

# Hungry bone-like syndrome in a patient with bone-metastatic prostate cancer following radiotherapy: a case report

## Abstract

Hungry bone syndrome is a rare complication that usually occurs after parathyroidectomy. In the literature, only a few cases have been reported in prostate cancer patients almost all with osteoblastic bone metastases. We present the case of a 63-year-old patient with osteolytic bone metastases from prostate cancer, who developed a hungry bone syndrome after starting radiotherapy and concurrent hormone ablation with bicalutamide. The patient developed severe electrolyte disturbances, including hypocalcaemia, hypophosphatemia, hypokalaemia, and hypomagnesemia, along with elevated parathyroid hormone (PTH) levels. In this case, we assume that therapy in the presence of osteolytic bone metastases triggered the hungry bone syndrome, most likely through osteoblast activation leading to redistribution of minerals into bone tissue.

Volume 8 Issue 4 - 2025

Amir Medhioub,<sup>1</sup> Georg Bollig<sup>2,3</sup>

<sup>1</sup>Department of Internal Medicine, Helios Clinic Schleswig, Germany

<sup>2</sup>Department of Anesthesiology, Intensive Care Medicine, Palliative Medicine and Pain Therapy, Helios Clinic Schleswig, Germany

<sup>3</sup>University of Cologne, Faculty of Medicine and University Hospital, Center for Palliative Medicine, Germany

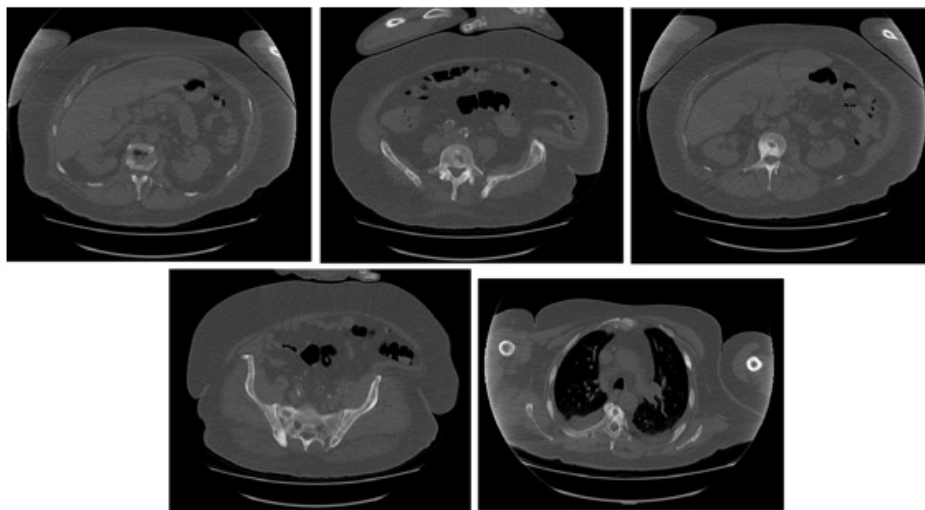
**Correspondence:** Georg Bollig, Abteilung für Anästhesie, Intensivmedizin, Palliativmedizin und Schmerztherapie, Helios-Klinikum Schleswig, St. Jürgener Str. 1-3; 24837 Schleswig, Germany, Email [georg.bollig@helios-gesundheit.de](mailto:georg.bollig@helios-gesundheit.de)

**Received:** December 29, 2025 | **Published:** December 31, 2025

## Case presentation

A 63-year-old male patient was referred to our clinic with newly developed acute renal failure and anaemia. On admission, he was in a reduced general condition, with progressive asthenia over several months and a weight loss of 30 kg within the last 7 months. At the time of diagnosis, the patient weighed 109 kg and had a height of 180 cm. The patient also reported long-standing back pain. His general practitioner had already ordered a cervical spine CT, which raised strong suspicion of a hematologic-oncologic process. Gastroscopy showed kissing ulcers in the duodenal bulb (Forrest Ib anterior wall,

Forrest IIb posterior wall), which were considered the cause of the anaemia. Proton pump inhibitors were started, and the anaemia was corrected with blood transfusions. Further CT scans and laboratory tests excluded classic plasmacytoma but raised suspicion of prostate cancer. The CT-scan revealed multiple bone metastases (Figure 1), with severe spinal canal stenosis at L4/5. Specific tumour markers (PSA) were measured, and because of the elevated values, a spinal MRI was performed. Altogether, the findings confirmed metastatic prostate cancer. Laboratory results showed an ionized calcium of 1.51 mmol/L and normal potassium levels. Total PSA was above 5000 µg/L.



**Figure 1** CT scan performed by the Radiology Department at Helios Clinic Schleswig demonstrating osteolytic bone metastases secondary to prostate cancer.

The patient case was discussed in the regional tumour board which recommended a palliative treatment approach: stabilizing and pain-relieving radiotherapy to the most affected areas, alongside an initiation of hormone ablation using bicalutamide. On day 10, a subcutaneous depot injection for androgen deprivation was planned. In the meantime, the patient had another hospital admission in a regional hospital due to constipation and bowel obstruction in another hospital. After that he was discharged to his own home and referred to the mobile palliative care team. About two months after his first treatment in our hospital, the patient was re-admitted through his specialized palliative care physician. The palliative care physician sent the patient to the hospital only a few days after starting palliative treatment in the patient's home because of general condition degradation, with new manifestation of hallucinations, and panic attacks. Laboratory tests on readmission to our hospital showed severe electrolyte disturbances: hypocalcaemia (ionized 0.49 mmol/L; total 0.95 mmol/L), hypophosphatemia (0.45 mmol/L), hypokalaemia (1.7 mmol/L), and hypomagnesemia (0.36 mmol/L), along with elevated PTH (395 ng/L). Vitamin D3 was normal (31.4 ng/L), and the patient also presented with transfusion-dependent normocytic, normochromic anaemia. He was treated with red blood cell transfusions and targeted electrolyte replacement. Due to his very low life-threatening potassium level, he was admitted to the ICU, where he was placed on electrolyte infusions with continuous monitoring and frequent blood tests. Under this treatment, it was possible to normalize the hypokalaemia over 3 days. We evaluated the patient as stable and found no further need for ICU monitoring. After some days on the ICU the patient was transferred to the palliative unit to complete the treatment of the electrolyte disorder and to

organize his further palliative treatment and to the patient stayed in the palliative unit for the next three weeks, during which he required daily oral and intravenous electrolyte supplementation. Discharging the patient home in his condition was somewhat complicated. The patient required more assistance than we were able to arrange for him outside an institutional setting. Therefore, he was discharged to a short-term nursing home, where he could benefit from more intensive care. Because of continuing pain, dyspnoea and reduced ability to move he was readmitted to the palliative care unit of our hospital. After some days in the unit, he was sent to another hospital to receive radiation for his bone metastases.

During the weeks he spent in the palliative unit, the patient had an abnormally elevated consumption of calcium, magnesium, and potassium. Although he received high doses of electrolyte supplementation with a daily electrolyte supplementation comprising oral potassium chloride 7,200 mg (12 capsules × 600 mg; ≈ 96 mmol potassium), intravenous potassium 40 mmol, calcium 3,750 mg, vitamin D 1,200 IU, and intravenous magnesium 3 g the blood test results consistently remained under the normal level. This continued until, at one point, there was suddenly no further electrolyte consumption, and the test results showed normal levels without any correction over many days. The last blood samples taken before transfer to the hospice showed electrolytes as follows: calcium 1.94 mmol/L (just under the normal minimum value of 2.00 mmol/L), potassium 4.6 mmol/L (within the normal range), hypomagnesemia with 0.62 mmol/L (just under the normal minimum value of 0.70 mmol/L). The patient is at present in the hospice. A summary of the patient clinical course and hospitalisations is shown in Table 1.

**Table 1** Summary of the patient's clinical course

Hospitalisation dates	Clinical course	Length of stay (days)
Pre-clinical	Progressive weakness, severe weight loss, back pain, and anaemia over several months. Outpatient imaging raised suspicion of an oncologic process.	Unknown
Initial hospital care 04.07.25 – 12.07.25	Admission for acute renal failure and anaemia. Gastroscopy revealed duodenal ulcers. Imaging and laboratory diagnostics confirmed metastatic prostate cancer. A palliative treatment approach was initiated.	8 days
ICU 08.09.25 – 10.09.25	Re-admission with acute deterioration, hallucinations, and panic attacks. Severe, life-threatening electrolyte disturbances required intensive monitoring and intravenous replacement therapy.	2 days
Palliative care unit (first stay) 10.09.25 – 18.09.25	Transfer after ICU stabilization. Persistent excessive electrolyte consumption despite high-dose oral and intravenous supplementation. Symptom control and discharge planning.	8 days
Post-clinical / Transitional care 18.09.25 – 17.11.25	Discharge to institutional care with ongoing palliative support between hospital stays. Continued functional decline and symptom burden.	59 days
Palliative care unit (readmission) 17.11.25 – 24.11.25	Re-admission due to pain, dyspnoea, and reduced mobility. Further radiotherapy for bone metastases. Gradual spontaneous normalization of electrolyte levels.	7 days
Radiology and radiation therapy 24.11.2025 – 12.12.2025	Radiation therapy for analgesia of the bone metastases in another hospital	17 days
Hospice 12.12.2025 – 30.12.2025 ongoing	The patient was after radiation therapy admitted to a nearby hospice where he again was treated by the mobile palliative care team	19 days ongoing

## Discussion

From our point of view this case represents a hungry bone-like syndrome following radiotherapy, likely caused by activation of osteoblastic cells within osteolytic bone metastases, atypically seen in prostate cancer. The patient was stabilized with electrolyte substitution in the ICU, and he was transferred to the palliative care unit to complete his treatment once there was no more life-threatening disorder and organize his discharge. The massive electrolyte consumption had disappeared almost two months after the initial radiotherapy. After the final hospital stay, the patient was discharged to a hospice under specialized outpatient palliative care by our mobile palliative care team.

Hungry bone syndrome (HBS) is a rare but serious complication, most often described after parathyroidectomy in patients with primary or secondary hyperparathyroidism. It occurs due to a sudden shift of calcium and phosphate into newly forming bone, which can lead to profound and prolonged hypocalcaemia.<sup>1</sup> In bone metastatic prostate cancer, bone metabolism plays a central role in both disease progression and response to therapy. Treatments such as antihormonal therapy and radiotherapy can further alter bone metabolism. In this report a case of a patient with osteolytic metastatic prostate cancer who developed a hungry bone syndrome after the initial radiotherapy cure and hormone ablation is presented.

In the presented patient case, the syndrome most likely arose from activation of osteoblastic cells within the osteolytic metastases, which are not typical for a prostate Cancer, causing rapid mineral uptake from the bloodstream and subsequent severe electrolyte disturbances. The patient that at the initial diagnosis presented with elevated calcium levels, had developed a profound hypocalcaemia and multiple other electrolyte imbalances only after therapy began. This course differs from most cases described in the literature, which typically occur in patients with osteoblastic metastases at the time of the diagnoses or long before the initial therapy. Only a few reports on similar cases do exist. Garla et al. described HBS in a 50-year-old patient with osteoblastic prostate metastases, where radiotherapy led to long-term stabilization.<sup>2</sup> Berruti et al. reported significant improvement of laboratory parameters with pamidronate therapy over 12 weeks.<sup>3</sup> In a series of 829 oncologic patients with hypocalcaemia, Ferraz Gonçalves et al. found 37 cases of prostate cancer in which HBS was a major cause of this hypocalcaemia, especially in osteoblastic metastases.<sup>4</sup> Navarro et al. described a patient in whom correction of hypocalcaemia without sufficient calcium and magnesium supplementation led to tetanic symptoms due to previously undiagnosed HBS-related hypocalcaemia associated with diffuse prostatic osteoblastic metastases.<sup>5</sup> Sakai et al. reported HBS in a patient with gastric cancer and osteoblastic metastases, who died one month later during palliative care, which, in our case, is unlikely according to our gastroscopy findings.<sup>6</sup> Our case adds to these observations and the scientific knowledge about the topic by showing that hungry bone syndrome can also occur in the context of atypical osteolytic metastases after began of the therapy-a rare reported scenario. It highlights the need for careful monitoring of electrolytes and awareness of this potential complication especially in a non-classical metastatic pattern.

## Ethical aspects and ethical consideration

At admission, the ethical question was raised as to whether intensive care treatment was appropriate in a palliative patient. A

key issue was whether resuscitation should be attempted in the event of acute electrolyte-related cardiac arrest. After interdisciplinary discussion with the intensive care unit team, palliative medical team, gastroenterology, and urology teams, intensive care treatment was approved to address the reversible, life-threatening electrolyte disorder. However, once the patient was stabilized, he was immediately transferred to the palliative care unit to continue treatment in line with the agreed palliative goals. This treatment was in accordance with the patient wish to receive palliative treatment and life-prolonging treatment as far as possible.

The patient has provided informed consent to publish his case including data about his disease, medication and laboratory and radiology images.

## Conclusion

Hungry bone syndrome can also occur in patients with osteolytic prostate cancer metastases during radiotherapy and hormone therapy. Therefore, one should be aware of this rare condition. Close monitoring of calcium, phosphate, magnesium, and potassium is essential in patients with bone metastases receiving treatment and an early electrolyte replacement can prevent life-threatening complications. If wished by the patient and indicated ICU-treatment may be a part of the treatment options to prolong the patients life, to provide best possible palliative care and to relieve suffering.

## Acknowledgments

None.

## Conflicts of interest

The authors declares that there are no conflicts of interest.

## References

1. Witteveen JE, van Thiel S, Romijn JA, et al. Hungry bone syndrome: still a challenge in the postoperative management of primary hyperparathyroidism: a systematic review of the literature. *Eur J Endocrinol.* 2013;168(3):R45–R53.
2. Garla VV, Salim S, Kovvuru KR, et al. Hungry bone syndrome secondary to prostate cancer successfully treated with radium therapy. *BMJ Case Rep.* 2018.
3. Berruti A, Sperone P, Fasolis G, et al. Pamidronate administration improves the secondary hyperparathyroidism due to bone hunger syndrome in a patient with osteoblastic metastases from prostate cancer. *Prostate.* 1997;33(4):252–255.
4. Ferraz Gonçalves JA, Costa T, Rema J, et al. Hypocalcemia in cancer patients: an exploratory study. *Porto Biomed J.* 2019;4(4):e45.
5. Navarro J, Oster JR, Gkonos PJ, et al. Tetany induced on separate occasions by administration of potassium and magnesium in a patient with hungry bone syndrome. *Miner Electrolyte Metab.* 1991;17(5):340–344.
6. Sakai K, Tomoda Y, Saito H, et al. Hungry bone syndrome and osteoblastic bone metastasis from gastric cancer. *QJM.* 2020;113(12):903–904.