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(CASE REPORT)

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Parathyroid Water-Clear Cell Hyperplasia: Case Report and a Brief Review of the Literature

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Abstract

Parathyroid water-clear cell hyperplasia (WCCH) is an exceptionally rare cause of primary hyperparathyroidism, characterized by the multiglandular replacement of parathyroid tissue by large vacuolated hyperplastic parathyroid cells. Its clinical significance lies in its rarity and the diagnostic difficulties it presents, particularly the frequent failure of preoperative localization studies.

We present a case of a 49-year-old female who presented with an eight-month history of fatigue, nausea, and weakness. Biochemical evaluation revealed mild hypercalcemia with markedly elevated parathyroid hormone (iPTH), consistent with primary hyperparathyroidism. Preoperative neck ultrasound and sestamibi SPECT/CT were equivocal, suggesting possible multiglandular disease without localizing a dominant adenoma. A multidisciplinary tumor board recommended bilateral neck exploration. During the operation, surgeons discovered that each of the four parathyroid glands was enlarged, weighing a combined 31.6 grams. A total parathyroidectomy with forearm autotransplantation was performed. Histopathology confirmed diffuse WCCH. The patient developed transient but severe postoperative "hungry bone syndrome" requiring aggressive supplementation, with eventual normalization of calcium and successful autograft function. At two-year follow-up, she remains normocalcemic and asymptomatic.

This case highlights that WCCH can present with massive glandular enlargement despite only mild-to-moderate biochemical abnormalities. It underscores the unreliability of standard localization imaging for WCCH. It reinforces the essential role of bilateral neck exploration as the definitive diagnostic and therapeutic procedure when multiglandular disease is suspected.

Keywords: Parathyroid; Water-clear cell hyperplasia; Water-clear cell adenoma; Parathyroid hormone; Hyperparathyroidism; Immunohistochemistry

1. Introduction

WCCH is a rare and distinctive cause of primary hyperparathyroidism (PHPT), accounting for less than 1% of cases in contemporary series. [1]

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Classic WCCH is typically a multiglandular disease, often with marked enlargement of the upper glands before the lower glands are affected. [2] Histologically, the glands are composed of large polygonal cells with abundant clear, finely vacuolated cytoplasm, thought to reflect dilated intracellular organelles. [3] Water-clear cells are not a component of normal parathyroid tissue but are believed to arise from chief cells and are associated with parathyroid hyperfunction. [1] The absence of a fibrous capsule helps distinguish WCCH from the rarer water-clear cell adenoma (WCCA), a distinction of clinical importance given differences in surgical management. [4]

Clinically, WCCH often presents mild hypercalcemia and modest elevations in parathyroid hormone (PTH) levels. [5] Symptoms are frequently nonspecific, such as fatigue, constipation, or abdominal discomfort, and imaging findings are variable and usually non-diagnostic. [6] Notably, gland size does not correlate with biochemical severity, as water-clear cells contain relatively low PTH per unit of tissue. [6] Definitive diagnosis relies on histopathologic evaluation, with immunohistochemical (IHC) studies typically showing PTH positivity. [7]

Given its multiglandular nature, limited or focused parathyroidectomy may result in persistent or recurrent disease, underscoring the importance of accurate recognition and appropriate surgical planning. [8] Owing to its extreme rarity, current understanding of WCCH remains limited, and further case documentation is needed to clarify optimal management and long-term outcomes.

2. Case presentation

2.1. Clinical Presentation

A 49-year-old Female with a past medical history of mild osteopenia and gastroesophageal reflux disease presented to her primary care physician, reporting progressive fatigue, persistent low-grade nausea, and vague abdominal discomfort over the preceding eight months. She also noted worsening mild weakness and occasional constipation. Initial outpatient laboratory screening, prompted by her fatigue, revealed mild hypercalcemia and elevated parathyroid hormone (iPTH). She was referred to endocrinology, where repeat biochemistry confirmed the findings. She was started on oral hydration and dietary calcium restriction and advised to increase fluid intake. Despite these conservative measures, her symptoms persisted over the subsequent three months, prompting referral to a head and neck endocrine surgeon for further evaluation and consideration of surgical intervention. Notably, she denied any personal or family history of nephrolithiasis, multiple endocrine neoplasia syndromes, or prior neck radiation.

2.2. Evaluation: Examination, History, Labs, and Imaging

Physical examination was unremarkable. No palpable neck mass, lymphadenopathy, or thyroid abnormality was identified. Family and personal history were negative for MEN-1, MEN-2, hyperparathyroidism-jaw tumor syndrome, or neurofibromatosis type 1.

Laboratory workup demonstrated serum calcium of 2.85 mmol/L (mildly elevated), iPTH of 186 pg/mL (markedly elevated), mildly elevated alkaline phosphatase, low-normal phosphorus, mildly reduced 25-hydroxyvitamin D, and 24-hour urinary calcium within the upper limit of normal. A DEXA scan confirmed low-grade osteopenia at the lumbar spine.

Neck ultrasound demonstrated bilateral multilobulated hypoechoic masses in both the superior and inferior parathyroid regions, with the largest lesion measuring approximately 4.2 cm on the left inferior position. Technetium-99m sestamibi SPECT/CT showed equivocal bilateral uptake with poor tracer retention, failing to localize a dominant adenoma clearly. The pattern suggested possible multiglandular disease rather than a single-gland process.

Radiographic differential diagnosis included: multiglandular parathyroid hyperplasia (primary or secondary), multiple parathyroid adenomas, parathyroid carcinoma with local spread, and ectopic or supernumerary parathyroid tissue.

2.3. Multidisciplinary Tumor Board Discussion

Given the imaging findings of bilateral enlargement of all four parathyroid glands and equivocal Sestamibi scan results, the case was presented to the multidisciplinary head and neck endocrine tumor board. Discussion centered on several key points: the strong likelihood of multiglandular hyperplasia versus multiple adenomas; the appropriate surgical strategy (subtotal parathyroidectomy versus total parathyroidectomy with auto transplantation); the need for bilateral neck exploration regardless of imaging; and the known limitations of sestamibi scintigraphy in water-clear cell lesions, which often show poor tracer uptake despite considerable glandular enlargement. Fine needle aspiration (FNA) was not considered for early evaluation, as it would not be useful to determine adenoma from hyperplasia.

The board recommended proceeding with bilateral neck exploration with intraoperative PTH monitoring and careful identification of all four parathyroid glands. The surgical plan included subtotal parathyroidectomy (three-and-a-half gland resection) or total parathyroidectomy with auto transplantation, with the final decision to be guided by intraoperative findings and frozen section pathology assessment.

2.4. Management, Pathology, IHC, Molecular Studies, and Final Diagnosis

The patient underwent bilateral neck exploration under general anesthesia with continuous intraoperative iPTH monitoring. All four parathyroid glands were identified and found markedly enlarged, with a characteristic mahogany-brown color. The glands weighed 7.2 g (left superior), 9.8 g (left inferior), 6.5 g (right superior), and 8.1 g (right inferior), all of which were significantly above normal parathyroid weight. Given the diffuse four-gland involvement identified intraoperatively and frozen section findings suggestive of diffuse hyperplasia, a total parathyroidectomy with auto-transplantation was performed. Approximately 50 mg of minced parathyroid tissue from the most normal-appearing portion of the right superior gland was autotransplanted into the left brachioradialis muscle with marking sutures for easier monitoring and potential re-excision if the disease recurs. Intraoperative iPTH declined by greater than 80% within 15 minutes of total gland excision.

Final histopathological examination of all four glands revealed diffuse replacement of normal parathyroid architecture by sheets of large polygonal cells with abundant pale, vacuolated, finely foamy "water-clear" cytoplasm and eccentric, round, dark-staining nuclei. No residual normal parathyroid tissue was identified in any specimen. There was no fibrous capsule, capsular invasion, vascular invasion, or mitotic activity. (Figure 1 A, B, C) Histomorphology excluded the possibility of clear cell adenoma (WCCA). In the specific context of WCCH or WCCA, the IHC profile typically confirms the parathyroid origin while ruling out malignancy. IHC panel demonstrated the following: PTH: Diffusely and strongly positive, p27: Positive (majority of cells), Bcl-2: Positive, PGP9.5: Negative, Parafibromin: Retained (intact expression), Ki-67: Very low proliferation index (<1%), Thyroglobulin / TTF-1: Negative, PAX-8 / CA-IX / PAX-2: Negative, excluding metastatic renal cell carcinoma, CD56 / Chromogranin / Synaptophysin / Calcitonin: Negative, excluding paraganglioma and medullary thyroid carcinoma, GCM2: Positive, confirming parathyroid origin.

No molecular or genetic studies for MEN-associated mutations were indicated, as WCCH is not linked to hereditary endocrine syndromes. Final diagnosis: Parathyroid water-clear cell hyperplasia (WCCH), all four glands, with primary hyperparathyroidism.

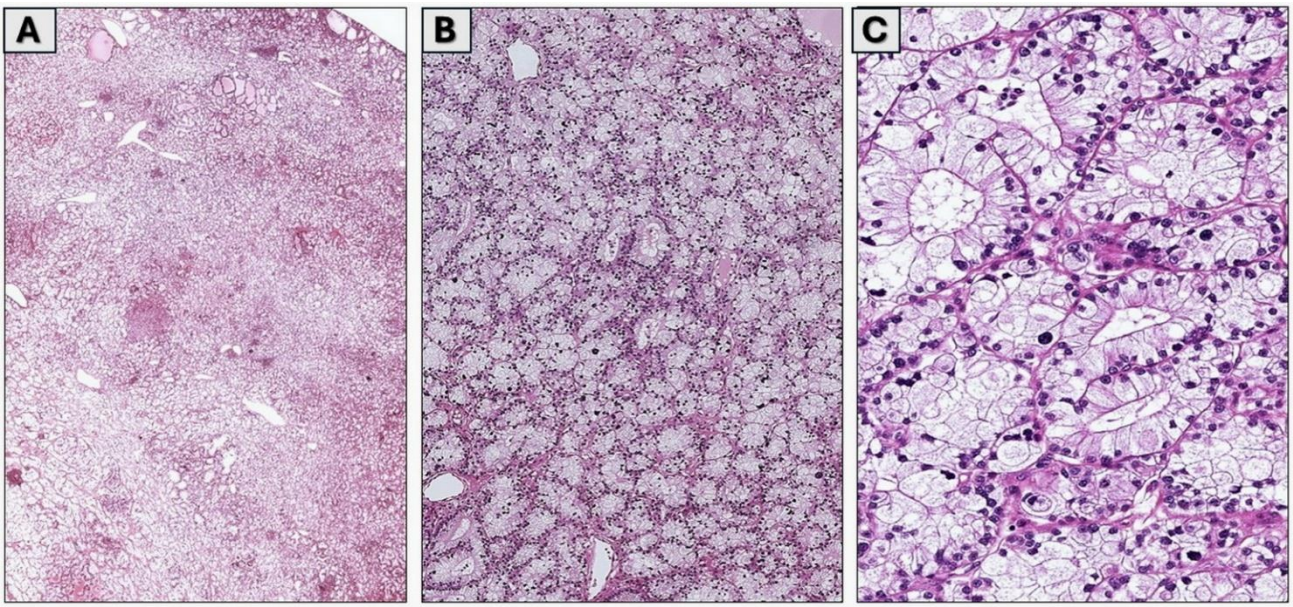
2.5. Outcome, Follow-Up, Complications, and Surveillance

Postoperatively, serum calcium and iPTH dropped precipitously. The patient developed transient severe hypocalcemia within 24 hours, requiring aggressive intravenous calcium gluconate supplementation followed by high-dose oral calcium carbonate (2-3 grams elemental calcium daily in divided doses) and calcitriol (0.5-1.0 mcg twice daily). This "hungry bone syndrome" reflected rapid skeletal calcium uptake following correction of chronic hyperparathyroidism.

Over the subsequent 6-8 weeks, calcium and vitamin D supplementation were gradually tapered as the auto-transplanted parathyroid tissue demonstrated functional engraftment. Serial iPTH measurements showed gradual recovery from undetectable levels to low-normal range by three months postoperatively.

The patient's symptoms of fatigue, nausea, and constipation resolved completely within the first month. She experienced no complications related to recurrent laryngeal nerve injury or permanent hypoparathyroidism. Long-term surveillance consisted of monitoring serum calcium, iPTH, phosphorus, and vitamin D every 3 months for the first year, then every 6 months thereafter. Repeat DEXA scanning was performed annually.

At two-year follow-up, the patient remained clinically well and normocalcemic on maintenance calcitriol (0.25 mcg daily) and calcium supplementation (500 mg daily). iPTH remained in the low-normal range, confirming successful autograft function. Bone density improved significantly in the lumbar spine and femoral neck. No clinical or laboratory evidence of recurrent hyperparathyroidism was identified.



1A**:** Low power view showing normal follicular structure of the parathyroid gland largely replaced by a solid, sheet-like, and nesting proliferation of cells, no thick capsule around the mass (H&E Stain X20).

1B: Intermediate power view showing the cells with clear cell features organized into solid sheets and small acini. (H&E Stain X40).

1C: High power view showing "water-clear" or "foamy" cells due to abundant cytoplasmic vacuoles. The cell membranes are sharply outlined, giving the tissue a distinct "plant-like" or "honeycomb" appearance. The nuclei are generally small, hyperchromatic, and located at the periphery of the cells. There is a lack of significant mitotic activity or atypia (H&E Stain X60)

Figure 1 Microscopic examination of excised parathyroid glands with water-clear cell hyperplasia

3. Discussion

3.1. Background (History, epidemiology, risk factors, and WHO classification):

Primary hyperparathyroidism (PHPT) has been recognized as a distinct clinical and pathological entity since the early twentieth century, with substantial advances in its histopathologic classification over time. [9] A single parathyroid adenoma accounts for the majority of PHPT cases (approximately 85%), while 10–15% involve multiglandular disease, including various forms of hyperplasia, such as WCCH. [10]

Water-clear cells were first described in the parathyroid gland in the mid-twentieth century. First described by Fuller Albright in 1934, WCCH has become increasingly uncommon, with its reported incidence declining markedly since the early twentieth century. [11] It predominantly affects women, with an approximate 6:1 female-to-male ratio and is typically not associated with a family history of PHPT or with multiple endocrine neoplasia syndromes. [10] Importantly, unlike other multiglandular forms of PHPT, WCCH has no established malignant potential. [2] Because of their morphologic resemblance to clear cell tumors of the neck, particularly renal cell carcinoma, their presence initially posed diagnostic challenges for pathologists. [5] WCCH is distinguished from classic chief cell hyperplasia by glands that are disproportionately enlarged relative to the degree of biochemical hyperparathyroidism. It typically involves more than one gland, reflecting varying degrees of hyperplastic change. [5] This multiglandular pattern, combined with imaging studies that may suggest single-gland disease, can complicate surgical planning and intraoperative decision-making.

Unlike certain other forms of parathyroid hyperplasia, WCCH is not associated with inherited endocrine neoplasia syndromes such as Multiple Endocrine Neoplasia type 1, Multiple Endocrine Neoplasia type 2, or hyperparathyroidism–jaw tumor syndrome, and no consistent underlying genetic or molecular abnormality has been identified. [12] Patients typically present symptoms attributable to hypercalcemia or are diagnosed incidentally through biochemical screening. [1]

According to the 2022 World Health Organization Classification of Tumors of Endocrine Organs, WCCH is categorized as a benign hyperplastic parathyroid disorder and considered a non-neoplastic multiglandular process. The WHO

emphasizes distinguishing WCCH from parathyroid adenoma and carcinoma based on architectural features, the absence of a fibrous capsule or vascular invasion, a low proliferative index, and intact parafibromin expression. [13] [14] [15] These distinctions carry significant implications for surgical management, recurrence risk, and long-term follow-up.

3.2. Pathogenesis, Pathophysiology

Parathyroid proliferative disorders are commonly associated with dysregulation of cell-cycle control. Alterations in the MEN1 tumor suppressor gene and overexpression of cyclin D1 (CCND1) promote unregulated cellular proliferation in many parathyroid neoplasms. [16] [17] [18] Mutations in CDC73 (HRPT2) are more frequently linked to aggressive parathyroid tumors but are uncommon in benign hyperplasia. In contrast to monoclonal adenomas, hyperplasia typically involves all four glands and reflects a generalized increase in cellular mass rather than clonal expansion. [13] [19]

Excess parathyroid hormone (PTH) secretion leads to hypercalcemia through several mechanisms: stimulation of osteoclast-mediated bone resorption via RANKL signaling, increased renal tubular calcium reabsorption, and enhanced vitamin D activation, which augments intestinal calcium absorption. Concurrently, increased renal phosphate excretion results in hypophosphatemia. [20] [21] These pathophysiologic processes explain the classic manifestations of hypercalcemia, commonly summarized by the mnemonic "stones, bones, groans, thrones, and psychiatric overtones," referring respectively to nephrolithiasis, reduced bone mineral density, gastrointestinal symptoms, polyuria, and neurocognitive disturbances. [22]

3.3. Comparative Analysis of our Case with Existing Literature. (Clinical-radiology-pathology, lab, management, and outcome)

This case of parathyroid water-clear cell hyperplasia (WCCH) aligns with and diverges from the existing literature in several key aspects. Clinically, the patient's presentation with nonspecific symptoms like fatigue, nausea, and constipation alongside mild hypercalcemia is consistent with modern presentations of primary hyperparathyroidism, which are often subtle. [1] However, the sheer size and weight of the glands (totaling over 31g) are remarkable and more pronounced than in typical chief cell hyperplasia. The literature suggests that, despite their large size, water-clear cell lesions may not produce proportionately high levels of PTH. [2] This finding corresponds with the marked, but not astronomically elevated, iPTH in this patient relative to the gland mass.

Diagnostically, the case highlights a known challenge: the unreliability of preoperative localization studies for multiglandular disease, especially WCCH. The equivocal Sestamibi scan is a classic feature, as water-clear cells often exhibit poor tracer uptake. This reinforces the literature's emphasis on the limitations of imaging in predicting four-gland hyperplasia and the need to plan for bilateral neck exploration when multiglandular disease is suspected. [1] [12]

From a management perspective, the decision to perform a total parathyroidectomy with autotransplantation is the standard of care for diffuse, four-gland hyperplasia. [23] The intraoperative findings and successful postoperative outcome vindicated this approach. The development of severe "hungry bone syndrome" is also a well-documented consequence following the correction of long-standing hyperparathyroidism, and its management with aggressive calcium and calcitriol supplementation was appropriate. [2]

This case reinforces the concept that WCCH, while rare, is a distinct entity that cannot be reliably distinguished from other forms of hyperparathyroidism preoperatively. It underscores the importance of surgeon preparedness for bilateral exploration and complex decision-making based on intraoperative findings, rather than relying solely on imaging.

4. What Have we learned from this case?

This case of WCCH provides several practical clinical insights. The primary diagnostic lesson is the unreliability of preoperative imaging in multiglandular parathyroid disease. The equivocal sestamibi scan, despite significant glandular enlargement, serves as a red flag that clinicians should not be falsely reassured by negative or ambiguous localization studies, particularly when biochemical data strongly suggest primary hyperparathyroidism. This experience reinforces existing knowledge that WCCH is a "great mimicker" and can be indistinguishable from more common causes preoperatively.

For management, this case highlights the continued importance of bilateral neck exploration as a crucial strategy when multiglandular disease is on the differential. The decision to proceed to total parathyroidectomy with

autotransplantation, guided by intraoperative assessment, was critical for achieving a definitive cure and preventing recurrent disease, which is a known risk with subtotal resection in hyperplasia. This challenges any trend toward more limited, image-guided exploration in patients in whom multiglandular disease cannot be excluded. The severe postoperative hungry bone syndrome also reminds clinicians to be vigilant and prepared for aggressive metabolic management after correcting chronic, severe hyperparathyroidism. Ultimately, this case strengthens the argument for a comprehensive surgical approach in suspected hyperplasia.

Abbreviations:

- Parathyroid water-clear cell hyperplasia (WCCH);
- Parathyroid hormone (iPTH),
- primary hyperparathyroidism (PHPT);
- Immunohistochemistry (IHC).

5. Conclusion

This case report details a rare instance of primary hyperparathyroidism caused by diffuse, four-gland WCCH. We present this case to highlight the significant diagnostic and management challenges posed by this rare histological variant. The clinical presentation was deceptively mild relative to the profound glandular enlargement discovered intraoperatively, and preoperative imaging failed to accurately localize the disease, reinforcing the limitations of sestamibi scintigraphy in WCCH. The value of this report to the medical community lies in its reinforcement of key clinical principles: maintain a high index of suspicion for multiglandular disease when imaging is equivocal and underscore the indispensable role of comprehensive bilateral neck exploration and intraoperative decision-making for definitive management. This case serves as a valuable reminder that despite advances in imaging, sound surgical judgment remains paramount in the successful treatment of complex parathyroid disease.

Compliance with ethical standards

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All authors make the following declarations:

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- Financial relationships: All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might be interested in the submitted work.

Statement of ethical approval

Ethical review and approval were not required for this study involving human participants. The paper has been sufficiently anonymized to maintain the patient's confidentiality.

Statement of informed consent

This study was conducted as a retrospective review of archival pathology material collected during routine clinical care. All data were fully de-identified prior to analysis. No patient contact or intervention was involved, and informed consent was not required in accordance with institutional policies.

Data access statement

All relevant data are included in the paper.

Author contributions

All authors contributed equally to producing this manuscript.

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