Complex Stroke Cases

Large vessel occlusion with poor anterior versus good posterior collateral flow

Clinical case

89 year old female brought to hospital because of a wake-up onset of left side weakness. Last time seen well 10 hours prior to arrival. The neurological assessment indicated a right total anterior circulation syndrome (National Institutes of Health Stroke Scale - NIHSS 18).

Imaging

Brain NCCT: Right frontal hypodensity consistent with recent middle cerebral artery (MCA) ischemic stroke (Figure 1.2, J). CTA: Right MCA occlusion (M1 segment)

CTP (Figure 1.1): Decreased CBV (A and E) and CBF (B and F) with prolonged MTT (C and G) and DT (D and H) in the anterior territory of the right MCA. In comparison, increased CBV with normal CBF and MTT and DT in the posterior territory of the right MCA. Figure 1.2: Automated core-penumbra maps show anterior right MCA core (total volume 70.4mL), with a posterior MCA penumbra (total volume 98.6mL). Dynamic CTA (Figure 1.2), derived from CTP acquisition, shows early and late filling of the MCA territory at the circle of Willis level (K) showing rapid and near complete collateral filling of the posterior/inferior MCA territory beyond the right M1 occlusion. L: Early and late filling of the MCA territory at the corona radiata level showing delayed and incomplete collateral filling of the right anterior/superior MCA territory. Figure 1.3: same case processed with RAPID (A and G = CBV; B and H = CBF; C and I = MTT; D and J = Tmax, E and K = core map defined by rCBF <30%; F and L = penumbra map defined by Tmax >6 seconds). Note there is good concordance between the two software. RAPID automated core-penumbra maps measured the core at 76 mL and the penumbra at 103mL.

Comments

The patient has recent, but established, right frontal infarction on NCCT suggesting time of onset >6 hours. Note the difference between the much larger region of reduced CBF compared to the smaller region of reduced CBV (Fig 1.1 and Fig1.3). This reflects the good posterior MCA collateral flow seen on dynamic CTA, preserving CBV posteriorly. Note also the differing severity of delay time within the perfusion lesion. The most severe delay is in the core in the anterior and deep territory of the MCA, with less severe delay in in the posterior territory of the MCA (again reflecting better collateral flow). Importantly, note the estimated CBF core on both RAPID and MIStar underestimate the extent of hypodensity seen on NCCT. This suggests that there was more severe ischaemia prior to imaging, leading to infarction, but that improved collateral flow has led to increased CBF above the 30% core threshold. The improved collateral flow is also represented here by regions of elevated CBV and shorter DT/Tmax within regions that are hypodense on NCCT (note this is easier to appreciate on the less 'smoothed' MIStar maps - Fig 1.1).

In this case, no acute thrombolytic/endovascular treatment was offered. The presence of extensive, established infarction was a contraindication for thrombolysis. Moreover, this patient was a wake-up stroke. New evidence for treating wake up strokes with endovascular clot retrieval based on perfusion parameters is limited to cases with moderate-high NIHSS and relatively small core in the maps (smaller than 21mL in patients >80 years old), and without major ischaemic change on NCCT (1).

Practice Tip: Always look at all components of the multimodal CT exam (including NCCT, CTA, raw perfusion maps, and the core/penumbra maps). In this case there is 'mismatch' between the hypodensity core on NCCT (larger) and the CTP-calculated core. There is a

good pathophysiologic reason for this in this case. As a rule of thumb, such 'mismatch' can occur in 3 circumstances:

- Artefact (see later in review). This is not the case here.
- Reperfusion prior to CTP. This is not the case here – the patient had a persistent proximal M1 occlusion.
- Persistent occlusion with infarction leading to hypodensity on NCCT, but improvement in collateral flow at time of CTP leading to CBF being above the threshold for CTP estimated core.

Proximal occlusion with excellent collaterals

Clinical case

77 year old male brought by ambulance to the Emergency Department (ED) with an 80 minute evolution of right sided weakness and speech disturbance. Initial clinical assessment revealed a left total anterior circulation syndrome, NIHSS 23.

Initial imaging

Brain NCCT: No acute ischemic changes. CTA: Proximal left MCA (M1 segment) occlusion with some (non-occlusive) thrombus in the left terminal internal carotid artery.

CTP (Figure 2.1): Decreased CBV (A, F) and CBF (B, G) in the left basal ganglia and part of the left insula, with more prolonged DT (D, I) in this area. DT is only mildly prolonged in the rest of the MCA territory with relatively normal left cortical CBF and CBV. Prolonged MTT in most of the left MCA territory (C, H). Automated core-penumbra maps (autoMIStar, L) show core 11.7m and penumbra 19.8mL. Dynamic CTA shows early and complete filling of MCA branches beyond the occlusion (Figure 2, K).

Similar results using RAPID software (Figure 2.2): Decreased CBV (A and E) and CBF (B and F) with larger area of prolonged MTT (C and G). Tmax (D and H) is prolonged mildly in the cortical MCA regions, but is more severely prolonged in the left basal ganglia. Automated core-penumbra maps show a core of 12 mL and a penumbra of 36mL.

Progress

Patient received therapy with intravenous alteplase followed by thrombectomy resulting in complete recanalization of the MCA. Total time of ischemia (onset to recanalization): 270 minutes. Patient progressively improved, NIHSS: 5 at 24 hours, NIHSS: 2 at 72 hours post procedure. 24 hour MRI showed presence of an acute ischemic lesion involving left lenticular nucleus and left corona radiata.

Comment

Another case that shows the importance of collateral flow in acute ischemic stroke. Compare the size of the perfusion lesion in this patient to the previous case with similar occlusion locations. This patient, despite a proximal M1 occlusion, has a relatively small total perfusion lesion (DT>3 seconds) of 31.5mL. This reflects much better collateral flow than in the previous patient. Nonetheless, this patient clearly should be treated aggressively as they have target mismatch.

The case also highlights a well-known phenomenon, that dominant hemisphere stroke will often cause a very high NIHSS due to the bias of the scale towards language deficits, and so may overestimate the 'true pathophysiologic deficit'. Nonetheless, the presence of 'clinical-core mismatch' to select patients for reperfusion therapy, has been revisited by the recent DAWN study(1). There is previous data indicating that clinical-core mismatch is highly

specific (>85%) for the presence of some tissue at risk (penumbra), but it has poor sensitivity (only detects presence of penumbra in 46% of cases)(2), and the extent of clinical-core mismatch does not correlate well with the volume of the penumbra. This is exactly the finding in the current case. Use of a clinical-core mismatch criteria may unnecessarily exclude some patients with imaging defined penumbra from reperfusion therapy, although the current patient had both perfusion-core mismatch and clinical-core mismatch, thus there were no doubts he needed aggressive reperfusion therapy.

Practice Tip: Note the marked difference in the size of the penumbra (as defined by DT >3 seconds or Tmax >6 seconds) compared to the visual MTT lesion. There is considerable data showing MTT overestimates tissue truly at risk (by assessing follow-up infarct in patients without recanalization)(3-7). This is because the MTT lesion often includes regions of benign oligaemia (due to excellent collaterals) that mean the tissue is unlikely to infarct even without recanalization. Both Tmax and DT have been shown to be significantly more accurate than MTT at predicting tissue that infarcts without recanalization/reperfusion. Tmax and DT are also more direct measures of collateral flow, whereas MTT reflects fluctuations in both CBV and CBF (MTT = CBV/CBF). However, both the overall visual Tmax lesion and the visual DT lesion are actually quite similar to the total MTT lesion. It is simply that most of the visual Tmax lesion is <6 seconds and the visual DT lesion is <3 seconds, due to excellent collateral flow. Therefore, the automated software does not identify the large part of the hypoperfused left MCA territory as penumbra. It is important to note, however, that both software identified this patient as having target mismatch, and therefore had a high probability of good outcome with successful reperfusion therapy.

Proximal occlusion with poor collaterals

Clinical case

79 year old male, admitted to the Coronary Care Unit for an elective coronary angioplasty. The patient had a witnessed sudden onset of speech arrest and right side weakness whilst an inpatient. The acute stroke team was notified rapidly, who confirmed the presence of a left total anterior circulation syndrome, NIHSS: 24. Multimodal CT was commenced 20 minutes after symptom onset.

Initial imaging

Brain NCCT: No acute changes. CTA: Proximal left MCA occlusion (mid M1, after origin of the left temporal artery). Dynamic CTA derived from CTP acquisition (Figure 3, M). Left-Coronal view, early and late CTA phases showing antegrade filling of inferior temporal branch but minimal retrograde filling of distal MCA branches beyond occlusion. Right - Axial view, early and late CTA phases showing minimal retrograde filling of distal MCA branches beyond the occlusion, indicating very poor collateral supply.

CTP (Figure 3): Relatively small region of decreased CBV (A, G) in the deep left MCA territory with more extensive reduced CBF in deep and cortical MCA territory (B, H). Prolonged MTT (C, I) and DT (D, J) in the entire left MCA territory. Automated core-penumbra maps (autoMiStar, Figure 3, N) show a large left MCA core (volume 95 mL) and extensive surrounding penumbra (volume 257.9mL). However, much of the penumbra has very severe delay (DT>6s lesion = 199mL), indicating poor collateral flow (E, K). MRI-DWI 48 hours post thrombectomy (F, L): Extensive MCA infarction despite technically successful mechanical thrombectomy.

Progress

Complete recanalization of left MCA was achieved with a total time to ischemia of 160 minutes. However, the patient had minimal improvement at 24 hours (NIHSS 20). Brain MRI performed at 48 hours after stroke onset showed extensive acute left MCA infarction. The patient succumbed 6 weeks later.

Comment

This patient suffered an in-hospital stroke, with witnessed symptom onset and relatively short total time of ischemia (160 minutes). However, the outcome was very poor despite early and complete recanalization. The initial imaging, performed just 20 minutes after symptom onset, showed presence of an extensive perfusion lesion, with a relatively large core (95 mL), and a large penumbra. Despite the large penumbra, there was evidence of poor collateral flow, both qualitatively (on dynamic CTA) and quantitatively (delay time >6s lesion close to 200 mL).

This case and the previous shared similar occlusion sites, and also similar pre-morbid factors and age. The recanalization time was also faster (100 minutes less) in the current patient. A 'clock' based approach would suggest that the second case should have the better outcome. These cases are good examples to support the concept that treatment selection should be not be solely based on an 'onset to intervention time' approach, and that the 'tissue clock' and the 4 Ps are more important.

Note the relatively preserved CBV in this case. Imaging occurs at a single timepoint in a dynamic process. If the collateral flow is very slow (as demonstrated by very prolonged DT or Tmax) the CBF will be reduced (as in this case), but the total blood volume may still be relatively preserved. Drop in CBV to very low levels is probably the last phase in this dynamic process, and likely reflects total failure of local vessel autoregulation, as well as

poor collateral flow(8). Thus, measures of contrast delay (DT, Tmax) may be very prolonged with relatively preserved CBV, if imaging occurs at a timepoint before there is failure of local autoregulation.

This is a classic case of 'futile' reperfusion with large core and very poor collaterals (9). There is data to show a core >70 mL means probability of benefit from IV lysis is very low(10), but we are currently uncertain where the upper core volume threshold is to say there is no benefit from thrombectomy. Indeed, some have questioned withholding reperfusion therapy (particularly thrombectomy) on the basis of a large core and/or a malignant mismatch pattern (11). 'Malignant mismatch' is where the core may be relatively small (not the case in this patient), but the penumbra is still quite large, but is composed of tissue with severe delay. These patients appear to do poorly even if they achieve reperfusion with IV lysis. This might indicate that infarction of the penumbra with severe delay may be just 'around the corner'(9, 12) and the (often delayed) reperfusion seen with IV lysis may not be timely enough to save the tissue from infarction. As this case demonstrates, the chances of tissue salvage may be relatively low even with effective endovascular therapy. We suggest that seeing such an imaging profile should, at the very least, make the treating neurologist and interventionalist be very guarded about response to therapy and prognosis when discussing acute treatment options with the family. More data is needed on outcomes following thrombectomy for both groups of patients: those with large CBF cores and those with cores <70 mL, but with large volumes of penumbra where the delay is severe (e.g. Tmax >12 seconds or DT > 6 seconds).

Practice tip: This case highlights the problem of assessing collaterals with single phase (or even multiphase) CTA. If one only had the first timepoint (e.g. an arterial-weighted CTA) to assess on CTA then it would be assumed collateral flow is very poor. Conversely, if one only

had the second timepoint it might be assumed collateral flow was at least moderate. Collateral flow may be reliably (and quickly) quantified by assessing the Delay Time or Tmax maps. In this case there is a very large lesion with severe delay (DT >6 seconds), more than double the size of the (already quite large) core.

Spontaneous reperfusion

Clinical case

61 year old male, with no vascular risk factors, who was brought to the ED with one hour of left sided weakness. On arrival at hospital, the patient's family felt there had been some improvement in his weakness. The neurological assessment revealed moderate left and leg weakness, with left facial palsy, and dysarthria (NIHSS 8).

Imaging

Brain NCCT: No acute ischemic changes. CTA: No intra or extracranial occlusion.
CTP (Figure 4): Right parietal hypoperfusion, with tiny core and small penumbra (Fig 4, K). The raw perfusion maps on the higher slice (F-I) show a typical pattern seen in acute ischemia that corresponds with the core-penumbra map (decreased CBV and CBF with prolonged MTT and DT). However, the small region of hypoperfusion is surrounded by increased CBV (A, F) and CBF (B, G), along with decreased MTT (C, H) and DT (D, I) throughout much of the right MCA territory. MRI-DWI (E, J) at 24 hours shows a tiny right cortical infarct (J) within the acute hypoperfusion area.

Progress

Given the early partial recovery and, more importantly, the imaging pattern indicating spontaneous reperfusion, it was decided not to give the patient intravenous tPA. The patient was discharged asymptomatic (NIHSS 0) after 3 days.

Comments

It is difficult to tell clinically and with NCCT alone if the mechanism of improving an acute stroke patient is spontaneous reperfusion, or a temporary fluctuation reflecting improved collateral flow beyond a persistent occlusion. Multimodal CT can give the answer. In this case we see a pattern of hyperperfusion surrounding a small area of hypoperfusion (13). Based on the clinical picture and the imaging pattern it is highly probable the patient had a large vessel occlusion (e.g M1 or proximal M2) that spontaneously lysed with fragments embolising to a more distal branch of the arterial circulation (M4 segment) that are beyond the resolution of CTA to detect.

Practice tip: A pattern of spontaneous reperfusion (increased CBV and CBF + decreased MTT and DT) may precede major clinical improvement. In cases such as the current (where there is still a significant clinical deficit) the treatment decision is unclear. It is still uncertain whether the patient could gain some benefit by be treated with tPA. We would generally decide based on the size of the residual hypoperfusion lesion. Our data suggests there is little to gain from treatment if it is <15 mL(14). Nevertheless, ongoing trials, as TEMPO-2, targeting the question of thrombolysis for minor stroke with an occlusion on CTA and/or perfusion lesion on CTP, could help to clarify this dilemma (15).

Lacunar stroke

Clinical case

63 year old male, smoker, presented to ED after 4 hours of dysarthria with mild right sided arm and leg weakness. NIHSS: 3. Clinical diagnosis was of a lacunar syndrome.

Imaging

Brain NCCT (Figure 5, E): The width of left internal capsule may be slightly wider than the right. No vascular occlusion was present on CTA.

CTP (Figure 5, A-D): Normal CBV (A), decreased CBF in the left internal capsule (B), with prolonged MTT (C) and DT (D) in the same area, this is topographically consistent with the clinical presentation. (Figure 5)

Brain MRI (F): 24 hour MRI-DWI showing infarction in the left internal capsule matching with the prolonged MTT and DT area seen on acute CTP.

Progress

The patient presented with minor motor symptoms (limb drift only), so thrombolysis was not given. He improved over the next 12 hours, with an NIHSS of 1 at 24 hours.

Comment

The ability of CTP to detect very small lesions has traditionally thought to be limited. However, a recent publication (16) assessed the value of CTP in lacunar infarction and found that the sensitivity was highest for MTT compared to Tmax, CBV and CBF (Figure 5).

The benefits of thrombolysis for lacunar stroke remain uncertain, but we suggest that a significant motor deficit, particularly in the presence of a matching perfusion lesion, is a strong indication for treatment.

Practice Tip: If a lacunar stroke is suspected carefully assess all perfusion maps, especially MTT and DT. As the perfusion lesion is small in lacunar stroke it can be difficult to distinguish between a true lesion and 'noise'. Thus, it is important to focus on the brain location that may be affected based on the clinical lacunar syndrome (e.g. corona radiata, internal capsule).

Acute versus subacute infarction

Clinical case

75 year old male presented 90 minutes after sudden onset of left sided weakness and sensory deficit, NIHSS 7. No clinical contraindications to thrombolysis. The patient was said to have had a TIA 3 days prior (with similar symptoms) at another hospital.

Imaging

Brain NCCT: Hypodensity in right parietal region, which is not hyperacute (likely subacute).

Brain CTA: Normal.

CTP: Automated core-penumbra maps (autoMiStar), showed right parietal hypoperfusion (core 3 mL, penumbra 36 mL) (Figure 6, M). CTP source images (CTPSI) (Figure 6, E and K) - apart from the hypodensity seen on NCCT (blue arrows) there is more extensive hypodensity outside the acute perfusion lesion (red arrows). This was not obvious on acute NCCT. There is also evidence of subtle reperfusion (white arrows) - Increased CBF and decreased MTT and DT (Figure 6, A-D / G-J). Brain NCCT 2 hours later shows acute intracerebral haemorrhage into the region of subacute infarction with subarachnoid and intraventricular extension.

Progress

This patient received thrombolysis and deteriorated soon thereafter with symptomatic intracerebral haemorrhage.

Comment

The acute NCCT and CTPSI showed hypodensity suggestive of recent (subacute) infarction. CTPSI may be better to identify this than NCCT (as in this case). The symptoms three days prior likely resulted in the subacute infarction (meaning this was not a TIA despite resolution of symptoms).

Practice Tip: Beware of any hypodensity on NCCT or CTPSI outside of the perfusion lesion (especially if it is adjacent). This suggests recent spontaneous reperfusion, which may increase bleeding risk after thrombolysis(14). In this case, the subacute lesion was (correctly), not identified as infarct core by the software, as reperfusion had already occurred with restoration of perfusion (indeed, the raw perfusion maps show evidence of hyperperfusion in this region). Always check the raw perfusion maps – you may also see evidence of spontaneous reperfusion that correlates with the hypodensity on NCCT/CTPSI (increased CBV and CBF, reduced MTT and Delay Time/Tmax).

Part 2 – Stroke Mimics and Stroke Artefacts

Stroke mimics are common in the emergency department, with prevalence of mimics varying between series, from 1.8% (17) to 16.7% (18,19). Our experience, particularly when DWI (rather than clinical diagnosis alone) is used as to confirm acute ischaemia, suggests that these figures are significantly underestimated (20). Multimodal CT may provide some clues to the true aetiology of mimics, including focal seizures, migraine with aura, or brain tumour. We also find that completely normal multimodal CT in a patient with an acute focal

neurologic deficit (bearing in mind CTP may miss brainstem ischemia or lacunar infarcts) can be a pointer towards a psychogenic ('functional') stroke mimic. We do not believe it is good medicine to treat such patients with thrombolysis (21). For the same reason, we recommend that multimodal CT should also be applied to all telestroke consultations prior to a treatment decision (22).

Migraine with aura

Clinical case

67 year old male with a past medical history of hypertension and migraine. He was brought to the hospital two hours after of a progressive onset of headache associated with visual disturbance, followed by speech disturbance. The stroke team noted presence of a fluctuating mixed mild aphasia, no visual field deficits were present.

Imaging

Brain NCCT and CTA: Normal.

CTP (Figure 7): Increased CBV (A) and CBF (B) in the left insula / parietal cortex. Slightly prolonged DT (D) more posteriorly, with normal MTT (C).

Progress

There was progressive recovery of the aphasia and headache over 12 hours. Brain MRI, including DWI, performed 24 hours after the episode was normal, and a diagnosis of migraine with aura was made.

Comments

Migraine with aura is one of the most common stroke mimics seen in the ED. Some of clinical features described here, such as the progressive onset of visual disturbance (rather than sudden onset) are unusual in stroke. Of course, when the patient has aphasia it can be impossible to obtain a precise history. Despite the atypical presentation, with a patient in their 60's, stroke should always be carefully considered. In this case, the acute CTP changes could be consistent with early reperfusion following acute ischemia, with increased CBF and CBV, although typically the DT and MTT would be reduced.

In a classic paper, Olesen (23) described that migraine with aura commenced with a decrease in regional CBF (when the aura appears) followed by an increase on CBF (when the headache predominates). Nevertheless, clinical presentations are not so straightforward and the timing of headache and aura is often overlapping. In this case, symptom onset was 2 hours prior to imaging, so hyperperfusion changes would be expected in migraine with aura. If the imaging assessment were performed earlier, hypoperfusion might be seen, in these cases the presence of a perfusion deficit crossing vascular territories can help to differentiate migraine from stroke (24). However, in our experience, it is very rare to see focal hypoperfusion in migraine. If one does not see focal hyperperfusion in migrainous cases, usually the CTP is normal.

Practice Tip: Focal hyperperfusion (corresponding topographically with the symptoms) must be carefully looked for when migraine with aura is suspected. However, this pattern may also represent spontaneous reperfusion after acute ischaemia. A combination of hypoperfusion and hyperperfusion (as in case 4) means stroke is much more likely than migraine.

Complex partial seizure

Clinical case

86 year old female was assessed in ED after a 60 minute duration of speech arrest. Neurological examination was normal. Initial brain NCCT and MRI (including DWI, and post-contrast T1) showed no acute change and no focal lesion, although there was generalized moderate cortical atrophy. A transient ischemic attack affecting left MCA territory was the clinical diagnosis.

Three days later, during admission, the patient experienced a similar episode, lasting several hours. The examination revealed a fluctuating mixed aphasia, with, again, no motor deficit. Altered conscious state was also observed during the episode. CTP was performed during the event.

Imaging

Brain NCCT and CTA: Normal.

CTP (Figure 8, A-E): A Increased CBV in the left insula B: Increased CBF in the left insula. C: Decreased MTT time on the left insula D: Normal automated core-penumbra maps. E: Decreased DT in the left insula.

MRI-DWI (F): No acute ischemia.

Progress

Based on the presence of stereotyped episodes with altered level of consciousness, complex partial seizures were the working diagnosis. After initiation of sodium valproate no further episodes were described. EEG showed focal slowing in the left temporal region.

Comment

As in migraine with aura, perfusion changes described in seizure are bimodal. In patients with ictal activity focal hyperperfusion occurs initially, with increased CBF and CBV, and decreased DT and MTT (25). This is followed by post ictal focal hypoperfusion (decreased CBF and CBV) with prolonged MTT and decreased CBV and CBF (26). Although the CTP pattern observed in this patient is consistent with an ictal event, increased CBV and CBF, along with decreased MTT and DT, could also be consistent with reperfusion after acute ischemia, but this did not fit with the clinical picture (recurrent episodes, and normal DWI).

Interestingly, both previous cases (migraine with aura, and complex partial seizure) had focal increases in both CBF and CBV, but with different DT/MTT patterns. Theoretically, the decrease in DT and MTT observed in seizures may be related to increased local metabolism. However, in migraine there is no increase in metabolism, thus reactive vasodilation could potentially explain the slightly prolonged DT (Figure 7).

Practice tip: In patients with presenting with an acute focal neurologic deficit suspected as stroke, the CTP pattern of hyperperfusion is increased CBV/CBF, and decreased MTT/DT. However, this can also occur with an ictal event. Careful correlation between the clinical picture (clinical history, onset and evolution of the symptoms) and the CTP pattern is required. A mixture of hypoperfusion and hyperperfusion is more suggestive of stroke (Case 4).

Tumour

Clinical case

65 year old female, was brought to ED because of a wake-up onset of unusual behaviour associated with speech arrest. The neurological examination showed presence of a fluent

aphasia, with some mild impairment of comprehension (difficulties with complex commands) and fluctuating level of alertness. No motor disturbance.

Imaging (Figure 9)

Brain NCCT (E) showed presence of oedema (note effacement of cortical sulci) affecting the left parietal cortex. CTA: Normal.

CTP: Increased CBV (A) and CBF (B), prolonged MTT (C) in the left parietal cortex. (Figure 9) CTP source image (D) shows contrast enhancement in the left parietal cortex. (F) Hyperintense lesion on FLAIR.

Progress

The patient suffered a generalized tonic-clonic seizure minutes after CT, so the hyperperfusion seen on CTP could be related to an ictal component, but its appearance is much more typical of a hypervascular tumour with neovascularization. Note the difference between the previous case in the hyperperfusion pattern on CTP (which likely reflects ictal hyperperfusion). The CTP pattern in this case is not typical of ischemia. Taking into account the clinical picture described, the patient's presentation was likely due to a partial complex seizure that generalized after the CT.

Comment

In such cases, where there is doubt between a hypervascular lesion and an ictal event, follow-up perfusion imaging could be helpful to differentiate. However, in this case the abnormal NCCT and CTP source image (although not the same as a contrast-enhanced CT, CTP SI may potentially give somewhat similar information) point to the correct diagnosis. In this

patient, MRI confirmed presence of an infiltrative lesion, and biopsy revealed an infiltrative anaplastic astrocytoma.

Practice Tip: Although perfusion maps were quite abnormal in this case, it is always important to review the NCCT and CTPSI carefully, as these give the clue to the diagnosis.

Common CT perfusion artefacts

No software is perfect, and stroke patients sometimes move in the scanner. Thus, what appears to be a 'lesion' on CTP can be related to motion artefact. Other common artefacts can include chronic hypoperfusion related to a chronic infarct, or slow contrast injection which can lead to low contrast signal and widespread spotty 'noise' artefact which, at times, may be called a 'lesion' by automated software. Slow contrast injection due to either a poorly placed IV cannula or low cardiac output can also lead to truncation of the contrast bolus. Always check that the venous outflow curve has returned at near baseline by the end of the acquisition(27). Otherwise CBV and CBF may be underestimated (potentially overestimating core) (Figure 10: Arterial (red) and venous (blue) input function curves, with presence of an abnormal arterial and venous curve (dampened curves)).

We stress that a good stroke clinician must always assess whether a perfusion 'lesion' is topographically consistent with the clinical presentation. Always beware of bilateral perfusion 'lesions', generally these reflect artefact, unless the patient has basilar ischaemia.

Streak artefact

Clinical case

59 year old female, with sudden onset of left sided weakness. Neurological assessment at 3 hours showed right total anterior circulation syndrome (NIHSS 20).

Imaging (Figure 11)

NCCT (3hours 20 minutes after symptom onset): Subtle but extensive right temporoparietal hemisphere ischemic changes. CTA: Right terminal internal carotid occlusion with MCA extension.

CTP: CBV (A, G), CBF (B, H), MTT (C, I), DT (D, J). Diagonal lines across the raw perfusion maps, this is classic streak artefact due to patient motion. The AIF curve (M) shows several peaks that are typically seen with patient motion. This has led to erroneous underestimation of the automated core/penumbra maps E, K), which are much less than the overall area of extensive right hypodensity seen on CTPSI (F, L)

Comment

Streak artefact is due to patient movement during the CTP acquisition. In general, this artefact is relatively easy to identify, the raw CTP maps will show a striped (diagonal) perfusion deficit that do not follow a vascular territory. In these cases, CTP maps should not be taken into account in the treatment decision-making process.

Practice Tips:

- Always review the AIF as a cross-check on scan quality - Compare the difference between an abnormal AIF curve (M) with a normal AIF curve of an ideal case (N). Note that some perfusion software (e.g. RAPID, MIStar) allows review (and editing) of individual timepoints for motion.

- Always carefully review the CTPSI for hypodensity. Because CTPSImay provide somewhat similar information to a contrast-enhanced CT, the hypodensity may be more apparent than on NCCT.
- Beware of acute hypodensity outside the perfusion lesion – this indicates the infarct core on CTP is underestimated and can be due to:
    - partial reperfusion, or
    - perfusion lesion artefact (as in this case), or
    - improvement in collateral flow at time of CTP.
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