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REVIEW ARTICLE

Pattern-driven neuroimaging of intracranial hemorrhage: CT/MRI findings and their clinical implications - A comprehensive review article.

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ABSTRACT… Intracranial hemorrhage (ICH) represents a significant and growing healthcare burden in Pakistan, with an increasing number of patients presenting to tertiary care hospitals. This rise is attributed largely to contemporary lifestyle factors and prevailing traffic conditions. The etiologies of ICH are diverse, encompassing trauma, hypertension, cerebral amyloid angiopathy, hemorrhagic transformation of ischemic infarction, cerebral aneurysms, arteriovenous malformations and fistulas, vasculitis, tumor-related hemorrhage, and venous sinus thrombosis. Accurate interpretation of neuroimaging is critical for early diagnosis and effective management; however, trainee doctors frequently encounter challenges in recognizing the imaging features associated with different types of ICH, which may lead to diagnostic delays or inappropriate treatment decisions. This review aims to give a thorough insight of the characteristic imaging patterns of intracranial hemorrhage across modalities, with a focus on practical, pattern-based interpretation. By consolidating key diagnostic features and correlating them with underlying pathology, this article seeks to support trainee physicians in improving diagnostic accuracy, enhancing patient outcomes, and contributing to the standardization of care in clinical settings.

Key words: Arteriovenous Malformations, Cerebral Amyloid Angiopathy, Computed Tomography, Cerebral Aneurysm, Epidural Hemorrhage, Hypertensive Hemorrhage, Intracranial Hemorrhage, Subarachnoid Hemorrhage, Subdural Hematoma, Traumatic Brain Injury.

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INTRODUCTION

One of the major medical concerns in Pakistan is intracranial hemorrhage, accounting for approximately 13–15% of all strokes.[1] The estimated incidence of ICH is around 40 / 100,000 person-years, with a one-month mortality rate ranging from 40–42%.[3] Given the diverse etiologies of ICH and its involvement of multiple intracranial compartments[2], it is essential for treating physicians to possess a thorough understanding of neuroimaging to accurately identify the location, extent, and volume of hemorrhage.[4] Such knowledge is critical for assessing the likelihood of secondary cerebral injury and guiding timely, and urgent management.

Computed tomography remains the imaging modality of choice in the emergency evaluation of patients with suspected or confirmed ICH due to its rapid acquisition, wide availability, and high sensitivity in detecting acute hemorrhage.[4,5] Pattern recognition on CT not only aids in differentiating the underlying causes of ICH but also provides essential information for prognostication and therapeutic decision-making.

AIM

To analyze characteristic imaging patterns across CT and MRI modalities and correlate them with clinical severity, prognosis, and treatment pathways.

OBJECTIVES

- To summarize the common and uncommon causes of intracranial hemorrhage.
- To describe the characteristic imaging features of different types of intracranial hemorrhage on computed tomography (CT) and other relevant modalities.
- To highlight pattern-based approaches that can aid trainees in differentiating between hemorrhage subtypes.
- To provide guidance on the clinical relevance of imaging findings for appropriate patient

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management.
- To promote standardization in diagnostic interpretation and improve decision-making among physicians.

Inclusion Criteria

This review focuses on imaging patterns of intracranial hemorrhage resulting from the following etiologies:
Trauma: including epidural, subdural, and intraparenchymal hemorrhage secondary to head injury. Hypertension: hypertensive intraparenchymal hemorrhage. Cerebral Amyloid Angiopathy: lobar hemorrhages associated with amyloid deposition. Hemorrhagic Conversion of Ischemic Infarction: post-stroke hemorrhagic transformation. Cerebral Aneurysms: including ruptured aneurysms causing subarachnoid hemorrhage. Cerebral Arteriovenous Malformations (AVMs): spontaneous hemorrhage from AVMs. Dural Arteriovenous Fistulas (dAVFs): intracranial bleeding associated with fistulas. Vasculitis: hemorrhages secondary to inflammatory vascular disorders. Venous Sinus Thrombosis: hemorrhagic infarcts due to impaired venous drainage.

Exclusion Criteria

Studies and cases were excluded from this review if they involved:
Non-intracranial hemorrhagic conditions such as spinal hemorrhage or extracranial bleeding. Pediatric specific hemorrhages unless directly relevant to the adult patterns discussed.Isolated traumatic microbleeds or diffuse axonal injury without significant hemorrhage. Hemorrhages secondary to rare or experimental interventions not widely applicable in clinical practice. Animal studies or in vitro imaging studies not correlating with human pathology. Reports lacking imaging correlation or adequate radiological documentation.

Traumatic Intracranial Hemorrhage

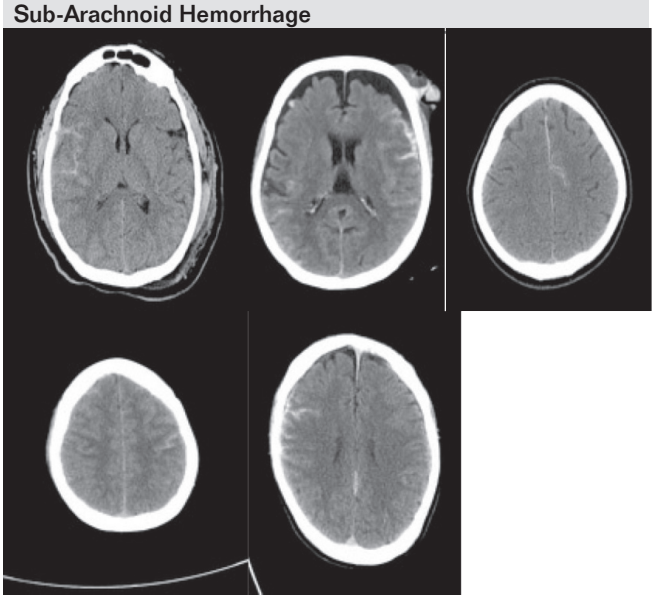
Trauma is one of the commonly found cause of intracranial hemorrhage (ICH), resulting from damage to intracranial arteries and veins that leads to bleeding within or on the surface of the brain.[2] Traumatic ICH can present in various forms, including subarachnoid hemorrhage (SAH), epidural hematoma, subdural hematoma, hemorrhagic parenchymal contusions, and cerebral microhemorrhages secondary to shear injury.[2] Each type of hemorrhage exhibits distinct radiological characteristics, clinical implications, and management considerations, which will be discussed in detail in the following sections.

Traumatic Subarachnoid Hemorrhage (SAH)

Traumatic subarachnoid haemorrhage (tSAH) is described as the existence of blood in the subarachnoid spaces, which is usually localised in the superficial sulci along the cerebral convexities.[6] It is the second most frequently observed acute brain injury on CT scans in individuals with traumatic brain injury (TBI), occurring in approximately 35% of cases[7], with reported ranges between 11% and 60%. Unlike non-traumatic subarachnoid hemorrhage, which frequently involves the basal cisterns and the lateral or Sylvian fissures, tSAH is predominantly found in the cerebral sulci. It often occurs adjacent to skull fractures and parenchymal contusions. When tSAH involves basal cisterns[8], it shows a predilection for the superior cistern or the cistern of the great cerebral vein.

FIGURE-1

Sub-Arachnoid Hemorrhage



Epidural Hematoma

Epidural Hematoma (EDH) is an acute neurosurgical emergency characterized by the accumulation of blood in the epidural space, situated between the inner surface of the skull and the dura mater, the outermost protective membrane covering the brain. EDHs most commonly arise from skull fractures

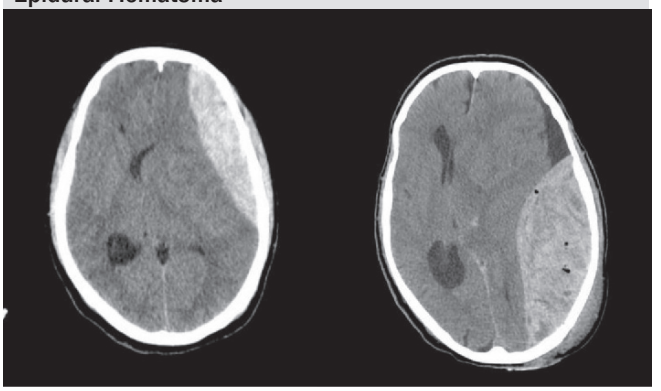
that lacerate underlying blood vessels, resulting in the accumulation of blood in this potential space.[9,4] Although less frequent than subdural hematomas, EDHs are more commonly observed in young adults.[10]

Diagnosis of an acute EDH is typically established using a head CT scan, which reveals a hyperdense, biconvex (lentiform) collection confined to the epidural space. A distinguishing feature of EDHs is that their spread is limited by cranial sutures, which act as anatomical barriers, helping clinicians estimate the hematoma's extent and plan management.[9] As the hematoma enlarges, it initially expands laterally to the suture margins and subsequently raises in the superficial-to-deep dimension, producing characteristic biconvex shape.[11]

Clinically, EDHs exert pressure on the brain parenchyma, leading to symptoms such as headache, confusion, vomiting, seizures, or loss of consciousness.[10] The severity of symptoms is largely dependent on the hematoma's size and location. Prompt recognition and intervention are critical, as untreated EDHs can result in irreversible brain injury or death. Surgical evacuation, usually via craniotomy, remains the definitive treatment in cases of significant mass effect or neurological deterioration.

FIGURE-2

Epidural Hematoma



Subdural Hematoma

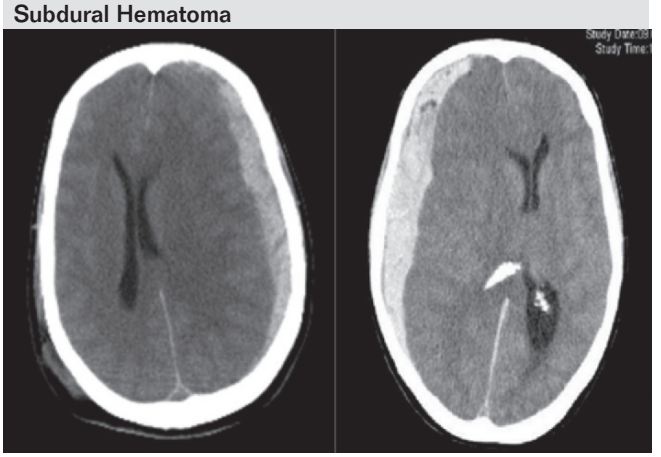
Acute subdural hematomas (ASDH) can be found to be hyperdense collections on head CT scans, located within the subdural space, which lies in the space separating the arachnoid and pia layers of the meninges.[12] Traumatic ASDH remains one of the most lethal forms of head injury. The timing of surgical intervention for clot evacuation is widely recognized as a critical determinant of patient outcome.[7,8]

Adjacent bony features may obscure small subdural hematomas on CT images. To enhance detection, radiologists can adjust CT window settings to better differentiate the density of blood from surrounding bone.[14,15] A window width of 130 with a window level of 30 has been recommended for optimal visualization.[14]

Subdural hemorrhage occurs beneath the dura mater, allowing it to spread without restriction across cranial sutures, which helps distinguish it from epidural hematomas, which are typically confined by suture lines.[14] Medially, the falx cerebri limits the spread of subdural hematomas, whereas superiorly and inferiorly, the tentorium cerebri serves as a boundary. These hematomas tend to extend along the ipsilateral aspect of the structures without opposition.

FIGURE-3

Subdural Hematoma



Hemorrhagic Parenchymal Contusion

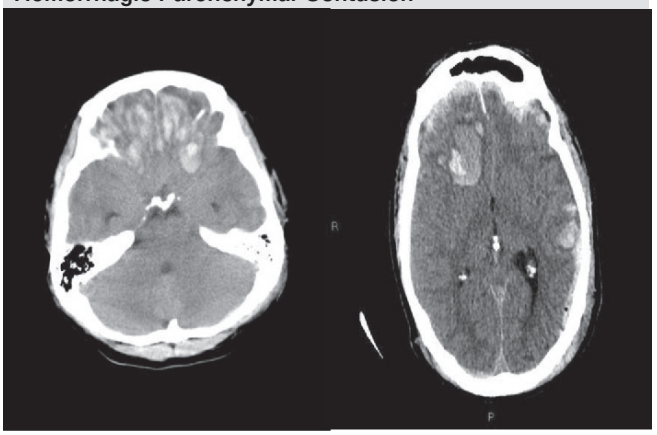
Hemorrhagic parenchymal contusions, also known as brain bruises, are a type of traumatic brain injury that typically results from considerable head movement and forceful impact.[16,17] These contusions are defined by localized areas of bleeding within the brain tissue, most often affecting the undersurface and poles of the frontal and temporal lobes. They appear as hyperdense regions of hemorrhage on CT scans, caused by injury to microvascular arteries or veins.[17]

Contusions can evolve over time, sometimes expanding in size, and multiple contusions may be present in a single patient.[18] These injuries can lead to substantial brain damage and loss of function in the affected areas. Prompt medical evaluation is crucial, as early detection and intervention can significantly improve the chances of recovery.[18]

Hemorrhagic parenchymal contusions often occur either at the site of impact (coup) or contralaterally (contrecoup) because the brain shifts within the cranial cavity following the initial force.[17] This mechanism means that a head injury can produce both direct and secondary damage.[17] Because of the potential for progression, follow-up imaging is often recommended to monitor for increases in contusion size.[18] Awareness of brain injury symptoms and timely medical attention are essential to optimize outcomes.[18]

FIGURE-4

Hemorrhagic Parenchymal Contusion



Cerebral Microhemorrhage (Diffuse Axonal Injury).

Diffuse axonal injury (DAI) is a serious type of brain trauma caused by sudden rotational or linear forces on the brain, such as during a car accident or a fall. This motion causes the brain to move and twist inside the skull, resulting in damage to the long nerve fibers, called axons, that connect different brain regions. Such injuries primarily affect the brain's white matter, which is essential for communication between different regions of the brain.[2,7]

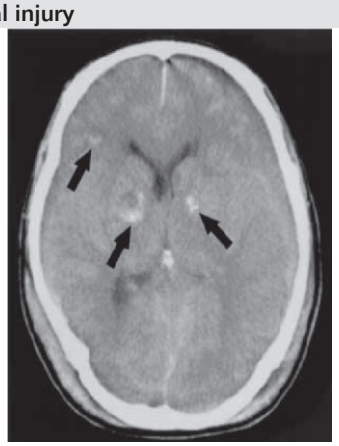
Cerebral microhemorrhages are smaller forms of post-traumatic hemorrhagic parenchymal contusions that commonly occur in the brain's white matter. These tiny areas of bleeding can further compromise white matter integrity and may contribute to additional neurological complications.[19]

The consequences of diffuse axonal injury are often widespread and can result in coma and damage to multiple brain regions. Importantly, the changes caused by DAI are frequently microscopic, making them difficult to detect with conventional imaging approaches for example computed tomography or magnetic resonance imaging. Therefore, accurate diagnosis relies heavily on detailed clinical assessments.[19]

In conclusion, diffuse axonal injury is a complex and serious neurological condition that predominantly affects the brain's white matter. Its subtle and microscopic nature often makes detection challenging, emphasizing the need for careful clinical evaluation to ensure proper diagnosis and management.[20]

FIGURE-5

Diffuse axonal injury



Intraparenchymal Hemorrhage Due to Hypertension

Intraparenchymal hemorrhage (IPH), often caused by chronic hypertension, which commonly occurs in patients with age falling between 60s and 70s and is associated with a high mortality rate of 30–50%.[21] On head CT imaging, IPH appears as a region of increased density within the brain tissue, most frequently involving the basal ganglia, cerebellum, or occipital lobes.[21]

When IPH occurs in the cerebral cortex, particularly in non-traumatic cases, alternative etiologies beyond

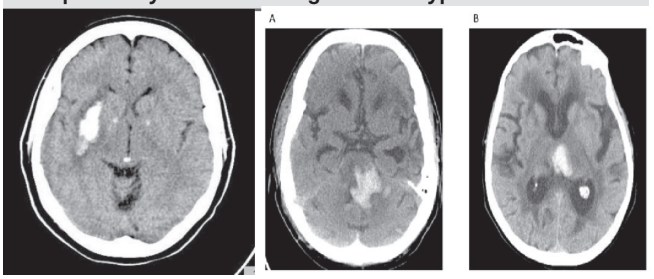
hypertension should be considered.[22] Similarly, patients under 50 years of age presenting with IPH require evaluation for other potential causes, including underlying brain neoplasms or vascular malformations. Hematoma size varies widely, from small collections (<1–2 cm) with minimal effect on surrounding brain tissue to large hematomas causing significant mass effect or even brain herniation.

Pathophysiologically, chronic hypertension leads to early arteriolar smooth muscle hypertrophy. This process is followed by the loss of smooth muscle cells and the accumulation of collagen. This process can result in arterial occlusion or ectasia, predisposing to Charcot-Bouchard aneurysm formation and subsequent intracerebral bleeding.[23]

Management of IPH often includes serial imaging with CT or MRI to monitor hematoma expansion or surrounding edema, which may necessitate interventions such as surgical decompression or hematoma evacuation.[24] In acute settings, CT angiography (CTA) has become increasingly valuable. Delayed-phase CTA can detect active contrast extravasation in the hemorrhage, a finding known as the Spot Sign.[25] The presence of the Spot Sign is strongly associated with an increased risk of hematoma expansion and worse clinical outcomes, serving as a critical marker to guide urgent medical or surgical intervention.[25]

FIGURE-6

Intraparenchymal hemorrhage due to hypertension



Intraparenchymal Hemorrhage Due to Cerebral Amyloid Angiopathy

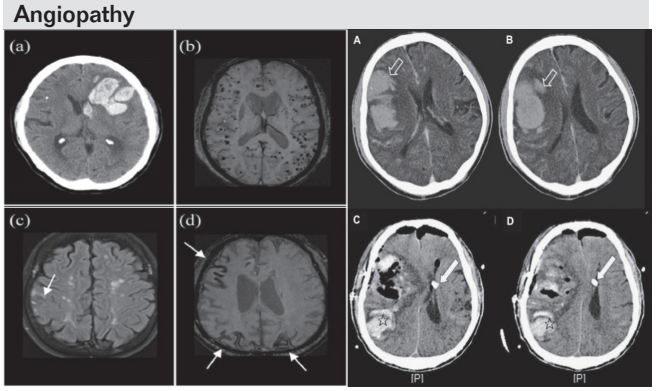
Cerebral amyloid angiopathy (CAA) involves the accumulation of amyloid-β protein in the walls of cerebral blood vessels, leading to weakened vascular integrity. This vascular fragility can result in cerebral microhemorrhages, sulcal subarachnoid hemorrhage, or larger intracerebral hemorrhages.[26] Sulcal SAH associated with CAA differs from vasculopathy or vasculitis in that it predominantly affects individuals over 60 years of age and is often accompanied by transient motor or sensory deficits.[26] Additional intracerebral hemorrhages may also be present.

When CAA causes IPH, imaging characteristics can help distinguish it from hypertensive hemorrhages. CAA-related IPH typically occurs in the subcortical white matter adjacent to the cerebral cortex, while sparing the basal ganglia, posterior fossa, and brainstem.[27] The Boston criteria are widely applied to estimate the probability that intraparenchymal hemorrhage is related to cerebral amyloid angiopathy, using the pattern and number of cerebral hemorrhages and microbleeds.[28] While confirmation of CAA ultimately requires histopathologic examination via brain biopsy.[29], CT imaging often provides the first clue, typically demonstrating a hyperdense intra-axial hemorrhage in the subcortical region.[28]

Other imaging features may include widespread hypoattenuation of the cerebral white matter bilaterally, suggesting underlying microangiopathic pathology, although this finding is not present in all individuals with CAA. MRI of the brain can offer additional diagnostic evidence, as susceptibility-weighted imaging (SWI) or gradient-recalled echo (GRE) sequences often demonstrate multiple small areas of susceptibility blooming within the bilateral cerebral white matter.[30]

FIGURE-7

Intraparenchymal Hemorrhage Due to Cerebral Amyloid Angiopathy



Cerebrovascular Causes of Intracranial Hemorrhage

Intracerebral hemorrhage is a serious neurological

condition that can result from a variety of cerebrovascular diseases. Among these, non-traumatic cerebrovascular disorders are a common cause of ICH. Morphology and anatomical distribution of intracerebral hemorrhage often vary according to the specific underlying lesion and its location within the brain.[31]

Frequently encountered cerebrovascular causes of intracerebral hemorrhage include hemorrhagic transformation of ischemic infarcts, aneurysms, arteriovenous malformations (AVMs), dural arteriovenous fistulae (DAVFs), vasculitis or other vasculopathies, mycotic aneurysms, and thrombosis of cortical veins or dural venous sinuses.

- Hemorrhagic conversion of ischemic infarction occurs when an area of brain previously affected by ischemia develops secondary hemorrhage.
- Aneurysms are abnormal vascular outpouchings that can rupture, leading to intracerebral bleeding.
- Arteriovenous malformations (AVMs) are tangled networks of abnormal vessels prone to rupture and hemorrhage.
- Dural arteriovenous fistulae (DAVFs) are abnormal connections between arteries and veins in the dura mater, which can cause ICH due to arterialized venous pressure.
- Vasculitis or vasculopathy involves inflammation or structural damage to cerebral vessels, which can precipitate hemorrhage.
- Mycotic aneurysms are rare aneurysms caused by infectious involvement of the arterial wall.
- Cortical venous or venous sinus thrombosis refers to the formation of blood clots in cerebral veins or dural sinuses, which may lead to secondary hemorrhage.

Hemorrhagic Conversion of Ischemic Infarction

An ischemic infarct develops when a cerebral artery is obstructed, typically due to a thrombus or embolus, resulting in compromised blood flow and tissue ischemia. A serious complication of ischemic infarction is hemorrhagic conversion, in which bleeding occurs within the infarcted brain tissue.[32] This complication can affect up to 43% of patients and is more likely following intravenous thrombolysis or trans-arterial recanalization procedures.[33,35]

The severity of hemorrhagic transformation is classified into four types:

- HI1 (Hemorrhagic Infarction 1): Petechial hemorrhage along the margin of the infarct.
- HI2 (Hemorrhagic Infarction 2): Confluent petechial hemorrhage within the infarcted tissue.
- PH1 (Parenchymal Hematoma 1): Hematoma involving ≤30% of the infarct with mild mass effect.
- PH2 (Parenchymal Hematoma 2): Hematoma involving >30% of the infarct with significant mass effect.

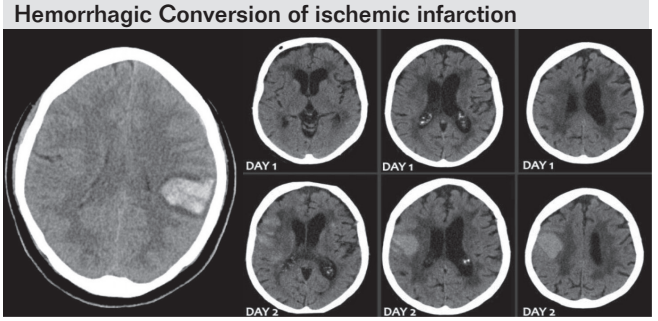
Among these, only PH2 is associated with substantial clinical consequences.[34]

Patients with ischemic strokes often undergo repeated neuroimaging to track potential hemorrhagic conversion and post-stroke vasogenic edema.[36] If neurological function deteriorates in the subsequent days, urgent CT or MRI is indicated to identify symptomatic hemorrhagic transformation or worsening edema, which may occasionally require surgical decompression.[37]

Reperfusion hemorrhage is a specific type of bleeding that can occur following successful endovascular therapy for stroke. Detection may be challenging due to contrast "staining" from cerebral angiography. Emerging techniques, such as dual-energy CT, have shown promise in distinguishing true hemorrhagic transformation from contrast extravasation, although these approaches are still under investigation.[38]

FIGURE-8

Hemorrhagic Conversion of ischemic infarction



Cerebral Aneurysms

Cerebral aneurysms are localized dilations of arteries on the cerebral surface, indicating regions of arterial wall fragility susceptible to rupture.[39]

When a cerebral aneurysm ruptures, it commonly causes the abrupt onset of an intense headache, frequently described by patients as the most severe they have ever experienced, due to subarachnoid hemorrhage and irritation of the dura.[39]

Computed tomography (CT) of the head serves as the main diagnostic tool for detecting acute subarachnoid hemorrhage (SAH), with a sensitivity approaching 100% during the initial 6 to 24 hours following symptom onset.[40] SAH resulting from aneurysmal rupture most commonly includes involvement of the basal cisterns, corresponding to the usual sites of cerebral aneurysms, and may spread widely throughout the subarachnoid space.[40] Depending on the hemorrhage volume and aneurysm location, blood may also track into the ventricles or brain parenchyma.

The modified Fisher scale is commonly employed to assess both the volume and pattern of subarachnoid hemorrhage and to estimate the likelihood of cerebral vasospasm after aneurysm rupture.[41]

FIGURE-9

Cerebral Aneurysms

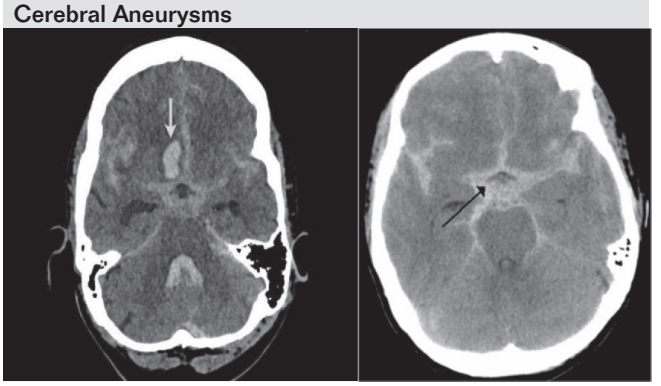
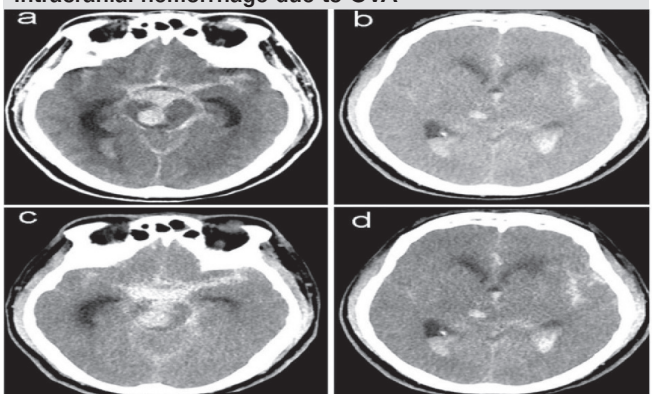


FIGURE-10

Intracranial hemorrhage due to CVA



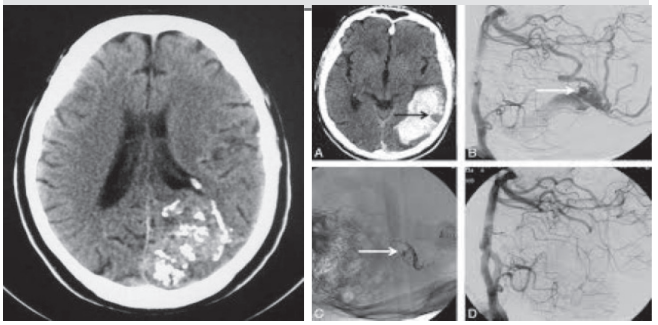
Cerebral Arteriovenous Malformations

Cerebral arteriovenous malformations (AVMs) are rare vascular abnormalities in which blood flows directly from cerebral arteries to veins through a tangle of small vessels, known as the "nidus," without an intervening capillary network.[42] These lesions are usually congenital or arise shortly after birth, with an estimated prevalence of 0.1% and an annual risk of intracranial hemorrhage of 2–4%. Intracerebral hemorrhage remains the most common clinical manifestation of cerebral AVMs.[42]

Rupture of a cerebral AVM frequently leads to intraparenchymal hemorrhage, intraventricular hemorrhage (IVH), or subarachnoid hemorrhage, all of which appear as hyperdense regions on head CT within the affected compartments.[43] AVM rupture is most commonly observed in younger patients, and the occurrence of IPH—especially in children—should prompt consideration of this diagnosis.[44] Evaluation of cerebral AVMs can be performed using CT angiography, MRI with MR angiography, or digital subtraction angiography (DSA). DSA is recommended for all patients with ruptured AVMs to identify nidal or perinidal aneurysms, which may necessitate urgent endovascular or surgical treatment to prevent recurrent bleeding.[45]

FIGURE-11

Cerebral AV-Malformations



Dural Arteriovenous Fistulae

Dural arteriovenous fistulae (dAVFs) are vascular abnormalities in which blood flows directly from dural or cerebral arteries into dural venous sinuses or cortical veins. They represent roughly 10–15% of intracranial arteriovenous shunts.[46,47] In contrast to cerebral AVMs, dAVFs do not possess a vascular nidus and are typically considered acquired lesions, often developing after trauma or dural venous sinus thrombosis, although the exact mechanism of their

formation remains uncertain.[47]

Clinical presentation is variable and may include headache, tinnitus, cranial nerve deficits, symptoms of increased intracranial pressure, or intracranial hemorrhage.[47] Ruptured dAVFs most commonly manifest as subarachnoid or intraparenchymal hemorrhage, which appears as hyperdense regions on acute computed tomography. Increased vascularity—arterial or venous—near major dural venous sinuses on CT angiography may suggest an underlying dAVF, although standard CTA is limited by poor temporal resolution. Time-resolved CTA has been shown to improve detection sensitivity.[48]

Magnetic resonance imaging is increasingly utilized for dAVF evaluation. Susceptibility-weighted imaging and arterial spin labeling sequences demonstrate high sensitivity for detecting arteriovenous shunting, potentially surpassing the diagnostic accuracy of CT and CTA.[49] However, direct comparisons between these advanced MRI techniques and CTA remain limited.

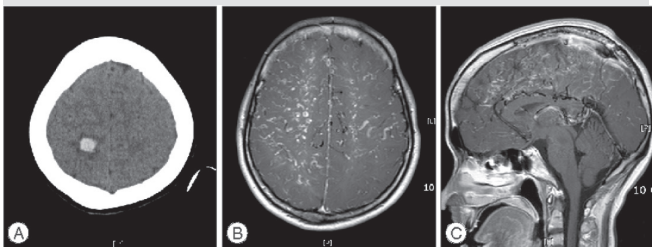
The likelihood of a dAVF rupturing largely depends on how the venous blood drains. The Cognard and Borden grading systems stratify hemorrhage risk based on venous drainage patterns.[48,50] Arteriovenous shunting increases venous sinus pressure, which can retrogradely transmit to cortical veins (Cognard IIB and IIA+IIB) or directly into cortical veins (Cognard III and IV), predisposing them to rupture and ICH. Patients exhibiting more severe clinical symptoms tend to have a greater risk of rupture. Digital subtraction angiography (DSA) continues to be the preferred method for assessing the angioarchitecture of dAVFs, while advanced MRI techniques, such as arterial spin labeling, can accurately find both the presence of a dAVF and cortical venous reflux that may necessitate treatment.

Cortical Venous or Venous Sinus Thrombosis

Dural venous sinus thrombosis (DVST) and cortical venous thrombosis are relatively rare but potentially challenging conditions to diagnose, with imaging playing a critical role in their identification.[51] Intracerebral hemorrhage caused by DVST or cortical venous thrombosis typically manifests as a headache, although patients may also exhibit signs of increased intracranial pressure or experience seizures.[51] DVST is more prevalent than cortical vein thrombosis and can arise from multiple factors, such as skull base infections, dehydration, hypercoagulable conditions, or compression by meningiomas or other dural tumors.[52]

FIGURE-12

Dural Arteriovenous Fistulae

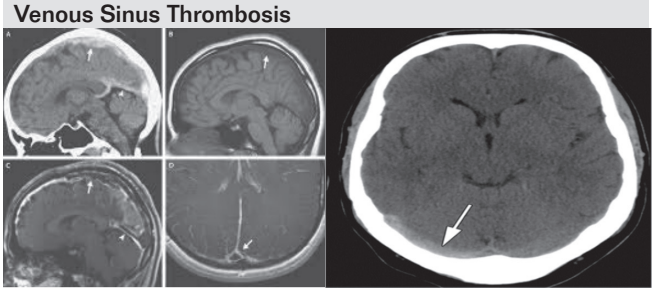


"Intracerebral hemorrhage associated with venous thrombosis generally does not correspond to an arterial distribution. Instead, it often occurs near the gray-white matter junction rather than along the cortical surface. When a hyperdense parenchymal hemorrhage is observed outside a typical arterial territory, DVST should be considered, especially if the major dural venous sinuses appear hyperdense.[53]

Definitive diagnosis typically requires additional imaging, such as CT venography, MR venography, or contrast-enhanced brain MRI. These studies can reveal the thrombosis as a filling defect in the affected venous structures or as a signal void on MR venography.[54] After treatment with anticoagulation or endovascular venous thrombolysis, non-invasive vascular imaging may be used to track the resolution or progression of DVST or cortical venous thrombosis.[54]

FIGURE-12

Venous Sinus Thrombosis

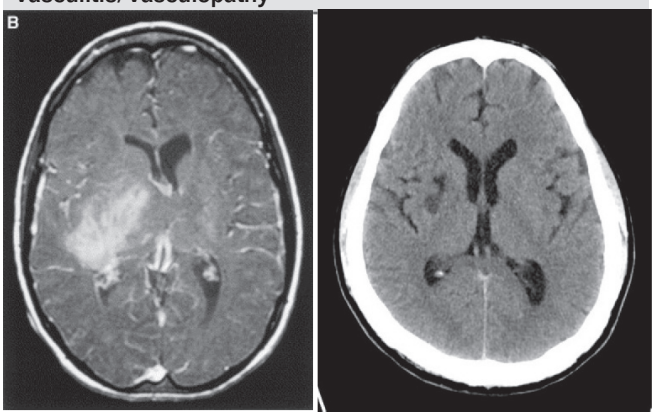


Vasculitis or Vasculopathy

Cerebral arterial vasculitis often appears with a variety of clinical manifestations, including headaches, behavioral changes, focal neurological deficits, or intracranial hemorrhage.[55] Among the types of ICH associated with vasculitis or vasculopathy, sulcal subarachnoid hemorrhage over the cerebral convexities is the most common.[56] Acute sulcal SAH resulting from vasculitis or vasculopathy is most reliably identified on head CT, which demonstrates hyperdensity within the affected cerebral sulci. Magnetic resonance imaging can also identify sulcal SAH, often visualized as hyperintensity on FLAIR or signal drop-out on GRE/SWI sequences.[57] In cases of non-traumatic sulcal SAH, further evaluation with digital subtraction angiography is warranted, particularly when computed tomography angiography is negative, to ensure accurate diagnosis of underlying vasculitis or vasculopathy.[58]

FIGURE-13

Vasculitis/Vasculopathy



Mycotic Aneurysms

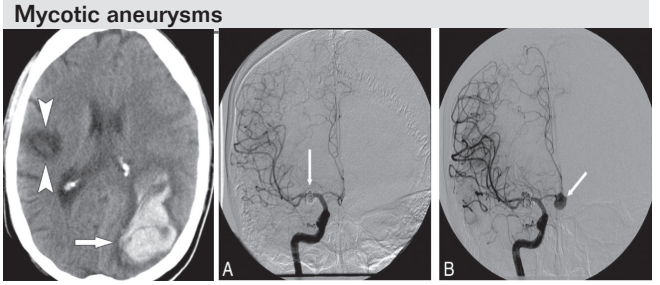
Mycotic aneurysms are outpouchings of arteries that typically occur in the distal cerebral vasculature.[59] These abnormalities represent pseudoaneurysms and often result from thromboembolic occlusion of distal vessels accompanied by inflammatory changes, which can cause minor ruptures at the occlusion site. Common etiologies include infective endocarditis, thrombi associated with prosthetic heart valves and various other cardiac abnormalities.[59]

Mycotic aneurysm rupture can result in sulcal subarachnoid hemorrhage (SAH), mimicking the distribution seen in vasculitis. In the acute stage, hyperdense areas are often visible within the cerebral sulci, particularly near the vertex. When mycotic aneurysms arise from more proximal intracranial vessels, the SAH may exhibit a more diffuse pattern. On GRE MRI sequences, these aneurysms may appear as hypointense foci within the subarachnoid space or adjacent to the gray-white matter junction.[60]

Non-invasive vascular studies, including CT angiography or MR angiography, can detect small arterial outpouchings or localized vessel dilation. However, small mycotic aneurysms can be missed by these modalities. Digital subtraction angiography (DSA) continues to be the preferred method for identification and characterization of these lesions and can also detect additional unruptured mycotic aneurysms.[61]

FIGURE-14

Mycotic aneurysms



CONCLUSION

Intracerebral hemorrhage (ICH) is a serious condition with a high risk of mortality. The wide variability in its imaging appearance reflects the diverse underlying pathologies. However, by carefully considering factors such as the pattern of hemorrhage, patient symptoms and demographics, and findings on vascular or post-contrast imaging, clinicians can often establish an accurate diagnosis. A thorough and systematic evaluation of each patient is essential to guide appropriate management and optimize outcomes.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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2	**Bella Virk:** Writing, data collection.
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