



perforation may not immediately produce symptoms of peritonitis, making an early diagnosis difficult and causing delay to treatment.³ Gallbladder perforation or rupture after blunt trauma should be a consideration in patients with symptoms and signs of an acute abdomen and shock at the time of admission or during hospitalisation. Cholecystectomy is the preferred treatment for isolated traumatic rupture of gallbladder.⁴

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LETTERSTO THE EDITOR

Clinical-scientific notes

Focal hyperperfusion on ictal cerebral perfusion computed tomography in partial seizures mimicking acute stroke

Perfusion computed tomography (PCT) utilizes computed tomography (CT) data sequentially acquired from pre-determined locations during intravenous contrast injection.¹ Commercially available softwares use raw PCT data to calculate the cerebral blood volume (CBV), meant transit time (MTT) and cerebral blood flow (CBF) maps, providing a rapid assessment of cerebral perfusion.¹ PCT facilitates acute stroke thrombolysis by confirming the presence and extent of an ischaemic penumbra. Other applications include assessment of vasospasm post subarachnoid haemorrhage, quantifying microvascular permeability in intracranial tumours and evaluating cerebrovascular reserve prior to bypass or neuroendovascular procedures for intracranial vascular stenosis.² However, PCT use in epilepsy has not been extensively explored.

An 85-year-old woman presented with 2 hours of aphasia and right-sided weakness. She suffered previous left frontoparietal strokes with residual non-fluent dysphasia. On examination, she was mute and had left gaze deviation. Right-sided upper motor neuron pattern of facial weakness was accompanied by moderate right arm and leg weakness. During the assessment, she developed right arm jerking, which persisted during CT scanning.

Non-enhanced CT brain (Fig. 1) showed two chronic infarcts in the left middle cerebral artery territory. PCT

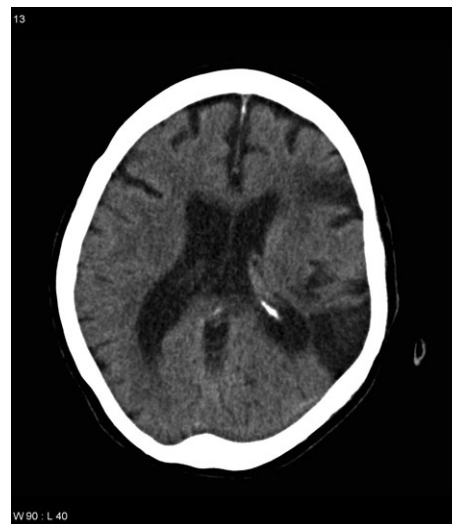


Figure 1 Non-enhanced axial computed tomography of the brain. Two chronic cortical infarcts are present in the left middle cerebral artery territory anteriorly and posteriorly. The intervening parenchyma appears normal.

(Fig. 2) demonstrated reduced CBV and CBF at these sites. However, in the peri-Sylvian region between these two infarcts, an area of increased CBV and CBF was seen, associated with reduced MTT.

Thrombolysis was not considered as clinical and radiological features were suggestive of focal seizures.

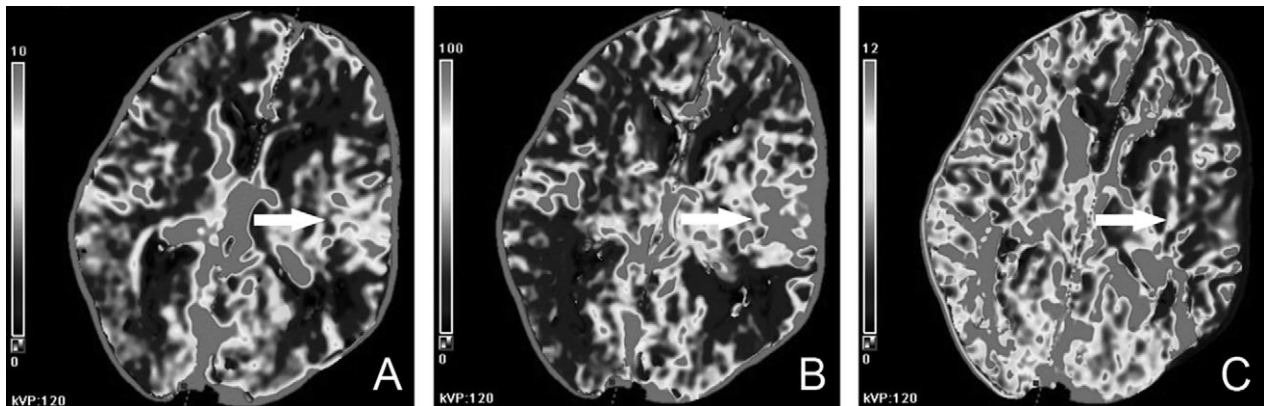


Figure 2 Perfusion computed tomography. (A) Cerebral blood volume (CBV) (mL/100 g) image demonstrates low CBV involving the two chronic left-sided infarcts compared with the normal right hemisphere. Between these two infarcts is a region of increased CBV (arrow). (B) Cerebral blood flow (mL/100 g/min) image demonstrates low CBF involving the chronic left-sided infarcts best appreciated in the posterior infarct. The region between these two infarcts demonstrates increased cerebral blood flow (arrow). (C) Mean transit time (s) image demonstrates reduced mean transit time (arrow) in the region between the two chronic infarcts.

Intravenous benzodiazepines terminated her right arm jerking. An EEG performed 24 h later showed excessive slowing and epileptiform discharges over the left mid-temporal region. She remained seizure-free on antiepileptic medication and her symptoms resolved over 3 days.

Seizure-related presentations can mimic stroke, making the initial assessment challenging. Thrombolysis for acute stroke, which aims at salvaging the ischaemic penumbra, demands accurate and timely diagnosis.¹ PCT assesses differences in regional blood flow.³ The combination of relative MTT and absolute CBV with dedicated thresholds¹ can differentiate the ischaemic penumbra (prolonged MTT, normal or increased CBV, reduced CBF) from the infarct core (prolonged MTT, markedly reduced CBV and CBF)³ and assist in identifying candidates for revascularization.

CBF to the epileptogenic region varies during different phases of a seizure. Blood flow increases at onset, but upon seizure termination, the 'post-ictal switch' phenomenon is seen where CBF transiently but dramatically reduces.⁴ This is primarily reflective of underlying neuronal metabolism.⁵ Not surprisingly, post-ictal PCT abnormalities are common, occurring in up to 37%, especially if PCT is performed within 2 hours of seizure termination.⁶ A frequent post-ictal PCT pattern is focal hypoper-

fusion (prolonged MTT, reduced CBV and CBF) not conforming to a vascular territory and often sparing the basal ganglia.⁶

In our patient, PCT was performed during the ictal phase, which produced a distinct picture of focal hyperperfusion of the epileptogenic region. The lateralization and localization of the perfusion abnormality correlated well to the clinical and electroencephalogram findings.

A stroke-directed CT protocol, including non-enhanced CT, PCT and CT angiography (CTA), can rapidly identify patients suitable for thrombolysis.³ In the presence of atypical PCT findings of hypoperfusion not conforming to traditional vascular distribution and the absence of corresponding large vessel occlusion on CTA, seizure should be considered.⁶ Similarly, in the presence of focal hyperperfusion on PCT, seizure should also be considered. The full spectrum of PCT abnormalities and their correlation to different stages of a seizure require further clarification.

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Sodium valproate – a rare cause of eosinophilic pleural effusion

A 45-year-old man was commenced on sodium valproate, 500 mg twice a day, for bipolar disorder that was not controlled with Mirtazapine. Five days later, he developed a non-productive cough and pleuritic chest pain that required hospital admission. On examination he was afebrile with oxygen saturation of 96% on room air, tachypnic and had bibasal dullness and no signs of heart failure. Chest radiograph confirmed bilateral pleural effusion and a computed tomography pulmonary angiogram excluded pulmonary embolism and any pulmonary parenchymal pathology.

A diagnostic, right-sided pleural aspirate revealed red blood cells 45 900/μL, white cell count 4000/μL, eosinophils 39%, mononuclear cells 46%, polymorphs 21%, pH 7.5, protein 50 g/L, glucose 8.5 mmol/L and lactate dehydrogenase (LDH) 202 U/L. Aspirate to serum, protein and LDH ratio, was 66% and 91% respectively. Aspirate had no bacterial growth or any malignant cells.

Haematology and biochemistry investigations revealed peripheral eosinophilia of $7.92 \times 10^9/L$, C-reactive protein 69 mg/L and erythrocyte sedimentation rate

50 mm/h. Antinuclear antibody was 1:160. Rheumatoid factor, antineutrophil cytoplasmic antibody and antibodies to extractable nuclear antigen were negative. An echocardiogram revealed preserved cardiac function and normal pulmonary artery pressure.

Drug-related pleural effusion was considered to be the most likely explanation and valproate was discontinued. He was treated with simple analgesics.

A week after hospital discharge he was readmitted to the hospital with similar symptoms. Repeat left aspirate had similar characteristics with 49% eosinophils. He was discharged home a day later.

Clinical review in 1 and 2 months revealed progressive resolution of the effusion with no recurrence of symptoms.

Our literature review revealed only 11 published cases of pleural effusion associated with valproate. Development of effusion in this series ranged from 1 to 9 months (Table 1). Our patient developed the effusion within 5 days of starting therapy. The majority of patients (91%) had an exudative effusion and 81% of them had an exudative eosinophilic effusion. Patients in this series were having between 250 mg and 1500 mg of valproate, the majority of them (78%) were having 1000 mg or more a day.

Table 1 Cases of pleural effusion associated with sodium valproate

Case report	Age (years)	Sex	Indication and dose	Duration of therapy before symptoms	Pleural fluid eosinophilia (%)	Characteristic of effusion	Resolution after cessation	Rechallenge
André <i>et al.</i> ¹	57	Female	Epilepsy, 1000 mg/day	NR	Lymphocytic	Exudative	6 months	No
Bullington ²	25	Male	Neurosarcoidosis, 250 mg/day	NR	75	Exudative	1 month	No
Catalan ³	19	Female	Psychosis, 1000 mg/day	2 months	51	Exudative	6 months	No
Fernandez-Perez ⁴	64	Female	Schizophrenia, 1500 mg/day	2 months	35	Exudative	4 months	No
Joshi <i>et al.</i> ⁵	48	Female	Epilepsy, 1500 mg/day	NR	44	Exudative	6 months	No
Kaufman ⁶	42	Male	Epilepsy, schizophrenia, 1000 mg/day	9 months	62	Exudative	Several days	No
Kravetz ⁷	34	Male	Schizophrenia, 1500 mg/day	2 months	40	Exudative	6 weeks	Yes
Savvas <i>et al.</i> ⁸	44	Female	Epilepsy, NR	20 days	40	Exudative	2 weeks	No
Shaib ⁹	38	Male	Epilepsy, schizophrenia, NR	NR	45	Exudative	10 weeks	No
Tonnellier ¹⁰	50	Female	Epilepsy, 1500 mg/day	1 month	65	Exudative	6 weeks	No
Tryfon <i>et al.</i> ¹¹	70	Male	Epilepsy, 500 mg/day	4 months	Neutrophils 65%	Transudative	15 days	Yes

NR, not reported.