ORIGINAL RESEARCH PAPER
Volume - 13 | Issue - 08 | August - 2024 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

THYROID STORM IN A SETTING OF ACUTE ISCHEMIC STROKE- A RACE AGAINST TIME

Internal Medicine

Dr Sona Mitra* Consultant Physician, Department of General Medicine, PIMSR, Parul University, Waghodia, Gujarat*Corresponding Author

Dr. Arti Muley Professor & HOD, Department of General Medicine, PIMSR, Parul University, Waghodia, Gujarat

Dr. Kuldeep Viramgama Junior Resident, Department of General Medicine, PIMSR, Parul University, Waghodia, Gujarat

Dr. Parth Patel Junior Resident, Department of General Medicine, PIMSR, Parul University, Waghodia, Gujarat

ABSTRACT

Thyroid (or thyrotoxic) storm is a rare syndrome of acute exacerbation of thyrotoxicosis that occurs in association with increased levels of circulating thyroid hormones. Many studies have reported thrombotic events in patients of hyperthyroidism, and suggested few mechanisms like embolism due to atrial fibrillation, role of autoantibodies and hypercoagulable state. However, there is lack of larger analytical studies and recommendation of treatment strategy in such cases. We present a case of 43- year-old Indian male patient with untreated hyperthyroidism presenting with thyroid storm and neurological deficits due to ischemic stroke to highlight the need of regular treatment and prevention of atherosclerosis in such cases.

KEYWORDS

Thyroid storm, Ischemic stroke, Hypercoagulable state

Introduction:

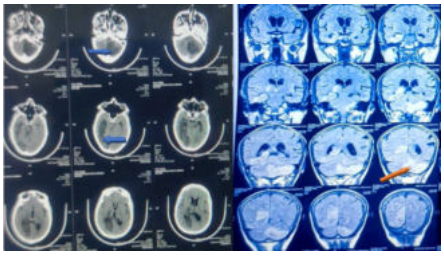
Thyroid (or thyrotoxic) storm is a rare syndrome of acute exacerbation of thyrotoxicosis that occurs in association with increased levels of circulating thyroid hormones. [1,2] It is a life-threatening medical emergency with an overall mortality as high as 8% to 25% despite modern advancements in treatment and supportive measures. [3] Common precipitating factors include infection, surgery, trauma, excess iodine intake and medication non-compliance. [3-4] The clinical presentation of thyroid storm includes high grade fever, profuse sweating, tachycardia, irritability, anxiety, delirium, nausea, vomiting, and diarrhea. In severe cases, heart failure, shock, and coma may be present [4-5].

Thyroid storm coexisting with ischemic stroke is a rare presentation. Ischemic stroke can either act as the acute illness that precipitates the thyroid storm or can be the direct result of the storm. Thyroid hormone dysregulation is known to affect the coagulation pathway which may itself increase the risk of ischemic and embolic events.[6] There is, however, no clear recommendation for anticoagulation therapy in patients experiencing thyroid storm. This case report describes a 43-year-old Indian male patient with untreated hyperthyroidism presenting with neurological deficits who developed thyroid storm post hospitalisation.

Case: A 43-year-old thin built male patient with history of irregularly treated hyperthyroidism, hypertension and Type II Diabetes mellitus was brought to the Emergency department of our institute with history of altered sensorium, left sided weakness, difficulty in swallowing and dysarthria since 2-3 days. There was no history of fever, headache, tingling or numbness or root pain, sweating, nausea, vomiting or diarrhoea. Upon initial evaluation patient was conscious, but irritable. He had fever (Temp - 99.6F), tachycardia (Heart rate-125 beats/min) and elevated blood pressure (170/100mmHg). On systemic examination, patient was thin built, tone was increased in left upper and lower limb. Power was normal on right side while it was zero on the left side. Coordination couldn't be checked. Corneal reflex, Jaw jerk and Gag reflex all were normal. Right sided pupil was 2 mm reactive to light and left sided pupil 2 mm sluggishly reactive to light. Planter reflex on right side was flexor and on left side was extensor. Deep tendon reflexes were exaggerated on left side. On sensory system examination all sensations were found to be normal. There was no nystagmus or ptosis. Cranial nerve examination also did not reveal any abnormality. Respiratory system examination revealed bibasilar crepts on auscultation. Rest of the systemic examination was within normal limits.

All routine blood investigations were sent and the patient was shifted to ICU for further management. CT scan of head revealed ill defined hypodensity involving right occipito-temporal lobe, ganglio-capsular region, right thalamus with left cerebellar hemisphere and vermis with no signs of haemorrhage. (Figure 1A) MRI Brain showed acute infarct in right Posterior cerebral artery (PCA) territory and left Superior cerebellar artery (SCA) territory. (Figure 1B) Dual antiplatelet therapy, statins and antihypertensives were started.

Figure 1: A. CT Brain without Contrast- ill defined hypodensity involving right occipito-temporal lobe and left cerebellar hemisphere. B. MRI Brain-Right PCA territory & Left SCA territory infarct.



Complete blood count, renal function tests, liver function tests and lipid profile were all normal. (Hemoglobin-13.4g/L, Leucocyte count-10400 cells/cmm, platelet count-3,16,000 cells/cmm. Blood urea-56 mg/dL, S. Creatnine-0.9mg/dL). However, he had hypernatremia, uncontrolled blood sugar and altered thyroid levels. (S. Sodium - 155mg/dl, S. Potassium-3.5mg/dl, HbA1C-10.4, Free T3-8.37, Free T4-256, TSH-0.01). ECG showed sinus tachycardia, Chest X ray was suggestive of pulmonary edema and 2D Echo showed concentric left ventricular hypertrophy with EF of 55% and grade 1 LV diastolic dysfunction. Her Burch and Wartofsky score was > 45 which was indicative of thyroid storm. Hence, methimazole 20 mg every four hours and diltiazem 60 mg thrice daily were started. In addition, glucocorticoids (IV Hydrocortisone 100 mg thrice daily), Potassium iodide 5 drops (50 mg iodide/drop [0.05 mL]) orally every six hours and antipyretics were also started. He was closely monitored thereafter to look for any further deterioration of neurological status. On the very next day, patient developed atrial fibrillation (AF) (Rate-150/min) with high grade fever (104F) and tachypnoea (RR-30/min). Atrial fibrillation converted to normal sinus rhythm and the patient became afebrile within 24 hours; however, he remained persistently tachycardic. Thus, the ongoing treatment was continued with observation.

On the Day 5 of admission neurological status of the patient deteriorated and oxygen saturation dropped to 90% at room air. Patient became drowsy and was arousable only to deep painful stimulus. Hence the patient was electively intubated. Repeat CT head showed increase in size of infarct with mass effect causing midline shift in posterior fossa. He was taken for Emergency Decompression Craniotomy. Post operative status of the patient remained guarded where the patient needed multiple inotropic support. On day 6, patient went into cardiorespiratory arrest and succumbed soon after.

Discussion: Thyroid storm, also known as thyrotoxic crisis, is an acute, life-threatening complication of hyperthyroidism, with sudden multisystem involvement. Thus, it is very important to recognize it early and start aggressive treatment to reduce mortality. Literature suggests that thyroid storm occurs in no more than 1%–2% of hospitalized thyrotoxic patients [7], but its incidence in a large population has not been fully investigated.

Timely diagnosis of thyroid storm is challenging and is often missed due to non-specific symptoms, which resembles an accentuated catabolic state. The diagnosis of thyroid storm is based on clinical findings of hyperthyroidism accompanied with manifestations of multi-organ failure. Diagnosis can further be supported by thyroid function tests with very low TSH and elevated free T3 and T4 levels. [1-3] High grade fever, cardiovascular involvement, gastrointestinal manifestations and central nervous system manifestations are common. Fever of 104° F to 106°F with sweating is a key presenting feature. Cardiovascular manifestations include tachycardia of more than 140 beats/minute, heart failure with pulmonary edema, hypotension, arrhythmia and death from cardiac arrest. Gastrointestinal (GI) symptoms include nausea, vomiting, diarrhea, abdominal pain, intestinal obstruction, and acute hepatic failure. Central nervous system involvement includes agitation, anxiety, delirium, psychosis, or coma. Physical examination findings may include high temperature, tachycardia, systolic hypertension, orbitopathy, enlarged thyroid gland (Goitre), hand tremors, moist and warm skin, hyperreflexia, and jaundice. [1,2,7]

Ischemic stroke presenting with thyroid storm is a rare occurrence. Ischemic stroke as a result of thyroid storm has two possible mechanisms: atrial fibrillation and hypercoagulable state. Sinus tachycardia is the most common electrocardiographic finding in thyrotoxicosis, but atrial fibrillation can also occur in 5-15% cases. [8] Atrial fibrillation is a well-known risk factor for stroke and occurs in 10% to 35% of thyrotoxicosis patients with an increased risk in older patients above the age of 60 years. [8.9] Thyrotoxicosis also promotes a hypercoagulable state due to a shortened activated partial thromboplastin time, increased fibrinogen levels, and increased factor VIII and factor X activity [6], predisposing a patient to stroke regardless of heart rhythm. A recent study by Antonijevic N et al. [10] on 64 hyperthyroid patients found that high levels of factor VIII, vWF, fibrinogen, PAI-1, and anticardiolipin antibodies along with other hemostatic factors contribute to the presence of a hypercoagulable state in patients with hyperthyroidism. In addition, in recent years, some studies have highlighted that the immune responses are profoundly involved in the progression of endothelial dysfunction and arteriosclerosis [11]. Several cases have provided evidence for the association between thyroid diseases and arterial damage [12,13]. A study showed the correlation between hyperthyroidism and intracranial arterial stenosis in stroke patients [14]. In addition, thyroid dysfunction and positive thyroid autoantibodies were shown in pediatric patients with moyamoya disease [15]. It is unclear whether thyroid dysfunction or positive thyroid autoantibodies were associated with vascular diseases.

Although these mechanisms have been documented, there are no specific or clear-cut recommendations regarding anticoagulation therapy in patients with thyrotoxicosis.

A few case reports/series have documented the association between thyrotoxicosis and ischemic stroke, although no large randomized controlled trials have assessed it. Alex Gonzalez-Bossolo et al. [16] reported a case of 53- year-old Hispanic man who experienced a left middle cerebral artery (MCA) stroke and global aphasia with coexisting thyroid storm. In this case, the fever and palpitations preceded the development of paralysis, confirming that thyrotoxicosis lead to ischemic stroke. Snyder S. et al [17], reported a case of 63 year old male patient with left sided weakness and dysarthria. This patient was a known case of hyperthyroidism on irregular treatment which could have precipitated thyroid storm. Yuen et al.[18] reported in their study of 210 subjects with thyrotoxicosis, 21 (10%) had atrial fibrillation, of which 24% of the patients had developed systemic emboli. A recent prospective cohort study from Taiwan, 3176 patients with hyperthyroidism were followed for 5 years and compared with subjects without hyperthyroidism. They found that the risk for developing a stroke was 1.44 times greater in the hyperthyroidism cohort compared with the control group after adjusting for several confounders [19]. However, larger randomized controlled studies may be needed for identifying specific risk factors for development of atherosclerotic events in cases of hyperthyroidism, it is apparent that regular treatment of hyperthyroidism with high index of suspicion and regular measures for prevention of atherosclerotic events may be recommended for patients with hyperthyroidism

Conclusion: Ischemic stroke can either act as the acute illness precipitating the thyroid storm or can be the direct result of the storm. An RCT is required to identify risk factors for development of ischemic events, but in view of the reported studies, regular treatment and prevention of atherosclerosis may be a meaningful strategy to reduce ischemic events in hyperthyroid patients.

REFERENCES:

1. Chiha, M., Samarasinghe, S., & Kabaker, A. S. (2013). Thyroid storm. Journal of Intensive Care Medicine, 30(3), 131–140.
2. De Almeida, R., McCalmon, S., & Cabandugama, P. K. (2022). Clinical review and update on the management of thyroid Storm. Mo Med, 119(4), 366–371.
3. Ross, D. S., Burch, H. B., Cooper, D. S., Greenlee, M. C., Laurberg, P., Maia, A. L., Rivkees, S. A., Samuels, M., Sosa, J. A., Stan, M. N., & Walter, M. A. (2016). 2016 American Thyroid Association Guidelines for Diagnosis and Management of Hyperthyroidism and Other Causes of Thyrotoxicosis. Thyroid : official journal of the American Thyroid Association, 26(10), 1343–1421.
4. Ylli, D., Klubo-Gwiezdzinska, J., & Wartofsky, L. (2019). Thyroid emergencies. Polish archives of internal medicine, 129(7-8), 526–534.10.20452/pamw.14876
5. Angell, T. E., Lechner, M. G., Nguyen, C. T., Salvato, V. L., Nicoloff, J. T., & LoPresti, J. S. (2015). Clinical features and hospital outcomes in thyroid storm: a retrospective cohort study. The Journal of clinical endocrinology and metabolism, 100(2), 451–459..
6. Franchini, M., Montagnana, M., Manzato, F., & Vescovi, P. P. (2009). Thyroid dysfunction and hemostasis: an issue still unresolved. Seminars in thrombosis and hemostasis, 35(3), 288–294.
7. Pokhrel, B., Aiman, W., & Bhusal, K. (2022). Thyroid Storm. In StatPearls. StatPearls Publishing.
8. Chen, Z. C., Wu, N. C., Chang, C. L., Ho, C. H., Liao, C. T., Chiang, C. Y., & Chang, W. T. (2019). Risk of ischaemic stroke in thyrotoxic atrial fibrillation. Clinical endocrinology, 91(4), 561–570.
9. Goldstein, S. A., Green, J., Huber, K., Wojdyla, D. M., Lopes, R. D., Alexander, J. H., Vinereanu, D., Wallentin, L., Granger, C. B., & Al-Khatib, S. M. (2019). Characteristics and Outcomes of Atrial Fibrillation in Patients With Thyroid Disease (from the ARISTOTLE Trial). The American journal of cardiology, 124(9), 1406–1412.
10. Antonijevic, N., Matic, D., Beleslin, B., Mikovic, D., Lekovic, Z., Marjanovic, M., Uscumlic, A., Birovljev, L., & Jakovljevic, B. (2024). The Influence of Hyperthyroidism on the Coagulation and on the Risk of Thrombosis. Journal of clinical medicine, 13(6), 1756
11. Yoshida, Y., Hiwasa, T., Machida, T., Kobayashi, E., Mine, S., Matsushima, J., Takiguchi, M., & Iwadate, Y. (2018). Elevation of Autoantibody in Patients with Ischemic Stroke. Neurologia medico-chirurgica, 58(7), 303–310.
12. Gini, B., Lovato, L., Cianti, R., Cecotti, L., Marconi, S., Anghileri, E., Armini, A., Moretto, G., Bini, L., Ferracci, F., & Bonetti, B. (2008). Novel autoantigens recognized by CSF IgG from Hashimoto's encephalitis revealed by a proteomic approach. Journal of neuroimmunology, 196(1-2), 153–158.
13. Moodley, K., Botha, J., Raidoo, D. M., & Naidoo, S. (2011). Immuno-localisation of anti-thyroid antibodies in adult human cerebral cortex. Journal of the neurological sciences, 302(1-2), 114–117.
14. Zhang, X., Chen, Z., Shi, Z., & Lou, M. (2012). Correlation between thyroid autoantibodies and intracranial arterial stenosis in stroke patients with hyperthyroidism. Journal of the neurological sciences, 318(1-2), 82–84.
15. Li, H., Zhang, Z. S., Dong, Z. N., Ma, M. J., Yang, W. Z., Han, C., Du, M. M., Liu, Y. X., Yang, H., Liu, W., Duan, L., & Cao, W. C. (2011). Increased thyroid function and elevated thyroid autoantibodies in pediatric patients with moyamoya disease: a case-control study. Stroke, 42(4), 1138–1139.
16. Gonzalez-Bossolo, A., Gonzalez-Rivera, A., & Coste-Sibilia, S. (2016). Cerebrovascular Accident due to Thyroid Storm: Should We Anticoagulate?. Case reports in endocrinology, 2016, 5218985.
17. Snyder, S., & Joseph, M. (2020). The Perfect Storm: A Case of Ischemic Stroke in the Setting of Thyroid Storm. Cureus, 12(5), e7992.
18. Yuen, R. W., Gutteridge, D. H., Thompson, P. L., & Robinson, J. S. (1979). Embolism in thyrotoxic atrial fibrillation. The Medical journal of Australia, 1(13), 630–631.
19. Sheu, J. J., Kang, J. H., Lin, H. C., & Lin, H. C. (2010). Hyperthyroidism and risk of ischemic stroke in young adults: a 5-year follow-up study. Stroke, 41(5), 961–966.