Focal seizures with reversible hypodensity on the CT scan

Sir: When a postictal abnormal computed tomographic (CT) finding corresponds anatomically to the presumed ictal focus in patients with focal seizures, the abnormality is thought to reflect the existence of a pathological process responsible for the occurrence of the seizures. The CT abnormality is usually not considered to be the result of the ictal and interictal metabolic disturbances which are known to occur in and around the seizure focus. We report a case whose transient postictal focal CT abnormality appears to have been the direct consequence of recurrent focal seizure activity.

An 18-year-old right handed student was in good health until ten days prior to admission when he woke up with headache and noted he had bitten his tongue. Three days prior to admission he experienced spontaneous twitches of the right corner of the mouth and the right hand. He was unable to talk during this focal seizure which lasted several minutes. Over the next 12 hours he had four identical focal seizures. Previous medical and family history were unremarkable, blood pressure was 115/70 mm Hg, auscultation of the heart was normal. Neurological examination was normal. A CT scan on the day of admission showed a large hypodense left frontal area without displacement of the lateral ventricle or midline structures (fig). Contrast injection showed a peripheral rim of enhancement. EEG, ECG, chest radio- graphs, serum electrolytes and full blood count were normal. He was treated with phenobarbitone 150 mg nocte. A four vessel angiogram performed six days after admission showed no abnormalities of the aortic arch, the carotid and vertebral arteries. The intracerebral circulation was normal in the arterial, capillary and venous phases. A presumptive diagnosis of left fronto-temporal glioma was made. Three weeks after admission a stereotaxic biopsy under CT monitoring revealed two specimen of normal cerebral tissue. The hypodense area appeared less extensive and a transient right facial weakness was noted after the procedure. One month after admission a CT scan with and without contrast enhancement was normal. Anticonvulsant therapy was gradually withdrawn after twelve months. Over the next two years he remained free of seizures and the CT scan remained normal.

This young man's repeated focal seizures suggested an underlying focal pathologic process. The CT scan showed a large hypodense area which location was corresponding to the presumed ictal focus. This led us to perform a surgical diagnostic procedure. The adequate location of the biopsy was confirmed by CT monitoring and postoperative right facial weakness. The hypodense image had no features of mass effect and disappeared within one month. The CT remained normal over the two year follow up. The nature of the hypodensity remained uncertain. Traumatic contusion and a neoplasm were excluded. Viral meningoencephalitis and other infectious processes were unlikely as there was no fever, no EEG abnormality and no histologic clues of an inflammatory process. Cerebrovascular disease was more difficult to exclude. Arterial thrombosis or emboli from a proximal artery was unlikely in view of the patient's age, and were excluded by angiography. There was no evidence of valvular disease, cardiomyopathy or arrhythmia to account for cardiogenic emboli. Also the absence of EEG abnormalities over the large hypodense area and the complete reverse of the CT features within one month did not suggest underlying cerebral infarction. A vascular malformation may present with focal epilepsy of sudden onset. Several histologically proven cases have been reported in detail in whom angiography did not reveal the vascular mass. These occult arteriovenous malformations are shown on the unfused CT scan as an area of increased attenuation, an appearance quite different from the pictures shown in our case. Also, there was no histological clue to suggest a vascular malformation. We are left with the idea that the transient frontobasal hypodensity was related to the recurrent seizure activity.

The development of cerebral position emission computed tomography has led to new insight in the ictal and interictal metabolic disturbances around the seizure focus. These studies confirmed the known ictal increase of perfusion of the cortical epileptic focus concomitant with the increase of local cerebral glucose utilisation. The local hypoxia, accumulation of lactic acid and loss of vascular autoregulation have not been quantified. Several of these factors are thought to account for the transient abnormal radionucleide brain images shown within three days after the acute onset of idiopathic focal epilepsy in children. Cerebral angiography performed during focal status epilepticus has demonstrated transient focal arteriolar and capillary blush and early filling veins corresponding to the idiopathic seizure focus. Reports of transient focal CT abnormalities in relation to focal epilepsy are rare. To the best of our knowledge only one case had been reported in detail, prior to 1984, that of a two year old child in focal status epilepticus. The lack of similar other reports is remarkable as focal epilepsy is widely investigated by CT because the diagnostic yield of underlying structural brain disease is known to be high. Recently a paper by Rouger et al reports four adolescents with focal epilepsy and transient CT abnormalities. Although the time span between the discovery of the abnormal CT scan and the seizures is very variable two cases are very similar to our report. In conclusion, we think that repeated focal seizure activity may cause a reversible abnormal CT image of decreased density. Although this seems extremely rare, it appears advisable to repeat the CT scan in those circumstances before subjecting the patient to more invasive diagnostic procedures.

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Fig. CT scan after contrast injection on admission (a, b). A left frontal hypodensity without mass effect shows a peripheral rim of enhancement. One month later, the CT scan after contrast injection is normal (c, d).
References


Transitional hemiparesis—a cautionary tale: coexistence of phaeochromocytoma and intracranial aneurysm

Sir: The following salutary case history may prove of interest to your readers.

A 40-year-old right-handed caucasian male experienced sudden onset of tinnitus one evening. The following morning, he noted marked weakness and numbness of his left arm and leg and was admitted to hospital. On examination, he had a left hemiparesis, sparing the face, and impaired sensation to pin prick and light touch over his left arm and leg. He was hypertensive (BP 190/100 mm Hg) and treated with hydralazine and a beta blocker. The hemiparesis completely resolved after three days. The patient's haematocrit, serum urea and electrolytes, serum cholesterol, fasting lipids and blood glucose were normal. Whilst the results of urinalysis for catecholamines were awaited, additional investigations revealed normal skull and abdominal x-rays; a normal intravenous urogram and a normal head computerised tomogram (CT scan). Lumbar puncture yielded clear cerebrospinal fluid (CSF) with no red cells, less than 1 white cell and 30 mg% of protein. A right carotid angiogram demonstrated an aneurysm of the right middle cerebral artery but was otherwise normal. Surgical obliteration of the aneurysm was recommended. The patient requested transfer to a centre nearer his home prior to surgery and he was, therefore, referred to our unit. The diagnosis at this stage was (1) hypertension (2) prolonged reversible ischaemic neurological deficit (3) aneurysm of the right middle cerebral artery.

On arrival, the patient had no abnormal neurological signs. After withdrawing his anti-hypertensive therapy, he remained normotensive. In view of the clinical picture, a complete series of intracranial angiograms including the neck vessels was requested. The patient was cooperative and angiography was performed under mild sedation. However, attempted femoral catheterisation was associated with the onset of hypertension (BP 270/120 mm Hg) and the examination was aborted. It was thought that anxiety was responsible for the hypertensive episode although the patient denied this. His blood pressure returned to normal as soon as he returned to the ward. A repeated attempt at angiography under heavier sedation (Pethidine 100 mg, Droperidol 6 mg) was again aborted because of a hypertensive episode (BP 250/130 mm Hg). Urinalysis for catecholamines was requested since those performed at the referring hospital were still unavailable. Direct questioning did not reveal any history of headaches, sweating or palpitations. It was decided to perform angiography under general anaesthesia. Soon after anaesthetic induction, a hyper- tensive crisis occurred (BP 240/150 mm Hg) which was accompanied by sweating, salivation and ventricular extrasystoles. Prompt treatment (10 mg intravenous phentolamine) by the vigilant anaesthetist controlled this. Angiography did not reveal any further vascular abnormality. After repeated direct questioning of the patient, we obtained a history of occasional headaches, nocturnal sweating attacks and palpitations—none of which had he considered significant enough to warrant divulging to the admitting doctor. A provisional diagnosis of phaeochromocytoma was made and confirmed later by the results of our urinalysis for catecholamines (18-μmol normetadrenaline/24 hrs). The patient's blood pressure was controlled with Propranolol and Phenonybenzamine. A tumour of the right adrenal gland was demonstrated by whole body CT and this was subsequently removed. Histological examination confirmed the diagnosis of phaeochromocytoma. The intracranial aneurysm was successfully clipped at a later date. There was no evidence of a previous haemorrhage from the aneurysm when it was displayed at operation.

We are not aware of a reported association between intracranial aneurysms and phaeochromocytomas, although hypertension may contribute to the development of aneurysms.1 Usually, an intracranial aneurysm is identified as a cause of a sub- arachnoid haemorrhage although on occasion the presentation may be that of a tumour or transient cerebral ischaemia. Vascular anomalies of the posterior fossa, including a basilar artery aneurysm may present with episodic hypertension and mimic a phaeochromocytoma as may posterior fossa tumours.7 Phaeo- chromocytoma usually causes episodic sustained hypertension but it may present with cortical blindness or transient neurological deficits. The mechanism responsible for the latter has not been elucidated but it is possible that arterial spasm, probably involving perforating vessels arising from the proximal middle cerebral artery, may have been responsible for the transient neurological deficit that our patient experienced. Interestingly, a recent report identified large numbers of neurophyside Y (NPY) producing cells in phaeochromocytomas8 and plasma NPY levels were raised in patients with phaeochromocytomas. NPY has been shown to have vasoconstrictor activity and it is possible that release of this neuropeptide may have contributed to impaired cerebral perfusion in our patient but we admit that this explanation is speculative.

This case study illustrates the pitfalls which may be encountered when two coincidental dangerous pathological conditions are encountered in the same patient.

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