Emergency Patient Care

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A fifty four year old lady presented to the Emergency Room on the 1St of November 1999, at 1 300 hours, with a complaint of severe headache. At 1000 hours (lie same day she suddenly developed an incapacitating. splitting left frontoparietal headache, which radiated to the occipital region and the neck. She felt nauseous and vomited thrice There was no loss of consciousness, no aura and no visual disturbance. Assuming a severe migraine attack she took Ergotamine but her headache continued to worsen. She was a patient of migraine and was on Methysergide for the last several years. For the last 3 years she was taking Atenolol for her hypertension. There was no history of epilepsy, trauma or recent exertion. She described having a similar but less intense headache about a week back but without nausea and vomiting and with neck pain, which lasted about 12 hours and responded poorly to ergotaminc. There was no history of any other drug usage.

1) What are the main differentials for her headache?[1]
a) Migraine
b) Meningitis
c) Acute CVA
d) Sub-Arachnaid Haemorrhage
e) Subdural/ Epidural bleed

2) Which of the following drugs can cause acute headaches?
a) Nitroglycerine
b) Indomethicin
c) Mcthotrexate
d) Alpha- agonists
e) Reserpine

On examination the patient was a middle aged lady sitting on the bed and holding her head with both hands. She looked irritated but was fully conscious with a OCS of 15/15 and oriented in place and person but not in time. Her pulse was 88/rn in E3P 150/100 mm Ig and temperature was 38.5°C. The resident quickly jumped to the CNS examination. The pupils were bilaterally equally reactive, fundi showed left subhaloid hemorrhage[2] with no papilledenia, no nystagmus or opthalmoplegia, no focal neurological signs (including a downgoing Babinski and normal reflexes) or neck stiffness.

Based on the history and examination presumptive diagnosis of Subarachnoid Hemorrhage (SAIl) was made.

3) Based on the history and exams, which of the following points were consistent with the diagnosis of SAIl?
a) Age
b) Sex
c) Lack of focal neurological signs
d) Absence of men ingeal irritation
e) Acute onset and incapacitating nature of headache
f) Hypertension
g) Raised body temperature
h) Left subhyaloid hemorrhage
i) Unilaterality of headache

4) What is the epidemiology of SAl-I?

5) The following are the most common etiologies of SAH. Which is the most common?

a) Aneurysms
b) AV Malformations
c) Bleeding Diathesis
d) Trauma
e) Anticoagulants
f) Miscellaneous vascular abnormalities
6) Which of the following is the most common type of aneurysm causing SAH?
a) Mycotic
b) Saccular or Berry aneurysms
c) Charcot-Bouch ard in içro neurysms
Ninety percent of Saccular aneurysms present with acute rupture ($AI-I); 7% with compression symptoms (visual field defects, opthalmoplegia, facial pain); 3% incidental on angiograms.
7) What is the common congenital abnormality seen in association with saccular aneurysms?
a) Neurofibromatosis
b) Spina Bifida
c) Adult polycystic kidney disease.
In the meanwhile she was started on IV fluids (1.5 litres daily maintenance), CBC and lytes were sent, and pulse oximetry was done. She was advised a complete bedraest in a dimly lit room. A repeat physical exam showed the development of neck stiffness with a positive Kerning\'s and E3ruzundski sign and photophobia.
8) What is the usual time course of development of neck stiffness after SAH?
Prompt arrangements were made for CT Scan and neurosurgical con su lat ion was taken.
9) Why was a MRI not done?[1]
The CTScan showed localized collection of blood in the left Sylvian fissure with no blood in the ventricular system. This was suggestive of a Middle Cerebral Artery blood. An angiogram was planned in preparation for su rgeiy.
10) 10% CT Scans are inconclusive? What are other diagnostic options?
She was then given Pethidine 75 mg intramuscularly (im) for her headache, Prochlorperazine 12.5 mg im as an antiemetic, enema was given to prevent constipation and straining which increases the chance of rebleed. Phenytoin was given orally in preparation for surgery. Atenelol was given to nornialize blood pressure.
Her angiograni was done which showed a small (<6mm) aneursni in the left proximal Middle Cerebral Artery. Surgery for aneurysm clipping was planned the following day.
She was then started on Tranexamic acid 6g/day IV bolus Q.D., Niniodipine 360mg/day P.O. in 6 divided doses and on neurological consult her atenelol was withdrawn.
11) Why were the above measures undertaken?
She had a successful microsurgery the following morning and was kept under observation for development of complications. After I week she was discharged with regular follow-ups.
The following are the most frequent complications of SAH. Intracranial Extracranial
Rebleeding
Cerebral lscheniia/ infarct
Hydrocephalus
Hematoma
Epilepsy
Myocardial Infarction
Cardiac arrhythmias
Pulmonary edema
Gastric stress ulcers
Cranial nerve palsies especially cranial nerve Ill

12) What is the prognosis of SAIl?

Answers

1) ci

2) a; b ; e

3) a: b ; c (cranial nerve palsies are common at presentation) e; f; g ; h

4) 10-15/100, 00 population with a 3:2 femalepreponderance

5) a

6) b

7) c

8) Neck stiffness develops in about 2/3rd patients over a period of 4-24 hours after the SAN episode. It lasts between 3 days and 3 weeks on the average. Maybe accompanied by back pain and radicular limb pain.

9) MRI tak.es longer to perform: it is less sensitive than a CTSCAN in the early stage of the bleed.

10) An LP can he to look for R BC count and the development of Xanthochromia (pale yellow coloration of CSF indicating Hb breakdown). If .both LP and CT are negative. SAN is excluded. If surgery is planned, an angiogram is required to localize the aneurysm.

11) Tranexamic acid (antifibrinoly tic) is given for prophylaxis of rebleeding. which occurs in 30% of patients within a month of SAH with a mortality of up to 70. Nimodipine (as a vasodilator) and the withdrawal of atenelol is done to prevent cerebral ischemia / infarct which develops in as many as 55% of patients peaking b/w the 7-12 after SAH. This is usually secondary to vasosasm. or less often due hypovolemis (which is associated with SAH) and decreased cerebral perfusion pressure due to raised IC P).[4]

12) 43% die as a result of first SAl-I. 5 year survival for SAl-I with prompt treatment is 30%.

References

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