

An Unusual Presentation due to Usual Problem : Cerebellar Ataxia due to Hypothyroidism: A Case Report

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ABSTRACT

Auto-immune Hypothyroidism is one of the common causes of hypothyroidism. The usual clinical features of auto-immune hypothyroidism are constipation, fatigue, cold intolerance and weight gain. Rarely it can present with neurologic problems like reversible cerebellar ataxia, dementia, peripheral neuropathy, psychosis and coma. A 37-year-old lady presented with the history of gait-ataxia and generalized body pains. Investigations revealed hypothyroidism. Anti-TPO antibody was positive. Thyroid replacement therapy was started and patient improved completely within 2 weeks. Case report is targeted to spread awareness that hypothyroidism should be considered in all patients who present with acute onset of cerebellar ataxia.

Keywords: Ataxia, tremor, hypothyroidism,

INTRODUCTION

Cerebellar ataxia (CA) can rarely arise from autoimmune disorders, including Sjogren's syndrome, type I diabetes mellitus (DM), Stiff person syndrome, and celiac disease.^[1] Hypothyroidism is a common problem in clinical practice. In almost all the cases, the neurologic manifestations occur along with the systemic features of the disease. However, symptoms of neurologic dysfunction may be the presenting feature in some patients. Untreated hypothyroidism may also be associated with CA

in 5.52% of cases^[1], although the onset is usually more rapid, occurring over several months, the ataxia is reversible with thyroid hormone replacement.^[2,3]

Our patient presented with only cerebellar ataxia as presenting complaint and no neurological cause could be attributed. Further workup showed altered thyroid profile. Hypothyroidism should be considered in all cases of cerebellar ataxia as it is a reversible cause of ataxia. We report this case to emphasize the fact that acute cerebellar ataxia can be sole presenting feature of hypothyroidism.

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CASE REPORT

A 37-year-old lady presented to our hospital with history of truncal ataxia since 2 weeks with generalized body pain and constipation since one year. Unsteadiness was acute in onset and progressive in nature. There

Acute Intestinal Presentation due to Typhoid: A Case Report

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ABSTRACT

Intestinal presentation is one of the commonest forms of typhoid fever. It is characterized by abdominal pain, tenderness, and sometimes by localized or generalized ileocolitis. The clinical picture is often non-specific, and the diagnosis is usually confirmed by blood and bone marrow cultures. In this report, we describe a case of acute intestinal presentation of typhoid fever in a young male patient. The patient presented with abdominal pain, fever, and malaise. The diagnosis was confirmed by blood and bone marrow cultures. The patient responded well to treatment with antibiotics and supportive care.

INTRODUCTION

Typhoid fever is a systemic infection caused by *Salmonella typhi*. It is characterized by a prolonged fever, malaise, and sometimes by localized or generalized ileocolitis. The clinical picture is often non-specific, and the diagnosis is usually confirmed by blood and bone marrow cultures. In this report, we describe a case of acute intestinal presentation of typhoid fever in a young male patient. The patient presented with abdominal pain, fever, and malaise. The diagnosis was confirmed by blood and bone marrow cultures. The patient responded well to treatment with antibiotics and supportive care.

CASE REPORT

A 25-year-old male patient presented to the hospital with abdominal pain, fever, and malaise. The patient had been ill for about a week. The pain was localized to the right lower quadrant. The fever was intermittent, with temperatures ranging from 38°C to 40°C. The patient also had a loss of appetite and some weight loss. The physical examination revealed tenderness in the right lower quadrant. The blood and bone marrow cultures were positive for *Salmonella typhi*. The patient was treated with ceftriaxone and supportive care, and he made a full recovery.

was no history of weakness of limbs, headache, vomiting, convulsions or loss of consciousness. There was no history of trauma to the head, fever or drug intake. On examination her vitals were normal. Cognitive functions were normal. Neurological examination showed gait ataxia, dysarthria and dysmetria on finger-nose and heel-to-knee tests. Dysdiadochokinesia of both hands was noted, The gait was wide-based and ataxic. Tandem walking was impaired. Her fundus was normal. Her power and reflexes were normal. Sensory system was normal. There was no evidence of autonomic dysfunction.

Her investigations revealed: Hb: 14.3g%, TLC: 6,000 /mm³, DLC: N44, L40, E16, ESR: 60mm, Platelet count: 2, 92, 000/mm. Serum electrolytes, blood sugar, renal and liver function tests were normal. HIV was negative. Her chest X-ray was normal. MRI Brain was normal. In further evaluation of cerebellar ataxia T3, T4, TSH were ordered and reports were: T3:0.410ng/ml (Ref: 0.7-2.0), T4: 0.77ng/ml (Ref: 4.5-11), TSH:>100mcIU/ml (Ref: 0.4-4.2). The Serum anti-TPO antibody was elevated.

Her lab investigations suggested hypothyroidism (auto-immune). She was started on 100µg of thyroxine. She improved completely within 2 weeks.

DISCUSSION

Hypothyroidism is one of the causes of acute onset (usually reversible) ataxia.^[4] Stroke, viral encephalitis and drugs can also cause acute cerebellar ataxia. Mass lesions in the posterior fossa, infections such as HIV, deficiency

syndromes of B1 and B12, alcohol and paraneoplastic syndromes are causes of subacute onset cerebellar ataxia in an adult. Hypothyroidism has been recognized as a cause of gait ataxia.^[5] Hormone Replacement therapy has reversed the cerebellar symptoms in most patients^[6], suggesting that symptoms were due to endocrine mediated dysfunction of the cerebellum. In some patients with hypothyroidism, the cerebellar deficits have persisted despite adequate treatment.^[7] Our patient was started on 100µg of thyroxine and she improved completely within 3 weeks with resolution of the symptoms.

In a study^[7] it is postulated that there were two separate mechanisms resulting in cerebellar dysfunction in patients with thyroid disorders of hypothyroidism is not associated with auto-immunity, the endocrine disorder produces cerebellar dysfunction that could be reversed by thyroid replacement. In patients with autoimmune thyroiditis not reversed by thyroid replacement therapy, auto-immune mediated cerebellar degeneration was a likely mechanism.^[7] Our patient showed an excellent response to treatment.

In summary, we present a case of cerebellar ataxia in a patient with hypothyroidism, where patient became completely asymptomatic after hormone replacement therapy. Classical signs of hypothyroidism may not be clinically obvious in patients presenting with ataxia.^[8] We would like to stress the fact the unusual symptom can be due to usual problem which can be dealt with little awareness.

CONCLUSION

This case report is aimed to highlight the fact that sometimes unusual presentation can be there for usual problems. Cerebellar ataxia can be the only presenting complaint in patients of Hypothyroidism and such cases should be tested for thyroid hormones and antithyroid antibodies (anti-TPO).

Hypothyroidism is a reversible cause of ataxia. Hence clinicians should keep such possibilities in mind and so appropriate treatment can be started without wasting time.

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