All of them progressed well. The patient who died had already severe prior comorbidities. We performed a systematic survey on PubMed of English articles (case reports and reviews) in humans and we analyzed our 5 cases along with the 108 articles about the use of plasmapheresis in thyroid storm from 1970 to 2020 and we compare them to 394 ones of conventional treatments in past 10 years. Our objective was to evidence plasmapheresis is not related to a higher mortality of patients who underwent to it. We found 7% of mortality in both groups. The chi square test showed an Odds Ratio of (CI 95%) = 1,091 reinforcing there is no relation between number of deaths and treatment type. **Conclusion:** Plasmapheresis is a therapeutic option with few reports in the literature and without clear guidelines about indication criteria or better timing to initiate it. The statistical analysis showed that 3 or more organ dysfunctions in thyroid storm are related to higher death rates. Its early employment within 24 hours of the initial symptoms and the prompt normalization of free T4 are related to lower mortality. It is a safe and effective therapy that allows thyroid storm patients to be compensated to receive definitive treatment with lower chances of death. **Reference:** Ono Y, Ono S, Yasunaga H, Matsui H, Fushimi K, Tanaka Y. Factors Associated With Mortality of Thyroid Storm: Analysis Using a National Inpatient Database in Japan. *Medicine (Baltimore)*. 2016;95(7):e2848.

Thyroid

THYROID DISORDERS CASE REPORT

Plasmapheresis as First Line Therapy for Thyrotoxicosis in a Critically Ill Patient

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Introduction: The role of plasmapheresis (TPE) in thyrotoxicosis management is not well established. Its use may be determined on an individualized basis (1). We report a case of a critically ill patient where TPE was utilized as first-line therapy for refractory thyrotoxicosis. **Clinical Case:** A 33-year-old woman with Graves' disease complicated by medication non-adherence presented with rapidly ascending paralysis and bulbar weakness. Primary work up was consistent with acute inflammatory demyelinating polyneuropathy (AIDP) based on EMG findings of motor fiber polyneuropathy with demyelinating features. Laboratory evaluation revealed uncontrolled hyperthyroidism (TSH <0.05 uU/mL, N 0.3-4.2 uU/mL; fT4 3.9 ng/dL, N 0.6-1.5 ng/dL; tT3 318, N 60-160 ng/dL). Initially, there was low concern for thyrotoxicosis based on a Burch-Wartofsky score of 15 (2). Standard dose methimazole and aggressive beta-blockade were initiated. Hospital course was complicated by hypoxic respiratory failure due to progressive paralysis requiring intubation and septic shock from Klebsiella pneumonia requiring initiation of pressors and broad-spectrum antibiotics. Biochemical evaluation showed increasing fT4 (3.8 ng/dL) and tT3 (419 ng/dL) levels. Burch-Wartofsky score increased to 55, consistent with a thyrotoxic crisis. Due to the patient's critical condition, TPE was rapidly initiated along with standard therapy for thyrotoxic crisis (high dose methimazole, esmolol drip, stress dose corticosteroids, cholestyramine, and potassium iodide) as a bridge to definitive management with thyroidectomy. Rapid clinical improvement with a decline in fT4 levels (3.8 to 2.1 ng/dL) was noted after initiation of TPE with normalization in fT4 (1.5 ng/dL) and tT3 (54 ng/dL) after three sessions. Thyroidectomy was pursued after clinical stabilization. Surgical pathology showed diffuse papillary hyperplasia consistent with Graves' disease. Due to persistent respiratory failure, the patient underwent tracheostomy placement. Repeat EMG revealed severe myopathic dysfunction without demyelinating features favoring a diagnosis of acute thyrotoxic myopathy over AIDP. Patient was ultimately discharged to a long term acute care facility due to slow neurological recovery. **Conclusion:** TPE should be considered as first line management in conjunction with conventional medical therapy in critically ill patients with thyrotoxicosis as a bridge to thyroidectomy due to rapid time to effect and patient stabilization. **References:** (1) Padmanabhan A, et al. J Clin Apher. 2019 Jun;34(3):171-354. (2) Bahn Chair RS, et al. Thyroid. 2011 Jun;21(6):593-646.

Thyroid

THYROID DISORDERS CASE REPORT

Postpartum Thyroid Abnormalities and Systemic Lupus Erythematosus: Is There a Link?

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Introduction: Postpartum Thyroiditis (PPT) is an autoimmune disorder characterized by destruction of the thyroid gland within the first year after delivery. Systemic Lupus Erythematosus (SLE), another autoimmune disease, has been associated with a spectrum of thyroid disorders. While the prevalence of thyroid diseases in patients with SLE is increased, the association between SLE and PPT is not well known. The infrequency of encountering SLE and PPT makes abnormal thyroid tests in the postpartum period a diagnostic challenge.

Clinical Case: A 27-year-old G1P1001 who was five months postpartum and not breast feeding was referred to Endocrinology clinic for evaluation of abnormal thyroid function tests. Past medical history was significant for SLE with renal and pericardial involvement. SLE was well controlled, treated with hydroxychloroquine. Family history was significant for hypothyroidism in her mother.

She was asymptomatic and appeared clinically euthyroid. Vitals were stable and physical exam was negative for goiter, nodule or orbitopathy. Lab results at two months postpartum showed an elevated TSH of 3.87 UIU/mL (Normal 0.40-3.8 UIU/mL) and at four months postpartum TSH was low at 0.012 UIU/mL. Repeat labs at five months postpartum continued to show a low TSH at 0.007 UIU/mL with mildly elevated Free T4 at 1.7 ng/dL (Normal 0.6-1.6 ng/dL) and elevated Free T3 of 6.0 pg/mL (Normal 2.1-3.8 pg/mL). Anti-thyroid peroxidase antibodies (TPO), thyroid