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STORMY SEAS: MANAGING THYROID STORM TREATMENT-RELATED COMPLICATION WITH BETA-BLOCKER TOXICITY

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INTRODUCTION/BACKGROUND
Thyroid storm is a life-threatening condition involving multiple organ systems due to thyrotoxicosis. The standard treatment often includes the preferred option of the beta blocker (BB) propranolol. However, usage of BB in thyroid storm management was linked to cardiogenic collapse due to its toxicity. We present a case of thyroid storm who was treated with a beta-blocker and developed toxicity.

CASE
A 65-year-old female presented with shortness of breath, palpitations, fever, and diarrhoea. In the Emergency Department, she was fully conscious but agitated. She had tachycardia with signs of heart failure. Urgent TFT showed suppressed TSH <0.01 m IU/L and elevated T4 level of 44.12 pmol/L. Her Burch and Wartofsky Score was 70. Thus, diagnosis of thyroid storm with thyrotoxic cardiomyopathy was made. She was started on propylthiouracil, Lugol's Iodine, steroids, Propranolol 40 mg QID.

After 8 hours of treatment, she became drowsy, developed junctional bradycardia and hypotension. Appropriate resuscitation with IV Atropine and inotropes was started. Excluding other causes of hypotension with bradycardia, we considered beta-blocker toxicity. Subcutaneous glucagon was initiated. Within one day, inotropes were weaned off.

Thyroid storm can lead to lethal complications. The presentation ranges from thermoregulatory, neurologic, gastro-hepatic, cardiac dysfunctions to circulatory collapse and shock. The treatment includes BB, anti-thyroid drugs, and potassium Iodide or Lugols Iodine, along with hydrocortisone. Second-line options may include lithium, dialysis, or plasmapheresis.

Beta blockers work by reducing hyperadrenergic states and blocking the peripheral conversion of T4 to T3. They can have adverse effects such as peripheral coldness, syncope, bradycardia, hypotension, circulatory collapse, and even cardiac arrest. Glucagon is the first-line antidote for BB toxicity.

CONCLUSION
The use of beta blockers in treating thyroid storm requires close monitoring due to the risk of devastating cardiogenic collapse.

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NODULAR PRETIBIAL MYXEDEMA FOLLOWING TREATMENT OF GRAVES' DISEASE

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INTRODUCTION/BACKGROUND
Pretibial myxoedema (PM) is an uncommon manifestation of Graves' disease (GD). Typically, thyroid dermopathy may present as non-pitting oedema with indurated skin giving a 'peau d'orange' appearance. Less commonly, patients may present with other variants such as plaques, nodules and elephantiasic type lesions.

We report a case of biopsy-proven PM in a patient with GD.

CASE
An 80-year-old woman presented with multiple painless nodules over both shins with gradual increase in size over 6 months. She had a history of hypertension and difficult-to-manage GD, complicated by thyroid storm, atrial fibrillation, and heart failure. Her thyroid function fluctuated from hypo- to hyperthyroidism within weeks. She refused radioactive iodine ablation and was subsequently controlled with a block and replace regimen. She did not have any constitutional symptoms, preceding trauma, or insect bite. Clinically, she was euthyroid and did not have any active thyroid eye disease.

On examination, there were multiple ill-defined, firm, non-tender, flesh-coloured nodules over both shins. Her FT4 was 18.12 pmol/L (12.0 - 22.00) and TSH <0.005 m IU/L (027 – 4.20). A punch biopsy revealed fragmented collagen fibres with conspicuous mucin deposits over the reticular dermis and subcutaneous layer, consistent with PM. She was started on potent topical corticosteroids with marked improvement in her skin lesions.