ALCOHOL RELATED CEREBELLAR DEGENERATION: A CASE REPORT

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ABSTRACT

Alcohol related cerebellar degeneration in one of the feared complications with unsatisfactory recovery that requires high index of suspicion for identification. We report a case of 62 years alcohol consumer female who presented with ataxia and dysarthria of subacute onset. She demonstrated bilateral cerebellar signs on examination with MRI brain suggestive of bilateral cerebellar atrophy.

KEYWORDS

Ataxia, Cerebellar atrophy, SARA score

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INTRODUCTION

Alcohol use is a leading risk factor for the global disease burden linking its consumption to more than 60 acute and chronic diseases.[1] Among them, alcohol related cerebellar degeneration is considered one of the commonest form of acquired cerebellar ataxia.[2] The pathophysiology remains unclear but the various proposed mechanisms includes excitotoxicity, dietary factors especially thiamine deficiency, oxidative stress, compromised energy production and cell death.[3] Radiological finding of this disorder is cerebellar atrophy with anterior superior cerebellar vermis being predominantly affected.[4] Tissue volume loss in this area is due to either shrinkage or atrophy of Purkinje cells, large nerve cells that make up much of the vermis. Figure 1 shows intracellular mechanisms underlying ethanol-induced cell death and abnormal development.[3] Cerebellar shrinkage is most noticeable in older alcoholics with at least a 10-year duration of alcoholism.[5] The onset of the cerebellar symptoms usually occurs at middle age, with a significant history of chronic alcohol abuse. Permanent cerebellar deficits are observed among alcoholics, and they persist even with alcoholic abstinence.[1] The cerebellum is the structure that controls the stability of the gait. Chronic cerebellar diseases and acute alcohol intoxication affect cerebellar function; the mechanism behind this has been depicted in figure 1. Subacute or chronic onset of ataxia, nystagmus, dysarthria, dizziness and cognitive dysfunction are major neurological manifestations of this disease.

CASE REPORT

A 62-year female patient presented to emergency department in December 2024 with an alleged history of fall injury with impact over her head and bilateral shoulder resulting in bilateral clavicular fracture with a small wound over her forehead. She mentioned that she had an episode of dizziness followed by imbalance while walking and she accidentally fell from her stairs. She complained that she has developed dizziness and imbalance since last one month for which she did not seek medical treatment. While speaking to her we noticed that her speech was not clear. She was able to comprehend words spoken to her but could not respond with clarity. She gave history that she developed slurred speech for the same duration of time. On further inquiry, she admits regular consumption of homemade local alcohol since last 42 years; approximately 30 grams per day. She was a daily drinker with an AUDIT-C score of 9. The AUDIT-C score is The Alcohol Use Disorders Identification Test (AUDIT-C) is scored on a scale of 0–12, with score ≥ 4 in male and ≥ 3 in female considered positive. Positive score indicates greater likelihood of alcohol use. Family history was not significant for similar illness in the family. On examination, her vital parameters were stable. She had bilateral cerebellar signs with a SARA score of 25. She had difficulty in articulation of words, could not coordinate alternate hand movements, developed past pointing while reaching target, had a wide base gait with tendency to fall while walking. No nystagmus or rebound phenomenon were observed. Cranial nerve examination was normal. Romberg's test was negative. Other motor and sensory system examinations were normal. She was admitted to the medical ward and routine blood investigations were sent. She had a hemoglobin of 12.5 gm/dl with MCV of 91 fl, serum sodium 139 mEq/L, serum potassium 4.2 mEq/L, with a normal blood sugar level of 99 mg/dl. Her serology was non-reactive with a negative VDRL test. Her liver function was deranged, and ultrasonography showed fatty changes in liver. Liver function test was normal. Serum vitamin B12 level was 320 pg/ml and TSH was 0.53 uIU/ml. MRI brain with whole spine screening was done which revealed bilateral cerebellar hemisphere atrophic changes with prominent CSF spaces with no abnormality of spine. She was advised for alcohol abstinence and was treated conservatively with thiamine supplementation 200 mg intravenous thrice daily. She got symptomatically better during the 5 days stay at our hospital.

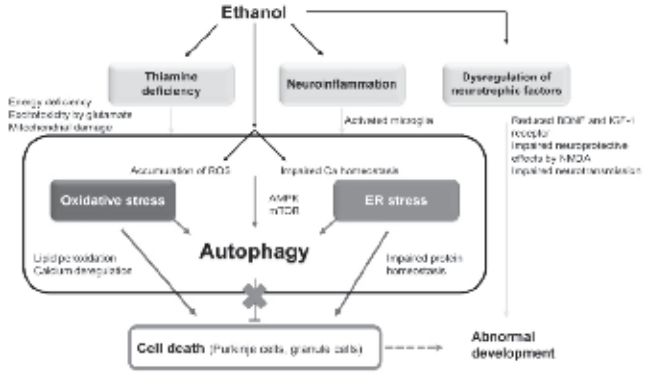

Figure 1. Shows intracellular mechanisms underlying ethanol-induced cell death and abnormal development

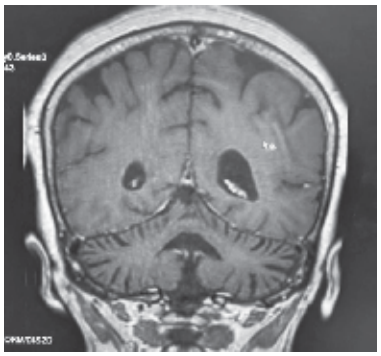

Figure 2. T1 Coronal image showing prominent extra axial CSF Spaces in between the lobules of cerebellum

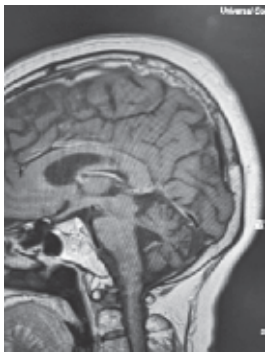

Figure 3. T1 sagittal image showing prominent extra axial CSF Spaces in between the lobules of cerebellum

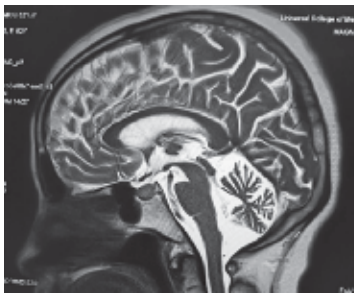

Figure 4. T2 sagittal image showing diffuse atrophy of cerebellum more prominent on vermis

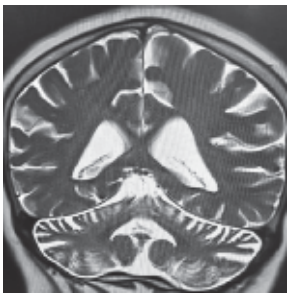

Figure 5. T2 coronal image showing difuse atrophy of cerebellum more prominent on vermis

DISCUSSION

Alcohol related cerebellar degeneration is common among middle aged patients ranging from 40 to 75 years with mostly female predominance.[6] Our patient was also female with age of 51 years. Perhaps the most common form of cerebellar dysfunction, alcoholic cerebellar degeneration, is frequently observed in alcoholics after around 10 or more years of heavy drinking.[7] Our patient had also consumed alcohol for 42 years in moderate amount. Alcoholic cerebellar ataxia typically evolves gradually over weeks to months but may also occur abruptly or progress over many years.[6] In our case, she had developed symptoms gradually over one month. Conditions may improve after abstinence from alcohol consumption.[8] SARA score is a clinical scale that is based on a semi-quantitative assessment of cerebellar ataxia on an impairment level with score ranging from 8 to 40.[9] Our patient had SARA score of 25. Her clinical examination revealed findings consistent with bilateral cerebellar dysfunction with intact higher mental function, sensory and motor examination. Other causes of cerebellar dysfunction include multiple sclerosis, tumors, cerebellitis, vascular disorders including stroke etc. This patient didn't have a previous history of similar attack and no radiological findings suggesting alternative diagnosis. Her electrolytes were normal, vitamin B12 level and thyroid function were normal and venereal disease research laboratory (VDRL) test for syphillis was negative. Neuroimaging findings in patients with alcohol related cerebellar degeneration includes marked cerebellar atrophy in superior vermis on sagittal T1-weighted brain MRI.[10] Our case also demonstrated prominent extra-axial CSF space in between the lobules of cerebellum on T1 images (Figure 2 and 3) as well as diffuse cerebellar atrophy (more prominent of vermis) on T2 images (Figure 4 and 5).

CONCLUSION

Alcohol related cerebellar degeneration is an easily identifiable and treatable condition though the outcomes are variable. Any chronic alcohol consumers presenting with symptoms of cerebellar dysfunction should be promptly evaluated for alcohol related cerebellar degeneration. However, other conditions with similar presentation like demyelinating disorders, vascular disorders like stroke, autoimmune causes, tumors must be ruled out.

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CONFLICT OF INTEREST

None

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