

Patient:	Exam Date:	05/09/2007
MRN :	DOB:	02/20/1954
Referring Physician: XMRI, SECOND OPINION	FAX:	888-886-2486

MR OF THE BRAIN WITHOUT CONTRAST

Multiple studies are provided for review. The following studies are reviewed:

Noncontrast head CT dated 4/26/2007
Contrast-enhanced MRI of the brain dated 5/9/2007
CT angiogram of intracranial and cervical circulation dated 5/13/2007
Noncontrast head CT dated 5/28/2007
Noncontrast head CT dated 6/22/2007
Noncontrast head CT dated 8/5/2009

Multiple additional medical records were reviewed, including reports from Dr. , Dr. and Dr. .

Brief summary of findings:

Head CT dated 4/26/2007:

Endotracheal and nasogastric tubes are noted on the scout view. There is a large area of abnormal low attenuation and edema in the bilateral cerebellar hemispheres involving inferior and parasagittal areas of the cerebellar hemispheres, likely ischemic stroke. There is mass effect of the fourth ventricle with resultant ventriculomegaly. There is mild herniation through the foramen magnum and ascending herniation through the tentorium cerebelli. There is no intracranial hemorrhage. There is chronic appearing small stroke involving right anterior lentiform and right caudate nuclei.

MRI of the brain dated 5/9/2007:

The MRI is labeled as contrast enhanced MRI. However, on the several sequences are available for review, including axial noncontrast FLAIR, diffusion sequence, axial T1 fat-sat sequences and several selected images from other sequences.

There is abnormal FLAIR hyperintense signal in the bilateral parasagittal inferior cerebellar hemispheres with mild swelling but without compression of the fourth ventricle and without herniation at this time. There is patchy diffusion restriction within



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inferior cerebellum. There is mild enhancement in the bilateral inferior cerebellum, corresponding to the areas of abnormal signal on the FLAIR sequence. This is consistent with evolution of now subacute ischemic stroke seen on the prior CT. There is evidence of a ventricular shunt with gliotic path to the ventricle from the burr hole in the right posterior parietal lobe. There is no ventriculomegaly at this time. There is chronic small stroke in the right caudate and lentiform nuclei. There is no acute intracranial hemorrhage.

There is abnormal T1 hyperintense signal in the region corresponding to the expected location of the right vertebral artery. This is suspicious for right vertebral artery dissection. However, the study is not conclusive and confirmation is needed with CT angiogram.

CT angiogram of intracranial and cervical circulation dated 5/13/2007:

Tracheostomy and nasogastric tubes are noted.

Patient is status post suboccipital craniectomy. There is small pseudomeningocele at the site of the craniectomy. There is gliosis involving bilateral parasagittal inferior cerebellum. There is mild ventriculomegaly.

The right vertebral artery is congenitally hypoplastic. There is complete occlusion of flow within V2 segment of the right vertebral artery at the level of the C1 arch and C2, corresponding to the abnormal T1 hyperintense signal on the prior MRI, consistent with acute or subacute vertebral artery dissection.

The left vertebral and carotid arteries are normal, without dissection or occlusion. There is no vascular stenosis. The left vertebral artery is dominant and the right vertebral artery is hypoplastic. Intracranial circulation is normal without dissection, stenosis, aneurysm, or arteriovenous malformation. There is a burr hole in the right posterior parietal bone, without shunt catheter.

Head CT dated 5/28/2007:

Patient is status post suboccipital craniectomy. There is small postsurgical fluid collection, possibly pseudomeningocele at the site of the craniectomy. There is gliosis involving bilateral parasagittal inferior cerebellum. In comparison to prior study, there

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is mild new ventriculomegaly. There is no transtentorial or foramen magnum herniation at this time. There is no acute intracranial hemorrhage.

Head CT dated 6/20/2007:

Patient is status post midline suboccipital craniectomy and decompression of the posterior fossa. There is postsurgical fluid collection, possibly pseudomeningocele at the site of the craniectomy. There is no acute intracranial hemorrhage. There is gliosis and encephalomalacia in the bilateral cerebellar hemispheres. The small chronic infarction in the right lentiform nucleus extending to the right caudate nucleus. There is worsening hydrocephalus.

Head CT dated 8/5/2009:

In comparison to prior study, there is a new ventricular shunt entering via right posterior parietal burr hole. The shunt catheter traverses the right lateral ventricle, crosses the midline and terminates in the left frontal horn. There has been interval decompression of the previously seen hydrocephalus. There is no ventriculomegaly at this time. Patient is status post midline suboccipital craniectomy and decompression of the posterior fossa. There is stable appearance of postsurgical fluid collection, possibly pseudomeningocele at the site of the craniectomy. There is no acute intracranial hemorrhage. There is gliosis and encephalomalacia in the bilateral cerebellar hemispheres. The small chronic infarction in the right lentiform nucleus extending to the right caudate nucleus.

Overall conclusion:

Patient suffered large cerebellar stroke in late April of 2007, as a result of vertebral artery dissection. Review of the provided medical record reveals a history of previous trauma approximately 13 months prior to the stroke. However, there is no mention of the cervical spine trauma in the medical records provided for review. Vertebral artery dissection is one of the potential complications of the trauma, which usually happens in the setting of a known cervical trauma. Vertebral artery dissection may remain

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asymptomatic initially, although, it usually manifests clinically within one week of trauma in majority of patients. Furthermore, the stenosis resulting from vertebral artery dissection usually significantly improves or resolves within the 2-3 months after dissection. The vertebral artery dissection on the current study appears to be acute or subacute. The vessel is hyperintense on the T1-weighted sequence, which indicates that the blood is in methemoglobin state, which is usually acute to subacute. In my opinion, it isn't possible to conclude that the vertebral artery dissection is secondary to a trauma from 13 months earlier, especially since there is no documentation of cervical spine trauma at that time. Furthermore, one of the risk factors for spontaneous vertebral artery dissection is prolonged neck hyperflexion. It appears that the patient suffered vertebral artery dissection while painting and possibly has been in the prolonged neck hyperflexion at the time.

References:

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-Electronically Signed by:

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