Case report

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Illustrative case of acromegaly caused by sparsely granulated somatotroph adenoma

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Background: Acromegaly arises from excessive growth hormone (GH) secretion, most often due to a pituitary adenoma. The disorder is typically indolent and may remain undiagnosed until mass-effect symptoms occur. Histological subtype, particularly the granulation pattern, influences clinical behavior and treatment response.

Case description: We describe the case of a 52-year-old man who presented with a two-year history of progressive enlargement of his hands and feet, facial coarsening, and recent-onset headache with visual blurring. Physical examination revealed classical acromegalic features and mild bitemporal hemianopia. Serum GH was markedly elevated (50 ng/mL) with other pituitary hormones within normal limits. MRI demonstrated a contrast-enhancing sellar–suprasellar lesion compressing the optic chiasm. The patient underwent endoscopic transnasal transsphenoidal excision of the mass. Histopathology confirmed a sparsely granulated somatotroph adenoma positive for PIT-1 and GH. Postoperative recovery was uneventful.

Conclusion: Sparsely granulated somatotroph adenomas exhibit greater invasiveness and reduced medical responsiveness than densely granulated variants. Early diagnosis and timely surgical resection remain crucial for visual preservation and endocrine control.

Keywords: *acromegaly; growth hormone; pituitary macroadenoma; sparsely granulated somatotroph adenoma; endoscopic transsphenoidal surgery*

Introduction

Pituitary adenomas account for approximately 10–15% of all primary brain neoplasms and are among the most common intracranial tumours. Growth hormone (GH)-secreting adenomas make up roughly 6–14% of these lesions and they usually exhibit the clinical condition of acromegaly, which is marked by systemic problems, coarse facial alterations, and gradual expansion of acral regions. Based on immunohistochemistry and electron microscopy, somatotroph adenomas are divided into two types: sparsely granulated somatotroph adenomas (SGSAs), which are far less common, and densely granulated somatotroph adenomas (DGSAs).

SGSAs are recognised for their more aggressive biological behaviour in contrast to DGSAs, which are generally smaller, less invasive, and show a positive response to first-line somatostatin analogues. They frequently have a higher proliferative index, are larger at presentation, and show expansion of the suprasellar or cavernous sinuses. Their characteristic histology includes sparse secretory granules and fibrous structures that are cytokeratin-positive. In terms of immunohistochemistry, SGSAs are often negative for other anterior pituitary hormones but positive for PIT-1 and growth hormone. They are less responsive to standard treatment with octreotide, likely because they exhibit differential somatostatin receptor expression, primarily SSTR5 over SSTR2.

Differentiating between SGSA and DGSA is uncommon, but it has significant clinical implications since it affects prognosis, treatment plans, and long-term follow-up procedures. Headaches, vision field impairments, and the possibility of hypopituitarism are among the major mass effects of macroadenomas that frequently result from delayed diagnosis. Although, SGSAs are linked to increased recurrence rates, necessitating careful, ongoing multidisciplinary therapy that includes oncology, neurosurgery, endocrinology, and pathology specialists.

In this report, we describe the case of a 52-year-old man with a sellar–suprasellar pituitary macroadenoma, sparsely granulated somatotroph adenoma histology, and characteristic acromegaly symptoms. By reviewing the pertinent literature and presenting this case, we aim to highlight the diagnostic challenges, pathological characteristics, and therapeutic implications of SGSAs. Acromegaly arises from sustained overproduction of GH, typically caused by a pituitary somatotroph adenoma. The disorder progresses slowly, often remaining undiagnosed for years until the development of coarse facial features, acral hypertrophy, or symptoms related to local tumor expansion. Pituitary macroadenomas may compress the optic chiasm, resulting in visual field deficits, and can impair pituitary function through mass effect.

Among GH-producing adenomas, the sparsely granulated subtype is recognized for its relatively rapid growth, invasiveness, and reduced responsiveness to medical therapy. We describe a case of such a tumor presenting with characteristic acromegalic changes and early visual field involvement, managed surgically via an endoscopic transsphenoidal approach.

Case report

A 52-year-old man presented with a two-year history of progressive enlargement of both hands and feet, accompanied by facial broadening and mandibular prominence. Over the preceding two months, he had developed intermittent headaches and gradually progressive blurring of vision. There was no history of trauma, fever, or systemic illness.

Clinical examination

The patient was well built and well nourished. Vital parameters were within normal limits (BP 130/80 mm Hg, HR 54/min, SpO_2 96% on room air). Facial examination revealed coarse features with frontal bossing, thickened lips and nose, prognathism, and macroglossia. Both hands and feet were enlarged with widened soft tissues (***Fig. 1***). There was no pallor, lymphadenopathy, or peripheral edema.

Neurological examination showed the patient to be alert and oriented with normal higher mental functions. Cranial nerve assessment revealed mild bitemporal hemianopia on perimetry. Pupils were equal and reactive to light; the remaining cranial nerves were intact, and motor strength was normal in all limbs. Systemic examination was otherwise unremarkable.

Investigations

1. Routine hematological and biochemical investigations were within reference limits.

2. Hormonal evaluation:

- Growth Hormone: 50 ng/mL (markedly elevated)
- Insulin like Growth Factor 1 (IGF-1): 650.5 ng/mL
- ACTH, Cortisol, TSH, T3/T4, prolactin, LH, FSH, and testosterone: within normal limits.

3. Magnetic Resonance Imaging:

MRI of the sellar region demonstrated a T1-hypointense, T2-hyperintense, contrast-enhancing mass involving the sella turcica with suprasellar extension compressing the optic chiasm and the cavernous sinus (***Fig. 2***). Imaging findings were consistent with a pituitary macroadenoma abutting over the carotid artery with the cavernous sinus. According to the modified Knosp classification the lesion was graded as 3 A.

Surgical management

The patient underwent endoscopic trans-nasal trans-sphenoidal resection of the tumor (***Fig. 3***). Intraoperatively, the mass appeared soft, greyish-pink, and moderately vascular. Gross total excision was achieved without complications. Postoperative recovery was uneventful, with gradual improvement in headache and visual symptoms.

Postoperative investigations

1. Routine hematological and biochemical investigations were within normal limits.

2. Hormonal evaluation:

- Growth Hormone: 32.5 ng/mL (markedly elevated)
- Insulin like Growth Factor-1 (IGF-1)– 1: 550ng/mL
- Cortisol – 4.4 µg/dL
- TSH, T3/T4, prolactin, LH, FSH, and testosterone: within normal limits.

Postoperative imaging

Postoperative day 4 CT Brain Plain: postoperative sellar hematoma and pneumocephalus with postoperative changes in the sellar region in brain parenchyma (***Fig. 4***).

Follow-up investigations (4 months postoperatively)

1. Routine hematological and biochemical investigations were within normal limits.

2. Hormonal evaluation:

- Growth Hormone: 26.80 ng/mL (markedly elevated)
- Insulin like Growth Factor – 1 (IGF-1): 400 ng/mL
- Cortisol – 5.26 µg/dL
- TSH, T3/T4, prolactin, LH, FSH, and testosterone: within normal limits.

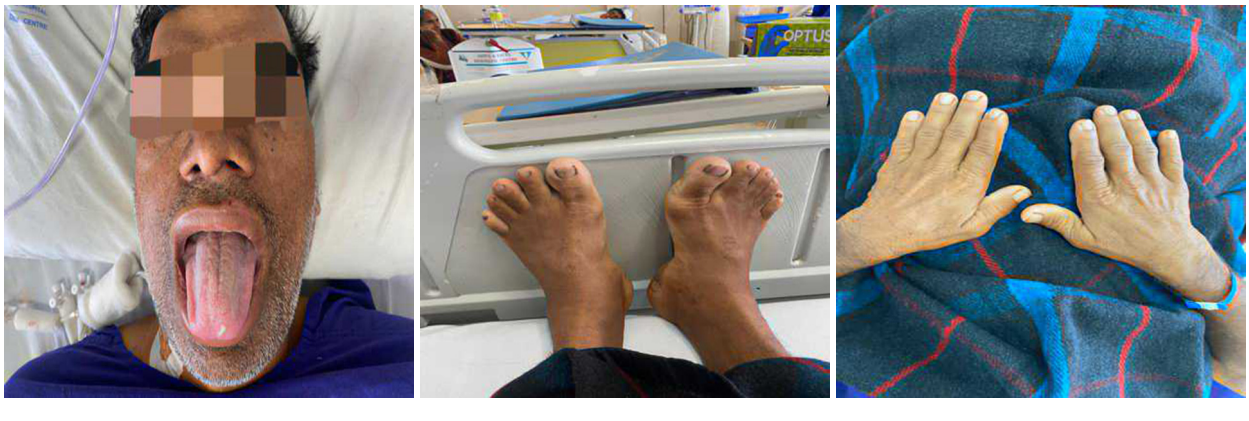


Fig. 1. Acromegalic features of the patient

Note. Photographs were obtained with the patient's informed consent.

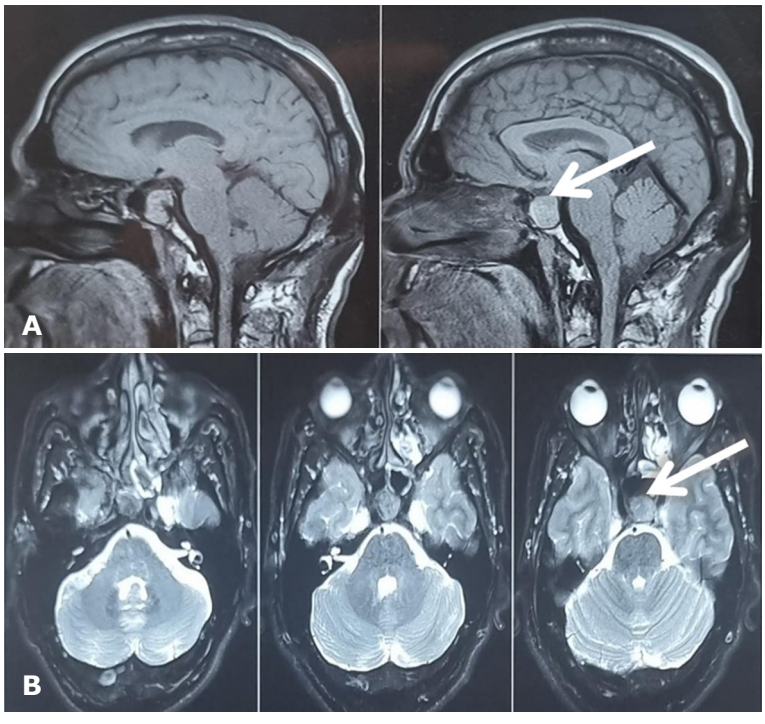


Fig. 2. Contrast enhancing suprasellar lesion suggestive of pituitary macroadenoma: sagittal (A) and axial (B) projections

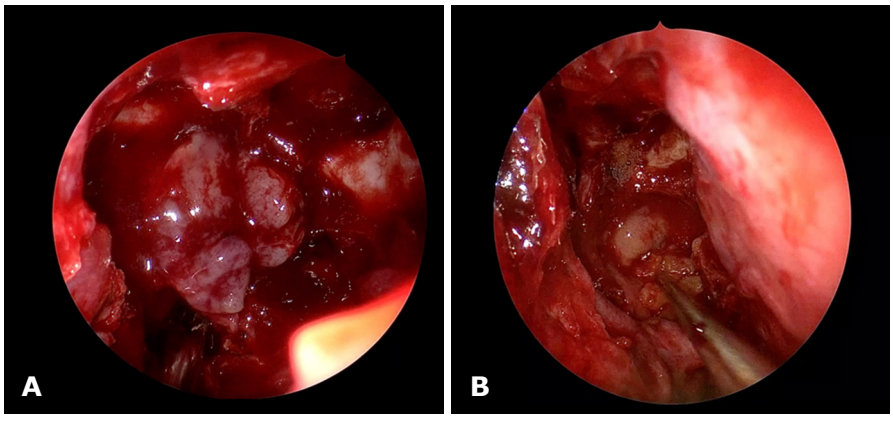


Fig. 3. Intraoperative images. A – Intraoperative pituitary tumor resection, B – Sellar Region after pituitary tumor resection reconstructed with Haddad Flap and supportive tissue with topical haemostatic agents

The patient remained clinically stable from the first postoperative day onward and follow – up imaging was subsequently performed.

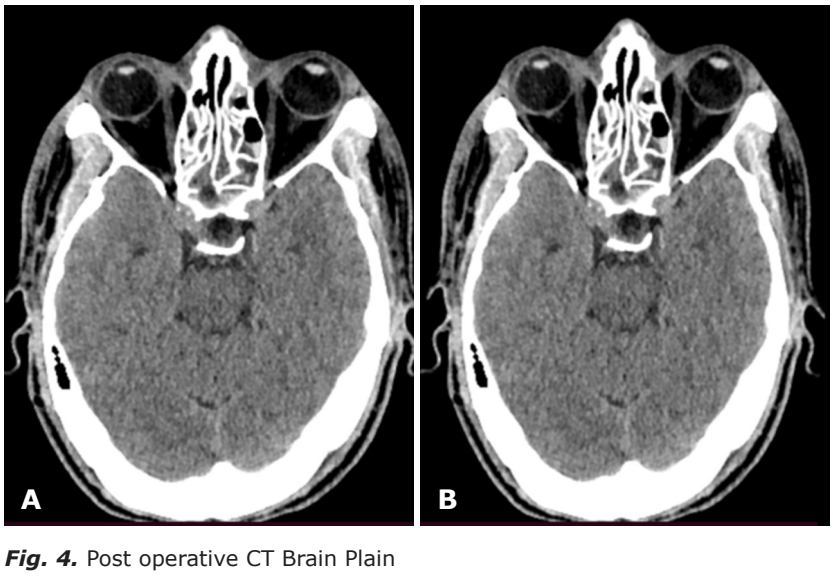


Fig. 4. Post operative CT Brain Plain

Magnetic Resonance Imaging (4 months postoperatively)

MRI of the sellar region demonstrated a T1-hypointense, T2-hyperintense, contrast-enhancing mass of 6mm. Imaging findings were consistent with residual pituitary macroadenoma (***Fig. 5***).

Histopathology and immunohistochemistry

Histopathological evaluation reported by NIMHANS, Bangalore. Microscopic examination revealed a pituitary adenoma composed of uniform cells arranged in sheets, nodules, and organoid nests. The tumor cells exhibited round-to-oval nuclei with stippled chromatin and moderate eosinophilic cytoplasm. Numerous paranuclear eosinophilic inclusions (fibrous bodies), characteristic of the sparsely granulated somatotroph subtype were noted. Mitotic figures were rare, and foci of vascular congestion and hemorrhage were present (***Fig. 6***).

Immunohistochemical profile
- Cytokeratin (CK): highlights many fibrous bodies

Transcription factor
- PIT-1: positive
- T-PIT and SF-1: negative

Pituitary hormones
- GH: positive
- PRL, ACTH, TSH, LH, FSH: negative

Ki-67 MIB-1 labelling index 3-4%

Final Diagnosis: Sparsely granulated somatotroph adenoma (GH-secreting pituitary macroadenoma)

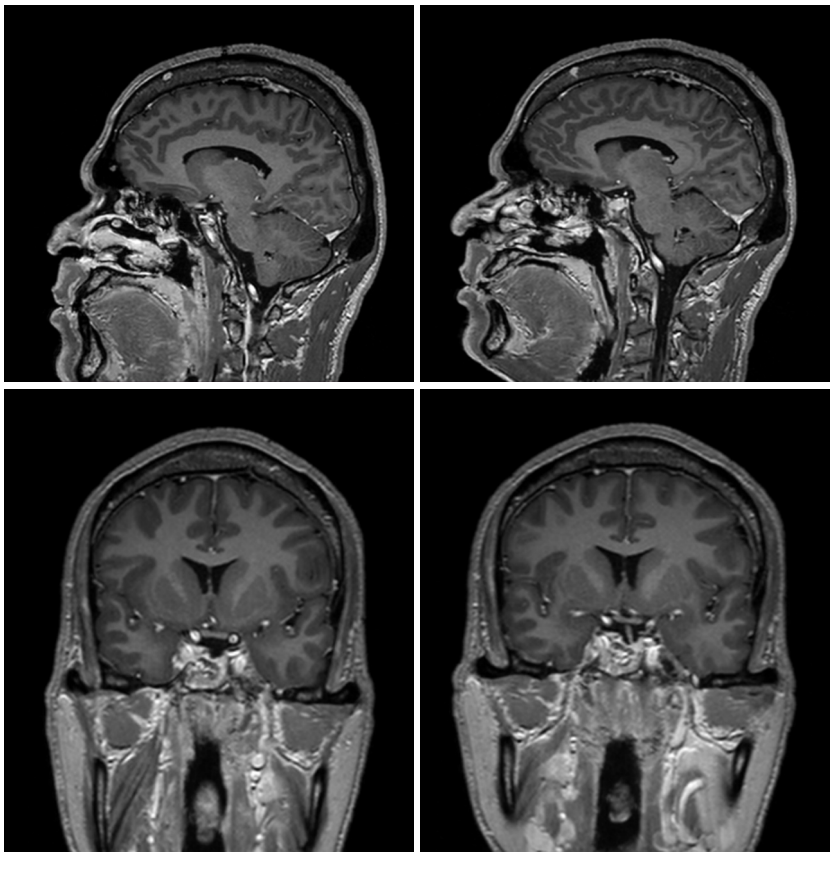
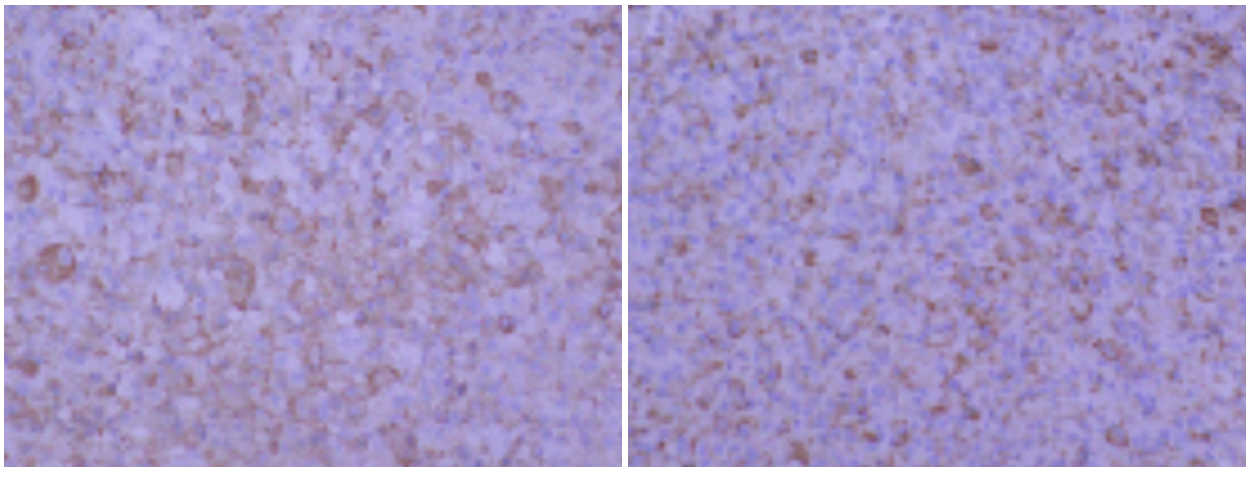
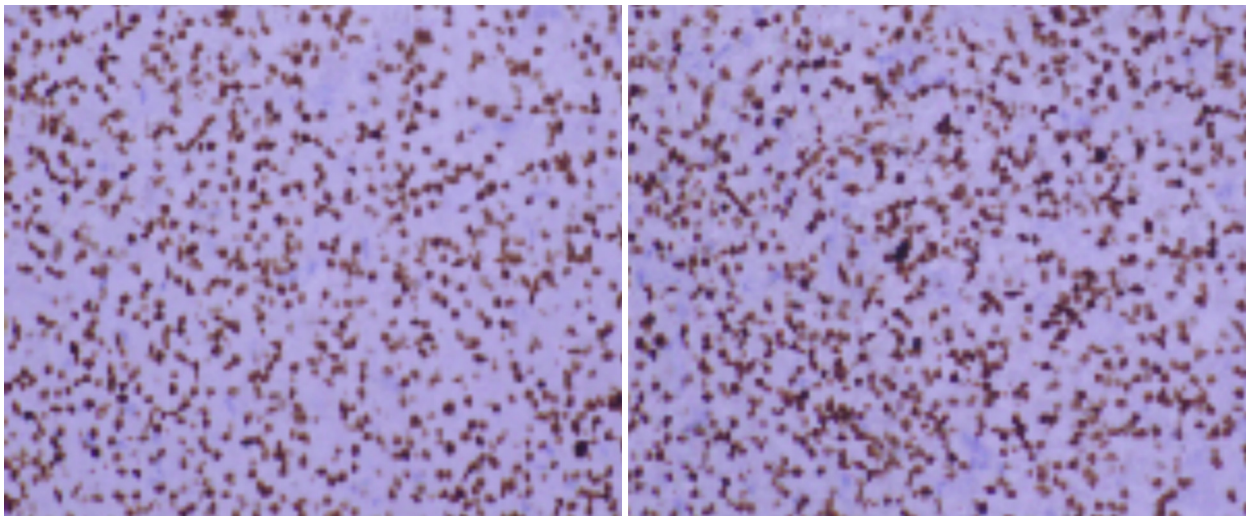


Fig. 5. Postoperative MRI brain with contrast: sagittal (A) and frontal (B) projections

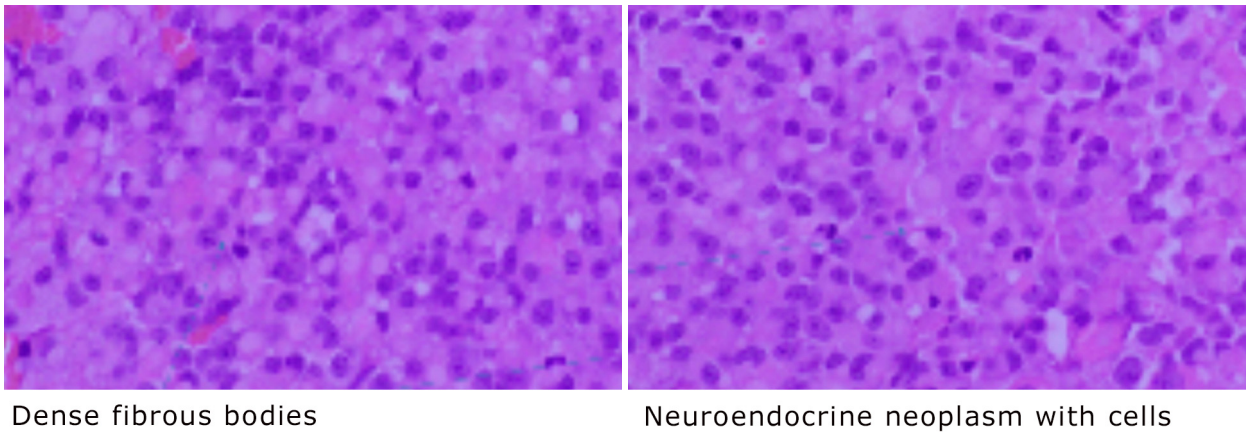
GH – IHC Positive



Pit1 – IHC x 100 Positive



H & E x 200



Dense fibrous bodies

Neuroendocrine neoplasm with cells arranged in sheets and vague nodules and sinusoidal pattern

Fig. 6. Histopathological and immunohistochemical findings

Discussion

Pituitary adenomas account for approximately 10–15% of all intracranial tumors; among these, GH-secreting adenomas represent 6–14% of cases. Based on their granulation patterns, somatotroph adenomas—the second most frequent pituitary neuroendocrine tumors [1, 2]—can be divided into two subtypes:

1. Sparsely granulated somatotroph adenomas (SGSAs) and

2. Densely granulated somatotroph adenomas (DGSAs).

Prevalence: A small percentage of all somatotroph tumours are sparsely granulated somatotroph adenomas, which are regarded as a more invasive and high-risk subtype [3-5].

Histopathology: Histologically, SGSAs are defined by chromophobic, poorly cohesive cells that are grouped in sheets or nests. They also often include paranuclear fibrous structures and light eosinophilic cytoplasm. On the other hand, DGSAs typically exhibit diffuse GH immunostaining and acidophilic cytoplasm [3, 6, 7].

In contrast to DGSAs, SGSAs exhibit lower rates of *GNAS* mutations and are PIT-1 positive immunohistochemically [8-10].

Sparsely granulated tumors are characterized by increased proliferative and invasive capacity, typically reflected in a higher Ki-67 (MIB-1) labelling index [9, 11-14].

PIT-1 positivity is a defining feature, whereas T-PIT and SF-1 are negative, aligning with somatotroph cell lineage.

The rare and distinct subtype of growth hormone (GH)-secreting pituitary adenomas known as sparsely granulated somatotroph adenomas (SGSAs) is distinguished by histological, and IHC markers. These tumours frequently pose diagnostic and therapeutic challenges because they are more aggressive, respond poorly to standard treatment, and recur more frequently than densely granulated somatotroph adenomas. [15-17].

Clinically, the present case is consistent with the typical acromegalic characteristics of SGSAs, including gradual acral enlargement, facial deformity, and related symptoms such as headaches and visual field abnormalities, especially bitemporal hemianopia caused by chiasmal compression. The pituitary aetiology was further reinforced by the absence of other systemic disorders and the lack of diurnal change in headache. Our patient's increased GH level (GH = 50 ng/mL) matched the clinical severity. Hypopituitarism, which is sometimes seen in large macroadenomas, or a plurihormonal adenoma were ruled out by the normal levels of other pituitary hormones.

Histopathologically, sparse secretory granules and paranuclear fibrous bodies distinguish SGSAs from densely granulated somatotroph adenomas. In the present case, cytokeratin (CK) immunostaining highlighted the fibrous bodies. By showing positive expression of GH and PIT-1 transcription factors and negative expression of other anterior pituitary hormones and transcription factors (TPIT, SF-1) [18-20], immunohistochemical staining was able to identify SGSAs. A stronger proliferative potential is indicated by a raised Ki-67 (MIB-1) labelling index of 3–4%, which is associated with a higher likelihood of aggressive behaviour and recurrence [21, 22].

From a therapeutic perspective, the main treatment for SGSAs is surgical resection using the transsphenoidal technique, which aims for gross complete excision while maintaining endocrine and neurological function. Surgery alone, however, is frequently insufficient due to the aggressive nature of these adenomas and the prevalence of partial resections. Owing to lesser somatostatin receptor subtype 2 (SSTR2) expression compared with densely granulated versions, adjuvant therapies such as radiation and medical therapy with somatostatin analogues show varied efficacy in SGSAs and result in lower response rates. Temozolomide is one of the novel medicines and chemotherapeutics that have shown promise in aggressive and resistant cases. Therefore, to maximise results and create customised treatment regimens, multidisciplinary strategy comprising endocrinologists, neurosurgeons, radiation oncologists, pathologists, and molecular geneticists is crucial [23-27].

Regular hormonal, clinical, and radiological follow-up is necessary because of the observed high recurrence rates, particularly in tumours with higher Ki-67 indices and inadequate resection. Timely intervention improves the prognosis when recurrence or residual disease is identified early [28, 29].

Research by Petersenn *et al*. has shown that SGSAs occur more frequently in patients under 40 years of age. Our male patient is the minority group within an already uncommon tumour subtype, despite the literature suggesting a slight female prevalence in SGSAs. SGSAs may paradoxically exhibit worse biochemical reactions to medical therapy than DGSAs, despite the fact that they frequently present with elevated GH levels because of their bigger size at diagnosis. SGSAs are less susceptible to traditional somatostatin analogues owing to their distinct somatostatin receptor expression patterns, characterized by a more frequent expression of SSTR5 than SSTR2 [30].

The mainstay of treatment for pituitary macroadenomas remains surgical excision via transsphenoidal approach. However, due to their invasive nature, SGSAs require multimodal management, including repeat surgery, radiotherapy, and advanced medical therapy with newer somatostatin analogues or temozolomide in refractory cases [8, 9, 31, 32]. Long-term monitoring is mandatory because of the high risk of recurrence, particularly in tumors with elevated Ki-67 index [11, 12].

Importance of Multidisciplinary Management

The rarity and aggressiveness of SGSAs demand an individualized, multidisciplinary approach encompassing endocrinology, neurosurgery, pathology, radiation oncology, and molecular genetics for optimal long-term outcomes [8, 9, 33].

Conclusion

In conclusion, we report a rare case of a sparsely granulated somatotroph pituitary macroadenoma presenting with progressive acral enlargement and visual field impairment. Prompt recognition of acromegaly and timely surgical intervention are critical to prevent

irreversible visual loss and systemic complications. Histopathological characterization is essential for predicting tumor behavior and guiding postoperative management.

SGSAs represent an uncommon and clinically challenging subset of growth-hormone secreting pituitary adenomas, with unique pathological and clinical features. Comprehensive multimodal management and careful long-term follow-up are essential to optimise patient outcomes and minimise morbidity.

Disclosure

Conflict of interest

The authors declare no conflict of interest.

Informed consent

Informed consent was obtained from the patient.

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