due to the redistributory changes of blood flow in face of hypoglycemia. Earlier reports indicate that hypoglycemia in the absence of cerebrovascular stenosis can cause reversible injury of internal capsule and splenium, demonstrated on MRI in a patient with transient hypoglycemic hemiparesis [11]. Kim et al. [11] reported complete resolution of the lesions within 12 h after appropriate correction of hypoglycemia, as did neurological deficits, indicating that the lesions are reversible cytotoxic edema in nature. However, in the current case the lesions were demonstrable on the third day after the onset of hemiparesis, refuting the possibility of reversible cytotoxic edema. Lacunar pontine strokes can present in a similar fashion; however, the temporal relation between the onset of hypoglycemia, neurologic deficit and partial resolution of deficit with dextrose infusion in this patient clearly establishes the cause and effect relationship.

We propose that the presence of basilar stenosis made the patient vulnerable to pontine infarct in the setting of hypoglycemia-induced cerebral flow patterns. This is the first reported case of such hypoglycemic brain injury resulting in pontine infarct in a patient with diabetes and basilar artery stenosis. Underlying cerebrovascular disease is a frequent occurrence in the elderly, diabetic population. Knowledge of vertebro-basilar stenosis may lead the physician to more fastidious management of blood glucose.

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Brain Haemorrhage and Cerebral Vasospasm Associated with Chronic Use of Cannabis and Buprenorphine

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A 34-year-old woman presented with subacute-onset headache. Her medication consisted of oral buprenorphine 5 mg o.d. in treatment for heroin addiction, which she had stopped using 8 years before. No recent change in the dose of buprenorphine was made. She had smoked 2–6 cannabis cigarettes per day since the age of 22. The day of symptom onset, she smoked 4 cannabis cigarettes followed by another one 50 min after headache onset. No alcohol or other drugs were used. Except for tobacco, no other vascular risk factors were present. Clinical examination was normal. The blood pressure was 145/86 mm Hg on admission and normalized spontaneously 1 h later. Blood count, CRP, sedimentation rate, renal and liver function tests, ANF, lupus anticoagulant, anticardiolipin antibodies, ANCA, and serology of HIV, syphilis, hepatitis B and C were normal. Urine toxicological screening was negative for cocaine, heroin and amphetamines, and strongly positive for cannabis. Unenhanced brain CT showed a right temporal lobe haemorrhage (fig. 1) without enhancement after contrast administration. No underlying acute brain infarction was seen on diffusion-weighted MRI. Transoesophageal echocardiography was normal. Angiography revealed diffuse multifocal arterial narrowing (fig. 1) in the absence of aneurysm or arteriovenous malformation. The buprenorphine dose was decreased to 2 mg o.d. and the cannabis use was reduced to 3–4 cigarettes per week. Three months later, complete disappearance of arterial abnormalities was seen on angiography.

Discussion

Drug abuse is a risk factor for both haemorrhagic and ischaemic stroke [1]. Haemorrhagic stroke is most often seen with sympathomimetic drugs (e.g. cocaine, amphetamine and ephedrine) [2, 3]. Possible aetiological mechanisms include acute hypertension, failure of cerebrovascular autoregulation, underlying aneurysm or arteriovenous malformation, coagulopathy, vasculitis and early haemorrhagic transformation of infarction. Cerebral vasoconstriction has been described in association with cocaine, amphetamines, ephedrine, phenylephrine, LSD and heroin [2, 4–8]. Drug-induced vasculitis is often difficult to distinguish from vasospasm caused by these agents. Arterial narrowing disappearing after decrease in drug dose, like in our patient, favours vasospasm over vasculitis. Brain haemorrhage and cerebral vasoconstriction have never been described in association with cannabis or buprenorphine. In our patient, both drugs were well tolerated for a long period of time. One might question if there is a causal relationship between radiological abnormalities and these drugs. It is likely that vasoconstriction was related to (at least one of) these drugs, since angiographic abnormalities disappeared after decrease in the dose of both drugs. The pathophysiological role of

Fig. 1. A Brain CT showing a right temporal lobe haemorrhage. **B** Angiography reveals diffuse multifocal segmental vasoconstriction in anterior and middle cerebral artery.

vasoconstriction in brain haemorrhage is unclear. A possible mechanism is that when vasospasm subsides and perfusion is restored, artery rupture occurs. A drug-induced shift of upper limit of cerebrovascular autoregulation towards lower blood pressure levels together with a cannabis-induced transient arterial hypertension may have played a role in the pathogenesis of brain haemorrhage in our patient. A delayed and possible synergic effect of both drugs on cerebral autoregulation can be suspected.

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Middle Cerebral Artery Dissection Gives Rise to Giant Serpentine Aneurysm

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A cervico-cephalic artery dissection is defined as hematoma within the artery wall. Spontaneous dissecting aneurysms of the middle cerebral artery (MCA) have been implicated in stroke. The giant serpentine aneurysm (GSA) is a partially thrombosed giant aneurysm (>2.5 cm) with tortuous vascular channels and separate entrance and outflow pathways [1–3]. The GSA is often situated on the MCA or its branch vessels [4]. Its pathogenesis has not been elucidated. We report a case in which a right MCA dissection was diagnosed after a stroke. Eleven years later, a second stroke occurred in the same territory, leading to the discovery of a GSA.

Case Report

In 1992, a 37-year-old woman suffered a sudden attack of weakness on the left side of the body. She was free from vascular risk factors and took no prescribed drugs. The CT scan revealed a right striatocapsular ischemia. Laboratory investigation failed to explain the stroke. The selective bilateral carotid, vertebral and renal angiograms showed only a very irregular aspect of the MCA, with multiple zones of narrowing interspersed with zones of dilatation (fig. 1a). The diagnosis of dissection of the MCA was proposed. The course of the disease was positive. Arterial hypertension was discovered during hospitalization. Antihypertensive and antithrombotic drugs were prescribed. Eleven years later, the patient presented sudden, persistent anesthesia and weakness of the left arm. No other neurological or general anomalies were observed. MRI in the frontal T₁-weighted sequence revealed a partially thrombosed aneurysm, suggestive of a GSA, on the insular segment of the MCA (fig. 1b, c).