

Acromegaly: FAQs for Health Care Professionals



Recognition and Referral

When should I suspect acromegaly?

Acromegaly is rare — roughly 25,000 people in the United States — but it is under-diagnosed rather than over-diagnosed, and the median delay from symptom onset to diagnosis runs to several years. The acral and facial changes that define it develop too slowly to be noticed by the patient, the family, or a clinician who sees them regularly.



The higher-yield trigger is not appearance but pattern. Consider testing when a patient under 65 has accumulated an unexplained cluster of the following, particularly if the combination is disproportionate to their age or body habitus:

- Obstructive sleep apnea
- Bilateral carpal tunnel syndrome
- Type 2 diabetes or impaired glucose tolerance
- Hypertension
- Degenerative arthropathy, especially in large joints, at a young age
- Hyperhidrosis
- Headache, or visual field loss
- Menstrual irregularity or infertility in women; erectile dysfunction or low libido in men

Direct questions that patients answer accurately: has your ring size or shoe size changed in adulthood? Have your teeth spread apart, or has your bite changed? Comparing a current photograph with one from ten years earlier is a genuinely useful clinical manoeuvre.



Who should be screened even without a suspicious cluster?

- Any patient with an incidentally discovered pituitary adenoma
- Patients with MEN1, familial isolated pituitary adenoma, Carney complex or McCune-Albright syndrome
- First-degree relatives of patients with a known AIP mutation or familial pituitary adenoma

Routine screening of unselected populations — including unselected sleep apnea or diabetes clinics — is not recommended.

At what point should I refer to endocrinology?

Refer on the basis of an elevated age- and sex-adjusted IGF-1, or on strong clinical suspicion regardless of IGF-1. You do not need to complete confirmatory testing or imaging before referring; sequencing those tests and interpreting borderline results is where specialist input helps most. Refer to a pituitary centre with a high-volume neurosurgeon where one is accessible — surgical outcome correlates strongly with surgeon volume.



Biochemical diagnosis



What is the right first test?

A single serum IGF-1, interpreted against an age- and sex-specific reference range. IGF-1 integrates growth hormone secretion over time and is stable across the day, so it does not need to be fasting or timed.

A random growth hormone level is not a screening test. GH is secreted in pulses; a low value does not exclude acromegaly and a high value does not establish it.



What can make IGF-1 misleading?

IGF-1 is the right first test but not an infallible one. Interpret with care in the presence of:

Factor	Effect and implication
Assay and reference range variability	IGF-1 assays are not fully harmonized. Use the same laboratory for serial measurements, and interpret against that laboratory's own age-stratified range.
Age	IGF-1 falls with age. An "upper normal" adult value may be inappropriate for an older patient, and vice versa.
Pregnancy	Placental GH raises IGF-1. Testing during pregnancy is unreliable; defer where possible.
Oral estrogen	Lowers IGF-1 and can mask acromegaly. Transdermal estrogen has less effect.
Poorly controlled diabetes, malnutrition, hepatic or renal failure, hypothyroidism	Lower IGF-1, risking a false negative in a patient whose comorbidity was itself the reason to suspect the diagnosis.
Adolescence	Physiologically high IGF-1; interpret against pubertal stage.

How is the diagnosis confirmed?

With growth hormone suppression during a 75 g oral glucose tolerance test. GH is sampled at baseline and at intervals over two hours. In health, glucose suppresses GH; failure to suppress below the assay-specific nadir threshold confirms acromegaly.

Two points matter in practice. First, the nadir threshold depends on the sensitivity of the GH assay used, and modern ultrasensitive assays use a lower cut-off than older ones — confirm the threshold your laboratory applies rather than relying on a remembered number. Second, glucose intolerance itself can blunt suppression, so results in patients with diabetes need specialist interpretation.

Where IGF-1 is unequivocally elevated and the clinical picture is characteristic, the OGTT does not always add information; that judgement sits with the endocrinologist.



Imaging

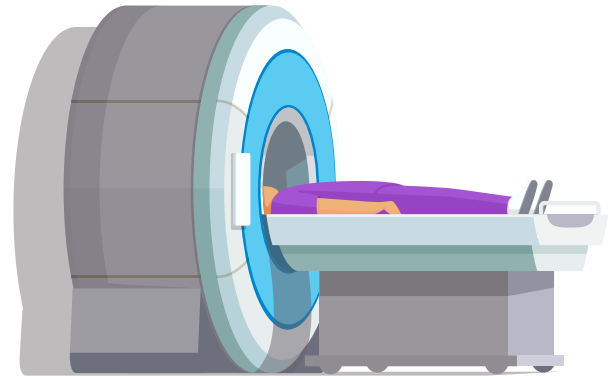


What imaging, and when?

Imaging follows biochemical confirmation – not the other way round. Pituitary incidentalomas are common, and imaging first risks anchoring on a lesion that is not the cause.

Dedicated pituitary MRI with gadolinium and thin sections is the study of choice; a standard brain MRI is not adequate. Report should specify tumor size (micro- versus macroadenoma), cavernous sinus invasion, and relationship to the optic chiasm. CT is the fallback where MRI is contraindicated, and is inferior for soft tissue detail.

If MRI shows no pituitary lesion in a patient with confirmed biochemical acromegaly, consider an ectopic GHRH-secreting tumor – rare, usually a neuroendocrine tumor of the lung or pancreas – and measure serum GHRH.



Does the patient need visual field testing?

Formal perimetry is indicated when the tumor abuts or compresses the optic chiasm, and before and after surgery in those cases. Confrontation testing is not sensitive enough to exclude a bitemporal defect.

Comorbidity assessment at diagnosis



What should be evaluated once the diagnosis is made?

Comorbidity drives the excess mortality in acromegaly, and much of it is treatable independently of the tumor. A baseline assessment at diagnosis, repeated at intervals thereafter, is standard practice:



System	Baseline assessment
Cardiovascular	Blood pressure, ECG, echocardiography, lipid panel. Cardiomyopathy, valvular disease and arrhythmia are all over-represented.
Metabolic	Fasting glucose and HbA1c. Insulin resistance is direct GH effect and often improves with disease control.
Respiratory	Screen for obstructive sleep apnea; polysomnography where symptoms or a positive screening tool suggest it.
Gastrointestinal	Colonoscopy at diagnosis, with surveillance intervals guided by findings and by disease activity.
Endocrine	Full anterior pituitary assessment — thyroid, adrenal, gonadal axes and prolactin. Hypopituitarism may coexist at presentation.
Skeletal	Vertebral fracture assessment. Fracture risk is raised even when bone density appears normal, so DXA alone is not sufficient.
Thyroid	Clinical neck examination; ultrasound where nodules are palpable. Routine ultrasound screening of everyone is not universally recommended.
Neuro-ophthalmic	Visual fields where the tumor approaches the chiasm.
Psychological	Ask directly about mood, anxiety and body image. Under-recognized and under-treated in this population.

Treatment



What is the treatment sequence?

Transsphenoidal surgery is first-line for most patients, performed by a high-volume pituitary neurosurgeon. Remission rates are substantially better for microadenomas and non-invasive tumors than for large invasive macroadenomas.



Medical therapy is used when surgery does not achieve biochemical control, when the patient is not a surgical candidate, and sometimes preoperatively. Radiation therapy – conventional, stereotactic radiosurgery or proton beam – is generally third-line, reserved for residual or recurrent disease not controlled by surgery and medication.

What are the medical options?

Class	Agents and route Practical notes
Somatostatin receptor ligands (first-generation)	<ul style="list-style-type: none"> Octreotide LAR (Sandostatin® LAR), IM monthly Lanreotide (Somatuline® Depot), SC every 4 weeks Oral octreotide (Mycapssa®), capsules Mainstay of medical therapy. Common adverse effects: gastrointestinal upset, gallstones, worsening glycaemia in some patients.
Somatostatin receptor ligand (second-generation)	<ul style="list-style-type: none"> Pasireotide LAR (Signifor® LAR), IM monthly Greater efficacy in some patients resistant to first-generation agents, but with higher rate of hyperglycaemia – monitor glucose closely.
Selective SST2 nonpeptide agonist	<ul style="list-style-type: none"> Paltusotine (Palsonify®), oral once daily FDA approved September 2025 for adults with inadequate response to surgery, or for whom surgery is not an option. First once-daily oral option in this class.
Growth hormone receptor antagonist	<ul style="list-style-type: none"> Pegvisomant (Somavert®), SC daily Blocks GH action peripherally rather than reducing secretion. Normalizes IGF-1 in a high proportion of patients. GH levels are not interpretable on treatment – monitor IGF-1 only. Monitor liver enzymes; tumor size still needs MRI surveillance.
Dopamine agonists	<ul style="list-style-type: none"> Cabergoline, oral Bromocriptine, oral Modest efficacy as monotherapy; most useful with mildly elevated IGF-1, in co-secreting tumors, or as an add-on. Cabergoline is generally preferred over bromocriptine.



What are we monitoring, and how often?

Biochemical control is judged on an age-normalized IGF-1 within the reference range, together with a suppressed or normal GH. On pegvisomant, IGF-1 alone is used, because the drug does not lower – and may raise – circulating GH.

Typical practice is IGF-1 at around 12 weeks after starting or changing therapy, and at least annually once stable, with pituitary MRI at intervals determined by residual tumor and treatment. Comorbidity screening is repeated periodically. Surveillance is lifelong; recurrence years after apparently successful surgery is well described.

What happens after radiation?

Effect on hormone levels is gradual, often over years, so medical therapy usually continues in the interim. Progressive hypopituitarism is the principal late complication and can appear a decade or more afterwards, so annual pituitary function testing is required indefinitely.

Special situations and counselling



Should I test family members?

Genetic evaluation is worth considering in patients diagnosed under about 30, those with a macroadenoma at a young age, those with a family history of pituitary tumors, and those with features of a syndromic condition. AIP mutations and, in early-childhood-onset gigantism, X-linked acrogigantism are the main considerations alongside MEN1 and Carney complex. Refer for genetic counselling rather than testing ad hoc.



What about pregnancy?

Pregnancy is usually well tolerated. IGF-1 cannot be interpreted during pregnancy because of placental GH. Medical therapy is generally withdrawn before conception where the disease allows, and management should be planned in advance with an endocrinologist experienced in pituitary disease. Watch for tumor enlargement, gestational diabetes and hypertension.



How should I frame the diagnosis with a patient?

Three messages carry most of the weight in the first conversation:

- It is a benign tumor, not cancer. Patients frequently assume otherwise, and radiation therapy – if mentioned – reinforces that fear unless addressed directly.
- It is treatable, and effective treatment can restore normal life expectancy.
- The delay in diagnosis was not their fault, and it is not too late to act.

Many patients arrive angry that this was missed.



Be honest about what treatment does and does not reverse. Soft tissue swelling, headache, sweating, sleep apnea and glucose intolerance often improve substantially. Bony changes to the jaw and face generally do not, and established arthropathy may persist. Patients told only the optimistic half of that lose trust later.

Where should I direct patients?

Refer your patients to the AACE Patient Guide to Acromegaly at aace.com/acromegaly, to learn more about the signs and symptoms, diagnosis, treatment options, and management of acromegaly.

Appendix – Reference base and review checklist

- Katznelson L, Laws ER Jr, Melmed S, et al. Acromegaly: an Endocrine Society clinical practice guideline. *J Clin Endocrinol Metab*. 2014;99(11):3933–3951.
- Fleseriu M, Biller BMK, Freda PU, et al. A Pituitary Society update to acromegaly management guidelines. *Pituitary*. 2021;24:1–13.
- Melmed S, Bronstein MD, Chanson P, et al. A Consensus Statement on acromegaly therapeutic outcomes. *Nat Rev Endocrinol*. 2018;14(9):552–561.
- Giustina A, Barkan A, Beckers A, et al. A consensus on the diagnosis and treatment of acromegaly comorbidities: an update. *J Clin Endocrinol Metab*. 2020;105(4):e937–e946.
- 16th Acromegaly Consensus Conference (September 2024). Consensus on acromegaly complications: an update. *Pituitary*, 2026.
- Broder MS, Neary MP, Chang E, et al. Incidence and prevalence of acromegaly in the United States: a claims-based analysis. *Endocr Pract*. 2016.
- FDA prescribing information for octreotide, lanreotide, pasireotide, pegvisomant and paltusotine.
- AACE. Consensus statement on pre-operative therapy in the management of patients with acromegaly, 2015 – note that this covers pre-operative therapy only and does not span diagnosis or long-term management.

