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Uncommon Patterns of Intracranial Aneurysms: A Pictorial Essay

Intracranial aneurysms are relatively common, with a prevalence of approximately 4%. Unruptured aneurysms may cause symptoms mainly due to a mass effect, but the real danger is when an aneurysm ruptures, leading to a subarachnoid hemorrhage. Most aneurysms are asymptomatic and will not rupture, but they grow unpredictably and even small aneurysms carry a risk of rupture. There are many risk factors for the development of intracranial aneurysms, both inherited and acquired. Females are more prone to aneurysm rupture, with subarachnoid hemorrhage (SAH) 1.6 times more common in women. The prevalence of aneurysm increases in certain genetic diseases. There are four main types of intracranial aneurysms: saccular, fusiform (atherosclerotic or dolichoectatic type/congenital type), dissecting, and the mycotic type. The saccular type accounts for 90% of intracranial aneurysms. We observed some patients who were referred for diagnosis and further angiographic evaluation. They had presented with various histories and clinical findings. Their imaging findings such as CT scan, CTA, MRI and MRA findings were unusual. Every one of these patients has an uncommon form of aneurysm which could be misdiagnosed with other conditions or might need special and different treatment and knowledge of these situations could be of great help.

Keywords: Subarachnoid Hemorrhage, Fusiform Aneurysm, Dolichoectasia, Bilobed Aneurysm, Mycotic Aneurysm, Giant Aneurysm

Introduction

Subarachnoid hemorrhage (SAH) causes approximately 5% of all "strokes." The most common cause of nontraumatic SAH is ruptured intracranial aneurysm (85% of causes). Intracranial aneurysms are generally classified according to their phenotype (gross pathologic appearance). Three general categories are recognized: (1) Saccular aneurysms (also known as "berry" aneurysms); (2) fusiform aneurysms; and (3) the rare, recently-described "blood blister-like" aneurysms. Saccular aneurysms are round or lobulated focal outpouchings that typically arise from areas of high hemodynamic stress; namely, major vessel bifurcations. Fusiform aneurysms are elongated, spindle-shaped dilation of vessels that can be associated either with atherosclerotic vascular disease (ASVD) or non-atherosclerotic pathology such as connective tissue disorders. All true intracranial aneurysms lack one or more layers of the normal arterial wall, usually the internal elastic lamina and a thinned or absent muscularis layer. Intracranial pseudoaneurysms lack all vessel wall layers and are typically a cavitated paravascular hematoma that may or may not communicate directly with the true arterial lumen. The wall of the rare but dangerous "blood blister-like" aneurysm is tissue-paper-thin. This entity, now well-known to neurosurgeons but rarely discussed in the imaging literature, is often subtle on, and underdiagnosed at cerebral angiography.[1]

The other type of aneurysm is the inflammatory (mycotic type) aneurysm. [2] Many risk factors are important for the development of intracranial

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aneurysms, both inherited and acquired. Females are more prone to aneurysm rupture, with SAH 1.6 times more common in women.[3-5]

Blood spilling into the cerebrospinal fluid (CSF) causes the symptoms of SAH.[6,7] The clinical manifestations of unruptured aneurysms, however, are much more subtle. Only 10-15% of intracranial aneurysms are symptomatic,[8,9] with the majority being identified incidentally during evaluation for other conditions. The symptoms are primarily due to the mass effect or possibly from minimal leakage of blood which irritates the meninges, though not enough to be classified as a hemorrhage.[3,10] These symptoms may be a warning sign of an impending rupture, as 10% to 43% of patients with SAH report experiencing a sentinel headache two months preceding the rupture.[11]

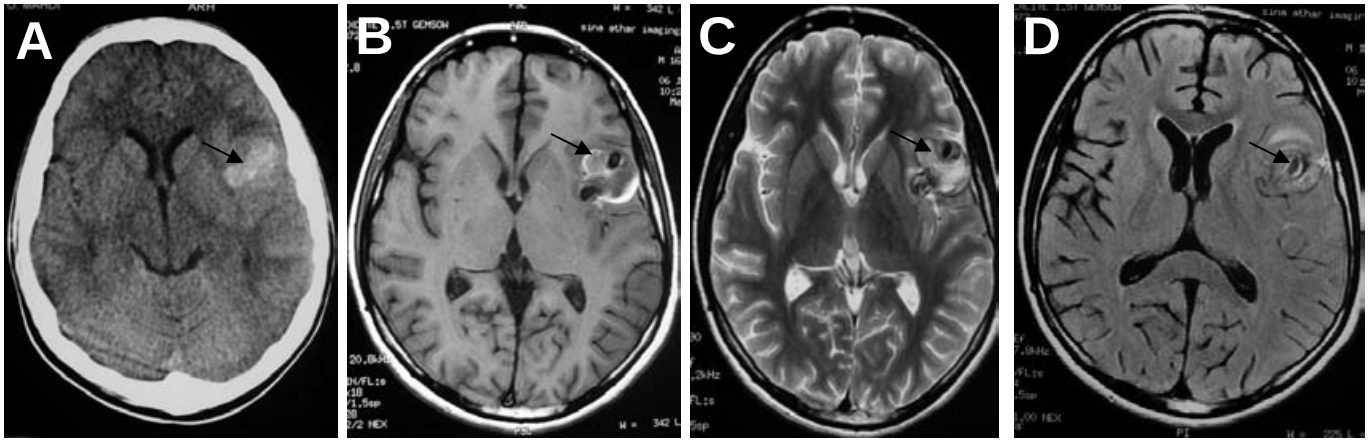
Natural history of unruptured aneurysms is such a hot-topic because knowing the likely course of aneurysms will play a pivotal role in determining the appropriate management. Unlike the more predictable course of abdominal aortic aneurysms, where the lesion grows in size and rarely ruptures before it reaches the threshold diameter of 5.0 cm, the data for intracranial aneurysms are much less clear. Despite criticisms with ISUIA (International Study of Unruptured Intracranial Aneurysms), this was indeed a landmark study, and the clinical guidelines are based largely on the data from this trial. In patients with incidental aneurysms smaller than 10 mm, the current recommendations are observation rather than intervention. Exceptions include patients with growing lesions and those approaching 10mm, lesions with irregular morphological and hemodynamic features, and patients with multiple first or second degree relatives with a history of aneurysmal SAH.[12]

The parameters suggesting that a patient should not undergo intervention include: lack of symptoms, aneurysm size larger than 7 mm, lesion in the anterior circulation, age older than 64 years, and no personal or family history of SAH. Similarly, patients younger than 50 years with symptomatic aneurysms larger than 25 mm located in the posterior circulation and a personal or family history of SAH should undergo intervention.[3] Of course, most cases fall somewhere in between these two extremes and the appropriate management is not obvious.

Case Presentation

Fusiform aneurysm of the left MCA: A 16-year-old boy presented with nausea, vomiting and loss of consciousness. Non contrast CT scan study showed SAH. There was no positive finding in his past medical history. In further studies, the evidence of fusiform aneurysmal dilatation of the left MCA was detected that was referred to our center for intervention procedure. At the time of admission, the boy was fully conscious and all cranial nerves, motor, sensory, gait and cerebellar examinations were intact. CT scan study without contrast revealed SAH in the left sylvian fissure (Fig. 1A). MRI study without Gd showed an abnormal signal in the left sylvian fissure and a signal void area within it (Figs. 1B-D) and also a nonspecific diffusion finding with a small area of restriction (Fig. 1E). DSA performed which showed fusiform dilatation of the left MCA from the distal part of M1 (Fig. 1F). The patient underwent surgery and partial clipping was performed along the longitudinal course of the superior part of segments M1-M2 of the left MCA (fusiform aneurysm). In the control angiography, 6 days after the surgery, there was remaining dilatation in distal M1, connecting to normal diameter inferior M2 trunk.

Vertebrobasilar dolichoectasia (atherosclerotic fusiform aneurysm of vertebrobasilar artery). A 68-year-old man presented with headache and dysarthria from 2 years ago and studies showed vertebrobasilar dilatation related to dolichoectasia. He was on medical therapy till 2 months ago when new symptoms — weakness and occasional diplopia—appeared. He had a long past history of hypertension. On admission, he was fully conscious and in the cranial nerve examination, findings showed ninth and tenth cranial nerve palsies and a disturbed gag reflex. In the motor examination, there is evidence of weakness in both upper and lower limbs with the force of 4/5. Sensory examination showed non-dermatomal patchy hypoesthesia in the right upper and lower limbs. The tendon reflexes were intact and he had an unsteady gait. There was some evidence of disturbed left cerebellar exams. CT scan without contrast showed a hyperdense lesion suspicious for a vascular lesion infront of the pons and brainstem (Fig. 2A). CECT and also Gd-MRI study revealed vertebrobasilar doli-



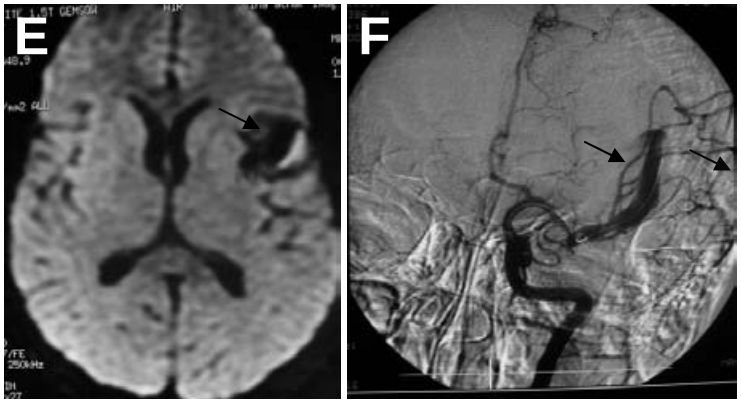


Fig.1. A 16-year-old boy presenting with nausea, vomiting and loss of consciousness.
A. Non-contrast CT scan study revealed SAH in the left sylvian fissure.
B-D. MRI study without Gd showed an abnormal signal in the left sylvian fissure and a signal void area within it.
E. A non-specific diffusion finding with a small area of restriction.
F. DSA showed fusiform dilatation of the left MCA from the distal part of M1.

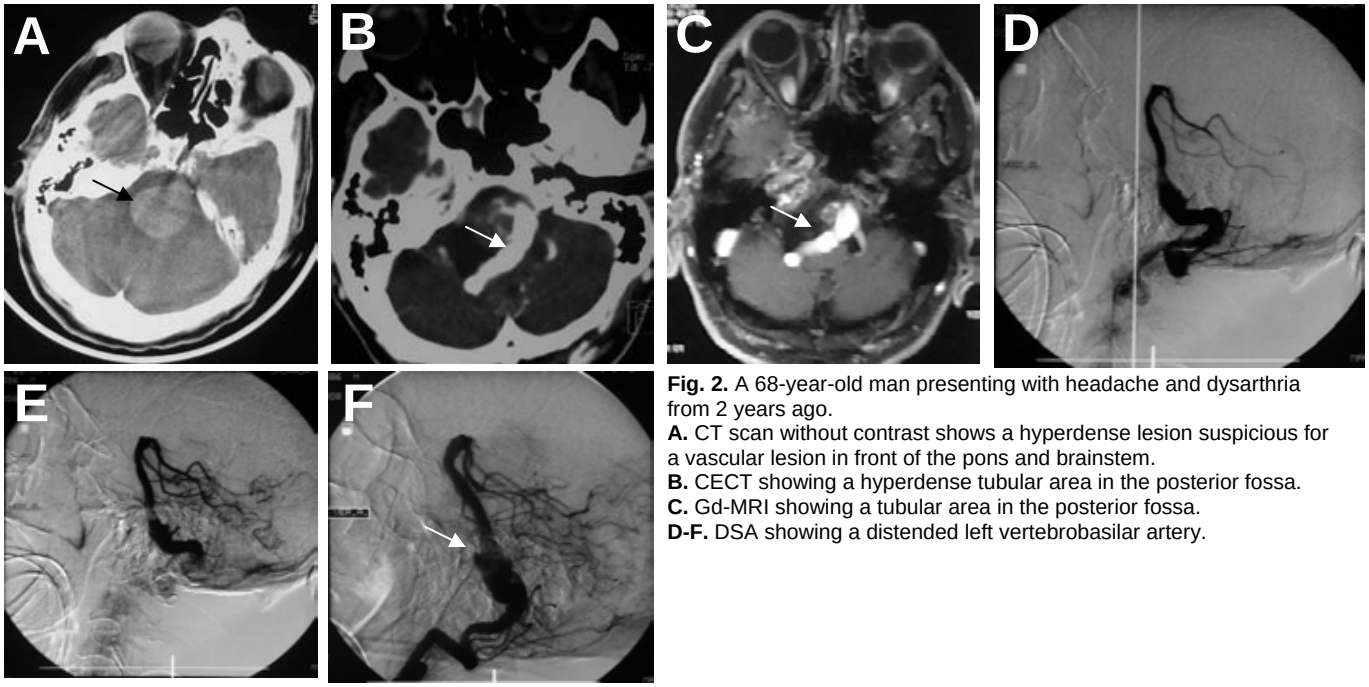


Fig. 2. A 68-year-old man presenting with headache and dysarthria from 2 years ago.
A. CT scan without contrast shows a hyperdense lesion suspicious for a vascular lesion in front of the pons and brainstem.
B. CECT showing a hyperdense tubular area in the posterior fossa.
C. Gd-MRI showing a tubular area in the posterior fossa.
D-F. DSA showing a distended left vertebrobasilar artery.

choectasia which was more severe on the left side (Figs. 2B & C) and was confirmed with DSA (Figs. 2D-F).

Giant aneurysmal dilatation of the intracavernous part of the right internal carotid artery. A 58-year-old man who was referred for the evaluation of progressive headache and occasional diplopia underwent a further angiographic study. In his past medical history there was no evidence of positive findings. At the time of the study he was fully conscious and in the cranial nerve examination he had right side sixth nerve palsy (Fig. 3A). There was no abnormal finding in the motor, sensory, gait and cerebellar exams. Imaging findings showed a giant aneurysmal dilatation

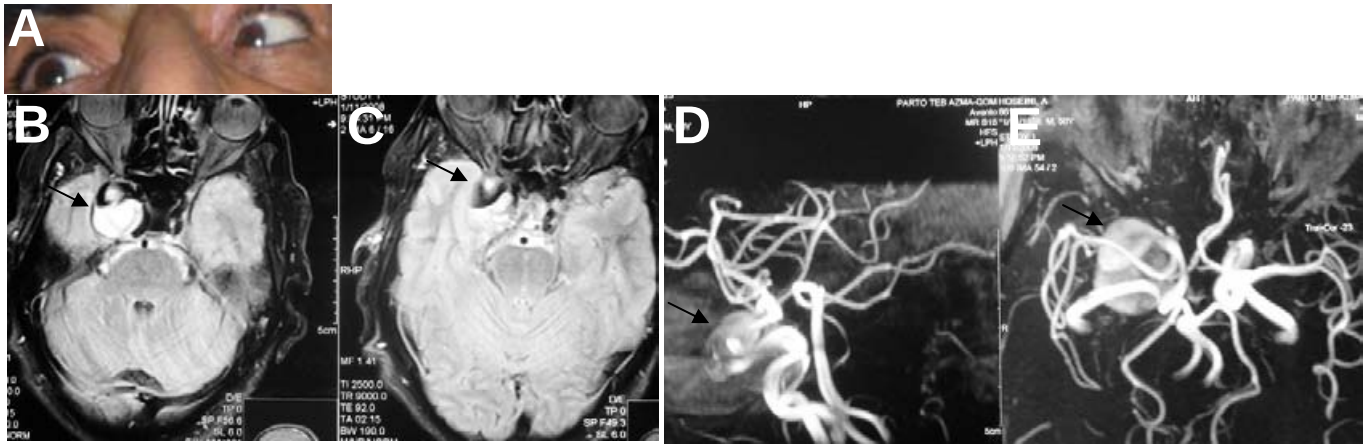


Fig. 3. A 58-year-old man with a past history of progressive headache and occasional diplopia.
A. Right side sixth nerve palsy in the cranial nerve examination.
B&C. Flair-sequence Brain MRI revealed a giant aneurysmal dilatation of the intracavernous part of the right internal carotid artery.
D&E. MR Imaging showing a giant aneurysmal dilatation of the intracavernous part of the right internal carotid artery.

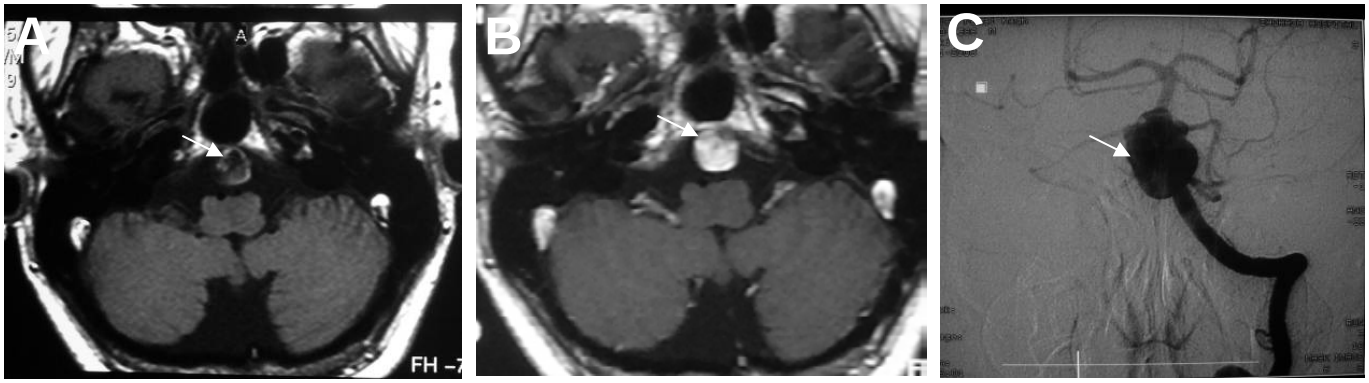


Fig. 4. A 51-year-old woman referred for angiographic study with a history of tinnitus in the left ear.
A. Non-contrast T1W MRI showing an abnormal signal void lesion in front of the pons.
B. T1W MRI study showing an abnormal signal void lesion in front of the pons with great enhancement after gadolinium study.
C. MRA imaging detected a giant basilar aneurysm.

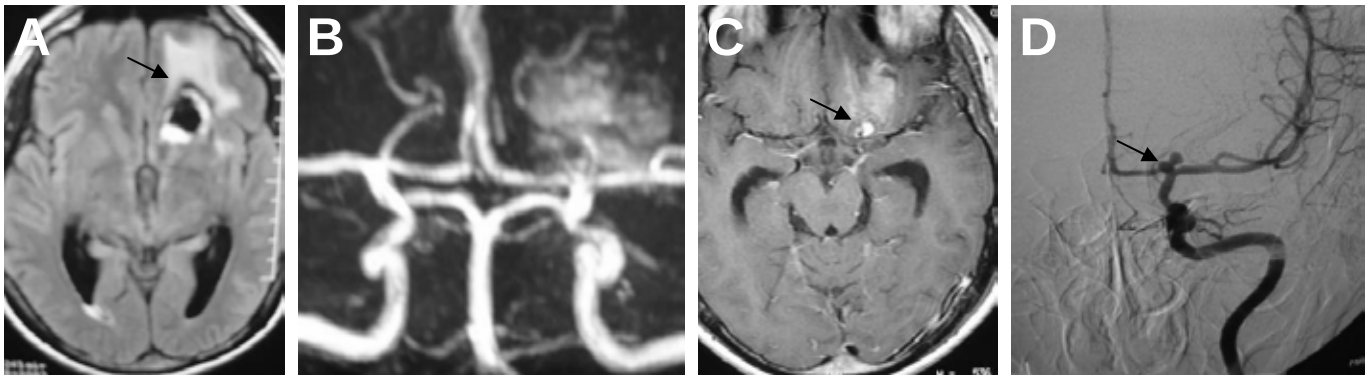


Fig. 5. A 30-year-old man with an acute onset of loss of consciousness and right hemiparesia.
A. MRI showing non-specific abnormal signal in the left frontal lobe.
B. MR angiography showing no significant lesion.
C. Gadolinium study in the left parasellar region detected an abnormal lesion which was suspicious for a bilobed aneurysm.
D. Conventional angiographic study confirmed a bilobed aneurysm of the left internal carotid artery's tip.

of the intracavernous part of the right internal carotid artery (Figs. 3B-E).

Giant basilar aneurysm. A 51-year-old woman was referred for angiographic study with a history of tinnitus in the left ear from 8 months ago and audiometric studies showed 50% hearing loss in both ears. MRI study incidentally showed probable aneurysmal dilatation in the prepontine cistern. There was no positive past medical history finding and his physical examination was negative. MRI study showed an abnormal signal void lesion in front of the pons with great enhancement after gadolinium study (Figs. 4A

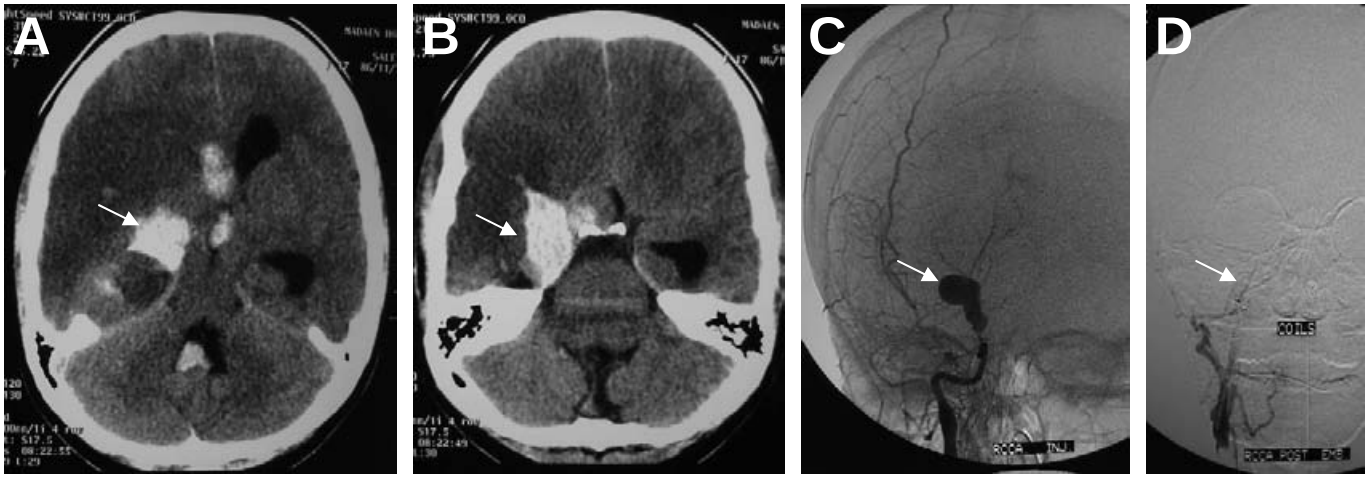


Fig. 6. A 17-year-old woman presenting with massive IVH, SDH, SAH, hydrocephaly and infarct in the right fronto-temporo-parietal lobe.
A. CT scan study without contrast demonstrating an infarction in the right fronto-temporo-parietal lobe.
B. CT scan study without contrast showed a hyperdense area in the right parasellar region.
C. DSA confirmed aneurysm of the C5 segment of the right ICA.
D. Post-coiled DSA (the arrow shows the coil in the location of the aneurysm).

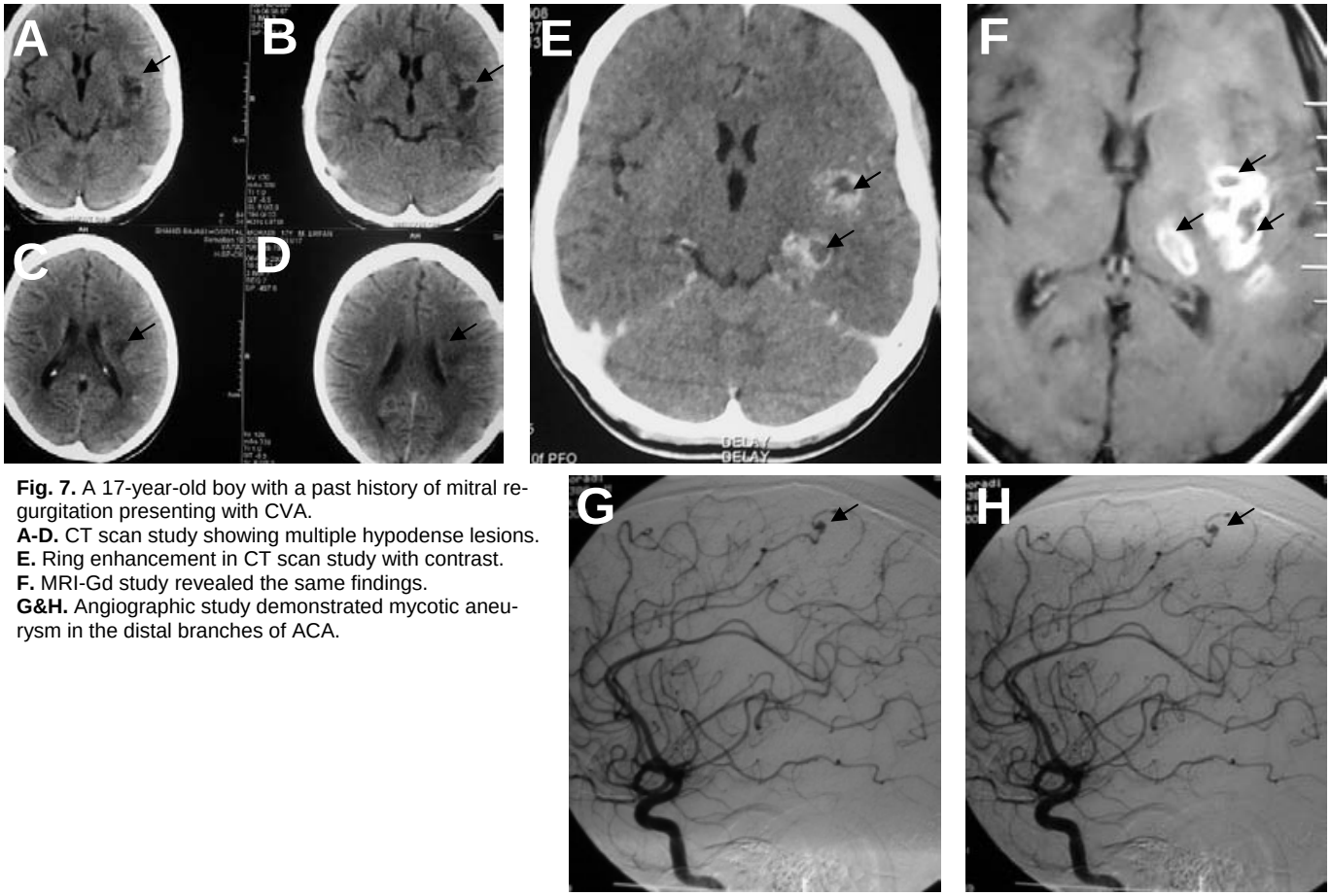


Fig. 7. A 17-year-old boy with a past history of mitral regurgitation presenting with CVA.
A-D. CT scan study showing multiple hypodense lesions.
E. Ring enhancement in CT scan study with contrast.
F. MRI-Gd study revealed the same findings.
G&H. Angiographic study demonstrated mycotic aneurysm in the distal branches of ACA.

& B). In the angiographic study, a giant basilar aneurysm was observed (4C).

Bilobed aneurysm of the internal carotid artery. A 30-year-old man with an acute onset of loss of consciousness and right hemiparesia underwent further evaluation for recognition of the source of the left frontal lobe ICH., MRI and MR angiography revealed nothing in favor of a vascular lesion (Figs. 5A & B), but with gadolinium study in the left para sellar region, an abnormal lesion was detected which was suspicious for a bilobed aneurysm (Fig. 5C); by conventional angiographic study a bilobed aneurysm of the left internal carotid artery's tip was confirmed (Fig. 5D).

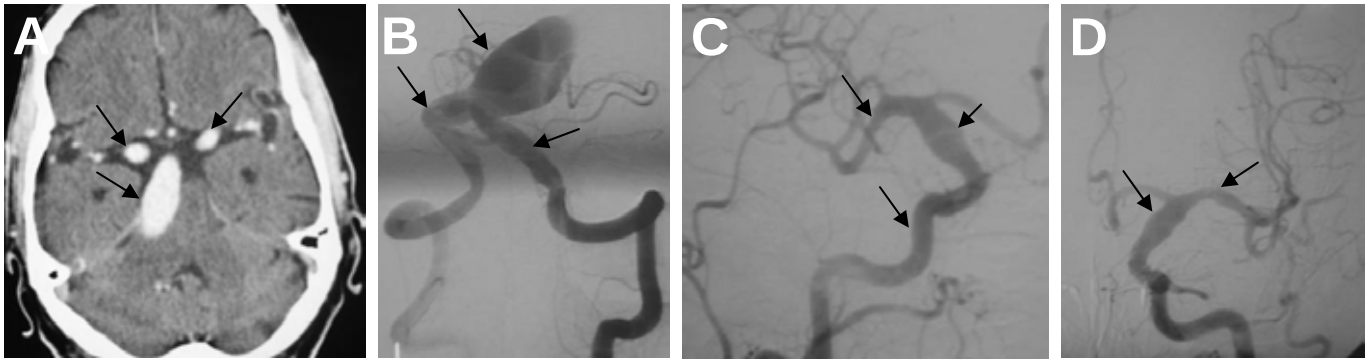


Fig. 8. A 70-year-old man with vertigo, pulsatile tinnitus and headache.
A. CT with contrast showed aneurysmal dilatation of the basilar artery, both internal carotid arteries and both middle cerebral arteries.
B-D. Digital subtraction angiography (DSA) revealed fusiform dilatation of the basilar and both vertebral arteries associated with the same dilatation of both internal carotid arteries and both MCA arteries.

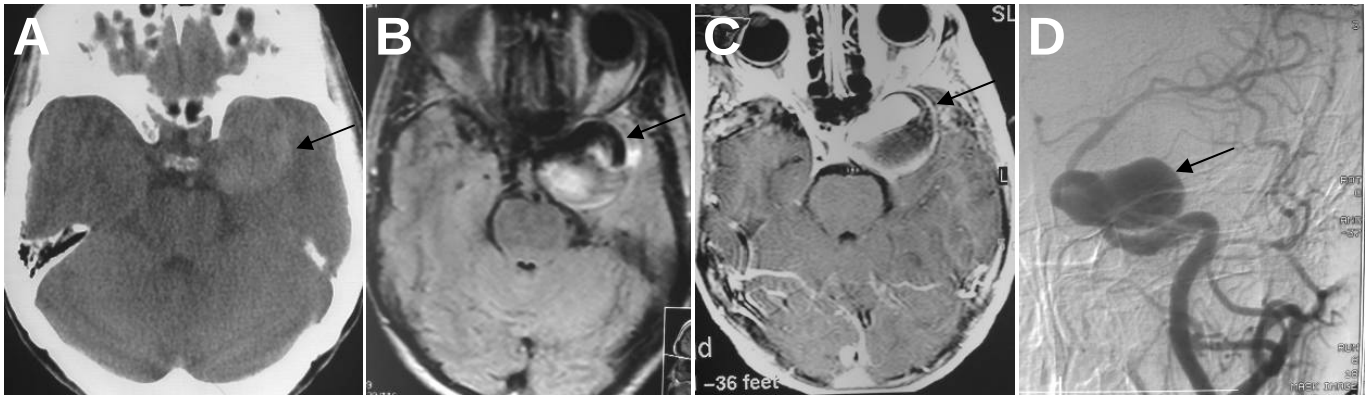


Fig. 9. A 67-year-old woman with the worst headache of her life, nausea, vomiting and loss of consciousness.
A. CT scan without contrast showed an abnormal round hyperdense lesion in the left parasellar region.
B-D. MRI (B&C) and DSA (D) showing giant aneurysm of the C4 segment of the left ICA with thrombosis.

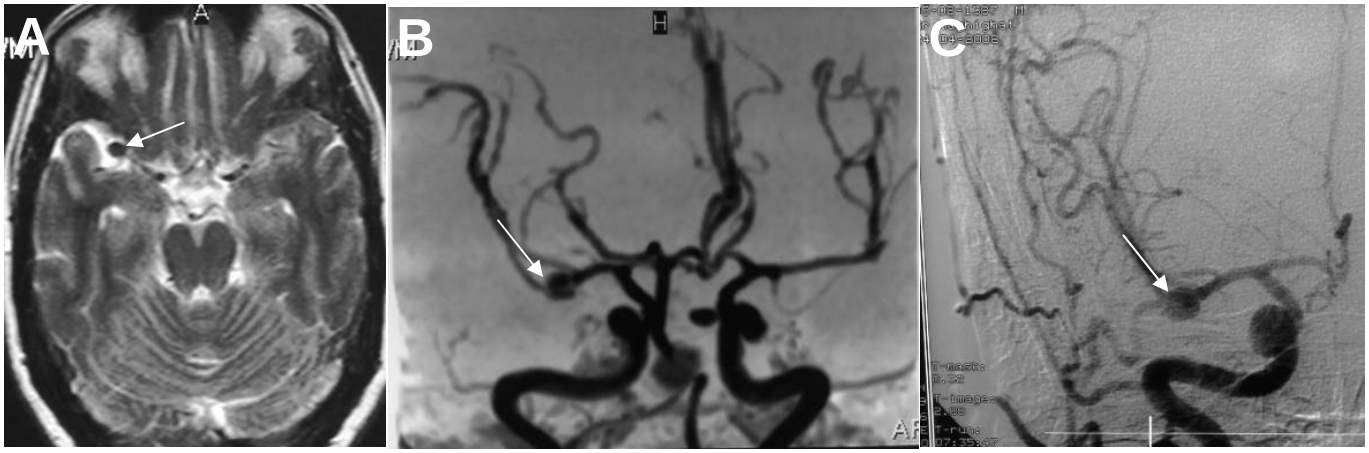


Fig. 10. A 30-year-old man admitted for further evaluation after previous episode of SAH.
A. MRI study revealed an abnormal signal void structure in front of right temporal lobe.
B&C. MRA (B) and angiographic study (C) showing aneurysm of the right MCA.

Aneurysm of the C5 segment of the right ICA. A 17-year-old female presented with massive IVH, SDH, SAH, hydrocephaly and infarction in the right fronto-temporo-parietal lobe (Fig. 6A). CT scan study without contrast showed a hyperdense area in the right parasellar region (Fig. 6B) and DSA confirmed aneurysm of the C5 segment of the right ICA (Fig. 6C) which coiled successfully (Fig. 6D).

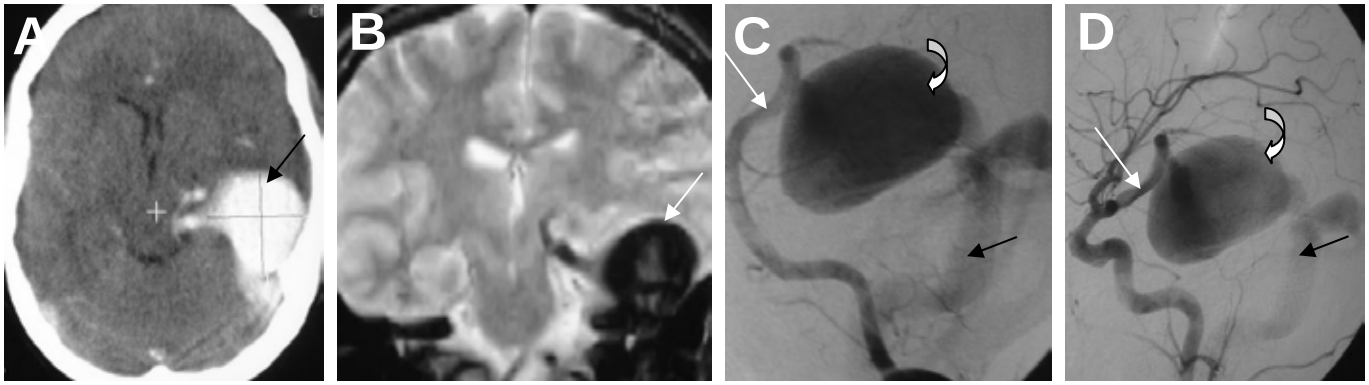


Fig. 11. A middle-aged man admitted for further evaluation for pulsatile headache and tinnitus.
A. CT scan with contrast showing an enhancing void structure in the left posterior fossa.
B. Signal void structure on MRI study.
C&D. DSA revealing an A-V fistula between the left PCA artery (white arrow) draining to the left lateral sinus (black arrow) through a giant aneurysm (curved arrow).

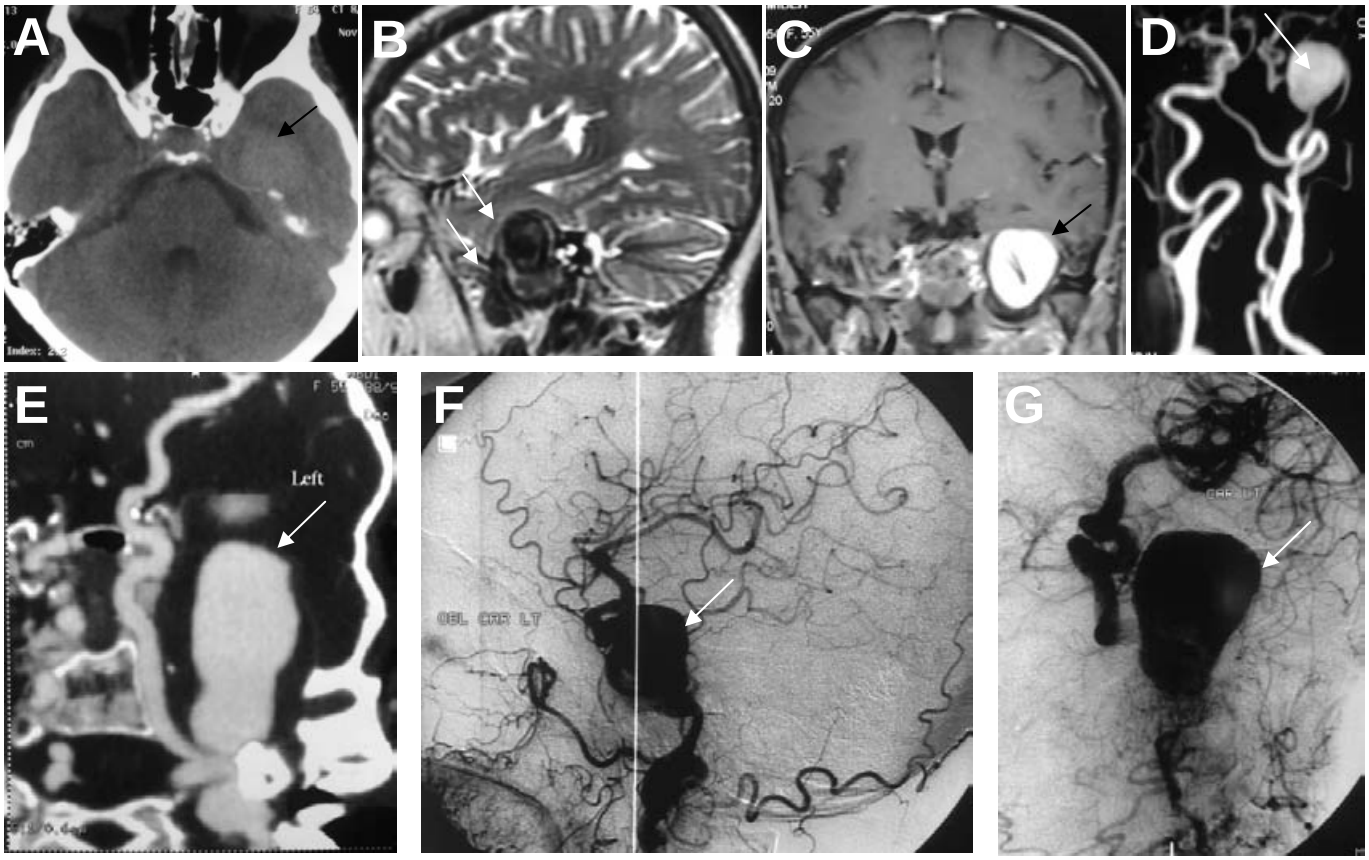


Fig. 12. A middle-aged woman with left hemifacial paresis and numbness.
A. CT scan study showed a round hyperdense mass in the left temporal lobe.
B-D. Sagittal T2W MRI (B), Coronal T1W MRI (C) and MRA (D) revealing giant aneurysm in the petrosal segment of the left internal carotid artery with both intra and extracranial components, containing thrombosis.
E. CTA confirmed the above diagnosis.
F&G. Angiographic study (DSA) confirmed the mentioned aneurysm.

Mycotic aneurysm. A 17-year-old boy with a past history of mitral regurgitation presented with hemiparesia and dysarthria. CT scan study showed multiple hypodense lesions (Figs. 7A-D) with ring enhancement in the contrast study (Fig. 7E). The MRI-Gd study revealed the same findings (Fig. 7F). In the angiographic study, mycotic aneurysm was detected in the distal branches of ACA (Fig. 7G & H). Due to the patient's age, underlying diseases (mitral regurgitation and recent endocarditis), the poor general condition, the high risk of rupture in infectious aneurysms and according to the references mentioning the theoretical hypothesis that implantation of foreign material—platinum coils—into an infectious lesion

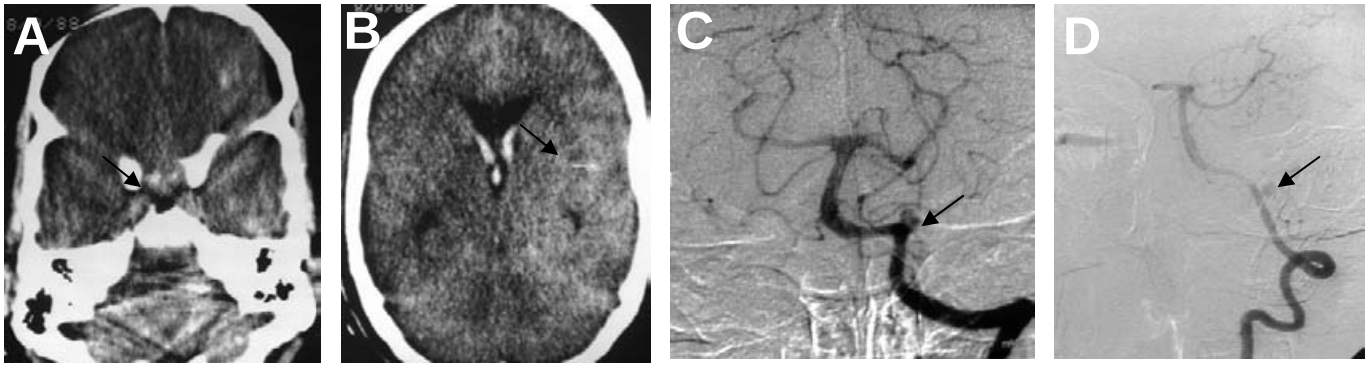


Fig. 13. A 42-year-old woman with SAH.
A&B. Brain CT scan without contrast (A) and with contrast (B) demonstrating SAH in the peri-mesencephal, around the brain stem and the supratentorial region.
C&D. DSA was performed for further evaluation and revealed saccular aneurysm of left vertebral artery in v4 segment (intracranial portion), below the PICA origin.

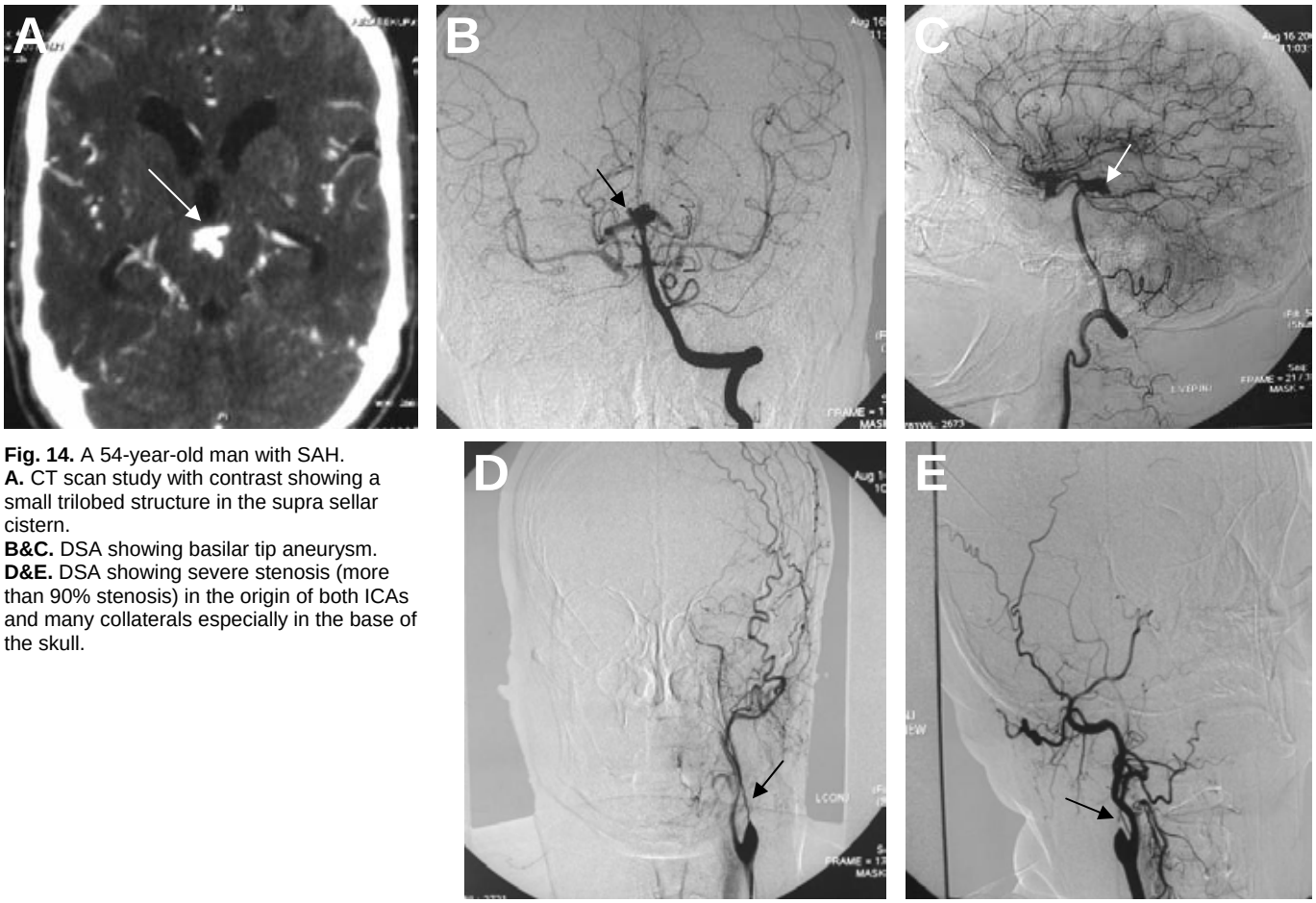


Fig. 14. A 54-year-old man with SAH.
A. CT scan study with contrast showing a small trilobed structure in the supra sellar cistern.
B&C. DSA showing basilar tip aneurysm.
D&E. DSA showing severe stenosis (more than 90% stenosis) in the origin of both ICAs and many collaterals especially in the base of the skull.

may worsen the problem does not apply for infectious intracranial aneurysms,[13] we decided to perform coiling as an easier and safer roption. One year later in the follow-up examination, the patient was well with no sequela.

Multiple fusiform aneurysms. A 70-year-old-man was admitted for further evaluation of vertigo, pulsatile tinnitus and headache. The patient had a history of poor controlled hypertension for several years. CT scan study with contrast surprisingly showed aneurysmal dilatation of the basilar artery, both internal carotid arteries and both middle cerebral arteries (Fig. 8A). Digital subtraction angiography revealed fusiform dilatation of basilar artery, and both vertebral arteries (Fig. 8B) associated with the same dilatation of both internal carotid arteries and both MCA arteries (Figs. 8C & D). Unfortunately, the patient died after two months without any intervention as a result

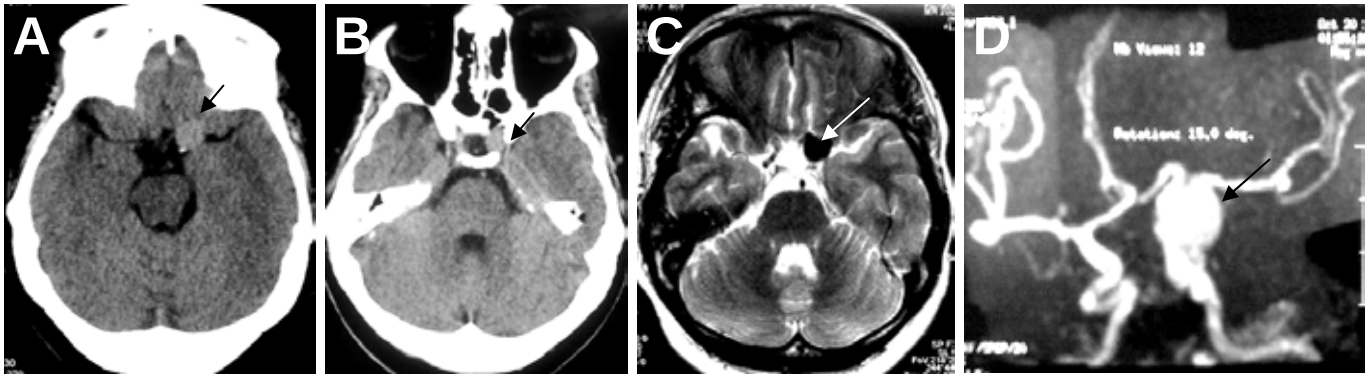


Fig. 15. A 48-year-old female after an episode of SAH.
A&B. Brain CT scan without contrast revealed a round hyperdense structure in the left side of the suprasellar region.
C&D. T2W MRI (C) and MRA (D) confirmed giant aneurysm of the supraclinoid part of ICA.

of massive CVA.

Giant aneurysm of the C4 segment of the left ICA. A 67-year-old female admitted for further evaluation of the worst headache of her life, nausea and vomiting, loss of consciousness from 9 days ago, under the therapy of SAH. In physical examination, left 3rd cranial nerve palsy, ptosis and midriasis were detected. CT scan without contrast showed an abnormal round hyperdense lesion in the left parasellar region (Fig. 9A), MRI study (Figs. 9B & C) and DSA (Fig. 9D) confirmed giant aneurysm of the C4 segment of the left ICA with thrombosis.

Aneurysm of the M2 segment of the right MCA. A 30-year-old man admitted for further evaluation after an episode of SAH. MRI study revealed an abnormal signal void structure in front of the right temporal lobe (Fig. 10A), MRA and angiographic study showed aneurysm of the M2 segment of the right MCA. (Figs. 10B & C). The interesting note in this case was obvious visualization of the aneurysm in simple MRI images.

A-V fistula associated with a giant aneurysm. A middle aged man was admitted for further evaluation of pulsatile headache and tinnitus. After performing CT scan with contrast, an enhancing void structure was presented in the left posterior fossa (Fig. 11A) which was signal void on MRI study (Fig. 11B) without gadolinium. DSA was performed for characterizing the lesion which revealed an A-V fistula between the left PCA artery draining to the left lateral sinus through a giant aneurysm (Figs. 11C & D). The patient underwent interventional treatment in another center.

Giant aneurysm in the petrosal (C2) segment of ICA. A 55-year-old woman who complained of left hemifacial paresis and numbness was admitted for further evaluation. CT scan study showed a round hyperdense mass in the left temporal lobe (Fig. 12A). In the MRI and MRA study, giant aneurysm in the petrosal segment of the left internal carotid artery with both intra and extracranial components, showed thrombosis (Figs. 12B-D). CTA and angiographic study (DSA) confirmed this diagnosis (Figs. 12E-G).

Severe bilateral ICA stenosis associated with basilar tip aneurysm. A 54-year-old man underwent CTA study after an episode of SAH. The study showed a small trilobed structure in the supra sellar cistern (Fig. 14A). Subsequently he underwent DSA which showed basilar tip aneurysm (Figs.14B & C) associated with severe stenosis (more than 90% stenosis) in the origin of both ICAs and many collaterals, especially, in the base of the skull (Figs. 14D & E)

Giant aneurysm of the supraclinoid ICA. CT scan study in a 48-year-old female after an episode of SAH revealed a round hyperdense structure in the left side of the suprasellar region (Figs. 15A&B). Consequent MRI and MRA confirmed giant aneurysm of the supraclinoid ICA (Figs. 15C & D).

Discussion

Intracranial aneurysms are relatively common, occurring in approximately 4% of the population. Most of these aneurysms are asymptomatic, carrying a small but real risk of rupture, resulting in a subarachnoid hemorrhage. The gold-standard for the diagnosis of intracranial aneurysms is DSA, although CTA, MRA, and transcranial Doppler sonography are also effective diagnostic tests. These non-invasive imaging modalities are more appropriate for serial monitoring

of the aneurysms because of the risks associated with invasive angiography. Our current understanding suggests that the annual risk of rupture is 1% or less. Large, irregularly shaped lesions arising from the posterior circulation are at an increased risk for rupture. The management strategies consist of observation, intravascular coiling, and surgical clipping.

Radiologists who work in CT and MRI fields must know about these unusual configurations and presentations of aneurysms, which may mimic neoplastic lesions, therefore may be misdiagnosed. It is necessary, especially for the neuroradiologist, to be expert in cranial angiography and diagnosis of vascular lesions.[12]

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