Cerebral Perfusion Imaging in Hemodynamic Stroke: Be Aware of the Pattern

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Summary

Reduction of the cerebral perfusion pressure caused by vessel occlusion or stenosis is a cause of neurological symptoms and border-zone infarctions. The aim of this article is to describe perfusion patterns in hemodynamic stroke, to give a practical approach for the assessment of colour encoded CT- and MR-perfusion maps and to demonstrate the clinical use of comprehensive imaging in the workup of patients with hemodynamic stroke.

Five patients with different duration cause and degree of hemodynamic stroke were selected. The patients shared the typical presentation with fluctuating and transient symptoms. All were examined by MR or CT angiography and MR or CT perfusion in the symptomatic phase. All patients were examined with diffusion weighted imaging. All five cases showed the altered perfusion patterns of hemodynamic insufficiency with a slight or marked increase in CBV in the supply area of the affected vessel and only slightly reduced or maintained CBF. The perfusion disturbances were most easily detected on the MTT maps. Border-zone infarctions were seen in all cases.

The typical pattern for hemodynamic insufficiency is characterized by increased CBV, normal or decreased CBF and prolonged MTT in the affected areas. The increased CBV is the hallmark of stressed autoregulation. Reading the color-encoded perfusion maps enables a quick and

robust assessment of the cerebral perfusion and its characteristic patterns. Internal border-zone infarctions can be regarded as a marker for hemodynamic insufficiency. Finding of the typical rosary-like pattern of DWI lesions should call for further work up.

Introduction

Reduction of the cerebral perfusion caused by vascular stenosis or occlusion is a common cause of neurological symptoms and border-zone infarctions in the peripheral parts of the cerebral vascular system. Imaging studies have shown an incidence of this stroke type as high as 19-64%¹⁻³.

The typical pattern of ischemic areas in the cortical and deep border-zones is easily visualized on diffusion weighted imaging (DWI) in the acute phase. Perfusion imaging can play an important role in identifying hemodynamic insufficiency and in grading its severity. With MR perfusion imaging (MRP) and CT perfusion imaging (CTP) there are diagnostic tools available at most hospitals enabling assessment of the cerebral perfusion^{4,5}.

The hemodynamics in cerebral hypoperfusion have been most notably studied with positron emission tomography (PET) and typical patterns of cerebral hypoperfusion have been described early⁶. However this knowledge is often not applied in clinical routine. Even in scientific papers and presentations perfusion

changes are often just characterized as “disturbed perfusion” mainly referring to the apparent but unspecific changes on mean transit time (MTT) or time-to-peak (TTP) maps. This superficial approach misses important information provided by the cerebral blood flow (CBF) and cerebral blood volume (CBV) maps.

In most cases visual interpretation of the color encoded perfusion maps allows a thorough assessment of the type, extension and severity of the perfusion alteration without need for time consuming ROI measurements

The aim of this article is to point out the typical perfusion patterns in hemodynamic stroke in cases with well defined disease, to give a practical approach for the use of color encoded CT- and MR-perfusion maps and to demonstrate the clinical use of comprehensive imaging in the workup of patients with a clinical presentation of hemodynamic stroke.

Material and Methods

Five representative cases with different degree, duration and cause of cerebral hypoperfusion examined with different equipment were chosen to demonstrate the altered perfusion patterns regardless of the underlying cause or the imaging modality. All cases were examined with MRI including DWI and either MRP or CTP. Case 1 was examined on a Siemens Vision 1.5 Tesla (Siemens Medical Systems, Erlangen, Germany). DWI was performed with spin echo – echo planar imaging (SE-EPI). MRP with dynamic susceptibility contrast (DSC) technique was performed with gradient echo – echo planar imaging (GE-EPI). Case 2 was examined on a Philips Intera 1.5 Tesla (Philips Medical Systems, Best, The Netherlands). DWI was performed with SE-EPI. CTP was performed on a GE Lightspeed 16 slice scanner (GE Healthcare, Waukesha, USA). Case 3 and 5 were examined on a Siemens Allegra 3 Tesla (Siemens Medical Systems, Erlangen, Germany). DWI was performed with a SE-EPI technique. MRP was performed with GE-EPI. Case 4 was examined on a Siemens Allegra 3 Tesla. DWI was performed with SE-EPI. CTP was performed on a Philips Brilliance 64 slice scanner (Philips Medical Systems, Best, The Netherlands).

MRP maps of the CBV, CBF and MTT were calculated with the Lund Perfusion Program (LUPE)⁷ utilizing the singular value decomposition (SVD) method⁸. The arterial input func-

tion (AIF) used for deconvolution calculation of CBF was determined from an artery in the Sylvian fissure on the not affected side. Histogram optimized color encoding was done by normalization to the cerebellar perfusion⁹.

The CTP parameter maps (CBV, CBF, MTT) in case 2 were calculated with the brain perfusion software provided in the GE Advantage Workstation (AW) (GE Healthcare, Waukesha, USA) and in case 4 with the brain perfusion software in the Philips Extended Brilliance Workspace (EBW) (Philips Medical Systems, Best, The Netherlands). The pericallosal arteries were used for determining the AIF and the confluens sinuum for calculating the venous outflow curve. Both software packages utilize non documented deconvolution algorithms. For color encoding the default values of the software packages were used.

Results

Patient history and imaging results

Case 1

A 68-year-old woman with diplopia due to a partially thrombosed giant aneurysm on the cavernous portion of the right internal carotid artery (ICA) was scheduled for balloon occlusion of the ipsilateral ICA. This was considered to be the most efficient and safe treatment at that time prior to the introduction of appropriate stent material. Test occlusion was neurologically uneventful and collateral circulation was angiographically considered satisfactory. Permanent occlusion was therefore carried out. Initially no complications occurred. However, 20 hours after treatment the patient developed left-sided hemiparesis and the MRI examination performed shortly thereafter showed ro-sary-like lesions in the deep internal border-zone and sporadic lesions in the cortical border-zones on DWI on the right side. On MRP a marked increase of CBV, prolonged MTT and maintained CBF was noted in the right hemisphere (Figure 1).

The patient was treated with prolonged heparin administration and hypervolemic hemodilution increasing the blood pressure 20-30 mm Hg above normal. She recovered during the following weeks and was almost back to normal five months later with only a minor left-sided weakness. On follow up MRI examinations the MRP changes normalized and no new ischemic lesions were seen.

Figure 1 Case 1. A 68-year-old woman with a giant aneurysm on the cavernous portion of right ICA (F). MR examination 20 hours after balloon occlusion of the aneurysm shows increased CBV (A), preserved CBF (B) and prolonged MTT (C) on the right side (bright represents long MTT, dark short). Acute infarcts are seen in the deep border-zone and in the posterior cortical border-zone on DWI (D,E). With kind permission from Springer Science+Business Media: *Neuroradiology, Diffusion- and perfusion-weighted MRI in therapeutic neurointerventional procedures*, Vol 43, 2001; p 665, Cronqvist M et Al, Figure 2.

Case 2

A 75-year-old woman was admitted to the emergency department because of dysphasia and weakness in the right arm and leg with onset one hour prior to admission. Plain CT was unremarkable but CT-angiography revealed a marked stenosis of the left middle cerebral artery with a length of 10 mm. Intravenous thrombolysis was considered, but the symptoms rapidly decreased and after two hours there was only slight dysphasia remaining. The initially considered thrombolysis was not carried out and the patient was put on aspirin. Two days after admission the patient again developed dysphasia and facial paresis. Plain CT showed no signs of infarction and CT-angiography showed increased stenosis. CTP showed slightly increased CBV in the left middle cere-

bral artery (MCA) territory and reduction of CBF in the border-zone mainly between the anterior cerebral artery (ACA)/MCA and the posterior cerebral artery (PCA)/MCA territories on the left side. MTT was prolonged in the entire distribution area of the MCA. DWI showed small acute infarcts with a rosary-like distribution in the deep white matter along the lateral ventricle on the left side (Figure 2). Because of increasing symptoms on the following day a successful balloon dilatation of the stenosis was carried. After treatment the patient rapidly improved. MRP performed three days after treatment showed normalized perfusion.

Case 3

A 37-year-old woman developed progressive motor and sensory deficits in her left arm face

Figure 2 Case 2. A 75-year-old woman with dysphasia and weakness in right arm and leg. CT angiography (E) revealed a marked stenosis of the M1 segment of left middle cerebral artery. CTP shows subtle increased CBV (A), slightly decreased CBF (B) and prolonged MTT (C) (blue represents long MTT, red short) in the left MCA territory. DWI (D) shows rosary-like acute infarcts in the internal border-zone on the left side.

and tongue. Plain CT at admission was unremarkable. Clinically multiple sclerosis was suspected, but MRI two days after the CT examination showed DWI lesions in the deep border-zones in the centrum semiovale on the right side. MR angiography showed occlusion of the right ICA. In addition a tight stenosis was found on the left side involving the most distal parts of the ICA and proximal parts of the ACA and MCA. MRP showed increased CBV, minimally decreased CBF and prolonged MTT in the MCA territory on the right side (Figure 3). Subsequently a diagnosis of Takayasu's arteritis was made based on arterial wall abnormalities in the right subclavian artery, in the aorta and a somatostatin receptor scintigraphy. During the initial investigation renal involvement was suspected and the patient was treated

with angiotensin-converting enzyme (ACE) inhibition for a short period. During this period of blood pressure lowering the weakness of her left arm worsened suddenly and an MR examination showed new DWI lesions in the left deep border-zone.

Case 4

A 64-year-old woman with a history of arterial hypertension was suffering from frequent episodes of mild paresis with short duration on her right side. Work up with MR and MRA showed a high grade, short stenosis of the intracranial part of the ICA on the left side and a few small infarctions on DWI in the deep border-zones. CTP showed a slight increase in CBV, an almost symmetrical distribution of CBF and prolonged MTT, which was most

Figure 3 Case 3. A 37-year-old woman with progressive motor and sensory deficit in her left arm face and tongue. MR angiography (E) disclosed an occlusion of the right ICA and a tight stenosis at the top of the left ICA affecting both the proximal ACA and MCA. MRP shows increased CBV (A), minimally decreased CBF (B) and prolonged MTT (C) (red represents long MTT, blue short) in the right MCA territory. DWI reveals rosary-like acute infarcts in the deep border-zone on the right side (D).

clearly seen in the border-zone between the distribution areas of the left ACA/PCA and the MCA (Figure 4).

Because of the frequent episodes of neurological symptoms and the evidence of border-zone infarctions the patient was scheduled for an angiography which confirmed the approximately 90% stenosis of the left ICA. Furthermore, a relatively poor pial collateral supply of the ACA and MCA supply areas of the left side was seen. Due to these findings a balloon dilation of the intracranial stenosis followed by stent application was performed, resulting in a restoration of the vessel lumen with a remaining stenosis of approximately 50%.

MRI including MRP the next day showed normalized perfusion and no new infarctions. The clinical status of the patient was clearly im-

proved with a cessation of the TIA like episodes.

Case 5

A 67-year-old woman with a history of transient ischemic attack (TIA) with left-sided symptoms was admitted to the Department of Neurology with a new attack of left-sided weakness and slurred speech. At admission the systolic blood pressure was elevated to 220 mm Hg. CT showed a small low attenuation area in the right centrum semiovale and doppler ultrasound did not show any significant stenosis of the carotid arteries. The condition was interpreted as a lacunar infarction and the patient improved spontaneously. Antihypertensive treatment was started and the patient was discharged.

Figure 4 Case 4. A 64-year-old woman with frequent episodes of mild right-sided paresis of short duration. CT perfusion measurements show a subtle increase in the CBV (A) on the left side, almost symmetrical CBF (B) and prolonged MTT (C) (blue/green represents long MTT, red short). DWI shows deep border-zone infarcts on the left side (D). Angiography reveals a short high grade stenosis of the intracanalicular part of the left ICA (E).

Three days later the patient was admitted again due to fluctuating left-sided weakness and hemianopia to the left. CT showed a new right-sided occipital infarct and CTA revealed a marked stenosis of the M1 segment of the right MCA (Figure 5). DWI showed acute infarcts in the centrum semiovale and in the occipital lobe on the right side. MRP showed markedly increased CBV, reduced CBF and prolonged MTT in the vascular territory of the right MCA (Figure 6). The patient was sent to the angio suite and a successful dilatation and stenting of the M1 segment was performed. Following treatment the hemiparesis and hemianopsia improves slightly but significant symptoms remain.

The pattern of CBV, CBF and MTT changes observed in the five cases are presented in Table 1 together with the distribution patterns of deep or cortical border-zone infarctions.

Table 1 Diagnosis, CBV / CBF changes on the color encoded maps at the state of hemodynamic insufficiency (one arrow means slight changes, two arrows apparent changes) and presence of cortical (CBZ) or deep border-zone (DBZ) infarcts.

Case	Cause	CBV	CBF	MTT	CBZ	DBZ
1	ICA occlusion, iatrogenic	↑↑	=	↑↑	+	+
2	MCA stenosis	↑	↓	↑↑	–	+
3	ICA occlusion, Takayashu's disease	↑	↓	↑↑	–	+
4	ICA stenosis	↑	=	↑↑	–	+
5	MCA stenosis	↑↑	↓↓	↑↑	+	+

Figure 5 Case 5. A 67-year-old woman with an attack of left-sided weakness and dysphasia. Perfusion measurement shows markedly increased CBV (A), markedly reduced CBF (B) and prolonged MTT (C) in the supply area of the right MCA DWI shows acute infarcts in the deep border-zone area (E) and the cortical borderzone between the MCA and PCA (D). CTA shows stenosis of the M1 segment of the MCA (F).

Discussion

Hemodynamic disturbances of the cerebral perfusion as a primary cause of stroke have been known for more than half a century¹⁰ and several studies have shown that border-zone infarction counts for a significant part of all brain infarctions^{1-3,11}. Before the introduction of CT and MR in the routine work up of patients with the clinical presentation of stroke, pathological studies indicated that border-zone infarctions count for approximately 10 % of all brain infarctions. Imaging studies based on CT performed later showed an incidence of 19% to 64%¹⁻³.

DWI plays an important role in identifying the often subtle border-zone infarctions and both MRA and CTA enables depiction of the underlying pathology e.g. stenosis or occlusion of brain supplying arteries. Perfusion studies

with MR and CT have been introduced in clinical practice during the recent years and can contribute important information on the distribution and degree of impaired cerebral perfusion.

The patients selected for this presentation shared a common clinical presentation with fluctuating symptoms and typical radiological findings of deep border-zone infarctions and disturbed hemodynamics. However, the duration and etiology of the reduced perfusion pressure are different.

The occlusion was iatrogenic with a short duration following balloon occlusion of the carotid artery in case 1. The symptoms and duration in cases 2 and 4 were observed during a couple of days and in case 3 symptoms were slowly progressive over several weeks. The cause of the stenosis in cases 2 and 4 is not clear, but might have been caused by dissec-

tion. The underlying cause of the perfusion deficits in case 3 was Takayasu's arteritis with multiple occlusions and stenoses with impairment of both the direct and the collateral supply. The stenosis in case 5 was probably caused by hypertension and general arteriosclerotic disease.

Border-zone infarctions

The etiology of border-zone infarctions in hemodynamic stroke is still a matter of debate and has been extensively discussed by Momjian¹². The typical pattern of deep border-zone infarctions with multiple, deep lesions along the border-zone between the deep arterial system of the MCA and the superficial arterial systems of the ACA, MCA and PCA has been stated as a marker for hemodynamic insufficiency¹³⁻¹⁶ and was seen in all our cases.

Cortical border-zone infarctions between the superficial arterial systems of the MCA and PCA respectively were seen in cases 1 and 5. These infarctions were located in the posterior cortical border-zone in case 1 with increased MTT on the MRP examination. It is difficult to tell whether the infarctions were caused primarily by the perfusion changes or by emboli from the interventional procedure or from the stenotic area. The posterior cortical border-zone infarctions in case 5 matched with an area with marked decreased CBF and increased CBV on the MRP map during the same examination, indicating a hemodynamic cause for this infarction.

The typical pattern of border-zone infarctions is difficult to detect by CT and can totally be missed in the acute phase as shown in case 2. Repeated CT examinations were interpreted as normal, whereas the small infarcts were readily disclosed on MRI, demonstrating that DWI is by far the most sensitive method to detect even minimal border-zone infarctions¹⁷.

Detection of the typical pattern of border-zone infarction in both cases 3 and 5 guided the diagnostic procedure to the final diagnosis of hemodynamic stroke. In particular, the existence of deep border-zone infarctions with normal doppler ultrasound of the carotid arteries, as in case 5, should be reason for complementary work up with perfusion studies and angiographic studies of the entire supraaortic vessel tree to reveal potentially perfusion impairment and stenoses outside the imaging window of ultrasound, as in all our cases.

Perfusion:

technical considerations and patterns

The principles of cerebral hemodynamics following the reduction of the perfusion pressure have been described in detail by Powers mainly based on the results of PET studies⁶. Today all hospitals with modern MR or CT equipment have the facilities to assess cerebral perfusion with MRP and CTP.

Even if there are specific advantages and drawbacks to each of these modalities, both are suitable for comprehensive imaging of stroke. The choice whether MR or CT is used for the assessment of the cerebral perfusion for a specific case is normally determined by factors such as availability and readiness.

In subacute situations MRI is preferable because of its combination of angiographic capabilities with high sensitivity for border-zone infarctions with DWI.

It is generally accepted that color encoding of the CBV and CBF maps significantly facilitates the visual assessment of perfusion patterns. Nevertheless there are no published standards or recommendations concerning the color scales in CTP and MRP. This lack of standard is reflected in our material by the fact that part of the the coloring in the diverse software packages is markedly different. For the CBV and CBF increasing values results in a shift from blue towards red in the rainbow scale both for CTP and MRP. However for the MTT an increase results in a shift from red to blue in the CTP packages, whereas the same increase results in a shift from blue to red in the MRP package. Apart from that, in our experience the default color scales in the CTP software packages (AW, GE and EBW, Philips) and the histogram optimized color encoding in the MRP software package LUPE^{7,9} generate reproducible and consistent coloring of the perfusion maps.

The main challenge in the primary assessment of cases with hemodynamic insufficiency, when examined with MRP or CTP, is to find a practical approach to extract the needed information about the location and the severity of the perfusion changes. Reading the color-encoded perfusion maps enables a quick and robust assessment of the cerebral perfusion and its characteristic patterns. ROI measurements are normally not useful for individual cases, when the distribution of the perfusion changes initially is unknown.

Despite different etiology and duration all cases showed the typical perfusion pattern following the dynamics of autoregulation in hemodynamic stroke, previously described in PET-based studies^{6,18}.

A practical approach for assessing the perfusion patterns is to start by analyzing the MTT maps. Disturbance of the cerebral perfusion is always most easily detected on the MTT maps, because the MTT, as a quota of the CBV and CBF, shows less pronounced differences between the white matter and the grey matter. Furthermore both the increase in CBV and the decrease in CBF results in a prolongation of MTT in the affected area as seen in all our cases. The changes in the CBV and CBF maps are often more subtle especially in hemodynamic stroke. However CBV and CBF maps provides crucial information on the characteristics and degree of the perfusion deficits and should therefore be analyzed carefully.

A slight (cases 2, 3 and 4) or marked (cases 1 and 5) increase in CBV in the supply area of the affected vessel reflects a dilatation of the resistive vessels in the hypoperfused region. This increase in CBV compensates for the apparent decrease of the perfusion pressure resulting in only slightly reduced (cases 2 and 3) or almost maintained (cases 1 and 4) CBF in the same region (table 1). The marked reduction of CBF in case 5 indicates a more severe reduction of the perfusion pressure. These perfusion patterns are not exclusively for hemodynamic stroke but can also occur in acute embolic stroke with e.g. M1 occlusion in combination with good collateral supply.

With the mechanism of autoregulation the direct impact of the decreased perfusion pressure on CBF is moderated. However increase in CBV by vasodilatation can only compensate for a drop of the perfusion pressure to a certain extent. With maximal vasodilatation further decrease of the perfusion pressure leads to a decrease of CBF as seen in cases 2, 3 and most clearly in case 5.

Significantly reduced CBV and CBF indicate the breakdown of autoregulation with imminent tissue ischemia as in acute ischemic stroke of any cause, not seen in any of our cases.

Increased CBV can be seen as the hallmark of hemodynamic insufficiency still compensated by autoregulation. The state of the CBF tells whether this compensation is sufficient (cases 1 and 4) or deficient (cases 2, 3 and 5).

Clinical impact

Patients with hemodynamic insufficiency are often highly sensitive for further decrease of the perfusion pressure due to their almost maximally stressed autoregulation. This susceptibility for blood pressure changes is well illustrated in case 1 where the patient recovered from increasing symptoms by an intentional temporary rise in blood pressure and in cases 3 and 5 where increased neurological symptoms followed an attempt to lower the patient's blood pressure. For that reason anti-hypertensive therapy in patients with clinical signs of hemodynamic stroke and vessel occlusions or significant vessel stenoses should be avoided as long as the cause of the hypoperfusion is untreated.

Perfusion studies in all the presented cases provided important information on the extent and grade of hemodynamic insufficiency. This information helps to differentiate whether the patient's symptoms are mainly due to the already established infarctions shown with DWI or due to hemodynamic insufficiency. Decreased CBF as in cases 2, 3 and 5 indicates hypoperfusion and deficient collateral supply, with a high risk for further hemodynamic infarctions.

DWI plays a key role in finding the often subtle border-zone infarctions in hemodynamic stroke. However, in our experience perfusion studies often yield critical information for the further treatment of these patients, especially when interventional treatment is considered or the effect of such procedures has to be followed up.

Conclusions

The typical pattern for hemodynamic insufficiency is characterized by increased CBV, normal or decreased CBF and prolonged MTT in the affected areas.

The increased CBV is the hallmark of stressed autoregulation. Internal border-zone infarctions in addition to perfusion changes can be regarded as a marker for significant hemodynamic insufficiency. Finding of the typical rosary-like pattern of DWI lesions should always call for further work up with perfusion and angiographic studies. Reading the color-encoded perfusion maps enables a quick and robust assessment of the cerebral perfusion and its characteristic patterns.

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