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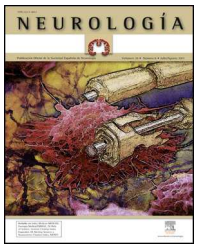

LETTERS TO THE EDITOR

Broad bean (*Vicia faba*) consumption and Parkinson's disease: a natural source of L-dopa to consider☆

Consumo de habas (*Vicia faba*) y enfermedad de Parkinson: una fuente natural de L-dopa a tener en cuenta

Dear Editor:

Scientists know that broad beans (*Vicia faba*) contain enough L-dopa to be pharmacologically active on patients with Parkinson's disease (PD). It has been shown that these beans increase plasma levels of L-dopa and improve motor function.[1] We present the case of a patient with PD who suffered very intense on-time dyskinesias linked to the consumption of this legume.

This 73-year-old man had a 10-year history of PD, now in stage III on the Hoehn and Yahr scale, and well-controlled arterial hypertension and diabetes mellitus. His cognitive condition was good and he was active and independent; in fact, he tended the legume and vegetable crops in his garden every day.

He was being treated with L-dopa plus carbidopa dosed at 800 mg/day and a dopaminergic agonist, cabergoline, dosed at 3 mg/day. Doses had not been adjusted in the past year. According to his family and his medical history, the patient experienced rigidity and slowness on rising in the morning and noticed only mild choreic dyskinesias toward late morning, before lunchtime (on-time dyskinesias). These events did not cause discomfort as he was used to them and experienced no other motor fluctuations at the time. He had never experienced hallucinations or psychiatric disorders, and slept well at night with no need for benzodiazepines or other sleeping aids.

Family members brought him to the emergency department some 2 hours after lunch when he suddenly displayed involuntary movements, motor agitation, nausea, profuse sweating, and aggressiveness. Examination revealed choreic movements (including ballistic movements) of the limbs, trunk, and neck, accompanied by cervical dystonia, profound sweating, tachycardia, anxiety, nausea without vomiting, and some level of verbal aggressiveness. Arterial blood pressure was 90/55 mmHg. Routine laboratory analyses, electrocardiogram, and chest radiography did not reveal any relevant results. The family did not report the patient's taking any new drugs or having more than the normal dose of his usual medication. Doctors decided to provide fluid therapy, sedation with diazepam dosed at 20 mg, and temporary withdrawal of the antiparkinsonian treatment. Since nothing appeared to have triggered the patient's syndrome, doctors asked the patient and his family about his eating habits. They responded that he had eaten broad beans (*Vicia faba*). They had harvested the first broad beans of the season that morning, and the patient reported that they were exceptionally tender and juicy that year due to abundant rainfall. Family members stated that the patient had eaten many raw unshelled beans before consuming a generous helping of cooked beans, for a total amount between 300 and 400 g. After 24 hours, once the patient had improved and displayed only mild choreic dyskinesia and no peripheral autonomic symptoms, his normal drug treatment was resumed. He had returned to his baseline state by 48 hours and was discharged the following day. Doctors gave the patient some dietary recommendations since he did not know that broad beans had this effect; he stated that he had not eaten them in 2 years, and they had never been 'problematic'.

Early sacred texts in Indian literature (second millennium BCE) refer to 'trembling' individuals who were prescribed a plant from the Fabaceae family to treat the condition. In 1913, Marcus Guggenheim was the first to isolate L-dopa (L-3,4-dihydroxyphenylalanine) from *Vicia faba* plants.[2] The broad bean (*Vicia faba*) is a typical member of, and lends its name to, the Fabaceae family; it is a herbaceous climbing plant with an upright stalk that is cultivated for its edible seeds. Broad beans are cultivated around the world, but they originated in the Mediterranean basin or Central Asia. The entire plant, including the leaves, stalks, pods, and beans, is a source of L-dopa. The amount of levodopa may vary considerably, depending on the species, climate zone, soil conditions, precipitation, and other factors. Scientists know that the pods and young beans contain more L-dopa than mature beans. Approximately 100 g of fresh or green broad beans may contain 50 to 100 mg of L-dopa.[3]

The literature describes anecdotal cases of patients with PD who showed substantial improvement after consumption of broad beans.[4] Studies carried out in healthy subjects and in patients with PD show that after controlled

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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.

References

1. Raguthu L, Varanese S, Flancbaum L, Tayler E, di Rocco A. Fava beans and Parkinson's disease: useful 'natural supplement' or useless risk? Eur J Neurol. 2009;16:e171.
2. Guggenheim M. Dioxyphenylalanina, a new amino acid from *Vicia faba*. Z Phys Chem. 1913;88:276.
3. Vered Y, Rabey JM, Palevitch D, Grosskopf I, Harsat A, Yanowski A, et al. Bioavailability of levodopa after consumption of *Vicia faba* seedlings by Parkinsonian patients and control subjects. Clin Neuropharmacol. 1994;17:138—46.
4. Apaydin H, Ertan S, Ozekmekçi S. Broad bean (*Vicia faba*) — a natural source of L-dopa — prolongs «on» periods in patients with Parkinson's disease who have «on-off» fluctuations. Mov Disord. 2000;15:164—6.
5. Kempster PA, Bogetic Z, Secombei JW, Martin HD, Balazs NDH, Wahlqvist ML. Motor effects of broad beans (*Vicia faba*) in Parkinson's disease: single dose studies. Asia Pac J Clin Nutr. 1993;2:85—9.
6. Rabey JM, Vered Y, Shabtai H, Graff E, Korczyn AD. Improvement of parkinsonian features correlate with high plasma levodopa values after broad bean (*Vicia faba*) consumption. J Neurol Neurosurg Psychiatry. 1992;55:725—7.
7. Nutt JG. Long-term L-dopa therapy: challenges to our understanding and for the care of people with Parkinson's disease. Exp Neurol. 2003;184:9—13.
8. Clavero-Ibarra P, Gil-Alzueta MC. Abordaje práctico de las discinesias en la enfermedad de Parkinson. Rev Neurol. 2012;54 Suppl. 5:S33—40.

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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 hypoaesthesia.

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