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Hemihypomimia in Parkinson's Disease

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A 55-year-old woman with a progressive gait disorder and difficulties in executing skilled movements with her right hand for 12 months was admitted for further evaluation. The patient had no previous history of stroke, Bell's palsy or other neurological diseases.

On admission, the neurological examination revealed a cogwheel rigidity and bradykinesia of the right upper and lower extremities. Reduced and slowed movements of the right-sided facial muscles were also observed, but only of the lower face (fig. 1). Both hypokinesia and bradykinesia were most pronounced when the patient spoke. Voluntary facial movements were less affected when she followed instructions; this was also the case for emotional movements (e.g., reflex smiling).

While the cranial MRI was normal, DaTSCAN-SPECT showed an asymmetry of the presynaptic dopamine transporter in the striatal region; there was a significantly lower intensity on the left side. The patient was diagnosed to have Parkinson's disease (PD) and was initially treated with levodopa (Madopar, 625 mg daily) and a dopamine agonist (Cabergoline, 0.5 mg per day). During this treatment, the hypokinesia and bradykinesia of the right upper and lower extremities and the right-sided hypomimia improved significantly.

A lateralization of motor signs in PD has only been reported to occur as an asymmetric hypo- and bradykinesia of the limbs, in particular as a typical feature of the early stages of PD [1, 2]. In our patient, the unilateral hypo- and bradykinesia also manifested in the lower face on the same side.

Parkinsonian bradykinesia is characterized by two main features: (1) patients underscale muscle force and (2) the deficit is often ameliorated when external cues are given to guide the movements. Generally, the basal ganglia motor output has access to the medial rather than the lateral motor cortical areas. Metabolic studies in patients with PD showed that there is underactivity of midline cortical motor areas (supplementary motor cortex), which is sometimes accompanied by an increase in activation of lateral premotor areas. This increased activation might be an active process of compensation and related to the improvement in performance that can be observed when external cues are given to guide movements [3, 4].

Medial cortical areas are more active in association with internally generated movements, whereas lateral areas are more active during externally cued movements. Underscaling of muscle force seems to be a particular problem in internally generated movements (e.g., facial expression while speaking) [5]. The underscaling of muscle force seems less of a problem when movements are externally cued. This was obvious in our patient whose voluntary facial movements were less affected when following instructions, whereas the hypo- and bradykinesia were best observed when the patient spoke. The sparing of the upper face might be explained by the fact that it is innervated bilaterally from the primary motor cortex [6].

Fig. 1. The 55-year-old patient, who was diagnosed to have PD, exhibits hemihypomimia of the right-sided facial muscles when speaking.

In conclusion, this is an exceptional case of right-sided hemiparkinsonism with an obvious unilateral brady- and hypokinesia of the face on the same side, i.e., hemihypomimia. Since this phenomenon has not yet been described in detail in the literature, the subtle lateralization of hypomimia may remain undetected in patients with PD – in particular, as a sign in the early stages of unilateral PD. It should, however, be considered in patients with PD.

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Reversible Visual Deficit and Corpus callosum Lesions due to Metronidazole Toxicity

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Introduction

Metronidazole is a 5-nitroimidazole antibiotic with neurological side-effects including neuropathy, seizures, encephalopathy and cerebellar dysfunction. Central nervous system (CNS) toxicity is rare. Magnetic resonance imaging (MRI) has shown variable supratentorial white matter lesions, including the corpus callosum and cerebellar grey matter lesions mainly involving the dentate nuclei. We report marked but reversible visual deficit, cerebellar and neuropathy symptoms after years of metronidazole treatment at normal doses.

Case Report

A 20-year-old man with ulcerative colitis complained of decreased visual acuity for 2 months. He had painful distal paraesthesia for 1 year. During the last few months, he had developed dysarthria and impaired coordination with both hands. He had been on metronidazole 1,500 mg daily for 2 years. The ophthalmologist noted a vision of 2/10 bilaterally and major disturbance of red-green colour discrimination. Pattern visual evoked potentials (VEPs) were absent (fig. 1), but low-amplitude flash VEPs were elicited with markedly prolonged latencies. MRI of the brain and optic nerves showed non-enhancing increased signal intensities on sagittal T_2-weighted images in the splenium and less conspicuously in the truncus and genu of the corpus callosum (fig. 2A) and normal optic nerves. Metronidazole was discontinued.

Two weeks later, when examined in the neurology department, vision had recovered to 8/10. Neither nystagmus nor other gaze abnormalities were observed. Limb coordination was slightly impaired. Sensory testing revealed dysaesthesia and allodynia at all extremities and astereognosia on both hands. Tendon reflexes were weak or absent.

Cerebrospinal fluid was normal, with no abnormalities on immunoelectrophoresis. Pattern VEPs could be elicited with low amplitudes and markedly increased P_1 latencies (fig. 1). Brainstem and somatosensory evoked potentials were normal. Electromyography revealed a purely sensory distal symmetric axonal neuropathy.

Repeat brain MRI scan after 2 and 8 months showed gradual though incomplete resolution of the increased signal in the corpus callosum (fig. 2B, C). The painful paraesthesia disappeared within 3 months after cessation of metronidazole. After 14 months, all other symptoms and signs had resolved and pattern VEP latencies had normalised (fig. 1). No visual or CNS symptoms or signs have recurred in a 3-year follow-up period.

Discussion

The incidence of metronidazole-induced CNS toxicity is low as compared with neuropathy, which is usually a distal symmetric sensory neuropathy with mainly small-fibre involvement and subclinical large-fibre involvement. Rare cases with metronidazole-related CNS toxicity have shown involvement of the corpus callosum. In 1995, Ahmed et al. [1] reported a patient with nausea, vomiting, confusion, ataxia, neuropathy and MRI lesions that symmetrically involved the supratentorial white matter, the corpus callosum and deep cerebellar grey nuclei. As in our patient, the lesions regressed after stopping metronidazole. The few other cases confirm ataxia and dysarthria as common symptoms and illustrate that the supratentorial white matter and cerebellar dentate nuclei are often involved [2–5].

Our patient presented with focal corpus callosum lesions mainly affecting the splenium. Multiple sclerosis is by far the most common disease with corpus callosum hyperintensities on T_2-weighted MRI. Transient T_2-weighted MRI hyperintensities in the splenium of the corpus callosum have rarely been reported in epilepsy patients and have been ascribed to vasogenic oedema due to antiepileptic drug toxicity possibly facilitated by vitamin deficiency [6]. The absence of oligoclonal banding in the cerebrospinal fluid, the reversibility of the clinical signs and symptoms, the reversible non-contrast enhancing MRI lesions and the absence of new neurologi-