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Reduced ^{123}I Ioflupane Binding in Bilateral Diabetic Chorea

Findings With ^{18}F FDG PET, ^{99m}Tc ECD SPECT, and ^{123}I MIBG Scintigraphy

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Abstract: We report a 64-year-old man with diabetic chorea whom we investigated with dopamine transporter SPECT, ^{18}F FDG PET, ^{99m}Tc ethylcysteinate dimer (ECD) SPECT, and ^{123}I metaiodobenzylguanidine (MIBG) scintigraphy. Dopamine transporter SPECT revealed reduced ^{123}I ioflupane binding in the bilateral striatum. ^{18}F FDG PET showed metabolic dysfunction in the bilateral striatum, as shown in earlier studies. ^{99m}Tc ECD SPECT revealed reduced brain perfusion in the bilateral caudate nucleus and putamen. ^{123}I MIBG scintigraphy revealed no cardiac sympathetic nerve dysfunction. Our case suggests a possible nigrostriatal presynaptic dopaminergic involvement in diabetic chorea.

Key Words: diabetic chorea, ^{123}I ioflupane, dopamine transporter SPECT, ^{18}F FDG PET, ^{123}I MIBG scintigraphy

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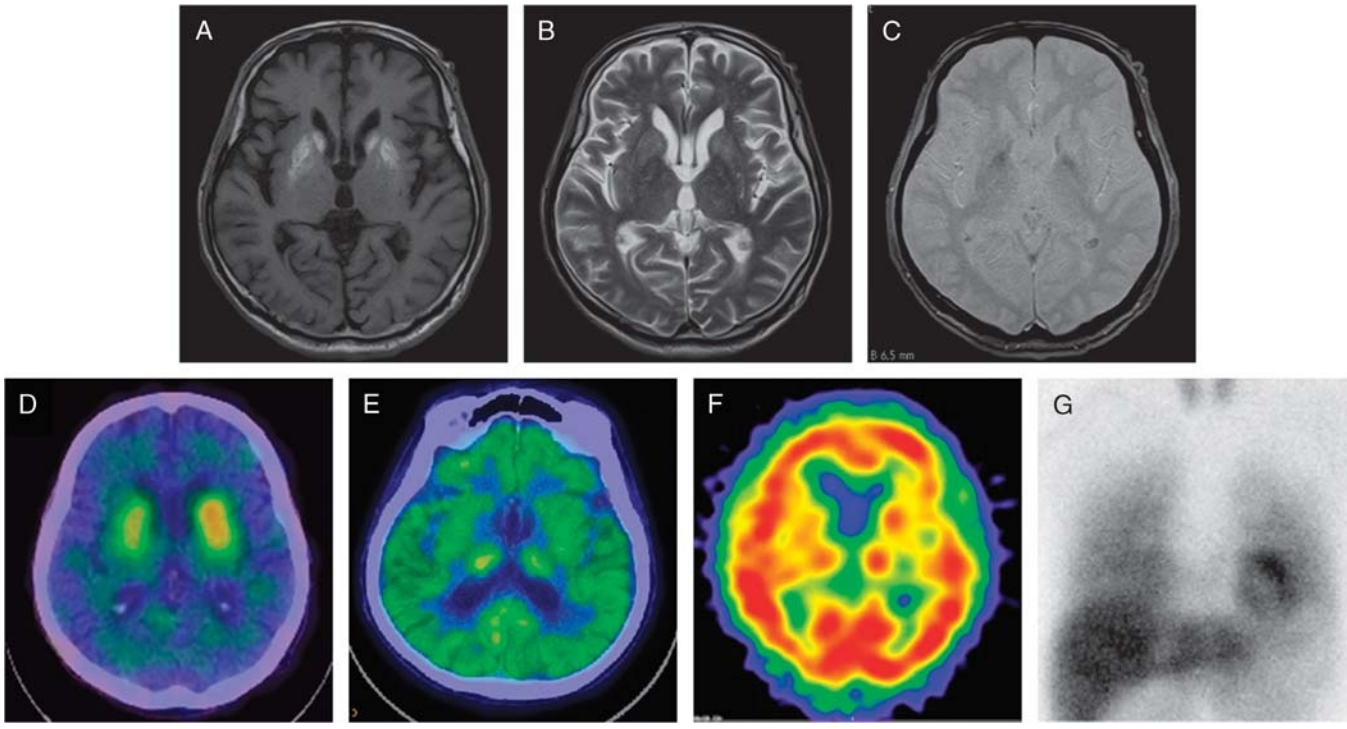

FIGURE 1. We present a 64-year-old man with subacute left-side dominant bilateral choreic movement (diabetic chorea) evaluated using a combination of dopamine transporter SPECT, ^{18}F FDG PET, ^{99m}Tc ethylcysteinate dimer (^{99m}Tc ECD) SPECT, and ^{123}I metaiodobenzylguanidine (MIBG) scintigraphy. We diagnosed him with diabetic chorea because of poorly controlled diabetes mellitus (HbA1c 13%–18%) and brain MRI showing pathognomonic hyperintensity on T1-weighted imaging in the bilateral caudate nucleus and putamen and right-side external segment of the globus pallidus (**A**). No significant abnormality on T2- (**B**) and T2*-weighted images (**C**) was found in the corresponding area. No significant old infarction or other differential diagnoses for chorea were identified.[1] Dopamine transporter SPECT showed bilaterally and right-side dominant reduced ^{123}I ioflupane binding in the caudate nucleus and putamen (**D**). The specific binding ratio[2] was 1.22 on the right and 1.36 on the left side (ELEGP was used as a collimator, and no scatter correction or attenuation correction was applied). The asymmetry dopamine transporter SPECT in diabetic chorea index (AI) of ioflupane binding was 10.7%, revealing a decline predominantly on the right side. This was consistent with the contralateral dominance of the chorea. No confounding medication for the evaluation with dopamine transporter SPECT was identified.[3] Brain ^{18}F FDG PET revealed decreased glucose metabolism (**E**) in the corresponding area, similar to the dopamine transporter SPECT, suggesting regional dysfunction in the bilateral striatum. There was no significant left-to-right difference in striatal ^{18}F FDG PET uptake, with an AI of 2.7%. ^{99m}Tc ECD SPECT revealed reduced brain perfusion in the bilateral caudate nucleus, putamen, and thalamus (**F**). ^{123}I MIBG scintigraphy showed no reduction in the H/M ratio, with 4.95 in the early phase and 5.03 in the late phase (**G**, late phase). We assumed that a low accumulation in the mediastinum caused this high H/M ratio. Treatment with dopamine antagonist and blood glucose control relieved most choreic symptoms within 4 weeks, and we are now successfully reducing his dopamine antagonist dose. Reduced glucose metabolism was found in the bilateral putamen and caudate nucleus in the ^{18}F FDG PET in our patient, suggesting regional striatal dysfunction. This was consistent with earlier studies on diabetic chorea.[4–7] Several theories regarding the underlying pathology of diabetic chorea have been proposed, including petechial hemorrhage,[7] metabolic abnormality because of hyperglycemia,[5,7] patchy striatal necrosis associated with vasculopathy,[6] and transient ischemia.[7] No conclusion has been reached to date. The only earlier report using dopamine transporter SPECT with hyperglycemic hemichorea-hemiballismus[8] revealed decreased ^{123}I ioflupane binding in the striatum as was observed in our case. Cardiac imaging with ^{123}I MIBG scintigraphy in our case revealed normal ^{123}I MIBG uptake by the cardiac sympathetic nerve, suggesting that coexisting Parkinson's disease, Lewy body disease, diabetic sympathetic nerve dysfunction, or heart failure were unlikely. This suggests nigrostriatal dopaminergic involvement: presynaptic neuronal terminal dysfunction, reduced density of nerve terminals, or reduced density of dopamine transporter is conceivable.[8,9]

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