**Letter to the Editor**
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# Striatum Involvement in LGI1 Limbic Encephalitis

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## TO THE EDITOR

Kim *et al.*[1] recently reported a 37-year-old male patient with LGI1-related limbic encephalitis (LE) who presented with recurrent episodes of selective amnesia, seizure-like activity, confusion, and personality change. The patient showed a significant improvement after steroid therapy. Interestingly, the patient also presented intermittent sensory symptoms described as "flow of an electric current" and "seizure-like shivering". Sensory symptoms have been extensively described in patients with LGI1-antibody encephalitis.[2,3] These patients experience multifocal seizure localisations with multiple semiologies, in addition to the characteristic faciobrachial dystonic seizures (FBDS) and some of them may remain subclinical.[2] In a recent paper by Aurangzeb *et al.*,[2] sensory semiologies were reported to be as common as FBDS, and were most frequently thermal or shivering sensations.

Five years ago, I reported a 30-year-old patient with right FBDS associated with serum positivity of LGI1 antibodies and gadolinium-enhancing lesion involving the left caudate and globus pallidus.[4] After about one year, this patient relapsed during low-dose tapering regime of prednisolone and the relapse did not consist in FBDS, but was characterized by brief and frequent episodes (approximately 40 per day) of tingling and numbness involving the right arm and face. These sensory seizures showed a complete response, after increasing the dosage of prednisolone. Voltage gated potassium channel (VGKC)-complex/LGI1-antibodies test resulted positive at the time of relapse and ictal and interictal electroencephalograms (EEGs) were still normal (Plantone, 2016, unpublished data).

There are two points that should be highlighted. The first one regards the characterization of these sensory episodes. It is still controversial whether these episodes can be classified as "sensory epileptic auras" not followed by FBDS or as "pure sensory seizures". In fact, sensory auras have been reported to precede some motor events during the course of the disease. EEG changes are not frequently recorded during these sensory seizures. One can speculate that the sensory symptoms experienced by LGI-1 LE patients could be related to the involvement of the striatum. There are very interesting evidences in literature supporting striatal epileptogenicity,[5] and the absence of EEG changes could be an indirect evidence of the so called "subcortical epileptic seizures" in LGI1 LE patients. Strictly linked to the first, the second point of this discussion regards the involvement of striatum in sensory responses. For a long time, striatum has been regarded as a virtually pure motor structure, even if there were old studies focusing on sensory properties of striatal neurons.[6] Substantial evidence indicates that dorsolateral striatal neurons respond to sensory stimulation; however, the origin of these sensory inputs is still matter of debate.[7] Some authors support the hypothesis that these inputs come almost entirely from sensorimotor cortex,[8,9] whereas others suggest that other brain regions play a significant role[10-13] and they focus particularly on several thalamic nuclei involved in processing sensory information, projecting to the dorsal striatum. Primarily the centromedian-parafascicular complex and potentially also posteromedial nucleus may have both a significant influence on striatal sensory responsiveness.[10] One can argue that the involvement of the striatum can be demonstrated with clinical magnetic resonance imaging in a significant percent-

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age of the patients with LGI1 LE[14]; however, the transient nature of this signal alteration and the lack of advanced imaging sequences in routine clinical practice may underestimate the involvement of striatum in LGI1 LE pathogenesis.

In conclusion, the debate on the clinical, radiological and neuropsychological features of LGI1 LE is helping to better characterize the pathogenesis of the disease, and will probably further clarify the role of the brain the structures involved.

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