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Patient #1

Differences between Hypoparathyroidism and HBS [1]

Hypoparathyroidism	Hungry bone syndrome
Low PTH	High PTH
Hypocalcemia	Hypocalcemia
Low 1,25 vitamin D	High 1,25 vitamin D
Hypercalciuria	Hypercalciuria absent
Hyperphosphatemia	Hypophosphatemia
Magnesium typically normal	Hypomagnesemia

HBS in patient with postoperative hypoparathyroidism after KT [1]

- Following renal transplantation, functioning allograft, but in the total absent of PTH, was unable to increase endogenous 1,25 vitamin D production and consequently higher intestinal calcium absorption to compensate for MBD
- Calcium supplementation via dialysis had also stopped

Teriparatide in kidney transplant patients with severe hypoparathyroidism [2]

- 3 case reposts
- Indication: symptomatic hypoparathyroidism and difficulty in taking the medication
- Dose: 20 ug/day Duration: 30 days
- Benefit: improves symptoms, corrects serum calcium and reduces requirements for calcium and calcitriol, improve the quality of life

Learning points

- Medical professionals should be aware of intractable hypocalcemia and delayed graft function in patient with postoperative hypoparathyroidism and recently receiving KT
- Successful use of teriparatide for the treatment of hypoparathyroidism and HBS following kidney transplantation

Reference

1. Mahajan A, Narayanan M, Jaffers G, Concepcion I. Hypoparathyroidism associated with severe mineral bone disease postrenal transplantation, treated successfully with recombinant PTH. Hemodialysis International. 2009;13(4):547-50.
2. Leon L, Rial MC, Curcio D, Casadei D. Teriparatide in kidney transplant patients with severe hypoparathyroidism. Nefrologia. 2013;33(4):601-2

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Patient #2

Thyrotoxicosis factitia

All causes of thyrotoxicosis due to ingestion of excess thyroid hormone may arise from

- Intentional, surreptitious or iatrogenic ingestion
- Accidental ingestion

Thyrotoxicosis factitia may be difficult to diagnose especially in case of surreptitious use of thyroid hormone. This condition requires a high index of clinical suspicion. Clinical manifestations of thyrotoxicosis can range from mild to life-threatening condition such as thyroid storm. Laboratory thyroid function test shows suppression of TSH, elevated serum FT3, FT4 (the predominant type of FT3 or FT4 depends on specific type of thyroid hormone ingestion eg., levothyroxine or triiodothyronine).

Clues for diagnosis

- Absence of goiter
- Suppressed serum thyroglobulin (While serum thyroglobulin rises in other causes of thyrotoxicosis)
- Low radioactive iodide uptake
- Ratio T3/T4 < 20 from ingestion of levothyroxine
- High fecal T4 content
- Treatment with cholestyramine

Treatment

- Discontinuation of exogenous thyroid hormone ingestion
    - The mainstay of treatment
    - However, the patient always denies ingestion of thyroid hormone.
- Beta-blocker if severe symptoms of thyrotoxicosis
- Massive thyroid hormone overdose
    - Gastric lavage
    - Induced emesis
    - Activated charcoal
    - Cholestyramine
    - Refractory case: plasmapheresis, exchange transfusion

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1. Werner & Ingbar's the Thyroid. 10th edition
2. Mariotti S et al. N Engl J Med. 1982;307(7):410-2.
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4. Bouillon R et al. Thyroid. 1993;3:101-103.
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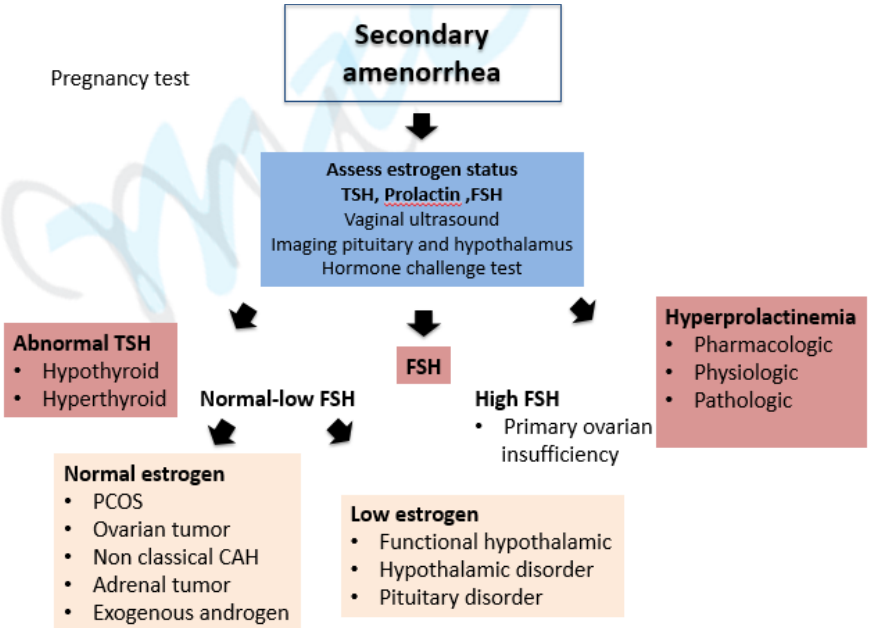
Patient #3



Adapted from Williams 's Endocrinology edition 13

and Berek & Novak' Gynecology edition 15

Cystic prolactinoma

- Cystic prolactinoma is a variant of prolactin-secreting pituitary adenoma.
- Cystic regions occupying > 50 % of the whole volume of the tumor.
- The mean age of diagnosis is 30–40 years.
- Men have greater tendencies for cyst formation: large tumor in this group.

Etiology

- Arising after the resolution of a preceding hemorrhage into the adenoma
- Necrosis of the tumor
- Occurred spontaneously
- Conjunction with other factors such as radiation therapy, trauma and use of anticoagulants

Clinical features

Hyperprolactinemia

Amenorrhea or oligomenorrhea and galactorrhea in women

Decreased libido and erectile dysfunction in men

Mass effect

Headaches, visual field defects, partial or panhypopituitarism, cranial nerve defects

Treatment

- Dopamine agonists should be considered for cystic prolactinoma in the **absence of clear indications for early surgical intervention**.
    - A trial of dopamine agonists could also be considered for cystic prolactinomas compressing the optic chiasm without visual deficits.
    - Close radiologic follow up and serial visual examinations.
- Early surgical intervention: trans sphenoidal approach
    - Symptomatic apoplexy with neuro-ophthalmic signs.
    - CSF leak which can occur in cases of invasion of the sphenoid sinus and tumor shrinkage with the use of dopamine agonists.
    - Resistance or intolerance to dopamine agonist.
    - Patient preference

References

1. Faje A, Chunharojrith P, Nency J et al (2016) Dopamine agonists can reduce cystic prolactinomas. J Clin Endocrinol Metab 101:3709–3715
2. Afif Nakhleh, Naim Shehadeh , Management of cystic prolactinomas: a review Pituitary (2018) 21:425–430