

INDICATION: 48 years-old Male with left-sided posterior headache.

COMPARISON: None.

TECHNIQUE: Axial CT scan images were performed of the head without administration of intravenous contrast. Multiplanar reconstructions in the sagittal and coronal plane were reviewed. Subsequent, CT angiogram of the intracranial and neck vasculature was performed. 3-D MIPs are provided for interpretation.

FINDINGS:

There is hyperdense appearance of proximal left transverse sinus with linear hyperdensity extending along the left tentorial leaflet. Additionally, there is slight hyperdensity involving the proximal aspect of the right transverse sinus. On postcontrast imaging, no contrast opacification is noted involving the mid and distal left transverse sinus as well as left sigmoid sinus.

There is filling of the right transverse and sigmoid sinus. Superior sagittal sinus and confluence of sinuses demonstrate contrast opacification.

There is no loss of gray-white distinction seen to suggest acute infarct. There is no acute parenchymal hemorrhage. There is no evidence of mass effect, midline shift, or herniation.

Ventricles and sulci are age-appropriate. There is no hydrocephalus. Basal cisterns are patent. No abnormal intra or extra axial fluid collection is present.

Paranasal sinuses and mastoid cells are clear. Orbital contents appear grossly unremarkable. Calvarium and overlying soft tissues appear unremarkable.

Patent anterior and posterior circulation without hemodynamically significant stenosis or aneurysm formation.

Origins of the great vessels appear patent. Bilateral common carotid arteries as well as internal carotid arteries appear patent. Bilateral vertebral arteries appear patent. No hemodynamically significant stenosis or aneurysm formation.

Scattered subcentimeter bilateral cervical lymph nodes are likely reactive in etiology.

IMPRESSION:

Hyperdense appearance of proximal left transverse sinus with linear hyperdensity extending along the left tentorial leaflet. On postcontrast imaging, no contrast opacification is noted involving the mid and distal left transverse sinus as well as left sigmoid sinus. These findings are compatible with left transverse and left sigmoid sinus thrombosis with associated thrombosis of left tentorial vein.

Contrast opacification is noted involving right transverse and sigmoid sinus, reflecting patency.

No intracranial hemorrhage. No evidence of evolving territorial infarction.

Patent intracranial and neck vasculature without hemodynamically significant stenosis or aneurysm formation.

INDICATION: 48 years-old Male with left-sided transverse and sigmoid sinus thrombosis.

TECHNIQUE: Multiplanar multisequence MRI of the brain before and after administration of intravenous contrast. Additionally, 3-D time-of-flight MRA of the intracranial vasculature was performed. MRV of the intracranial vasculature was performed. MRA of the neck vessels was performed. 3-D MIPs are provided for interpretation.

COMPARISON: Head CT/CTA performed on 12/24/2017.

FINDINGS:

There are scattered punctate foci of T2/FLAIR hyperintensity within the periventricular white matter involving bilateral, right greater than left frontal lobes, bilateral parietal lobes, as well as bilateral temporal lobes. A vague 1.2 x 1.2 cm nonenhancing T2/FLAIR hyperintense lesion within the left frontal centrum semiovale/periventricular white matter is also noted.

There is no acute parenchymal hemorrhage, mass effect, midline shift, or herniation. No restricted diffusion is identified to suggest acute territorial infarct.

There is no abnormal postcontrast enhancement.

Ventricles and sulci are normal in size, shape, and position. Basal cisterns are patent. No hydrocephalus is identified. No abnormal intra or extra axial fluid collection is present.

Minimal mucosal opacification involving bilateral ethmoid air cells. Remaining paranasal sinuses are clear. Mastoid cells are clear. Orbital contents appear grossly unremarkable. Calvarium and overlying soft tissues appear unremarkable.

Brain MRA:

Patent anterior and posterior circulation without hemodynamically significant stenosis or aneurysm formation.

Brain MRV:

There is lack of flow related enhancement involving the left transverse sinus with some inherent T1 hyperintensity within the left distal transverse and sigmoid sinus without any flow related enhancement compatible with thrombosis. Right transverse and sigmoid sinuses appear patent. Apparent filling defect within the right jugular vein could be artifact versus a small nonocclusive thrombus. Superior sagittal sinus appears patent. Susceptibly artifact within the left tentorial veins likely represents presence of thrombus.

Neck MRA:

Bilateral common carotid arteries, internal carotid arteries, as well as bilateral vertebral arteries appear patent without hemodynamically significant stenosis or aneurysm formation.

IMPRESSION:

Brain MRI:

1. No evidence of acute territorial infarction, mass effect, midline shift, herniation, hydrocephalus, or intracranial hemorrhage.
2. Scattered punctate foci of T2/FLAIR hyperintensity within the periventricular white matter involving bilateral, right greater than left frontal lobes, bilateral parietal lobes, as well as bilateral temporal lobes could be infectious/inflammatory

etiology or sequelae of small vessel ischemic changes.

3. Vague 1.2 x 1.2 cm nonenhancing T2/FLAIR hyperintense lesion within the left frontal centrum semiovale/periventricular white matter could also be infectious/inflammatory etiology, although a low-grade neoplastic process is not completely excluded.

Brain MRV:

1. Thrombosis of left transverse and sigmoid sinus as well as proximal left jugular vein. Susceptibly artifact within the left tentorial veins likely represents presence of thrombus.
2. Apparent filling defect within the right proximal jugular vein could be artifactual versus a small nonocclusive thrombus.
3. Superior sagittal sinus as well as right transverse and sigmoid sinuses appear patent.

Head and neck MRA:

Patent intracranial and neck vasculature without hemodynamically significant stenosis or aneurysm formation.