

consumption of broad beans, plasma levodopa levels remain at higher levels for longer periods in patients than in healthy volunteers. This is probably caused by the potent long-term effect of carbidopa on peripheral decarboxylase.⁵ The increase in plasma levodopa in PD patients after broad bean consumption is accompanied by significant improvement in motor symptoms; in some cases, intense dyskinesia will disappear.⁶ The pathophysiology underlying motor fluctuations, and dyskinesias in particular, has yet to be fully explained.⁷ Studies have demonstrated the presence of peripheral and central pharmacokinetic factors as well as central pharmacodynamic factors. Central factors are considered more important, and include pulsed dopaminergic stimulation (non-physiological) of striatal receptors and loss of ability to store dopamine in the striatum.⁸ The appearance of motor fluctuations in response to L-dopa indicates disease progression in PD. In our patient, whose long history of PD indicates severely impaired nigrostriatal terminals, a diet rich in broad beans provoked dopaminergic hyperstimulation that caused severe central nervous system effects (agitation, chorea) and peripheral autonomic effects.

From a nutritional standpoint, broad beans are an excellent foodstuff and a potential means of combating the effects of PD, but doctors must inform patients about their effects. This clinical case illustrates a fact that is often overlooked: natural levodopa is present in certain foods that are frequently consumed.

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Recurrent lacunar ischaemic stroke due to resistance to antiplatelet treatment: examining the need for personalised antithrombotic therapy[☆]



Ictus lacunar recurrente por resistencia al tratamiento antiagregante plaquetario: hacia la necesidad de una terapia antitrombótica individualizada

Dear Editor:

A significant percentage of patients with stroke will experience recurrence.^{1,2} Some of these cases of recurrence may arise due to resistance to antiplatelet treatment.³ We

describe the case of a patient who suffered several recurrent strokes, a situation attributed to insufficient analysis of risk factors; however, the possibility of resistance to antiplatelet treatment was not examined.

Our patient was a 51-year-old woman who was hospitalised for dysarthria in October 2006. Her cardiovascular risk factors comprised heavy smoking habit (2.5 packs of cigarettes daily), diabetes (with diabetic retinopathy), arterial hypertension, and dyslipidaemia. There was no other relevant family or personal history. She was treated with atorvastatin 20 mg, insulin, and acetylsalicylic acid (ASA) 500 mg. At time of admission, the patient displayed mild right-sided hemiparesis and dysarthria. Cranial magnetic resonance imaging (MRI) revealed a left pontine stroke (Fig. 1A). The neurovascular study (MRI angiography and carotid duplex ultrasound) showed clinically diffuse silent carotid atheromatosis causing stenosis of 60% on the right side. Cardiology study results were normal. A blood panel revealed high triglyceride levels (190 mg/dL) and high glycaemia (265 mg/dL). The stroke was attributed to microangiopathy and the patient's drug regimen was adjusted by adding clopidogrel and increasing the statin dose to 80 mg daily.

In September 2008, the patient came to the emergency department because of right-sided hypoesthesia.

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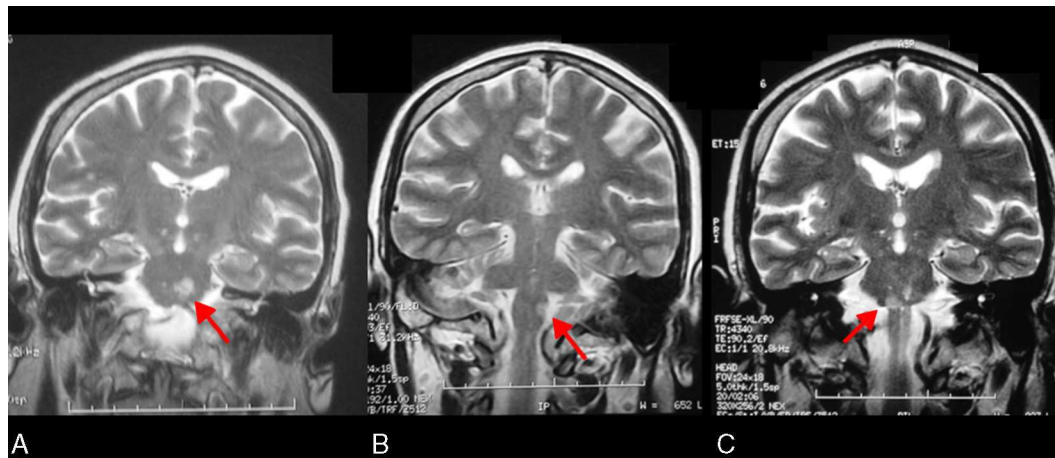


Figure 1 Cranial MRI, coronal T2-weighted sequences. (A) October 2006. Subacute left-sided lacunar pontine stroke (arrow). (B) September 2008. Subacute left-sided lacunar medullary stroke (arrow). (C) April 2011. Subacute right-sided lacunar pontine stroke (arrow).

Neurological examination revealed facio-brachio-cranial hypoesthesia preceded by mild paresis. Duplex carotid ultrasound showed bilateral carotid atheromatosis similar to that in the previous study. Transcranial duplex ultrasound yielded normal results. The blood count, including microbiology study and antiphospholipid antibodies, showed no abnormalities. Cranial MRI revealed a left medullary stroke (Fig. 1B). The patient remained on clopidogrel; low molecular-weight heparin (nadroparin calcium 0.6 mL SC daily) was added and later withdrawn after the cardiology study. The transthoracic ultrasonography in that study revealed ventricular hypertrophy and discrete left atrial enlargement. A routine CT angiography showed diffuse atheromatosis with no other alterations.

In April 2011, the patient was hospitalised for left hemiparesis. A neurological examination recorded exacerbation of initial dysarthria, left hemiparesis (3/5 in arm, 2/5 in leg), mild right hemiparesis, and bilateral Babinski sign. Cranial MRI revealed a right pontine stroke (Fig. 1C), and CT angiography indicated diffuse atheromatosis. High blood pressure and hyperglycaemia were also recorded while the patient was hospitalised. Once again, the diagnostic impression was microangiopathy plus poor control over cerebrovascular risk factors, and the patient continued to take antiplatelet drugs.

In November 2011, the patient stopped taking all drugs due to difficulty swallowing and was admitted due to poor clinical status. A neurological examination yielded similar results to previous ones, in addition to severe dysphagia. The patient could walk using supports and required assistance with most activities, including basic tasks. In light of the recurrent strokes and worsening clinical state, doctors suspected a neoplastic aetiology and performed a full work-up to rule out that suspicion. The haematology department completed a platelet study that yielded the following results: Platelet Function Analyser (PFA) collagen/epinephrine test 94 seconds (85-165), PFA collagen/ADP 86 seconds (71-118), and PFA P2Y 71 seconds (0-106). These tests did not show an antiplatelet effect occurring with either ASA or clopidogrel, even after raising the dose to 150 mg/day. The patient began treatment with cilostazol.

Earlier studies have described lacunar strokes as being the most frequently responsible for pure motor hemiparesis. Their main localisations are the internal capsule and the pons, as is the case in our patient.¹ Recurrence in lacunar stroke entails progressive motor and cognitive impairment. These patients will come to be dependent on others for activities of daily living, and this situation is costly. Today, tight control over cerebrovascular risk factors and use of antiplatelet drugs are the pillars of treating lacunar stroke. However, despite the benefits of antiplatelet drugs, many patients experience recurrent strokes.²⁻⁴ This phenomenon may be explained in part by resistance to antiplatelet treatment,⁴ defined as failure by the agent to block its target even when used at the correct dosage.^{5,6} In our hospital, platelet function is measured using the PFA collagen/epinephrine and PFA collagen/ADP tests for ASA and the PFA P2Y test for clopidogrel. In our patient, results on all tests were below the threshold for an antiplatelet effect. Higher doses of ASA (≥ 325 mg/day) and strict adherence to treatment may successfully overcome resistance to ASA in some patients.⁵ Maintaining clopidogrel at 150 mg/day is an option for patients who are poor responders.⁶ Although both approaches were used in our case, we observed no response. On the other hand, results from the SPS3 study, showing that antiplatelet bitherapy with ASA and clopidogrel was not superior to ASA in monotherapy in patients with lacunar stroke, influenced the decision to not administer this treatment to our patient.⁷

We believe that this case illustrates the importance not only of strict control over cerebrovascular risk factors, but also of administering personalised antiplatelet drugs. Measuring the response to antiplatelet drugs is an option in clinical practice that may help establish effective antithrombotic treatment, and should be carried out at least in those patients with recurrent cerebral infarcts.

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Posterior territory stroke: endovascular treatment in patients with an occluded artery of Percheron[☆]



Ictus de territorio posterior. Tratamiento endovascular en pacientes con oclusión de la arteria de Percheron

Dear Editor:

The artery of Percheron is a single thalamoperforating artery that supplies the bilateral medial thalami and also irrigates the midbrain to a greater or lesser extent.¹ Occlusion of this variant artery is infrequent.^{2,3} We present 2 cases of occlusion of the artery of Percheron that resolved with endovascular treatment.

The first case was a 53-year-old man with a history of arterial hypertension, diabetes, high cholesterol levels, and obesity. During coronary catheterisation for acute myocardial infarction, he presented a decreased level of consciousness and conjugate gaze palsy. Examination revealed drowsiness, partial comprehension of commands, acute dysarthria, bilateral eyelid ptosis (more pronounced in the left eye), left-sided mydriasis with non-reactive pupil, and upgaze palsy. He was able to move his arms and legs normally. No loss of strength or lack of coordination was observed in the limbs. Computed tomography (CT) yielded no significant findings. Cerebral angiography revealed foetal origin of the right posterior cerebral artery and occlusion of the right P1 segment. A 0.10" microguide wire was advanced as far as the right posterior cerebral artery (P2 and P3 segments) while mechanical disruption of the thrombus was being performed in the right P1 segment with a Gateway[®] balloon (Boston Scientific, Natick,

MA, USA). The balloon measured 1.5 mm × 9 mm and had been used previously for basilar artery angioplasty. The deflated balloon was passed through the occluded segment several times to achieve patency of the P1 segment and the single paramedian thalamic artery (artery of Percheron) (Fig. 1). Fibrinolytic treatment was not administered since the patient was anticoagulated with high doses of heparin for the cardiac catheterisation procedure. Since the calibre of the P1 segment was unknown, we did not inflate the balloon or use a stentriever device to avoid artery damage. Neurological examination after treatment showed ataxia and normal levels of consciousness. A follow-up brain CT yielded no significant findings. One week later, minimal bilateral eyelid ptosis, diplopia on upgaze, and ataxia persisted.

The second patient was an 81-year-old man with Meniere disease and convergent strabismus. Six hours before he was admitted, the patient presented dizziness similar to that occurring in Meniere disease, with drowsiness, lack of verbal response, and poor motor coordination of the right limbs. The patient was comatose and presented complex ophthalmoplegia with miosis at time of admission. Pain stimuli elicited flexion of the left limbs, whereas the right limbs showed no response. The patient displayed an extensor response to right-sided plantar stimulation (Babinski sign). Cranial CT yielded normal results; intravenous thrombolysis was not performed since first symptoms appeared 6 hours before admission. Arteriography revealed foetal origin of the left posterior cerebral artery with left hypoplastic P1 segment which presented a filling defect. A Mirage[®] microguide wire measuring 0.08" (ev3, Irvine, CA, USA) was passed through the P1 segment to break up the clot, but catheterisation was not possible. Consequently, 500 000 IU urokinase was administered at a rate of 10 000 IU/min at the origin of the P1 segment, resulting in patency of the P1 segment and of the artery of Percheron, which was detected after fibrinolysis treatment (Fig. 2). After the procedure, neurological examination (excluding gait) revealed a previously observed tendency of the eyes to converge, involuntary eye closure, and hypoacusia. Three days later, results from the neurological examination were normal. Brain MRI scans at 72 hours yielded normal results.

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