



BMH Med. J. 2017;4(3):81-83 **Editorial**

## Acute Endocrinology Unrelated To Diabetes Mellitus

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### Abstract

Endocrine emergencies related to diabetes is very common. There are many non-diabetic emergencies in the field of Endocrine Surgery which can be quite challenging. An attempt is made here to look at these common endocrine emergencies.

**Keywords:** acute endocrinology, non-diabetic

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### Acute adrenal insufficiency

First described by Thomas Addison in 1855. Prevalence is 35-120 per million. The usual etiology of primary adrenal failure includes autoimmune conditions, tuberculosis, metastasis, drugs like antifungals, HIV infection and adrenal hemorrhage. Secondary adrenal insufficiency is due to the pituitary disease and results only in glucocorticoid deficiency. Adrenal crisis is usually precipitated by sepsis. Abrupt stoppage of steroids can also precipitate a crisis. Can also happen in patients who undergo bilateral adrenalectomy and do not take the steroid supplements. Clinical features may be shock with unexplained hypoglycemia and hyponatremia. Patient may have associated abdominal pain. Hyperpigmentation is a cardinal sign of primary adrenal failure. The basic assessment includes measurement of electrolytes, cortisol, ACTH. Short synacthen tests helps differentiate primary and secondary adrenal failure. Treatment includes fluid resuscitation with normal saline, IV hydrocortisone; later shift to oral steroids and Fludrocortisone.

### Acute hypercalcemia

Primary hyperparathyroidism (PTH mediated) and malignancy account for 90% of these cases. Non PTH mediated hypercalcemia is commonly due to osteolytic bone metastasis (PTHrP related), Hypervitaminosis D (Chronic granulomatous diseases), drugs (Thiazides), lithium and Vitamin A toxicosis. Hyperthyroidism, acromegaly and pheochromocytoma are also associated with hypercalcemia. Acute hypercalcemia presents with non specific symptoms like weakness, confusion, anorexia, vomiting, polyuria. ECG may reveal short QT interval, bradycardia, AV block.

Calcium levels  $> 12\text{mg}\%$  is an indication for prompt intervention. In such a scenario the underlying etiology can be evaluated later. Treatment includes rehydration with IV saline. Bisphosphonates (Pamidronate/ Zoledronate) are used to lower the high calcium. The action of bisphosphates begins in 24-48 hours and it may take up to 4 days for calcium to normalize. The effect of IV bisphosphonates may last a few weeks. Calcitonin has rapid onset of action within 2-6 hours of administration and results in fall in calcium up to  $2\text{mg}\%$ . However overall effect of calcitonin may be inconsistent, partial and transient. Glucocorticoids, calcimimetic agents and diuretics also used in appropriate cases.

### **Acute hypocalcemia**

This is usually due to a low PTH. Neck surgery especially thyroid, parathyroid surgery, autoimmune conditions, chronic renal disease and vitamin D deficiency. Paresthesia, muscle cramps, tetany are the clinical features. Diagnostic tests include estimation of serum calcium, phosphorus, PTH, Vitamin D, renal function and serum magnesium. The treatment of hypocalcemia depends on the severity. Serum calcium less than  $7.5\text{mg}\%$  with associated tetany needs IV calcium either as infusion or by bolus injections. Infusion can be started at  $2\text{mg}/\text{Kg}/\text{hr}$  and tapered every 12 hours and stopped. Biochemical hypocalcemia without symptoms can be managed with oral calcium carbonate. Associated vitamin D deficiency has to be corrected with calcitriol or Vitamin D2. Associated hypomagnesemia needs to be corrected to achieve normocalcemia in resistant cases.

### **Thyroid storm**

Life threatening condition. Has severe symptoms and signs of thyrotoxicosis. The diagnosis is based on clinical features which includes high fever, tachycardia, cardiac failure, hypotension, gastrointestinal symptoms and neurological manifestations like apathy, coma and stupor. Burch and Wartofsky have formulated a rating system to identify whether the patient is in thyroid storm or is having only thyrotoxicosis. Physical examination in a suspected case of thyroid storm may reveal a goiter with or without ophthalmopathy.

The thyroid profile will reveal the biochemical evidence of hyperthyroidism. The treatment is a multimodality therapy. Supportive care, correction of precipitating event and targeting all the therapeutically accessible points in the thyroid hormone synthesis, secretion and peripheral action pathways. Inhibition of thyroid hormone synthesis is achieved with thionamides (methimazole/ carbimazole), inhibition of thyroid hormone release by lithium carbonate or iodine therapy, inhibition of T4 to T3 (active form) conversion by PTU, corticosteroids, non selective beta blockers. In resistant cases plasmapheresis is done. In cases where the patient has to undergo surgery a rapid blockade can be done; however even with rapid blockade patient is safe to be operated by 6th to 8th day. Beta blocker (Propranolol)  $60\text{-}80\text{mg}$  6th hourly, Dexamethasone  $2\text{mg}$  6th hourly, saturated solution of potassium iodide (SSKI,  $38\text{mg}$  iodide/drop) 5 drops 6th hourly is used for rapid preparation. After clinical manifestations of thyroid storm are, controlled long term anti thyroid medications/ radio iodine therapy/ thyroidectomy can be planned.

### **Myxedema coma**

A medical emergency with high mortality (50-60%). Defined as a severe form of hypothyroidism with depressed mental status, hypothermia and reduced function of other organs. Most of these patients are elderly women. A hypothyroid individual can develop myxedema coma due to many exacerbating and precipitating factors. These include hypothermia, congestive cardiac failure, GI bleed, myocardial infarction, burns, trauma and medications. History of these patients may reveal prior thyroidectomy, radio iodine therapy or past history of thyroxine therapy. In 5% of these patients myxedema may be part of central hypothyroidism. Physical examination reveals the classical features of hypothyroidism like dry skin, hoarseness, macroglossia, delayed tendon reflexes etc. Hypothermia is a clinical clue to the state of myxedema in a hypothyroid subject. Cardiac

manifestations include ECG changes, recurrent pericardial effusion and congestive heart failure. Evaluation of TSH/ T4 will clinch the diagnosis but there can be associated hypoglycemia and hyponatremia. Hypoadrenalism should also be ruled out in these patients.

On admission to ICU initiation of mechanical ventilation should not be delayed so as to prevent the hypoxia and hypercapnia. Adequate care is needed to treat the associated hypothermia, hypotension and hyponatremia. Glucocorticoid therapy 100mg IV 8th hourly is initiated. IV T4 and T3 is initiated. T4 200-400 micrograms is given daily. T3 is provided at 10 micrograms thrice daily.

### **Pheochromocytoma crisis**

Patient with already diagnosed or undiagnosed pheochromocytoma presents with life threatening hypertensive crisis or cardiogenic shock. A detailed history in such patients reveal a past history of episodic attacks of headache, sweating and palpitations. Few of these patients may also be on more than two anti-hypertensive agents. Diagnosis is confirmed by estimating the plasma metanephrines or the 24 hour urinary metanephrines. Confirmation can be done by CT scan or MRI scan of the adrenal gland. IV sodium nitropruside is used to control the hypertensive crisis. Phenoxybenzamine produces irreversible, non-competitive, alpha 1 and 2 blockade and in the acute setting a dose of 1mg/Kg in 250ml of 5% dextrose is used over 4 hours. Once patient is stable patient can be started on Prazocin to control the hypertension and later operate.

### **Pituitary apoplexy**

Sudden hemorrhage into the pituitary gland. This can occur into a pre-existing pituitary adenoma. Clinical presentation may vary from mild symptoms to adrenal crisis, coma and sudden death. Sudden onset of severe headache, diplopia and symptoms of hypopituitarism indicates the diagnosis. In the acute setting cortisol deficiency has to be promptly corrected.

Apart from these, insulinomas are known to present in various forms of emergencies due to the hypoglycemia. Frequent history of loss of consciousness, coma, epileptic attacks and neuropsychiatric symptoms.

To conclude, the endocrine emergencies, even though uncommon are life threatening. Although endocrine emergencies usually occur in patients known to have endocrine pathology, its important not to miss them in the de novo presentations. Aggressive supportive care, steroid replacement and other deficient hormones have shown to decrease the morbidity and mortality associated with endocrine emergencies.

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