“THE QUIET CEREBELLUM”-AN UNUSUAL RADIOLOGICAL SIGNATURE OF ASYMPTOMATIC CEREBELLAR INVOLVEMENT IN A DIABETIC PATIENT WITH HYPOGLYCEMIA

DR SREEDEVE SHAMRITHA,DM NEUROLGY RESIDENT, THANJAVUR MEDICAL COLLEGE
DR ARUN RAJ.E,PROFESSOR AND HEAD OF DEPARTMENT
DR SEKAR, ASSISTANT PROFESSOR,THANJAVUR MEDICAL COLLEGE

INTRODUCTION

Cerebellum is affected in wide variety of conditions including cerebellar malformations, vascular pathologies, infective & inflammatory conditions, toxic and metabolic processes, demyelinating diseases, neurodegenerative disorders and neoplasms. Cerebellar abnormalities on imaging are typically associated with clinically evident ataxia, dysarthria or co ordination deficits. We report a unique case of isolated cerebellar abnormality in neuroimaging in an asymptomatic patient.

CASE REPORT

56 Year old male known hypertensive and diabetic on oral hypoglycemic agents which was switched to dual oha 6 months prior to admission presented with recurrent episodes of giddiness, sweating and palpitations on and off for past 3 months which improved in minutes after consumption of sugar or food. Patient attributed those events to hypoglycemia on own and did not seek medical attention. He had no seizures or motor deficits or altered sensorium. No History of Unsteadiness or incordination or visual disturbances. Patient was admitted to our hospital during the evaluation he had 2 -3 episodes of documented hypoglycemia (60 mg/dl) following which his diabetic drugs were modified with no recurrence of events after.

No recent history of fever or vaccination. No History of loss of appetite or weight loss. No history of intake of any other medications. He is an occassional alcoholic, not a smoker. No trauma/prior hospital admissions.

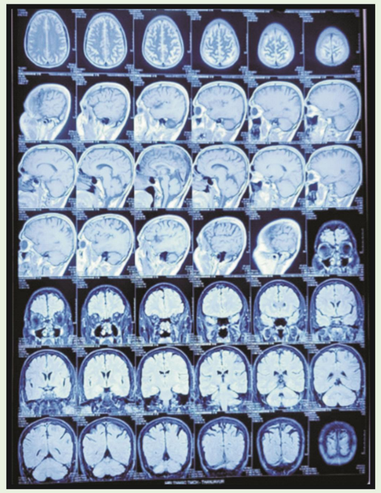
On Examination:

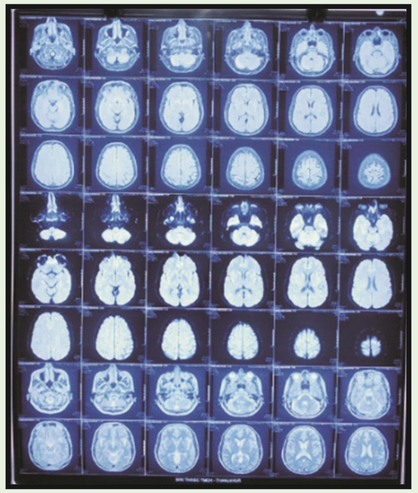
Patient moderately built and nourished with no pallor or clubbing lymphadenopathy. Higher mental functions and cranial nerve examinations were normal. Motor and sensory examination was normal. Reflexes were present and plantar was bilateral flexor. Cerebellar examination revealed no axial or appendicular incordination, tandem walking was intact. Rhomberg was negative. Bedside autonomic tests were normal. Cadiac, respiratory and abdomen examinations were also normal.

Blood investigations:cbc-normal, no evidence of elevated counts. Renal and liver function test were normal. Electrolytes including sodium, potassium, calcium, magnesium were normal. CT brain, chest and ct abdomen were normal. Cardiac evaluvation done-normal.

VEP, EEG and nerve conduction studies were normal.

MRI brain revealed T2/Flair hyperintensities in bilateral posteroinferior cerebellum with involvement of right more than left without diffusion restriction. Possibility of metabolic cause.





DISCUSSION

This case illustrates an uncommon radiological pattern of cerebellar involvement since involvement did not fit into arterial territory, vascular causes were ruled out. No recent evidence of infection & absence of contrast enhancement ruled out infective, inflammatory and demyelianting conditions. Paraneoplastic workup was negative. Based on his history and evaluavation, this was most likely to be due to hypoglycemia presenting only with autonomic symptoms. Adult hypoglycemia can present wih widerange of symptoms even leading to hypoglycemic coma. Hypoglycemia causing imaging abnormalities can predominantly affect grey matter involving cortex, neostriatum and hippocampus or 2) predominant white matter involvement affecting periventricular white matter, internal capsule and splenium of corpus callosum or 3) mixed pattern. mostly spares cerebellum. In this patient unique cerebellar involvement without symptoms and without diffusion restriction and resolution of imaging findings after a month mostly points out to hypoglycemia causing above mentioned change. Probable explanation could be predliction to affect purkinjie cells in posterior cerebellum since they are glucose dependent and prone to metabolic stress. Early radiological evidence may precede clinical manifestations.

CONCLUSION

This appears to be the first report of isolated asymptomatic cerebellar involvement in hypoglycemia. Only nine previous cases describe middle cerebellar peduncle lesions. In Diabetics with vague symptoms, incidental cerebellar MRI findings should prompt evaluation for hypoglycemia. Early recognition and glycemic optimization are essential to prevent permanent injury.

REFERENCE

1. G. Bathla, B. Policeni, A. Agarwal Neuroimaging in Patients with Abnormal Blood Glucose Levels American Journal of Neuroradiology May 2014, 35 (5) 833-840.
2. Ma JH, Kim YJ, Yoo WJ, Ihn YK, Kim JY, Song HH, Kim BS. MR imaging of hypoglycemic encephalopathy: lesion distribution and prognosis prediction by diffusion-weighted imaging. Neuroradiology. 2009 Oct;51(10):641-9.
3. Jeon SJ, Choi SS, Kim HY, Yu IK. Acute Acquired Metabolic Encephalopathy Based on Diffusion MRI. Korean J Radiol. 2021 Dec;22(12):2034-2051.
4. D.E. Kim, S.H. Park, S.K. Kim, et al. Hypoglycemia-induced cerebellar dysfunction and quantitative positron emission tomography study. Neurology 2000;55;418-422
5. Romero SH, Torres DV, Rivera DM. Bilateral middle cerebellar peduncle compromise due to hypoglycemic encephalopathy: A case report and literature review. Radiol Case Rep. 2022 Sep 28;17(12):4599-4602.
6. Arora R. Imaging spectrum of cerebellar pathologies: a pictorial essay. Pol J Radiol. 2015 Mar 16;80:142-50.