Management of thyroid and Parathyroid disorder

Rungnapa Laortanakul, MD
Nov.2018
Outline

- Thyrotoxicosis
- Hypothyroidism
- Thyroid nodule
- Primary Hyperparathyroidism
A 61-year-old woman, Nervous, anxious, periodic heart pounding

- Pulse 120/min, regular
- No exophthalmos, No lid lag, No lid retraction
- Thyroid not enlarged
- Other : normal

<table>
<thead>
<tr>
<th>Test</th>
<th>Value</th>
<th>Range</th>
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<tbody>
<tr>
<td>FreeT4</td>
<td>1.1 ng/dL</td>
<td>(0.6 – 1.6)</td>
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<td>FreeT3</td>
<td>5.5 ng/dL</td>
<td>(2.39-6.79)</td>
</tr>
<tr>
<td>TSH</td>
<td>0.001 uIU/mL</td>
<td>(0.38-5.33)</td>
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</table>
Subclinical hyperthyroidism

- Normal serum T4 and T3
- Subnormal TSH (<0.5 mU/L)
- Misnomer term!!

Causes of subclinical hyperthyroidism are the same as the causes of overt hyperthyroidism

- Excessive thyroid replacement
- Toxic nodular goiter
- Toxic MNG
- Graves’ disease
Hyperthyroidism with a normal or high radioiodine uptake

- Autoimmune thyroid disease
  - Graves' disease
  - Hashitoxicosis
- Autonomous thyroid tissue
  (uptake may be low if recent iodine load led to iodine-induced hyperthyroidism)
  - Toxic adenoma
  - Toxic multinodular goiter
- TSH-mediated hyperthyroidism
  - TSH-producing pituitary adenoma
  - Non-neoplastic TSH-mediated hyperthyroidism
- HCG-mediated hyperthyroidism
  - Hyperemesis gravidarum
  - Trophoblastic disease

Hyperthyroidism with a near absent radioiodine uptake

- Thyroiditis
  - Subacute granulomatous (de Quervain's) thyroiditis
  - Painless thyroiditis (silent thyroiditis, lymphocytic thyroiditis)
  - Postpartum thyroiditis
  - Amiodarone (also may cause iodine-induced hyperthyroidism)
  - Radiation thyroiditis
  - Palpation thyroiditis

- Exogenous thyroid hormone intake
  - Excessive replacement therapy
  - Intentional suppressive therapy
  - Factitious hyperthyroidism

- Ectopic hyperthyroidism
  - Struma ovarii
  - Metastatic follicular thyroid cancer
Differential diagnosis

- **Central hypothyroidism**
  - Some patients have low serum TSH and normal (but usually low or low-normal) free T4 and T3

- **Nonthyroidal illness**
  - especially those receiving high-dose glucocorticoids or dopamine, may have low serum TSH and low-normal free T4 and T3

- **Recovery from hyperthyroidism**
  - Serum TSH concentrations may remain low for up to several months after normalization of serum T4 and T3 in patients treated for hyperthyroidism or recovering from hyperthyroidism caused by thyroiditis.
Subclinical hyperthyroidism

- Increased risk of AF
- Decrease in bone mineral density (BMD)

- Insufficient data for treatment of subclinical hyperthyroidism in ...
- TSH between 0.1 and the lower limit of normal
- Younger patients (<65 years of age) with TSH <0.1 mU/L
  - A decision for individual management is based upon patient characteristics and preferences
Autonomous nodule evaluated by Thyroid scan/uptake

Indications for treatment of endogenous subclinical hyperthyroidism in nonpregnant adults*

Does the patient have any of the following risk factors for complications of subclinical hyperthyroidism?
- Age 65 years or older
- Cardiovascular disease or its risk factors
- Osteoporosis or its risk factors

Yes
- What is the TSH?
  - <0.1 mU/L
    - Treat the underlying cause of hyperthyroidism
  - 0.1 mU/L to lower limit of normal
    - Is the risk factor for complications of subclinical hyperthyroidism:
      - Cardiovascular disease
      - Osteoporosis
        - Yes
          - Does the patient have either:
            - Symptoms of hyperthyroidism
            - An autonomous nodule
              - Yes
                - Reasonable options include treatment of the underlying cause or close observation
              - No
                - Observation
            - Observation
        - No
          - Observation
  - <0.1 mU/L
    - What is the TSH?
  - 0.1 mU/L to lower limit of normal
    - What is the TSH?

No
- What is the TSH?
  - <0.1 mU/L
    - What is the TSH?
  - 0.1 mU/L to lower limit of normal
    - What is the TSH?

*Check TFT q 6 months
TREATMENT

- Thionamides: PTU, MMI
  - MMI is almost exclusively used
    - Longer duration of action, allowing for once-daily dosing, more rapid efficacy, and lower incidence of side effects
  - PTU is preferred during the 1st trimester of pregnancy (because of the more significant teratogenic effects of methimazole) and in patients who have minor drug reactions to methimazole
  - For Graves patients taking methimazole as primary therapy, it should be continued for 12 to 18 months.
- For autonomous nodule: radioiodine or surgery
- Radioiodine / Surgery
**TREATMENT**

**MMI dose**

- **Free T4 : 1-1.5 x upper limit of normal**: 5 to 10 mg
- **Free T4 : 1.5-2 x upper limit of normal**: 10 to 20 mg
- **Free T4 : 2-3 x upper limit of normal**
  - with larger goiters and more severe hyperthyroidism: 20 to 40 mg

- Assessed at 4-6 week intervals until stabilized on maintenance thionamide therapy, then at 3-6 month intervals.
- Patients with persistently low serum TSH after mo> 6 months of therapy with a thionamide are unlikely to remission when the drug is stopped.
A 61-year-old woman, DM, AF, bipolar disorder, esophagus cancer (Sx & RT)

- Fatigue, weight gain, and constipation
- Metformin, lithium, amiodarone
- Pulse 60/min, irregular
- Thyroid not enlarged
- Other : normal

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Subclinical hypothyroidism

- Normal serum T4
- Elevated serum TSH
- Misnomer term!!

Causes of subclinical hypothyroidism are the same as the causes of overt hypothyroidism

- Hashimoto’s thyroiditis
- Prior ablative
- Antithyroid drug therapy
- Prior partial thyroidectomy
- External radiation therapy
Differential diagnosis

- During the period of recovery from nonthyroidal illness, where a transiently elevated serum TSH is seen after a period of TSH suppression
- Following the hyperthyroid phase of subacute, painless, or postpartum thyroiditis, where mild hypothyroidism is usually, but not always, transient
- Assay variability
- The presence of heterophilic antibodies can interfere with TSH measurements in immunometric assays.
## Causes of hypothyroidism

### Primary
- **Chronic autoimmune thyroiditis**
- **Thyroid injury**: partial thyroidectomy or other neck surgery, radioactive iodine therapy, external radiotherapy of the head & neck
- **Thyroid infiltration** (amyloidosis, sarcoidosis, hemochromatosis, Riedel's thyroiditis, cystinosis, AIDS, primary thyroid lymphoma)
- **Inadequate replacement therapy** for overt hypothyroidism

### Thyroiditis
- **Thyroiditis**: subacute thyroiditis, postpartum thyroiditis, painless thyroiditis

### Secondary
- **Central hypothyroidism** with impaired TSH bioactivity

### Other
- **Drugs** impairing thyroid function
- **Toxic** substances, industrial and environmental agents
- **TSH receptor gene mutations; Ga gene mutations**
Drugs impairing thyroid function

- Iodine and iodine-containing medications
  - amiodarone, radiographic contrast agents
- Lithium carbonate
- Cytokines (especially interferon alpha)
- Aminoglutetimide
- Ethionamide
- Sulfonamides
Amiodarone and thyroid dysfunction

- Risk ~ 2-30%
- Depending upon underlying thyroid status, dietary iodine intake, and subclinical thyroid disorders
- Intrinsic drug effects
- Extrinsic drug effects
Extrinsic drug effect

- Wolff-Chaikoff effect
  - Iodine transport and thyroid hormone synthesis are transiently inhibited

- “Fail to escape” from the Wolff-Chaikoff effect
  - Autoimmune thyroid disease

- Jod-Basedow
  - Autonomous function
Normal, euthyroid individuals

Acute phase
- T4: ↑ 20-24%
- T3: ↓ 30%
- rT3: ↑ 20%
- TSH: ↑ upper norm

After 3-6 months
- T4: ↑ upper norm
- T3: ↓ lower norm
- rT3: ↑ upper norm
- TSH: ↔ normal
Amiodarone-induced hypothyroidism

- Hashimoto's thyroiditis or positive antithyroid antibodies more likely to develop persistent hypothyroidism.

- Amiodarone is usually not discontinued unless it fails to control the underlying arrhythmia.

- Replacement with T4 while amiodarone is continued.

- Hypothyroidism may persist after withdrawal of amiodarone in patients who have underlying chronic autoimmune thyroiditis.
Amiodarone-induced thyrotoxicosis (AIT)

**TABLE 2. AIT-differential diagnosis and therapy**

<table>
<thead>
<tr>
<th>Baseline thyroid condition</th>
<th>Type I AIT</th>
<th>Type II AIT Destructive thyroiditis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Toxic nodular goiter</td>
<td>“Latent” Graves’</td>
<td>Normal thyroid</td>
</tr>
<tr>
<td>Thyroid exam</td>
<td>Normal size or diffuse goiter. Bruit may be present.</td>
<td>Normal size or diffuse goiter</td>
</tr>
<tr>
<td>Ultrasound</td>
<td>Diffuse goiter</td>
<td>Heterogeneous pattern</td>
</tr>
<tr>
<td>CFDS</td>
<td>Normal or increased flow</td>
<td>Decreased flow</td>
</tr>
<tr>
<td>Thyroid autoantibodies</td>
<td>Present</td>
<td>Generally absent</td>
</tr>
<tr>
<td>Absent</td>
<td>Normal or high</td>
<td>Very high</td>
</tr>
<tr>
<td>Normal or increased flow</td>
<td>Low, normal, or high</td>
<td>Very low</td>
</tr>
<tr>
<td>IL-6</td>
<td>Normal or high</td>
<td></td>
</tr>
<tr>
<td>24-h radiiodine uptake</td>
<td>Low, normal, or high</td>
<td></td>
</tr>
<tr>
<td>Therapy</td>
<td>Methimazole or Propylthiouracil</td>
<td>Prednisione</td>
</tr>
<tr>
<td>Methimazole or</td>
<td>Methimazole or Propylthiouracil</td>
<td>Rarely surgery</td>
</tr>
<tr>
<td>Perchlorate may be</td>
<td>Perchlorate may be necessary</td>
<td></td>
</tr>
<tr>
<td>necessary</td>
<td>? ¹³¹-I</td>
<td></td>
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<tr>
<td>? ¹³¹-I</td>
<td>Surgery</td>
<td></td>
</tr>
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</table>

Prednisolone 40 to 60 mg/day
FOLLOWING PATIENTS RECEIVING AMIODARONE

BASELINE
Thyroid exam, TPO Ab*, TSH, T4, FTI (or FT4) T3 (or FT3)

EVERY 6 MONTHS
Thyroid exam, TSH, T4, FTI (or FT4) T3 (or FT3)

Serum TSH↓
No change
T4, FTI (or FT4) T3 (or FT3)
Frequent observations

Serum TSH↑
Increased
T4, (or FT4) T3 (or FT3) vs previous values
AIT

If TPO Ab positive, increased risk of AIH

AIT
AIH
CONSEQUENCES
OF SUBCLINICAL HYPOTHYROIDISM

- Progression to overt hypothyroidism
  - Annual rate of progression to overt hypothyroidism ~2 - 4 %
- Increase risk of CVD: conflict
- Lipids: associated btw elevated TSH and total & LDL chol.
- Decreased fertility: not well defined
- Non-alcoholic fatty liver disease (NAFLD): correlated with TSH
- Europsychiatric symptoms: conflict
For patients not treated with T4, monitor TSH and free T4 initially at 6 months and, if stable, yearly thereafter.

Convincing symptoms of hypothyroidism or growing goiter.
Subclinical hypothyroidism

- For women trying to conceive who have ovulatory dysfunction or infertility,
  - Elevations in TSH can be defined using 1\textsuperscript{st} trimester-specific TSH reference ranges.

- For pregnant women,
  - Elevations in TSH should be defined using population and trimester-specific TSH reference ranges.
If trimester-specific reference ranges for TSH are not available in the laboratory

The following reference ranges are recommended:

- **First trimester**: 0.1–2.5 mIU/L
- **Second trimester**: 0.2–3.0 mIU/L
- **Third trimester**: 0.3–3.0 mIU/L
Levothyroxine (LT₄)

- Half-life: 7 days
- About 80% of the hormone is absorbed relatively slowly.
- Typical dose: 1.6-1.8 µg/kg IBW per day
- Complete equilibration of free T₄ ~ 6 weeks
- Adverse effects: Bone loss, AF

Monitoring
- Primary hypothyroidism: TSH
- Secondary hypothyroidism: free T₄
Inadequate replacement therapy for overt hypothyroidism

- Inadequate dosage
- Noncompliance
- Drug interactions
  - iron, calcium carbonate, cholestyramine, dietary soy, fiber, etc.
- Increased T4 clearance
  - phenytoin, carbamazepine, phenobarbital, etc.
- Malabsorption
A 61-year-old woman, Anterior neck mass

- Clinical euthyroidism

- Growth rate
- Compressive symptoms
- History of neck radiation
- Family history of thyroid cancer, MEN 2

Size, Shape Consistency Movable Lymph node
<table>
<thead>
<tr>
<th>Benign Nodules (95%)</th>
<th>Carcinomas (5%)</th>
</tr>
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<tbody>
<tr>
<td>Hyperplastic nodules (85%)</td>
<td>Papillary (81%)</td>
</tr>
<tr>
<td>Adenomas (15%)</td>
<td>Follicular and Hurthle cell (14%)</td>
</tr>
<tr>
<td>Cysts (&lt;1%)</td>
<td>Medullary (3%)</td>
</tr>
<tr>
<td></td>
<td>Anaplastic (2%)</td>
</tr>
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</table>
Clinical findings in favor of malignant thyroid nodules

**Historical features**
- Young (<20 years) or Old (>60 years)
- Male
- Neck irradiation during childhood or adolescent
- Rapid growth
- Recent changes in speaking, breathing or swallowing
- Family history of thyroid malignancy or MEN2
Clinical findings in favor of malignant thyroid nodules

**Physical examination**

- Firm and irregular consistency
- Fixation to overlying tissues

- Vocal cord paralysis
- Regional lymph adenopathy
Evaluation of Newly Discovered Thyroid Nodules

- Nodules >1 cm should be evaluated

- There may be nodules <1 cm that require evaluation because of...
  - Suspicious US findings
  - Associated lymphadenopathy
  - History of head and neck irradiation
  - History of thyroid cancer in one or more first-degree relatives
  - Rapid growth and hoarseness
  - Pertinent physical findings suggesting possible malignancy
Low TSH

1²³I or ⁹⁹Tc scan

Hyperfunctioning

Evaluate and Tx for hyperthyroidism

History, physical, TSH

Normal or high TSH

Diagnostic US

Not functioning

Nodule on US
Do FNA

Result of FNA

Elevated TSH
Evaluate and tx for hypothyroidism

Elevated TSH
Evaluate and tx for hypothyroidism

Normal TSH
FNA not indicated

No nodule on US

Result of FNA

Nondiagnostic
- Repeat US guided FNA
- Non-diagnostic
- Close F/U or surgery

Malignant PTC
- Pre-op US
- Surgery
- Not hyperfunctioning

Suspicious for PTC
- Hürthle cell neoplasm

Interminate
- Follicular neoplasm
- Consider $^{123}$I scan if TSH low normal

Benign
- Follow
- Hyperfunctioning
Outline

- Thyrotoxicosis
- Hypothyroidism
- Thyroid nodule
- Primary Hyperparathyroidism

Hypercalcemia
A 61-year-old man, lung cancer

- Fatigue, weight loss, and constipation
- Drowsiness 1 day
- Pulse 110/min, regular
- Decrease breath sound right lung
- Other: normal

Serum calcium 13 mg/dL
Vitamin D synthesis and metabolism

Previtamin D3 $\rightarrow$ vitamin D3 via thermal isomerization

major source

Preparation process:

2. Skin absorption: Vitamin D3 is absorbed through the skin.
3. Liver conversion: In the liver, vitamin D3 is converted to 25-hydroxyvitamin D.
4. Kidney activation: In the kidney, 25-hydroxyvitamin D is activated to 1,25-dihydroxyvitamin D.

Dietary intake sources:

- Vitamin D3 (fish, meat)
- Vitamin D2 (supplements)

1,25-dihydroxyvitamin D maintains calcium balance in the body.
Parathyroid hormone
Vitamin D
sunlight → skin → 7-dehydrocholesterol → vitamin D₃ → food → liver → 25(OH)D₃ → (25-hydroxylase) → kidney → (1-hydroxylase) → 1,25(OH)₂D₃ → PTH activation → parathyroid glands → ↑PTH → ↑[Ca++]₇ECF → small intestine → ↑Ca++ absorption → kidney → ↑bone resorption → phosphate excretion → bone → ↑Ca++ reabsorption
PTH action

- Hypocalcimia $\rightarrow$ stimulate PTH secretion
- Bone $\rightarrow$ $\uparrow$ bone resorption $\rightarrow$ $\uparrow$ Ca, PO$_4$
- Kidney $\rightarrow$ $\uparrow$ Ca resorption
  $\uparrow$ PO$_4$ excretion
  $\uparrow$ Calcitriol

- Net effect is $\uparrow$ Ca $\downarrow$ PO$_4$
Calcitriol
1,25-dihydroxyvitamin D

- Small intestine → ↑ resorption of Ca,PO$_4$
- Bone → ↑ bone resorption
- Kidney → ↑ excretion of Ca,PO$_4$

Net effect is ↑ Ca ↑ PO$_4$
Hypercalcemia

- Clinical depend on time onset and level
- Normal level is 8.5-10.5 mg/dl
- Calcium level 10.5-12 mg/dl asymptomatic
- Calcium level >12 usually symptomatic
Clinical presentation

- Renal
  - Nephrolithiasis/ Nephrocalcinosis
  - Nephrogenic DI
  - Polydipsia/ Polyuria
  - Distal RTA
  - Renal insufficiency
  - Chronic hypercalcemic nephropathy
    ➔ nephrocalcinosis
GI
Constipation, Anorexia, Abdominal pain, Pancreatitis, Increase gastrin secretion → PU

CNS
Anxiety, Depression, Cognitive dysfunction, Confuse, Psychosis, Coma

CVS
Calcify valve, HT, Shortened QT interval

Skeleton
Gout, Pseudogout, Chondrocalcinosis, Osteoporosis, Osteopenia, Osteitis fibrosa cystica
Causes of hypercalcemia

Parathyroid-dependent hypercalcemia

- 1° hyperparathyroidism
- 3° hyperparathyroidism
- Familial hypocalciuric hypercalcemia (FHH)
- Lithium-associated
- Antagonist autoantibodies to the calcium-sensing receptor
Parathyroid-independent hypercalcemia

- Neoplasm
  - PTHrP dependent
  - Other humoral syndromes
  - Local osteolytic dz (including metastasis)
- PTHrP excess (non-neoplasia)
- Excess vit D action
  - Ingestion of excess vit D or vit D analogues
  - Topical vit D analogues
  - Granulomatous dz
  - Williams’ syndrome

- Thyrotoxicosis
- Adrenal insufficiency
- Renal failure
  - ARF
  - CRF with aplastic bone dz
- Immobilization
- Jansen’s dz
- Drugs:
  - vit A intoxication
  - milk-alkali syndrome
  - thiazide diuretics
  - theophylline
Hyper Ca

Hemoconcentration or Serum protein abnormality

Clinical evaluation
Hx, PE, e'lyte, BUN, Cr, PO₄, ALP

S.PTH

Normal

S&S malignancy

PTH dependent

Search for occult malignancy
Chest radiograph
Serum/urine IEP
Mammogram
Abdominal/chest CT

PTH independent

Evaluate for Other causes of PTH-independent

NO

Select appropriate tx, consider bisphosphonate

YES
Work up

- Ca, PO₄ level
- Film find evidence of bone abnormality
- Find the solid organ tumor e.g. CXR
- Alkaline phosphatase → bone lysis
- Hypercholoric metabolic acidosis suggest hyperPTH
- TFT → hyperthyroidism
- Cortisol → adrenal insufficiency
- Intact PTH level → hyperparathyroidism
- PTH-rP, vit D
- Urine calcium
A 61-year-old man, lung cancer

- Fatigue, weight loss, and constipation
- Drowsiness 1 day
- Pulse 110/min, regular
- Decrease breath sound right lung
- Other: normal

Serum calcium 13 mg/dL
Serum iPTH: suppress

Hydration
Loop diuretic
Bisphosphonate IV
Calcitonin SC
Tx primary CA
A 61-year-old man, healthy, check-up

- Check-up
- Pulse 80/min, regular
- Other: normal

Serum calcium 13 mg/dL

High PTH
Primary hyperparathyroidism

Present in one of four ways:

1. Asymptomatic hypercalcemia detected by routine biochemical screening

2. Symptomatic hypercalcemia

3. During evaluation for manifestations of hyperparathyroidism such as osteopenia, osteoporosis, or nephrolithiasis

4. Rarely, hyperparathyroid bone disease (osteitis fibrosa cystica) or parathyroid crisis
Primary hyperparathyroidism

- Adenoma
- Carcinoma
- Glandular hyperplasia
- MEN 1
- MEN 2A
- Familial hyperparathyroidism
## Multiple endocrine neoplasia

### Type 1
- Primary hyperparathyroidism (>90 percent)
- Pituitary tumors (10 to 20 percent)
  - Prolactinoma
  - Growth hormone-secreting
  - Corticotropin-secreting
  - Non-hormone-secreting
- Enteropancreatic tumors (60 to 70 percent)
  - Gastrinoma (Zollinger-Ellison syndrome)
  - Insulinoma
  - Vasoactive-intestinal polypeptide-secreting
  - Glucagonoma
  - Pancreatic polypeptide-secreting
  - Non-hormone-secreting
- Other

### Type 2A
- Medullary thyroid cancer (>90 percent)
- Pheochromocytoma (40 to 50 percent)
- Parathyroid hyperplasia (10 to 20 percent)
- Cutaneous lichen amyloidosis

### Type 2B
- Medullary thyroid cancer
- Pheochromocytoma
- Other
  - Mucosal neuromas
  - Intestinal ganglioneuromas
  - Marfanoid habitus

### Familial medullary thyroid cancer
- Variant of 2A
- Medullary thyroid cancer
Figure 1. A) Hip X-Ray of first case showing a long and laterally-displaced femoral neck; B) fourth patient clinical characteristics; C) mucosal neuromas in the tongue of fourth patient.
Band keratopathy

Subepithelial Ca-PO$_4$ deposits in the cornea
Sub-periosteal resorption
Cystic brown tumors

Chondrocalcinosis
Asymptomatic PHPT candidates for surgery

1. Serum calcium (> 1 mg/dL upper limit of normal)
2. BMD by DXA: T-score ≤−2.5
   (at lumbar spine, total hip, femoral neck, or distal 1/3 radius)
3. Creatinine clearance <60 mL/min
4. 24-hour urine for calcium >400 mg/day and increased stone risk by biochemical stone risk analysis
5. Age < 50 years

- Vertebral fracture by radiograph, CT, MRI, or VFA
- Presence of nephrolithiasis or nephrocalcinosis by radiograph, ultrasound, or CT

PHPT = Primary hyperparathyroidism

J Clin Endocrinol Metab 2014; 99:3561
Symptomatic PHPT

- Cohort studies: after parathyroidectomy
- BMD & fracture rate: improve
- Cognitive function: improve
- Kidney stones: incidence declines after surgery
- CVD & premature death: decrease

PHPT = Primary hyperparathyroidism
Sestamibi scan

- Noninvasive
- Most popular
- Localize abnormal parathyroid glands
- Quality varies
- Nonlocalizing sestamibi
  - Multigland disease
  - Small parathyroid gl.
  - Coexistent thyroid dz
Treatment of hypercalcemia

- Saline therapy
- Loop diuretic
- Initiated routinely once fluid repletion had been achieved to further increase urinary calcium excretion
- Calcitonin
- Bisphosphonate
- Dialysis