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# Computed tomography perfusion stroke mimics on RAPID commercial software: A case-based review

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**Abstract:**

Acute ischemic stroke (AIS) is a leading cause of morbidity worldwide and can present with nonspecific symptoms, making diagnosis difficult. Many neurologic diseases present similarly to stroke; stroke mimics account for up to half of all hospital admissions for stroke. Stroke therapies carry risk, so accurate diagnosis of AIS is crucial for prompt treatment and prevention of adverse outcomes for patients with stroke mimics. Computed tomography (CT) perfusion techniques have been used to distinguish between nonviable tissue and penumbra. RAPID is an operator-independent, automated CT perfusion imaging software that can aid clinicians in diagnosing strokes quickly and accurately. In this case-based review, we demonstrate the applications of RAPID in differentiating between strokes and stroke mimics.

**Keywords:**

Acute ischemic stroke, emergency medicine, neuroimaging, stroke mimics

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## Introduction

Acute ischemic stroke (AIS) is a medical emergency that results from an embolic or thrombotic incident that reduces blood flow to the brain and can result in a multitude of symptoms and long-term complications. Therefore, early diagnosis and intervention leads to improvement in clinical symptoms and prevention of permanent brain damage. Stroke mimics are stroke-like conditions that may present with clinical signs of a real stroke, but treatment differs significantly. Approximately 25%–30% of symptomatic stroke cases are thought to be stroke mimics such as seizures, migraines, tumors, and posterior reversible encephalopathy syndrome.[1,2]

Due to the high cost, limited availability, and time limitations of magnetic resonance imaging (MRI), computed tomography (CT) techniques are typically used for the diagnosis of AIS in most clinical settings.[3,4] Commonly utilized techniques for AIS diagnosis include CT perfusion (CTP), noncontrast CT (NCCT), and CT angiography (CTA).[5-8] Previous studies have shown that the use of multiple CT techniques improves the detection of infarction and overall infarct volume.[8,9] Many landmark stroke trials have validated the use of advanced perfusion techniques, such as CTP, for stroke management in patients eligible for endovascular intervention.[3-6] The results of CRISP, DAWN, DEFUSE 2, and DEFUSE 3 trials have prompted the inclusion of perfusion imaging in the American Heart Association guidelines for the management of patients with AIS.[7] Recent studies have also demonstrated the increased utility of CTP in comprehensive CT evaluation to detect stroke mimics.[10-13] Siegler *et al*.[10] found that



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comprehensive CT with CTP had a negative predictive value of 91% in detecting stroke mimics.

Several CTP artificial intelligence platforms have emerged to aid in the differentiation between strokes and stroke mimics and inform treatment decisions. In many of the aforementioned studies, the utility of platforms such as RAPID (Ischemia View, Menlo Park, CA) has been validated in the differentiation between AIS and stroke mimics.[3-6,11-14] In addition, RAPID has demonstrated success in detecting penumbra in AIS secondary to large vessel occlusion. RAPID outperformed similar commercial platforms in predicting final infarct volume in recanalized patients.[15] Recent studies have also shown that RAPID can recognize perfusion changes in stroke mimics.[10,16]

The aim of this case-based review is to demonstrate imaging characteristics of various stroke mimics that have been detected on CTP using RAPID software.

## Vascular Etiologies

There are several vascular etiologies that can present with ischemic stroke-like symptoms. These include hemorrhagic stroke, cerebral venous thrombosis, arteriovenous pial malformations, arteriovenous dural fistula, aneurysms, and cavernomas.

### Vasospasm

Cerebral vasospasm refers to the narrowing of the arterial vessels and has a high rate of mortality and morbidity along with high rehospitalization rates. Vasospasm usually occurs several days after subarachnoid hemorrhage and is most severe between the 4$^{th}$ and 7$^{th}$ days of hemorrhage.[15] Cerebral vasospasm may or may not be symptomatic but typically presents with focal neurological symptoms or loss of consciousness secondary to ischemia. More than 50% of patients with vasospasm develop permanent neurological deficits.[15] Vasospasm may also result in death.[16] Imaging characteristically shows diffuse narrowing of the vessels without intervening regions of normal caliber.

This is demonstrated in the case of a 39-year-old female patient who presented to the emergency department (ED) with right hemiparesis after developing a left posterior communicating artery aneurysm. Examination findings were suspicious for vasospasm, and the patient underwent angiography that showed severe stenosis of the left supraclinoid internal carotid artery (ICA) and A1 segment as well as mild vasospasm of the left M1 segment [Figure 1a].

CTA studies showed diffuse vasospasm [Figure 2], while CTP demonstrated impaired perfusion and ischemia in the right cerebral hemisphere [Figure 3]. After intra-arterial administration of Verapamil, there was a significant improvement in the degree of vasospasm [Figure 1b].

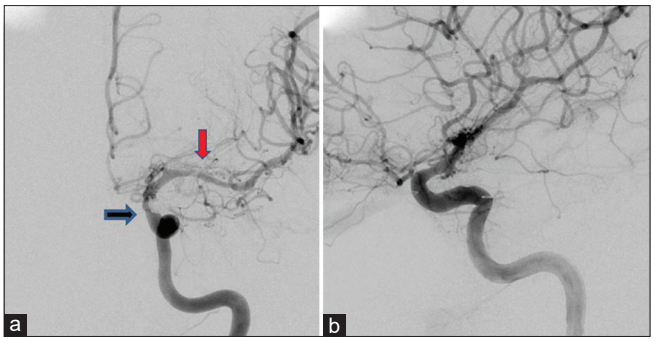


**Figure 1:** (a) Severe stenosis of left supraclinoid ICA and A1 segment (black arrow) and mild vasospasm of left M1 segment (red arrow). (b) After intra-arterial administration of verapamil, there is a significant improvement in vasospasm. ICA: Internal carotid artery

### Capillary telangiectasia

Capillary telangiectasias are dilated capillaries found in normal tissues of the brain. Radiological investigations have reported distribution in multiple regions of the brain, but they are most commonly seen in the pons, temporal lobe, medulla, or caudate and have a benign clinical course. Capillary telangiectasias are typically incidental findings associated with minor hemorrhage and gliosis, unlike cavernous angiomas.[17,18]

Capillary telangiectasia was discovered in a 44-year-old female patient who was admitted to the hospital [Figure 3]. She had a previous history of migraines that presented as progressively worsening headaches with photophobia and phonophobia. She subsequently developed right-sided facial droop, which prompted her to visit the ED.

The radiological markers of capillary telangiectasias are difficult to diagnose on CT and angiographic imaging.In this case, RAPID enhanced the appearance of capillary telangiectasias.[19] CTP findings included increased cerebral blood volume (CBV) and cerebral blood flow (CBF) and mildly increased Time-to-Maximum (TMax) and mean transit time (MTT), which can be attributed to slow flow within the lesion.

## Venous Thrombosis

### Superior sagittal sinus thrombosis

Superior sagittal sinus (SSS) thrombosis is a rare cerebrovascular condition that is frequently associated with diseases that cause hypercoagulability, local bloodstream stasis, and vessel wall abnormalities, which can contribute to the development of thrombosis.[20,21] Symptoms of high intracranial pressure

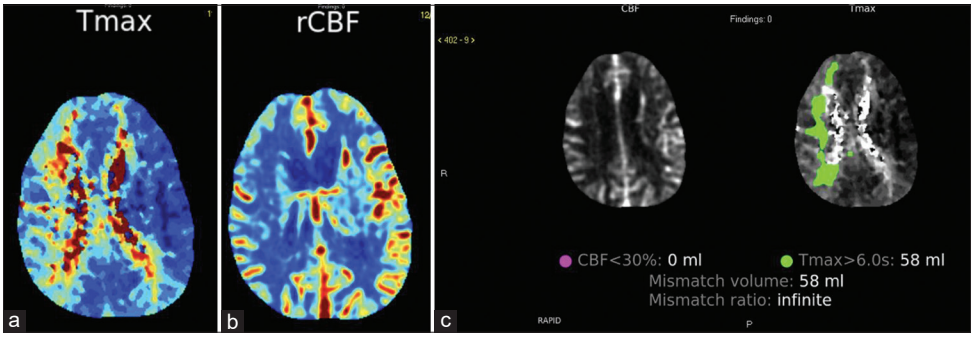


**Figure 2:** Perfusion imaging during vasospasm shows increased TMax in right cerebral hemisphere (a) with subtle decreased CBF. Although there are no areas of CBF <30%, small areas of slightly decreased CBF are present (b and c). TMax: Time-to-maximum, CBF: Cerebral blood flow

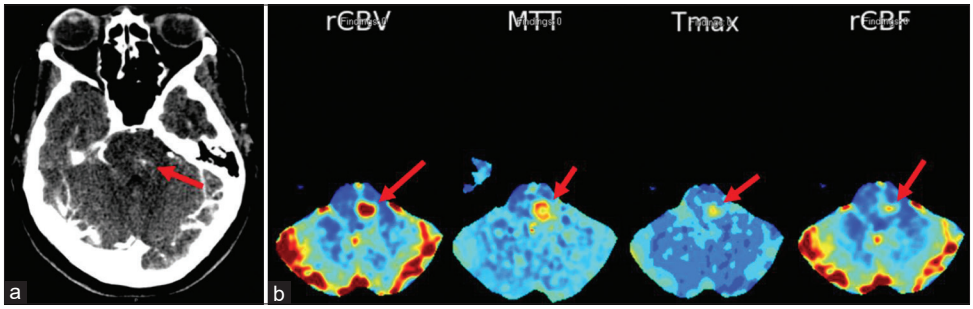


**Figure 3:** CT angiogram shows faint enhancement in left posterior aspect of pons (red arrow) with capillary telangiectasia (a). CTP shows associated increased CBV, CBF and mildly increased TMax and MTT (red arrows) reflective of slow flow within lesion (b). CT: Computed tomography, CTP: computed tomography perfusion, CBV: Cerebral blood volume, CBF: Cerebral blood flow, TMax: Time-to-maximum, MTT: Mean transit time

may be accompanied by focal neurological deficits, including headache, focal epilepsy, and hemiparesis in younger patients.[22]

This is demonstrated in the case of a 45-year-old female patient who presented with left-sided weakness. CT studies revealed the presence of SSS thrombosis and right cortical vein extension with nonhemorrhagic right parasagittal venous parenchymal abnormality [Figures 4 and 5].

Follow-up CT venography studies revealed recanalization of SSS with resolved thrombosis and encephalomalacia in the preceding signal abnormality location [Figure 6]. The thrombus may have resulted from a hypercoagulable state secondary to oral contraceptive use. Perfusion imaging shows increased TMax and MTT in the high right front parietal region, with symmetric CBV and CBF. These findings are compatible with delayed perfusion, which is a chronic phenomenon likely due to collateralization following the recanalization of long-standing thrombosis.

## Seizure Etiologies

### Postictal

The postictal state refers to the time period between seizure cessation and the return to the baseline condition. It may last for 30 min and is marked by disorienting symptoms such as headache, confusion, nausea, drowsiness, and hypertension.[13,23]

The postictal state may show temporary signal differences in brain imaging. CT studies have shown the presence of swelling in a small area of the cortex, which may have been involved in the seizure.[24]

We present a case of a 34-year-old female patient who was found by her partner in an unresponsive condition with pooling of oral secretions. When she presented to the hospital, NCCT was performed but showed no acute abnormality. However, perfusion imaging demonstrated subtle decreases in CBV and CBF and increased TMax in the region of the left temporal lobe with no abnormality in the region of basal ganglia [Figure 7].

This finding of subtle perfusion asymmetry with decreased CBV and CBF and qualitative slight increase of TMax in the left temporal lobe without the involvement of basal ganglia and or specific vascular territory is compatible with the postictal state of the patient at the time of examination (approximately 3 h after the seizure activity).

### Todd paralysis, postictal state

Todd's paralysis is a transient focal weakness of the hand, arm, or leg that can occur after partial seizure activity within that limb.[25] The incidence of Todd's paralysis has

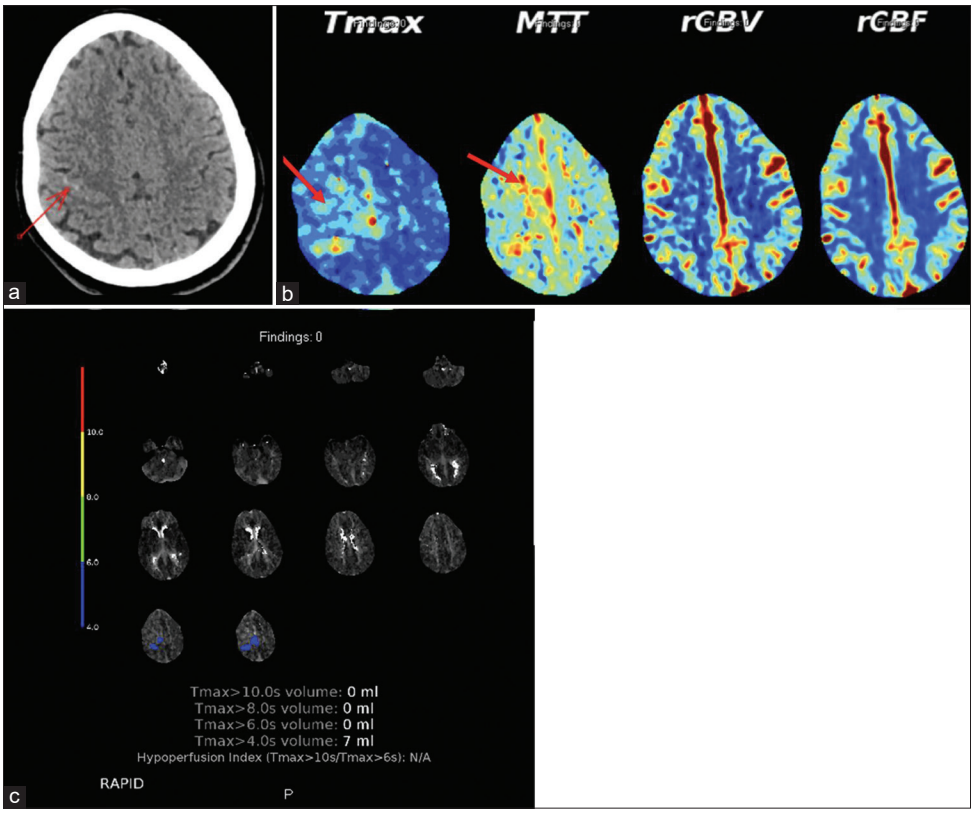


**Figure 4:** NCCT Head demonstrated increased attenuation of a right cortical vein in the region of precentral gyrus (a) suggestive of cortical vein thrombosis. Hypoattenuating area in the right parasagittal frontal lobe (red arrow) with loss of gray white matter differentiation is suspicious for cytotoxic edema. (b and c) Perfusion imaging shows increased TMax and MTT in the high right frontoparietal region (red arrows), with symmetric CBV and CBF, compatible with delayed perfusion. TMax: Time-to-maximum, MTT: Mean transit time, CBV: Cerebral blood volume, CBF: Cerebral blood flow, NCCT: Noncontrast computed tomography

been found to be around 13% in patients with seizures confirmed on video electroencephalography (EEG).[25] In patients with suspected Todd's paralysis, the NCCT is generally unremarkable unless there is the presence of an underlying condition.

We present a case of a 20-year-old male with Kearns-Sayre syndrome who presented to the ED with left arm weakness and acute confusion. Neurological examination revealed left-sided neglect along with the motor symptoms. CT study of the head revealed the absence of acute bleed [Figure 8].

CTP studies showed signs of decreased perfusion in the distribution of the right middle cerebral artery (MCA). CTA showed patent intracranial vessels, with a slightly decreased caliber of the right ICA. Continuous EEG (cEEG) preliminarily showed slowing without epileptiform activity on the right. EEG features improved with lorazepam, and he was started on levetiracetam 500 mg twice/day. Repeat EEG revealed right frontal epileptiform activity in the form of occasional runs and diffuse background slowing. On follow-up MRI, there was no residual abnormality in the right MCA territory. His symptoms appear to be linked to focal seizures; however, the cause of the seizures has not been identified.

## Brain Tumors

### Glioblastoma multiforme

Glioblastoma multiforme (GBM) is a malignant tumor that affects individuals of all ages, especially patients over the age of 50 years. GBM is difficult to treat and associated with poor prognosis.[26] On imaging, GBM presents with multiple discrete areas of contrast-enhancing tumor embedded with T2/fluid-attenuated inversion recovery signal abnormality.

GBM is illustrated in the case of a 75-year-old male patient who presented to the ED with dysphagia that started 1 h before arrival as well as a history of intermittent posterior headaches for the past few months. There is a direct ED-to-MRI pipeline in for patients who present with stroke-like patients in this institution, so the patient underwent an MRI to rule out acute infarct. MRI demonstrated a large left temporal lobe mass with heterogeneous enhancement, central necrosis, and peripherally increased CBF and volume that most likely represents features of a glioblastoma. The TMax was found to be marginally enhanced (>4 s) along the medial portion of the mass, which shows a shift in hemodynamics and delayed perfusion due to vasogenic edema around the tumor [Figure 9].

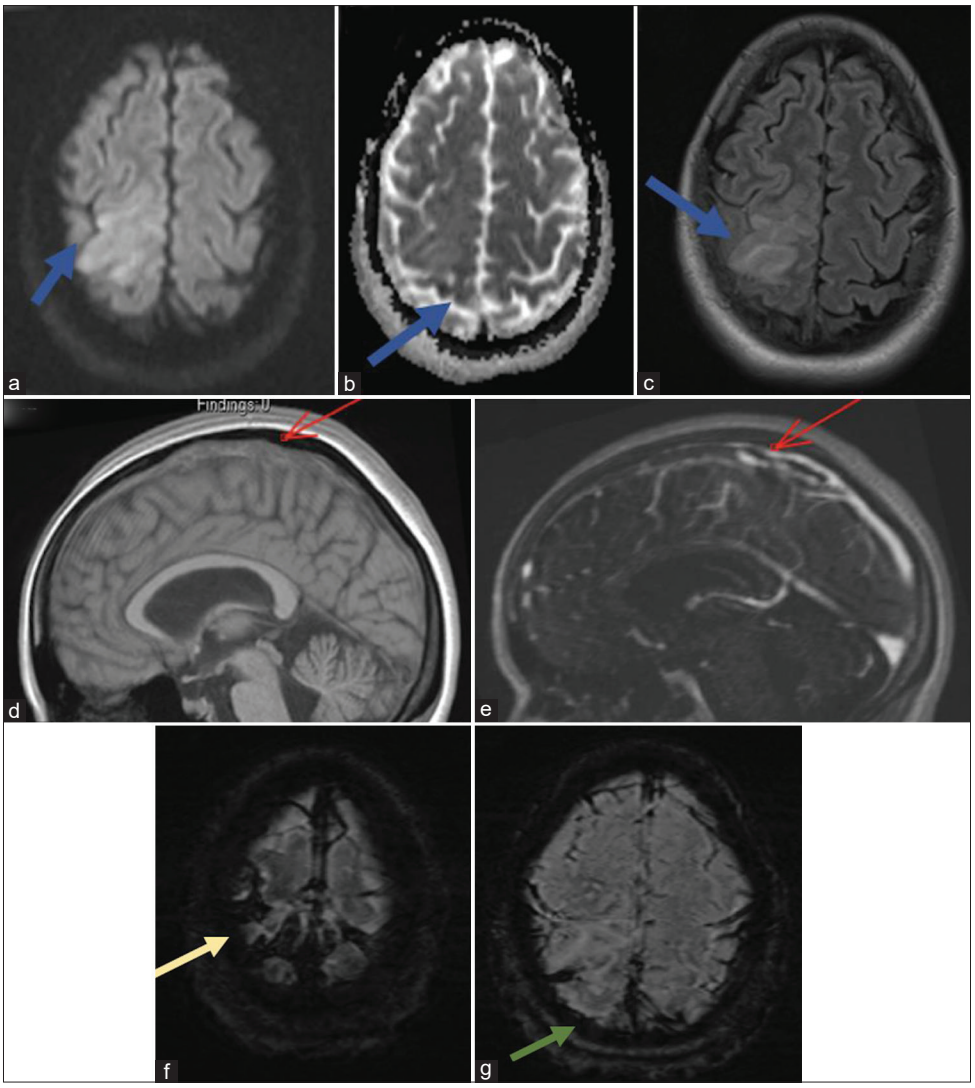


**Figure 5:** Follow-up MRI confirms SSS thrombosis and cortical vein extension (d and e) with nonhemorrhagic right parasagittal venous parenchymal abnormality (red arrows). DWI hyperintensity (a) has T2 shine-through on ADC (b) showing vasogenic and not cytotoxic edema (blue arrows) confirmed on FLAIR (c). SWI shows susceptibility corresponding to the SSS and cortical veins (yellow arrow) compatible with intraluminal thrombosis (f). Additional SWI image shows SAH (g, green arrow). SSS: Superior sagittal sinus, FLAIR: Fluid attenuated inversion recovery, DWI: diffusion-weighted imaging, ADC: Apparent diffusion coefficient, SWI: Susceptibility weighted imaging, SAH: sub-arachnoid hemorrhage

### Brain metastatic lesion

Brain metastases represent approximately 25% of all brain tumors in hospitalized patients. Lung cancer, breast cancer, colon cancer, kidney cancer, and melanoma have been shown to most commonly lead to metastases to the brain.[26-28] Tumor growth causes compression of nearby structures, resulting in headache, personality changes, memory loss, and seizures.[27]

We present the case of an 81-year-old male with metastatic rectal cancer who presented to the hospital with stroke-like symptoms, including acute confusion and dysarthria. The results of NCCT showed multiple hyper-dense round lesions involving the left frontal and left mesial temporal lobes of the brain without any signs of acute infarction [Figure 10]. Perfusion studies showed an increase in CBV and CBF. Slightly increased (TMax >6 s) was found along the medial margin of the left frontal lobe mass extending to adjacent left frontal white matter, likely related to underlying change in hemodynamics and vasogenic edema surrounding the tumor [Figure 10].

## Discussion

Understanding the prevalence and pattern of perfusion abnormalities in stroke mimics can help distinguish them from stroke [Table 1]. In this review, we have demonstrated the utility of RAPID software in identifying these patterns to improve diagnostic speed and accuracy. It is important to note the official specific Food and Drug Administration (FDA) approvals for the clinical use of RAPID. Per FDA approval, RAPID CTP can be "used by physicians to aid in the selection of AIS patients with

known occlusion of the intracranial ICA or proximal MCA for endovascular thrombectomy" if contraindications are not present.[29] These contraindications include issues

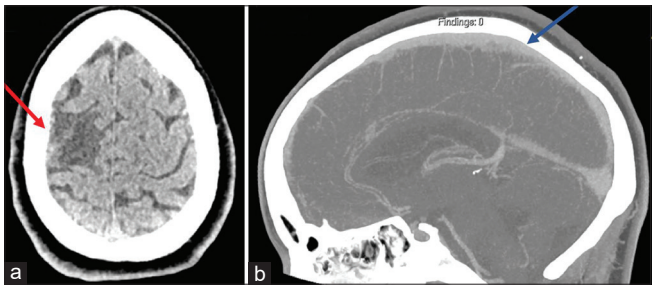


**Figure 6:** Follow up CTV demonstrated encephalomalacia (red arrow) in the area of prior signal abnormality (a) and recanalization of SSS (blue arrow) with resolved thrombosis (b). CTV: Computed tomography venography, SSS: Superior sagittal sinus

**Table 1: CTP findings in non-AIS etiologies**

| Non-AIS etiologies | CTP findings |
|---|---|
| Cerebral venous thrombosis | Decreased CBF and variable CBV with increased Tmax |
| Vasospasm | Increased Tmax in corresponding cerebral hemisphere with subtle decreased CBF and variable CBV dependent on extent of ischemia |
| Capillary telangiectasia | Increased CBV and CBF and mildly increased Tmax and MTT |
| Seizure, postictal state | Subtle decreases in CBV and CBF and increased Tmax |
| GBM | Peripherally increased CBF and CBV, variable Tmax marginally enhanced Tmax (>4 s) along the medial portion of the mass |
| Brain metastases | Increase in CBV and CBF, slightly increased (Tmax>6 s) variable Tmax |

CBF: Cerebral blood flow, CBV: Cerebral blood volume, MTT: Mean transit time. Tmax: Time to maximum, AIS: Acute ischemic stroke, CTP: CT perfusion, CT: Computed tomography, GBM: Glioblastoma multiforme

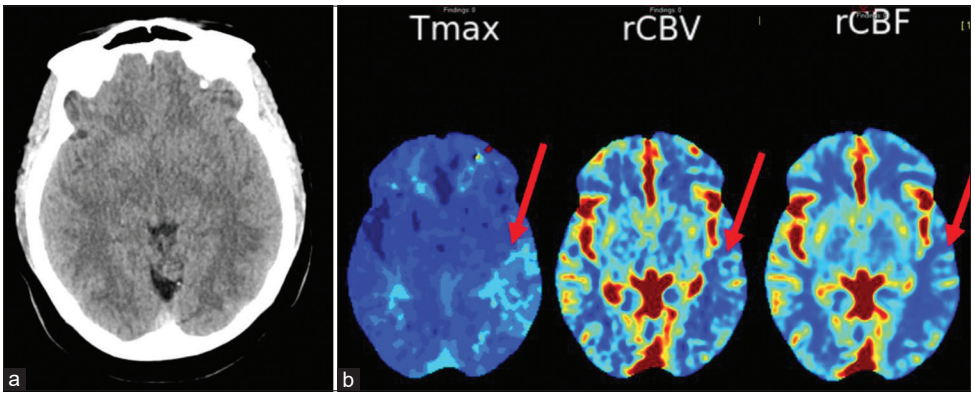


**Figure 7:** NCCT shows no acute abnormality (a). Subtle perfusion asymmetry with decreased CBV and CBF and qualitative slight increased TMax in the left temporal lobe (b, red arrows) in non-territorial distribution, compatible with postictal state. NCCT: Noncontrast computed tomography, CBV: Cerebral blood volume, CBF: Cerebral blood flow, TMax: Time-to-maximum

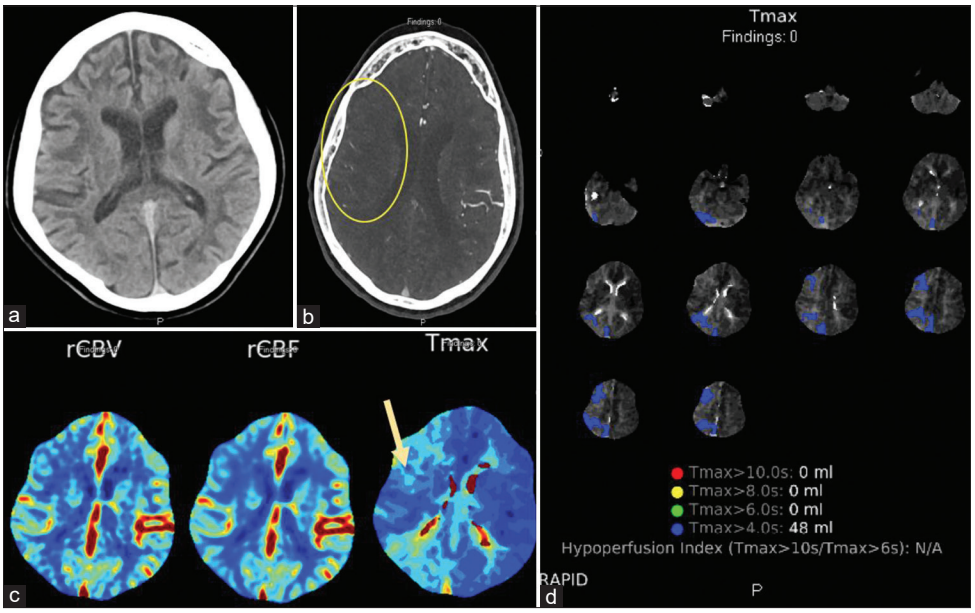


**Figure 8:** Todd paralysis. (a) No abnormality on NCCT. (b and c) Perfusion imaging shows increased TMax in the right MCA territory (yellow arrow) with preserved CBV and CBF. d) Mild transit delay in the right cerebral hemisphere of Tmax > 4 seconds. NCCT: Noncontrast computed tomography, TMax: Time-to-maximum, MCA: Middle cerebral artery, CBV: Cerebral blood volume, CBF: Cerebral blood flow

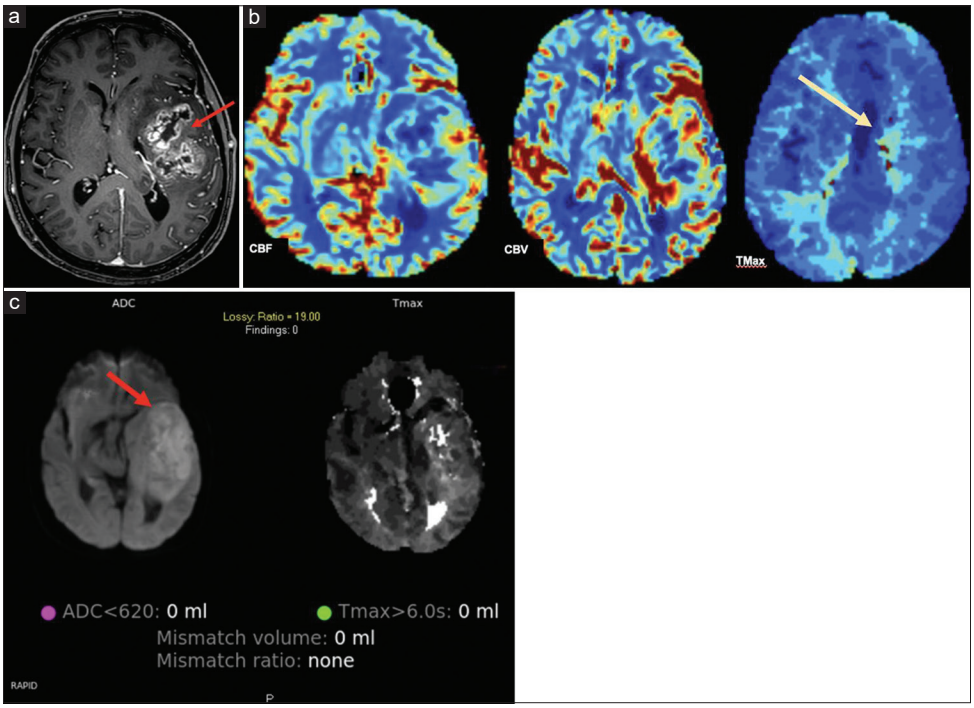


**Figure 9:** Stroke protocol MRI showed a large left temporal lobe mass (red arrow) with heterogeneous enhancement, central necrosis, and peripheral increased CBF (yellow arrow) and volume that most likely represents features of a high-grade glioma/glioblastoma (a, c). TMax is minimally increased (>4 sec) along the medial aspect of the mass (b, yellow arrow)

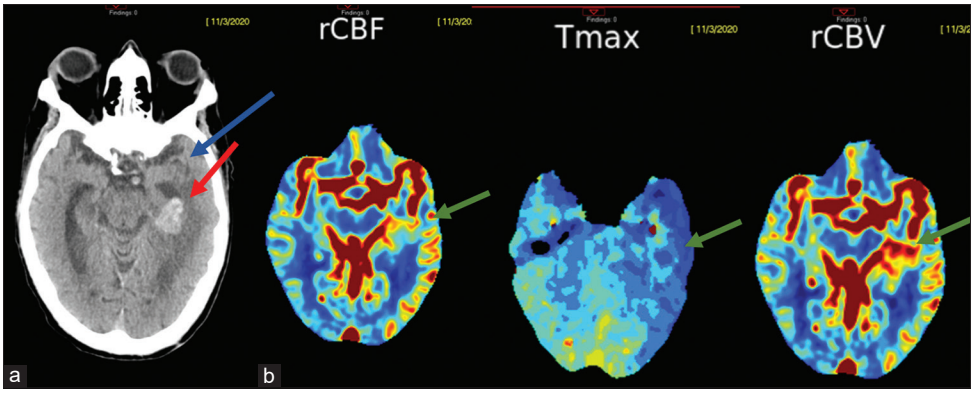


**Figure 10:** NCCT shows hyperdense mass in mesial temporal lobe (a, red arrow) and left frontal lobe (b, blue arrow). Elevated CBV and CBF in peripheral rim. Slightly increased TMax surrounding the lesions due to vasogenic edema (green arrow). NCCT: Noncontrast computed tomography, CBV: Cerebral blood volume, CBF: Cerebral blood flow, TMax: Time-to-maximum

with bolus quality, the presence of motion artifact, and hemorrhage.[29]

There are several limitations to the interpretation of this technology. Previous studies have shown perfusion abnormalities in various stroke mimics.[10,16,30-33] Seizures and interictal states have been linked with cerebral hyper/hypoperfusion, depending on the timing of image acquisition.[10] Similarly, in our seizure case, perfusion imaging showed increased TMax in the left temporal lobe with no abnormality in the basal ganglia. The correlation of perfusion imaging findings with clinical symptoms and arterial location is vital to ensure the accurate diagnosis of stroke versus stroke mimics. This principle is demonstrated again in our patient with GBM and our patient with brain metastasis secondary to rectal cancer. Brain tumors may show ischemic patterns despite their vascularity.[10] In addition, rCBF may be greater due to angiogenesis, but the MTT and TMax may be delayed, as seen in our cases where the TMax was marginally increased.[10] Other factors, such as changes in hemodynamics and the presence of vasogenic edema, may also contribute to the observed delay in TMax.

Another potential limitation of RAPID implementation is artifact. Siegler *et al*. noted that the presence of artifact, likely due to patient motion, can be corrected by the RAPID motion estimates function.[10] However, there

are no confirmed thresholds for which the onset or peak of excess motion causes significant perfusion artifact.[10] Further studies are needed to identify this threshold.

Siegler *et al*. found that the incidence of TMax abnormalities was significantly greater in patients with stroke than those with stroke mimics.[10] That is, patients with stroke mimics were more likely to have normal TMax. In cases of abnormal TMax patterns, more stroke mimic patients had TMax patterns that did not correspond with clinical symptoms compared to patients with stroke. Fully discordant TMax abnormalities were associated with stroke mimics with high negative predictive values.[10] In this study, many of the penumbral patterns did not match the expected arterial distribution given the clinical symptoms and CTA findings. CTP results should not be used alone. Clinicians must also ensure that the penumbral patterns visually correlate to established arterial territories and clinical findings prior to tPA or thrombectomy. In all cases, expert review of imaging is still required as RAPID is used in the context of all other clinical information.

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Nil.

## Conflicts of interest
Dr. V. Yedavalli is a consultant for RAPID (IschemaView, Menlo Park, CA) and MRIOnline (Cincinnati, OH, USA) but has no financial disclosures on the subject matter or materials discussed in the article or with any company making a competing product. All other authors have no relevant financial or nonfinancial interests to disclose.

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