

Clinical Case Review

Assessment of Left MCA Wake-Up Stroke Using Vitrea® Software

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INTRODUCTION

The patient is a 64-year-old male brought to the West Virginia University (WVU) Hospital Emergency Department (ED) at 9 a.m. The patient's wife reported that the patient was having slow, altered speech since 5:00 a.m. The patient was last seen normal at 10:00 p.m. the previous night. The patient's wife mentioned that the patient was restless during the night and got up twice to pass loose stools, but they did not communicate with each other, so she could not confirm whether he was having speech problems at that time or not.

The patient was seen by the Neurology team at 9:00 a.m. and deemed to be outside the window of stroke treatment options. There was no complaint of headache, no altered mental status, no muscle weakness, no history of seizures, and no urine/bowel incontinence. Exam on admission showed an elderly gentleman in bed with no acute distress, blood pressure of 148/90 with a pulse of 89, respiratory rate of 20 and temperature was 36.8 Celsius. Neurological exam showed poor memory recall (0/3 after 20 minutes). Speech exam showed anomia and expressive aphasia. The patient was unable to name common objects that were being showed to him and could not describe their function and purpose. There was also right and left disorientation and finger agnosia. Orientation, ophthalmoscopy, attention and knowledge were appropriate. Cranial nerves, reflexes, motor and sensory systems were intact too. His National Institute of Health Stroke Scale (NIHSS) rating was estimated to be 7-9. Acute stroke was highly suspected.

METHOD

Labs were drawn and a non-contrast head Computed Tomography (CT) was ordered. Images were processed with a Vitrea® workstation. Blood flow in the brain is visualized to rapidly interrogate the cerebral vasculature and flow abnormalities with vascular defects.

FINDINGS

The non-contrast head CT volumes were loaded into the Vitrea system using the Head CT protocol. A relatively narrow window was used to maximize the difference between gray matter and white matter in order to exclude the presence of hemorrhage (which would preclude further treatment). Signs of early stroke were analyzed including blurring in the gray matter. Images depicted a bright area in the left Sylvian fissure (see *Figure 1*). Although no other changes were appreciated, closer examination of the left Sylvian fissure using CT perfusion and CT angiogram (CTA) was required.

Figure 1: Image Depicting a Bright Area in the Left Sylvian Fissure

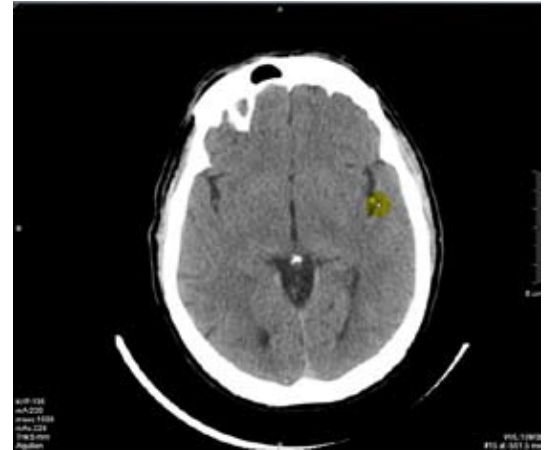
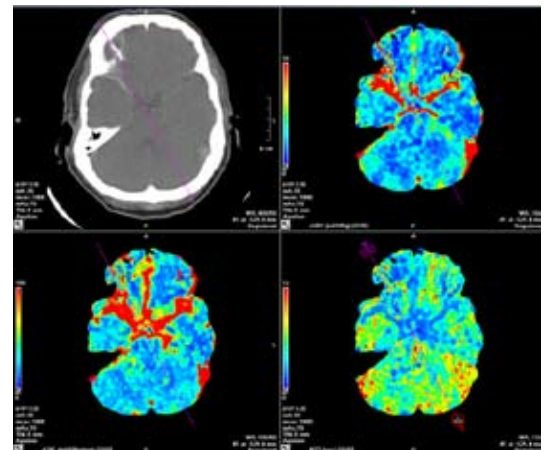


Figure 2: Perfusion Analysis



Clinical Case Review

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The first of two sets of perfusion volumes showed the vessel inputs were appropriate and assessed for motion artifact. After running Motion Correction, the system computed perfusion analysis (see Figure 2) after which the midline was straightened.

Moving up through the images, some asymmetries in the insular region underlying the left Sylvian fissure in the form of increased mean transit time (see Figure 3) were visualized, along with some lateral temporal lobe blood flow involvement. Both evidenced perfusion abnormalities on the left. However, decreased blood volume was not appreciated.

Moving a little higher in the images, evidence of a posterior division left middle cerebral artery (MCA) stroke (see Figure 4) is seen. The blood volume indicated viability of the brain due to good collateral flow despite the presence of a fairly large area of perfusion deficit.

Following this, perfusion trends using the Time-to-Peak map (which indicates how long it takes to get maximum enhancement in a particular area) and the Delay map (which indicates delay in bolus arrival) (see Figure 5) were evaluated. Areas of the brain at most risk tend to have high delay. Using these parameters, a small stroke and good collateral blood flow are identified.

A second set of perfusion volumes were processed using the Brain CT Perfusion protocol in the Vitrea system. After review of the first arterial input, Motion Correction to segment a better vessel was utilized. After segmenting an anterior cerebral artery, the system processed the data (see Figure 6). An abnormality in one of the M2/M3 distal branches of the left MCA along with good collateral supply (which tends to minimize overall stroke volume) are seen.

Figure 3: Visualization of Perfusion Abnormalities in the Left Brain

Figure 4: Identification of Left MCA Stroke

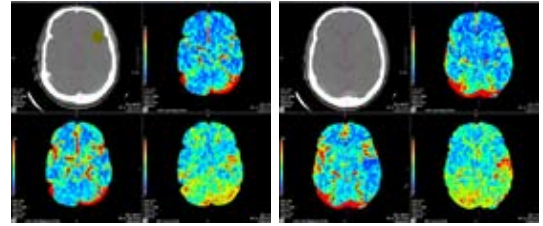


Figure 5: Time-to-Peak and Delay Maps

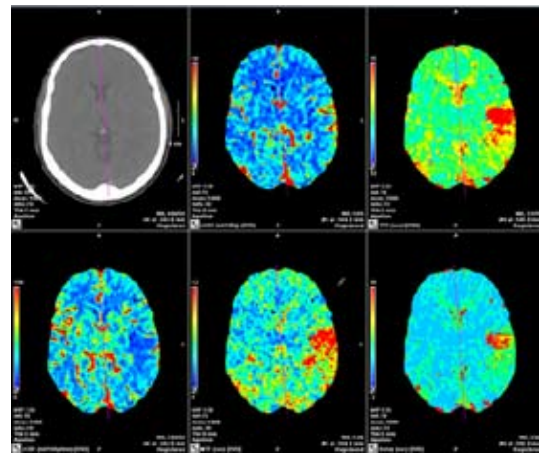
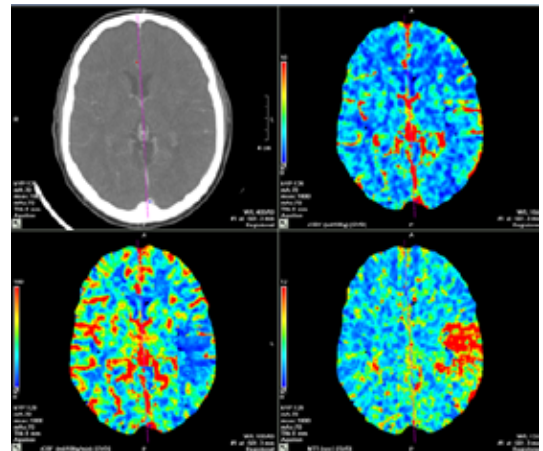


Figure 6: Visualization of Stroke in Distal Branches of Left MCA



Clinical Case Review

Assessment of Left MCA Wake-Up Stroke Using Vitrea Software

Continuing to move up through the images, elevated mean transit time and decreased blood flow (see *Figure 7*) are noted. The infarct was primarily limited to the insula and lateral temporal lobe. Additional areas of decreased blood flow are identified, but they still appeared viable.

Having localized the stroke site, the CTA volumes are evaluated. 2D imaging is utilized to evaluate the vessels all the way up, noting normal branching pattern in the aortic arch (see *Figure 8*).

The left common carotid artery was followed up to the area where the abnormality had been localized. Some atherosclerotic changes near the bifurcation and calcification in the wall (see *Figure 9*), but no stenosis are noted. No donor lesion or any obvious source for the cause in this patient is identified, therefore a cardiac source was suspected.

Following the skull base carotid artery up to the left MCA (see *Figure 10*) was visualized. A left M2 occlusion in a distal Sylvian branch extending about 8 mm was viewed. (Thicker MIPS could be employed if this were a more complex case in which localization was more difficult to achieve, since thicker sections allow better appreciation of asymmetry.)

The impression from images was the stroke likely occurred around 5 a.m. rather than 10 p.m. (when the patient was last observed normal). However, the patient fell outside the traditional stroke treatment window, so no treatment was given. Even so, he was expected to do very well due to his collateral flow. A follow-up MRI enabled comparison of the perfusion information and CTA to the actual MRI diffusion outcome.

Figure 7: Mean Transit Time Elevation and Decreased Blood Flow

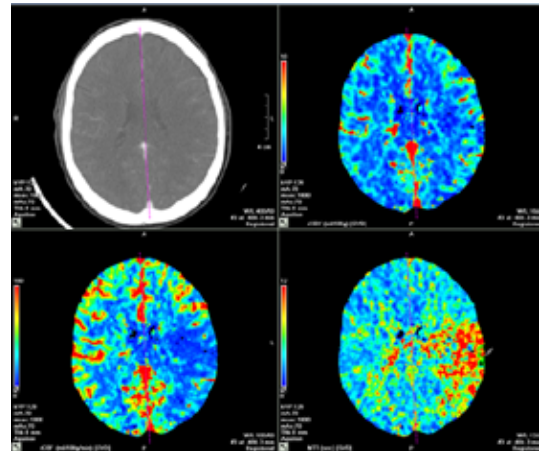


Figure 8: 2D Visualization of Vasculature

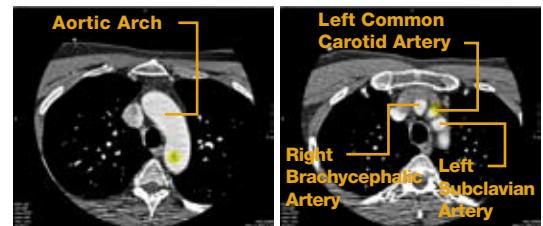
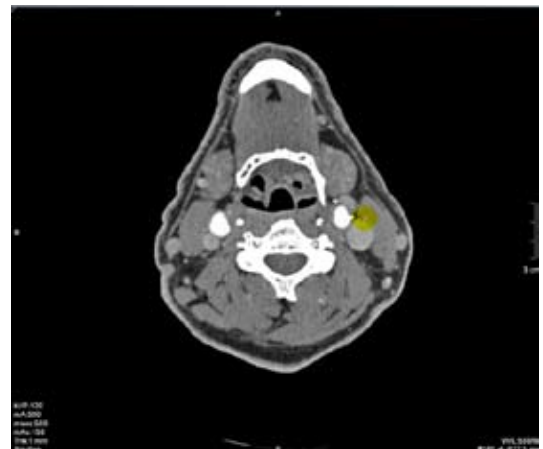


Figure 9: Visualization of Atherosclerotic Changes



Clinical Case Review

Assessment of Left MCA Wake-Up Stroke Using Vitrea Software

The diffusion-weighted sequence using the Head MR protocol was observed, noting that MRI is the gold standard for determining brain cell survival. The brain stem and cerebellum were unremarkable in this view. Some diffusion abnormality isolated in the gray matter (which has higher metabolic needs than the white matter) that extended out laterally into the posterior temporal region (see Figure 11) was appreciated. This relatively small MCA distribution infarct was consistent with the previously viewed acute stroke in the left MCA distribution supplying the Broca area. It was suspected that the patient (who had very good collateral flow) had partially lysed the lesion on his own before much more infarction occurred.

Finally, the follow-up MRI perfusion volumes were viewed in Softread to reveal whether the patient had recanalized. Increased Mean Time to Enhance in just a limited section of the brain (see Figure 12) was observed, indicating that the larger area of perfusion abnormality noted on admission had minimized. The patient did partially lyse the lesion but without complete restoration of normal flow based on MRI perfusion images (mean time to enhance) showing some delay.

CONCLUSION

At initial presentation, the patient had a normal CT with the exception of brightness along one of the Sylvian fissures, indicating a small arterial lesion. CT perfusion demonstrated very little cerebral blood volume abnormality and significant areas of viable brain due to collateral blood flow. Images showed acute left MCA stroke with posterior division distribution leading to expressive aphasia. Because no donor lesion was observed, a complete cardiac work-up to evaluate for a hole in the heart or a patent foramen ovale was recommended.

At discharge, the patient was still unable to complete sentences, but could identify common objects. Neurologic and systemic examinations were unchanged from admission. On follow-up MRI, a small infarct on the left insula and left posterior temporal lobe was still visible. At one-month, the patient had residual numbness and tingling in the right arm and right leg. He also reported some difficulty in reading and with dullness of taste. However, full strength had returned and the patient was able to carry out activities of daily living without any significant deficit.

While this patient fell out of the traditional time window for stroke treatment, he would probably be a candidate in the future as practicing communities move toward perfusion imaging as a guide to treatment therapies.

Figure 10: Localization of Left MCA Occlusion

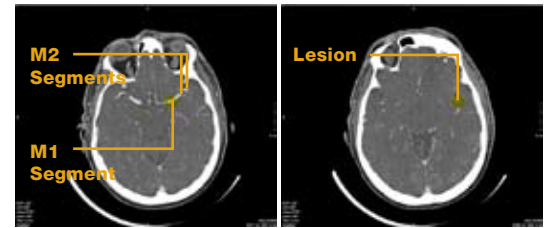


Figure 11: MRI Visualization of MCA Distribution Infarct

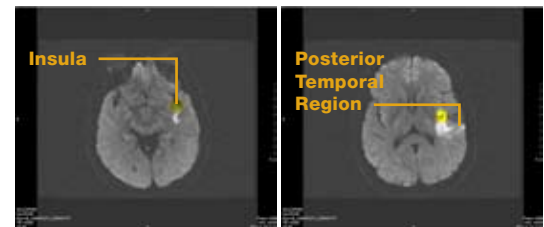


Figure 12: Visualization of Mean Time to Enhance

