

Multiple Osteolytic Lesions With a "Superscan" on Bone Scintigraphy as the Presenting Manifestation of a Parathyroid Adenoma in a 29-Year-Old Woman: A Case Report

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Abstract

Overt osteitis fibrosa cystica is an uncommon presentation of primary hyperparathyroidism (PHPT) in young adults. A 29-year-old woman presented with low back pain of one year's duration. Lumbar computed tomography (CT) showed multiple osteolytic lesions, and bone scintigraphy showed a metabolic "superscan". Biochemistry showed the following: parathyroid hormone (PTH) 1807.3 pg/mL, hypercalcemia (3.06 mmol/L), hypophosphatemia, alkaline phosphatase 1489 U/L, and severe vitamin D deficiency. Ultrasound and dual-phase sestamibi single-photon emission computed tomography/computed tomography (SPECT/CT) localized a single left inferior parathyroid lesion. Parathyroidectomy yielded an encapsulated adenoma (~5.9 g) without capsular or vascular invasion. PTH fell to 30.9 pg/mL on day 1 but rose to 310.3 pg/mL by day 64, while calcium remained mildly low (2.03 mmol/L), consistent with compensatory skeletal remineralization and vitamin D deficiency rather than proven residual disease. A neuropsychiatric event at 13 months was of uncertain relationship to PHPT.

Categories: Nuclear Medicine, Radiology, Endocrinology/Diabetes/Metabolism

Keywords: bone scintigraphy, hungry bone syndrome, parathyroid adenoma, primary hyperparathyroidism, spect/ct, superscan

Introduction

Primary hyperparathyroidism (PHPT) results from the autonomous overproduction of parathyroid hormone (PTH); a solitary parathyroid adenoma accounts for approximately 80–85% of cases [1,2]. Where biochemical screening is routine, PHPT is usually detected as asymptomatic mild hypercalcemia in middle-aged adults, and overt skeletal disease has become infrequent [3,4]. When advanced bone disease does occur, it can dominate the picture and pose a diagnostic challenge, particularly in a young patient in whom malignancy is more readily suspected.

Chronic PTH excess accelerates osteoclastic resorption, progressing from subperiosteal resorption to osteitis fibrosa cystica, in which expansile lytic lesions ("brown tumors") may mimic malignancy [5]. On bone scintigraphy, diffusely accelerated skeletal turnover can produce a "superscan"-intense, symmetric osseous uptake with faint or absent renal activity. Although classically associated with diffuse skeletal metastases, a superscan is also well recognized in metabolic bone disease, including hyperparathyroidism, renal osteodystrophy, and osteomalacia [6], and distinguishing these causes is essential. Ultrasound and technetium-99m (^{99m}Tc) sestamibi scintigraphy with single-photon emission computed tomography/computed tomography (SPECT/CT) are first-line localization modalities [7–9].

We describe a young woman in whom unexplained multiple lytic lesions and a metabolic superscan led, through integrated biochemistry, bone scintigraphy, and sestamibi SPECT/CT, to the localization and histopathologic confirmation of a parathyroid adenoma. The case illustrates the diagnostic challenge of a superscan with multiple lytic lesions in a young adult and the difficulty of interpreting a secondary postoperative PTH rise when follow-up is incomplete.

Case Presentation

A 29-year-old woman presented to the orthopedic department of our hospital with persistent low back pain of one year's duration. The pain had begun without identifiable trauma or other precipitating event and had not improved despite repeated medical evaluations elsewhere. Physical examination revealed tenderness between the fourth and fifth lumbar spinous processes. The straight-leg-raising test was negative, and muscle strength, cutaneous sensation, and deep tendon reflexes of both lower extremities were normal; no pathologic reflexes were elicited. No palpable neck mass was present, and there were no documented symptoms of nephrolithiasis, polyuria, polydipsia, constipation, or mood disturbance.

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Initial laboratory investigations at the time of presentation are summarized in Table 1. Biochemical evaluation demonstrated marked hyperparathyroidism consistent with PHPT. Serum alkaline phosphatase (ALP), creatinine, 25-hydroxyvitamin D, and 24-hour urinary calcium were not documented in the available records.

Parameter	Patient value	Reference range
Parathyroid hormone (pg/mL)	1807.3	12.4-76.8
Total calcium (mmol/L)	3.06	2.11-2.52
Phosphate (mmol/L)	0.56	0.85-1.51
Alkaline phosphatase (U/L)	1489	35-100
Creatinine (μmol/L)	41	41-81
25-Hydroxyvitamin D (ng/mL)	<4.2 (severe deficiency)	30-100
24-hour urinary calcium	Not performed	-

TABLE 1: Initial biochemical investigations.

Lumbar CT without contrast, performed before the biochemical diagnosis, showed multiple irregular osteolytic lesions of indeterminate cause involving the lumbar vertebrae, sacrum, and bilateral iliac bones (Figure 1A-1B). To map skeletal involvement, whole-body bone scintigraphy was performed two hours after the injection of 740 MBq (20 mCi) of 99mTc methylene diphosphonate (MDP) on a dual-head gamma camera with a low-energy high-resolution collimator, 140-keV photopeak with a 15% window, planar matrix 256×1024 at 18 cm/min (zoom 1.0), and SPECT matrix 128×128 (32 views, 15 s/view); SPECT/CT used 130 kVp with adaptive tube current and 5-mm slices. Planar images showed diffuse, symmetrically increased skeletal uptake with poor renal and bladder visualization, consistent with a superscan (Figure 1C); SPECT/CT showed multiple osteolytic lesions in several ribs, the thoracolumbar spine, the sacrum, and both iliac bones with heterogeneous uptake (Figure 1D). Because the CT lesions were indeterminate, the images were initially interpreted with a broad differential that included malignancy; attribution to hyperparathyroid bone disease was made only after the markedly elevated PTH and calcium became available. The patient had no known primary malignancy or constitutional features of neoplasia.

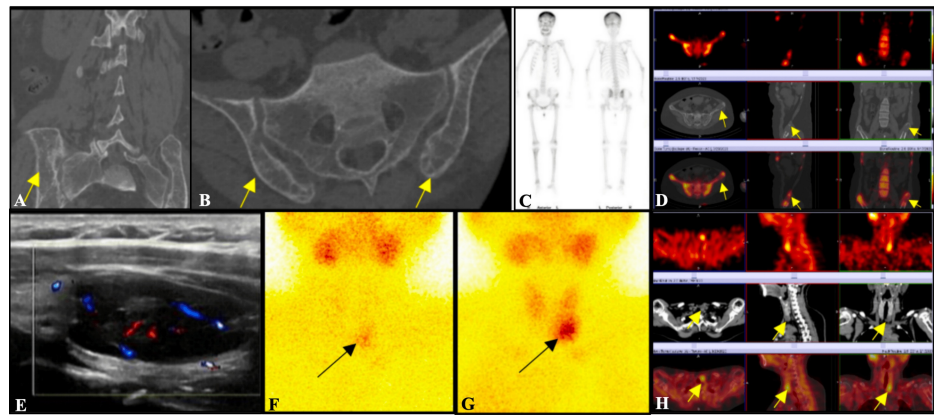


FIGURE 1: Imaging findings in a 29-year-old woman with primary hyperparathyroidism due to a parathyroid adenoma.

(A, B) Coronal and axial lumbar CT images showing multiple irregular osteolytic lesions (arrows) involving the lumbar vertebrae, sacrum, and bilateral iliac bones.

(C) Whole-body planar ^{99m}Tc -MDP bone scintigraphy (two hours after 740 MBq/20 mCi; low-energy high-resolution collimator, 140 keV $\pm$ 15%, matrix 256 $\times$ 1024, 18 cm/min) showing diffusely and symmetrically increased skeletal uptake with poor renal and bladder visualization, consistent with a "superscan".

(D) Fused pelvic SPECT/CT image from the same single-tracer MDP examination (SPECT 128 $\times$ 128, 32 views; CT 130 kVp, 5-mm slices) showing osteolytic lesions with heterogeneous radiotracer uptake (arrows).

(E) Cervical color Doppler ultrasound showing a well-defined hypoechoic, hypervascular solid nodule (2.84 $\times$ 1.29 cm) posterior to the inferior pole of the left thyroid lobe.

(F, G) Early (15 min, F) and delayed (120 min, G) planar images from a dual-phase single-tracer ^{99m}Tc -sestamibi examination (740 MBq/20 mCi; matrix 256 $\times$ 256, zoom 2.67; no pertechnetate subtraction) showing a focal focus inferior to the left thyroid lobe (arrows) that persists after thyroid washout.

(H) Fused neck sestamibi SPECT/CT image (CT 130 kVp, 5-mm slices) showing a well-circumscribed low-density lesion (1.7 $\times$ 1.2 cm) inferior to the left thyroid lobe with markedly increased uptake (arrow), consistent with a hyperfunctioning parathyroid adenoma.

CT: computed tomography; ^{99m}Tc -MDP: technetium-99m methylene diphosphonate; SPECT/CT: single-photon emission computed tomography/computed tomography

Cervical color Doppler ultrasound showed a well-defined hypoechoic hypervascular solid nodule measuring 2.84 $\times$ 1.29 cm posterior to the inferior pole of the left thyroid lobe (Figure 1E). Parathyroid scintigraphy was then performed as a dual-phase single-tracer ^{99m}Tc -sestamibi study (740 MBq/20 mCi) with planar imaging at 15 and 120 min and neck SPECT/CT; no ^{99m}Tc -pertechnetate dual-tracer acquisition or digital subtraction was used. Acquisition used a low-energy high-resolution collimator, 140 keV/15% window, planar matrix 256 $\times$ 256 (zoom 2.67, 3-min acquisition), and SPECT matrix 128 $\times$ 128 (zoom 1.0; 32 views, 15 s/view), with CT at 130 kVp, adaptive current, and 5-mm slices. Early images showed thyroid activity with a focal focus at the left inferior pole (Figure 1F); delayed images showed uniform thyroid washout with persistent focal uptake (Figure 1G), localized by SPECT/CT to a well-circumscribed low-density lesion measuring 1.7 $\times$ 1.2 cm immediately inferior to the left thyroid lobe (Figure 1H). Ultrasound and sestamibi were concordant for a single left inferior hyperfunctioning lesion. The dimensions differed between modalities; correlation with the fixed specimen (3.0 $\times$ 2.5 $\times$ 1.5 cm; see below) showed that the ultrasound measurement (2.84 cm in greatest imaged dimension) was closer to the pathological size than the SPECT/CT measurement (1.7 cm), which underestimated the lesion.

The patient underwent resection of the left inferior parathyroid gland together with bilateral partial thyroidectomy. The thyroid procedure was indicated by coexisting thyroid nodules documented on preoperative ultrasound, a right-lobe nodule categorized as Thyroid Imaging Reporting and Data System (TI-RADS) 4a, a left-lobe colloid cyst (TI-RADS 2), and multiple left-lobe nodules (TI-RADS 3), with no enlarged cervical lymph nodes; histopathology of the resected thyroid showed nodular goiter with focal fibrosis on the right and nodular goiter on the left, with no malignancy. Intraoperatively, the remaining parathyroid glands were explored and appeared unremarkable; intraoperative PTH monitoring was not performed. Histopathology demonstrated an encapsulated parathyroid neoplasm measuring 3.0 $\times$ 2.5 $\times$ 1.5 cm and weighing approximately 5.9 g, composed predominantly of chief cells in sheets and nests with uniform nuclei; no capsular or vascular invasion was identified, consistent with a parathyroid adenoma. Mitotic

count, Ki-67 index, and parafibromin immunohistochemistry were not performed. Back pain improved markedly after surgery. A neck ultrasound on day 64 showed no sonographically evident residual parathyroid lesion; a negative ultrasound cannot, however, exclude small or ectopic hyperfunctioning tissue, and no postoperative sestamibi study or other localization investigation was performed.

Given the young age and unusually severe phenotype, hereditary causes were considered. No suggestive family history or clinical evidence of multigland disease was found, and the other three glands appeared normal at exploration, but formal genetic evaluation (e.g., MEN1, CDC73-related hyperparathyroidism-jaw tumor syndrome, CASR-related disorders, and other familial syndromes) was not performed [10].

Postoperative PTH and calcium trajectory

The postoperative course and calcium/vitamin D treatment are summarized in Table 2. Values are discrete documented measurements. PTH fell to within the reference range on day 1, supporting the removal of the dominant hypersecreting tissue, but rose again during documented hypocalcemia and remained elevated on day 64, when calcium was still mildly below the lower reference limit. No postoperative sestamibi study, dual-energy X-ray absorptiometry (DXA), or biochemistry beyond day 64 is available.

Time relative to surgery	PTH (pg/mL) (12.4-76.8)	Total calcium (mmol/L) (2.11-2.52)	Calcium/vitamin D treatment
4 days before surgery	1807.3	3.06	-
Day 0 (surgery)	628.7	Not documented	IV 10% calcium gluconate, four 20-mL ampoules (2 g each)
Postoperative day 1	30.9	Not documented	-
Postoperative day 2	Not documented	Not documented	IV 10% calcium gluconate, four 20-mL ampoules (2 g each)
Postoperative day 3	196	1.77	-
Postoperative day 4	Not documented	Not documented	IV 10% calcium gluconate, two 20-mL ampoules (2 g each)
Postoperative day 7	Not documented	1.89	Oral calcium acetate 0.667 g once daily; vitamin D2 0.125 mg (50,000 IU) once daily for 5 days
Postoperative day 10	Not documented	2.04	Oral calcium acetate continued
Postoperative day 14	241	2	Oral calcium acetate (total duration not documented)
Postoperative day 64	310.3	2.03	Neck ultrasound: no sonographically evident residual parathyroid lesion

TABLE 2: Perioperative calcium and postoperative PTH trajectory and treatment (relative time; surgery=day 0).

PTH: parathyroid hormone

Late clinical follow-up: neuropsychiatric presentation

Thirteen months after surgery, the patient attended the neurology outpatient clinic with an acute neuropsychiatric presentation characterized by incoherent speech, self-muttering, disrobing, nocturnal elopement, and refusal to eat. Using the terminology of the original record, the attending neurologist documented a primary outpatient diagnosis of "mental disorder"; a more specific psychiatric or neurologic diagnosis was not available. No PTH, calcium, phosphate, or related biochemical tests were performed at that visit. The event is therefore reported as a late neuropsychiatric episode of uncertain relationship to PHPT rather than an established sequela (Figure 2).

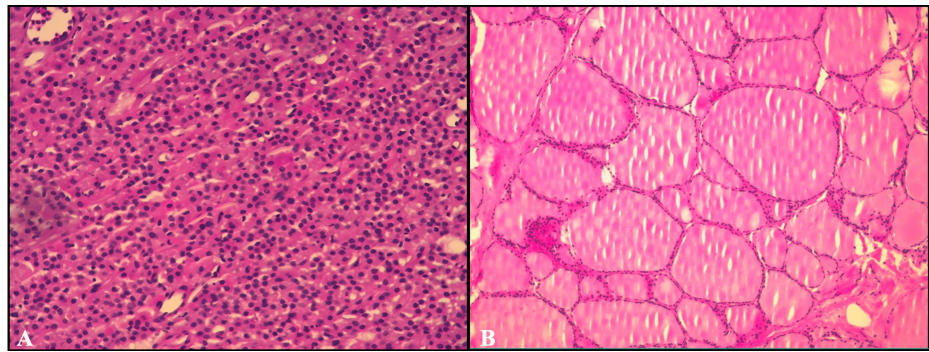


FIGURE 2: Histopathological findings of the excised lesion (hematoxylin and eosin staining).

(A) The parathyroid neoplasm is composed of densely packed chief cells arranged in sheets and nests, with uniform round nuclei and scant eosinophilic cytoplasm; no significant atypia or mitotic activity is seen, consistent with a parathyroid adenoma (magnification $\times 200$).

(B) The tumor is demarcated from the adjacent thyroid tissue by a fibrous capsule; thyroid follicles filled with eosinophilic colloid are seen alongside the adenoma, without capsular or vascular invasion (magnification $\times 100$).

Discussion

Advanced hyperparathyroid bone disease in a young patient

PHPT predominantly affects adults aged 50–70 years and is about twice as common in women; occurrence in the third decade is unusual [1,2]. In screened populations, the classic "stones, bones, groans, and psychic moans" presentation has been largely replaced by incidental asymptomatic disease, and overt osteitis fibrosa cystica with brown tumors is now seen in fewer than 5% of patients, persisting mainly where diagnosis is delayed [3–5]. The markedly elevated ALP (1489 U/L, ~ 15 -fold the upper reference limit) and the year-long diagnostic delay in this patient indicate advanced high-turnover skeletal disease at presentation.

The superscan and the differential diagnosis of multiple lytic lesions

A superscan reflects a systemic increase in bone turnover [6]. Diffuse skeletal metastases are the best-recognized cause [11], but an identical planar appearance occurs in hyperparathyroidism, renal osteodystrophy, and osteomalacia [6,12]. Here, the remarkably homogeneous, symmetric involvement of the axial and appendicular skeleton in a young patient without known malignancy favored a metabolic origin, but planar and SPECT/CT findings are non-specific: multiple osteolytic lesions with heterogeneous uptake overlap with malignant and other metabolic processes, and the decisive evidence was biochemical (the markedly elevated PTH, hypercalcemia, and hypophosphatemia) rather than the scintigraphic pattern or SPECT/CT morphology alone.

Because the patient had no history or features of malignancy, an extensive oncologic work-up was not pursued; serum and urine protein electrophoresis and immunofixation were not performed. Several benign causes of multiple lytic lesions were nevertheless considered. Renal osteodystrophy was effectively excluded by normal renal function (creatinine 41 $\mu\text{mol/L}$). Osteomalacia was considered in view of the profound vitamin D deficiency (25-hydroxyvitamin D < 4.2 ng/mL), but osteomalacia produces low or low-normal, not elevated, calcium, and the frank hypercalcemia, hypophosphatemia, and extreme PTH elevation argued against it as the primary process; vitamin D deficiency coexisted and was treated. Polyostotic fibrous dysplasia typically shows expansile ground-glass lesions, which were absent. Langerhans cell histiocytosis and giant cell tumor have characteristic distributions and do not explain the diffuse symmetric superscan or the biochemical triad. Multiple myeloma produces punched-out, often photopenic lesions [11,13]; the PHPT biochemistry strongly favored hyperparathyroid bone disease, although without electrophoresis a coexisting plasma-cell disorder cannot be formally excluded. The unifying diagnosis of osteitis fibrosa cystica with brown tumors rests on the lytic lesions, metabolic superscan, classic PHPT biochemistry, concordant localization, and histopathology.

Functional imaging and presurgical localization

Ultrasound is operator dependent and does not characterize gland function, whereas sestamibi identifies functionally hyperactive tissue, and SPECT/CT adds anatomic localization; dual-phase sestamibi has a sensitivity of approximately 80–90% for solitary adenomas [7–9,14–16]. In this case, the two modalities were concordant in location but not in size (ultrasound 2.84×1.29 cm; SPECT/CT 1.7×1.2 cm). The fixed specimen measured $3.0 \times 2.5 \times 1.5$ cm, so the ultrasound measurement approximated the pathological greatest dimension, whereas SPECT/CT underestimated it; we report this as a documented intermodality discrepancy.

resolved by specimen correlation rather than as presumed ultrasound overestimation [15].

Surgery, pathology, and the adenoma-carcinoma distinction

Parathyroid carcinoma accounts for under 1% of PHPT and is suggested by very high calcium and PTH, severe target-organ disease, and a large, heavy gland; histologic distinction rests on capsular or vascular invasion and invasive growth, whereas cytologic atypia alone is insufficient [17]. Our lesion was encapsulated with no capsular or vascular invasion identified on histopathology, supporting adenoma, and there was no gross invasion at surgery. We note, however, that the gland weighed approximately 5.9 g and measured 3 cm, exceeding the size/weight range (~3–3.5 g) that should prompt particularly careful exclusion of carcinoma; weight and size alone are non-specific, but the absence of reported mitotic count, Ki-67 index, and parafibromin immunohistochemistry limits complete characterization, and standardized pathology review, including parafibromin staining, which helps distinguish adenoma from atypical adenoma and carcinoma [18], has been recommended to the treating team. Our conclusion rests on the histopathologic (not intraoperative) findings: absence of gross invasion at surgery does not itself exclude carcinoma because invasion is a microscopic determination. Intraoperative exploration found the other glands unremarkable, arguing against multigland disease, but without intraoperative parathyroid hormone (ioPTH) monitoring, functional confirmation of complete resection was not obtained; the Miami criterion (>50% ioPTH decline 10 minutes after excision) could not be applied [19].

Postoperative PTH dynamics

The day-1 fall from 1807.3 to 30.9 pg/mL strongly supported the successful removal of the dominant hypersecreting gland but, particularly without ioPTH and in light of the later rise, does not by itself establish the removal of all hyperfunctioning tissue. PTH then rose to 196 pg/mL on day 3 with marked hypocalcemia (1.77 mmol/L), 241 pg/mL on day 14 (calcium 2.00 mmol/L) and 310.3 pg/mL on day 64 (calcium 2.03 mmol/L, still below the 2.11 mmol/L lower limit), despite calcium and short-course vitamin D supplementation.

Three non-mutually exclusive explanations apply. First, and best supported here, compensatory stimulation from skeletal remineralization: in severe high-turnover bone disease (ALP ~15-fold elevated), rapid post-resection mineralization generates prolonged calcium demand, a mild or protracted form of hungry bone syndrome [20], and the documented profound hypocalcemia with intensive calcium requirement supports this. Second, severe vitamin D deficiency (25-hydroxyvitamin D <4.2 ng/mL), only briefly replaced, would maintain a secondary PTH drive. Third, a small residual or ectopic focus cannot be excluded; the negative day-64 ultrasound reduces but does not eliminate this possibility.

Crucially, persistent PHPT cannot be established in the presence of hypocalcemia: the patient never showed postoperative hypercalcemia and remained mildly hypocalcemic on day 64. Taken with the profound osteitis fibrosa cystica, the elevated ALP, and severe vitamin D deficiency, the pattern is more consistent with a secondary, compensatory PTH response than with residual autonomous disease. Biochemical cure is conventionally defined by persistent normocalcemia (with normal PTH where measured) for at least six months [1,2]; follow-up here ends at day 64, so sustained remission cannot be assessed. The patient should be recalled for PTH, total and ionized calcium, phosphate, ALP, renal function, 25-hydroxyvitamin D, and DXA, with adequate calcium/vitamin D repletion, and sestamibi re-imaging only if hypercalcemia or rising PTH suggests persistence.

Hereditary considerations

PHPT in the third decade warrants consideration of inherited disease, including MEN1, MEN2A, CDC73-related hyperparathyroidism-jaw tumor syndrome, CASR-related disorders, and familial isolated hyperparathyroidism [10]; a monogenic diagnosis is found in a meaningful proportion of tested patients, particularly with young age, multigland disease, or a suggestive family history. Our patient had no reported family history, apparently single-gland disease, and normal-appearing remaining glands, but genetic testing was not performed and remains recommended given her young age and severe phenotype [10].

Neuropsychiatric manifestations

Neuropsychiatric symptoms are a recognized component of PHPT, ranging from anxiety, depression, and cognitive slowing to, rarely, frank psychosis [21,22]. Mechanistic inference is not possible here: 13 months postoperatively, the patient had an acute episode recorded only as "mental disorder", without contemporaneous PTH or calcium measurements or a specific diagnosis. We therefore present it as a late neuropsychiatric event of uncertain relationship to PHPT; it can be neither attributed to persistent hyperparathyroidism nor excluded as a coincidental primary psychiatric disorder.

Limitations

This is a single retrospective case. Twenty-four-hour urinary calcium was not measured, serum and urine electrophoresis/immunofixation were not performed, and no DXA was obtained. Early postoperative calcium monitoring was incomplete, and follow-up ends at day 64, so sustained biochemical remission cannot be

assessed; no ioPTH monitoring or postoperative localization study was performed. Ki-67, parafibromin, and mitotic activity were not reported for an adenoma that weighed ~5.9 g, precluding complete exclusion of an atypical adenoma and motivating pathology review. Genetic testing for hereditary PHPT was not performed. The brief vitamin D replacement (five days) and calcium supplementation of undocumented total duration may have been inadequate for the degree of deficiency and bone disease. No biochemistry accompanied the late neuropsychiatric visit, so its relationship to PHPT remains undetermined.

Conclusions

PHPT due to a parathyroid adenoma can present in a young adult as multiple osteolytic lesions with a "superscan", a pattern more commonly associated with diffuse metastatic disease. A homogeneous symmetric superscan with lytic lesions in a young patient should prompt early calcium and PTH measurement; here, the biochemical constellation, rather than imaging alone, was decisive, with bone scintigraphy defining skeletal extent and dual-phase sestamibi SPECT/CT localizing the adenoma. Histopathology showed an encapsulated adenoma without identified invasion, although the absence of Ki-67/parafibromin studies warrants pathology review. The postoperative PTH rebound with persistent mild hypocalcemia, markedly elevated ALP, and severe vitamin D deficiency is most consistent with compensatory skeletal remineralization with vitamin D deficiency rather than proven residual disease; with follow-up limited to day 64, sustained biochemical remission cannot be claimed, and long-term biochemical and bone-density follow-up with adequate calcium/vitamin D repletion is warranted. The month-13 neuropsychiatric event was of uncertain relationship to PHPT and should not be read as an established sequela or the basis for a specific surveillance recommendation.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Yong Yang, Xia Ma, Wei Cui, Jingxia Wang

Critical review of the manuscript for important intellectual content: Yong Yang, Xia Ma, Wei Cui, Jingxia Wang

Supervision: Yong Yang

Acquisition, analysis, or interpretation of data: Xia Ma, Wei Cui, Jingxia Wang

Drafting of the manuscript: Xia Ma

Disclosures

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