Medicine
TEAM 437

MED437
KING SAUD UNIVERSITY

Hemorrhagic Stroke

Objectives :

1. Etiology
2. Pathophysiology
3. Clinical presentation
4. Diagnosis and imaging
5. Treatment

Done by :

Team leader: Salem Al-Ammari
Team members: -Haifaa Taleb -Alfahdah Alsaleem
-Mohammed Alasqah

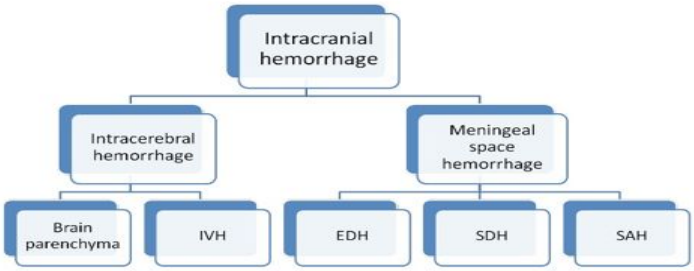
Revised by :

Aseel Badukhon

Resources :

Doctor's slides + Team 436
Lecturer: Dr. Yousef Mohammad
Same as 436 slides: Yes

Introduction



Epidemiology :

○ Asian countries have a higher incidence of intracerebral hemorrhage than other regions of the world.

○ A higher incidence of intracerebral hemorrhage has been noted in Chinese, Japanese, and other Asian populations, possibly due to environmental factors (eg, a diet rich in fish oils) and/or genetic factors.

○ Annually, more than 20,000 individuals in the United States die of intracerebral hemorrhage.

○ Intracerebral hemorrhage has a 30-day mortality rate of 44%.

○ Pontine or other brainstem intracerebral hemorrhage has a mortality rate of 75% at 24 hours. In general, If you have a hemorrhage in the brain stem it's too bad with a high mortality rate.

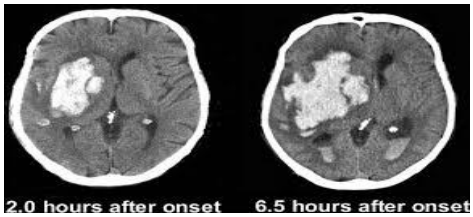
○ US strokes, approximately 87% are ischemic. and 13% are hemorrhagic.
Of those 87% that are ischemic, about 80% of them are non-cardioembolic.

○ Incidence of intracerebral hemorrhage increases in individuals older than 55 years and doubles with each decade until age 80. As long as they have many risk factors. Especially with uncontrolled or chronic HTN.

○ Ischemic has better prognosis in comparing to Hemorrhagic .

Mortality and disability :

Overall, 40% mortality at 1 month and 54% at one year.
Only 12-40% are functionally independent long term.
In 2010, 62.8 million lost daily with ICH1 compared to 39.4 million in ischemic stroke.



Risk factors :

The best method is prevention rather than treatment.

1.HTN . The most most most important risk factor.

2.Excessive ETOH2 use.

3.Smoking.

4.Obesity and physical inactivity.

5. Age . older

6. Ethnicity/Race.

7. Medications . Such as warfarin, anticoagulation drugs, and Aspirin .

8. Sympathomimetics. Like amphetamine and cocaine.

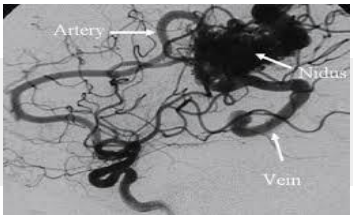
9. Fen-phen, was an anti-obesity treatment in the past which was pulled out from the market.

- A study was done here in KSA found that around 43%-45% of HTN cases are undiagnosed which is similar to other populations, and even those who are diagnosed around half of them are uncontrolled.

- 3 months ago, 3 international studies, found out that the use of aspirin as a primary prevention in people above 50 years of age with no cardiac diseases has no efficacy, rather it increases risks of hemorrhage.

Etiology :

Hypertensive ICH	-Essential -Eclampsia	
Non-hypertensive ICH	-Vascular Malformation: AVM, Aneurysm, Cavernous hemangioma causes seizures -Bleeding disorders/anticoagulant such as hemophilia	-Amyloid angiopathy elderly especially those with alzheimer -Trauma -Tumor -Drug abuse: amphetamine, cocaine, PPA
Other causes	-CVT Common in females due to OCP	-Intracranial neoplasm -Moyamoya Gradual endovascular hyperplasia (stenosis) & you will have collaterals



1-In elderly pt with Alzheimer's (deposition of amyloid in blood vessels). A 70yo coming with hemorrhagic stroke think of amyloid angiography it will show multiple hemorrhage. DON'T DO BIOPSY
2-AVM (as shown in the image) is common in younger patients.

Pathophysiology :

- **Primary immediate effect:**
  - Hemorrhage growth.
  - Increase ICP.
- **Secondary effect:**
  - Downstream effect.
  - Edema.
  - Ischemia

Site:

1. basal ganglia (40-50%).
2. lobar regions (20-50%).
3. thalamus (10-15%).
4. pons (5-12%)
5. cerebellum (5-10%).
6. other brainstem sites (1-5%) .

Clinical presentation :

Clinically you can't differentiate between ischemic and hemorrhagic stroke so you have to do CT to all patients, if CT was normal (ischemic stroke) because it take 12 hrs to show in CT. Hypodensity (don't give tPA)

- Alteration in level of consciousness (approximately 50%).
- Nausea and vomiting (approximately 40-50%).
- Headache (approximately 40%).They develop headache due to the collaterals.
- Seizures (approximately 6-7%).
- Focal neurological deficits:
  - **Putamen** - Contralateral hemiparesis (Mainly) , contralateral sensory loss, contralateral conjugate gaze paresis, homonymous hemianopia, aphasia, neglect, or apraxia.
  - **Thalamus** - Contralateral sensory loss (Mainly) , contralateral hemiparesis, gaze paresis, homonymous hemianopia, miosis, aphasia, or confusion.
  - **Lobar** - Contralateral hemiparesis or sensory loss, contralateral conjugate gaze paresis, homonymous hemianopia, abulia, aphasia, neglect, or apraxia.
  - **Caudate nucleus** - Contralateral hemiparesis, contralateral conjugate gaze paresis, or confusion.
  - **Brainstem** - crossed signs facial weakness in one side and sensory loss on the other side, eye movement problems, vertigo and coma.

*Intracerebral hemorrhage: 1- Hypertensive hemorrhage, 2- Intravenous hemorrhage.

Hypertensive hemorrhage :

The most important cause (from 1 most common to 5, in order)

- Putamen.
- Thalami.
- Pontine.

- The best management here is admit to ICU and be control

- Cerebellum.
- Lobar.

IMPORTANT :- **CVT or VST** (from Harrison's Neurology in clinical medicine)

Venous Sinus thrombosis of the lateral or sagittal sinus or of small cortical veins.

- **Occurs** mainly as a complication of **Oral contraceptives use** , and other causes like pregnancy, postpartum, IBD, meningitis, and dehydration. also seen in patients with blood disorders.
- **Patient present** with **headache** and may have a focal neurological signs like paraparesis and seizures. the patient may develop signs of Increased intracranial pressure (**like papilledema**) and coma.
- **CT is normal** . Venous sinus occlusion is readily **visualized by MRI or CT venography.**
- **Treat** by: Intravenous **heparin** regardless of the presence of intracranial hemorrhage.

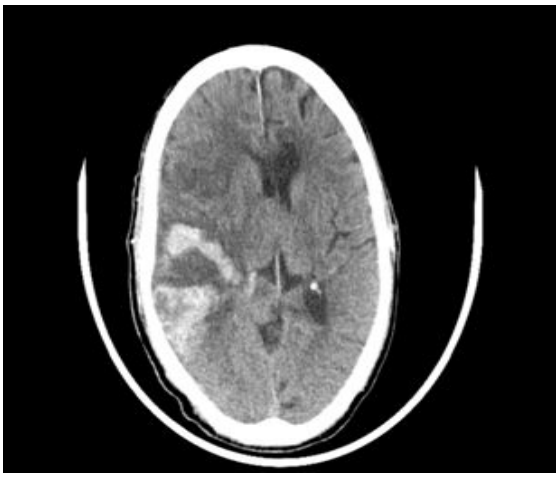
Hemorrhagic Transformation (HI 1/2 – PH 1/2) :

> 50% have some hemorrhage

- 0.6%-3% >> untreated patients.
- 6% in treated patients. **Risk Factors:**
- Older age.
- larger stroke size.
- cardioembolic stroke etiology.
- anticoagulant use

- fever.
- hyperglycemia.
- low serum cholesterol.

- Acutely elevated systolic blood pressure.
- **thrombolytic therapy/recanalization.**



Meningeal space hemorrhage: *MSH: 1- SAH , 2- SDH , 3- EDH.

Subarachnoid Hemorrhage :

- Usually due to aneurysm rupture.
- Can be perimesencephalic SAH.
- Treat by: Surgery: Coil / Clip , Medication: NIMOTOP/ NIMODIPINE , Strict BP control.
- Need to check Sodium Levels, Treat the Hyponatremia.
- Need to check Urine output.
- Treat the Hydrocephalus.
- treat the Vasospasm. with a vasodilator (Nimodipine).

investigations :

Laboratory studies:

- CBC.
- Coagulogram.
- Electrolyte.
- others.

Imaging:

- CT brain w/o contrast. Normal image doesn't exclude ischemic stroke.

CT-brain:

- Demonstrates acute hemorrhage as hyper dense signal intensity.
- Multifocal hemorrhages at the frontal, temporal, or occipital poles suggest a traumatic etiology.
- Hematoma volume can be approximated by (AxBxC)/2.
- Iodinated contrast may be injected to increase screening yield for underlying tumor or vascular malformation.

Vessel imaging:

CT angiography permits screening of large and medium-sized vessels for AVMs, vasculitis (String of beads appearance), and other arteriopathies.

The Workup:

1.CT head - no contrast.

2.CTA head/neck - suspect vascular etiology.

- Careful interpreting noncon CT head after CTA or other dye study.

3.MRI brain(it is very important and it shows you the cause of the hemorrhage)- with gado if looking for neoplasm.

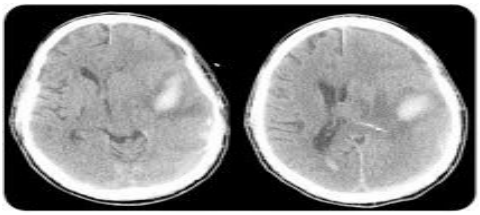
4.MRA/MRV - if allergic to CT dye or if you're looking at venous outflow.

5.Cerebral angiography. avoid it as it carries risk of stroke.

Cerebral Edema: (management)

- Use the ventilator to manage CO2.

- Get the Sodium levels up .
- Mannitol 3% or even 23.4% (requires central line). Given for brain herniation that's caused by the cerebral edema.



Treatment :

Control BP!!!!

a study, divided people with hemorrhage, to three groups, one group they lowered their blood pressure to between 180-160, second group between 160-140 and third group from 140-120. All of them have the same result at the end. So low BP does not cause ischemia .

Guidelines – reduction of SBP to 140 is safe

- Anderson/Qureshi studies – Interact 2 and ATACH 2. ○ Not clear if SBP > 220

- Use labetalol and/or nicardipine drip to titrate blood pressure. Between 15-23% of patients > hematoma expansion in first few hours. (A word about penumbra).

EBP nursing care (evidence based practice):

- Watch for neuro decline.
- Type and cross with your labs!.
- HOB > 30.

- Head midline.
- Prevent vagal maneuvers.

- Control SBP .
- Treat hyperglycemia.

- Treat hyperthermia.
- Seizure prophylaxis.
- DVT prophylaxis ● Aspiration precautions. ● Treat Infections.

-Typically, do not make patients DNR within the first 48 hours

-Surgery only for superficial lobar or cerebellar hemorrhage.

-The best treatment is always prevention

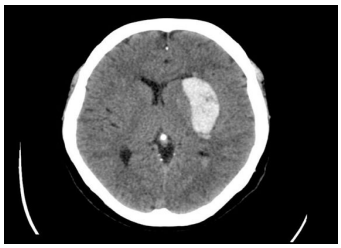
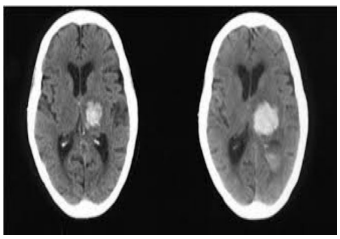
Surgery :

- EVD (external ventricular drains)
  - CLEAR III trial – no outcome benefit with vent use of tP
- Craniotomy
  - Depends on etiology.
  - Depends on (AC/APT status)11.
  - Depends on timing.
  - Depends on location

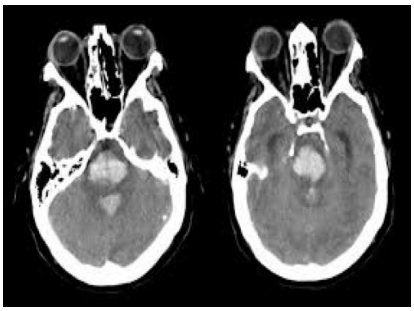
STICH II – no overall favorable outcome.
MISTIE II – MIS techniques.
MISTIE III – underway.

- Cerebellar ICH.

Imaging interpretation:

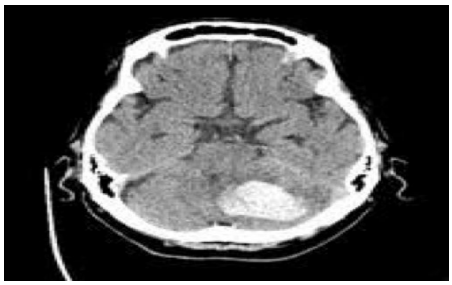
Putamen hemorrhage	Thalamic hemorrhage
	
If young patient 25 y/o come with Putamen hemorrhage, think about other causes such as (AVM, cavernous angioma or drugs like amphetamine)	The patient present with right sided loss of sensation, in general any lesion (hemorrhagic or ischemic) in thalamic will affect the sensation on the contralateral side.

Pontine hemorrhage



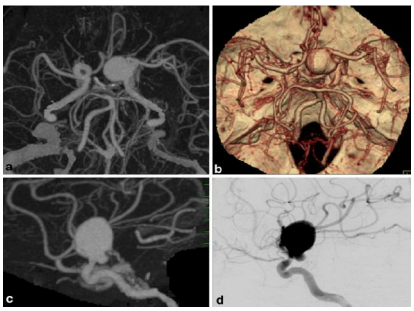
The worst prognosis --> to the grave (really really bad). Both side are affected, the patient is comatose. Quadriplegia.

Cerebellar hemorrhage



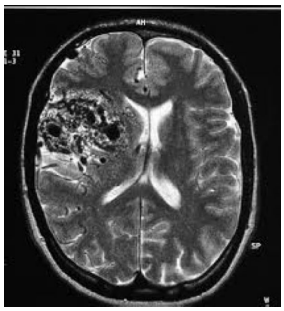
The surgery is easy with excellent prognosis. You can also remove one hemisphere of cerebella. You have to monitor the patient in ICU, it may compress the brainstem.

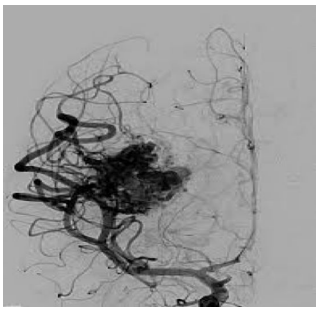
Aneurysm



It is going to rupture and patient is going to die,when patient comes with SAH it is only leaking. how do we treat it? they should undergo CT angiogram of the brain to coil it. it is done through a catheter through the groin and then they inject coil to close the aneurysm. Coiling is now the first line treatment.

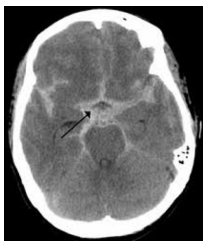
AVM

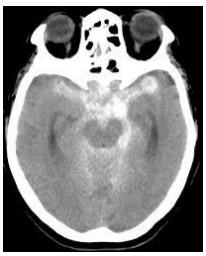




Any young patient with this typical MRI with worms like appearance. Any young patient comes with hemorrhage you have to rule out AVM. How to treat it ? First endovascular embolization to clot the vessels to prevent or reduce bleeding during surgery.

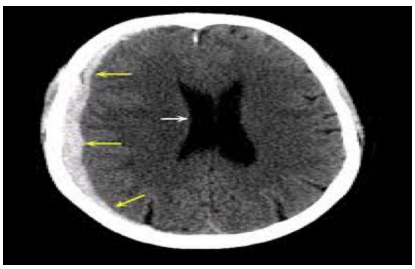
SAH





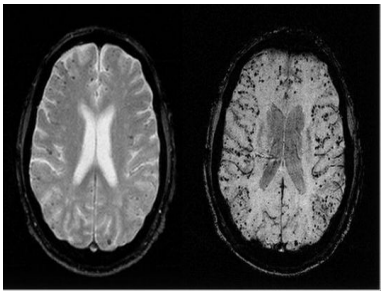
severe headache but the most important is the acuity of the pain. You should ask the patient how it occurred and what he/she was doing. Consider anything SUDDEN as SAH until proven otherwise. If you miss it, it will rupture and it has a high mortality and morbidity. How to rule it out ? First do CT scan for blood , but 10% is normal in CT. with this history you have to do spinal tap looking for blood in CSF, if Lumbar puncture also is negative we can rule it out. If it is positive you have to do CT angio to demonstrate an aneurysm which is a major cause of SAH

SDH



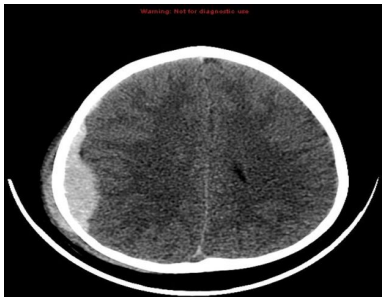
It is bleeding in meningeal veins or bridging veins, and the patient may have this without any trauma or minor trauma which may the patient forget about it. So we need a neurosurgeon to evacuate. it crosses suture lines.

Amyloid angiography



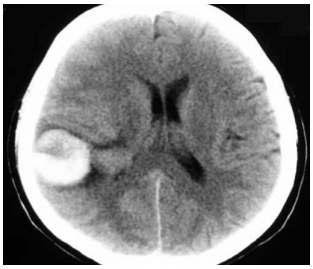
Hypertensive hemorrhage and Amyloid Angiopathy.Which is Deposition of amyloid protein on the vessel wall making it very fragile. -All these small dots are hemorrhage. - To differentiate between hypertensive and amyloid, we Do a sequence MRI in amyloid you will see multiple old and new hemorrhages this is indicative of amyloid. It means that the patient has been bleeding every now and then. - high recurrence rate. no definitive treatment, it may be associated with Alzheimer. - can't undergo surgery if you touch the vessel it will bleed. There's nothing you can do. :(

EDH



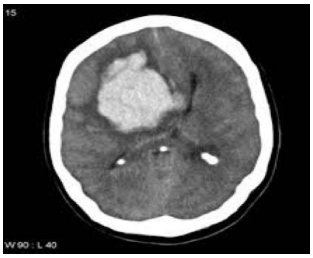
It is usually associated with trauma or fracture of the skull. It is very serious you need to watch the patient very closely, surgeons have to interfere very quickly because it's an arterial hemorrhage and they can compress and expand very quickly. It does not cross the suture line.

Tumor with ICH



We have to do an MRI to pick up tumors and know which type it is, whether a metastasis or primary. Very hyperdense then it's a hemorrhage, but if Hypodense it is a tumor.

Lobar hemorrhage



It is hemorrhage from warfarin with high INR in this case, it could be due to HTN also. It has a very bad prognosis, we can do a surgery if it is superficial.

Summary:

Hemorrhagic Stroke

high mortality rate (50% at 30 days).

Risk Factor:

HTN, Alcohol binge, Smoke, Obesity, Age, Race (Asians), Medications, Sympathomimetics (cocaine)

Causes

a. HTN (particularly a sudden increase in BP) is the most common cause
b. Ischemic stroke may convert to a hemorrhagic stroke (old, **large** stroke)
c. amyloid angiopathy (associated with Alzheimer)
d. brain tumors
e. Vascular malformation:
   a.**AVM** (young and drug free), Aneurysm, Cavernous hemangioma.
f. Bleeding disorders/**anticoagulant**.
g. Trauma.
h. Drug abuse: amphetamine, cocaine, PPA (in young always drug screen)
i. CVT (young female using OCP)
j. Moyamoya (CT-angio:"puff of smoke")

Locations: Basal ganglia > lobar regions > thalamus > Pons > cerebellum

But in ★ Hypertensive hemorrhage: **Putamen >** Thalami > Pontine > Cerebellum > Lobar

Invest:

CBC, Coagulogram, Electrolyte
CT w/o contrast (will show Hyper density)
CT-angio (to look for the cause: aneurysm, AVM)
MRV (CVT)

Ischemic VS Hemorrhagic: Only by CT
Ischemic 12-24 changes will appear
Hemorrhagic immediately

Management:

1. Admit to ICU, Control BP, Watch for neuro decline, DVT prophylaxis
   Cerebral Edema Management: Ventilator, Sodium, Mannitol
2. Surgery : only for cerebellar hemorrhage ~

Subarachnoid Hemorrhage

Berry aneurysm (associated with PCKD)

Complications:

-Vasospasm (prevent by nimodipine for 21 days)
-Hydrocephalus
-Hyponatremia (put on hypertonic Saline)

Treatment: Intervention and inject coil in the aneurysm

*~Pontine hemorrhage lead to locked in syndrome (aware but can only blink)

Questions

A 35-year-old previously healthy woman suddenly develops a severe headache while lifting weights. A minute later she has transient loss of consciousness. She awakes with vomiting and a continued headache. She describes the headache as “the worst headache of my life.” She appears uncomfortable and vomits during the physical examination. Blood pressure is 140/85, pulse rate is 100/min, respirations are 18/min, and temperature is 36.8°C (98.2°F). There is neck stiffness.

Physical examination, including careful cranial nerve and deep tendon reflex testing, is otherwise normal. Which of the following is the best next step in evaluation?

a. CT scan without contrast

b. CT scan with contrast

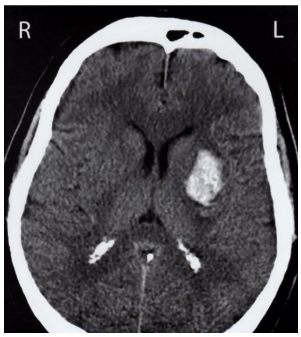
c. Cerebral angiogram

d. Holter monitor

e. Lumbar puncture

The answer is a. An excruciating headache with syncope requires evaluation for subarachnoid hemorrhage (SAH). This occurs with leakage or rupture of an intracranial aneurysm, usually located at an arterial bifurcation in the anterior cerebral circulation. Rupture may occur spontaneously or at times of exercise. About 2% of persons have “berry” aneurysms. Fortunately only a small percentage of these persons ever experience rupture, which may be fatal. The headache that precedes or accompanies SAH is severe and often described as a “thunderclap” headache, meaning that it reaches its maximum intensity in seconds. Migraine may also cause severe headache, but usually reaches maximum intensity in 5 to 30 minutes. Syncope occurs in about one-half of patients with SAH and is thought to be due to accompanying cerebral artery spasm. Blood in the cerebrospinal fluid irritates the meninges and may cause neck stiffness. Suspected subarachnoid hemorrhage mandates CT scanning as the initial test. In about 90% of patients, there will be enough blood to be visualized on a noncontrast CT scan. A contrast CT scan sometimes obscures the diagnosis because, in an enhanced scan, normal arteries may be mistaken for subarachnoid blood. If the CT scan is normal, a lumbar puncture will establish the diagnosis by demonstrating blood in the cerebrospinal fluid (CSF). As opposed to CSF blood from a traumatic lumbar puncture, the CSF blood does not clear with continued collection of fluid. Cerebral angiography is necessary to assess the need for surgery and to detect other aneurysms, but it is usually delayed because angiography may precipitate spasm, especially if performed immediately after the acute rupture. Holter monitor might be helpful in unexplained syncope but would not address the severe headache. Electroencephalography is sometimes used to diagnose seizures in a patient with unwitnessed and unexplained syncope, but would not be appropriate until subarachnoid hemorrhage has been excluded.

One day after undergoing a left carotid endarterectomy, a 63-year-old man has a severe headache. He describes it as 9 out of 10 in intensity. He has nausea. He had 80% stenosis in the left carotid artery and received heparin prior to the surgery. He has a history of 2 transient ischemic attacks, 2 and 4 months ago. He has hypertension, type 2 diabetes mellitus, and hypercholesterolemia. He has smoked one pack of cigarettes daily for 40 years. He drinks 1–2 beers on weekends. Current medications include lisinopril, metformin, sitagliptin, and aspirin. His temperature is 37.3°C (99.1°F), pulse is 111/min, and blood pressure is 180/110 mm Hg. He is confused and oriented only to person. Examination shows pupils that react sluggishly to light. There is a right facial droop. Muscle strength is decreased in the right upper and lower extremities. Deep tendon reflexes are 3+ on the right. There is a left cervical surgical incision that shows no erythema or discharge. Cardiac examination shows no abnormalities. A complete blood count and serum concentrations of creatinine, electrolytes, and glucose are within the reference range. A CT scan of the head is shown. Which of the following is the strongest predisposing factor for this patient's condition?



A) Degree of stenosis
B) Use of asprin
C) Perioperative heparin
D) Hypertension
E) Smoking

Anser is D

A 50-year-old man is brought to the emergency department because of severe headache over the past hour. He also reports nausea and one episode of non-bloody vomiting. He has a history of hypertension and type 2 diabetes mellitus. He does not smoke or drink alcohol. Medications include enalapril and metformin, but he states that he does not take his medications on a regular basis. His temperature is 37°C (98.6°F), pulse is 80/min, and blood pressure is 190/110 mm Hg. He is oriented to person but not place or time. Physical examination shows decreased muscle strength in the right leg and arm. Deep tendon reflexes are 3+ in the right upper and lower extremities. A noncontrast CT scan of the head shows a solitary hyperdense lesion surrounded by hypodense edema in the left cerebral hemisphere. Which of the following is the most likely underlying cause of this patient's symptoms?

A. Intracranial neoplastic cell growth
B. rupture of the small penetrating artery
C. rupture of bridging veins
D. rupture of saccular aneurysm
E. rupture of middle meningeal artery
F. rupture of an arteriovenous malformation

Answer is B