



Atypical Diabetic Striatopathy with Hypokinetic Parkinsonism and Levodopa Responsiveness



Suk-Kyeong Kee, MD¹, Gi-Wook Kim, MD, PhD^{1,2}, Yu Hui Won, MD, PhD^{1,2}, Sung-Hee Park, MD, PhD^{1,2}, Myoung-Hwan Ko, MD, PhD^{1,2}, Jeong-Hwan Seo, MD, PhD^{1,2}, Da-Sol Kim, MD, PhD^{1,2}

¹ Department of Physical Medicine and Rehabilitation, Jeonbuk National University Medical School, Jeonju, Korea
² Research Institute of Clinical Medicine, Biomedical Research Institute of Jeonbuk National University Hospital

Introduction

- Diabetic striatopathy (DS) is a rare neurologic complication of nonketotic hyperglycemia, typically presenting as hyperkinetic chorea or ballism. It is classically characterized by T1-weighted striatal hyperintensity on MRI.
- Hypokinetic presentations and the coexistence of DS with idiopathic Parkinson's disease (iPD) are extremely uncommon. To report a rare case of DS presenting with a parkinsonian phenotype and significant levodopa responsiveness

Case presentation (59/F)

Chief complaints	Progressive gait disturbance
Onset	1 month before presentation
Vector	Long-standing diabetes mellitus with severe hyperglycemia
Associated symptoms	Bilateral hand tremor, bradykinesia, postural instability
Past history	Diabetes mellitus
Previous medication	Levodopa (initiated at local clinic)
Neurologic examination	<ul style="list-style-type: none"> ✓ Masked facies (Fig 1-A) ✓ Stooped posture (Fig 2-B) ✓ UPDRS part III score: 30 ✓ Hoehn and Yahr stage 3
Diagnostic assessment	<ul style="list-style-type: none"> ✓ Laboratory test: HbA1c 16.2% (marked chronic hyperglycemia) ✓ Brain MRI(Fig 2) <ol style="list-style-type: none"> 1) Bilateral striatal T1 hyperintensity (consistent with diabetic striatopathy) 2) DWI: Small focal hyperintense lesion in left basal ganglia (suggestive of acute infarction)
Therapeutic intervention	<ul style="list-style-type: none"> ✓ Levodopa dose titration ✓ Intensive glycemic control ✓ Rehabilitation program including gait training, functional mobility training, postural stability training

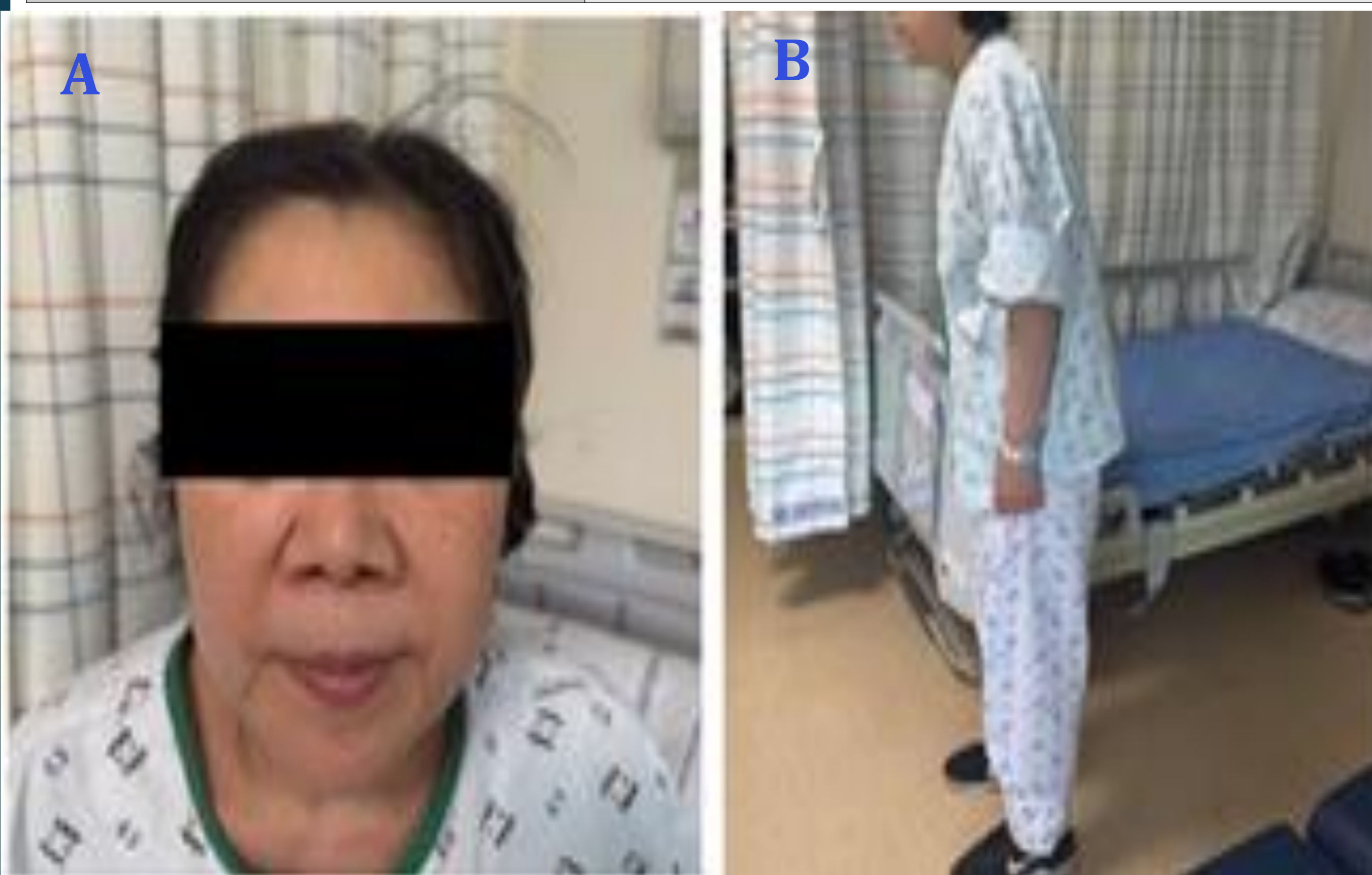


Fig.1 A) Masked face and B) Stooped posture

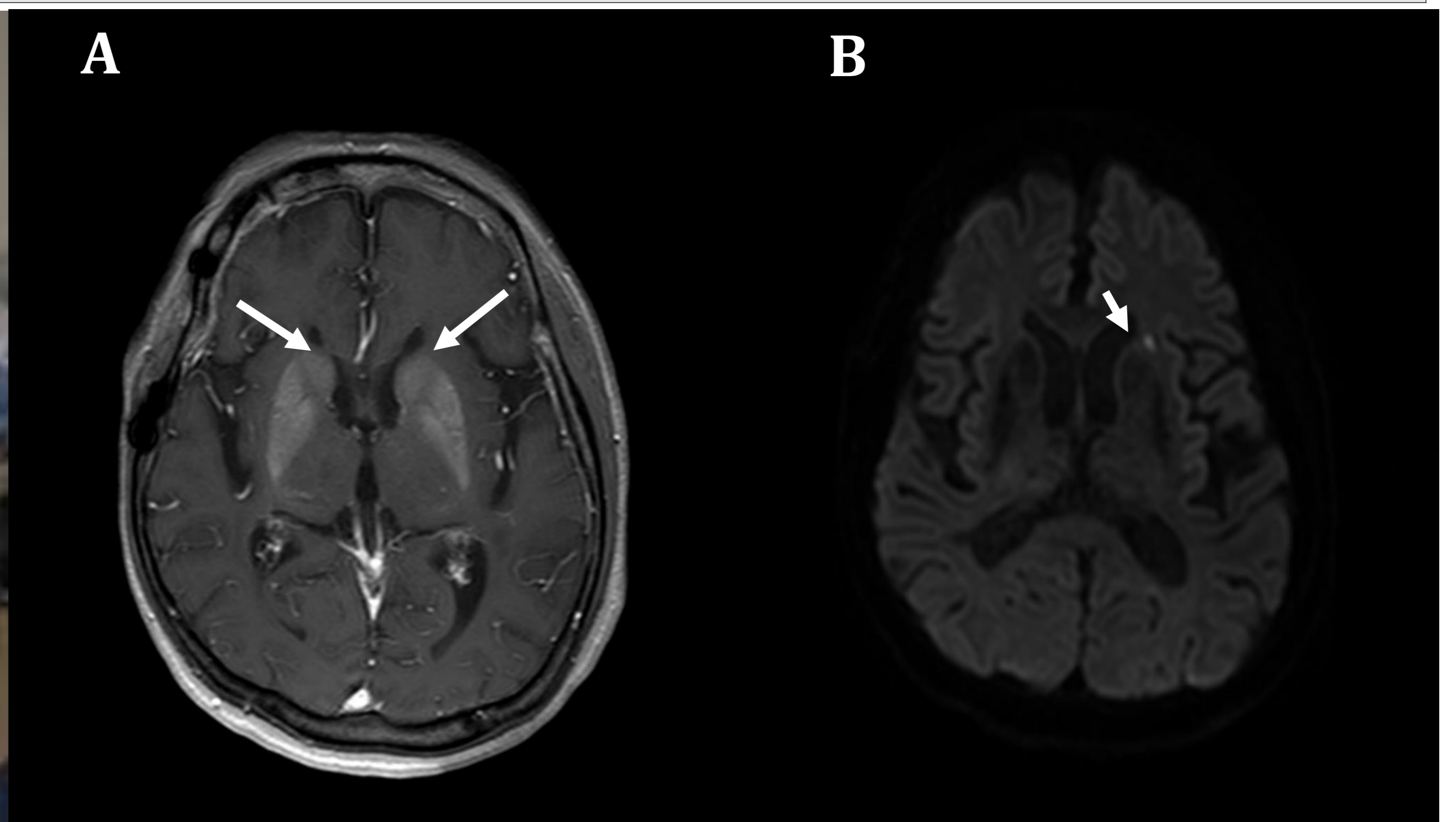


Fig.2 (A) Bilateral striatal hyperintensity axial T1-weighted imaging(arrows) (B) Small focal hyperintense lesion in the left basal ganglia(white arrowhead) on diffusion-weighted imaging

Outcome

Medication

- Glucose control : Diabex 500mg BID, Trajenta 5mg D, Tresiba 6 units before breakfast
- Dopaminergic therapy : Levodopa titration
- Equifina(MAOI-B) was co-administered to control motor symptoms

Rehabilitation

- Gait training using Morning walks and Angelegs
- Applied auditory stimulation to maintain a consistent cadence
- OT, Dysphagia therapy, Speech and language therapy

Electrodiagnostic study

- Findings : No evidence of abnormal findings

Functional improvement (Fig.3)

- Substantial improvement in gait and posture
- Regained ability to ambulate with improved postural stability

Date	MMSE	BNT	MBI	BBT	UPDRS
03.08	27	46	64	35	30
03.15	28	50	74	42	26
03.22	30	53	82	46	22

Fig.3 Progressive improvement in cognitive and functional outcomes (MMSE, BNT, MBI) and BBT, UPDRS part 3

Conclusion

- Diabetic striatopathy (DS) usually presents as hyperkinetic movements, but this case highlights an **atypical hypokinetic phenotype**. Levodopa responsiveness in DS patients should raise suspicion for **coexisting idiopathic Parkinson's disease (iPD)**.
- Meaningful functional recovery can be achieved through a combination of **dopaminergic therapy, aggressive glycemic control, and tailored rehabilitation**