Neuroimaging Highlight

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Contralesional Spasticity of Conjugate Gaze in Striatocapsular Infarction

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A 44-year-old man developed sudden non-fluent aphasia and right hemiplegia due to left striatocapsular infarction (Figure). Neurologic examination revealed gaze deviation to the right with eyes closed, but not with eyes open (Video). There was no spontaneous or gaze-evoked nystagmus, even after elimination of visual fixation. Leftward pursuit was impaired in a craniotopic frame of reference, and horizontal saccades were hypometric in both directions. Head impulse test was normal in the horizontal plane and there were no visual field defects. The contralesional gaze deviation with eye closure persisted for ten days.

Gaze deviation with eye closure, but not with eyes open has been reported primarily in patients with lesions involving the medulla and cerebellum (ocular lateropulsion),[1,2] but our patient had no infratentorial lesions. A similar phenomenon has been also described for supratentorial lesions as the name of perverted Bell's phenomenon or spasticity of conjugate gaze (SCG), which is characterized by gaze deviation predominantly to the contralesional side with forced or attempted eye closure;[3-6] therefore, the ocular motor symptom observed in our patient is compatible with SCG. SCG has been reported primarily in

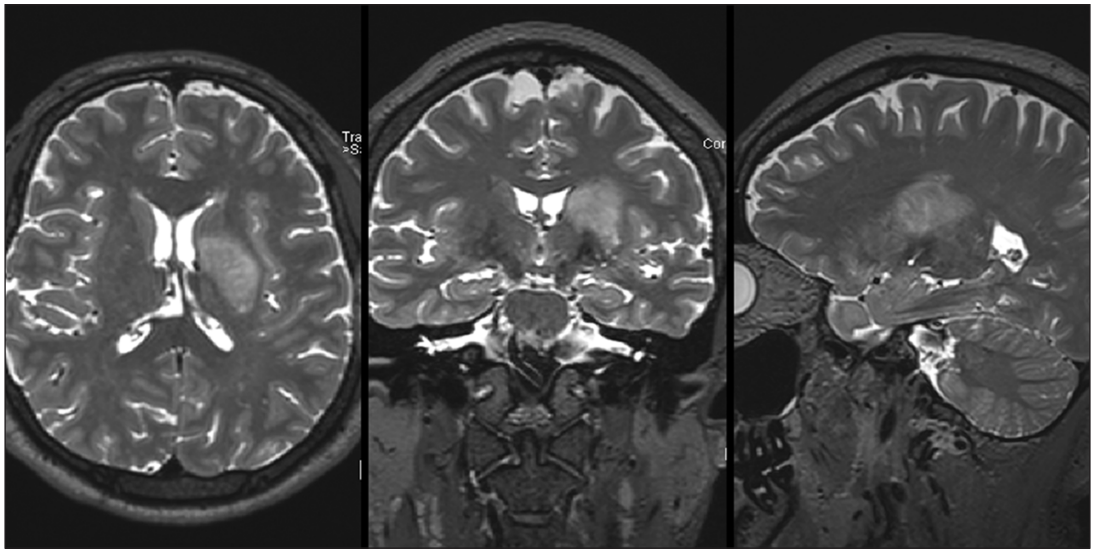

Figure: *T2-wighted MRIs show a lesion involving the left putamen, globus pallidus, and internal capsule. The head of the caudate nucleus is spared. There are no identifiable lesions in the cortex or infratentorial areas.*

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parietotemporal lesions,[4,5] but, to the best of our knowledge, has never been reported in isolated striatocapsular infarction.

Although the mechanism has not yet been established, SCG in our patient could be explained by tone imbalance of eye movements including saccades, pursuit, and the vestibulo-ocular reflex.[7] Although our patient had impaired smooth pursuit to the left which could potentially cause rightward gaze deviation and pursuit paretic nystagmus, there was no spontaneous nystagmus with visual fixation.[7] Since the head impulse test was normal and spontaneous nystagmus was not present after elimination of visual fixation, vestibular imbalance may not have been the cause of gaze deviation in this case, either.[7] Therefore, imbalance of saccadic tone might have resulted in SCG in our patient. It has been suggested that the basal ganglia control saccadic eye movements.[8] The cortical saccadic commands arrive at the head and body of the caudate nucleus. About half of the caudate nucleus neurons directly project to the substantia nigra pars reticularis, which consequently activates the ipsilateral superior colliculus and initiates contralateral saccades (direct pathway).[8] The other half of the caudate neurons project to the substantia pars reticularis via the external segment of the globus pallidus and suppress the superior colliculus and contralateral saccades (indirect pathway).[8] Therefore, in the present case, more damage to the indirect inhibiting pathway and resultant disinhibition of the superior colliculus may have caused contralesional SCG.

Our patient showed bidirectional hypometria during visually-medicated saccadic examination. This finding could be different from ocular lateropulsion, which shows asymmetric horizontal saccades. Although bidirectional hypometria in SCG cannot be simply explained, a previous study has suggested that the saccadic neurons in the caudate nucleus works differently according to the saccadic paradigm.[9] Stimulation of the caudate nucleus neurons resulted in spontaneous contralateral saccades, but contralateral saccades were suppressed during the purposive saccadic paradigm.[9] Therefore, the saccadic command circuit in the basal ganglia may be highly complex, and lesions in the basal ganglia may result in various saccadic abnormalities including SCG and bidirectional hypometria.

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The patient (C-H Kyung) in the present study agreed to the use of his clinical information, medical records, and video data for publication for the purpose of academic research and teaching. The patient consent form was provided as supplementary data.

Disclosure

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Author's contribution

Jin-Woo Park acquired the data and drafted the manuscript; Jeong-Yoon Choi acquired the data, analyzed the data, and drafted the manuscript; Jin-Man Jung acquired the data and made critical revisions to the manuscript; Do-Young Kwon acquired the data and made critical revisions to the manuscript; Moon-Ho Park acquired the data and made critical revisions to the manuscript; Ji-Soo Kim analyzed the data and made critical revisions to the manuscript.

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