

Transient dystonia following magnetic resonance imaging in a patient with deep brain stimulation electrodes for the treatment of Parkinson disease

Case report

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✓ Data from previous studies have shown that magnetic resonance (MR) imaging of the head can be performed safely in patients with deep brain stimulators. The authors report on a 73-year-old patient with bilaterally implanted deep brain electrodes for the treatment of Parkinson disease, who exhibited dystonic and partially ballistic movements of the left leg immediately after an MR imaging session. Such dystonic or ballistic movements had not been previously observed in this patient. In the following months, this focal movement disorder resolved completely. This case demonstrates the possible risks of MR imaging in patients with deep brain stimulators.

KEY WORDS • Parkinson disease • deep brain stimulation • magnetic resonance imaging

EVEN though relevant health problems associated with MR imaging studies performed in patients with deep brain stimulators had not been reported, such studies initially were not performed in these patients because of safety considerations. Then, data from in vitro and in vivo studies showed that MR imaging could be performed safely in patients in whom deep brain stimulators had been implanted.^{5,10-12} Subsequently, the use of MR imaging was established in patients with deep brain stimulators for the postoperative control of electrode location^{2,10} and for functional MR imaging studies.^{7,11} In this report we describe the first adverse effect of MR imaging, which consisted of a transient focal dystonia and ballism in a patient in whom bilateral subthalamic electrodes were implanted for the treatment of Parkinson disease.

Case Report

Implantation of DBS Electrodes. In this 73-year-old woman with refractory Parkinson disease of the tremor-dominant type, quadripolar DBS electrodes (length 51 cm, model #3383; Medtronic, Inc., Minneapolis, MN) were implanted into the left STN on September 6, 2001, and into the right STN 5 days later. The electrodes and their contacts

consisted of platinum-iridium, and the proximal connector to the stimulator (Kinetra, model no. 7428; Medtronic) was made of a nickel alloy. Following electrode implantation, the patient was placed on a regimen of 3 × 50 mg levodopa. As usual, the implantation of the stimulators was planned for a later time (September 17 in this patient); until then, external stimulation was performed. The postoperative results were good: when the external stimulators for both sides were turned on (the on state), tremor disappeared from the right side and decreased substantially on the left side. A slight dysarthria was the only side effect. These stimulation effects ended immediately when both stimulators were turned off (the off state). Hyperkinesias were not noticed during the on state or the off state.

Magnetic Resonance Imaging Studies and Complications. On September 14, an MR imaging study of the head was conducted using a standard transmit/receive CP head coil (1-tesla unit, Expert; Siemens, Erlangen, Germany). Four conventional spin echo sequences were performed, two in the sagittal and two in the coronal orientation. Imaging parameters were as follows: sagittal TR 570, TE 15, matrix 192 × 256, FOV 230 mm, 11 slices, thickness 3 mm, acquisition time 3:01 min; coronal TR 570, TE 15, matrix 192 × 256, FOV 200 mm, 11 slices, thickness 3 mm, acquisition time 2:41 min. The external stimulators were removed before beginning the MR imaging examination. During MR imaging, both leads were fixed outside the coil in a straightened manner. Inspection revealed no evidence of defective insulation of the leads. Results of MR imaging were normal

Abbreviations used in this paper: DBS = deep brain stimulation; FOV = field of view; MR = magnetic resonance; STN = subthalamic nucleus.

and demonstrated the correct positioning of both DBS electrodes (Fig. 1). The patient noticed no dysesthesias or other symptoms during MR imaging.

Immediately after leaving the MR imaging device, however, the patient exhibited dystonic and single ballistic movements of the left leg, which had never been observed before. The main movement consisted of continuous dystonic dorsal extensions of the left foot combined with an outward rotation. To a smaller extent, intermittent dystonic extensions occurred in the left knee joint. In addition, sudden ballistic leg movements with an abduction of the whole left leg could be observed. All these movements increased when the stimulator for the left side of the body was turned on with the same stimulus parameters used before MR imaging. Therefore, the stimulator for the left side remained turned off at first. On a computerized tomography scan obtained on the same day, there was no evidence of a larger focal brain lesion; however, metal artifacts on the scan impaired our ability to judge the brain tissue surrounding the electrode tips. The antiparkinsonian medication regimen of 3×50 mg levodopa was not changed.

Implantation of Stimulators and Postprocedure Course. On September 17, the stimulators for both electrodes were implanted into the left infraclavicular fossa. In the following weeks, the dystonic and ballistic movements diminished continuously. In December 2001, the stimulator for the left side was reactivated at a low stimulus intensity (amplitude 1.5 V, impulse duration 120 μ sec, frequency 160 Hz) without a recurrence of the movement disorders. During stimulation at higher intensities, however, the dystonic and ballistic movements of the patient's left leg returned. In February 2002, the stimulator for the left side was programmed to a higher stimulus intensity (amplitude 3.5 V, impulse duration 120 μ sec, frequency 160 Hz), a setting at which tremor completely disappeared. Despite the higher stimulus intensity, dystonic or ballistic movements did not recur any other time.

Discussion

The dystonic and partly ballistic movements of the patient's left leg occurred immediately after MR imaging; there had been no dystonic or ballistic movement any time before the imaging study. This temporal correlation between the MR imaging study and the first occurrence of hyperkinesias strongly indicates the presence of a lesion in the right STN induced by MR imaging: ballistic hyperkinesias occur immediately after lesioning of the STN, as observed in patients with therapeutic^{1,6,9} and other kinds of acute STN lesions.⁴ Furthermore, such lesion-induced ballistic hyperkinesias reveal a clinical course similar to that in our patient.^{1,4,6,9} Note that our patient initially exhibited hyperkinesias 3 days after electrode implantation; therefore, a minisubthalamotomy effect following electrode implantation is unlikely as a possible explanation for the hyperkinesias, given that such effects occur immediately after, and decrease usually within the first 2 days of, electrode implantation.⁸ The stimulation parameters and the medication administered before and after MR imaging were identical in our patient. For this reason, the hyperkinesias cannot be based on a change in stimulus parameters or antiparkinsonian medication. In consideration of these points, we assume

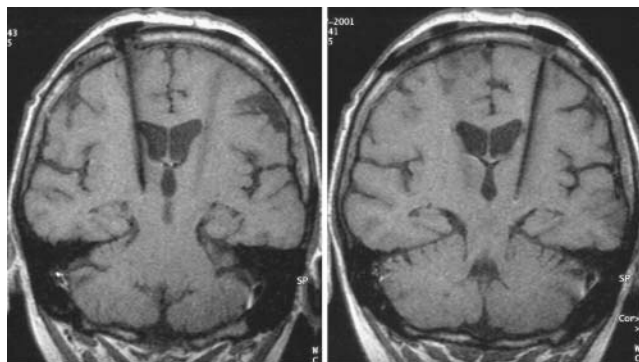


FIG. 1. Coronal MR images obtained after implantation of subthalamic electrodes, demonstrating their correct positioning on the right (left) and left (right) sides. There was no structural lesion after electrode implantation. Because of the electrode artifacts, the tracks of the probes appear to be broader than they were in reality.

the presence of an acute lesion of the right STN that was caused by MR imaging. Unfortunately, the patient did not consent to any further MR imaging studies.

We can only speculate on the reason for this side effect of MR imaging, which has not been previously reported in a patient with deep brain stimulators. A current in the implanted leads that causes heating and consecutive thermal tissue damage can be based on two mechanisms. First, the MR imaging device generates magnetic fields that induce a current in a coiled wire located inside or near the emitting coil, according to the Faraday law. This is unlikely in our case, however, because the wires were not coiled. Second, a straightened wire in the coil acts as an antenna that is sensitive to the oscillating electric field.^{3,11} One could assert that this latter effect happened in our patient because the straightened wires were partially located within the coil; therefore, a current could be induced and cause a small brain lesion. In view of the bilateral subthalamic implants, it is unclear why this effect manifested in only one side in our patient.

Conclusions

Our case illustrates that MR imaging in patients with implanted deep brain stimulators may pose a potential risk, despite data from several *in vitro*^{5,12} and *in vivo*^{2,7,10-12} MR imaging studies, which were performed with no side effects in patients. A transcranial Doppler ultrasonography study of the basal ganglia may be a safer alternative for checking DBS electrode locations.

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