Endocrine Emergencies in hospital: Not everything is Diabetes

Prof.Khwaja Nazim Uddin
MBBS FCPS FRCP FACP
Professor of Medicine BIRDEM



Spectrum of Endocrine Emergencies

- Acute hypercalcemia
- Acute hypocalcaemia
- Acute adrenal insufficiency
- Myxedema coma
- Thyroid storm
- Pituitary apoplexy
- Pheochromocytoma crisis

Attention

Acute Hypercalcaemia

History of polyuria and polydipsia

Dehydration

Bone pain

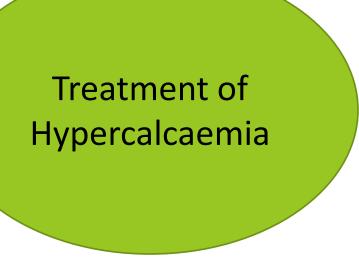
Confusion

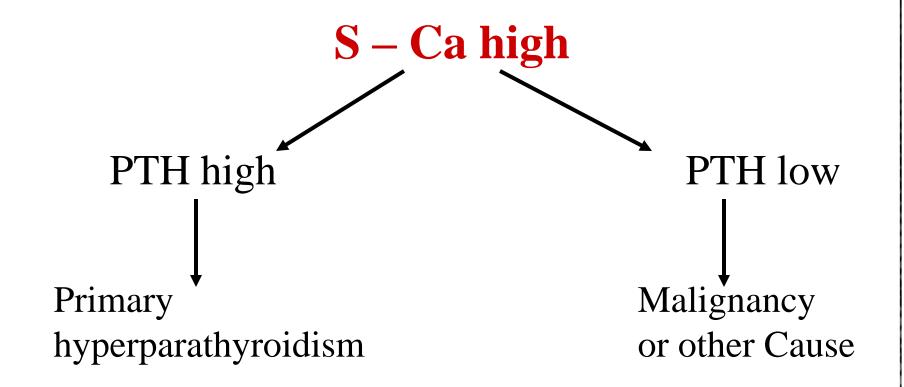
Anorexia

Constipation

 Commoner than hypocalcaemia

- Volume repletion and diureses
 - NaCl 0.9% 4 L in first 24 h
 - Loop diuretics (furosemide has calciuretic effects)
- Bisphosphonates IV (Pamidronate)
- Corticosteroids (prednisone 30 60 mg daily) are the drugs of choice if granulomatous disease or vit A or D intoxication is the cause
- **-** &
- Find out causes





S - Ca > 3.0 is 90% of the time of malignant origin

Practically we see the total calcium & correct with albumin

Acute Hypocalcaemia

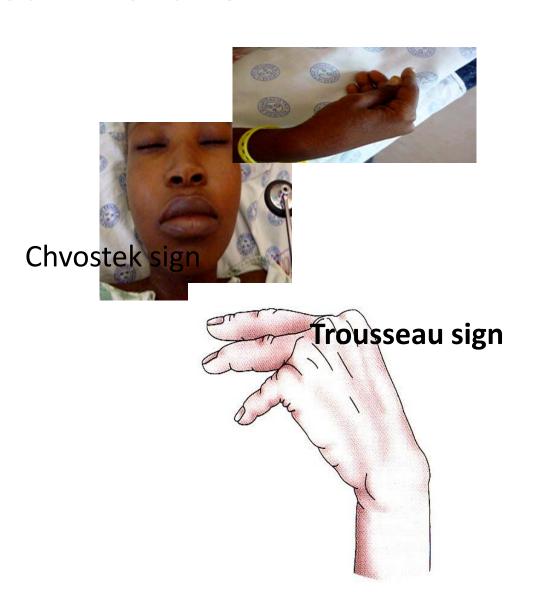
 Extremely common in the inpatient setting & estimated to occur upto 88% of patients in ICU & 26 % of non ICU ward.

- First correct low Mg⁺⁺
- Calcium gluconate 10 ml of 10% solution IV over 5 – 10 min and repeat as necessary in cases with frank generalized tetany
- Slower continuous infusion of Calcium gluconate in less acute cases
- Oral calcium and vitamin D preparation

Treatment of Hypocalcaemia

Clinical Picture

- Signs
 - Hyperreflexia
 - Hypotension
 - Bradicardia
 - Prolonged QT interval
- Arrhythmias
- Symptoms
- tetany
 - Perioral numbness
 - Tingling parasthesias
 - Muscle cramps
 - Carpopedal spasm
 - Seisures



S total Ca⁺⁺, Practically we see the total calcium & correct with albumin

- ionized Ca⁺⁺
- S PO₄++
- S Mg⁺⁺
- Plasma PTH
 - Low in hypoparathyroidism
 - High in hungry bones syndrome
- 25(OH)D₃ and 1,25 (OH)D₃
- S Amylase and Lipase
- ECG

Biochemical

- Insufficient Parathyroid Hormone surgical, familial, autoimmune, or idiopathic
- Insufficient response to PTH pseudohypoparathyroidism
- Insufficient Vitamin D rickets, osteomalasia Acute hypomagnesaemia
- Reduced 1,25(OH)vit D
 - Chronic renal insufficiency
 - Acute systemic illness
 - Drugs: ketoconazole, doxorubicin, cytarabine
- Increased uptake of Ca in bone
 - Osteoblastic metastases
 - Hungry bone syndrome
- Complexing of Ca from the circulation
 - ↑ albumin binding in alkalosis
 - Acute pancreatitis with formation of Ca soaps
 - Transfusion related citrate complexing

Precision Of Causes

Apprehension

Acute Adrenal Insufficiency

**Usually presents as an acute process in a patient with underlying chronic adrenal insufficiency

Apprehension/ Suspicion

- Hypotension
- Hypontremia
- Fatigue
- Pyrexia



Hydrocortisone

- 100 mg IV stat then 50 mg4 hly for 24 h
- Taper slowly over the next72 h
- When oral feeds is tolerated change to oral replacement therapy
- Overlap the first oral and last IV doses
- Replace salt and fluid losses with 5% dextrose in normal saline IV(may require several liters)

Management of Acute Adrenal Insufficiency

a) Do not wait for documentation of diagnosis if suspect draw blood (Cortisol and ACTH) institute therapy

B) Inj. Dexamethasone IV every 6 hrs

- Electrolytes: Hyponatremia and hyperkalemia (Hyponatremia might be obscured by dehydration)
- Random cortisol: is not helpful unless it is very low (<5 mg/L) during a period of great stress
- ACTH:Basal ACTH will be raised in primary adrenal insufficiency but not in secondary
- ACTH (cosyntropin) stimulation test/: shortsynacthen test
 - Failure of cortisol to rise above 552 nmol/L 30 min after administration of 0.25 mg of synthetic ACTH IV
- CT of abdomen :will reveal enlargement of adrenals in patents with adrenal hemorrhage, active TB or metastatic malignancy

Lab Diagnosis

- Causes of Primary adrenal insufficiency
 - Auto-immune .TB , Metastatsis
- Acute destruction of the adrenals
 - Sepsis, DIC or ,anticoagulant therapy
- Drugs. Ketoconazole, Phenytoin and rifampicin

Precipitating factors

- a)Following stress- .
- b) Sudden withdrawl of steroid
- d) Sudden destruction of pituitary Gland
- e) Injury
- f)Surgery without adrenal support
- g) unabsorbed glucocorticoid medication due to vomiting

Causes of Acute
Adrenal
insufficiency

- Causes of secondary adrenal insufficiency
 - Pituitary or hypothalamic disease
- Withdrawal of steroid therapy in a patient on long term steroid therapy (adrenal atrophy)
- look for diseases like arthritis,BA

Causes of Acute Adrenal insufficiency (2) Patients with primary adrenal insufficiency may require mineralocorticoid therapy (fludrocortisone) when shifted to oral therapy

Treat precipitating diseases

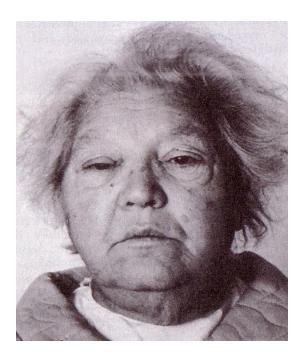
Management of Acute Adrenal Insufficiency

**Steroid Card—
counseling about
increased dose of
steroid during sick
days

Recognition

Myxedema Coma

- Typical clinical picture:
 - Elderly obese female
 - Becoming increasingly withdrawn, lethargic, sleepy and confused
- Slips into a coma



- 1.ICU admission may be required for ventilatory support and IV medications
- 2.Parenteral thyroxin (not readily available)
 - Loading dose of 300 400 μg (by NG tube)
 - Then 50 µg daily
 - 3.Slow rise of core temperature
 - 4.Take care of hypotension, hypoglycemia

Management of Myxedema coma is the end stage of untreated or inadequately treated hypothyroidism

5.Electrolytes

- Water restriction for hypernatremia
- Avoid fluid overload
- 6.Avoid sedation
- 7.Glucocorticoids
 - Controversial but necessary in hypopituitarism or multiple endocrine failure
 - Dose: Hydrocortisone 40 100 mg/ 6 hr for 1 week, then taper
 if initial serum cortisol was > 30 mg/dL, corticosteroids are unnecessary
 - Identify & treat concurrent precipitating illness

Management of Myxedema

Mortality is 40%, and is mostly due to underlying and precipitating diseases

Events Precipitating

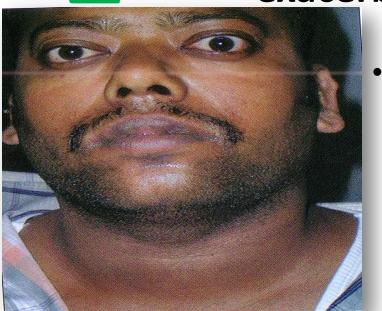
- CVD
- Myocardial infarction
- Infection
 - UTI
 - Pneumonia
- Gastrointestinal hemorrhage
- Acute trauma
- Administration of sedative, narcotic or potent diuretics



Thyroid Storm(1-2% hospital admission)



Acute life threatening exacerbation of thyrotoxicosis



- **Extreme form of Thyrotoxicosis**
 - 1) Stressful illness
- 2) Thyroid surgery
- 3) RAI administration
- 4) withdrawal anti thyroid drug

Specific management:

- B blocker: Propranolol, 40–80 mg orally 6 hourly(X sympathetic flow)
- Anti-thyroid ((decrease production)
 - Propylthiouracil, 150 mg every 6h h
 - /CarbimazoleMethimazole,
- May be administered per rectally
- Iodides(Decrease release & conversion of T4 to T3)

Ipodate sodium (500 mg /d orally)

<u>lugol,s iodine</u>)Saturated solution of <u>potassium iodide(</u>- 5 drops (250 mg) orally twice daily;

Dexamethasone I/V, 2 mg every 6 hours

- Cholestyramine 20 30 g/d
- Others:

Management

Supportive care

- Fluids, containing Glucose
- Oxygen
- Cooling
- Phenobarbital
- Multivitamins
- If indicated antibiotics or digoxin

Avoid Aspirin / use paracetamol

Laboratory diagnosis

- Free T4, free T3 elevated
- TSH suppressed
- Note that findings are not different than that of hyperthyroidism, but the difference is in the setting

Mortality dropped since the 1920's from 100% to 20 – 30%

Evaluation of underlying factors

Mortality most frequently associated with serious underlying medical conditions

Recognition

Pituitary Apoplexy

Infarction of tumor

- Hemorrage into cystic tumor
- It may occur in a normal gland during and after child birth, or with head trauma, surgery or in patient on anticoagulation therapy

- Symptoms of acute secondary adrenal insufficiency
 - Nausea vomiting, hypotension and collapse



- Severe headache and visual disturbance
- Bitemporal hemianopia
- N III palsy
- Meningeal symptoms with neck stiffness

Symptoms/ Signs

acute adrenal crisis +Pituitary tumor

Hormonal

 Dexamethasone 4 mg bd (glucocorticoid support and relief of cerebral edema) Management of Pituitary Apoplexy

Neurosurgical

Transsphenoidal pituitary decompression

***Treat adrenal crisis first then others viz thyroid replacement

After the acute episode the patient must be evaluated for multiple pituitary deficiencies

Presentation

Pheochromocytoma

- Triad of (paroxysm)
- 1.Headache 2.Palpitation
- 3.Sweating & Hypertension

Pheochromocytoma Crisis

Action of unopposed high circulating levels of catecholamines

 α - receptors: Pressor response

 β - receptors: positive ino- and chronotopic

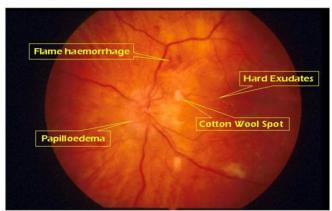
Pheochromocytoma Crisis:

Attacks build up over a few minutes and fade gradually over 15 min or can be more sustained (60 min)

Signs of end organ damage

Patients developing hypertensive crisis

during GA / SI Hypertensive Retinopathy - Grade 4



Clinical
Eeatures

Patients with unexplained heart failure

Management

- Crisis management(no need to confirm diagnosis)
- Hypertensive Crisis-

IV Nicardipine

Nitroprusside IV Infusion

Phentolamine – 1-5 mg bolus followed by infusion

- Preparation for surgery(6 weeks)
- Alpha antagonist(phenoxybenzamin) followed by beta blocker
- Surgical treatment
- Resection

Chronic case: Management <u>Diagnosis</u>

Biochemical: Urinary metanephrin/catecholamine

Plasma catecholamine/metanephrine

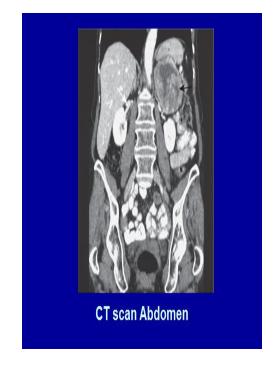
Localisation:CT/MRI/PET &Isotope studies

Treatment of HTN

 α - antagonists: Prazosin, Doxazosin, phenoxybenzamin

Non selective β - antagonist: Propranolol

Sustained release Nifidipine



Treatment with α - antagonists should precede β - antagonist treatment with 48 h to avoid exacerbation of the crisis

Summary

- Acute and chronic failure or hyper functioning of an endocrine gland may result in catastrophic illness or death
- Combined presence of multiple endocrinopathies, systemic illness and precipitating events pose challenge to physician
- It is important to appreciate and recognizes these abnormalities and manage them appropriately
- Simple measure like fluid replacement may be worthy initial step of management by a clinician

Conclusion

- Though relatively uncommon, endcrine emergencies other than those due to hyper and hypoglycemia are life threatening.
- Careful clinical evaluation and a high degree intellectual suspicion and targeted lab workup are the cornerstone to manage such cases

Thank you

