Emergency Bulletin
Hong Kong College of Emergency Medicine

Challenging subtle CT brain finding in a patient with syncope

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This is a 44-year-old lady who attended the emergency department for syncope with subsequent minor head injury that happened on same day. She enjoyed unremarkable past health except asthma. There was no neurological deficit. She also had another episode of transient dizziness a few days ago which did not lead to injury.

Upon consultation, she was fully conscious and ambulatory. Blood pressure and pulse were within normal limits. Chest and neurological exam were unremarkable. ECG showed sinus rhythm and was unremarkable. Point-of-care capillary glucose was 4.0 mmol/L. Laboratory tests including complete blood count, liver function test, renal function test, cardiac enzymes and high-sensitivity troponin I were also unremarkable.

A plain computed tomography of brain (CT brain) was obtained (Figure 1a) and was commented to be unremarkable.

Routine reporting of CT brain by radiologist for cases with head injury was not yet available by that time. Patient was discharged with symptomatic treatment.

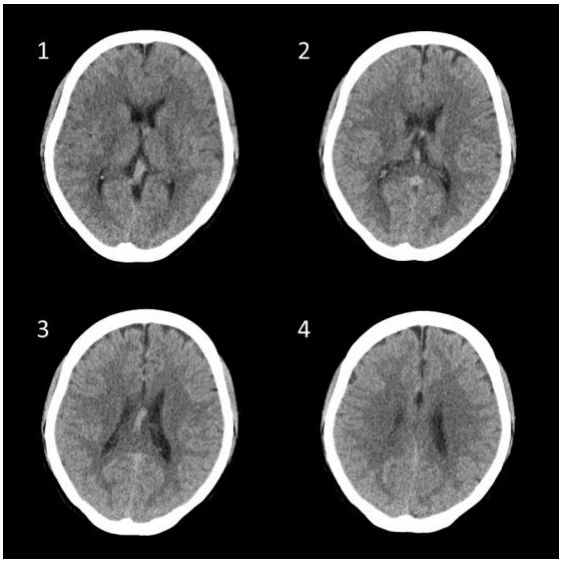

Figure 1a: CT brain images obtained in the first consultation

How to interpret CT brain?

There is a useful systematic approach for interpreting a plain CT brain with an easy-to-remember mnemonic: "Blood Can Be Very Bad and EENT". It includes the following items:

- *Blood*: intra-axial haemorrhage (intraparyenchymal, intraventricular) or extra-axial haemorrhage (subarachnoid, epidural, subdural); intradural blood clot indicating cerebral venous thrombosis
- *Cisterns*: blood in cisterns, or effacement / compression / obliteration of cisterns
- *Brain*: hyper- or hypodensity, grey-white matter differentiation, shift or asymmetry, or sulcal effacement / obliteration

- *Ventricles*: blood, hydrocephalus or effacement; displacement of ventricles indicating midline shift
- *Bone*: skull fractures or other bone lesions; occasionally may see C1 fracture that is frequently missed
- *Eye*: orbital fracture, proptosis, lens dislocation, retrobulbar haematoma
- *ENT*: ear canal especially temporal bone fracture, mastoid air cells; paranasal sinuses, air-fluid levels, nasal bone fracture, tripod fracture, nasopharyngeal mass

The CT brain actually showed a slightly hyperdense structure (35-45 Hounsfield Unit) draining to a prominent internal cerebral vein near the foramen of Monro (Figure 1b). The finding was subtle and missed at the initial assessment.

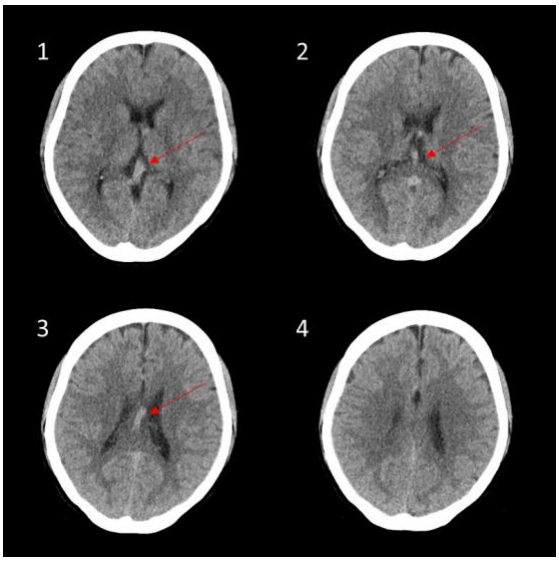

Figure 1b: hyperdense lesion (35-45 Hounsfield Unit) pointed by red arrows

Progress of the patient

On the next day, the patient was called back for an abnormal CT brain report. Another CT brain was repeated and showed similar findings. Admission was suggested but patient requested discharge against medical advice.

She was brought in 12 days after the 1st A&E attendance, for confusion and abnormal behaviour. She complained of headache with vomiting. This time CT brain (Figure 2) showed right thalamic haemorrhage with bilateral intraventricular haemorrhage and hydrocephalus. She was then admitted to the neurosurgery ward.

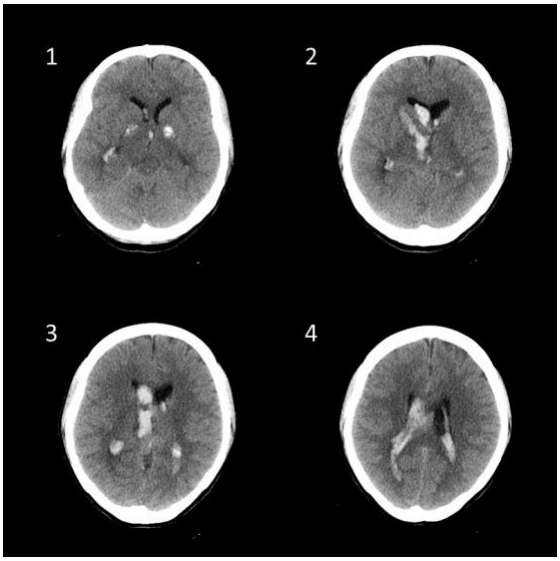

Figure 2: CT brain at the third A&E attendance showing right thalamic haemorrhage with bilateral intraventricular haemorrhage and hydrocephalus

The subsequent cerebral CT angiogram (CTA) (Figure 3), digital subtraction angiography (DSA) (Figure 4) and magnetic resonance imaging angiography (MRI+MRA) confirmed the presence of arteriovenous malformation in right basal ganglia, right thalamus and right choroid plexus draining into internal cerebral vein. Stereotactic radiosurgery was offered but patient requested transferal to a private centre. Upon follow-up at 2 months, she displayed no focal neurology. No surgery was done, and patient recovered with conservative management. The follow-up MRI+MRA was arranged at 7 months later.

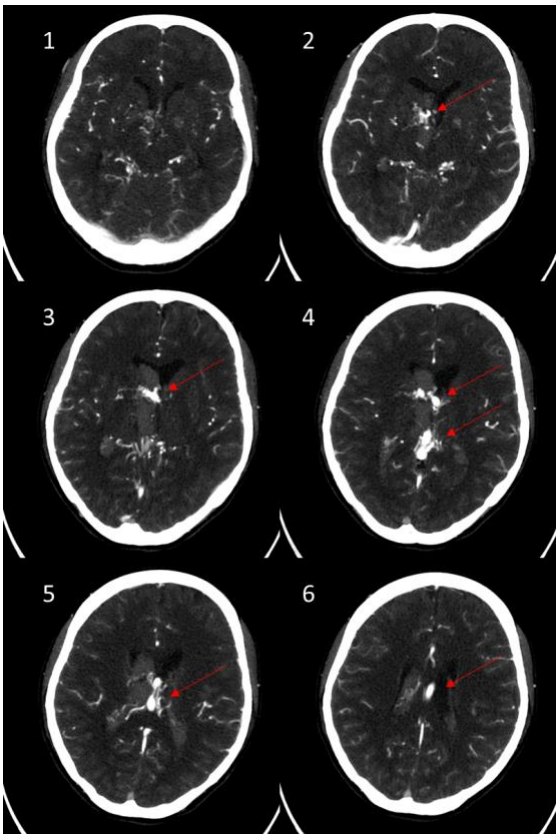

Figure 3: CTA arterial contrast phase; the nidus of AVM is pointed by red arrows

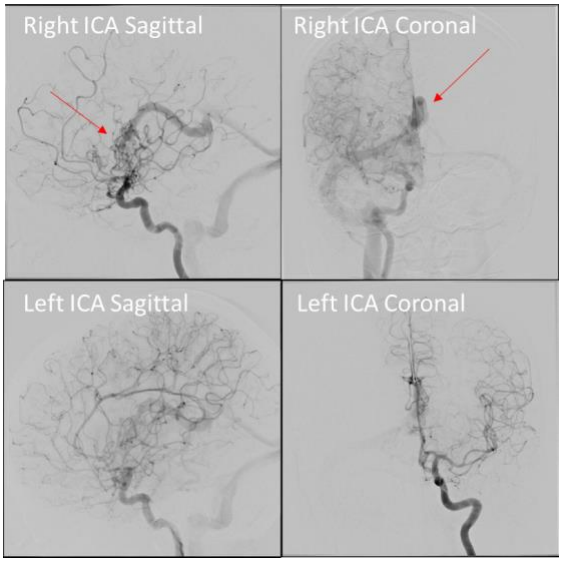

Figure 4: DSA images; nidus of AVM is pointed by red arrows

Central nervous system vascular malformation

Central nervous system vascular malformations can be classified by haemodynamic, components, and location (Table 1).[1,2] Brain arteriovenous malformations (AVM) and cerebral cavernous malformation (CCM) are the most found with detection rate reported to be 1.4 and up to 0.56 per 100 000 adults per year, respectively. The clinical presentation can vary from incidental finding in asymptomatic person to headache, seizure, or even intracranial haemorrhage.

Components	Pathology	Haemodynamics
Arterial	Arteriovenous malformations (AVM)	High flow
	Dural arteriovenous fistula (dAVF)	High flow
	Pial arteriovenous fistula (pAVF)	High flow
	Cerebral proliferative angiopathy	High flow
	Carotid cavernous fistula (CCF)	High flow
Venous	Cerebral cavernous malformation	Low flow
	Developmental venous anomalies	Low flow
	Sinus peri-crani	Low flow
	Cerebral varix	Low flow
Capillary	Capillary telangiectasia	Low flow

Table 1: Types of central nervous system vascular malformations; locations are not mentioned for simplification

The pathogenesis of brain AVM (also known as cerebral AVM) is unclear but thought to be congenital. The key feature is the lack of capillary bed in an abnormal tangle of vessels where arteries directly drain into veins, constituting a vascular shunt. It is comprised of 3 parts, the feeding arteries, a nidus, and the draining veins (Figure 5). The nidus may be seen in scan as diffuse or more commonly compact "glomerular". Brain AVM is equally seen in both genders and mostly affects patients of 20 to 40 years of age. There were post-mortem data showing that approximately 10% of brain AVM would become symptomatic during life. Among those symptomatic, up to 50% of patients

presented as intracranial haemorrhage, followed by seizures, headache, and focal neurologic deficits. Spontaneous intracerebral haemorrhage is more common than subarachnoid haemorrhage (SAH) or intraventricular haemorrhage (IVH). Brain AVM is related to 2% of all haemorrhagic stroke and is the leading cause of spontaneous intracerebral haemorrhage in patients younger than 35 years old.[1]

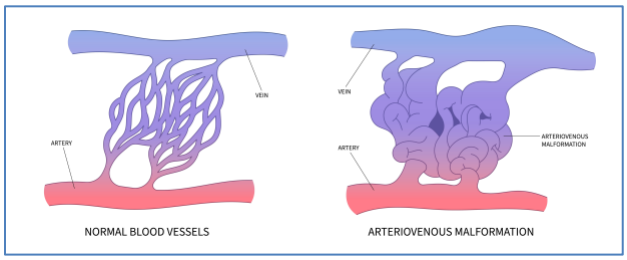

Figure 5: The appearance of an AVM. (Pepermpron/Shutterstock.com)

Brain AVM is difficult to diagnose with plain CT brain. The nidus is of blood density and usually appears hyperdense compared to adjacent brain. Dilated draining veins may also be seen.[3] CT findings may be subtle, as shown in the CT brain images of another patient (Figure 6).

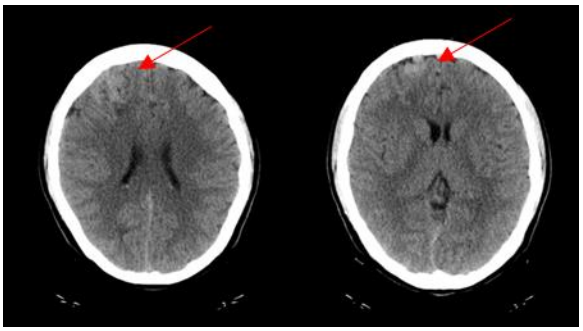

Figure 6: Plain CT brain of another patient with brain AVM at right frontal lobe, pointed by red arrows

Digital subtraction angiography (DSA) is the gold standard as it shows the detailed dynamic architecture and accurate sizing of nidus; but DSA is invasive in nature and carries definite though low risk of complications. Computed tomography angiography (CTA) or magnetic resonance angiography (MRA) are safe available alternatives if central nervous system vascular malformations is suspected.[15, 16]

Spontaneous intracerebral haemorrhage

Typical spontaneous intracranial haemorrhage, usually regarded as haemorrhagic stroke, occurs at deep brain structure commonly, such as basal ganglia, thalamus, pons and deep portions of the cerebellum.[4] Lobar haemorrhage can be due to chronic hypertension but is more associated with anticoagulant use and underlying vascular malformation.[5]

As emergency physicians, we have an important role to communicate with neurosurgeons, the acute stroke team or even the intensive care unit. Intracerebral Haemorrhage Score (ICH score) is recommended by AHA to achieve effective communication among teams. It consists of 5 parts, the Glasgow Coma Scale, the patient's age, the presence of infratentorial haemorrhage, the presence of intraventricular haemorrhage, and clot volume (Table 2).[6]

Component	Points
GCS	
3-4	2
5-12	1
13-15	0
ICH volume (cm^3)	
≥ 30	1
<30	0
Intraventricular haemorrhage	
Yes	1
No	0
Infratentorial origin of ICH	
Yes	1
No	0
Age (year)	
≥ 80	1
<80	0
Total Score	0-6

Table 2: The ICH Score with 5 components as independent predictors of 30-day mortality

Clot volume can be best estimated by using ABC/2 method, by calculating the product of the largest diameters (A & B) (in cm) and vertical height (C) (in cm) and divided by 2.[7] Most of the time, the CT scan is 5 mm cut in Hong Kong Hospital Authority Setting, so C is usually a product of number of slices and 0.5 cm by default. For illustration, there is another patient found intracerebral haemorrhage over right external capsule (Figure 7). The largest diameter is 30 mm and 15 mm at image 14. Its vertical height is 35 mm (span from image 10 to 17; 7 x 5 mm = 35 mm). The haematoma size is estimated to be 3 x 1.5 x 3.5 /2 = 7.875 ml.

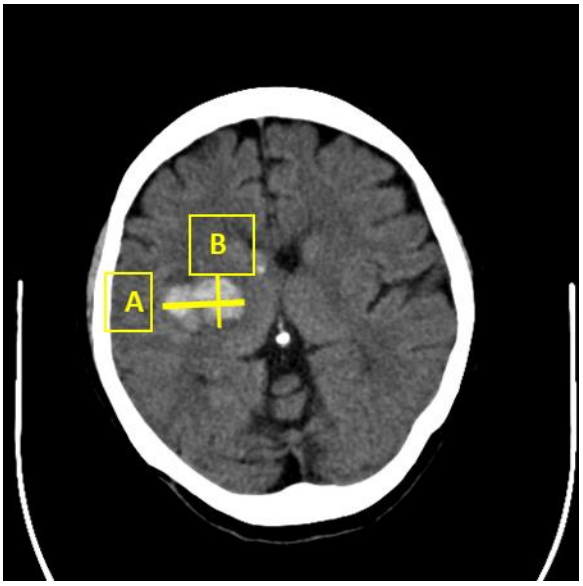

Figure 7: Illustration of ABC/2 method. A = 30 mm; B = 15 mm

With regards of its primary use in facilitating discussion in clinical severity, the initial ICH score can also provide prognostic value by predicting the risk of early death and death at 12 months and functional outcome. However, since the initial ICH score can be drastically affected by initial treatment, it should not be used as the primary means to decide for DNAR (Do Not Attempt Resuscitation) or withdrawal of medical treatment. There was an update in 2022 AHA guideline regarding this issue, stating that "aggressive care is reasonable to decrease mortality and improve functional outcome until at least the second full day of hospitalisation".[8]

The aim of treatment for spontaneous intracerebral haemorrhage focuses on preventing secondary brain damage, which may arise from complications including haematoma expansion, intraventricular haemorrhage, and mass effect.

Blood pressure control is believed to be beneficial in preventing haematoma expansion, yet the ideal target remains uncertain. Drawing conclusions from ATACH-2 and INTERACT2, AHA has recommended that continuous smooth and sustained blood-pressure-lowering therapy targeting systolic blood pressure (SBP) to a range of 130 to 140 mmHg is safe and may be reasonable in improving functional outcome, especially when patient presenting systolic blood pressure is between 150 and 220 mmHg. Nonetheless, acute lowering of SBP to less than 130 mm Hg is potentially harmful.[8]

For patients taking anticoagulants, reversal should be given as soon as possible according to the agents respectively, as poor outcome was reported to be as high as 6 times to those without anticoagulation.[9] Antiplatelet is another common antithrombotic prescribed besides anticoagulant. However, antiplatelet reversal by platelet transfusion is proven to be harmful in spontaneous intracerebral haemorrhage in comparison to conservative care in PATCH phase 3 in 2016.[10] This finding was also echoed in traumatic intracerebral haemorrage.[19,20] There has been no clear reason found but it is proposed that the harm of transfusion-associated risks outweighs the benefit. Platelet transfusion should be only reserved for patients with bleeding risk during neurosurgical procedures.[10]

In contrast to traumatic brain injury in CRASH3 trial, the clinical effectiveness of tranexamic acid is not well established in spontaneous cerebral haemorrhage, but it is safe to use with no increase in venous thromboembolism.[11] There is an ongoing trial on prescribing factor VIIa in order to limit expansion of haematoma

but there has been no significant difference in favourable patient outcome.[12]

Other general measures including euglycaemia, prevention of aspiration, thromboprophylaxis, maintaining euthermia, should be taken. Prophylactic antiepileptic drug is of unclear benefit. Intensive care with continuous electroencephalographic (EEG) monitoring and prompt treatment of seizure are reasonable.[8] Glucocorticoids are found to be of no clear benefit with potential risk in large scale studies.[17,18]

There is a common misunderstanding among emergency physicians that all intracerebral haemorrhage benefits from neurosurgery assessment and require urgent consultation of neurosurgeon.

The landmark study, Surgical Trial in Intracerebral Hemorrhage II (STICH II) trial, has found that early clot removal is not shown to have better outcome in comparison to conservative management, unless in certain circumstances.

Those who may benefit in haematoma evacuation are those with larger than 20 to 30 ml clot volume in moderate severity (GCS 5-12), lobar clots located within 1 cm of the cortical surface, and brainstem compression.[13] Despite lack of evidence, haematoma evacuation is often recommended in spontaneous cerebellar haemorrhage with lower threshold of clot volume larger than 15 ml.[14] Patients with hydrocephalus may also be benefited from placement of an external ventricular drain.

Learning objectives

1. Identify spontaneous intracerebral haemorrhage as one of the sinister causes of syncope
2. Analyse the plain CT brain in a systematic approach
3. Able to name the types of central nervous system vascular malformations
4. Choose the appropriate investigations for delineating central nervous system vascular malformation
5. Calculate the haematoma volume in imaging
6. Manage spontaneous intracerebral haemorrhage

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