International Journal of Health Sciences and Research
Vol.13; Issue: 6; June 2023
Website: www.ijhsr.org
Case Report *ISSN: 2249-9571*

Reversible Speech Dysfunction in a Case of Hashimoto's Encephalopathy: A Rare Manifestation

Kumar Anupam[1], Santhosh Kumar Singh[2], Rohit Vashisht[3], Suresh K Srinivasan[4], Balakrishnan Arivalagan[5], Srishthi Bhardwaj[6], Vamsi Kota[7]

[1]Dept of Internal Medicine, Command Hospital, Pune
[2,3]Dept of Internal Medicine AFMC, Pune
[4]Dept of Anesthesiology, Trinity Health Systems, Steubenville, Ohio
[5]Dept of Internal Medicine AFMC, Pune
[6]Dept of Internal Medicine, Chennai Medical College, Thirchy
[7]Dept of Internal Medicine Augusta University, Georgia

Corresponding Author: Dr Balakrishnan Arivalagan

DOI: https://doi.org/10.52403/ijhsr.20230624

ABSTRACT

Thyroid hormone plays a significant role in the neurocognitive development. Language disorders with thyroid agenesis and congenital hypothyroidism are well documented in the literature however language dysfunction with adult hypothyroidism is very rarely reported in the literature. Here we present a rare case of language dysfunction in a 33 years old male with hypothyroidism with elevated Anti thyroid peroxidase antibody (TPO) antibody titer with no other significant abnormality on thorough examination and detailed evaluation. He had no response with thyroxine replacement alone but responded dramatically with steroid therapy in anti-inflammatory doses. This case possibly highlights the probable role of steroid apart from standard indication like steroid responsive encephalopathy.

Keywords: Thyroid Hormone, Language disorder, Hypothyroidism, Anti TPO, Steroids

INTRODUCTION

Thyroid hormones are required for maturation of neuronal circuits in the intrauterine life [1,2]. Development defects in language are well known in thyroid agenesis and cretinism [3]. There is enough literature available on effects of thyroid hormones on language in childhood [4,5] but the language dysfunction occurring later in adult life as a result of acquired hypothyroidism remains an ill-defined entity.

CASE SUMMARY

A 33-year-old male with no addictions presented with difficulty in speech for a period of few months. He took some time to start answering a question. He used appropriate words but repeated the words many times before starting the next word. He was visibly annoyed with this, tried to correct himself, but the pattern kept repeating itself (Video 1). There was no definite slurring or neologisms. Despite repeated sessions of listening to him, the pattern did not seem to satisfy a specific type of aphasia. His intellect, memory, recall, comprehension and articulation were intact. He has no clinical evidence of cerebellar, motor, sensory, autonomic and other cortical dysfunction. He was diagnosed as a case of hypothyroidism about a year back and had been on suboptimal dose for the same. Despite

increase in the dose, his speech dysfunction persisted as shown in (Video 2).

His complete hematological, biochemical profile, Magnetic Resonance Imaging (MRI) brain, Cerebrospinal fluid (CSF) analysis and Electroencephalograph (EEG) were turned out to be normal (Table 1). His thyroid function test was deranged along with high serum anti TPO antibody titers (>1000 IU/ml), suggesting Hashimoto's hypothyroidism hence we optimized the dose of thyroxine along with addition of oral prednisolone in anti-inflammatory doses. Over a period of few days an improvement in his speech output was noticed and so the therapy was continued (Video 3). After a period of 03 weeks of therapy, slowness of his speech and repetition of words was restored to normal (Video 4).

Table 1

Investigation	**Results**	**Investigation**	**Results**
Hb(gm/dl)	14.0	**Urea(mg/dl)**	25
TLC (/cumm)	4600	**Creatinine (mg/dl)**	0.9
DLC	P70l20	**Sodium(mEq/dL)**	142
Plt (Lakh/cumm)	1.54L/cumm	**Potassium(mEq/L)**	4.2
FBS/PPBS (mg/dl)	104/114	**S. Bilirubin (Total/Direct) (mg/dl)**	0.9/0.2
ECG	NAD	**SGOT/SGPT(IU/L)**	36/74
HBsAg/Anti HCV/HIV	Negative	**TSH**	5.92 microL/ml (0.27-4.20)
TG/TC(mg/dl)	214/143	**T4**	5.37mcg/ml (5.10-14.10)
Urine RE and ME	NAD	**Chest X ray Posterio-anterior view**	NAD
T3	100 ng/dl (80-200)	**2D Echo**	NAD
Anti TPO	1000 IU/ml (<12)	**USG Abdomen**	NAD
USG Neck	Increased vascularity with no gland enlargement or regional lymphadenopathy.	**CSF for autoimmunepanel**	Negative
CSF (Cytology and biochemistry)	NAD	MRI BRAIN	NAD

ECG- Electrocardiograph, USG – Ultrasonography
TC/TG-Total cholesterol/Triglyceride,
RE/ME- Routine/Microscopic Examination
CSF- Cerebrospinal Fluid
TPO - Thyroid Peroxidase, TSH – Thyroid Stimulating hormone
HBsAg- Hepatitis B surface antigen
HCV- Hepatitis C Virus
HIV- Human Immunodeficiency Virus
MRI – Magnetic Resonance Imaging

DISCUSSION

Disorders of speech are classified as motor, Wernicke's, conduction aphasia and global aphasia. Dysarthria is arising out of in coordination of oropharyngeal, nasopharyngeal, vocal cords or mechanical dysfunctions of these structures. This patient did not have any feature to suggest the well understood aphasias or dysarthrias[6]. There was definite slowing of the initiation of speech and repetitions of words or syllables or phrases. He was able to comprehend the questions well and answered in the right context and content. There were no neologisms as well. The intonations of normal voice were missing and speech appeared monotonous, slow and repetitive. There was no slurring of speech. He could pronounce the vowels and consonants clearly albeit with a delay and frequent repeats.

Literature search on the subject revealed many studies among children and language dysfunction arising from congenital hypothyroidism [4,5]. Monica Barby et al[4] in their review has quoted found only 02 articles looking into the speech and the voice among the baby with congenital hypothyroidism. A few studies have been published in the journals of Otorhinolaryngology which objectively looked at the changes in structure of voice as analyzed by voice therapists [6].

Mohammad Zadeh et al[7] found significant voice and phonation abnormality in adults with hypothyroidism. However, the specificsof this type of language dysfunction seldom find a mention in literature.

Literature on the neurocognitive deficits in hypothyroidism is available and does mention the slowed cognition as a sign of hypothyroidism. Since language use is a cognitive function, we presume that the deficit seen in this patient was a part of hypothyroidism[8–10] as described by various authors.

In our case the deficit persisted despite being on thyroxine supplementation and responded dramatically after treatment with steroids over a period of few weeks. This aspect, steroid responsiveness, of such deficit has not been described in literature as the anti TPO Antibody titers were high we presume that the response was largely by limitation of the inflammatory process in the brain, albeit no reference for such response exists outside the common indication of altered sensorium in Hashimoto`s encephalopathy (steroid responsive encephalopathy) [11,12,13].

CONCLUSION

The present case of language dysfunction in Hypothyroidism and its significant response to treatment with steroid warrants detailed evaluation of these aspects in hypothyroidism patients and patient with language dysfunction. We may consider steroids in auto inflammatory doses for patients with hypothyroidism with language dysfunction with elevated anti TPO antibody titer with no response to thyroxine replacement.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understands that his name and initials will not be published and due efforts will be made to conceal his identity, but anonymity cannot be guaranteed.

Declaration by Authors

Acknowledgement: None

Source of Funding: None

Conflict of Interest: The authors declare no conflict of interest.

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How to cite this article: Kumar Anupam, Santhosh Kumar Singh, Rohit Vashisht et.al. Reversible speech dysfunction in a case of Hashimoto's encephalopathy: a rare manifestation. *Int J Health Sci Res.* 2023; 13(6):136-139.
DOI: *https://doi.org/10.52403/ijhsr.20230624*

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