## Endocrinology Referral Recommendations

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<td>The following diagnoses or symptoms are considered under Endocrinology:</td>
<td>Standard history and examination. Key points and appropriate investigations are indicated below:</td>
<td>Management options essentially depend on established diagnoses.</td>
<td>Referral guidelines are provided to clarify the primary/secondary interface. In some instances they will promote understanding between General specialist and Endocrinology specialist services as well.</td>
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<td>- Pituitary disorders, hyperprolactinaemia</td>
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<tr>
<td>Adrenal Insufficiency</td>
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<td>Acute:</td>
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<td>• cessation of glucocorticoid therapy</td>
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<td>• Early discussion with endocrinologist advised.</td>
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<td>• adrenal haemorrhage in severe illness</td>
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<td>• Treatment should be started pending results of cortisol, Synacthen testing in acutely ill patients with suspected adrenal insufficiency.</td>
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<td>Chronic:</td>
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<td>• Acute severe illness: hydrocortisone 50 mg 8-12 hourly IM or IV; IV saline.</td>
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<td>• autoimmune, Tb, other adrenal disease</td>
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<td>• Less severe illness, able to take oral medication: oral cortisone acetate 25 mg or hydrocortisone 20 mg 2-3 times daily initially.</td>
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<tr>
<td>• hypopituitarism</td>
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<td>• Maintainence therapy: cortisone acetate 12.5-25 mg AM, 12.5 mg PM or hydrocortisone 10-20 mg AM, 10 mg PM; also fludrocortisone 0.1 mg AM if primary adrenal insufficiency.</td>
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</tbody>
</table>

| Investigations:            |            |                    |                     |
| • electrolytes, creatinine |  | • Urgent:           |                     |
| • glucose                 |  | • suspected or confirmed acute adrenal insufficiency. |                     |
| • cortisol                |  | • severe untreated chronic adrenal insufficiency. |                     |
| • cortisol response to Synacthen (short Synacthen test) |  | Semi-urgent:       |                     |
| • renin, aldosterone      |  | • all other cases of suspected or confirmed adrenal insufficiency. |                     |
| • pituitary investigations if evidence of ACTH deficiency |  |                     |                     |
### Diabetes

#### Classification:
- Type 1
- Type 2
- Secondary:
  - pancreatic: haemochromatosis, chronic pancreatitis
  - endocrine: Cushing’s syndrome, acromegaly, phaeochromocytoma

#### Diagnosis:
- fasting plasma glucose >7.0 mmol/L
- random or 2 hour GTT plasma glucose >11.1 mmol/L

#### Type 1 vs Type 2:
- Type 1: ketonuria; detectable islet cell and/or GAD antibody, low c-peptide.
- Type 2: no detectable islet cell or GAD antibody; elevated or high-normal c-peptide.

#### Management Options
- **Type 1 diabetes: ‘sick day’ management:**
  - If vomiting or other acute illness not requiring immediate intravenous therapy:
    - do not omit insulin doses
    - frequent (at least four times daily) blood glucose tests
    - test each urine specimen for ketones
    - if ketonuria: administer 6 units short-acting insulin subcutaneously hourly until ketones clear; increase usual insulin doses as necessary
    - if persistent or increasing ketonuria, refer urgently

#### Referral Guidelines
- **Urgent:**
  - Type 1 diabetes:
    - all newly diagnosed patients
    - known patients with acute illness and ketonuria unresponsive to ‘sick day’ procedures
  - Type 2 diabetes:
    - acute illness with volume depletion, altered mental state, plasma glucose >25 mmol/L
- **Diabetes complications:**
  - foot ulceration with cellulitis, acute neuropathic arthropathy
  - high risk retinopathy

### Glucocorticoid excess (Cushing’s syndrome)

#### Usual causes:
- exogenous glucocorticoids
- ACTH-secreting pituitary adenoma
- ectopic ACTH secretion
- adrenal adenoma, carcinoma

#### Evaluation
- Weight gain, fat distribution, hirsutism not specific; thin skin, bruising, striae more reliable indicators.
- Measure 24 hour urine free cortisol and/or 0800-0900 plasma cortisol after 1 mg dexamethasone at 2300 to confirm or exclude cortisol excess.
- False positive results in obesity, polycystic ovary syndrome, depression, illness.

#### Management Options
- Early discussion with endocrinologist advised.

#### Referral Guidelines
- Semi-urgent or routine:
  - all patients with suspected or confirmed endogenous Cushing’s syndrome.
### Hirsutism

#### Usual causes:
- Idiopathic/ familial: increased androgen sensitivity.
- Idiopathic ovarian androgen excess (polycystic ovary syndrome).

#### Rare causes:
- Late onset congenital adrenal hyperplasia
- Cushing’s syndrome
- Functioning ovarian or adrenal tumour

#### Evaluation
- Teenage onset hirsutism with regular periods: idiopathic/familial.
- Teenage onset hirsutism with irregular periods: polycystic ovary syndrome.
- Progressive hirsutism with masculinisation, plasma testosterone >5 nmol/L consider Cushing’s syndrome, adrenal or ovarian tumour.

#### Investigations:
- Testosterone, SHBG,
- LH, FSH, prolactin
- Fasting glucose, lipids

#### Management Options
- Idiopathic/ familial:
  - Hair removal
  - Consider 4-6 month trial of spironolactone

- Polycystic ovary syndrome:
  - Hair removal.
  - Oral contraceptive pill or cyclical progestagen to restore regular periods.
  - Consider 4-6 month trial of spironolactone for acne, hirsutism.

#### Referral Guidelines
- Urgent, semi-urgent:
  - Progressive hirsutism, Cushingoid features, masculinisation, plasma testosterone >5 nmol/L.
- Routine:
  - Significant hirsutism without evidence of severe androgen excess.

### Hypercalcaemia

#### Usual causes:
- Primary hyperparathyroidism
- Malignancy: solid tumours, myeloma, other
- Sarcoidosis, other

#### Diagnosis:
- Elevated or high-normal PTH: primary hyperparathyroidism.
- Suppressed PTH: malignancy, other non-PTH mediated hypercalcaemia.

#### Evaluation
- Symptoms:
  - Often asymptomatic
  - Thirst, polyuria, renal colic
  - Anorexia, constipation, nausea, vomiting
  - Fatigue, confusion

- Investigations:
  - Serum total calcium, albumin OR ionized calcium
  - Electrolytes, creatinine, phosphate
  - Parathyroid hormone
  - Fasting AM urine calcium/creatinine
  - Bone densitometry

#### Management Options
- Severely symptomatic hypercalcaemia:
  - IV saline
  - IV pamidronate or zolendronate

- Primary hyperparathyroidism:
  - Parathyroidectomy
  - Observation

#### Referral Guidelines
- Urgent:
  - Severely symptomatic hypercalcaemia.
- Semi urgent:
  - All other symptomatic hypercalcaemia.
  - All non-PTH mediated hypercalcaemia.
- Routine:
  - Mild, asymptomatic hyperparathyroidism.
## Hypertension

### Endocrine causes of hypertension:
- **Primary hyperaldosteronism** (Conn’s syndrome)
- Phaeochromocytoma

### Primary hyperaldosteronism:
- Adrenal adenoma or bilateral hyperplasia
- Suppressed plasma renin, ‘normal’ or high aldosterone, high aldosterone:renin ratio

### Evaluation

**Possible endocrine hypertension:**
- Resistant, severe hypertension esp in younger adults
- Labile hypertension with adrenergic symptoms
- Unexplained hypokalaemia
- Adrenal mass

Many drugs affect renin and aldosterone secretion: early discussion with endocrinologist recommended

**Investigations:**
- Electrolytes, creatinine
- Renin, aldosterone
- 24 hour urine catecholamines

### Management Options

- Early discussion with endocrinologist advised.

### Referral Guidelines

**Urgent:**
- Suspected phaeochromocytoma
**Semi-urgent, routine:**
- Suspected primary hyperaldosteronism
- Adrenal ‘incidentaloma’
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<td>Hyperthyroidism</td>
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<td>Usual causes:</td>
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<tr>
<td>• Graves’ disease (+ ophthalmopathy)</td>
<td>• Is the thyroid gland enlarged? If so, is it diffuse or nodular, nontender or tender?</td>
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<tr>
<td>• Toxic multinodular goitre, adenoma</td>
<td>• Is there associated ophthalmopathy?</td>
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<tr>
<td>• Thyroiditis: incl subacute, post-partum, amiodarone</td>
<td>• Cardiac rhythm, evidence of cardiac failure?</td>
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<td></td>
<td>• Tests should include TSH, free T4, free T3, FBE, ESR, thyroid peroxidase (TPO) antibodies.</td>
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<td>• Consider isotope scan to determine cause if not clinically evident. Ultrasound is less helpful in this regard.</td>
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<td>• If hyperthyroid with Graves’ disease, consider starting carbimazole + beta blocker (after discussion with endocrinologist) followed by semi-urgent clinic appointment.</td>
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<td>• FBE essential before starting carbimazole or propylthiouracil; all patients must be warned of risk of drug-induced agranulocytosis.</td>
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<td>• Toxic multinodular goitre and adenoma usually best treated with iodine-131 without prior carbimazole therapy; beta blocker often indicated.</td>
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<td></td>
<td>• Hyperthyroidism caused by thyroiditis usually transient, unresponsive to carbimazole; beta blocker often indicated.</td>
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<td>• Consider anticoagulation if in atrial fibrillation.</td>
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All hyperthyroid patients should be referred to an endocrinologist.

Urgent:
• Clinically severe hyperthyroidism complicated by cardiac, respiratory failure.
• Neutropaenia in patients taking carbimazole or propylthiouracil.
• Possible tracheal or superior vena caval obstruction from retrosternal thyroid enlargement.

Semi-urgent:
• All other newly diagnosed hyperthyroid patients
• Recurrent hyperthyroidism
• Inadequate or unstable response to medication
• Intolerance of medication
### Diagnosis / Symptomatology

**Hypocalcaemia**

**Usual causes:**
- vitamin D deficiency
- hypoparathyroidism

**Causes of vitamin D deficiency:**
- lack of sunlight exposure
- malabsorption
- renal failure

### Evaluation

- Severe, symptomatic with elevated phosphate: hypoparathyroidism.
- Mild, asymptomatic with normal or low phosphate (unless renal impairment): vitamin D deficiency.

**Investigations:**
- total or ionized calcium
- phosphate, electrolytes, creatinine, ALP
- parathyroid hormone
- 25-hydroxy-vitamin D

### Management Options

- Calcium supplement.
- Ergocalciferol (vit D$_2$) or calcitriol.

### Referral Guidelines

**Urgent:**
- severe, symptomatic hypocalcaemia

**Semi-urgent, routine:**
- mild, asymptomatic hypocalcaemia.
### Hypoglycaemia

**Usual causes:**
- young, lean, fit adults
- impaired glucose tolerance, early Type 2 diabetes
- dumping syndrome

**Fasting:**
- insulin excess esp insulinoma
- liver failure
- hypoadrenalism
- growth hormone deficiency (esp children)
- sulphonylureas, insulin

Fasting hypoglycaemia often caused by insulinoma; reactive hypoglycaemia usually benign.

**Evaluation:**
- Fasting or postprandial symptoms?
- Relieved by carbohydrate?
- Low blood glucose at time of symptoms?
- Previous abdominal surgery
- Access to hypoglycaemic medication?

**Investigations:**
- capillary, plasma glucose at time of symptoms
- fasting plasma glucose and insulin
- prolonged (up to 72 hours) fasting may be needed to exclude or confirm fasting hypoglycaemia
- 2 hr glucose tolerance test to confirm or exclude diabetes, IGT
- prolonged GTT not helpful
- LFT’s, plasma cortisol

**Management Options:**
- Reactive hypoglycaemia:
  - avoid simple sugars; high complex carbohydrate diet
  - exercise, weight loss to reduce insulin resistance

- Fasting hypoglycaemia:
  - refer for urgent investigation, management

**Referral Guidelines:**
- Urgent:
  - all patients with fasting hypoglycaemia

- Semi-urgent:
  - suspected fasting hypoglycaemia

- Routine:
  - reactive hypoglycaemia nor responding to diet
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<td>Usual causes:</td>
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<td>• symptomatic hyponatraemia.</td>
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<td>InappropriateADH secretion:</td>
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<td>• SSRI's, other drugs</td>
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<tr>
<td>• hypothyroidism</td>
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<td>• intracranial pathology</td>
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<td>• chest pathology</td>
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<td>• abdominal malignancy</td>
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<td>Sodium depletion</td>
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<td>• diuretic therapy</td>
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<td>• vomiting, diarrhoea</td>
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<td>• adrenal insufficiency</td>
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<td>Oedematous states (cardiac failure, cirrhosis, nephrotic syndrome)</td>
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**Assessment:**
- Assess mental state.
- Assess volume status:
  - euvolaemic: inappropriate ADH secretion
  - hypovolaemic: sodium depletion
  - oedema: cardiac failure, cirrhosis, nephrotic syndrome

**Investigations:**
- electrolytes, creatinine
- serum and urine osmolality
- urine sodium

**Management Options:**
- Water retention caused by inappropriate ADH secretion usually readily responsive to fluid restriction.

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<td><strong>Hypothyroidism</strong></td>
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<td>Urgent, semi-urgent:</td>
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<td>While TSH measurement reliably detects primary (eg autoimmune) hypothyroidism, both TSH and thyroxine must be measured to exclude secondary hypothyroidism (eg. pituitary adenoma).</td>
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<td>• Hypothyroidism should generally be managed in the GP setting.</td>
<td>suspected or confirmed secondary hypothyroidism</td>
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<td>Routine:</td>
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<td>• problems with management of primary or secondary hypothyroidism</td>
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| Male hypogonadism          | • Low plasma total testosterone often due to low SHBG in overweight, insulin resistant men: normal free testosterone.  
| Usual causes:              | • Calculated ‘free androgen index’ unreliable indicator of free testosterone in men.  
| • hypopituitarism          | Investigations:  
| • Klinefelter’s syndrome, mumps orchitis, other testicular disease | • testosterone, SHBG  
| • Significance of age-related decline in total and free testosterone uncertain. | • LH, FSH, prolactin  
|                            | • bone densitometry  
|                            | • pituitary investigations as above if LH, FSH not elevated | • Options for testosterone replacement:  
|                            | • 2-4 weekly intramuscular testosterone esters  
|                            | • 4-6 monthly subcutaneous implants  
|                            | • Transdermal testosterone patches | Urgent, semi-urgent:  
|                            |                      | • suspected hypopituitarism  
|                            |                      | Semi-urgent, routine:  
|                            |                      | • confirmed hypogonadism |
### Diagnosis / Symptomatology

**Osteoporosis and Metabolic Bone Disease**

*Note: Core Services Report*

**Determinants of fracture risk:**
- bone density
- age
- postural instability
- previous fracture

**Important causes:**
- idiopathic, familial, aging
- alcohol, smoking
- male, female hypogonadism (incl postmenopausal)
- primary hyperparathyroidism
- glucocorticoid excess
- coeliac disease
- myeloma

### Evaluation

**Aims of clinical assessment:**
- estimate fracture risk
- exclude/detect specific causes of osteoporosis

**History:**
- falls, fractures
- smoking, alcohol
- glucocorticoid therapy
- early menopause, hypogonadism
- weight loss, diarrhoea, iron deficiency

**Examination:**
- height; kyphosis
- postural stability

**Investigations:**
- lateral X-ray thoracic and lumbar spine
- total or ionised calcium
- electrolytes, creatinine, 25-OH Vit D, alkaline phosphatase, TSH, FBE, ESR
- FSH, oestradiol, testosterone
- serum and urine protein electrophoresis
- coeliac disease serology

### Management Options

**Treatment options:**
- calcium; Vit D₂ if Vit D deficient
- weight bearing exercise
- oestrogen or testosterone if hypogonadal
- bisphosphonates

### Referral Guidelines

Most postmenopausal osteoporosis can be managed in general practice.

The following patients should be referred to an endocrinologist or osteoporosis clinic (routine):
- premenopausal
- male
- glucocorticoid-associated
- hyperparathyroidism
- other (suspected) metabolic bone disease
- unresponsive to or intolerant of therapy
- non-PBS indications for bisphosphonate therapy
### Paget’s Disease

**Most patients asymptomatic.**

**Causes of pain:**
- expansion, deformity, stress fractures of Pagetic bone
- articular surface involvement
- mechanical effects of deformity on adjacent joints

**Evaluation**
- Bone pain
- Progressive deformity
- Impaired hearing, other neurological effects

**Investigations:**
- X-ray, bone scan
- alkaline phosphatase
- calcium, electrolytes, creatinine

**Management Options**
- Oral or intravenous bisphosphonates for pain attributable to Pagetic bone involvement, as per PBS indications.

**Referral Guidelines**
- Urgent, semi-urgent: fracture, neurological involvement, heart failure
- Routine: pain attributable to Pagetic bone involvement

### Pituitary disorders

**Mass effects:**
- headache
- bitemporal hemianopia

**Hormone excess:**
- hyperprolactinaemia: galactorrhoea, amenorrhoea, erectile dysfunction
- Acromegaly
- Cushing's syndrome

**Hormone deficiency:**
- gonadotrophins, TSH, ACTH, growth hormone deficiency
- diabetes insipidus

**Evaluation**
- Consider possible mass effects, hormone excess, hormone deficiency in all patients with suspected pituitary disease.
- Hypopituitarism not excluded by ‘normal’ pituitary hormone levels.

**Investigations:**
- prolactin
- suspected Cushing’s syndrome: 24 hour urine free cortisol
- suspected acromegaly: growth hormone and IGF-1
- suspected hypopituitarism: FSH, LH and oestradiol or testosterone; TSH and thyroxine; ACTH and cortisol;
- computerised visual fields
- CT or MR pituitary imaging

**Management Options**
- Macro-, microprolactinoma:
  - cabergoline, bromocriptine
  - hormone replacement as needed
- Non-functioning adenoma:
  - hormone replacement
  - observation
  - surgery if visual impairment
- Acromegaly:
  - surgery
  - octreotide
- Cushing’s disease:
  - surgery

**Referral Guidelines**
- Urgent: visual impairment and/or severe headache with pituitary mass
- Semi-urgent or routine:
  - all other cases of suspected pituitary disease
### Polydipsia and Polyuria

**Usual causes:**
- diabetes mellitus
- hypercalcaemia
- hypokalaemia
- chronic renal failure
- primary polydipsia
- diabetes insipidus

**Evaluation:**
- Is polydipsia the cause (primary polydipsia) or consequence (hypercalcaemia, hypokalaemia, renal failure, diabetes insipidus) of polyuria?
- Fluid restriction is hazardous in patients with diabetes insipidus.

**Investigations:**
- glucose, electrolytes, calcium, creatinine
- serum and urine osmolality after supervised water deprivation

**Management Options:**
- Discuss with Endocrinologist

**Referral Guidelines:**
- Urgent or Semi-urgent:
  - severely symptomatic patients
- Semi-urgent or routine:
  - patients with less severe, long-standing symptoms

### Secondary amenorrhoea

**Usual causes:**
- pregnancy, lactation
- weight loss, exercise, illness (hypothalamic amenorrhoea)
- hyperprolactinaemia
- ovarian androgen excess (polycystic ovary syndrome)
- primary ovarian failure (premature menopause)

**Investigations:**
- beta-HCG
- prolactin, FSH, LH, oestradiol
- testosterone, SHBG
- Ovarian ultrasound has low specificity and sensitivity for PCOS.

**Management Options:**
- Hypothalamic amenorrhoea:
  - treat underlying cause cause
  - consider oestrogen replacement eg contraceptive pill
  - Hyperprolactinaemia: see ‘Pituitary Disorders’
  - Polycystic ovary syndrome: see ‘Hirsutism’

**Referral Guidelines:**
- Routine:
  - secondary amneorrhoea for investigation and management.
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<td>Thyroid enlargement</td>
<td>Symptoms:</td>
<td>Colloid, multinodular goitre:</td>
<td>Urgent:</td>
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<tr>
<td>Usual causes:</td>
<td>• recent enlargement</td>
<td>• observation</td>
<td>• severe pain</td>
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<tr>
<td>• colloid, multinodular goitre</td>
<td>• pain, tenderness</td>
<td>• surgery</td>
<td>• stridor</td>
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<tr>
<td>• Hashimoto’s thyroiditis</td>
<td>• hoarse voice, dyspnoea, dysphagia</td>
<td>• radioiodine</td>
<td>• malignancy</td>
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<tr>
<td>• colloid cyst</td>
<td>Signs:</td>
<td>Hashimoto’s thyroiditis:</td>
<td>Semi-urgent, routine:</td>
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<tr>
<td>• adenoma (non- or hyperfunctioning)</td>
<td>• diffuse goitre, multinodular goitre or solitary nodule</td>
<td>• commence thyroxine when TSH elevated</td>
<td>• uncertain diagnosis</td>
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<tr>
<td>• carcinoma</td>
<td>Investigations:</td>
<td>Solitary nodule:</td>
<td>• local symptoms</td>
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<tr>
<td>5% solitary nodules malignant.</td>
<td>• TSH; T₄, T₃ if TSH low</td>
<td>• benign: reassure, observe</td>
<td>• surgery or radioiodine required</td>
</tr>
<tr>
<td>Thyroid pain usually caused by:</td>
<td>• ESR</td>
<td>• hyperfunctioning adenoma:</td>
<td></td>
</tr>
<tr>
<td>• subacute thyroiditis</td>
<td>• thyroid peroxidase antibodies</td>
<td>• radioiodine</td>
<td></td>
</tr>
<tr>
<td>• haemorrhage into nodule</td>
<td>• fine needle aspiration cytology mandatory for solitary nodules, except if suppressed TSH i.e hyperfunctioning (benign) adenoma</td>
<td>• suspicious, malignant: surgery</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Subacute thyroiditis:</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>• anti-inflammatory medication, monitor thyroid function</td>
<td></td>
</tr>
</tbody>
</table>

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