Acute peripheral vestibulopathy in a cocaine addict

Cracking the vestibular nucleus

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A previously healthy 58-year-old man developed vertigo, imbalance, nausea, and vomiting shortly after smoking cocaine. He denied hearing loss, double vision, limb weakness, or sensory loss. Examination showed left-beating horizontal nystagmus with a torsional component, skew deviation, and positive right-sided head-impulse test. His urine tested positive for cocaine. Autoimmune workup, lipid panel, and echocardiography were unremarkable. MRI revealed acute right medial vestibular nucleus (VN) infarction (figure, A and B) and subcortical T2 hyperintensities (figure, C). Cerebral angiography was unremarkable. Presenting symptoms responded to meclizine and ondansetron, resolving by the third day.

Our patient presented with signs and symptoms consistent with acute peripheral vestibulopathy (APV), albeit for the skew deviation, in the context of acute right VN infarction. This unusual occurrence may be related to the VN's role in relaying and central processing of peripheral vestibular signals.¹ The exact mechanism of cocaine-induced ischemic stroke

Figure Isolated vestibular nucleus infarction after cocaine consumption

Brain MRI shows an acute infarct (arrows) that selectively involved the right vestibular nucleus at the pontomedullary junction level on diffusion-weighted imaging (A) and apparent diffusion coefficient mapping (B). Fluid-attenuated inversion recovery image depicting periventricular and subcortical white matter hyperintensities (C).

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remains unclear; however, likely mechanisms include cerebral vasospasm, enhanced platelet aggregation, and cardioembolism. In our patient, we attribute the acute infarction to transient vasospasm of the penetrating branch of the posterior inferior cerebellar artery supplying the VN. Isolated VN infarction mimicking APV has been reported in a few patients with ischemic stroke due to atherosclerosis²; however, this association has not been described following cocaine use. The presence of normal head-impulse testing, direction-changing nystagmus on eccentric gaze, and skew deviation (HINTS: head impulse, nystagmus, test-of-skew) suggests a brainstem stroke.² Of the 3, positive skew deviation has the strongest predictive value and can help identify dangerous brainstem strokes mimicking APV.³

REFERENCES

1. Kim HA, Lee H. Isolated vestibular nucleus infarction mimicking acute peripheral vestibulopathy. *Stroke* 2010;41:1558–1560.
2. Kim HJ, Lee SH, Park JH, Choi JY, Kim JS. Isolated vestibular nuclear infarction: report of two cases and review of the literature. *J Neurol* 2014;261:121–129.
3. Kattah JC, Talkad AV, Wang DZ, Hsieh YH, Newman-Toker DE. HINTS to diagnose stroke in the acute vestibular syndrome: three-step bedside oculomotor examination more sensitive than early MRI diffusion-weighted imaging. *Stroke* 2009;40:3504–3510.

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