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IMAGING INTRACRANIAL HEMORRHAGE PATTERNS TO HELP TRAINEE DOCTORS FOR DIAGNOSIS, GUIDANCE AND ASSISTING IN OVERALL MANAGEMENT

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ABSTRACT

Intracranial hemorrhage is a prevalent ailment in Pakistan,[1] and the number of patients being referred to tertiary care hospitals due to intracranial bleeding has been continuously increasing,[1] owing to contemporary lifestyles and prevailing traffic conditions.[2] The causes of intracranial hemorrhage are multifaceted, encompassing Trauma,[3] Hypertension, Cerebral Amyloid Angiopathy, Hemorrhagic Conversion of Ischemic Infarction, Cerebral Aneurysms, Cerebral Arteriovenous Malformation (AVM), Dural Atrioventricular Fistula (DAVF), Vasculitis, Tumor Bleed, and Venous Sinus Thrombosis. Trainee doctors may commit errors while interpreting Computed Tomography (CT) scans of such patients, resulting in suboptimal patient management. The principal objective of this paper is to aid trainee physicians in the diagnosis of pathology and standardization of treatment by scrutinizing the imaging of these patients.[4,5]

KEYWORDS: Trauma, Epidural Hematoma (EDH), Subdural Hematoma (SDH), Subarachnoid Hemorrhage (SAH), Computed Tomography (CT), Traumatic Brain Injury (TBI).

AIM AND OBJECTIVE

The aim and objective of this paper is to help assist the trainee doctors in the diagnosis and management of intra cranial hemorrhage due to various causes.

INCLUSION CRITERIA

Imaging patterns of intracranial hemorrhage due to trauma, hypertension, and cerebral amyloid angiopathy, hemorrhagic conversion of ischemic infarction, cerebral aneurysms, cerebral AVM (arteriovenous malformations), dural AV (arteriovenous) fistula, vasculitis, and venous sinus thrombosis are discussed.

EXCLUSION CRITERIA

ICH due to primary or metastatic neoplasms is not discussed.

INTRODUCTION

In Pakistan, intracerebral hemorrhage (ICH) constitutes a highly significant medical occurrence that contributes to 8.74% of all strokes.[1] The incidence of intracerebral hemorrhage (ICH) stands at approximately 40 per 100,000 person-years, with a mortality rate of 40-42% within one month of presentation.[3] Due to the diverse causes[2] of intracranial hemorrhage and its multiple intracranial compartments, it is imperative that the treating physician has a comprehensive understanding of neuroimaging to identify the location and volume of hemorrhage.[4] This knowledge helps determine the risk of impending cerebral injury and guides the often urgent treatment of the patient. CT (computed tomography) is the preferred technique in the emergency evaluation of patients with suspected or known intracranial hemorrhage, and it is highly recommended for this purpose.[4,5]

TRAUMATIC ICH

Trauma has the potential to trigger intracranial hemorrhage by harming the arteries and veins inside the skull, resulting in bleeding on or within the brain tissue.[2] This bleeding can manifest in different ways, such as subarachnoid hemorrhage (SAH), epidural hematoma (EDH), subdural hematoma (SDH), hemorrhagic parenchymal contusions, and cerebral micro-hemorrhage from shear injury.[2] Each of these forms of traumatic intracranial hemorrhage possesses

distinct qualities and will be elaborated upon in the subsequent sections.

TRAUMATIC SUBARACHNOID HEMORRHAGE (SAH)

Traumatic subarachnoid hemorrhage (tSAH) refers to the presence of blood within the subarachnoid spaces, typically in the superficial sulci along the cerebral convexities.[6] It is the second most common acute brain injury finding on CT (computed tomography) in patients with traumatic brain injury (TBI), occurring in approximately 35% (range 11-60%) of cases.[7] Unlike non-traumatic SAH, which is commonly seen in basal cisterns and the lateral or Sylvain fissure, tSAH is more commonly found in the cerebral sulci. It often occurs adjacent to fractures of the skull and contusions, and has a particular affinity for the superior cistern or cistern of the great cerebral vein when present in basal cisterns.[8]

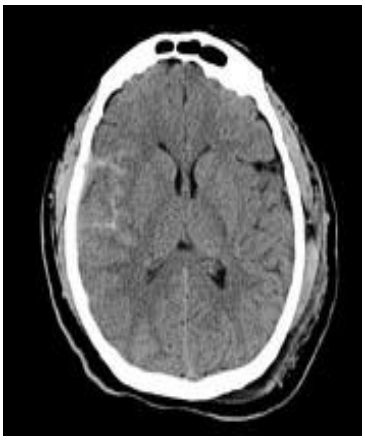
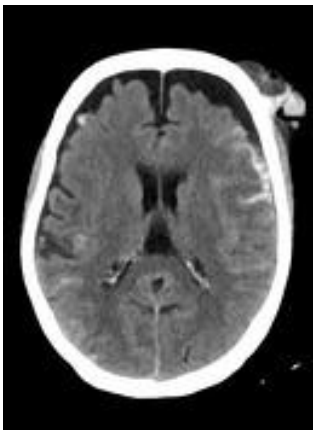


Fig 1



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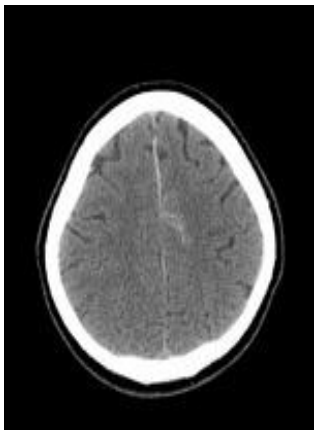

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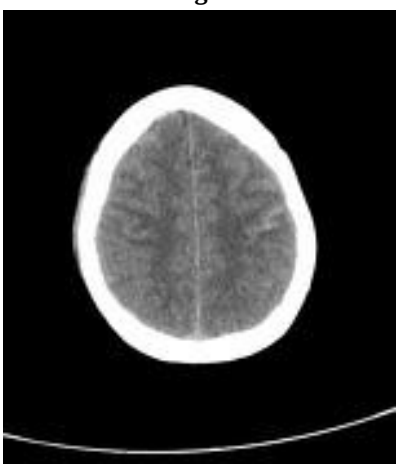

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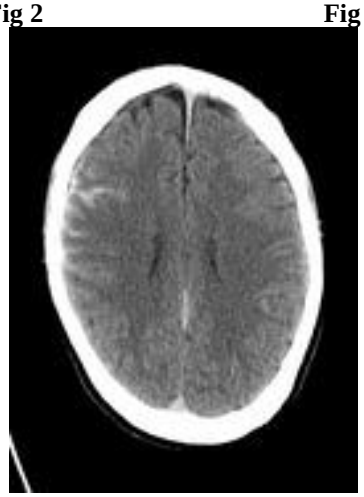


Fig 5

EPIDURAL HEMATOMA

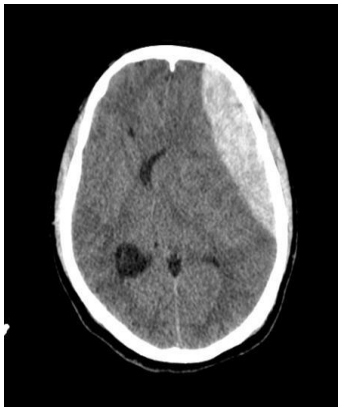
An Epidural Hematoma (EDH) is an acute emergency medical condition that occurs when blood accumulates in the epidural space between the inner table of the skull and the dura mater, the thick membrane that covers the brain. EDHs typically result from a skull fracture that tears an underlying blood vessel, causing blood to pool and build up in the epidural space.[9,4] Although less common than subdural hematomas, young adults are more likely to develop this condition.[10]

An acute epidural hematoma can be diagnosed using a head CT scan, which identifies a hyperdense collection in the epidural space. As this collection of blood exerts pressure on the brain, it can lead to symptoms such as headaches, confusion, and loss of consciousness.[10] However, one unique characteristic of epidural hemorrhage is that its extent is limited by the adjacent sutures between the bones of the skull, which can help doctors determine the best course of treatment.[9]

Ignoring an epidural hematoma can lead to severe complications, including permanent brain damage or death. As such, it is crucial to seek immediate medical attention if you experience symptoms of an EDH.

As a result of head trauma or injury, hemorrhaging can occur in the space between the skull and the outer layer of the brain known as the epidural space. As the volume of hemorrhage increases, the epidural blood extends along the inner table of the skull up to the nearest suture boundary. This boundary acts as a barrier that limits the extent of the hematoma.[9] As the epidural hemorrhage maximizes its lateral extension to the suture margins, the hemorrhage then increases in the superficial to deep dimension, which results in the so-called “crescentic” or

"biconvex" appearance of epidural hematomas.[11] This appearance is due to the pressure of the hematoma against the dura mater, the outermost layer of the meninges, which causes it to bulge.[11] The size and location of the hematoma determine the severity of symptoms, which can range from mild headaches to more severe neurological symptoms such as seizures, confusion, or loss of consciousness.[10] Therefore, prompt diagnosis and treatment are crucial in preventing further complications.



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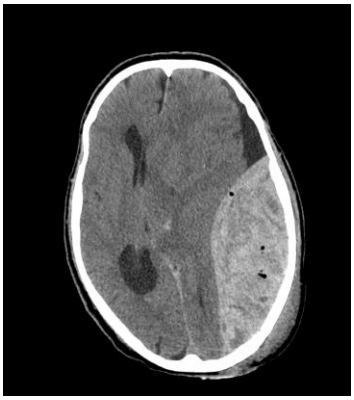
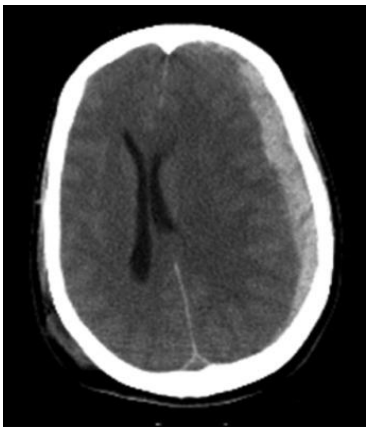


Fig 7

SUBDURAL HEMATOMA

Acute subdural hematomas can be easily identified on head CT scans as hyperdense hemorrhages located within the subdural space, which is situated between the arachnoid and pia mater.[12] Traumatic acute subdural hematoma remains one of the most lethal of all head injuries. It is widely held that the timing of operative intervention for clot removal is critical in determining overall outcome from acute subdural hematoma.[7,8] When detecting small subdural hematomas using CT scans, it's possible for them to be hidden by adjacent bony structures. To increase detection of these small hematomas, radiologists should adjust the window of the CT scan so that the density of blood is different enough from that of the adjacent bone.[14,15] A suggested window width of 130 with a window level of 30 is recommended for best results.[14]

Subdural hemorrhage occurs beneath the dura mater, which means its spread is not restricted by the bony sutures in the lateral dimension. As a result, subdural hematomas can cross suture lines, which differentiates them from epidural hematomas.[14] The falx cerebri confines subdural hematomas in the medial-to-lateral dimension, and the tentorium cerebri bounds them in the superior-to-inferior dimension. Subdural hematomas often spread along the ipsilateral structures unimpeded.



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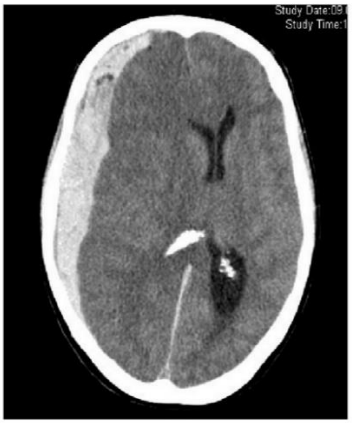


Fig 9

"CT scans of subdural hematomas"

HEMORRHAGIC PARENCHYMAL CONTUSION

Hemorrhagic parenchymal contusions, also known as brain bruises, are a type of traumatic brain injury that occurs due to significant head motion and impact.[16,17] These contusions are characterized by scattered areas of bleeding on the surface of the brain, usually along the undersurface and poles of the frontal and temporal lobes. The contusions are visible on CT scans as hyperdense hemorrhage within the brain tissue itself, and they result from micro vascular arterial or venous injury.[17]

It's worth noting that contusions can progress and expand over time, and in many cases, multiple hemorrhagic contusions may be present.[18] These contusions can

cause significant brain damage and result in a loss of function of the affected brain tissue. It is crucial to seek immediate medical attention if you suspect a brain injury, as the early detection and treatment of such injuries can significantly improve the chances of a full recovery.[18] These parenchymal contusions, which result from the impact of the head, are frequently located in the brain at the site of impact or on the opposite side, creating a "coup" and "counter-coup" pattern of head injury.[17] In other words, the impact of the head can cause not only immediate damage at the point of impact, but also a secondary injury at the opposite side of the brain due to the brain bouncing back after the initial impact. Therefore, it is essential to be aware of the symptoms of brain injury and to seek medical help without delay.[18] Hemorrhagic parenchymal contusions should prompt follow-up head imaging, as these contusions may grow in size in a relatively short period of time.[18]

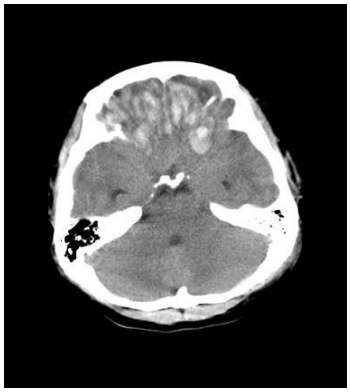


Fig 10

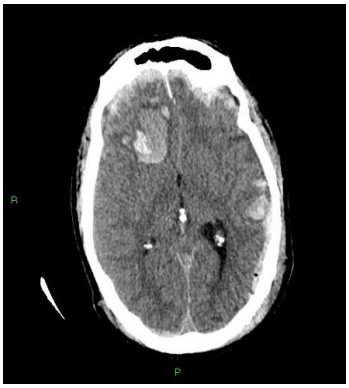


Fig 11

"CT scans of contusions"

CEREBRAL MICROHEMORRHAGE (DIFFUSE AXONAL INJURY)

Diffuse axonal injury is a severe medical condition that occurs when the brain suffers substantial damage due to rapid acceleration or deceleration, such as in a car accident or a fall. It happens when the brain shifts and rotates inside the skull, causing the tearing of its long connecting nerve fibers, known as axons.[7] This injury can cause significant damage to the brain's white matter, which is responsible for connecting different parts of the brain.[2]

Cerebral micro hemorrhage is a smaller form of post-traumatic hemorrhagic parenchymal contusion that frequently occurs in the white matter of the brain. Micro hemorrhages are small areas of bleeding that can damage the brain's white matter and lead to further complications.[19]

The effects of diffuse axonal injury are widespread and can result in coma and injury to many different parts of the brain.[19] The changes in the brain are often microscopic and may not be evident on CT or MRI (magnetic resonance imaging) scans. Therefore, physicians must rely on various clinical assessments to diagnose diffuse axonal injury accurately.

In conclusion, diffuse axonal injury is a severe and complex medical condition that can cause significant damage to the brain's white matter. The resulting changes in the brain are often microscopic and can be difficult to detect, necessitating careful and thorough clinical evaluations to diagnose and treat this condition.[20]

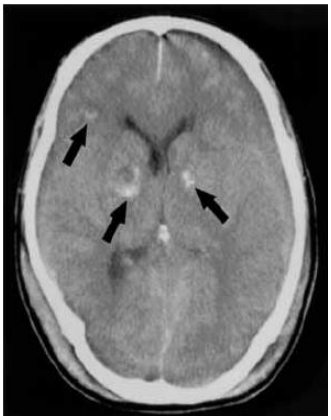


Fig. 12.

INTRAPARENCHYMAL HEMORRHAGE DUE TO HYPERTENSION

Intraparenchymal hemorrhage (IPH), which is caused by hypertension, usually affects patients in their 60s and 70s. The mortality rate for this condition is quite high, ranging from 30-50%.[21] Acute IPH can be identified through head CT scans as a hyperdense region of bleeding within the brain tissue. This bleeding typically occurs in the basal ganglia, cerebellum, or occipital lobes.[21]

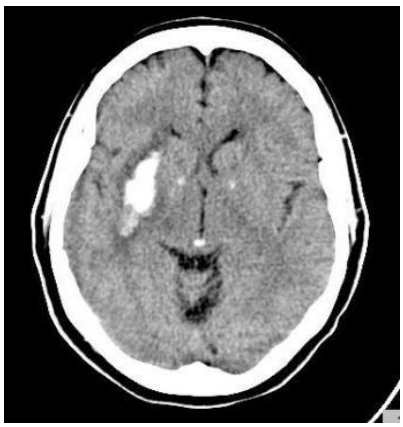
When a non-traumatic IPH (intraparenchymal hemorrhage) occurs specifically in the cerebral cortex, it is vital to consider other potential diagnoses beyond hypertension.[22] In addition, patients who are under 50 years of age and experience IPH should also be evaluated for other potential causes of bleeding, such as an underlying brain neoplasm or vascular malformation.

The size of the initial hemorrhage can range widely, from a relatively small hematoma (less than 1-2 cm) with no significant effect on the normal brain tissue nearby, to large hematomas that can cause significant local mass effect and even brain herniation.

In hypertension, early arteriolar smooth muscle growth is followed by smooth muscle cell death and collagen deposition. This eventually leads to arterial blockage or ectasia.[23] The latter causes Charcot-Bouchard aneurysm development and probable intracerebral bleeding.[23]

Patients with IPH (intraparenchymal hemorrhage) often undergo serial head imaging with CT and/or MRI to check for any expansion of the hematoma or mass effect caused by edema around the hemorrhage. These changes in the patient's condition may require a change in management, such as surgical decompression or evacuation.[24]

In the acute evaluation of intraparenchymal hemorrhage (IPH), CTA (Computed Tomography Angiography) is increasingly being used. The delayed phase CT images obtained after performing CTA of cerebral vessels can reveal active contrast extravasation as a hyperdense region of contrast pooling within the hematoma, which has been termed the "Spot Sign".[25] The presence of the Spot Sign is an important predictor of hematoma expansion and poor outcome. Therefore, this sign may be used for both prognostication and to guide more aggressive medical or surgical intervention. So, the Spot Sign[25] is a crucial marker that helps identify patients who require immediate medical or surgical intervention to prevent further complications.



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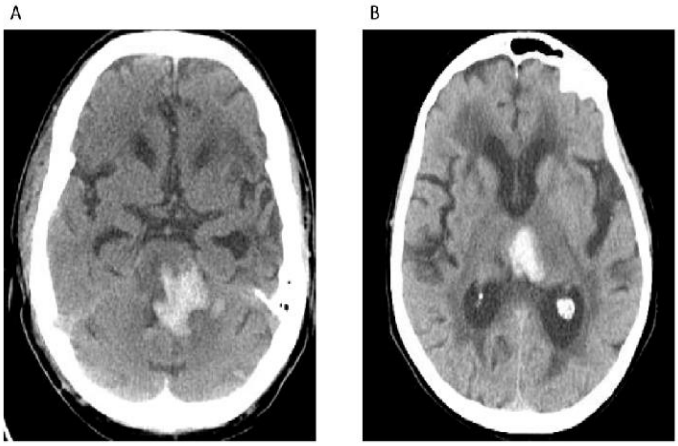


Fig 14

INTRAPARENCHYMAL HEMORRHAGE DUE TO CEREBRAL AMYLOID ANGIOPATHY

Cerebral amyloid angiopathy (CAA) occurs when amyloid-ß peptide builds up within the walls of cerebral arteries. This results in weakened arterial walls that can lead to cerebral micro hemorrhages, sulcal SAH, or larger cerebral IPHs.[26] Sulcal SAH caused by CAA is different from vasculopathy or vasculitis because it typically affects people over 60 years old and is associated with transient motor or sensation changes.[26] Additionally, there may be other areas of intracerebral hemorrhage present. In cases where intraparenchymal hemorrhage (IPH) is caused by cerebral amyloid angiopathy (CAA), specific imaging features help distinguish it from hypertensive hemorrhages. When IPH is caused by CAA, it is typically located in the white matter adjacent to the cerebral cortex, while sparing the basal ganglia, posterior fossa, and brainstem.[27] To determine the likelihood of IPH being secondary to CAA, the Boston criteria is used, which considers the number and distribution of cerebral hemorrhages and microhemorrhages.[28] Although a definitive diagnosis of CAA requires brain biopsy,[29] a hyperdense intra-axial hemorrhage in the subcortical region identified through CT is usually the first indication of IPH caused by CAA.[28] In addition, patients with CAA may show prominent diffuse white matter hypo-attenuation in both cerebral hemispheres, which indicates underlying micro-angiopathic changes. However, this finding is not always present in CAA patients. A brain MRI may further support the diagnosis of CAA through the presence of numerous small foci of susceptibility blooming in the bilateral cerebral white matter on GRE (Gradient-Recalled Echo) or SWI (Susceptibility-Weighted Imaging) sequences.[30]

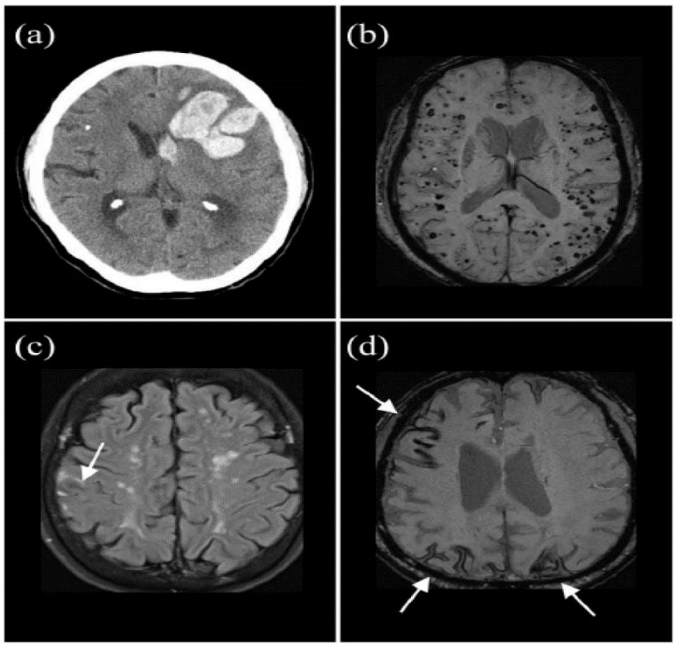


Fig. 15.

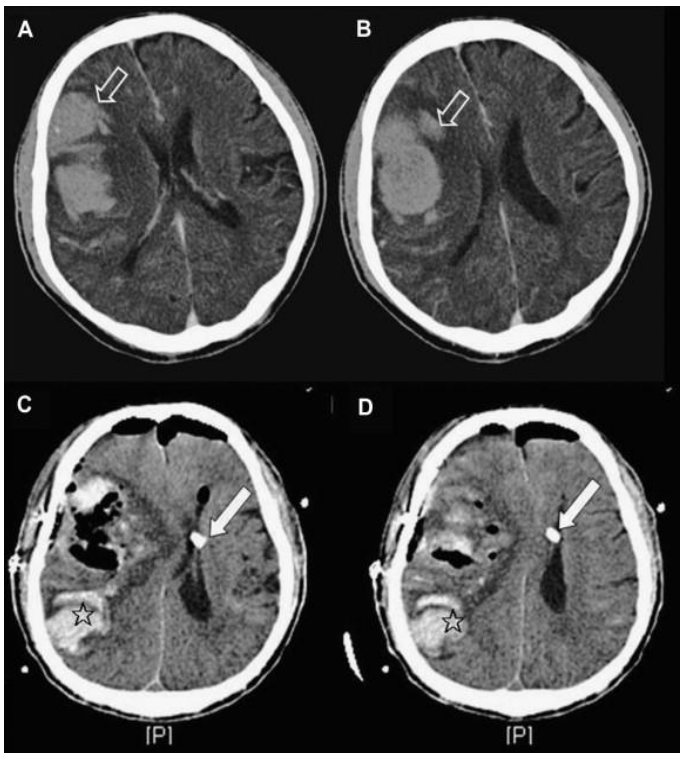


Fig. 16.

CEREBROVASCULAR CAUSES OF INTRACEREBRAL HEMORRHAGE

Intracerebral hemorrhage is a serious medical condition that can be caused by various cerebrovascular diseases. Among these, non-traumatic cerebrovascular disease is a common cause of intracerebral hemorrhage. The appearance and distribution of intracerebral hemorrhage secondary to cerebrovascular lesions can vary depending upon the specific causative lesion and its location in the brain.[31]

Some of the common cerebrovascular causes of intracerebral hemorrhage include hemorrhagic conversion of ischemic infarction, aneurysms, arteriovenous malformations (AVMs), dural arteriovenous fistulae (DAVF), vasculitis or vasculopathy, mycotic aneurysms, and cortical venous or venous sinus thrombosis.

Hemorrhagic conversion of ischemic infarction refers to the formation of hemorrhage in an area of the brain that has previously suffered from ischemic injury. Aneurysms

are abnormal bulges or out pouchings in blood vessels that can rupture and cause bleeding in the brain. AVMs are abnormal tangles of blood vessels that can also rupture and cause intracerebral hemorrhage. DAVFs are abnormal connections between arteries and veins in the brain that can cause intracerebral hemorrhage due to the high pressure in the arterial system. Vasculitis or vasculopathy refers to inflammation or damage to blood vessels, which can also lead to intracerebral hemorrhage. Mycotic aneurysms are rare and result from infection of the arterial wall. Finally, cortical venous or venous sinus thrombosis refers to the formation of blood clots in the veins that drain blood from the brain, which can cause intracerebral hemorrhage.

It is important to note that the treatment and prognosis for intracerebral hemorrhage can vary depending upon the underlying cause. Therefore, accurate diagnosis of the specific cerebrovascular disease that caused the intracerebral hemorrhage is crucial for effective treatment and management of the condition.

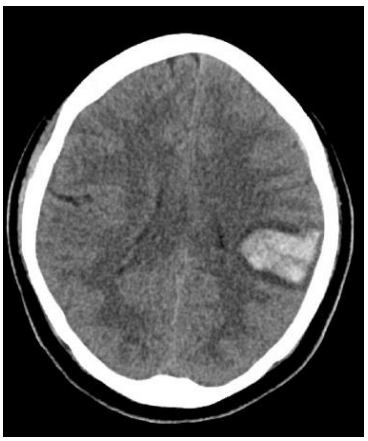
HEMORRHAGIC CONVERSION OF ISCHEMIC INFARCTION

Ischemic infarction is a medical condition that results from the blockage of a cerebral artery caused by a blood clot. The brain tissue that is affected by this condition is at risk of developing hemorrhagic conversion, which is a serious complication that occurs when bleeding into the damaged area of the brain.[32] This can happen in up to 43% of patients, and the risk of hemorrhagic conversion increases after intravenous or trans-arterial artery recanalization.[33,35]

The severity of hemorrhagic transformation that can occur after an ischemic infarction is graded using a spectrum of different types. Petechial hemorrhage along the infarcted tissue margin (Hemorrhagic Infarction type 1:HI1) and confluent petechial hemorrhage within the infarcted tissue (Hemorrhagic Infarction type 2:HI2) are the less severe types. Parenchymal hematoma involving 30% or less of the infarcted tissue with slight mass effect (Parenchymal Hematoma Type 1:PH1) is the third type, and parenchymal hematoma involving more than 30% of the infarcted tissue with significant mass effect (Parenchymal Hematoma Type 2:PH2) is the most severe type. Only PH2 (Parenchymal Hematoma Type 2) has significant clinical implications.[34]

Patients with ischemic infarction usually undergo serial neuroimaging scans to monitor for hemorrhagic conversion and the development of post-infarction vasogenic edema.[36] If a patient experiences a decline in their neurological status in the days following an ischemic stroke, prompt brain imaging by CT or MRI is necessary to rule out the development of symptomatic hemorrhagic conversion or worsening vasogenic edema. In some cases, this may require neurosurgical decompression.[37]

Reperfusion hemorrhage is a bleeding complication that can occur after successful endovascular stroke treatment. It may be difficult to detect because the iodinated contrast used in cerebral angiography can "stain" the brain. This is common in patients who have undergone successful endovascular stroke treatment. Dual-energy CT scanning has shown early promise in detecting hemorrhagic transformation in this patient population, although this approach is still under development.[38]



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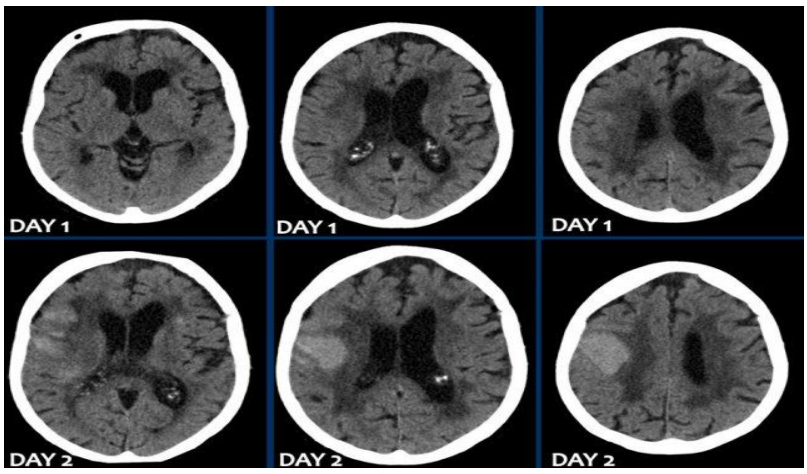
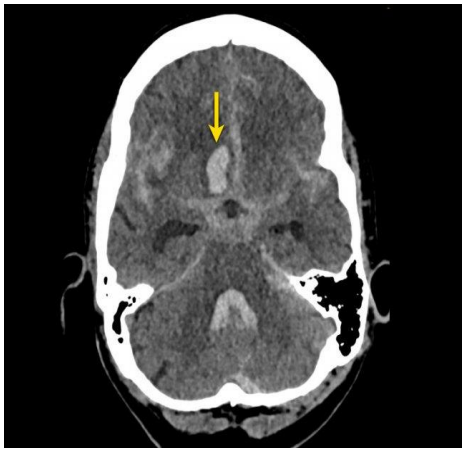


Fig. 18.

CEREBRAL ANEURYSMS

Cerebral aneurysms are localized dilations in arteries that traverse the cerebral surface, representing areas of arterial wall weakness that are predisposed to rupture.[39] The rupture of cerebral aneurysms is typically characterized by the abrupt onset of an intense headache, known as the worst headache of a patient's life, which results from hemorrhage into the subarachnoid space and irritation of the dura.[39]

Head computed tomography (CT) is an instrumental diagnostic modality that displays a sensitivity of nearly 100% for the detection of acute subarachnoid hemorrhage (SAH) in the initial 6-24 hours of symptom onset.[40] The SAH secondary to cerebral aneurysm rupture primarily involves the basal cisterns, where most cerebral aneurysms are situated, and frequently extends diffusely throughout the subarachnoid space.[40] Depending on the volume of the hemorrhage and the location of the ruptured aneurysm, SAH may also extend into the ventricles and brain parenchyma. The modified Fisher grade represents a widely used scale that quantifies the magnitude and distribution of the hemorrhage and predicts the likelihood of developing cerebral artery vasospasm after aneurysm rupture.[41]



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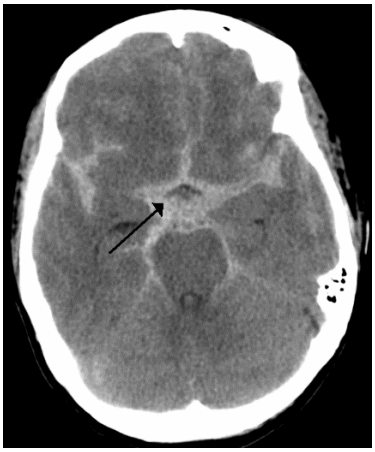

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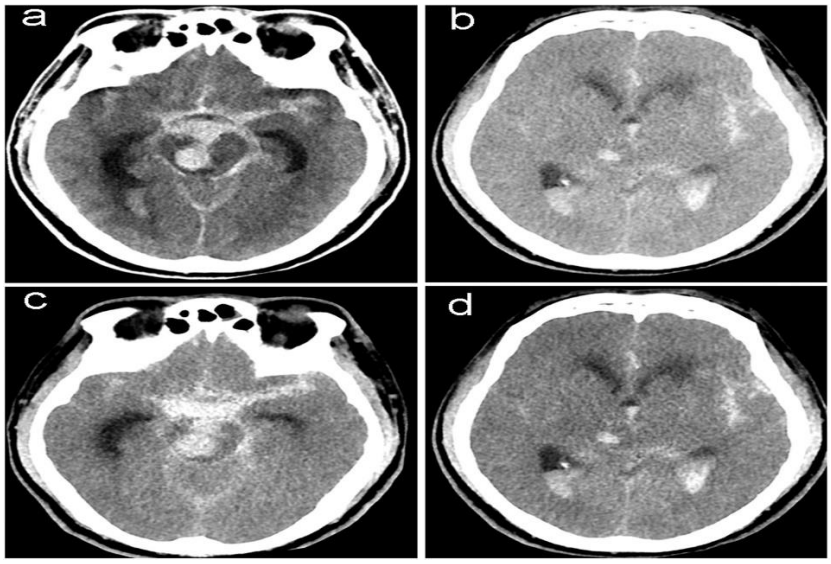

Fig. 21.

CEREBRAL ARTERIOVENOUS MALFORMATIONS

Cerebral arteriovenous malformations (AVMs) are infrequent lesions characterized by aberrant arteriovenous shunting between cerebral arteries and veins through various small channels without the presence of an intervening capillary bed (termed the "nidus").[42] These malformations are considered congenital or acquired shortly after birth and have a prevalence of 0.1%, with an estimated 2-4% annual risk of intracranial hemorrhage. However, ICH (intracerebral hemorrhage) is the most common manifestation of cerebral AVMs.[42]

The rupture of cerebral AVMs frequently results in intraparenchymal hemorrhage (IPH), intraventricular hemorrhage (IVH), or subarachnoid hemorrhage (SAH), which are identified by head CT in the acute setting as hyper density within these compartments.[43] Cerebral AVM rupture is most commonly observed in young patients, and the presence of IPH, particularly in

pediatric patients, should alert physicians to this diagnosis.[44] Cerebral AVMs can be identified by computed tomography angiography (CTA), magnetic resonance imaging with MR angiography, or digital subtraction angiography (DSA). DSA should be conducted in every patient presenting with a ruptured cerebral AVM to determine whether there is a nidal or perinidal aneurysm that may require immediate endovascular or surgical treatment to prevent a recurrent acute hemorrhage.[45]

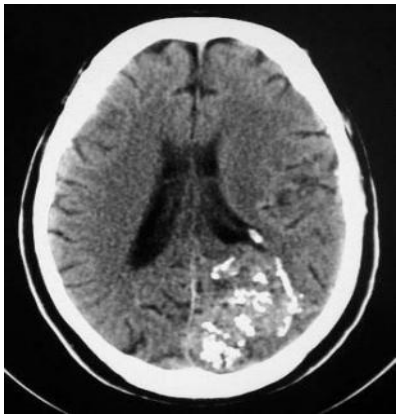

Fig. 22.

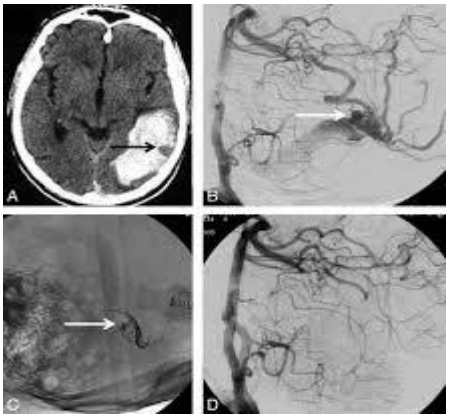

Fig. 23.

DURAL ARTERIOVENOUS FISTULAE

Dural arteriovenous fistulae (DAVF) are vascular lesions that are characterized by arteriovenous shunting due to direct fistulous connections between dural or cerebral arteries and the dural venous sinuses or cortical veins. These lesions account for approximately 10-15% of intracranial arteriovenous shunts.[46,47] Unlike cerebral arteriovenous malformations, DAVF lacks a vascular nidus and are believed to be acquired lesions that arise secondary to trauma or dural venous sinus thrombosis, although their etiology remains poorly understood.[47]

The clinical presentation of DAVF is varied and includes headache, tinnitus, cranial nerve deficits, symptoms of increased intracranial pressure.[47] In particular, ruptured DAVF is most commonly associated with subarachnoid hemorrhage or intraparenchymal hemorrhage, which is typically observed in the acute setting by computed tomography as a hyperdense hemorrhage in these spaces. The presence of an increased number of vessels, which may be arteries or veins, near the major dural venous sinuses on computed tomography angiography (CTA) may suggest an underlying DAVF. However, the lack of temporal resolution on CTA limits its utility in the detection of DAVF. Recent studies have shown that time-resolved CTA may increase the sensitivity of this modality in the detection of DAVF.[48]

Magnetic resonance imaging (MRI) is increasingly being used for the detection and evaluation of DAVF.[49] Specifically, susceptibility-weighted imaging and arterial spin labeling sequences have demonstrated excellent sensitivity for the detection of arteriovenous shunting, suggesting that MRI may be superior to CT and CTA in the detection of DAVF.[49] However, to our knowledge, there has been no direct comparison between MRI using these techniques and CTA.

The risk of DAVF rupture is based on the pattern of venous outflow from the fistula. The Cognard and Borden grading systems are commonly used to describe the risk of hemorrhage due to a DAVF.[48] The risk of hemorrhage is related to the venous egress of the fistula. Arteriovenous shunting due to DAVF increases the pressure in the venous sinus, which may result in the retrograde transmission of this increased pressure to cortical veins that drain into the sinus (Cognard IIB and IIA+IIB lesions) or into the cortical vein itself (Cognard III and IV lesions).[50] Cortical veins that are unable to accommodate the increased pressure may rupture and cause intracerebral hemorrhage. Patients presenting with more severe symptoms are also more likely to develop DAVF rupture.[48] These grading systems are based upon digital subtraction angiography, which remains the gold standard in the evaluation of the angioarchitecture of these lesions.[50] Advanced MRI techniques, such as arterial spin labels, have been shown to be highly accurate in identifying the presence of a DAVF and in determining whether there is cortical venous reflux that should prompt treatment.

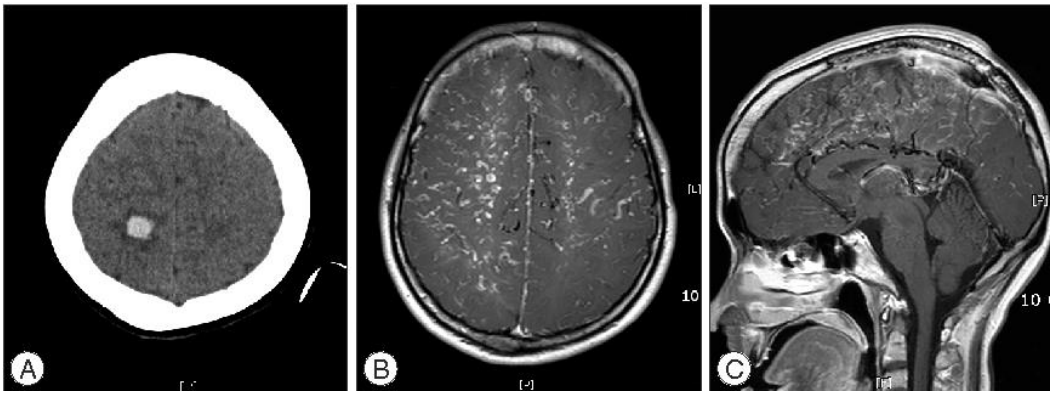

Fig. 24.

CORTICAL VENOUS OR VENOUS SINUS THROMBOSIS

Dural venous sinus thrombosis (DVST) and/or cortical venous thrombosis are infrequent but potentially confusing medical conditions, and imaging plays a crucial role in their identification.[51] Intracerebral hemorrhage due to DVST or cortical venous thrombosis usually manifests as a headache, although signs of elevated intracranial pressure or seizures may also occur.[51] DVST is more prevalent than cortical vein thrombosis, and it may result from a variety of factors, including skull base infections, dehydration, hypercoagulable states, and compression from meningiomas or other dural tumors.[52] Venous thrombosis-related ICH is usually not distributed in an arterial pattern and is centered closer to the grey-white matter junction rather than the cortex.

If a hyperdense parenchymal hemorrhage that does not conform to an arterial territory is identified, DVST may be suggested by the hyperdense appearance of the major intracranial dural venous sinuses.[53] To make an accurate diagnosis, further testing using CT venography, MR venography, or post-gadolinium brain MRI may be required. These tests will reveal the venous thrombosis as a filling defect within the affected venous structures or a lack of signal within these structures on MR venography.[54] After anticoagulation or endovascular venous thrombolysis treatment, non-invasive vascular imaging may also be used to track the progress of DVST or cortical venous thrombosis.[54]

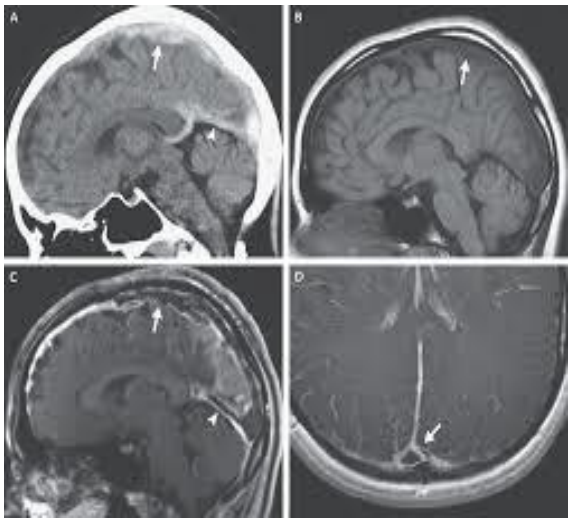

Fig 25

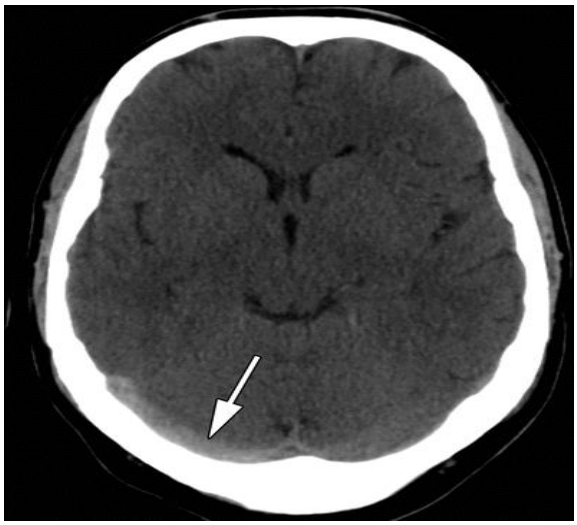

Fig26

VASCULITIS OR VASCULOPATHY

Cerebral arterial vasculitis can manifest through various symptoms including headaches, behavioral changes, neurologic deficits or intracranial hemorrhage.[55] Sulcal subarachnoid hemorrhage (SAH) in proximity to the cerebral convexity is the most common form of ICH that is caused by vasculitis or vasculopathy.[56] The identification of acute sulcal SAH due to vasculitis or vasculopathy is primarily performed by head CT, which shows hyper density within the cerebral sulci. Nonetheless, the same can also be identified by MRI through sulcal hyperintensity on FLAIR sequences or hypointense signal abnormality on GRE or SWI sequences.[57] Sulcal SAH in the absence of trauma requires further evaluation through DSA, particularly in cases where CTA results come out negative, to ensure a correct diagnosis of vasculitis or vasculopathy.[58]

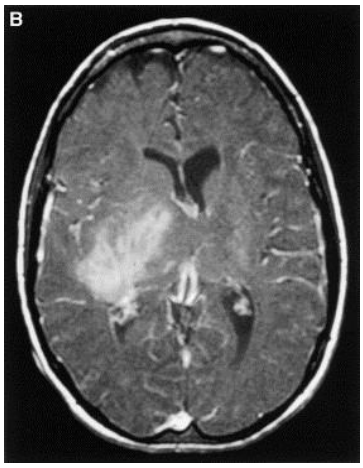

Fig.27

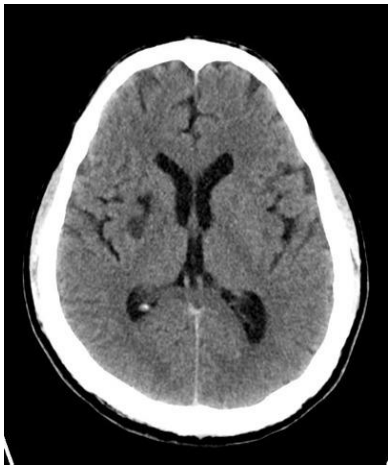

Fig 28

MYCOTIC ANEURYSMS

Mycotic aneurysms are arterial outpouchings that typically arise from distal cerebral arteries.[59] These lesions are actually pseudo-aneurysms, and they are commonly caused by distal vessel thromboembolic occlusion with associated inflammatory changes resulting in small tears at the site of vessel occlusion. Endocarditis or thrombi related to mechanical cardiac valves and other cardiac anomalies are common causes of mycotic aneurysms.[59]

The rupture of a mycotic aneurysm may result in sulcal SAH similar to vasculitis. Hyper density within the cerebral sulci, often near the vertex, is identified in the acute phase. Mycotic aneurysms arising from the more proximal intracranial vessels may present with a diffuse pattern of SAH. GRE MRI sequences may reveal mycotic aneurysms as foci of hypo intensity in the subarachnoid space or in proximity to the grey-white matter junction.[60]

CTA or MR Angiography may demonstrate a subtle arterial outpouching or a focal increase in the caliber of the affected vessel. However, non-invasive vascular imaging can miss small mycotic aneurysms which should be identified and characterized through DSA. Additionally, DSA can identify any other associated mycotic aneurysms that are unruptured.[61]

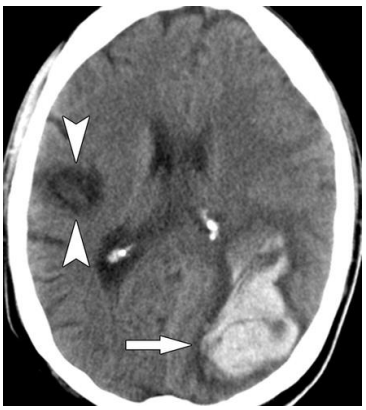

Fig. 29.

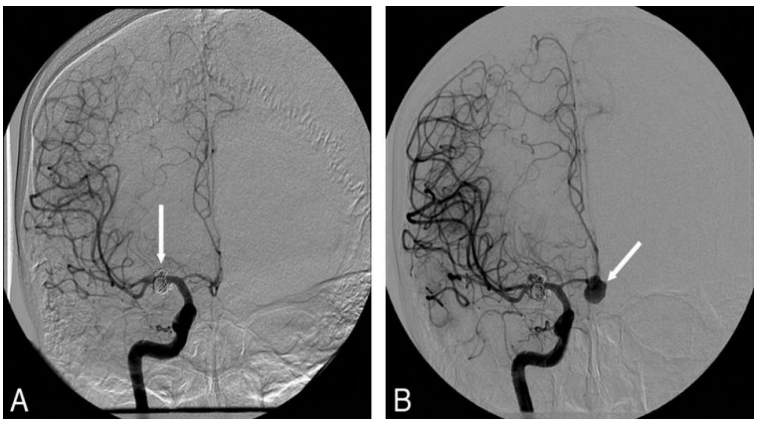

Fig. 30.

CONCLUSION

Intracranial hemorrhage is a serious medical condition that has a high mortality rate. One of the reasons why there is a wide variation in the imaging appearance of intracranial hemorrhage is due to the different types of pathology that can lead to this condition. However, by taking into account various factors such as the pattern of intracranial hemorrhage, patient symptoms and demographics, as well as associated vascular or post-contrast imaging, it is possible to arrive at a diagnosis in most situations. Therefore, healthcare professionals must conduct a thorough evaluation of the patient's condition in order to determine the best course of treatment.

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