

Pituitary tumours: recommendations for service provision and guidelines for management of patients.

SUMMARY OF A CONSENSUS STATEMENT OF A WORKING PARTY FROM
THE ENDOCRINOLOGY AND DIABETES COMMITTEE OF THE ROYAL COLLEGE OF PHYSICIANS AND
THE SOCIETY FOR ENDOCRINOLOGY IN CONJUNCTION WITH THE RESEARCH UNIT OF
THE ROYAL COLLEGE OF PHYSICIANS

Methods

Experts in the UK with an international reputation for clinical research as pituitary specialists were invited to produce background papers on specific aspects of the topic, summarising the relevant evidence from peer reviewed journals (see list of contributors at the end of this report). Because of the potential for bias in consensus development by small groups, the background papers were circulated to members of a larger working group before a workshop held at the Royal College of Physicians. The working group consisted of specialists in endocrinology, general physicians, pituitary surgeons, ophthalmologists, radiotherapists, gynaecologists, general practitioners (GPs), and representatives of the Pituitary Foundation on behalf of patients (see list of members of the workshop at the end of this report).

At the workshop there was a brief (10 min) introduction to each background paper, followed by rigorous debate (30 min per section) and the recording and agreement of the consensus view on management of each specific pituitary tumour (or craniopharyngioma) subtype. If there was more than one acceptable approach to management, they were incorporated as options of acceptable standards. The configuration of service provision for this patient group was also considered and an acceptable 'flow-through' scheme agreed (Fig 1). After the workshop a summary of the discussion was circulated to all contributors of the background papers so that these could be amended. A small group abbreviated the salient points in order to produce a concise consensus statement for good practice, evidence-based wherever possible. This was circulated to all members of the workshop, inviting comments and approval. The final document was agreed after three iterations.

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Recommendations for service provision for patients with pituitary tumours

- 1 Once the diagnosis is suspected, patients should be referred to a specialist centre for further assessment and treatment. Pituitary disease is rare, and professionals in primary and secondary care are unlikely to see enough patients to provide the full range of expertise required to take sole responsibility for management.
- 2 In view of the complex nature of these tumours and the possibility of other endocrine and general medical complications, it is usual for a clinical endocrinologist to assume responsibility for coordinating treatment and long-term follow-up.
- 3 At the specialist centre, which may be located across several sites, management plans should be agreed jointly by endocrinologists, pituitary surgeons and radiotherapists. Options include joint clinics or case discussion sessions. Neuroscience units should have formal links with an endocrinologist. Joint surgical/endocrinological review is desirable at least within the first 12 months after diagnosis and treatment.
- 4 Adequate facilities for visual field and acuity testing must be available. These can be managed by an endocrinologist with experience in pituitary related problems. However, supervision and quality control should be by an ophthalmologist, who should also be available for interpretation of tests and advice in complicated cases.
- 5 Pituitary imaging and interpretation should be performed in an experienced centre where this is done regularly. Magnetic resonance imaging (MRI) scan is preferable to computed tomography (CT) as it provides better anatomical definition prior to surgery and is more likely to identify non-tumorous lesions in the pituitary region. In urgent cases, or in patients in whom MRI is contraindicated, a CT scan is acceptable.
- 6 Pituitary function testing should be performed in an investigation unit with appropriate facilities for sample collection, handling, analysis and interpretation.

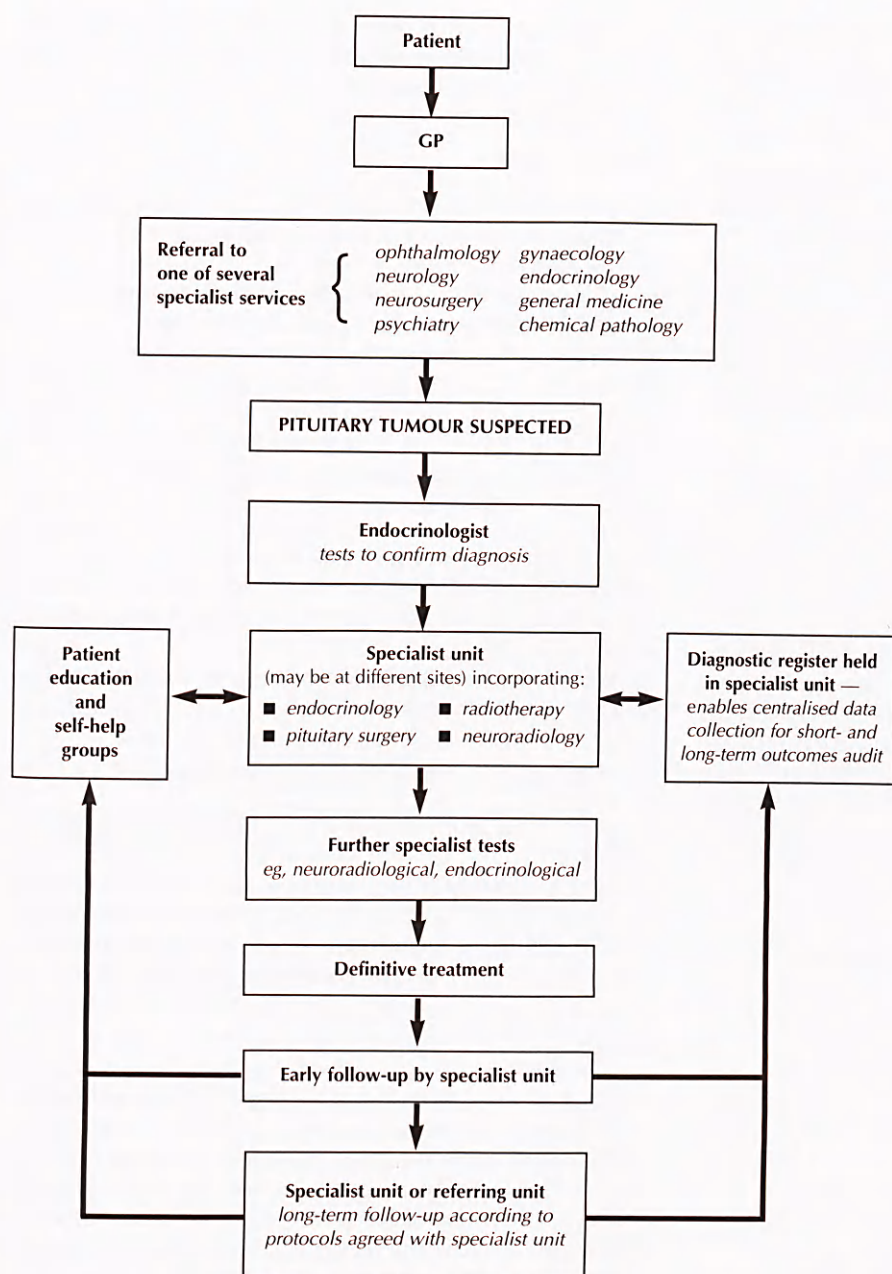


Fig 1. Recommended pathway for patients with pituitary tumours.

tation of results. All centres should have access to an accredited hormone assay service which participates in the National Quality Control Scheme.

- 7 Histological analysis, including routine immunohistochemistry, should be performed by an experienced histopathologist, usually a neuropathologist.
- 8 Surgery should be performed by surgeons specialising in pituitary surgery, able to choose the most appropriate method of operation for a patient with a 'non-functioning' tumour.

Additional superspecialisation, expertise and operative experience optimise outcome for a patient with a hormone secreting adenoma. Outcome data are required to determine the minimum number of operations that should be performed each year by a single surgeon in order to achieve the best results.

- 9 All patients should have access to and receive detailed explanation and advice from professionals conversant with all aspects of their condition, including likely immediate and long-term outcomes. Advice may be given jointly by an

endocrinologist and pituitary surgeon. Patients should be informed about self-help groups such as the Pituitary Foundation and the Child Growth Foundation. Printed literature should be provided where available.

- 10 Specialist fertility advice should be available for patients intending to conceive.
- 11 Access to psychiatric services is essential.
- 12 Paediatric patients will require support from a psychologist/educational psychologist.
- 13 Registers of all patients should be held at the endocrine unit and include all information necessary to conduct audit and provide both short- and long-term outcome data.
- 14 Protocols agreed between specialist centre and other local professionals should be available to enable some aspects of long-term follow-up to be provided in referring units.

Prescribing for patients with pituitary disease

GPs may be asked to prescribe appropriate drugs for these patients in the community, provided adequate shared care treatment plans have been agreed locally with both clinicians and purchasing authorities. Experimental treatments should usually remain under specialist prescription. Provision of routine medication may be expected through the GP, once the patient's condition and therapeutic regimen have been optimised.

Objectives of treatment are broadly defined as:

- Restoration of a feeling of well-being.
- Relief of pressure symptoms.
- Treatment of hormone excess symptoms.
- Restoration of or substitution for specific system deficits.
- Prevention of tumour regrowth.
- Lowering of increased long-term mortality.

Guidelines for the management of non-functioning tumours

Introduction

- Incidence: 10/million/year.
- Mortality in patients with hypopituitarism, associated with non-functioning tumours or their treatment, is increased 2–3 fold.

Pre-operative diagnostic tests

Formal test of visual acuity and fields. MRI scan to delineate size and anatomical relations of tumour is important for determining the surgical approach. Contrast enhancement is required to distinguish tumours from other pituitary mass lesions.

Serum prolactin result is *essential* before operation

on *any* patient; if elevated, it could be due to prolactinoma or stalk compression, and referral to an endocrinologist is advisable.

Hormonal tests

- To assess replacement therapy requirements before definitive treatment, particularly if this is to be delayed.
- To provide baseline from which to determine effect of treatment on pituitary function.
- To exclude unexpected hypersecretory state.

Therapeutic options

IN ABSENCE of pressure signs/symptoms

There is no medical therapy which reliably shrinks non-functional tumours. No intervention is advised, with close observation of vision and tumour growth and regular serial MRI scans (typically 6 monthly or annually).

Hypophysectomy/adenomectomy or external radiotherapy are other approaches.

IN PRESENCE of pressure signs/symptoms

Urgent transsphenoidal surgical debulking is the preferred route; in rare instances craniotomy may be required. Peri-operative hormonal treatment should be undertaken in consultation with an endocrinologist. A decision should be made on post-operative radiotherapy when results of surgery are known.

Follow-up

Visual field assessment: regular visual assessment (acuity and fields) is required.

Radiological: early imaging to detect residual tumour, usually 2–3 months post-operatively. In some circumstances, an immediate post-operative scan may be required. Follow-up to detect tumour regrowth is required, a suggested schedule being 6–12 months, then at 1–5 year intervals.

Endocrinological: early assessment of cortisol requirement may be performed before discharge. In many patients glucocorticoid cover is maintained until first post-operative assessment, usually 6–12 weeks after surgery. Improvement or normalisation of deficiencies may occur and requires assessment. If deficiency persists at 12 weeks, this is likely to be permanent.

External irradiation

Post-operative

Practice varies from routine use post-operatively to withholding external irradiation until recurrence is apparent. The possibility of external irradiation

should be considered for surgically treated cases if tumour removal is incomplete. Further data are required to identify patients with a high risk of recurrence to enable post-operative external irradiation to be confined to this high-risk group.

Follow-up

Visual assessment: as for surgery alone.

Radiological: the frequency of scans is currently uncertain; late recurrence can occur, and clinical circumstances should dictate frequency of scans.

Endocrinological: the effects of external irradiation are most marked in the first 2 years and slowly progressive thereafter. Lifelong hormonal follow-up is required, optimal intervals are unknown but suggested at 6 months, 1 year, 2 years, then bi/triennially.

Functional testing and replacement therapy

Functional testing

Many of these tests should be performed under the guidance of an endocrinologist. Reference to how specific tests are performed can be found in standard endocrinology texts.

Adrenocorticotrophin (ACTH)

Random blood measurements of ACTH and cortisol and 24-hour urinary free cortisol are of limited value in screening for ACTH deficiency. An impaired cortisol response to insulin-induced hypoglycaemia indicates ACTH deficiency. Patients with ischaemic heart disease, an abnormal resting ECG, dysrhythmias or epilepsy must be excluded from this test. A physician should be available close to the patient to administer intravenous hydrocortisone and/or glucose as necessary. Alternative tests are the glucagon or short synacthen test.

Thyrotrophin (TSH)

Measurement of serum TSH and thyroxine (free or total T4) is sufficient to determine TSH deficiency.

Gonadotrophins

No biochemical tests are required in women with a regular menstrual cycle. In women with oligo/amenorrhoea, luteinising hormone (LH), follicle-stimulating hormone (FSH) and prolactin measurements are useful, combined with assessment of oestrogen status (eg serum oestradiol, progestogen withdrawal test).

Measurement of serum LH and FSH, with appropriate target organ hormone (oestradiol or testosterone), helps diagnose hypogonadism as these may be inappropriately low.

Growth hormone (GH)

Children: children suspected of GH deficiency require dynamic tests in a specialist paediatric endocrine centre. Recommended dynamic tests include the insulin hypoglycaemia test, which is not recommended for children under 10 years. Alternatives are glucagon and arginine stimulation. Sex hormone priming is necessary in hypogonadal and pre- or peri-pubertal patients.

Adults: GH reserve may be assessed during insulin hypoglycaemia or the glucagon test. Other tests (eg insulin-like growth factor (IGF)-1 or the binding protein (BP) IGFBP-3) may be of value.

Vasopressin (ADH)

If the 24-hour urine volume is less than 3 litres and plasma sodium is normal, diabetes insipidus is unlikely. A water deprivation test, followed by desmopressin administration is usually necessary to confirm ADH deficiency. Adequate glucocorticoid replacement must be provided to avoid spurious results.

Therapeutic options

ACTH

Hydrocortisone is the preferred glucocorticoid replacement. Prednisolone is acceptable, but not recommended for children. Dosage is adjusted individually on clinical and biochemical grounds. Patients must be educated to increase oral glucocorticoids (2–4 fold) with illness and to seek medical advice for systemic therapy if necessary. Self-administered parenteral hydrocortisone may be useful for travel abroad.

MedicAlert or SOS bracelets/pendants are recommended in addition to carrying a steroid card.

TSH

L-thyroxine is the treatment of choice, the dosage being monitored clinically and by serum thyroid hormone levels.

Gonadotrophins (LH and FSH)

In women, cyclical oestrogen and progestogen, or lower-dose sequential hormone replacement therapy should be given until at least age 50, assuming there is no contraindication. Treatment of women with infertility and pubertal induction in both girls and boys should be performed in centres with appropriate expertise.

Testosterone replacement therapy in men can be given orally, by intramuscular injection, patches, or implant. Stimulation of spermatogenesis will require input from specialist centres.

GH

Children: biosynthetic GH is given under supervision from centres with specialist auxological and endocrine expertise. Regionally agreed, shared care protocols should be in place to facilitate primary care involvement.

Adults: GH has potential beneficial actions after growth has ceased; long-term efficacy of GH therapy in adult GH deficiency is currently being evaluated.

ADH

Desmopressin is the treatment of choice, given intranasally or orally in divided doses. Parenteral therapy is also available.

Peri-operative management

Patients should be managed jointly by the surgeon and endocrinologist. Systemic glucocorticoid therapy is usually required. The hydrocortisone dose should be reduced over several days to a maintenance regimen, until post-operative pituitary endocrine evaluation. Antibiotic prophylaxis against meningitis is advisable with transsphenoidal surgery.

Post-operative diabetes insipidus is common, but often transient. It may be managed by fluid replacement, but desmopressin should be given if it is prolonged (>2 days).

Long-term care and follow-up

Once replacement therapy has been optimised, patients should receive long-term specialist follow-up at 6–12 monthly intervals. Testing for additional pituitary hormone deficiency should be performed at regular intervals (annually) in patients receiving radiotherapy since hormone deficiencies may develop years later.

Management of craniopharyngiomas and peripituitary tumours

Introduction

The incidence of craniopharyngioma is approximately 9% of all intracranial tumours in childhood. Poor prognostic features are a young age, presentation with hydrocephalus, and hypothalamic involvement. First presentation may be in adult life.

Presentation

Acute presentation is usually with raised intracranial pressure, one-third of patients requiring urgent surgical decompression. Visual impairment is common, and usually severe before it is recognised; squints, which may be non-paretic, may also occur.

Presentation with growth failure due to an endocrine deficiency occurs in 20–30% of cases.

Diagnostic tests

When possible, the child should be assessed by an ophthalmologist, psychologist, neurologist and endocrinologist. Pre-operative endocrine assessment of thyroid status, adrenal reserve, and posterior pituitary function is required, and a pre-operative MRI scan is mandatory.

Therapeutic options plus outcomes

Acutely raised intracranial pressure will require urgent surgery. The most effective form of therapy is an attempt at complete excision, usually by craniotomy. Recurrence after initial surgical procedure is common. Morbidity figures are similar for complete resection and subtotal excision followed by elective radiotherapy. Preservation of hypothalamic function should be one of the most important aims of treatment.

Radiotherapy may prevent or slow down the rate of growth following partial surgical resection, but should not usually be given to children younger than 6–7 years.

The treatment of recurrent cysts is by surgical drainage, installation of local chemotherapy or intracavitary radiation using yttrium.

Long-term care and follow-up

Follow-up is usually coordinated by an endocrinologist, with input from specialists in neurology, psychology and ophthalmology. Transfer to an adult endocrinologist will be required as lifelong specialist follow-up is essential. Gynaecological advice should be sought early.

Endocrine follow-up will include the management of hypothalamic obesity, the re-establishment of normal statural growth with growth hormone replacement treatment as well as the establishment of puberty and maintenance of sexual maturation.

Hypothalamic syndrome, particularly diabetes insipidus in combination with absent thirst, requires careful monitoring. Fluid balance crises may be associated with intercurrent illness.

Psychological and educational difficulties caused by partial sightedness, blindness and neurological impairment require specialised resources.

Diagnosis and management of Cushing's syndrome**Introduction**

Hypercortisolism must be differentiated from conditions with similar clinical features (eg obesity, hirsutism).

It is suggested that differential diagnostic tests should be performed only in centres with appropriate technical and endocrinological expertise.

Diagnostic tests

Screening tests include 24-hour urinary free cortisol, overnight dexamethasone suppression and a low-dose dexamethasone suppression test. If clinical suspicion of Cushing's syndrome is high, specialist investigation and confirmatory tests may be necessary even if screening tests are negative.

Differential diagnosis

None of these tests is completely reliable in all cases and 'atypical' responses are common.

The most useful tests are:

- High-dose dexamethasone test (suppression points to pituitary-dependent disease).
- Plasma ACTH (unmeasurable in adrenal tumours).
- Plasma potassium (hypokalaemia favours ectopic ACTH secretion).
- Corticotrophin-releasing hormone (CRH) test (an exaggerated response of serum cortisol over basal suggests pituitary-dependent disease).
- Bilateral inferior petrosal sinus sampling performed with CRH administration is a valuable test for determining pituitary dependence.
- Contrast-enhanced MRI scan is preferable to CT to identify pituitary adenomas.

Treatment

Pituitary Cushing's disease

Transsphenoidal selective adenomectomy by an experienced surgeon is the treatment of choice. Bilateral adrenalectomy and/or pituitary radiotherapy and adrenal blocking drugs are appropriate where pituitary surgery fails or is contraindicated.

Ectopic ACTH secretion

Surgery, with excision of tumour if possible. Adrenal blocking drugs, bilateral adrenalectomy, chemotherapy and/or radiotherapy may be indicated in selected cases.

Adrenal adenoma

Unilateral adrenalectomy by an experienced surgeon is the treatment of choice. After successful surgery, hypoadrenalism is inevitable for a period of months or

years and requires appropriate replacement therapy and follow-up.

Adrenal carcinoma

Surgical excision of the tumour, if possible. Palliative adrenal blocking drug therapy is often required. Chemotherapy (usually mitotane $\pm$ other drugs) and/or radiotherapy may be indicated in selected cases.

Preparation for surgery

Patients with florid Cushing's syndrome represent a significant peri-operative risk, particularly for abdominal surgery. Medical treatment should always be considered prior to surgery in order to restore cortisol levels to normal and resolve the clinical syndrome.

Assessment of outcome after therapy

Pituitary surgery

Assessment of biochemical 'remission' can be performed within 7 days of surgery. Hypoadrenalism in the immediate post-operative period is usual after complete removal of tumour; careful observation is required.

Specialist long-term follow-up is indicated in all patients, with regular measurement of urinary free and/or plasma cortisol day profiles.

Follow-up

Patients with Cushing's syndrome of all causes require regular follow-up in a specialist endocrine clinic at least until complete recovery of the normal hypothalamo-pituitary-adrenal axis. Patients who have had adrenal adenomas removed might be discharged to GP care at this stage but all other causes require life-long follow-up to detect recurrence, development of Nelson's syndrome, and to monitor steroid replacement therapy (if needed).

Diagnosis and management of acromegaly

- Incidence: 4–6 new cases/million/year.
- Acromegaly is associated with a 2–3 fold increase in mortality.

Tests

Initial tests

- Random GH (<1.0 $\mu\text{u/l}$ excludes active acromegaly).
- IGF-1 elevated above local age-related normal range.

Diagnostic/specialist tests

- A 75 g oral glucose tolerance test (suppression of serum GH to <1 $\mu\text{u/l}$ excludes acromegaly).
- Pituitary MRI scan.
- Visual fields.
- Prolactin.
- Tests of other pituitary function (see section on functional testing and replacement therapy).

Objectives of treatment

Restoration of GH secretion to currently acceptable safe values (mean value <5 $\mu\text{u/l}$), normalisation of IGF-1 and to retain normal pituitary function.

Therapeutic options plus outcome

No active treatment only when long-term sequelae unlikely to modify prognosis.

Transsphenoidal adenomectomy

Treatment of choice for most cases is transsphenoidal adenomectomy. Serum GH levels to aim for are basal mean value <5 $\mu\text{u/l}$, suppressing to <2 $\mu\text{u/l}$ after glucose loading. Assess 6–12 weeks post-surgery. If surgery leaves some residual tumour, options include repeat surgery, pituitary radiotherapy, medical treatment or all three.

Medical treatment

If long-term medical treatment required, first consider bromocriptine up to 20 mg/day. Efficacy of bromocriptine can be determined within 3 months (only effective in 10–20%); if ineffective, use somatostatin analogue.

Somatostatin analogue: indicated in bromocriptine non-responders, those in whom surgery is contra-indicated or unsuccessful, and while awaiting the effects of pituitary radiotherapy. It is effective in 40–60% of cases. Watch for gallstone development.

External irradiation to the pituitary

Not usually first-line treatment. If used, patient may need additional medical treatment while radiotherapy takes effect.

Indications:

- Residual tumour post-operatively if GH still elevated.
- Somatostatin analogue/bromocriptine non-responder.
- Unfit for or refusal of surgery.

Watch for hypopituitarism.

Long-term care and follow-up

Post-operative

If GH <2 $\mu\text{u/l}$, review annually or less frequently with GH profile and IGF-1. Enquire about headaches and check visual fields if GH >2 $\mu\text{u/l}$. Repeat MRI scan if GH rises consistently during follow-up.

Somatostatin analogue treatment

Measure GH and IGF-1 6 monthly or more frequently until stabilised. Consider biliary ultrasound (before and during treatment).

Radiotherapy

1–3 yearly assessment of pituitary reserve to detect hypopituitarism; measure GH, IGF-1. If on somatostatin analogue, take off yearly to determine 'late' effects of radiotherapy.

Colonoscopy

In view of reports of increased incidence of colonic premalignant and malignant lesions, colonoscopy should be considered.

Diagnosis and management of hyperprolactinaemia

Introduction

Single blood samples from normals sometimes show mildly elevated prolactin levels (twice upper limit of normal). Pathological hyperprolactinaemia is commoner in women than men. Associated hypogonadism reduces bone density in the long term.

Causes

Drugs, hypothyroidism, polycystic ovarian syndrome, hypothalamic-pituitary disease.

Prolactin $>5,000$ $\mu\text{u/l}$ usually indicates a prolactinoma; 60–70% of hyperprolactinaemic women have microprolactinomas, and most hyperprolactinaemic men have prolactinomas.

Tests

Screening

- Elevated prolactin level – confirm on at least two occasions.
- Pregnancy test.
- Thyroid function test.

Diagnostic

Pituitary MRI is indicated in patients with confirmed hyperprolactinaemia not due to pregnancy, drugs or

hypothyroidism. Assessment of visual fields and hypothalamic-pituitary-adrenal status if macroadenoma present or hypopituitarism suspected.

Management

Microprolactinoma and idiopathic hyperprolactinaemia

Symptomatic: use dopamine agonists, and follow-up at 3 and 12 months, then annually with prolactin measurement. Withdraw treatment every 2–3 years to check for remission. Consider pituitary microsurgery if patient intolerant of drugs, has no return of menses or normalisation of serum prolactin by 12 months, or because of patient preference.

Asymptomatic with menses: simply observe, with regular measurement of prolactin. MRI scan if prolactin levels rise or symptoms develop.

Macroprolactinoma

Use dopamine agonists. Follow-up at 3, 6, 9, 12 months, then annually with monitoring of prolactin levels. Repeat MRI at 3–6 months, then annually until prolactin normalised and/or evidence of maximal tumour shrinkage. Most patients will require long-term treatment, but continue reducing and possibly stopping every 3–5 years to check for the small number who maintain normal prolactin levels.

Treat hypogonadism, hypoadrenalism and hypothyroidism, but check need for long-term replacement when prolactin normalised and/or tumour shrinkage, usually by 6–9 months.

Consider surgery and/or radiotherapy if patient intolerant of drugs, or if prolactin levels/tumour size continue to increase despite maximal dopamine agonist therapy. Consider surgery in those presenting with visual or cranial nerve problems if no improvement occurs within 7 days of initiation of dopamine agonist treatment.

Outcomes

Dopamine agonist treatment

Normoprolactinaemia and ovulatory cycles are restored in over 80% of women with idiopathic hyperprolactinaemia or microprolactinomas within 6 months, and up to 90% within one year of starting therapy. Most (up to 90%) prolactinomas show significant reduction in size, often maximal by 6 months but more slowly in a significant number. Visual field improvement occurs in 80–90%.

Surgery

Different series report 'cure' rates after surgery with preservation of normal pituitary function in 40–90% of cases if the pre-operative prolactin level is

<10,000 mu/l. In patients whose prolactin has returned to normal after surgery, a recurrence rate of 10–40% has been reported at 5 years (usually in the first year). Surgery for macroprolactinomas is less often (30–60%) associated with normalisation of prolactin, and there is a higher incidence of hypopituitarism and recurrence.

Pregnancy

Women on bromocriptine or quinagolide should generally stop therapy and obtain a pregnancy test if a period is missed. Consider continuation if macroprolactinoma present. There are no adverse effects of bromocriptine on mother or fetus when used before or during pregnancy.

Clinically significant expansion occurs in only 1–5% of microprolactinomas and up to 10% of macroprolactinomas during pregnancy. Patients should be checked every two months throughout pregnancy for signs or symptoms of tumour expansion. If they occur, use dopamine agonists. Surgery is an option for those who are intolerant, fail to respond, or prefer this approach.

Breast feeding: women with macroprolactinomas should be advised that there may be a risk of enlargement of the adenoma if they breast feed.

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Functional testing and replacement therapy

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 Diagnosis and management of acromegaly
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Acknowledgements

The development of this document was made possible by a grant from the NHS Executive Central Audit Funds to the authors and the Research Unit of the Royal College of Physicians.

A shorter version of this summary will also be published in other journals.

NEW TITLE

Pituitary tumours: recommendations for service provision and guidelines for management of patients.

Copies of the full recommendations and guidelines can be obtained by sending a cheque for £10.00 (overseas £12.00) to: The Publications Department, Royal College of Physicians, 11 St Andrews Place, London NW1 4LE.