertebral artery or posterior inferior cerebellar artery
- vertigo & components of the lateral medullary syndrome
(vi) ophthalmic artery
- monocular vision loss

NB: abducens nerve palsy is a false localising sign of raised ICP

investigations

I. imaging:
(i) CT
- 90% sensitive within the 1st 24hrs, 80% sensitive at 3 days & 50% sensitive at 1 week
- can detect ICH, mass effect and hydrocephalus
- false -ve can result from small vol SAH or severe anaemia
- intraparenchymal bleeding may occur with middle communication artery & posterior communicating artery aneuryms
- interhemispheric & intraventricular haemorrhages may occur with anterior communicating artery aneuryms
(ii) cerebral angiography
- assesses vascular anatomy, current bleeding & presence of other aneurysms
- negative in 10-20% of patients with SAHs
(iii) MRI
- sensitivity in detecting blood is equal or less than CT
- mostly used to identify possible AVMs not visible of angiography

II. lumbar puncture
- indicated if patient has possible SAH & negative CT findings
- LP may be negative less than 2 hours after symptom onset & is most sensitive at 12 hours after symptom onset
- RBCs in CSF that remain elevated in sequential tubes
- xanthochromia is usually seen by 12 hours

medical complications

I. cardiac abnormalities
- cardiac abnormalities are common in the first 48 hours after SAH. ECG changes include:
(i) tall peaked T waves
(ii) ST segment depression
(iii) prolonged QT
- it appears that ECG changes do not represent myocardial ischaemia although cardiac enzymes may become mildly elevated
- in rare cases 'stunned myocardium' may occur with impaired myocardial contractility, hypotension and pulmonary oedema. This phenomenon is usually transient lasting only a few days
- cardiac rhythm disturbances occur in about 30-40% of patients although life-threatening cardiac arrhythmias are rare
- neurogenic pulmonary oedema may occur

II. blood pressure abnormalities
- blood pressure is often elevated after SAH and is associated with a greater risk of rebleeding and vasospasm
- early on management of blood pressure focuses on preventing rebleeding
- after clipping or coiling the risk of rebleeding is virtually eliminated and spontaneous elevations in blood pressure should be allowed to occur without intervention because the risk of exacerbating vasospasm with hypotension is now the predominant concern

III. disturbances in salt and water balance
- occur in approximately 1/3rd of patients
- hyponatraemia and hypovolaemia are correlated with an increased risk of symptomatic vasospasm and poor outcome
- although hyponatraemia was once attributed to SIADH, later evidence has suggested that both sodium and water are lost. In fact, when administered 2-3L of fluid a day as many as half patients develop intravascular volume contraction. The mechanisms of this 'cerebral salt wasting' are unknown

neurosurgical complications

I. Rebleeding
- rebleeding is heralded by sudden worsening of headache, vomiting and development of a new neurological deficit or arrhythmia
- occurs in up to 1/3rd of patients and is often fatal
- the risk of rebleeding is greatest during the first 24 hours and declines rapidly over the next 2 weeks
- rates of rebleeding are highest in women, those with a poor clinical grade, those in poor medical condition and those with elevated systolic blood pressure

II. Hydrocephalus
- hydrocephalus occurs because of disturbances in CSF flow or reabsorption; subarachnoid blood may impair CSF reabsorption at arachnoid granulations and ventricular blood may obstruct its flow
- acute hydrocephalus can develop within hours of subarachnoid haemorrhage often in the absence of intraventricular blood; it may also develop gradually at any time ever weeks later
- usually it manifests as an insidious decline in level of responsiveness

III. Vasospasm
- defined as segmental or diffuse narrowing of intracerebral arteries, vasospasm is a leading cause of morbidity and mortality following SAH.
- can be detected angiographically in up to 70% of patients almost half of whom become symptomatic
- the onset of vasospasm is delayed, most commonly developing in the latter half of the first week after the initial haemorrhage and it may persist for up to 3 weeks
- the strongest predictor of vasospasm is the amount & distribution of subarachnoid blood in the initial CT scan with the greatest risk occurring in those having subarachnoid blood in the basal cisterns or layers of blood 1mm thick or greater in the cerebral fissures
- focal neurological deficits reflect the territories of the arteries involved and may fluctuate with exacerbation due to hypovolaemia or hypotension
- serial transcranial doppler has been used to screen for vasospasm with criteria for vasospasm based on absolute linear blood flow velocity [mild >120cm/sec, moderate >160cm/sec, severe >200cm/sec]. The sensitivity is about 80% when compared with angiography.

initial treatment

Initial Stabilisation:
- initial steps in evaluation of a patient with a suspected SAH should include assessment of ability to protect the airway and level of neurological function
- if the patient is lethargic or agitated, elective intubation should be considered before angiography

Routine Care & Monitoring:
- the routine monitoring of all patients with acute SAH should include serial neurological examinations, continuous ECG monitoring and frequent determinations of blood pressure (or continuous monitoring)
- because seizures increase the risk of rebleeding, anticonvulsants are indicated if seizures occur. The value of prophylaxis anticonvulsants in patients who have not had a seizure is not known
- dexamethasone is used by some to reduce meningeal irritation and intra- and post-operative oedema but there is no convincing evidence of its efficacy

(i) fluid management:
- a stable intravascular volume should be maintained by isotonic fluids
- in some patients with severe cerebral salt wasting large volumes of fluid are required to prevent intravascular volume contraction
- hyponatraemia can be treated with restriction of free water

(ii) hypertension
- initial attempts to treat hypertension should consist of analgesics and nimodipine; other antihypertensives should follow if needed

Surgical & Endovascular Treatment:
- the definitive way to prevent rebleeding is to obliterate the aneurysm by surgically clipping its neck
- the optimal timing of surgery is controversial but in patients with a good clinical grade, favourable neurological outcome is more common when surgery is performed early (within 72 hours)
- endovascular techniques using either detachable balloons or electrolytically detachable coils that thrombose the aneurysm have been used to repair acutely ruptured aneurysms
- initial experience with endovascular techniques was limited to patients who were poor surgical candidates owing to anatomical or medical considerations
- subsequent reports in which surgical considerations did not heavily influence the selection of patients, similar outcome was obtained for the two treatments
- a controversial randomized trial revealed that in patients with ruptured cerebral aneurysms, for which both endovascular coiling or surgical clipping are treatment options, the outcome in terms of disability at 1 yr is better for patients undergoing endovascular coiling

treatment of complications

(i) rebleeding
- initial management directed at the prevention of rebleeding includes avoiding situations that produce that sudden changes in the transmural pressure across the wall of aneurysm (ie sudden increases in vascular pressure or decreases in ICP). Sedation is indicated if a patient is agitated. Measures should be taken to minimise cough and Valsalva maneouvres.
- multiple clinical trials have demonstrated that antifibrinolytic agents such as aminocaproic acid and tranexamic acid reduce the risk of rebleeding but this benefit is offset by an increased incidence of vasospasm & hydrocephalus
- with the advent of early surgery and endovascular treatment, the use of these agents has declined
- definitive treatment is accomplished by obliteration of the aneurysm by coiling or clipping

(ii) hydrocephalus
- the decision to treat is usually based on the CT appearance of enlarging ventricles in a patient whose level of consciousness is deteriorating to the point of obtundation. Upon placement of a ventriculostomy, the CSF pressure is reduced slowly to lessen the risk of aneurysm rerupture
- CSF drainage via ventriculostomy may be needed for many days to determine whether a permanent shunt is required

(iii) vasospasm
a. prevention
- routine measures to prevent or reduce the effects of vasospasm include mechanical removal of subarachnoid blood at the time of surgery, administration of nimodipine, avoidance of intravascular volume contraction and hypotension

b. treatment
1. haemodynamic augmentation
- because of the disparity in the incidence of vasospasm detected angiographically, by transcranial doppler and clinically, there is disagreement about how aggressively to treat assymptomatic vasospasm when it is detected
- treatment of symptomatic vasospasm in patients with secured aneurysms begins with haemodynamic augmentation in which blood volume and cardiac output are optimised with fluids and blood pressure is increased with vasoactive agents to enhance CBF and prevent cerebral infarction. The initial step is to correct hypovolaemia; no data exist to indicate that hypervolaemia is better than euvolaemia. If there is no response to fluid then vasoactive agents are required to increase MAP although it is unclear whether augmenting volume, cardiac output or MAP is the most efficacious intervention.
- goals for intravascular volume and blood pressure should be defined as a percent change from baseline (beginning with about a 15% change) rather than aiming for prespecified levels. Although defining goals is useful for to guide therapy, the degree of haemodynamic augmentation should be titrated against the patient's neurological status; thus, if a goal is reached and there is no neurologic improvement the goal should be reassessed
- haemodynamic augmentation is weaned gradually over several days, guided by neurological status

2. endovascular treatment
- endovascular treatment of constricted vessels consists of either balloon angioplasty or intra-arterial infusion of agents such as papaverine or nicardipine. Angioplasty on the proximal segments of vasospastic cerebral vessels yields impressive angiographic changes but clear clinical efficacy has been difficult to establish because it is often used in conjunction with haemodynamic maneouvres. Papaverine produces clear vasodilation and improvement in global blood flow but the response is often transient with vasospasm returning after 24-48 hours. Multiple treatments are often required for sustained effect.
- the timing of endovascular therapy is debated but it is generally used if after a few hours the response to haemodynamic augmentation is inadequate

grading

1. Fisher scale (CT scan appearance)
group 1 - no blood detected
group 2 - diffuse deposition of subarachnoid blood without clots or layers of blood greater than 1mm
group 3 - localised clots and/or vertical layers of blood 1mm or greater in thickness
group 4 - diffuse or no subarachnoid blood but intracerebral or intraventricular clots

2. Hunt & Hess grading system / WFNS scale:
grade I - assymptomatic or mild headache / GCS 15, no motor deficit
grade 2 - moderate-severe headache, meningism and no weakness / GCS 13-14, no motor deficit
grade 3 - mild alteration in mental status with weakness / GCS 13-14, motor deficit
grade 4 - obviously depressed level of consciousness and/or hemiparesis / GCS 7-12 +/- motor deficit
grade 5 - posturing or comatose / GCS 3-6, motor deficit present or absent

prognosis

- up to 25% die before reaching the hospital
- mortality reaches as high as 40% within the first week due to complications including rebleeding, hydrocephalus, & delayed ischaemia due to vasospasm

prognosis varies with grade:
- grade I 70% survival; grade II 60% survival; grade III 50% survival; grade IV 40% survival; grade V 10% survival

predictors of poor prognosis include high grade, older age, pre-existing medical illness, blood >1mm thick on CT, seizures, cerebral oedema, basilar artery aneurysm and symptomatic vasospasm