Annals of Case Reports
Ali M, et al. Ann Case Rep: 11: 102608
www.doi.org/10.29011/2574-7754.102608
www.gavinpublishers.com

OPEN ACCESS

GAVIN PUBLISHERS

Case Report

Severe Primary Hyperparathyroidism with Atypical Parathyroid Neoplasm, Brown's Tumour, and Incidental Adrenal Adenoma

Mohamed Ali*, Nihal Mani, Anthony Sony

Department of Endocrinology, North Tees Hospital, UK

*Corresponding author: Mohamed Ali, Department of Endocrinology, North Tees Hospital, UK

Citation: Ali M, Mani N, Sony A. **(2026)**. Severe Primary Hyperparathyroidism with Atypical Parathyroid Neoplasm, Brown's Tumour, and Incidental Adrenal Adenoma. Ann Case Report. 11: 2608. DOI: 10.290112574-7754.102608

Received: 22 April 2026; **Accepted:** 27 April 2026; **Published:** 29 April 2026

Summary

Primary hyperparathyroidism (PHPT) is a condition most clinicians will encounter in its milder form an incidental hypercalcaemia, a modest PTH elevation, and a patient who feels reasonably well. What is far less common is the picture we encountered here: a patient in hypercalcaemic crisis with a jaw lesion, kidney injury, and a parathyroid mass that immediately raised the question of malignancy. Parathyroid carcinoma is responsible for fewer than 1% of PHPT cases, though its true incidence is likely underreported given the diagnostic difficulty [1,2].

We describe a middle-aged woman who presented with an adjusted calcium of 3.62 mmol/L, PTH of 157 pmol/L, and a maxillary Brown's tumour. During a subsequent admission she developed AKI in the context of ongoing hypercalcaemia. Staging CT also picked up an adrenal lesion, which added an additional layer of diagnostic complexity before surgery could safely proceed. Histopathology ultimately returned an atypical parathyroid neoplasm (TNM8: pTisN0) - a diagnosis that sits in an uncomfortable middle ground between benign and overtly malignant, and which carries its own long-term surveillance implications.

Background

PHPT is most often caused by a single benign adenoma, and many patients are identified incidentally on routine bloods without any symptoms at all. The stereotypical "*bones, stones, groans and psychic moans*" presentation is now relatively uncommon in the developed world, largely because of earlier biochemical detection. Severe PHPT is a different matter. When PTH climbs to many times the upper limit of normal and calcium approaches or exceeds 3.5 mmol/L, multi-organ involvement becomes a real clinical risk [2].

Brown's tumours are something of a forgotten entity in modern medicine. They are osteolytic, giant-cell rich lesions caused by localised areas of intense osteoclastic activity driven by chronic PTH excess. On imaging they can look alarming mimicking primary bone tumour or metastatic disease and without the clinical context of hypercalcaemia they are easily misinterpreted [13].

The distinction between benign adenoma, atypical neoplasm, and carcinoma remains genuinely difficult, even with histopathology in hand. The literature reflects this honestly: multiple case reports have described situations where preoperative assessment pointed one way and the final pathology went another [3,4].

Case Presentation

Our patient was a middle-aged woman with Class III obesity (BMI 42.12 kg/m²) referred by her GP after hypercalcaemia was found incidentally during investigation of persistent fatigue. She was otherwise reasonably well she mentioned polyuria but denied bone pain, abdominal symptoms, or any cognitive changes. There was no family history of endocrine tumours or anything that might

suggest a hereditary syndrome. Examination was unremarkable aside from a right-sided gingival swelling she had noticed a few weeks earlier, and no cervical lymphadenopathy or neck mass was palpable at that point.

What struck us early on was the biochemical severity. PTH at 157 pmol/L is not a number you see with a run-of-the-mill adenoma. The combination of that PTH level, the calcium, and the subsequent imaging findings kept parathyroid carcinoma firmly in mind throughout the workup, even before histopathology was available [2,5].

Investigations

Biochemical Workup

Initial bloods confirmed PHPT of significant severity. We calculated the urine calcium:creatinine clearance ratio at 2.7, which comfortably excluded familial hypocalciuric hypercalcaemia (FHH) an important early step before committing to surgical planning. The patient was subsequently admitted on a second occasion with a frank hypercalcaemic crisis; at that point the corrected calcium had risen further to 3.88 mmol/L and creatinine was 130 µmol/L, meeting criteria for AKI Grade 1. The key numbers are shown in Table 1.

Adjusted Calcium: 3.62–3.88 mmol/L (Reference: 2.20-2.60 mmol/L).
PTH (initial): 157 pmol/L (Reference: 1.8-7.8 pmol/L).
Creatinine (at crisis): 130 µmol/L (Reference: 45-84 µmol/L).
Alkaline Phosphatase (ALP): 360 IU/L (Reference: 30-130 IU/L).
Urine calcium: creatinine clearance ratio: 2.7 (FHH threshold: <0.02).

Table 1: Biochemical Parameters.

Imaging and Histopathology

SPECT-CT parathyroid imaging showed intense tracer uptake at the inferior pole of the right thyroid lobe, providing reasonable preoperative localisation. It is worth noting that SPECT-CT has well-recognised limitations in the context of large or atypical glands; the pattern of uptake here was consistent with a hyperfunctioning lesion but did not definitively separate carcinoma from adenoma [7-9].

The gingival biopsy was a key piece of the puzzle. Histopathology showed giant-cell-rich tissue consistent with a Brown's tumour confirming that this was not a primary oral malignancy, as had been a concern, but rather a skeletal manifestation of chronic PTH excess. Brown's tumours of this kind, particularly involving the jaw, have been described in the context of both benign and malignant hyperparathyroidism [13].

Staging CT demonstrated a 33 mm right parathyroid mass. Tumour size is one of the few preoperative clues that can help raise suspicion for carcinoma; lesions exceeding 3 cm are repeatedly flagged in the literature as a risk factor for malignant or atypical behaviour [1,5,7].

The CT also showed an incidental 12 mm left adrenal lesion. This required characterisation before surgery could proceed safely particularly given the need to exclude a phaeochromocytoma, which would carry significant anaesthetic risk. Adrenal CT washout measured 12 Hounsfield Units, which is essentially diagnostic of a lipid-rich benign adenoma. Plasma-free metanephrines were normal. With phaeochromocytoma excluded and MEN-1 considered unlikely given the clinical picture, we were comfortable proceeding to parathyroidectomy [13].

Differential Diagnosis

The main differentials considered during the workup were:

Parathyroid carcinoma: This was the primary concern given PTH >20 times the upper limit of normal, AKI, and a large mass. The literature suggests that PTH exceeding twice the upper reference limit should always prompt suspicion for malignancy [2,5].

Atypical parathyroid neoplasm: A tumour exhibiting some worrying features histologically thick fibrous capsule, fibrous banding, mitotic figures but falling short of definitive carcinoma criteria. This is where our patient ultimately landed, and it carries its own surveillance implications [4,10].

Benign parathyroid adenoma: Remained possible but felt less likely given the biochemical severity and tumour size. Standard adenomas do not typically produce PTH at this level.

MEN-1: Always worth considering when there are multiple endocrine findings. The adrenal adenoma raised this briefly, though the absence of family history and a normal pituitary screen made it less likely. Genetic counselling was discussed.

Treatment

The immediate priority was getting the calcium down. The patient was admitted for aggressive intravenous rehydration, and loop diuretics were used to promote calciuresis. When calcium remained elevated despite these measures, cinacalcet was started at 30 mg twice daily and titrated to 60 mg twice daily. Cinacalcet works by allosterically activating the calcium-sensing receptor, effectively lowering PTH secretion, and it is a useful bridge while awaiting surgery [13].

Once phaeochromocytoma had been excluded and the patient was medically stable, she underwent right parathyroidectomy and right hemithyroidectomy. The decision to include the hemithyroidectomy was deliberate when you cannot exclude carcinoma preoperatively, en bloc resection incorporating the ipsilateral thyroid lobe is the recommended approach. The rationale is that it reduces the risk of leaving behind microscopic disease or causing tumour capsule rupture, both of which are associated with significantly worse outcomes [4,7,9].

Outcome and Follow-Up

Final histopathology reported an atypical parathyroid neoplasm (TNM8: pTisN0). Features included fibrous banding and increased cellularity, but there was no unequivocal vascular or capsular invasion to satisfy criteria for carcinoma. It is an uncomfortable diagnosis in some ways the malignant potential is higher than a standard adenoma, yet it does not warrant the same staging workup or adjuvant treatment as frank carcinoma [4,5,10].

Postoperatively, the biochemistry normalised quickly. Calcium came down to the normal range within days, and renal function recovered with eGFR returning to >60 mL/min/1.73m². The patient was discharged with a plan for regular follow-up in the endocrine clinic, including serial calcium, PTH, and periodic neck imaging. Given the atypical classification, this follow-up is indefinite rather than time-limited [4,5,11].

It is worth noting that rapid biochemical normalisation after surgery is generally a good sign in cases where PTH and calcium remain elevated postoperatively, incomplete resection or occult metastatic disease must be considered [5,11].

Discussion

A few things about this case are worth unpacking. The Brown's tumour probably caused the most alarm in the early stages a jaw lesion in a middle-aged woman raises obvious oncological concerns, and the biopsy was necessary both to characterise it and to reassure the multidisciplinary team that this was hyperparathyroidism-related bone disease rather than primary malignancy. Zelano et al. reported a similarly complex case where parathyroid carcinoma was accompanied by brown tumours, osteoporosis, renal stones, pancreatitis, and portal thrombosis a reminder of just how many organ systems PTH can affect when it is elevated for long enough [13].

The adrenal incidentaloma was a separate problem that needed resolving before any surgical intervention. It did not change the definitive management, but it had to be addressed proceeding to general anaesthesia with an uncharacterised adrenal mass is something that should always be avoided if there is any suspicion of phaeochromocytoma. In this case the CT washout and metanephrines gave us the reassurance we needed.

The histopathological result atypical parathyroid neoplasm reflects a genuine diagnostic grey zone. Several groups have written about this, and the problem is not going away: without a definitive molecular marker or a consistent set of histological thresholds, some tumours will always sit ambiguously between benign and malignant [3,10,16].

In terms of surgical decision-making, the literature is reasonably consistent: when carcinoma or atypical neoplasm cannot be excluded preoperatively, the safest approach is en bloc resection. Retrospective data from case series suggest that incomplete initial resection is the single biggest determinant of recurrence and poor outcome [4,6,7].

It is also worth acknowledging the difficulty of preoperative diagnosis in this context. Both ultrasound and sestamibi scanning gave us useful localisation information, but neither can reliably distinguish carcinoma from adenoma. Shi et al. noted that imaging findings in parathyroid carcinoma are often non-specific, and that a high index of clinical suspicion based on biochemistry is usually what drives the diagnosis [1].

For this patient, the plan going forward is long-term endocrine surveillance. Atypical neoplasms have a higher recurrence risk than standard adenomas, and at present there is no reliable way to predict which will and which will not recur. Case series data, including work by Tian et al. on delayed diagnoses following initial surgery, reinforce the importance of not discharging these patients early [5,11-18].

Learning Points

PTH levels significantly above the upper reference limit particularly when >10-fold elevated should prompt active consideration of atypical neoplasm or parathyroid carcinoma rather than assuming a standard adenoma.

Brown's tumours can present in unusual locations including the jaw and may mimic primary bone malignancy. The diagnosis rests on the combination of histology and the underlying biochemical context of hypercalcaemia and elevated PTH.

Incidental adrenal findings in the context of PHPT need systematic characterisation particularly to exclude phaeochromocytoma but should not delay definitive parathyroid surgery once benignity has been established.

When parathyroid carcinoma cannot be excluded preoperatively, en bloc resection incorporating the ipsilateral thyroid lobe is the appropriate surgical strategy and represents the most important

determinant of long-term outcome.

An atypical parathyroid neoplasm diagnosis requires lifelong surveillance. This is not a 'discharge and reassure' result patients need ongoing calcium, PTH, and imaging monitoring given the uncertain malignant potential.

Patient Perspective

The patient provided written informed consent for publication of this case report. She told us that the most difficult part of the journey was the period of diagnostic uncertainty not knowing whether the jaw lesion was cancer, and later not knowing whether the parathyroid itself was malignant. She found the multidisciplinary team approach reassuring and felt that the information she was given at each stage was clear and honest. She accepted the need for ongoing follow-up and understood the reason for it.

Acknowledgements

We would like to thank the multidisciplinary endocrine team at North Tees Hospital for their input into this case, and the pathology team for their detailed histopathological assessment.

Contributors

MA was responsible for drafting the manuscript and reviewing the clinical documentation. NM contributed to data collection and literature review. AS supervised the clinical management and reviewed the final manuscript. All authors approved the submitted version.

Competing Interests

None declared.

Provenance and Peer Review

Not commissioned; externally peer reviewed.

References

1. Shi C, Lu N, Yong YJ, Hai-Di C, Ai-Jun X. (2023). Parathyroid carcinoma: Three case reports. World J Clin Cases. 11: 5934-5943.
2. Saccone S, Nicolas Tarigo N, Soto V, Salom A, Ronco A. (2023). Parathyroid carcinoma: Case report and literature review. J Clin Med Surg. 3: 1072.
3. de Falco N, Santangelo G, Chirico F. (2021). Synchronous intrathyroidal parathyroid carcinoma and thyroid carcinoma: case report and review of the literature. BMC Endocr Disord. 21: 105.
4. Afellah M, Ben Ameur El Youbi A, Chahbouni S. (2025). Parathyroid carcinoma: a clinical case report and literature review. Cureus. 17: e89550.
5. Yin Z, Xu X, Cheng L, Kong W, He Y, et al. (2023). Functional and nonfunctional parathyroid carcinoma: two case reports and literature review. Discov Oncol. 14: 216.
6. Xu T, Zheng X, Wei T. (2024). Incidental synchronous intrathyroidal parathyroid carcinomas and papillary thyroid microcarcinoma: case report and literature review. BMC Endocr Disord. 24: 74.
7. Wu J, Sun Y, Zhang Q. (2024). Giant functional parathyroid carcinoma: a case report and literature review. Front Oncol; 13: 1310290.
8. El Mekkaoui M, Benyahia Z, Elouazzani H. (2024). Left supraclavicular parathyroid carcinoma: diagnostic difficulties through a case report. Pan Afr Med J. 48: 106.
9. Bouzayan L, Mabrouk M, Ait Ali H. (2021). Giant parathyroid carcinoma: diagnostic difficulties and therapeutic strategies. Ann Med Surg. 71: 102919.
10. Tian W, Hu C, Cao T, Chen D, Su X, et al. (2025). Case report: Delayed diagnosis of parathyroid carcinoma and two pulmonary recurrences and metastases. Front Oncol. 15: 1581911.
11. Sen M, Nagaoka R, Kazusaka H. (2021). Non-functioning oxyphilic parathyroid carcinoma: a case report. Surg Case Rep. 7: 68.
12. Ratniece M, Tauvena E, Pavlovičs S. (2022). Large parathyroid tumor 8 years after thyroid surgery: a case report. Case Rep Oncol. 15: 344-352.
13. Zelano L, Locantore P, Rota CA. (2022). Parathyroid carcinoma all-in-one, a rare life-threatening case with multiple systemic manifestations: case report and review of the literature. Front Endocrinol. 13: 881225.
14. Lenschow C, Fuss C, Kircher S. (2021). Case report: abdominal lymph node metastases of parathyroid carcinoma: diagnostic workup, molecular diagnosis, and clinical management. Front Endocrinol. 12: 643328.
15. Sherif M, El-Bolkainy N, Khattar N. (2023). Carcinoma of the parathyroid gland: a case report and review of literature. J Egypt Med Assoc. PP: 145.
16. Yu Z, Zhou J, Xie X, Li X, Marie-Laure MG, et al. (2025). Potential higher malignancy of nonfunctional parathyroid carcinoma: a case series study. Gland Surg. 14: 947-957.
17. Radu P, Garofil D, Tigora A. (2023). Parathyroid cancer a rare finding during parathyroidectomy in high volume surgery centre. Medicina. 59: 448.
18. Bao Y, Kang GJ, Wu X. (2023). Mediastinal parathyroid carcinoma: a case report and review of the literature. BMC Endocr Disord. 23: 90.