CASE REPORT

Anti-Thyroid Peroxidase/Anti-Thyroglobulin Antibody-Related Neurologic Disorder Responsive to Steroids Presenting with Pure Acute Onset Chorea

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Background: Pure acute onset chorea without encephalopathy has rarely been reported in anti-thyroid peroxidase (anti-TPO)/anti-thyroglobulin (anti-TG) antibody-related neurologic disorders responsive to steroids (ATANDS).

Case report: We report a 16-year-old female who presented with acute chorea without encephalopathy. Anti-TPO antibodies were found to be strongly positive (>1200 IU/ml) along with anti-thyroglobulin and anti-thyroid stimulating hormone receptor antibodies. After pulse intravenous methylprednisolone therapy (1 g/day for five consecutive days), all the movements seized, and she was discharged with oral prednisolone 30 mg/day with gradual tapering over next three months. After one year of follow-up, she is stable, drug-free, and never had any other problems.

Discussion: Anti-thyroid antibodies testing should be included in routine/conventional panel that is done for elucidating causes of chorea as ATANDS can be easily missed and is treatable with widely available, relatively low-cost drugs like steroids with a promising outcome.

Keywords: Anti-thyroid peroxidase/anti-thyroglobulin antibody-related neurologic disorders responsive to steroids; Anti-thyroid peroxidase antibodies; Anti-thyroglobulin antibodies; Autoimmune; Chorea; Movement Disorders; Review; Steroid-responsive encephalopathy associated with autoimmune thyroiditis

In autoimmune thyroid disorders, albeit known to affect only 1% of the population, focal or sub-clinical autoimmune thyroid inflammation can be found in around 15% of biochemically euthyroid population [1–3]. Anti-thyroid peroxidase (anti-TPO) and anti-thyroglobulin (anti-TG) antibodies are considered diagnostic markers of autoimmune thyroid disorders [1]. Neurological manifestations associated with autoimmune thyroid disorders have been frequently under-documented in literature [4], being the most protean amongst these disorders Hashimoto’s encephalopathy [5–7]. Spectrum of this disorder an range from subtle behavioral/personality changes to movement disorders, seizures, dementia, encephalopathy, stroke, coma and death [5–8]. Patients can also present with movement disorders without encephalopathy and cognitive impairment [5–7]. There is no pathognomonic clinical, serological, biochemical, electrophysiological or imaging markers [5–7]. In addition, there are no good predictors of treatment response to steroids; [9] in fact, in a recent study [9], only 31% patients completely responded to these drugs. Similarly, in other studies, only 56% and 36% patients with suspected Hashimoto’s encephalopathy responded to...
steroids [10, 11]. Despite this, response to steroids seems to be the only partially consistent feature of this disorder hence renamed as “steroid responsive encephalopathy associated with autoimmune thyroiditis” (SREAT) [12], but neither response to steroids nor association with thyroiditis is steadfast [13]. Termasarasab et al., [14] have recently proposed “anti-TPO/TG antibody-related neurologic disorders responsive to steroids (ATANDS)” to be the renamed entity that would include the complete spectrum. Reported movement disorders that have been associated with ATANDS can be either “encephalopathic” or “non-encephalopathic” [5–7, 14].

We had treated a patient with pure acute chorea who rapidly improved with corticosteroids. Here, we describe the case with a complete report. We also provide a review of the literature, which was performed to collect and summarize the present state of knowledge on movement disorders associated with ATANDS.

Case presentation
A 16-year-old female presented to the neurology outpatient department with complaint of acute onset involuntary weird and quirky movements of all four limbs for last four days, which were irregular, asymmetric, rapid, unpredictable, purposeless, jerky and flowing from distal to proximal and that disappeared completely during sleep. Her past medical history was unremarkable. No associated febrile episode, seizure, headache, visual disturbances, behavioral changes, personality changes, forgetfulness, attention problems, or self-care inadequacy were noted. She had no history of any drug intake for any disease or substance abuse in recent past. No history was suggestive of any connective tissue disorder or thyroid dysfunction. Nobody in family had any neurological disease. On completion of an unremarkable general survey, detailed neurological examination revealed generalized chorea involving all extremities (right > left) with classic Jack in the box tongue and Milkman’s grip signs. Precise and meticulous cognitive assessment failed to unveil any impairment. Neither motor weakness, nor sensory deficits, nor signs of meningeal irritation and cranial nerve deficits were noted. Slit lamp examination ruled out Kayser-Fleischer ring.

No cognitive domain seemed to be affected and family history was negative; therefore, Huntington’s disease, Huntington’s disease-like syndromes, dentatorubral-pallidolysian atrophy, and deposition disorders were virtually excluded. Hence, working diagnosis kept was acute onset generalized chorea without cognitive impairment. Differentials considered were: 1) metabolic chorea, 2) rheumatic chorea, 3) dysthyroidism associated chorea, 4) autoimmune chorea, 5) vascular chorea, and 6) chorea gravidarum.

Complete hemogram, thyroid, liver, kidney functions, electrolytes, arterial blood gas analysis and HbA1C were normal. A urine beta human chorionic gonadotropin and abdominal ultrasound ruled out any pregnancy. Serologies for human immunodeficiency virus, hepatitis C, and hepatitis B were negative. 24 hours urinary copper and serum ceruloplasmin levels were within normal range. Echocardiography, serial anti-streptolysin O titers, and anti-DNase B antibodies levels ruled out possibility of Sydenham’s chorea. Anti-Nuclear Antibody (ANA) screening using HEp-2 cells, ANA profile, antiphospholipid antibodies, and antineutrophil cytoplasmic antibodies (cANCA and pANCA) were found to be negative. Autoantibodies directed against voltage gated potassium channel and anti-N-methyl-D-aspartate receptor antibodies were also negative. Magnetic resonance imaging of brain, electroencephalogram and cerebrospinal fluid analyses were otherwise normal.

Serum anti-TPO antibodies were found to be strongly positive (>1200 IU/ml) along with anti-TG and anti-thyroid stimulating hormone receptor antibodies. Patient was put on pulse intravenous methylprednisolone therapy (1 g/day for five consecutive days). All the movements seized and she was discharged with oral prednisolone 30 mg/day with gradual tapering over next three months. Tests were rerun with similar results, but anti-thyroid stimulating hormone receptor antibodies, which were within normal range this time around. At present, after one year of follow-up, she remains stable, drug-free and without any other problems.

Discussion
ATANDS with associated movement disorders have been described previously (Table 1) [15–50]. We have reported a 16-year-old female with ATANDS who presented with acute pure chorea without encephalopathy. ATANDS presenting with chorea is exceedingly rare. For example, Miranda et al., [47] described a middle-aged female with acute onset rapidly worsening choreo-athetosis with dystonia and slurred speech which came out to be a case of ATANDS. Sharan A et al., [41] reported an aged female with ATANDS, who developed abrupt onset behavioral changes along with asymmetric florid chorea. Taurin G et al., [19] narrated behavioral abnormality with psychotic features along with bilateral and axial choreic movements in an elderly female. Our patient had no behavioral abnormalities or any other extrapyramidal or cerebellar features unlike those previously mentioned cases [19, 41, 47]. In all those cases steroid resulted in good yield alike our patient [19, 41, 47].

Etiopathogenetic factors for chorea are believed to be a) a hypersensitivity of dopaminergic receptors to dopamine due to a thyrotoxic state; [17, 51] b) derangements in cerebral perfusion as reported in cases of acute onset chorea associated with other etiologies [27, 52, 53] and substantiated by single-photon emission computed tomography and positron emission tomography imaging; [54, 55] c) autoimmune central nervous system vasculitis; [8, 56] and d) anti-thyroid antibody mediated effects on neurons [7, 57–59]. However, for neurological manifestations like chorea, anti-thyroid antibodies are extremely sensitive, but lack specificity [60]. And whether they are pathogenic or just a marker...
### Table 1: Movement disorders associated with anti-TPO/TG antibody-related neurologic disorders responsive to steroids.

| Author and year of publication | Age/Sex | Type of movement disorder | Thyroid status | Anti-thyroid antibody | Neuroimaging | Treatment | Outcome |
|-------------------------------|---------|----------------------------|----------------|-----------------------|--------------|-----------|---------|
| 1. Mehta AB et al., [15] 1981 | 17/F    | Torsion dystonia           | Primary hyperthyroidism/thyrotoxicosis | Anti-TG+ Anti-TPO+ | No data available | Carbimazole and radioactive iodine | Euthyroid, dystonia was very mild with a slight tendency for torticollis and scoliosis to the right, and for the right outstretched arm to hyperpronate, provided drug compliant |
| 2. Javaid A and Hilton DD [16] 1988 | 15/F    | Generalized choreoathetosis | Primary hyperthyroidism | Anti-TG+ Anti-TPO+ | No data available | Propranolol, carbimazole, tetrabenazine, chlorpromazine, and haloperidol | Refractory chorea. Chlorpromazine and tetrabenazine only partially suppressed it. At six months it was persisting. Haloperidol almost completely abolished chorea. It returned whenever she stopped taking haloperidol. Recurrence occurred 16 months after she first presented |
| 3. Baba M et al., [17] 1992 | 23/F    | Hemichorea                 | Primary hyperthyroidism/Graves’ disease | Anti-TG+ Anti-TPO- | No changes | Metoprolol, thiamazole and chlorpromazine | Improved |
| 4. Hernández Echebarría LE et al., [18] 2000 | 41/F    | Opsoclonus, myoclonus, and gait ataxia | Euthyroid → subclinical hypothyroidism | Anti-TPO+ | SPECT showed decreased perfusion in the left fronto-parietal region and in the right basal ganglia | Antibiotics, acyclovir and valproate followed by L-thyroxin and steroids | At one-year follow-up, CSF analysis, SPECT, and electroencephalogram were normal. Anti-TPO decreased. She remained well at the last visit, two years after the onset of neurologic symptoms. |
| 5. Taurin G et al., [19] 2002 | 77/F    | Bilateral and axial choreic movements | Primary hypothyroidism | Anti-TG+ Anti-TPO+ | No changes | Venous anomaly in hypothalamus | With 60 mg/day of prednisolone, chorea disappeared and reappeared again; on increasing dose to 80 mg/d, it disappeared. At 3 weeks, the patient was clinically normal. No relapse during 8 months of follow-up |
| 6. Erickson JC et al., [20] 2002 | 34/M    | Myorhythmia, myoclonus, and tremor | Primary hypothyroidism | Anti-TG+ Anti-TPO+ | No changes | IVMP followed by oral prednisolone | Moderate improvement with residual mild cognitive impairment and subtle facial myorhythmia |
| 7. Erickson JC et al., [20] 2002 | 38/M    | Palatal tremor             | Euthyroid          | Anti-TPO+ Anti-TG- | No changes | IVMP followed by oral prednisolone | Moderate improvement in seizures, but cognitive impairment persisting |

(Contd.)
| Author and year of publication | Age/Sex | Type of movement disorder | Thyroid status | Anti-thyroid antibody | Neuroimaging | Treatment | Outcome |
|--------------------------------|---------|--------------------------|----------------|----------------------|-------------|-----------|---------|
| Nagpal T and Pande S [21] 2004 | 52/F    | Parkinsonism and myoclonus | Subclinical hypothyroidism | Anti-TPO+ Anti-TG- | Cerebral atrophy | IVMP followed by oral prednisolone, PLEX, and finally by oral prednisolone | No improvement with IVMP, significant improvement 10 days after PLEX |
| Loh LM et al., [22] 2005       | 40/M    | Propriospinal or segmental myoclonus, Spasmatic truncal flexion | Primary hyperthyroidism/Graves’ disease | TRAB+ Anti-TG+ TSI+ Anti-TPO | No changes | Clonazepam and propylthiouracil | Euthyroidism established and symptoms improved |
| Tan EK et al., [23] 2006       | Middle aged/M | Bilateral postural hand tremor and task-specific dystonia-writer’s cramp | Primary hyperthyroidism/Graves’ disease | Anti-TG+ | No changes | Carbimazole | Euthyroidism achieved and symptoms improved |
| Guimaraes J et al., [24] 2007 | 60/M    | Painful legs and moving toes syndrome, bradykinesia, and dystonia | Primary hypothyroidism | Anti-TG+ Anti-TPO+ | Subcortical white matter lesions | Oral prednisolone | No improvement |
| Tan EK et al., [25] 2008       | 50/F    | Isolated orthostatic tremor | Primary hyperthyroidism/Graves’ disease | TRAB+ Anti-TG+ Anti-TPO+ | No changes | Carbimazole | Complete resolution |
| Ku CR et al., [26] 2008        | 42/F    | Generalized chorea | Primary hyperthyroidism/Graves’ disease | TRAB+ Anti-TG- Anti-TPO+ | No changes | IVMP, propylthiouracil, propranolol, trihexyphenidyl, ropinirole, donepezol and quetiapine | Improved |
| Yu JH and Weng YM [27] 2009    | 17/F    | Chorea | Primary hyperthyroidism/Graves’ disease | Anti-TPO+ | SPECT revealed decreased perfusion to the right anterior temporal cortex | Propylthiouracil and propranolol | Complete resolution |
| Broch L and Amthor KF [28] 2010 | 66/F   | Myoclonus, tremor | Euthyroid | Anti-TPO++ | No changes | Systemic steroids | Improved |
| Salazar R et al., [29] 2012    | 59/M    | Opsoclonus and gait ataxia | Euthyroid | Anti-TG+ Anti-TPO+ | No changes | IVIG/IVMP | After three months of therapy with corticosteroids improved, but not with IVIG |
| Liu MY et al., [30] 2012       | 75/M    | Paroxysmal kinesigenic dyskinesia | Unknown | Anti-TPO+ | No changes | IVMP followed by oral prednisone taper | Back to baseline in 20 days |

(Contd.)
| Author and year of publication | Age/Sex | Type of movement disorder | Thyroid status | Anti-thyroid antibody | Neuroimaging | Treatment | Outcome |
|--------------------------------|---------|--------------------------|----------------|---------------------|-------------|-----------|---------|
| Inoue K et al., [31] 2012      | 63/F    | Micrography, parkinsonian gait and tremor | Euthyroid | Anti-TPO+ | White matter ischemic changes | IVMP followed by oral prednisolone | Improved |
| Ryan SA et al., [32] 2012      | 48/M    | Myoclonus | Subclinical hypothyroidism | Anti-TPO++ Anti-TG- | No changes | Oral prednisolone | Improved |
| Park J et al., [33] 2012       | 16/M    | Asymmetric chorea | Primary hyper-thyroidism/Graves’ disease | TRAB+ Anti-TG+ | No changes | Propylthiouracil and propranolol | Improved |
| Nakavachara P et al., [34] 2013 | 14/M   | Choreo-athetosis | Primary hyper-thyroidism/Graves’ disease | Anti-TPO+ Anti-TG+ | No data available | Methimazole, propranolol, and IV potassium | Improved |
| Kaminska A et al., [35] 2013   | 23/F    | Hemi-chorea | Primary hyper-thyroidism/Graves’ disease | TRAB+ Anti-TPO+ | No changes | Thiamazole, prednisolone, haloperidol, thioridazine | Subsidence of symptoms |
| Ghoreishi E et al., [36] 2013   | 32/M    | Palatal myoclonus | Euthyroid | Anti-TG+ Anti-TPO+ | Bilateral striatal hyperintensity on T2WI | Oral prednisolone | Improved |
| Philip R et al., [37] 2014     | 18/M    | Myoclonus and tremor | Primary hyper-thyroidism | Anti-TPO+ | Non-specific white matter changes and pituitary hyperplasia | IVMP followed by oral prednisolone and L-thyroxin | Significant improvement |
| Saygi S et al., [38] 2014      | 12/M    | Motor tics | Euthyroid | Anti-TPO++ Anti-TG+ | No changes | Oral prednisolone | Improved |
| Rozankovic PB et al., [39] 2015 | 27/F    | Myoclonus-Dystonia and choreo-atetosis | Euthyroid | Anti-TPO+ | No changes | IVMP followed by oral Prednisolone | Complete resolution |
| Lee HJ et al., [40] 2015        | 30/M    | Ocular flutter, limb and gait ataxia, myoclonus, and truncal titubation | Euthyroid | Anti-TG+ Anti-TPO- | No changes | IVMP followed by oral prednisolone | Improved |
| Sharan A et al., [41] 2015      | 78/F    | Choreiform movements | Euthyroid | Anti-TPO+ | Atrophy | IVMP followed by oral prednisolone | Improved |
| Sheetal SK et al., [42] 2016    | 66/M    | Action-myoclonus, parkinsonism (corticobasal disease-variant-like) | Euthyroid | Anti-TPO+ Anti-TG- | Small right thalamic hematoma | IVMP followed by oral prednisolone | Improved |

(Contd.)
| Author and year of publication | Age/Sex | Type of movement disorder | Thyroid status | Anti-thyroid antibody | Neuroimaging | Treatment | Outcome |
|-------------------------------|---------|---------------------------|----------------|----------------------|--------------|-----------|---------|
| 30. Ramcharan K et al., [43] 2016 | 34/F | Bilateral hand postural tremor | Euthyroid | Anti-TG+ TRAB+ Anti-TPO- | No changes | Oral prednisolone | Improved |
| 31. Correia I et al., [44] 2016 | 61/F | Limb myoclonus | Primary hypothyroidism | Anti-TPO+ | SPECT revealed hypoperfusion in frontal, temporal, and parietal regions with left predominance | L-thyroxin, IVMP followed by oral prednisolone and azathioprine | Resolution |
| 32. Kelly DM et al., [45] 2017 | 64/M | Abdominal tremor and abdominal wall dyskinesia | Primary hyperthyroidism | Anti-TPO+ | No changes | Carbimazole and supportive | Complete resolution |
| 33. Keshavaraj A and Anpalagan J [46] 2018 | 23/F | Oro-lingual dyskinesia | Primary hyperthyroidism | Anti-TPO+ TRAB+ | No changes | IVMP, carbimazole, and propranolol | Significant improvement |
| 34. Miranda M et al., [47] 2018 | 34/F | Choreo-athetosis, dystonia and ataxia | Euthyroid | Anti-TG+ Anti-TPO+ | No changes | IVMP and IVIG | Complete resolution |
| 35. Miranda M et al., [47] 2018 | 61/M | Myoclonus-dystonia | Euthyroid | Anti-TPO+ | No changes | IVMP, IVIG, PLEX and rituximab | Incomplete resolution |
| 36. Mohd Fauz NA et al., [48] 2019 | 50/F | Relapsing-remitting opsoclonus-myoclonus-ataxia syndrome | Subclinical hyperthyroidism | Anti-TG+ Anti-TPO+ | Lesions in cortical and subcortical regions, pons and midbrain | IVMP and PLEX | Improved |
| 37. Delhasse S et al., [49] 2019 | 60/F | Complex dyskinesia, high-amplitude myoclonic jerks, mild chorea, and postural tremor | Primary hyperthyroidism / Graves’ disease | TRAB+ Anti-TG+ Anti-TPO+ Isotope scan+ | No changes | Carbimazole, propylthiouracil, radioactive iodine | Complete resolution |
| 38. Shree S et al., [50] 2020 | 60/F | Tremor, myoclonus and catatonia | Euthyroid | Anti-TPO+ | No changes | IVMP followed by oral prednisolone | Improved |

F: female; M: male; Anti-TPO: anti-thyroid peroxidase antibody; Anti-TG: anti-thyroglobulin antibody; TSI: thyroid stimulating immunoglobulin; TRAB: TSH receptor antibody; CSF: cerebrospinal fluid; SPECT: Single-photon emission computed tomography; IV: intravenous; MP: methylprednisolone; IG: immunoglobulin; PLEX: plasma exchange; MRI: magnetic resonance imaging; ‘+’: high/positive titer; ‘-’: low/negative titer.
of the disease or just an epiphenomenon, remains elusive [6, 7, 60, 61]. Presence of anti-thyroid antibodies in general population is well established even in absence of neurologic disorders and usually acting as a confounding factor in diagnosis [60]. Further, levels of anti-thyroid antibodies do not correlate well with disease severity and often persist in high levels after the treatment and clinical response [6, 62, 63].

In conclusion, our experience with the current case and our review of the literature strongly suggest that ATANDS/SREAT can rarely present with movement disorders alone and may be a definite separate entity. Anti-thyroid antibody testing should be included in routine/conventional panel that is done for elucidating causes of chorea as this disorder can be easily missed and is treatable with widely available, relatively low-cost drugs like steroids with a promising outcome.

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**Competing Interests**
The authors have no competing interests to declare.

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