International Journal of Radiology Research
www.radiologyjournal.in
E-ISSN: 2663-4562, P-ISSN: 2663-4554
Received: 20-07-2020; Accepted: 05-08-2020; Published: 24-08-2020
Volume 2; Issue 2; 2020; Page No. 04-11

Atypical imaging findings in radiological evaluation of non-traumatic intracranial hemorrhage

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Abstract
Introduction: The aetiology of non-traumatic intracranial haemorrhage ranges from the common ones like haemorrhagic stroke and aneurysmal rupture to the rare, but nonetheless important causes like haemorrhagic neoplasm and metastasis. In such a wide range of pathologies, imaging findings are very essential in making the diagnosis and thus deciding the course of management to prevent further complications. The objective of our study is to evaluate the role of CT in early diagnosis and establishing the importance of MRI in further characterization across this wide spectrum of presentation in non-traumatic parenchymal haemorrhage.
Methods: We retrospectively reviewed cases of non-traumatic cerebral hemorrhage in 20 patients referred for CT and MRI study to our department with proven or almost certain aetiologies for the hemorrhage.
We evaluated the clinical presentation and aetiology of the hemorrhage based on multimodality imaging findings.
Results: In our sample of 20 cases, the most common cause of non-traumatic haemorrhage was found to be Hypertension, followed by other important causes like Aneurysmal rupture, AV malformation and old infarcts with haemorrhagic transformation, cerebral amyloid angiopathy and cavernoma.

Keywords: non-traumatic, parenchymal hemorrhage, hemorrhagic neoplasm

Introduction

The common causes of non-traumatic intracranial haemorrhage include haemorrhagic stroke and aneurysmal rupture. Rare causes also include haemorrhagic neoplasm and haemorrhagic metastasis. In suspected cases of non-traumatic intracranial hematoma, a rapid assessment and imaging modality is required to determine the cause of bleed, to alleviate the clinical symptoms and to prevent life threatening complications. Although the age, comorbidities and onset of symptoms may provide a clue to the diagnosis, imaging plays a definitive in role in further management. The aim of our study is to exhibit the imaging findings in pathologies which can present as intracerebral hemorrhage. Considering the atypical and typical presentations of intracranial hemorrhage, we need to individualize the primary causes from secondary ones and this requires MRI for confirmation and further assessment after using CT as the primary modality for diagnosis, thus this study meanwhile aims to validate the role of MRI in customizing the management.

2. Material and Methods

This is a descriptive study of retrospectively reviewed cases of non-traumatic cerebral hemorrhage in 20 patients with proven or almost certain aetiologies for the hemorrhage who were referred for CT and MRI study to Department of Radio-diagnosis in our hospital during the period of June 2019 to June 2020. All our patients were imaged in our institution. CT scans were done using a Siemens SOMATOM SCOPE 32 slice CT scanner, whereas MRI was done using Siemens 1.5 T Magnetom Essenza MRI scanner. We evaluated the clinical presentation and aetiology of the hemorrhage based on multimodality imaging findings.

3. Results

In our sample of 20 cases, the most common cause of non-traumatic haemorrhage was found to be Hypertension - 35%, followed by Aneurysm rupture – 20%, AV malformation and old infarcts with haemorrhagic transformation – 15% each, cerebral amyloid angiopathy -10% and cavernoma – 5%. Sudden onset focal neurologic deficit was the most common presentation (n=20). In 11 cases headache with nausea and vomiting was the first symptom and in 7 cases, change in the level of consciousness was clinical presentation. 1 patient presented with seizures.

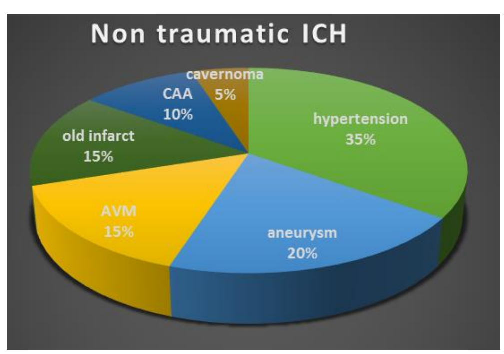

Fig 1: Pie Chart showing distribution of various causes of non-traumatic intracranial haemorrhage in our study

4. Discussion

The occurrence of ICH is roughly 25 per 100,000 person-years, and it has a death rate of 40% when calculated in a time period of one month of presentation [1] The most common cause of Intra cranial hemorrhage is trauma and Non-contrast CT of the head is the preliminary examination done to estimate the degree of acute brain injury due to trauma [2]. The other common causes of intracerebral haemorrhage are haemorrhagic stroke and subarachnoid haemorrhage due to a ruptured aneurysm. Clinical features are usually sudden onset of focal neurologic deficit, headache, altered level of consciousness, seizure, nausea and vomiting.

In neuroimaging, Plain CT i.e. Non-contrast computed tomography (CT) of the head is first line of investigation in view of its comparative safety, wide accessibility, less time vital to perform the investigation along with high sensitivity to acute ICH.

Dual energy CT is highly promising in diagnosis of haemorrhagic distribution however its usage is still in investigational phase [3].

Acute SAH causes enhanced density of the cerebrospinal fluid (CSF) spaces on CT (if there is satisfactory rise of the CSF haematocrit). CT angiography (CTA) and CT perfusion (CTP) can also be done to assist the plain CT in evaluation of acute ICH.

If undistinguishable findings on CT or in case of spontaneous ICH, MR is better modality in finding the cause of haemorrhage since CT is most sensitive to acute bleeding while MR is sensitive to both acute and chronic blood products because their presentation is more complex on MR relative to CT.

CT shows acute haemorrhage as a hyper density which over a period shows slow reduction in density over days to weeks depending on the size of the hematoma.

An increase of more than one third is noted in volume of intraparenchymal hematomas in at least 38 % of patients who were imaged initially in their clinical course [4].

Therefore, follow up is also recommended to observe the progression of hematoma using the various imaging modalities and MRI is especially helpful in assessing the age of hematoma.

Instead, in MRI as the blood products start changing from oxy-haemoglobin to ultimately ferritin and hemosiderin, the appearance of the hematoma changes on T1- and T2-weighted images. MR (often with MR angiography [MRA], MR venography [MRV], and/or MR perfusion) is usually performed following CT to assess the probabale cause of haemorrhage.

In case of haemorrhagic transformation of acute ischemic stroke, diffusion weighted (DW) MRI is the sequence of choice for swift and precise diagnosis can be made.

MR with gradient-recalled echo (GRE) imaging is sensitive to acute haemorrhage. The MR imaging protocol should imperatively comprise a T2*-weighted sequence because Hypertension associated hemorrhage has common presentation as deep grey nuclei and brainstem microbleeds [5].

Classification of haemorrhages

1. **Intra-axial haemorrhage:** Intracerebral haemorrhage - Basal ganglia haemorrhage, Lobar haemorrhage, Pontine haemorrhage, Cerebellar haemorrhage
2. **Extra-axial hemorrhage include:** Intracranial extra cerebral–Extradural, Subdural, Subarachnoid, Intra ventricular hemorrhages

The underlying aetiology in a patient with an intra cerebral haemorrhage is very broad. It includes:

1. Vascular malformations [arteriovenous malformation (AVM), Dural arteriovenous fistulas (d AVF), aneurysms, caver noma]
2. Haemorrhagic primary brain tumours or metastases
3. Infarction with haemorrhagic transformation
4. Haemorrhagic venous infarction in sinus thrombosis
5. PRESS
6. Drug abuse
7. Reversible cerebral vasoconstriction syndrome

On Basis of Location

In non-traumatic haemorrhage, knowing the location of a bleed is often important to determine the cause. The various locations of a non-traumatic intracranial haemorrhage can be anywhere from basal ganglia, caudate nucleus, thalamus, ventricles or subarachnoid spaces.

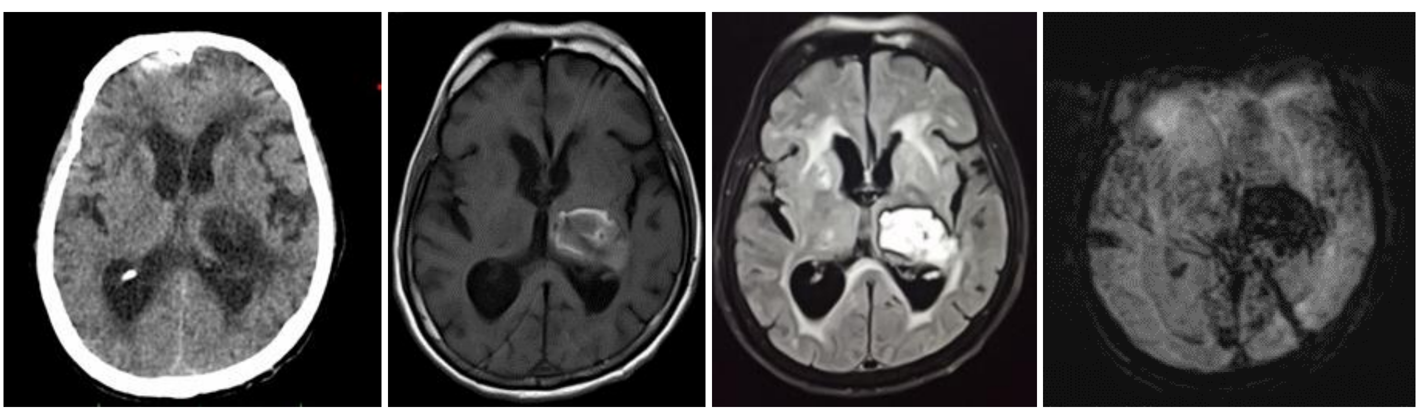

Fig 2: Hemorrhage in the left thalamus in a known hypertensive patient. Image (A) Axial CT without contrast - hypodense area in the left thalamic region with a surrounding near CSF density region. (B) Axial T1W image – the area appears isodense with a surrounding peripheral hyperintense rim. (C) Axial T2W image – same area appears predominantly hyperintense with thin hypointense rim (D) SWI shows blooming in the left thalamic region. Some motion artefacts are seen in the surrounding regions.

Lobar hematoma: Usually located in the periphery of a lobe. It is seen in cerebral amyloid angiopathy. Although, it can also be seen in hypertension, tumour, vascular malformation, venous infarction and many other diseases.

On Basis of Etiology

Hypertensive Hemorrhage

Hypertensive Intraparenchymal hemorrhage (IPH) usually has a mortality rate of 30-50% and the affected age group is sixth and seventh decades [6]. Most common sites of hypertensive haemorrhage include Putamen - External capsule, Thalamus, Pons, Cerebellum, Lobar white matter with or without intraventricular extension.

Cerebral Amyloid Angiopathy (CAA)

Cerebral amyloid angiopathy is a condition which is characterized by deposition of amyloid in areas like tunica media and/or tunica adventitia of small and medium-sized arteries in specific locations of cerebral cortex and leptomeninges.

The deposits of amyloid in the walls of the small leptomeningeal and cortical arteries make them prone for rupture and hemorrhage [7, 8]. Boston criteria is used for diagnosis of CAA associated SAH which is based on the distribution and number of microbleeds and intraparenchymal haemorrhages. However, a brain biopsy is required for a definitive diagnosis [9]. On CT it appears initially hyperdense with hypodense surrounding oedema, often exerts positive mass-effect whereas MRI presentation may vary according to age of bleed. Cerebral micro hemorrhage appear as round or ovoid 2-10 mm areas of cortico-subcortical (grey-white matter junction) hemorrhage, T2* sequences (GRE, echo-planar, SWI) depict them as blooming artifact, while it is not imaged on CT, T1 and T2/FLAIR sequences. This bilaterally symmetrical distribution of multiple small foci of blooming in white matter on GRE or SWI sequences is highly suggestive of CAA [10, 11].

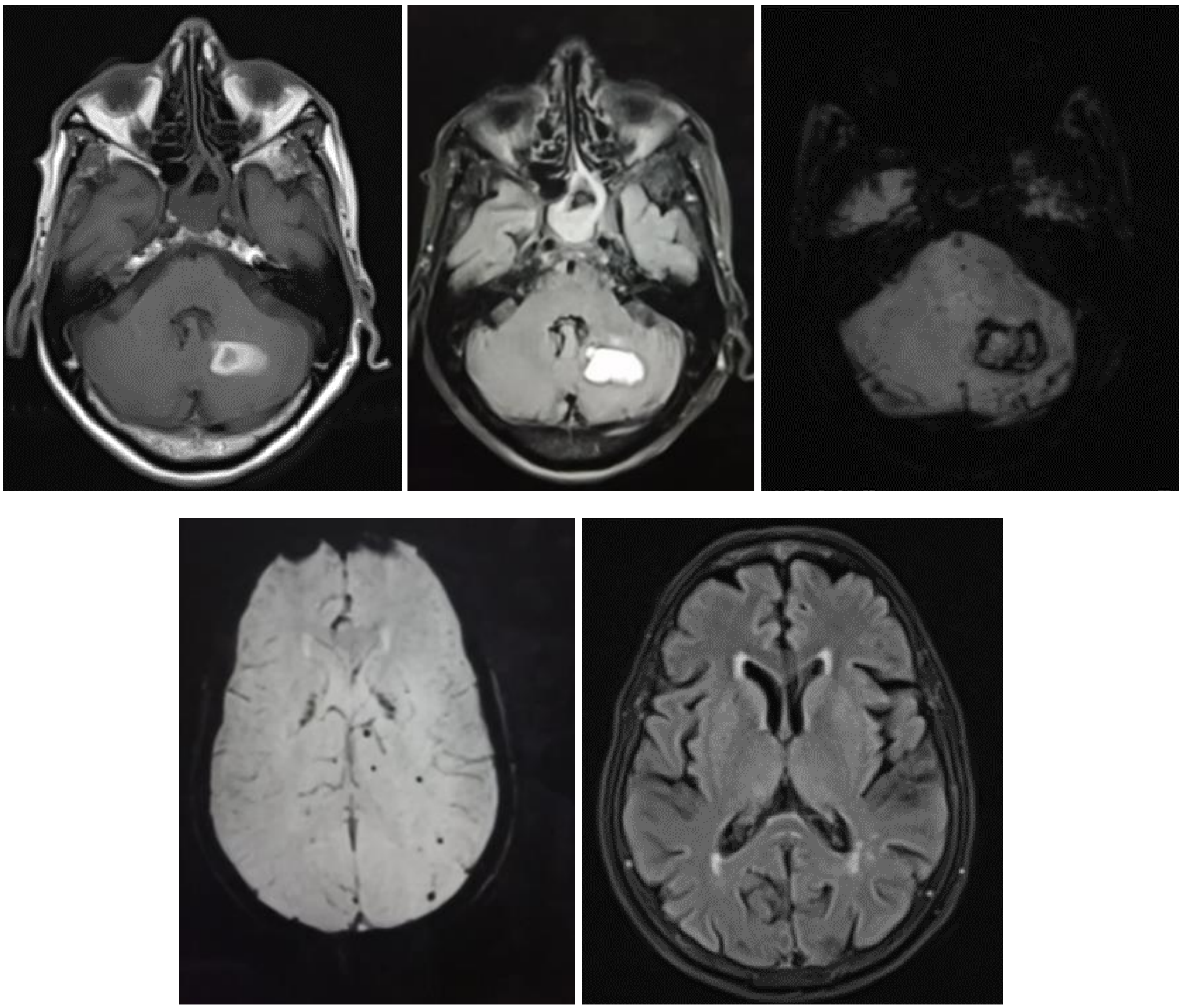

Fig 3: Cerebral Amyloid Angiopathy (CAA) (A) – Axial T1W image shows rounded isointense area with peripheral hyperintense rim in the left medial cerebellum which appears hyperintense in FLAIR Images (B) and with heterogenous areas of blooming in SWI sequences (C) SWI sequences at a higher level (D) showed multiple punctate areas of blooming suggestive of microbleeds seen in CAA, which are not seen in FLAIR images (E) at the same level.

Subarachnoid Hemorrhage

Subarachnoid: Subarachnoid hemorrhage (SAH) is presence of blood in the subarachnoid space which is a potential space between the arachnoid and pia mater. The most common cause for SAH is trauma whereas non-traumatic SAH is usually the consequence of rupture of aneurysm causing spread of blood into the cisterns. Post symptom period, in the first 6-24 hrs plain CT of Head has approximately 100 % sensitivity for detecting acute SAH [12]. The weakening of the wall of arteries result in focal outpouchings and in case of cerebral arteries, they are called cerebral aneurysms. The usual presentation is the outcome of sub-arachnoid hemorrhage and resulting dural irritation i.e. sudden onset of headache which patient describes as worst headache of life [13]. DSA with 3 D rotational angiography is strongly recommended, in case CTA comes negative but symptoms are highly suspicious of SAH due to aneurysm [14].

On CT: Shows Increased density in cisterns and along sulcal spaces. On MRI: It has a lower sensitivity relative to CT for detecting a SAH, especially in the acute phase. T2*gradient echo and FLAIR are extremely helpful in making diagnosis. The FLAIR images demonstrate hyper intense signal in the subarachnoid space.

Peri-mesencephalic SAH

- Non-aneurysmal form of SAH, occurs commonly anterior to pons and midbrain.
- May also be seen involving the basal, suprasellar cisterns, interhemispheric and sylvian fissures.

Venous infarction

Cerebral venous infarction is most commonly secondary to cerebral venous thrombosis and usually manifests with hemorrhage. It can lead to focal neurological deficits, or excessive oedema can result in symptoms from mass effect or hydrocephalus. Venous thrombosis should be suspected during the assessment of intracranial hemorrhage especially if it involves atypical areas, crosses arterial territories, or if there are infarcts with cortical sparing.

On CT Oedema & hemorrhage are seen in case of venous infarct. A specific sign called Cord sign is seen which refers to Cortical vein/ venous sinus hyper density whereas their filling defect or lack of opacification on contrast studies is called Empty delta sign. On MRI, Oedema and hemorrhage with variable restricted diffusion, dural venous sinusoidal flow void loss, altered signal in cortical vein or sinuses may be seen.

Venous ischemia or infarction usually shows reduced diffusion in DWI sequences unlike acute arterial infarction; however, its absence is not enough to rule out venous ischemia/infarction [15].

Cavernous Malformation

A cavernous malformation, also called cavernoma or cavernous haemangioma, is a vascular hamartoma. It is a benign mass composed of immature vessels which may bleed. Various stages of bleeding may be identified during imaging. Patient may be of any age, asymptomatic or present with intracranial haemorrhage. Location is usually supratentorial, but may less commonly present in the pons or cerebellum.

- CT images often appear normal or show rounded hyperdensities: blood products & calcification denoting acute haemorrhage.

MRI shows a complete hemosiderin ring of low signal surrounding a heterogeneous lesion of mixed signal intensity core called ‘popcorn’ lesions.

Fig 4: Cavernoma in a patient who presented with headache – (A) Axial Plain CT at the level of body of lateral ventricles show a small hyperdense focus involving the genu of corpus collosum. (B) Axial T1W image shows a small rounded hypo to isointense area which appears hyperintense with a surrounding hypointense rim in Axial FLAIR images (C) which shows blooming in SWI sequences (D).

Arteriovenous Malformation

A cerebral AVM represents arteriovenous shunting via abnormal vessels forming the nidus, a connection between the arteries and veins in absence of capillary bed [16] the vessels that make up the nidus are fragile and can rupture, which results in haemorrhage. The risk of ICH, most common presenting feature [17, 18, 19] of cerebral AVM is approximately 2-4% annually [20, 21] and the prevalence is 0.1 %. Rupture of cerebral AVM may result in SAH, Intraparenchymal hemorrhage or intraventricular hemorrhage, which can easily be diagnosed in acute cases on Head CT as a hyper density in respective locations [22].

CT with contrast shows hyperdense linear densities in a serpentine distribution commonly referred to as a "bag of worms", with little or no mass effect on adjacent brain. However small AVM's may appear a small bright focus on plain studies. Calcification is common. Uniform enhancement of all three AVM components (feeding arteries, nidus, draining veins) are seen.

MR Findings "honeycomb" of "flow voids" on both T1 and T2 scans with variable Contrast enhancement.

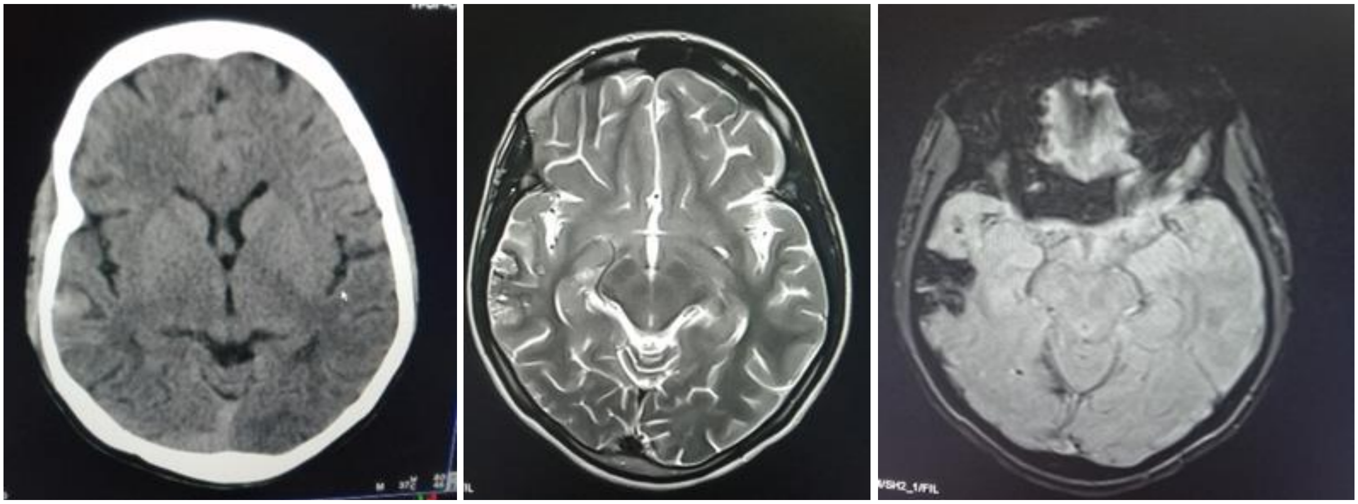

Fig 5: AVM in a patient who presented with headache – (A) Axial Plain CT show a peripherally located well defined hyper density in the right temporal lobe. (B) Axial T2W image shows multiple T2W/FLAIR hyperintense cortical based lesion with a surrounding hypointense rim. (C) SWI images show blooming of the area suggestive of multiple vascular channels.

Subdural Hemorrhage

Cause of Spontaneous hemorrhage into the subdural space may be intracranial hypotension or haemorrhagic dural metastases from breast carcinoma and choriocarcinoma or clotting disorders may also lead to spontaneous subdural hematoma (SDH). On plain Head CT, Acute subdural hemorrhage is seen as hyperdense hematoma located in space between pia mater and arachnoid, known as subdural space [23].

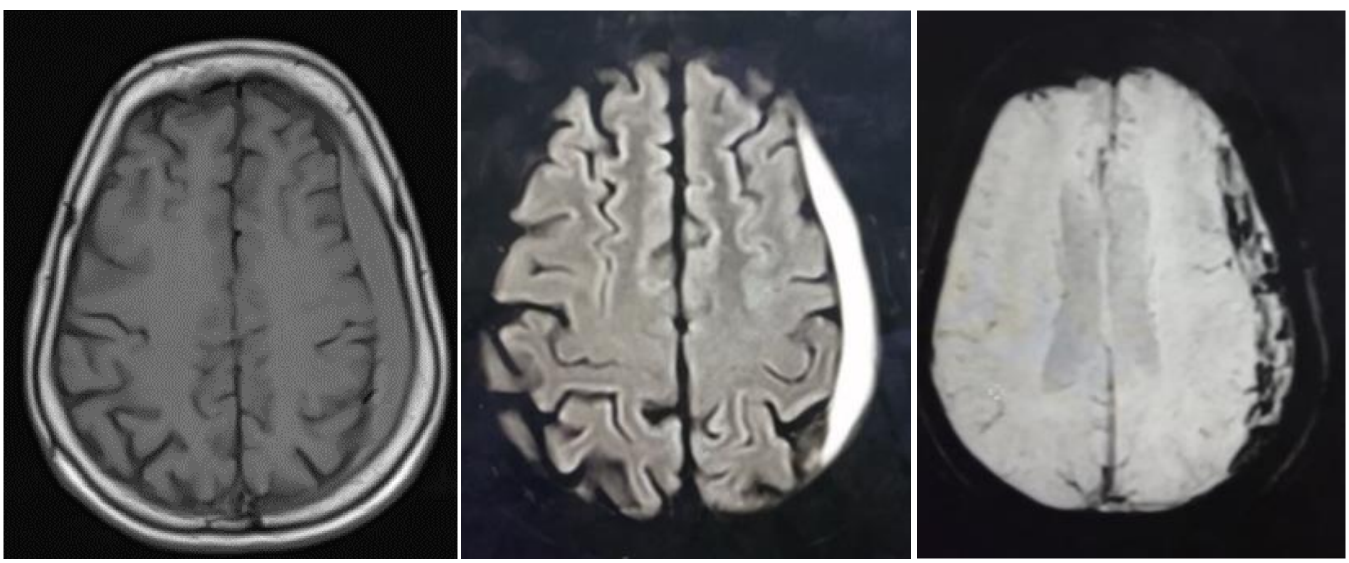

Fig 6: Subdural haemorrhage in a patient with left sided chronic headache – (A) Axial Plain T1W image shows a crescent shaped iso to hyperintense area between the inner table of skull and the cerebrum on the left side (B) Axial FLAIR at same level shows the area to be hyperintense with heterogenous areas of blooming seen in SWI sequence (C).

Infarction with haemorrhagic transformation

Haemorrhagic transformation can occur at a site of previously infarcted tissue and the risk of development of a transformation is increased following therapeutic recanalization of the thrombosed vessel. It should be noted that the term haemorrhagic transformation refers to two different processes- petechial hemorrhage and intracerebral hematoma It can occur spontaneously but more in those who receive anticoagulant therapy and even more in patients receiving thrombolytic therapy. Haemorrhagic transformation rarely occurs in the first 6 hours in patient who have not received treatment. Majority of haemorrhagic transformation occurs in the first 4 days post infarction. CT imaging shows large hematomas which may occur anywhere in an infarcted core. Underlying infarct when present can be identified as a region of low attenuation, with involvement of both the white matter and the overlying cortex. Hemorrhages can also appear as patchy areas, scattered throughout the infarcted tissue. MRI images may show restricted diffusion on DWI/ADC sequences. SWI are more sensitive compared to CT in identifying early or trace hemorrhage and may help direct therapeutic management (e.g. avoid thrombolysis).

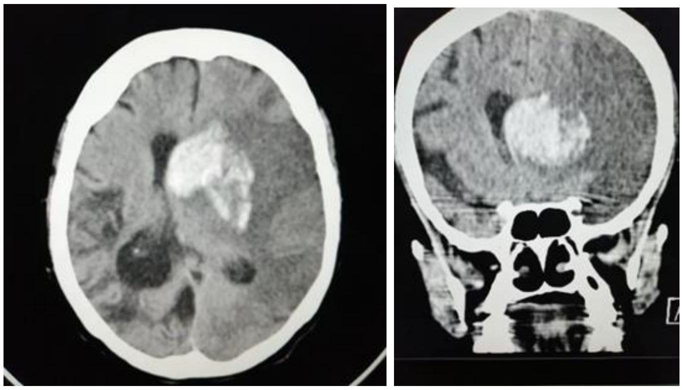

Fig 7: (A) Large acute MCA territory infarct with haemorrhagic transformation and midline shift and mass effect - Large ill-defined hypodense region is seen in left fronto-parietal region, with another comparatively hyperdense area medial to it with average HU +67 suggestive of haemorrhage. (B) Coronal reformatted CT image shows extent of the infarct which involving the left frontoparietal region with the hemorrhagic core inferomedial to it.

Hemorrhagic Neoplasm

Primary and metastatic brain tumours, both may have accompanied intra-parenchymal hemorrhage. Usually high-grade neoplasia has a propensity to bleed.

In an undiagnosed cerebral mass lesion, hemorrhage is a rare presentation but 15% of cases of Primary and secondary intracranial tumors have associated ICH.[24]

Primary brain tumors to show haemorrhage are

- Glioblastoma [GBM]: most common primary intracranial tumor to present with hemorrhage.
- Oligodendrogliomas: 20% of cases show intratumor hemorrhage.
- Anaplastic astrocytomas.

Hemorrhagic metastasis have a propensity for the grey-white matter junction, more commonly involving the anterior circulation [80%] and relatively less common location is the distribution of posterior circulation [20%]. Half of the metastasis present as a solitary lobar hematoma whereas the rest may show multiple lesions. The intracranial metastasis that have higher chances of bleed are- melanoma, choriocarcinoma, renal cell carcinoma, thyroid carcinoma and because of their higher incidence, breast carcinoma and lung cancer are more commonly encountered causes in general population. CT findings- Nonspecific Haemorrhage. Relatively extensive perilesional oedema may be a distinguishing feature. Some MRI findings seem to be more reliable in diagnosis of cerebral hemorrhage within the tumor or any malignancy.[25] MRI findings- Heterogenous lesion with areas of necrosis and hemorrhage. Extensive mass effect and surrounding oedematous tissue is usually present. Incomplete rim of hemosiderin may be seen in the periphery of the hemorrhage. In case of metastasis, similar lesions may be present at different sites. Diffusion restriction may be noted in case of primary tumor of highly cellularity.

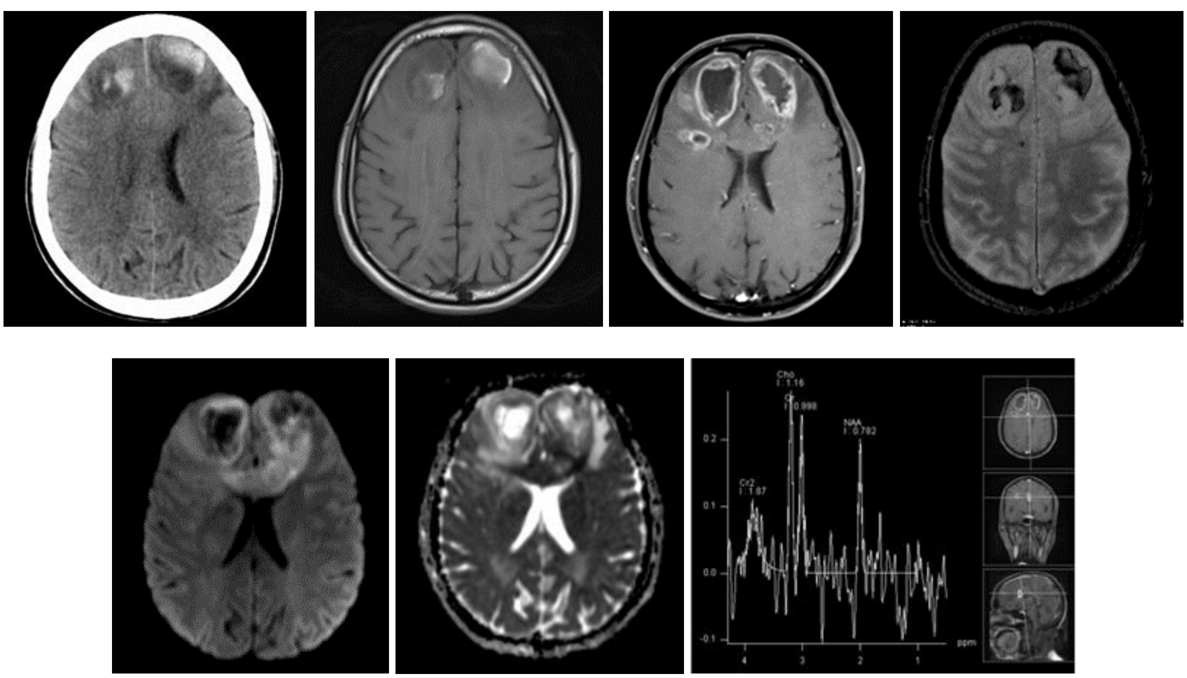

Fig 8: High grade haemorrhagic neoplasm (A) Axial CT at the level of lateral ventricles shows ill-defined heterogeneous density areas involving both frontal lobes. MR (B) shows the areas to be heterogenous appearing in T1W images with mixed hyperintense areas along with of low attenuation areas interspersed within it. (C) Post contrast T1W image shows peripheral ring enhancement with non-enhancing centre. Additionally two other small areas with similar enhancement patterns is seen surrounding the larger lesions. (D) SWI shows blooming of these lesions suggestive of haemorrhage (E) DWI and (F) ADC images at the same level shows an arc shaped area of restricted diffusion extending via the Corpus Callosum and connecting the lesions involving the frontal lobes (G) MR Spectroscopy of the lesion shows a Choline peak with low NAA levels suggestive of a neoplasm.

5. Conclusion

The aetiology of intracranial haemorrhage can range from a rupture of a small aneurysm to a more sinister cause like a haemorrhagic neoplasm. Although CT scan remains the mainstay of approaching a suspected case of intra cranial haemorrhage and will provide a quick and reliable diagnosis, a few require the need of higher modality for better visualization and characterisation of the lesion to and identify the underlying pathology prior to interventional procedures or surgery. MRI with its better soft tissue contrast and crucial imaging sequences adds as an adjuvant to confirm diagnosis and as a valuable problem-solving tool in indeterminate cases.

6. References

1. Van Asch CJ, Luitse MJ, Rinkel GJ, van der Tweel I, Algra A, Klijn CJ, *et al*. Incidence, case fatality, and functional outcome of intracerebral haemorrhage over time, according to age, sex, and ethnic origin: a systematic review and meta-analysis. Lancet Neurol. 2010; 9:167-176.
2. Shetty VS, Reis MN, Aulino JM, Berger KL, Broder J, Choudhri AF, *et al*. ACR appropriateness criteria head trauma. J Am Coll Radiol. 2016; 13:668-679.
3. Gupta R, Phan CM, Leidecker C, Brady TJ, Hirsch JA, Nogueira RG, *et al*. Evaluation of dual-energy CT for differentiating intracerebral hemorrhage from iodinated contrast material staining. Radiology. 2010; 257:205-211.
4. Brott T, Broderick J, Kothari R, *et al*. Early hemorrhage growth in patients with intracerebral hemorrhage. Stroke. 1997; 28:1e5.
5. Kinoshita T, Okudera T, Tamura H, *et al*. Assessment of lacunar hemorrhage associated with hypertensive stroke by echo-planar gradient-echo T2*-weighted MRI. Stroke. 2000; 31:1646e50.
6. Zhang LF, Yang J, Hong Z, Yuan GG, Zhou BF, Zhao LC, *et al*. Proportion of different subtypes of stroke in china. Stroke. 2003; 34:2091-2096.
7. Marder CP, Narla V, Fink JR, Tozer Fink KR. Subarachnoid hemorrhage: beyond aneurysms. AJR Am J Roentgenol. 2014; 202:25-37.
8. Field DK, Kleinig TJ. Aura attacks from acute convexity subarachnoid haemorrhage not due to cerebral amyloid angiopathy. Cephalalgia. 2011; 31:368-371.
9. Knudsen KA, Rosand J, Karluk D, Greenberg SM. Clinical diagnosis of cerebral amyloid angiopathy: validation of the Boston criteria. Neurology. 2001; 56:537-539.
10. Knudsen KA, Rosand J, Karluk D, Greenberg SM. Clinical diagnosis of cerebral amyloid angiopathy: validation of the Boston criteria. Neurology. 2001; 56:537-539.
11. Fisher M, French S, Ji P, Kim RC. Cerebral microbleeds in the elderly: a pathological analysis. Stroke. 2010; 41:2782-2785
12. Perry JJ, Stiell IG, Sivilotti ML, Bullard MJ, Emond M, Symington C, *et al*. Sensitivity of computed tomography performed within six hours of onset of headache for diagnosis of subarachnoid haemorrhage: prospective cohort study. BMJ. 2011; 343:d4277.
13. Brisman JL, Song JK, Newell DW. Cerebral aneurysms. N Engl J Med. 2006; 355:928-939.
14. Hochmuth A, Spetzger U, Schumacher M. Comparison of three-dimensional rotational angiography with digital subtraction angiography in the assessment of ruptured cerebral aneurysms. AJNR Am J Neuroradiol. 2002; 23:1199e205.
15. Makkat S, Stadnik T, Peeters E. Pathogenesis of venous stroke: evaluation with diffusion- and perfusion-weighted MRI. J Stroke Cerebrovasc Dis. 2003; 12:132e6.
16. Friedlander RM. Clinical practice. Arteriovenous malformations of the brain. N Engl J Med. 2007; 356:2704-2712.
17. Hofmeister C, Stapf C, Hartmann A, Sciacca RR, Mansmann U, terBrugge K, *et al*. Demographic, morphological, and clinical characteristics of 1289 patients with brain arteriovenous malformation. Stroke. 2000; 31:1307-1310.
18. Ondra SL, Troupp H, George ED, Schwab K. The natural history of symptomatic arteriovenous malformations of the brain: a 24-year follow-up assessment. J Neurosurg. 1990; 73:387-391.
19. Stapf C, Mohr JP, Pile-Spellman J, Solomon RA, Sacco RL, Connolly ES. Jr Epidemiology and natural history of arteriovenous malformations. Neurosurg Focus. 2001; 11:e1
20. Friedlander RM. Clinical practice. Arteriovenous malformations of the brain. N Engl J Med. 2007; 356:2704-2712.
21. Mohr JP, Parides MK, Stapf C, Moquete E, Moy CS, Overbey JR, *et al*. Medical management with or without interventional therapy for unruptured brain arteriovenous malformations (Aruba): a multicentre, non-blinded, randomised trial. Lancet. 2014; 383:614-621.
22. Lv X, Liu J, Hu X, Li Y. Patient age, hemorrhage patterns, and outcomes of arteriovenous malformation. World Neurosurg. 2015; 84:1039-1044.
23. Ivamoto HS, Lemos HP, Jr, Atallah AN. Surgical treatments for chronic subdural hematomas: a comprehensive systematic review. World Neurosurg. 2016; 86:399-418.
24. Salmaggi A, Erbetta A, Silvani A. Intracerebral haemorrhage in primary and metastatic brain tumours. Neurol Sci. 2008; 29:S264e5.
25. Atlas SW, Grossman RI, Gomori JM. Hemorrhagic intracranial malignant neoplasms: spin-echo MR imaging. Radiology. 1987; 164:71e7.