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Pictorial Review of Non-Traumatic Intracranial Haemorrhages (NTICH)

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Abstract: ***Background: Non-traumatic intracranial haemorrhages (NTICH) are a critical cause of morbidity and mortality, accounting for 10-15% of all strokes[1, 2]. Key etiologies include hypertension, vascular abnormalities, coagulopathies, aneurysms, and malignancies[2]. Early identification and precise diagnosis are essential for optimal patient management. Objective: This study presents a series of NTICH cases, illustrating the diversity of underlying causes and their characteristic imaging findings, to highlight the role of neuroimaging in diagnosis and management. Materials and Methods: We conducted a retrospective analysis of NTICH cases presenting between 2022 and 2023 at the Akash Institute of Medical Sciences and Research Centre, Devanahalli. Patients were evaluated using computed tomography (CT) and magnetic resonance imaging (MRI), with follow-up imaging where indicated. The cases include hypertensive intracerebral haemorrhage, haemorrhagic infarcts (arterial and venous), aneurysmal subarachnoid haemorrhage (SAH), cavernomas, tumour-associated haemorrhage, and haemorrhagic metastases. Results: Hypertension was the leading cause of NTICH, frequently presenting with intraventricular extension. Haemorrhagic infarcts demonstrated distinctive diffusion-weighted imaging (DWI) and susceptibility-weighted imaging (SWI) patterns, while aneurysmal SAH was identified via CT angiography. Rare entities such as tumour-related haemorrhages and haemorrhagic metastases were identified with characteristic CT and MRI features, aiding in differentiating them from other causes of intracranial haemorrhage. Conclusion: NTICH represents a complex spectrum of pathologies, with hypertension as the most common etiology. Advanced neuroimaging techniques like CT and MRI are indispensable for accurate diagnosis, guiding therapeutic decisions, and improving patient outcomes. Prompt recognition of the underlying cause is crucial for managing these life-threatening events.***

Keywords: Non-traumatic intracranial haemorrhage, CT, MRI, hypertensive haemorrhage, aneurysms, vascular malformations, haemorrhagic infarcts, spontaneous intracranial haemorrhage.

1. Introduction

Non-traumatic intracranial haemorrhage (NTICH) is a form of spontaneous bleeding within the brain parenchyma that occurs without external trauma. It accounts for approximately 10-15% of all strokes and is associated with high mortality and morbidity rates[1, 2]. NTICH is the second most prevalent stroke subtype globally, with the incidence expected to rise due to an aging population[2]. Despite advances in medical care, NTICH remains a major contributor to neurological disability and death, particularly among elderly patients[1, 2].

2. Background

Intracranial haemorrhage (ICH) is one of the most devastating forms of stroke due to its rapid onset and poor prognosis. It commonly results from rupture of weakened blood vessels, often related to chronic medical conditions such as hypertension and cerebral amyloid angiopathy[2, 3]. Advances in neuroimaging and medical management have improved early diagnosis and intervention, but outcomes remain poor, with high case-fatality rates[3]. ICH is also more common in specific populations, with increased incidence in men, older adults, and certain ethnic groups like African Americans and Asians[2].

Etiology

The primary etiologies of NTICH are chronic hypertension and cerebral amyloid angiopathy (CAA), with secondary causes including vascular anomalies, coagulation disorders, neoplasms, and drug use[2, 3]. Hypertension remains the most significant modifiable risk factor for ICH, particularly for deep brain haemorrhages in the basal ganglia, thalamus, and cerebellum[2]. Cerebral amyloid angiopathy, more common in the elderly, leads to lobar haemorrhages due to β-amyloid deposits in the small blood vessels[2, 3]. Other risk factors include anticoagulant use, often in patients receiving treatment for atrial fibrillation[1, 3].

Risk Factors

Hypertension is the most critical modifiable risk factor for NTICH, especially in deep-brain haemorrhages, followed by age, particularly in relation to CAA[1, 2]. Anticoagulant therapy significantly increases the risk of haemorrhage, especially in patients treated with vitamin K antagonists or newer anticoagulants like direct oral anticoagulants[2, 3]. Other important risk factors include heavy alcohol use, smoking, and drug abuse (e.g., cocaine, amphetamines)[2].

Hypertensive Intracerebral Haemorrhage

Hypertension remains the most common cause of NTICH, particularly affecting deep brain structures such as the basal ganglia, thalamus, and cerebellum. Hypertensive haemorrhages can be observed across all regions of brain, frequently extending into the ventricular system (intraventricular haemorrhage, IVH).

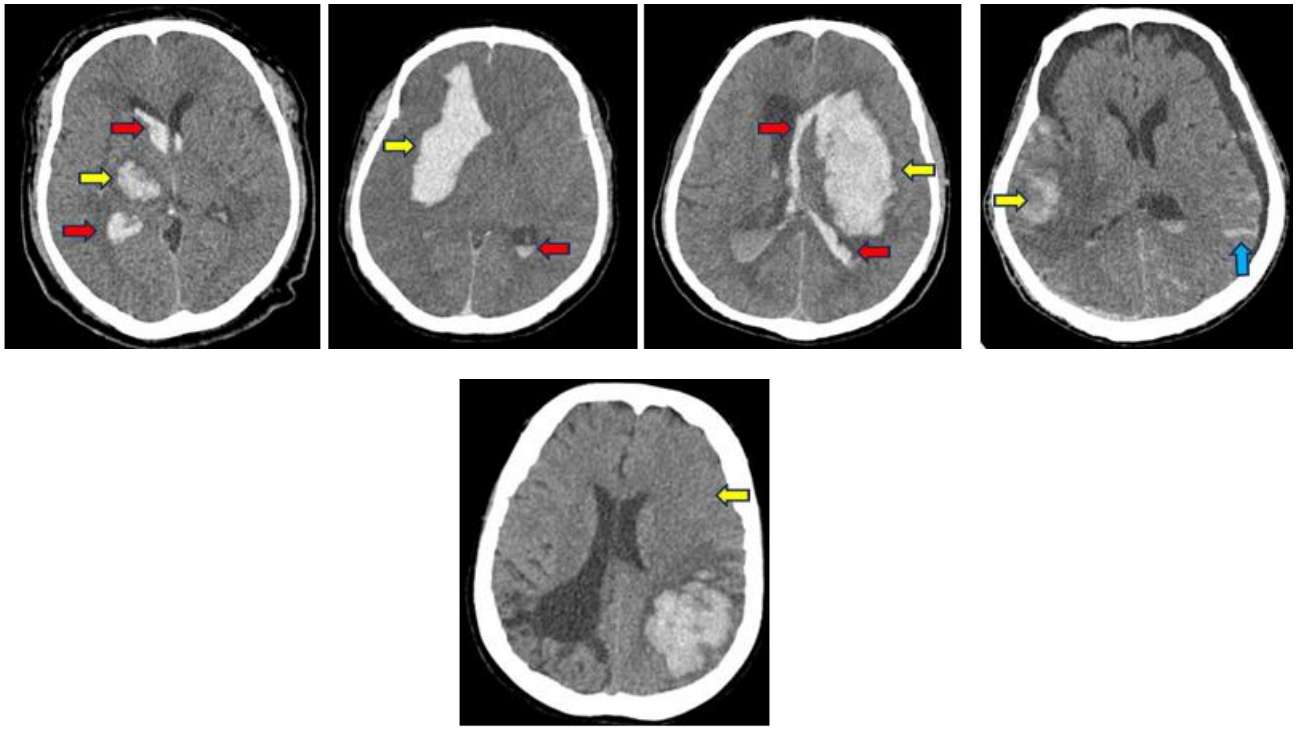


Figure 1: CT brain of different, known hypertensive patients shows, haemorrhages involving all regions of brain (yellow arrows), with intraventricular extension (red arrows) and few causing subarachnoid haemorrhage (blue arrows).

Haemorrhagic Arterial Infarcts

A case of a 70-year-old male presenting with right hemiparesis.

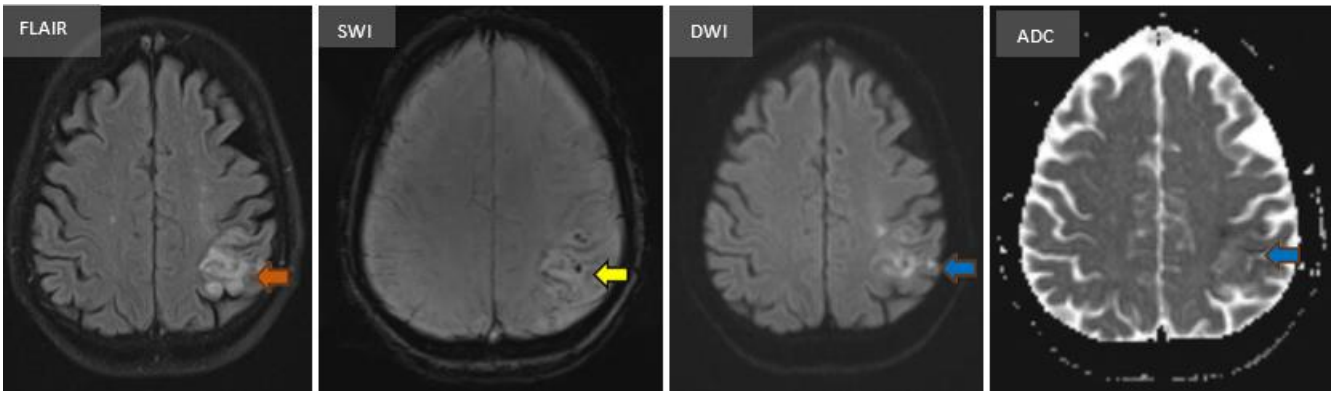
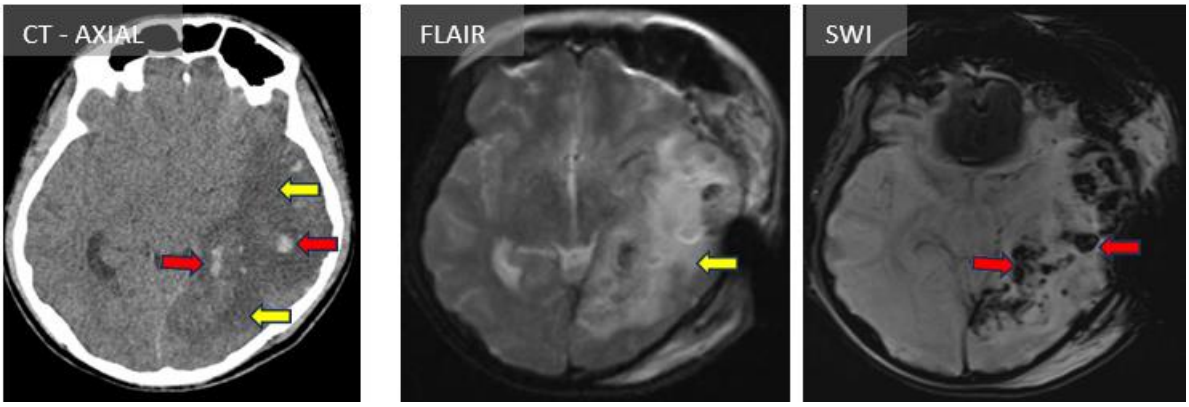


Figure 2: MRI brain stroke protocol shows, area of restricted diffusion (blue arrows) is noted involving both grey and white matter of the left parietal lobe associated with hyperintensity on FLAIR (orange arrow). On magnitude images of SWI, there is evidence of petechial hemorrhages (yellow arrows).

Haemorrhagic Venous Infarcts

A case of 19-year-old male presented with altered sensorium.



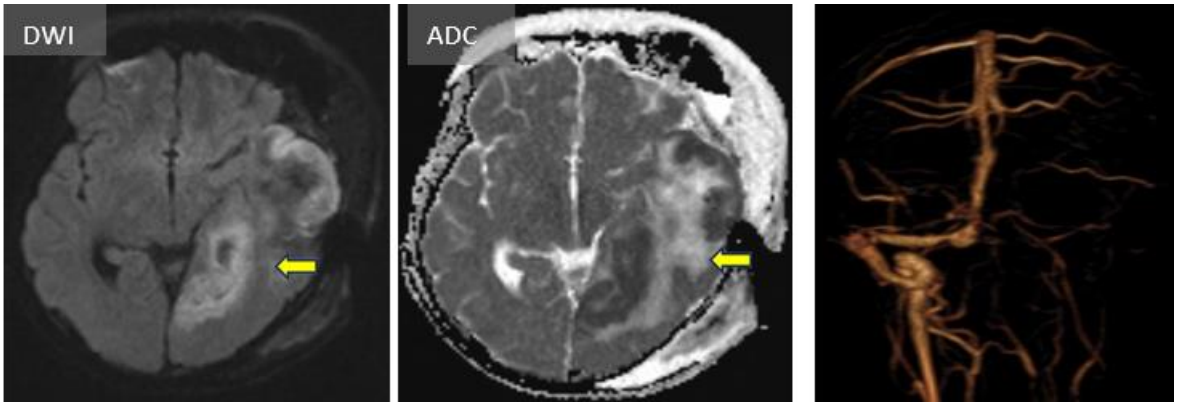


Figure 3: **CT brain shows,** ill-defined hypodensity in left temporal and medial occipital lobe *(yellow arrows)* with hyperdense areas within and mass effect causing significant midline shift. **MR venogram post decompression craniectomy shows,** a large area of signal abnormality involving left temporal lobe *(red arrows)* which is hyperintense on FLAIR with restriction on DWI and with evidence of extensive blood products on SWI. No flow related signal in left transverse and sigmoid sinuses.

Aneurysmal subarachnoid haemorrhage

A 72-year-old female, presented to causality with sudden onset headache and loss of consciousness

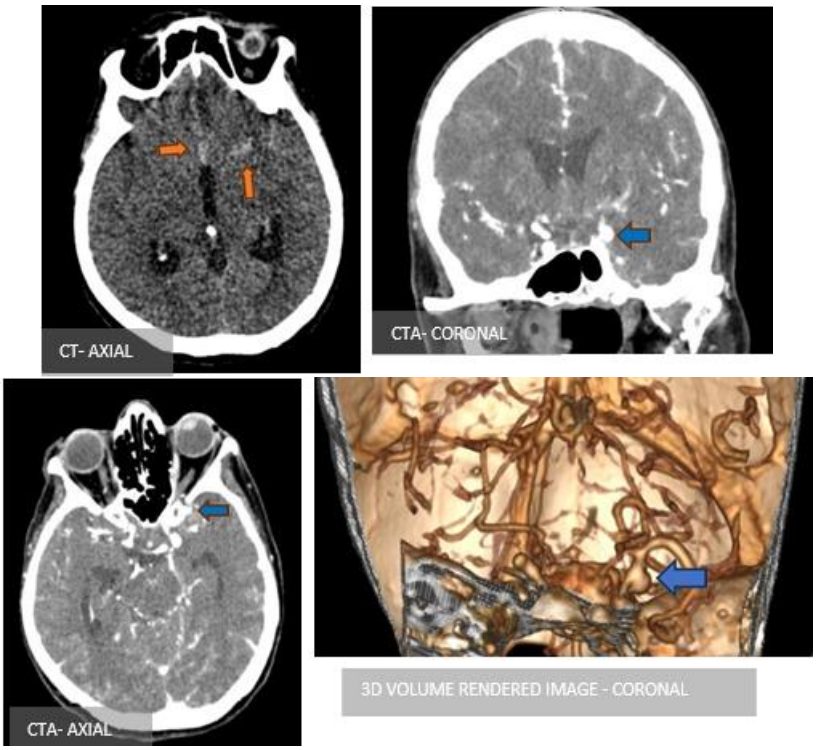


Figure 3: CT cerebral angiogram shows, Acute sub arachnoid hemorrhage (orange arrows) in the left sylvian fissure, anterior inter hemispheric fissure, peri mesencephalic and pre pontine cisterns on the left in the non- contrast images. A contrast filled out pouching arising from proximal supraclinoid segment of the left ICA which is consistent with an saccular aneurysm (blue arrows).

Cavernoma

A 60-year-old female with a history of sudden onset seizures and loss of consciousness.

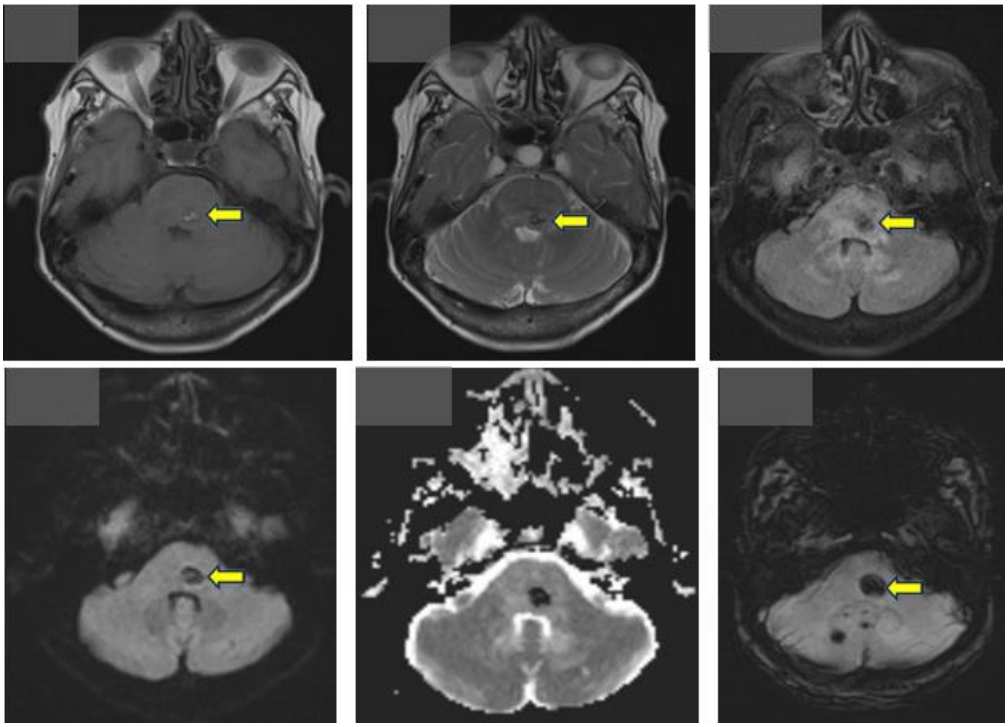
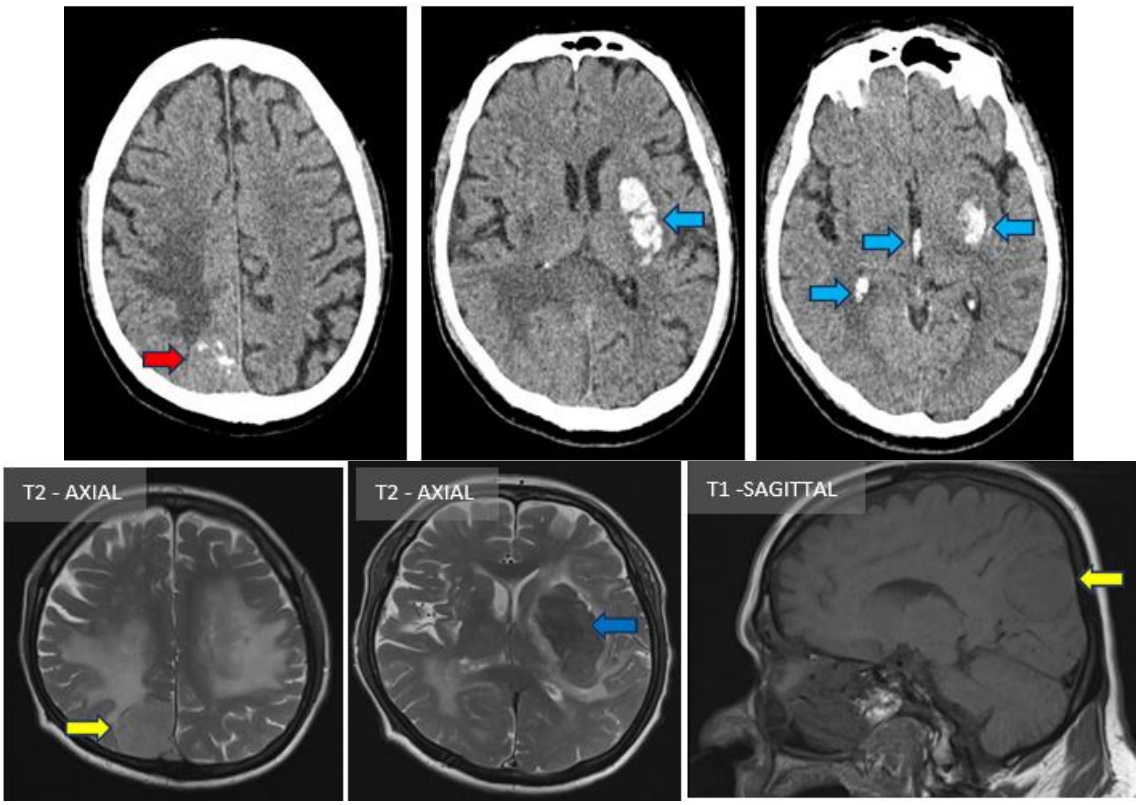


Figure 5: MRI brain shows, Evidence of old blood products in the right lentiform nucleus and right cerebellum. Focus of bleed in left half of pons *(yellow arrows)* which is T1WI hyperintense and T2WI hypointense, suggestive of early subacute blood

Tumour-associated haemorrhage

Haemorrhage secondary to brain tumours is rare but important to recognize.

An 85-year-old male presented with right hemiplegia associated with slurring of speech and giddiness.



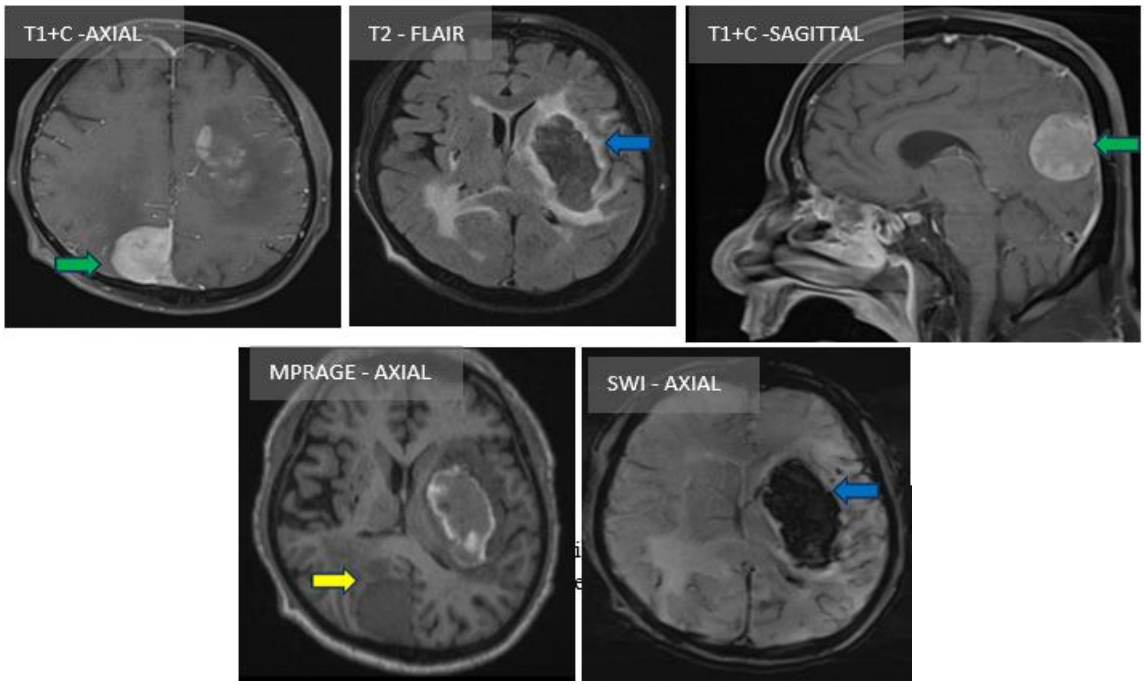


Figure 6: Plain CT brain shows, Extra axial isodense lesion along posterior falx to the right of midline showing calcifications within and significant perilesional white matter edema *(red arrows)*. Acute intraparenchymal hemorrhage in left capsuloganglionic region with perilesional edema and intraventricular extension of the hemorrhage *(blue arrows)*.

CEMRI of brain shows, A large extra axial space occupying lesion in the right inferior parietal region (yellow arrows) which is isointense on T1WI, hyperintense on T2WI without any evidence of blood products. Thin intense homogenous enhancement *(green arrows)* within the lesion. Significant perilesional vasogenic edema noted within the ipsilateral parietal lobe. A large intra parenchymal hematoma involving the left corona radiata and capsuloganglionic region *(blue arrows)* with extensive surrounding edema causing compression of left lateral ventricle and midline shift of to the right.

Haemorrhagic Metastases

A 42-year-old male with known embryonal cell carcinoma of left testis presented with sudden onset seizures.

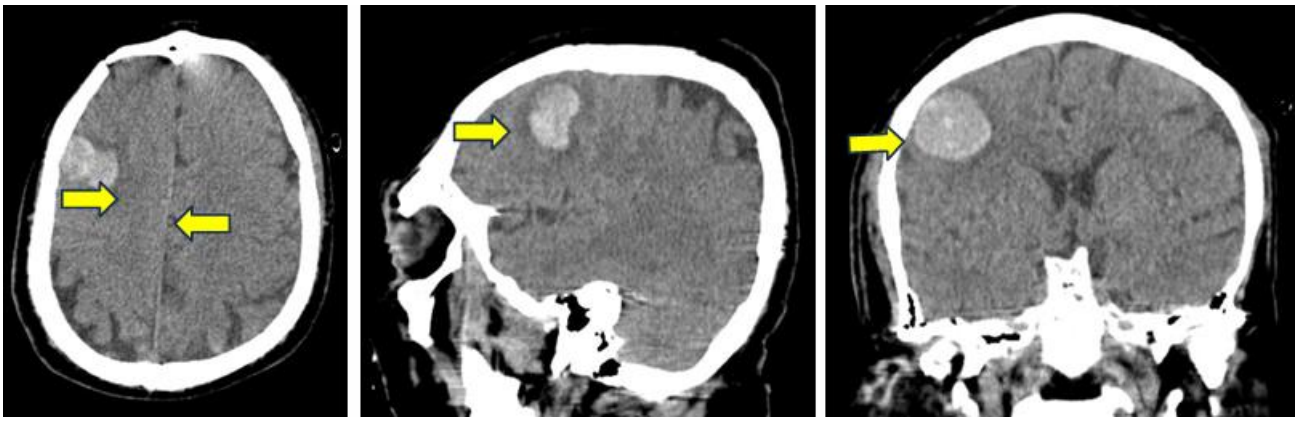


Figure 7: Plain CT brain shows, two fairly well-defined rounded closely opposed hyper densities in the right frontal lobe with hemorrhagic changes and perilesional edema *(yellow arrows)* causing mass effect in the form of effacement of adjacent sulci

3. Discussion

NTICH represents a complex pathology with diverse etiologies ranging from hypertension to vascular malformations and malignancies. Hypertensive haemorrhages, often located in the basal ganglia, are well-characterized on CT by their high density and possible ventricular extension. Haemorrhagic infarcts—both arterial and venous—demonstrate distinctive MRI findings, with DWI and SWI playing a critical role in identifying areas of infarction and microbleeds.

Aneurysmal SAH requires prompt diagnosis, typically via CT angiography, to localize the source of bleeding and assess the aneurysm morphology. AVMs, though less common, are associated with repeated haemorrhages, while tumour-related haemorrhage, although rare, should be considered in the differential, especially in patients with known malignancy or atypical site / morphology ofbleed.

4. Conclusion

Spontaneous intracranial haemorrhages pose significant diagnostic and therapeutic challenges. Hypertension remains the leading cause of NTICH, with other etiologies including haemorrhagic infarcts, aneurysms, vascular malformations, and malignancies. CT and MRI are invaluable tools in identifying the underlying causes and guiding treatment decisions. Given the high morbidity and mortality associated with NTICH, early diagnosis and intervention are paramount.

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