

Non-autoimmune hypothyroidism induced cerebellar atrophy

Saumya H Mittal*, Rakshith K C, Misri ZK, Shivananda Pai

Email: saumyamittal1@gmail.com

Abstract

A 45-year-old gentleman, non-diabetic, normotensive, non-alcoholic and non-smoker presented with the complaints of insidious onset, progressive imbalance while walking, with occasional falls for the past six to eight months. The patient had no other complaints. He had exaggerated reflexes and loss of vibration sense. He also had wide based ataxic gait, with inability to perform fine coordinated actions. He had associated nystagmus. MRI brain suggested cerebellar atrophy. The thyroid profile was TSH- 176.9 mIU/L, FT3- 0.963 pmol/L, FT4- 0.023 pmol/L and antibodies were negative. All other parameters like complete blood count, renal and hepatic function tests, peripheral smear, vitamin B12, folate levels, ultrasonogram abdomen and nerve conduction velocity were within normal range. We present a case of cerebellar atrophy in a patient of hypothyroidism with no autoimmune condition or antibody detected. This is a rare presentation and the exact mechanism behind this occurrence is unknown. This case reveals that the possible mechanism behind ataxia in hypothyroidism may be cerebellar atrophy. And there is an unknown mechanism for the cerebellar atrophy different from that known for autoimmune conditions and Hashimoto's thyroiditis.

Key words: Cerebellar atrophy, Hypothyroidism, Induced cerebellar atrophy, non-autoimmune hypothyroidism

Case report

We present the case of a 45-year-old gentleman, non-diabetic, normotensive, non-alcoholic and non-smoker who came to us with the complaints of imbalance while walking, associated with occasional falls for the past six to eight months. The patient had no other complaints and stated that the symptoms were slowly progressing and the frequency of falls was increasing. The patient had exaggerated reflexes and loss of vibration sense. He also had wide based ataxic gait, with inability to perform fine coordinated actions. He had associated nystagmus. He had no similar past or family history.

In view of the ataxia, an MRI brain was done, which showed cerebellar atrophy (figure 1). The patient's thyroid profile was TSH- 176.9 mIU/L, FT3-0.963 pmol/l, and FT4-0.023 pmol/l. The anti-TPO, anti-TG, TRAb antibodies were negative. All other parameters like complete blood count, renal and hepatic function tests, peripheral smear, serum vitamin B12 and folate levels, ultrasonogram abdomen and nerve conduction study were within normal range. Thyroid replacement at a dose of 100 mcg per day was initiated, and the patient showed marked improvement over the next two weeks. He was thereafter discharged and reviewed in OPD

Saumya H Mittal

MBBS, MD (Medicine), DNB (Neurology)
SR, DNB Neurology, KMC Hospital, Mangalore.

Rakshith K C

MBBS, MD (Medicine), DM (Neurology)
Associate Professor and HoD, KMC Hospital, Mangalore.

Misri ZK

MBBS, MD (Medicine), DM (Neurology)
Associate Professor, KMC Hospital, Mangalore.

ShivanandaPai

MBBS, MD (Medicine), DM (Neurology)
Associate Professor, KMC Hospital, Mangalore.

*Corresponding Author

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after two weeks, where he was found to be clinically stable and with improved gait.

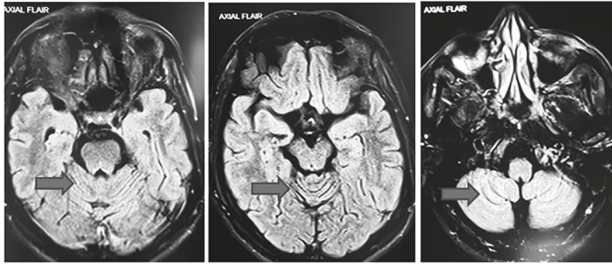


Figure 1: MRI brain AXIAL FLAIR suggestive of cerebellar atrophy

Discussion

Cerebellar disorders are organized by aetiology into inherited or idiopathic degeneration, nutritional disorders, neoplastic and paraneoplastic disorders, developmental disorders, disorders due to infection, vascular disorders, intoxications, trauma, metabolic disorders, and demyelinating and dysmyelinating disorders.¹

Causes of acquired cerebellar atrophy in children include prematurity, neonatal hypoxic ischemic encephalopathy, multiple sclerosis, post traumatic brain injury, paraneoplastic conditions, chronic epilepsy, radiation therapy and posterior fossa surgery, malnutrition and toxins, e.g., phenytoin, tacrolimus, heavy metals.²

Progressive non-familial adult onset cerebellar degeneration is caused by well recognized conditions including nutritional deficiencies, intoxications, hypoxia, paraneoplastic syndromes, and hyperthermia.³

Cerebellar ataxia is known with autoimmune disorders like stiff person syndrome, celiac disease, diabetes mellitus (type 1), and Sjogren's syndrome. The progress in such patients is usually slow. The cases of cerebellar ataxia associated with hypothyroidism are autoimmune conditions. In fact, it is considered that the cerebellar symptoms develop in the patients with hypothyroidism due to the attack by autoimmune mechanisms on the cerebellar neurons that leads to neuronal loss and gliosis.⁴

Cerebellar ataxia due to hypothyroidism is rare. Hypothyroidism has occasionally been known as a cause of gait ataxia. The exact mechanism of this is not known. Few authorities consider it to

be possibly due to cerebellar dysfunction, but not atrophy.^{3,4} Thyroid hormones have been known to be essential and important for the development of brain. Derangements of thyroid hormones in either wing of the spectrum can affect brain development. While the effects on intellectual functions are well recognized, effects on cerebellar development have been recorded.⁵ Similarly, a prior report on post-mortem examination suggested the degeneration of cerebellum in a patient of myxedema. The parts that were chiefly found to be affected were vermis, middle and superior peduncles.⁶

Conclusion

We present a case of cerebellar atrophy in a patient of hypothyroidism with no autoimmune condition or antibody detected. This is a rare presentation and the exact mechanism behind this occurrence is unknown. This case reveals that the possible mechanism behind ataxia in hypothyroidism may be cerebellar atrophy. And there is an unknown mechanism for the cerebellar atrophy different from that known for autoimmune conditions and Hashimoto's thyroiditis.

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