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ORIGINAL COMMUNICATION

Perfusion-CT imaging in epileptic seizures

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Abstract

Introduction PCT is used in the diagnosis of acute neurological syndromes, particularly stroke. We aimed to evaluate PCT abnormalities in patients with acute epileptic seizures or status epilepticus (SE).

Methods We collected patients undergoing acute PCT for the suspicion of acute ischemic stroke (AIS), who received a final diagnosis of focal seizures or generalised seizures with a post-ictal deficit, with or without concomitant AIS. PCTs were retrospectively analysed for the presence of hyper- and hypoperfusion, and results correlated with delay from seizure onset, aetiology, type of seizures and the presence of electrical SE.

Results Half of the 43 consecutively identified patients had regional PCT abnormalities—hyperperfusion in 13 (30%) and hypoperfusion in 8 (19%)—and 4 (9%) had AIS. Among patients with hyperperfusion, six (46%) had a focal deficit during imaging acquisition (two a normal clinical status, one altered consciousness and four ongoing seizure); nine (69%) of these patients had a SE; none had a stroke. All patients with hypoperfusion had focal neurological deficit; three (37%) of them a simultaneous ischemic stroke (in the remaining five, hypoperfusion was considered to be related to the seizure post-ictal phase). In the 22 with normal perfusion, 9 had a focal deficit (10 a normal clinical status, 2 altered consciousness and 1 ongoing seizure); 3 had a SE, and 1 had a stroke. Patients with SE featured a higher prevalence of hyperperfusion (9/13 [69%] vs. 4/30 [13%] without SE, $p = 0.00$).

Conclusion In patients with acute epileptic seizures, regional hyperperfusion on PCT may suggest an ongoing or recently resolved SE, whereas hypoperfusion may be due to post-ictal state or simultaneous AIS. These observations might help attributing focal deficits to epileptic seizures rather than stroke, allowing for targeted therapy.

Keywords Stroke · Epilepsy · Perfusion imaging

Introduction

Between 14 and 45% of patients presenting to the emergency room for a suspected ischemic stroke are affected by a non-stroke condition with similar clinical presentation (stroke mimics), the most frequent being epileptic seizures [1]. Adding to the confusion, infarctions (particulary cortical infarctions) sometimes have concomitant seizures that can mask the diagnosis of acute ischemic stroke (AIS) [2, 3]. Although IV thrombolysis carries a low haemorrhagic risk if administered to patients with a stroke mimic, improving the diagnostic accuracy between real stroke and imitators would prevent the administration of unnecessary reperfusion therapies [4].

Like ischemic stroke, epileptic seizures may lead to changes in cerebral perfusion that can be observed in the ictal and the post-ictal phase, the first featuring mainly focal hyperperfusion and the latter hypoperfusion [5]. These alterations are well demonstrated by SPECT imaging [5] and perfusion MRI [6] but the limited availability of these techniques hampers their clinical application in the emergency setting.

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On the other hand, PCT imaging is widely used in the diagnosis of AIS [7] and is becoming a validated tool for identification of patients with good potential for acute recanalization therapies [8]. However, the role of PCT in the acute evaluation of patients with epileptic seizures or status epilepticus (SE) has been explored only in single patient reports or smaller case series [9–12].

The aim of the present study is to describe the perfusion alterations seen on PCT in patients experiencing (A) focal seizures/SE or (B) generalised seizures/SE with a post-ictal neurological deficit. In addition, we assessed PCT changes depending on delay from seizure onset, seizure aetiology and type, clinical presentation and the presence of SE.

Methods

In our hospital, PCT following NCCT and CT angiography of head and neck is part of the emergency diagnostic workup for patients with a clinical suspicion of AIS [13] or TIAs. We included in this study patients who underwent PCT for suspected AIS or TIA within 24 h from symptom onset, but where further observation and workup lead to a diagnosis of (A) focal epileptic seizures/SE or (B) generalised seizures/SE followed by post-ictal deficit. Concomitant AIS was not an exclusion criterion. One of the authors maintained a list of all such patients admitted between February 2003 and August 2007, which then was retrospectively analysed.

The diagnostic workup of all patients in the study included an EEG within 24 h from hospital admission. Epileptic seizure (or SE) was diagnosed if clinical seizure activity was observed before or with the onset of new neurological deficits, or if a new focal neurological deficit was associated with electric epileptic activity on EEG. A diagnosis of simultaneous acute ischaemic stroke was sought by additional neuroimaging (brain CT or MRI with DWI) if patients had a persistent focal deficit for more than 1 h; in the absence of an ischemic lesion, such patient's deficit was considered a Todd's palsy.

According to our protocol, PCT images are acquired on a 64-row MSCT-GE (acquisition parameters: 80 kVp and 100 mA) and processed either on AW-Volumeshare 2 workstation or on custom Philips (Brilliance Workspace Portal) PCT post-processing software. For each patient MTT, CBF and CBV maps are calculated [14]. Detailed technical aspects have been described elsewhere [15].

A certified stroke neurologist (PM) and a neuroradiologist (PB), aware of the side of the neurological manifestations, independently analysed PCT data; in case of divergent imaging interpretation, a consensus was reached during joint reading of the images. Hyperperfusion was defined as increase in CBF and decrease in MTT. This qualitative visual analysis was preferred because of the lack of a precise quantitative definition of hyperperfusion [11, 16]. Hypoperfusion was defined on visual inspection of the raw PCT images as focally decreased CBF and increased MTT relative to the healthy side (clinically defined), independently of the CBV [17].

Patients' clinical status during PCT was classified into: normal, ongoing seizures or SE (defined as ongoing seizures for at least 5 min, or repetitive seizures without regaining a baseline clinical state [18]; diagnosis of nonconvulsive SE required an EEG), decreased level of consciousness (somnolence or stupor) without clinical seizure, and post-ictal deficit without clinical seizure.

We categorised the seizures/SE aetiology according to the international classification criteria valid during the observation period [19], while the dominant seizure semiology at the time of PCT was distinguished into focal without consciousness impairment (simple partial), focal with consciousness impairment (complex partial), and generalised [20]. EEG findings were categorised into normal, diffuse non-epileptiform, focal non-epileptiform, focal epileptiform and generalised epileptiform.

Statistical analysis

Comparisons between the three groups (hyperperfusion, hypoperfusion, normal perfusion) were performed with Fisher's exact test (categorical variables) and Kruskal–Wallis test (numerical variables), considering significant P-values < 0.05. Statistical analysis was performed with R statistical software (version 3.0.2, R Core Team [2013], R Foundation for Statistical Computing, Vienna, Austria).

These data, obtained from routine care during hospitalisation, were analysed after anonymization, which does not require formal ethical approval or patient consent under national law.

Results

During the observation period, 43 patients (20 women) fulfilled the inclusion criteria. Median age was 73 years and six patients were previously diagnosed with epilepsy. Two patients (5%) underwent repeat CT and 28 patients (65%) a brain MRI with DWI, while in the remaining 13 (30%) no follow-up imaging was considered necessary to clarify the diagnosis by the treating physician (none of these patients had hypoperfusion on acute PCT). PCT was abnormal in 21 patients (overall sensitivity of 49%): 13 (30%) had focal hyperperfusion, whereas 8 (19%) had focal hypoperfusion.

Hyperperfusion most frequently involved only the cortical grey matter, while focal hypoperfusion was more commonly extended to both cortical and subcortical regions ($p < 0.01$). Among patients with hyperperfusion, six

(46%) had a focal deficit during imaging acquisition (two a normal clinical status, one altered consciousness and four ongoing seizure); nine (69%) of these patients had a SE; none had a concomitant ischemic stroke. All eight patients with hypoperfusion had focal neurological deficits at the time of PCT; three (37%) of them had a simultaneous ischemic stroke (in the remaining five, hypoperfusion was considered to be related to the post-ictal phase of the seizure, including the one case with a nonconvulsive SE, resolved at the moment of PCT acquisition). In the 22 with normal perfusion, 9 (41%) had a focal deficit during PCT acquisition (10 a normal clinical status, 2 altered consciousness and 1 ongoing seizure), 3 of them (13%) a SE, and 1 (5%) a stroke.

We observed a significant association ($p = 0.01$) between the patients' clinical status at the time of PCT acquisition and imaging findings, with hypoperfusion exclusively observed in patients with focal neurological deficit and hyperperfusion associated with ongoing clinical seizure activity (Table 1). Hyperperfusion was found in 9/13 (69%) with a SE at any time ($p < 0.01$ if compared to patients without SE), in 4/5 (80%) for a SE still clinically persistent at the moment of PCT and in 5/8 (62%) for a SE already resolved before PCT. The frequency of PCT alterations was not significantly affected by the delay from symptom onset-to-PCT, the seizure type, nor the seizure aetiology (Table 1).

In four patients, epileptic seizures were part of the clinical manifestation of an AIS: three had focal clinical signs and focal hypoperfusion, one had neither focal symptoms nor perfusion alterations, but a medial frontal cortical lesion in the ACA territory was found on follow-up MRI. Of the former three patients, two were thrombolysed while the third was not initially recognised as stroke.

We present here three representative clinical situations of PCT findings in patients with acute epilepsy:

Case 1: hyperperfusion close to chronic stroke lesion

A 71-year-old man presented new-onset tonic–clonic movements of left limbs followed by persistent left hemiparesis. PCT performed at 180 min from symptom onset showed a focal area of hyperperfusion in the right parietal lobe, close to an area of hypoperfusion corresponding to a silent chronic ischemic lesion seen on non-contrast CT (Fig. 1). The left hemiparesis improved rapidly after the CT; therefore, IV thrombolysis was not administered. The EEG performed 5 h after symptom onset did not show any significant alteration. No follow-up imaging was performed. The final diagnosis was focal seizure related to the chronic ischemic lesion. The hyperperfusion observed on acute CTP was interpreted as a consequence of the focal epileptic activity.

Case 2: hypoperfusion related to acute ischemic stroke

A 76-year-old woman presented a generalised tonic–clonic seizure followed by left hemiparesis and neglect, with NIHSS of 11. The PCT at 130 min disclosed a focal right anterior MCA hypoperfusion (Fig. 2). She received IV thrombolysis 165 min after symptom onset. The EEG performed 210 min after symptom onset was normal. NIHSS was one at 24 h and zero at day 7. Brain MRI with diffusion-weighted imaging on day 3 confirmed an acute stroke in the hypoperfused region. The hypoperfusion observed on acute PCT was interpreted to represent the true acute ischemia.

Case 3: post-ictal hypoperfusion without old stroke

A 38-year-old man acutely presented aphasia, right-sided hemiparesis and severe psychomotor agitation requiring intubation and sedation with diprivan and midazolam. At his arrival in our hospital, 2 h and 40′ from symptom onset, sedation was withdrawn and neurological examination demonstrated normalisation of the right limb strength and persistent but gradually improving aphasia with Gerstmann syndrome and mild psychomotor agitation. NCCT and CT angiography were unremarkable, but PCT showed left posterior MCA territory hypoperfusion (Fig. 3). No antiepileptics were given and the patient completely recovered within few hours. A putative diagnosis of partial complex SE with post-ictal Todd's palsy was made, supported by the EEG performed on day 3 showed left frontal intermittent rhythmic activity. Follow-up brain MRI was normal. Given the absence of acute vascular occlusions on acute CTA and the absence of ischemic lesions on MRI, the hypoperfusion observed on acute CTP was considered related to the seizure and post-ictal deficit. No aetiology of the SE was identified.

Discussion

In patients with acute seizures or SE and focal symptoms undergoing PCT, we found focal perfusion abnormalities in about half of cases and AIS in 10%.

Hyperperfusion was found in one-third of patients, mainly during or after SE. Hypoperfusion was less frequent (one-fifth of patients), correlated with persistent focal neurological deficits and was explained in one-third of cases by an AIS. In these cases, it is likely that seizures were a consequence of stroke and hypoperfusion was primarily caused by stroke and not epilepsy.

Two previous studies on seizure patients reported a frequency of PCT alterations of 38% and 78%, respectively [10, 21]. Different patient selection and onset-to-PCT delay of 72 and 3 h, respectively, may in part explain these

Table 1 Patients' demographic details and PCT findings

	Total ($n=43$)	Hypo-perfusion ($n=8$)	Normal ($n=22$)	Hyper-perfusion ($n=13$)	p value
Age	72.9 (62.6–77.9)	68.2 (47.5–76.3)	73.3 (65.6–80.3)	73.1 (60.8–78)	0.45
Femal sex	20 (46.5%)	3 (37.5%)	12 (54.5%)	5 (38.5%)	0.61
Onset-to-PCT (h)	2.6 (1.7–4.4)	2.4 (1.6–3.3)	2.8 (1.8–4.7)	2.2 (1.7–5.2)	0.55
Onset-to-EEG (h)	11.2 (4.7–21.2)	17.2 (5.3–47.5)	14.7 (5.4–22.2)	7.8 (2.4–18.4)	0.18
Clinical state during PCT					0.01
Normal clinical status	12 (27.9%)	0 (0%)	10 (45.5%)	2 (15.4%)	
Altered consciousness	3 (7%)	0 (0%)	2 (9.1%)	1 (7.7%)	
Focal deficit	23 (53.5%)	8 (100%)	9 (40.9%)	6 (46.1%)	
Ongoing seizures	5 (11.6%)	0 (0%)	1 (4.5%)	4 (30.8%)	
Seizure type					0.58
Simple partial convulsive	2 (4.7%)	0 (0%)	2 (9.1%)	0 (0%)	
Simple partial nonconvulsive	5 (11.6%)	0 (0%)	4 (18.2%)	1 (7.7%)	
Complex partial convulsive	6 (13.9%)	0 (0%)	4 (18.2%)	2 (15.4%)	
Complex partial nonconvulsive	9 (20.9%)	4 (50%)	2 (9.1%)	3 (23.1%)	
Primary generalised	10 (23.3%)	2 (25%)	5 (22.7%)	3 (23.1%)	
Secondary generalised	11 (25.6%)	2 (25%)	5 (22.7%)	4 (30.8%)	
Status epilepticus	13 (30.2%)	1 (12.5%)	3 (13.6%)	9 (69.2%)	<0.01
EEG findings[a]					0.42
Normal EEG	7 (16.7%)	1 (14.3%)	6 (27.3%)	0 (0%)	
Diffuse non-epileptiform	5 (11.9%)	1 (14.3%)	3 (13.6%)	1 (7.7%)	
Focal non-epileptiform	17 (40.5%)	3 (42.9%)	8 (36.4%)	6 (46.1%)	
Focal epileptiform	13 (30.9%)	2 (28.6%)	5 (22.7%)	6 (46.1%)	
Seizure aetiology					0.56
New stroke	4 (9.3%)	3 (37.5%)	1 (4.5%)	0 (0%)	
Old stroke	12 (27.9%)	3 (37.5%)	6 (27.3%)	3 (23.1%)	
Neoplasm	2 (4.7%)	0 (0%)	2 (9.1%)	0 (0%)	
Metabolic	6 (13.9%)	0 (0%)	3 (13.6%)	3 (23.1%)	
Focal gliosis(scar)	2 (4.7%)	0 (0%)	1 (4.5%)	1 (7.7%)	
Other specific	8 (18.6%)	1 (12.5%)	4 (18.2%)	3 (23.1%)	
Unknown	9 (20.9%)	1 (12.5%)	5 (22.7%)	3 (23.1%)	
Thrombolysis	3 (7%)	2 (25%)	0 (0%)	1 (7.7%)	0.06
Lobe involved					0.37
Frontal	8 (38.1%)[b]	4 (50%)	n.a.	4 (30.8%)	
Parietal	1 (4.8%)	1 (12.5%)	n.a.	0 (0%)	
Temporal	1 (4.8%)	0 (0%)	n.a.	1 (7.7%)	
Occipital	3 (14.3%)	2 (25%)	n.a.	1 (7.7%)	
Fronto-parietal	6 (28.6%)	1 (12.5%)	n.a.	5 (38.5%)	
Multilobar	2 (9.5%)	0 (0%)	n.a.	2 (15.4%)	
Localisation					<0.01
Cortical grey matter	14 (66.7%)[b]	2 (25%)	n.a.	12 (92.3%)	
Cortical GM + WM	6 (28.6%)	6 (75%)	n.a.	0 (0%)	
Cortical + deep GM + WM	1 (4.8%)	0 (0%)	n.a.	1 (7.7%)	

For continuous variables median and interquartile range are displayed. For categorical variables column percentages are displayed. p value of Fisher's exact test for categorical variables and Kruskal–Wallis test for continuous variables

[a]EEG not performed in one patient

[b]Percentages calculated on the number of patients with abnormal perfusion ($n=21$)

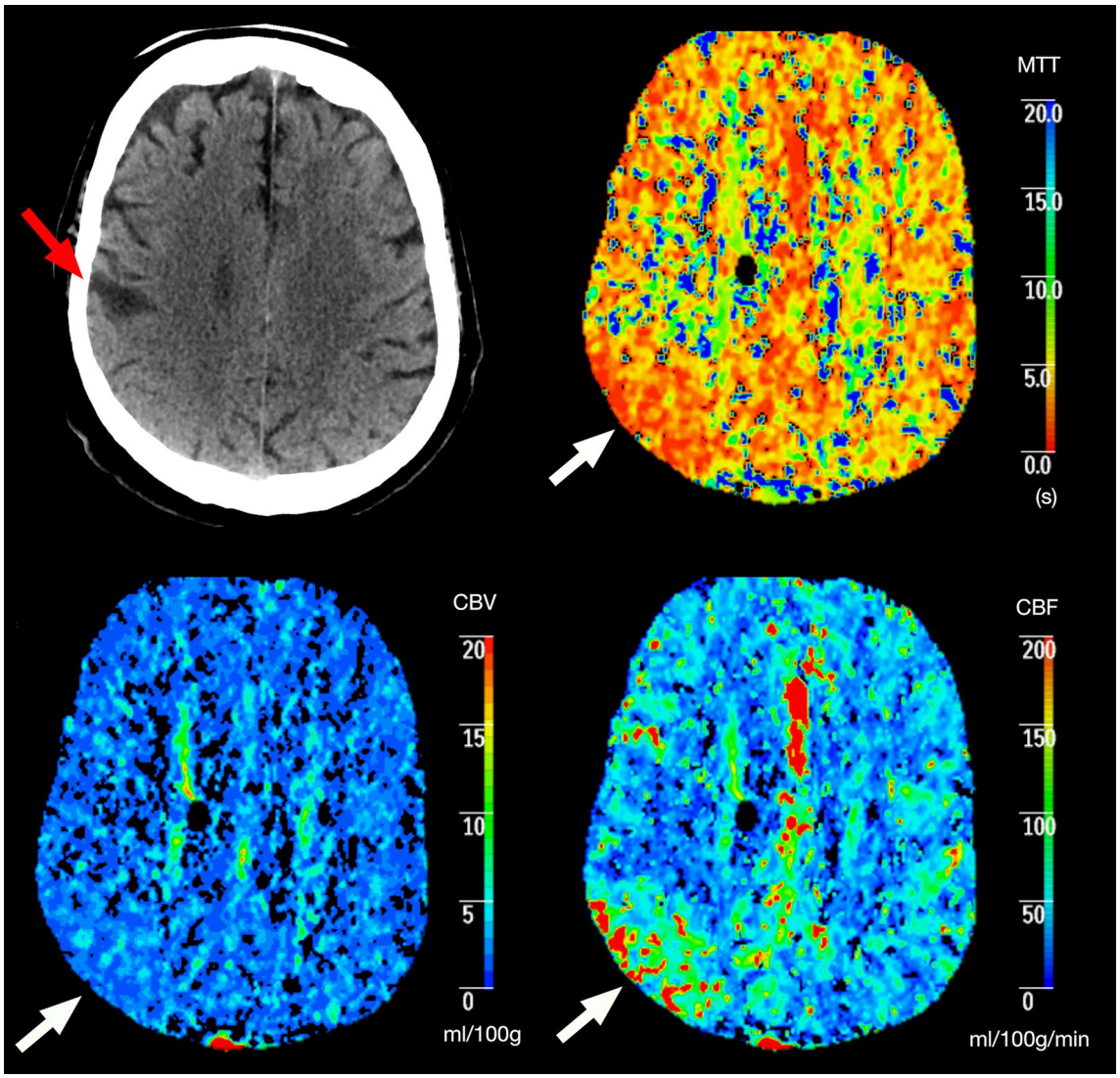

Fig. 1 Acute non-contrast CT (top left) showing an old ischemic lesion (red arrow); mean transit time (top right), cerebral blood volume (bottom left) and cerebral blood flow (bottom right) maps showing a focal area of hyperperfusion (white arrows) in the right parietal lobe, close to the area of hypoperfusion corresponding to the chronic stroke

discrepancies. In these studies, it was not possible to estimate the rate of concomitant AIS, since it was an exclusion criterion.

We identified a significant relationship between patients' clinical status and PCT findings: in patients with post-ictal neurological deficit we observed both hypoperfusion and hyperperfusion, however, while hypoperfusion was invariably associated to the presence of post-ictal palsy, hyperperfusion could be found also in patients with normal neurological status or ongoing SE.

Previous studies already showed that hyperperfusion, together with hypermethabolism, accompanies the abnormal synchronised discharge that characterises focal epileptic seizures [22]. In our patients hyperperfusion on PCT was frequently associated with ongoing or resolved SE. Its frequency was non-significantly higher for a SE still persistent at the time of PCT acquisition (80%) than for a SE already resolved (62.5%). The association between SE and hyperperfusion has already been described in a case series of 4 patients with nonconvulsive SE, and in a previous study on 19 patients with seizures and persistently altered mental status [11, 23]. Our observation suggests that hyperperfusion may persist also after the resolution of seizure activity, both in patients with and without post-ictal palsy. Hyperperfusion can also be observed in AIS or TIA during the reperfusion phase ("luxury perfusion") [17], but none of our four patients with acute stroke and seizures presented regional hyperperfusion, which in acute cerebral ischemia seems to be very rare [7/506 (1.4%) in our AIS registry [13] over the last 2 years, if PCT performed within 24 h; unpublished

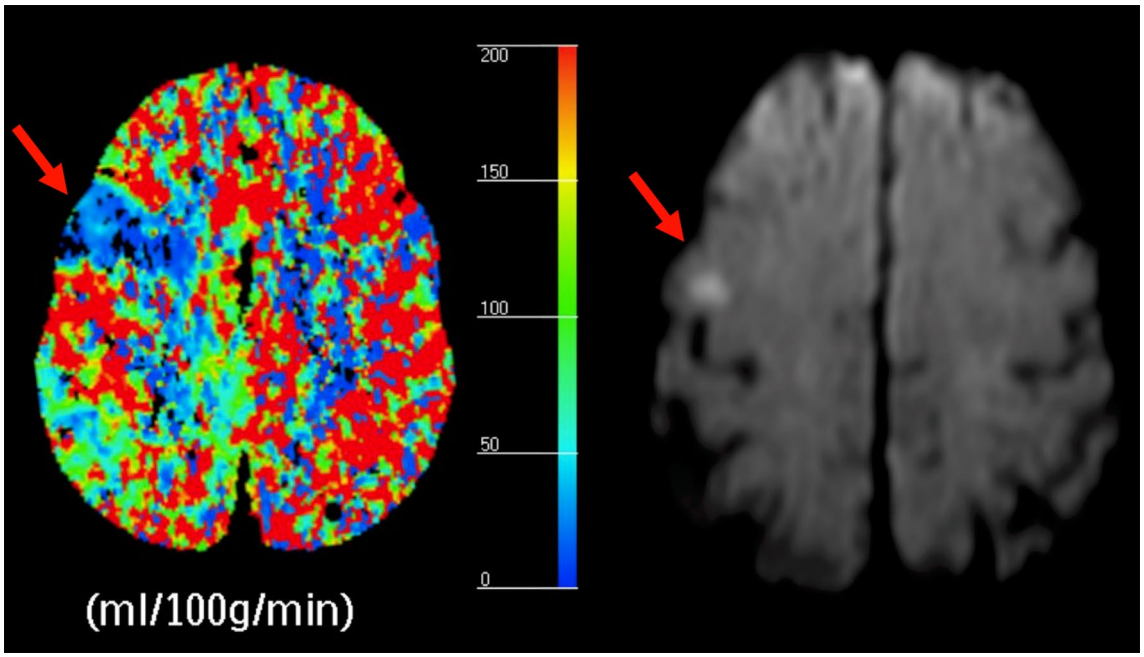

Fig. 2 Acute cerebral blood flow map (left) showing an hypoperfused area (arrow) in the superficial territory of the right middle cerebral artery, where 2 days later MRI with diffusion-weighted imaging (right) demonstrated an acute ischemic lesion

data]. Therefore, if hyperperfusion is observed in a patient with an acute focal deficit, an epileptic origin should first be considered, even in the absence of initial "positive" neurological symptoms.

Focal hypoperfusion, the diagnostic hallmark of AIS, was identified in a quarter of patients presenting a post-ictal focal deficit without acute ischemic lesion. These results, consistent with previous observations [10, 16, 24], suggest that focal hypoperfusion in patients with seizures does not invariably indicate the occurrence of an ischemic stroke, but it may also represents a functional alteration consequent to the seizure, that is more frequently observed is association with a post-ictal deficit. Recent data suggest that hypoperfusion results from arteriolar vasoconstriction induced by vasoactive substances released by neurons via COX-2-mediated pathways, as a consequence of the sustained synaptic activity occurring during seizure. Interestingly, the post-ictal deficit may directly result from hypoperfusion/hypoxia following seizure, and not from the seizure per se [25].

A more detailed analysis of PCT may be helpful to distinguish AIS from mimics, the latter often not respecting vascular territories and having milder degrees of hypoperfusion [10, 11, 26]. Still, when an acute ischemia is clinically suspected and PCT is normal or shows focal hypoperfusion, administration of IV thrombolysis should not be delayed, given its relative safety in patients with stroke mimics [27].

The clinical implications of our study are, therefore, threefold: (1) hyperperfusion on CTP in acute neurological deficits points towards an epileptic origin of the problems; (2) hypoperfusion may be due to acute ischaemia, or to a post-ictal state; in selected patients, this may be useful to initiate acute recanalization treatments, but in others, it would require more detailed imaging analysis; (3) PCT in patients with acute neurological deficits, even transient, should be performed as rapidly as possible to increase the likelihood of pathological findings; this has been recently demonstrated also for TIAs [28].

The strengths of our study are the number of included patients, making it, to the best of our knowledge, the largest cohort of acute seizure patients studied with PCT. Also, PCT was performed within 4.5 h after symptom onset in 80% of patients, making our results applicable to patients in the thrombolysis time window.

Limitations include the retrospective analysis and the limited number of acute strokes, not making possible a comparison between patients with and without AIS. Second, we did not conduct a quantitative evaluation of PCT, which might have increased the sensitivity, but would be less easily applicable to routine clinical practice. Still, our finding were comparable with previous studies in which PCT data were analysed quantitatively [11, 17]. Last, our results might have been partially influenced by the delay in performing the EEG, as determining whether a focal deficit at the time of PCT was due to post-ictal (Todd's) paralysis or ongoing focal seizure was not always possible due to lack of concomitant EEG.

In conclusion, patients with seizures may have focal hyperperfusion on acute PCT, especially in ongoing focal

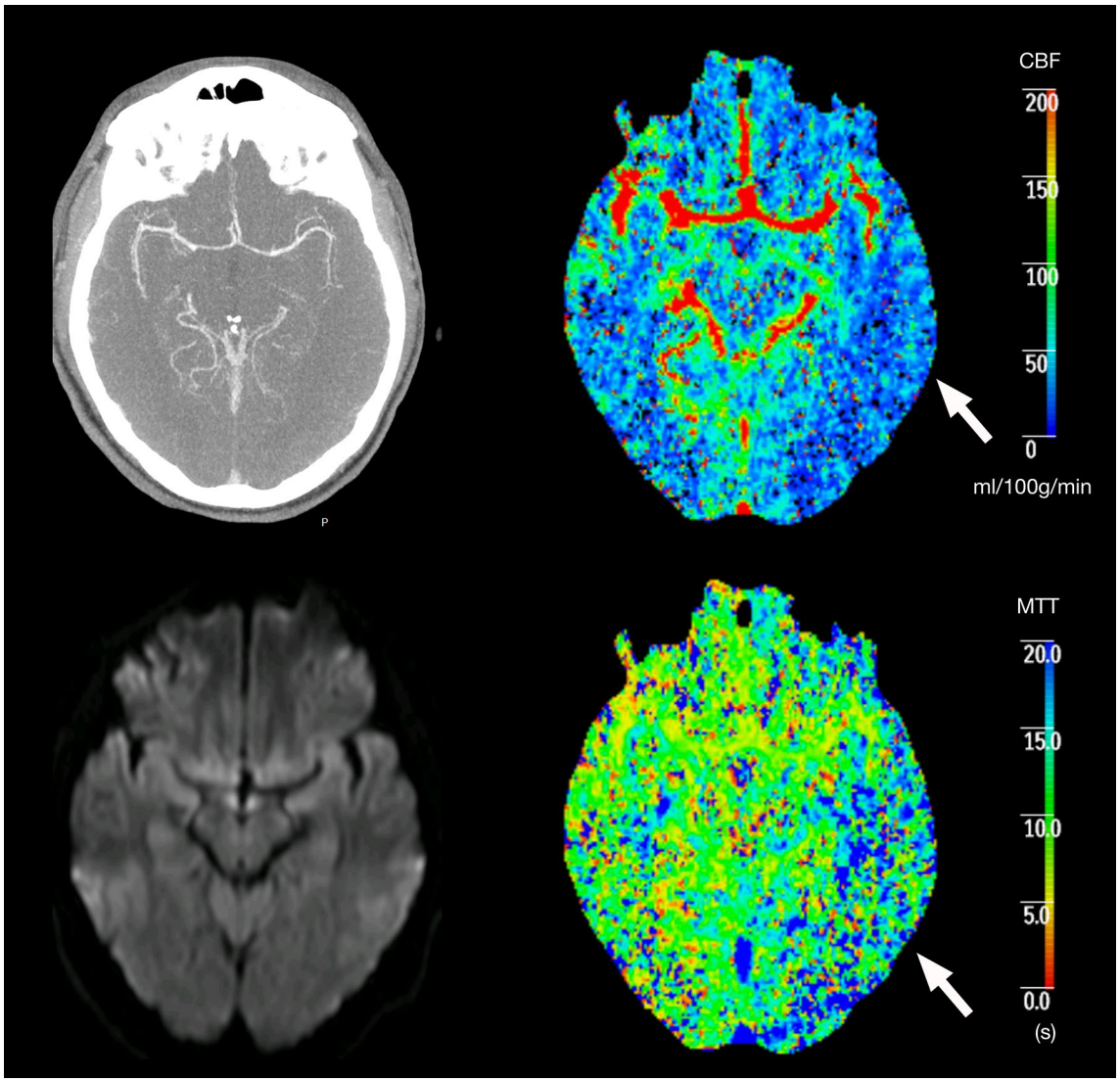

Fig. 3 Acute CT angiography (top left) showing absence of vascular occlusion, concomitant cerebral blood flow (top right) and mean transit time (bottom rigth) maps displaying a focal area of hypoperfusion (arrows) in the posterior territory of the left Middle cerebral artery and normal follow-up brain MRI (bottom left)

SE, focal hypoperfusion can be due to AIS simultaneous to seizure, or to a post-ictal state and it has a stronger correlation to concomitant clinical status than hyperperfusion. Such results may help to take acute treatment decisions in patients with new-onset neurological deficits. The definitive role of PCT in triaging patients with seizures and suspected AIS needs to be tested in further, larger prospective studies.

Compliance with ethical standards

Conflicts of interest Dr. Strambo, Dr. Rey, Dr. Rossetti, Dr. Maeder, Dr. Dunet and Dr. Browaeys have nothing to disclose. Dr. Michel received research grants from the Swiss Heart Foundation, and BMS; speaker fees from Bayer, Daiichi-Sankyo, Medtronic and Stryker; honoraria from scientific advisory boards from Boehringer-Ingelheim, Bayer, Pfizer and BMS; and consulting fees from Astra-Zeneca and Amgen. All this support is received by his institution (CHUV) and is used for stroke education and research.

Ethical standards Data were obtained from routine care during hospitalisation and analysed after anonymization, which does not require formal ethical approval or patient consent under national law.

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