ACTA SCIENTIFIC CLINICAL CASE REPORTS

Volume 6 Issue 9 September 2025

Case Report

Unraveling the Intricacies of Acute Respiratory Distress Syndrome in Metastatic Parathyroid Carcinoma After Laser lung Metastatectomy

Soumi Pathak* and Mamta Dubey

Department of Anaesthesia, Rajiv Gandhi Cancer Institute and Research Centre, India

***Corresponding Author:** Soumi Pathak, Department of Anaesthesia, Rajiv Gandhi Cancer Institute and Research Centre, India.

Received: August 18, 2025

Published: August 28, 2025

Abstract

Acute respiratory distress syndrome (ARDS) is a rare complication of hypercalcemia resulting from a parathyroid crisis, usually associated with acute kidney injury (AKI). The lack of familiarity with this condition can lead to uncertainty in management, ultimately resulting in severe and fatal outcomes. We are presenting a case of a 50-year-old female who had developed severe hypercalcemia and acute kidney injury (AKI) due to parathyroid carcinoma. Alarmingly, she deteriorated rapidly in the postoperative period after undergoing parathyroidectomy and lung laser metastectomy. She developed acute respiratory distress syndrome (ARDS) on postoperative day 2 with no evidence of cardiogenic pulmonary oedema or fluid overload. Importantly, all infection screenings sent multiple times returned negative. This case highlights the importance of awareness of the complications associated with parathyroid carcinoma and prompt intervention in managing these interconnected conditions.

Keywords: Acute Respiratory Distress Syndrome; Acute Kidney Injury; Hypercalcemia; Parathyroid Carcinoma; Parathyroidectomy

Introduction

Parathyroid storm is a rare but potentially life-threatening complication of primary hyperparathyroidism (PHPT). Although there is no uniform standard definition, it is typically characterized by a significantly elevated serum calcium (sCa) level, usually greater than 14 mg/dL, accompanied by a rapid decline in central nervous system, cardiovascular, gastrointestinal, and kidney functions. This situation underscores the importance of vigilant postoperative care and comprehensive management strategies for patients with parathyroid metastatic disease. Informed consent was obtained from the patient.

Case Report

A 50-year-old female patient with metastatic parathyroid carcinoma presented to our hospital with complaints of 5 kg weight loss over the past 2 to 3 months, a notable loss of appetite, and pain localized to the right lower limb. A PET-CECT scan revealed a nodular attenuation of 2.5/2.6 cm in the left tracheoesophageal groove, posterior to the left thyroid lobe. Additionally, there were subpleural and parenchymal nodules in both lung fields. An ultrasound of the neck showed a hypoechoic lesion posterior to the left thyroid lobe, infiltrating into the thyroid isthmus.

Her blood nvestigation revealed hypercalcemia (ca 2.15 mmol/l),serum calcitonin 2.96pg/ml, serum parathyroid hormone 750, creatinine of 2.12. All the other investigations were normal. Echocardiography revealed an Ejection fraction of 65. She was started on inj calcitonin 200 IU SC tds, and Inj Zolendronic 4mg infusion. Once her calcium level came down to 1.12, she was posted for en-bloc excision of left parathyroid glands with left hemithyroid lobe and bilateral VATS metastectomy. After premedication with raniti-

dine and granisetron, the patient was shifted to the operating theater. Standard monitors were applied, and a 16-gauge intravenous access was established. The left radial artery was cannulated for invasive blood pressure monitoring. Pre insicion PTH was 1344. Anaesthesia was induced with propofol, fentanyl, and vecuronium. Intubation was performed with a 7.5 size intraoperative neuromonitoring (IONM) endotracheal tube, and anesthesia was maintained with a combination of propofol, dexmedetomidine infusion, and a mixture of air, oxygen, and sevoflurane. A 5x4 cm mass was visualized originating from the left inferior pole of the thyroid, extending into the central compartment. The mass was encasing the left recurrent laryngeal nerve (RLN), and there was infiltration into the muscular fibres of the oesophagus. The V1 nerve was identified with an amplitude of 1124. The parathyroid gland was located using its autofluorescence, as detected by the blue channel of a modified Karl Storz near-infrared/indocyanine green endoscopic system. An intraoperative frozen section was sent to the pathology for margins. After hemithyroidectomy and left parathyroidectomy, comprehensive central compartment clearance was done, and oesophagal repair was done. R2 and V2 confirmed at amplitudes of 980 and 918, respectively.

Subsequently, a left double-lumen tube (DLT) No. 35 was inserted to facilitate bilateral video-assisted thoracoscopic (VATS) metastectomy. A total of ten lung nodules were resected and ablated using an ND YAG laser on the right side, while on the left side, seven nodules were excised and an inferior lingulectomy was performed. Additionally, mediastinal lymph node clearance was accomplished. The total duration of the surgical procedure was 13 hours blood loss was 200ml. The patient was subsequently transferred to the surgical intensive care unit (SICU) for elective ventilation. ABG done in the icu was normal.

The patient was extubated the next day after a trial with a T-piece. However, her oxygen requirement increased to 8 l/min by the evening. On postoperative day 1, the patient exhibited signs of respiratory distress, and an arterial blood gas (ABG) analysis revealed a PaO2 of 77 mmHg and a PaCO2 of 43 mmHg, with an oxygen saturation (SaO2) of 82% on room air. On auscultation, bilateral fine crackles were noted in the lungs. The patient was nebulised and commenced on high-flow oxygen therapy. Laboratory results indicated a D-dimer level of 2627, a procalcitonin level of 7.93, and an NT-proBNP level of 1360. Blood and sputum cultures were obtained for analysis, and naso and oropharyngeal swabs revealed no viral pathogens in the respiratory viral panel evaluation. A chest X-ray demonstrated bilateral widespread pulmonary infiltrates. Fig1 On the recommendation of infectious disease and microbiology specialists and respiratory physician, the patient was initiated on a course of broad-spectrum antibiotics for an atypical chest infection. However, blood, urine and sputum cultures collected exhibited no growth of organisms. Moreover, screening for atypical infections, and tuberculosis were negative. Serial levels of parathyroid hormone (PTH), serum calcium, phosphorus, and creatinine were obtained all showing declining trend except for serum creatinine which was 2.2. FIG2,3 On post-operative day (POD) 2, the patient was drowsy, and her arterial blood gas (ABG) analysis indicated hypoxia and hypercapnia. A central venous catheter was placed. Cardiogenic pulmonary oedema seemed unlikely, as an echocardiogram revealed an ejection fraction of 50% with no signs of decompensated heart failure. The patient was put on non-invasive ventilation (NIV).

On POD 3, she was started on injection methylprednisolone, injection furosemide, and nebulization every six hours with Foracort and Duolin. Her alertness, oxygen saturation, and chest X-ray findings improved. Additionally, her kidney function also improved, and she was gradually weaned off NIV and transferred to the ward on POD 15.

Discussion

Parathyroid carcinoma (PC) is an extremely rare form of cancer, accounting for less than 0.005% of all cancers worldwide. Its incidence is estimated to be between 3.5 and 5.7 cases per 10 million [1], people annually, representing approximately 1% of primary hyperparathyroidism (PHPT) cases. The peak incidence occuring in individuals during their fifth decade of life. Despite its low prevalence, PC significantly affects patient morbidity due to often severe hypercalcemia and the risk of local and distant metastases. The tendency for local and distant spread complicates surgical management and often requires multimodal treatment approaches. Our patient had an aggressive parathyroid carcinoma with thyroid and esophageal infiltration and lung metastasis.

After an En bloc resection of the left parathyroid with left lobe of the thyroid, she underwent bilateral lung metastectomy using laser which was crucial for achieving long-term disease control. Intraoperative fluorescence-guided imaging with indocyanine green (ICGT) was used to enhance precision in detecting parathyroid tissue. ICGT is particularly valuable in challenging cases where tumor is infiltrating the surrounding tissue and the extent of infiltration is unclear. It also helps in differentiating malignant from benign tissues [2].

Unfortunately, she developed Acute Respiratory Distress Syndrome (ARDS) on postoperative day 2, accompanied by a mild impairment of kidney function, as evidenced by a serum creatinine level of 2.2. All her cultures and viral panel results were negative,

and despite high-grade antibiotics, diuretics, and high-flow oxygen therapy, her condition did not improve. After ruling out infective, cardiac, and vasculitis, a diagnosis of parathyroid storm was made. A parathyroid crisis [3] has the potential to cause rapid lung calcification, which can compromise the integrity of the alveolar-capillary barrier, triggering a pro-inflammatory cytokine response and ultimately contributing to the development of ARDS.

Acute Respiratory Distress Syndrome (ARDS) is a rare complication of hypercalcemic crises, particularly in the context of Primary Hyperparathyroidism (PHPT) [5]. Holmes., *et al*. [6] were the first to report a case of ARDS in a patient undergoing a parathyroid crisis. The exact pathophysiological mechanisms leading to acute lung injury during hypercalcemic crises are not fully understood. The lungs are the preferential site for calcium deposition, largely due to their relative alkalinity, which results from lower local partial pressures of carbon dioxide or secretion of free hydrogen ions [7]. Calcium deposition initiates in the basement membrane of alveolar cells and can extend to involve entire alveoli and their associated capillaries. Autopsy findings in the past reported cases revealed extensive calcium deposition in alveolar cells, suggesting that this deposition compromise the alveolar-capillary barrier and contributed to lung injury. An experimental study by Chen., *et al.* [8] in conscious rats and isolated perfused rat lungs indicated that hypercalcemia can induce a sepsis-like syndrome and acute lung injury through elevated plasma nitrate/nitrite levels, increased free radicals, heightened pro-inflammatory cytokines, elevated procalcitonin, and increased activity of inducible Nitric Oxide Synthase (iNOS).

Metastatic calcification usually develops due to a prolonged period of hypercalcemia, but calcification can also occur acutely, as in our patient [9]. Most patients with metastatic pulmonary calcification suddenly become worse due to decreases lung compliance and diffusing capacity. MPC has also been reported in patients with normal calcium and phosphate levels, normal parathyroid hormone, and normal renal function [10,11].

Rehydration, calciuresis, and bisphosphonate therapy are effective for correcting the life-threatening manifestations of hyperparathyroid crises and preparing the patient for subsequent surgery. Once acute respiratory distress syndrome (ARDS) or acute lung injury (ALI) has manifested, only supportive treatment is recommended. This includes positive pressure ventilation, prone position ventilation, pulse steroid therapy, and maintaining calcium levels and the calcium-phosphate product within normal ranges.

In case of persistent hypercalcemia, aggressive management involves the use of 0.9% saline, an osteoclast inhibitor, such as calcitonin or a bisphosphonate. Gallium nitrate, another potent osteoclast inhibitor, may be used if bisphosphonate therapy is unsuccessful. Additionally, the somatostatin analogue octreotide has been shown to be effective in treating hypercalcemia of malignancy caused by the secretion of PTH-related protein [12-14].

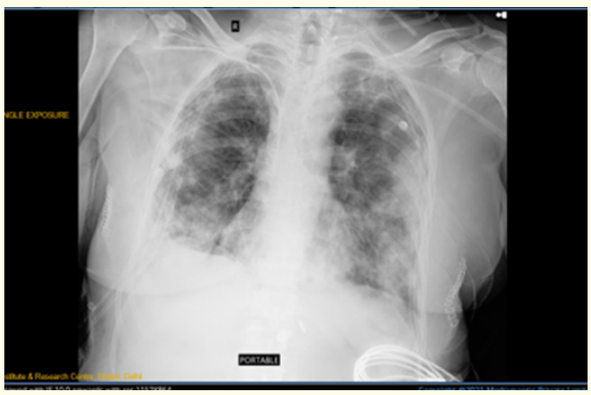

Figure 1: POD 2 chest xray showing ARDS picture

Figure 2: Trends of serum Calcium.

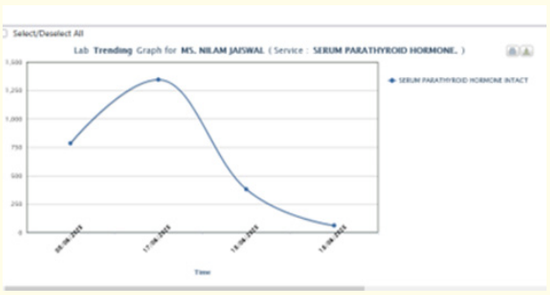

Figure 3: Trends of PTH .

Bibliography

1. Ullah A., *et al*. "Parathyroid Carcinoma: Incidence, Survival Analysis, and Management: A Study from the SEER Database and Insights into Future Therapeutic Perspectives". *Cancers (Basel)* 14.6 (2022): 1426.

2. Spartalis E., *et al*. "Intraoperative Indocyanine Green (ICG) Angiography for the Identification of the Parathyroid Glands: Current Evidence and Future Perspectives". *In Vivo* 34.1 (2020): 23-32.

3. Chow S., *et al*. "Parathyroid storm: rare manifestation of primary hyperparathyroidism". *CMAJ* 134 (1986): 503-504.

4. Ahmad S., *et al*. "Hypercalcemic crisis: a clinical review". *The American Journal of Medicine* 128 (2015): 239-245.

5. Hsu YH and Chen HI Acute respiratory distress syndrome associated with hypercalcemia without parathyroid disorders". *Chinese Journal of Physiology* 51 (2008): 414-418.

6. Holmes F., *et al*. "Pulmonary oedema in hypercalcaemic crisis". *Lancet* 303 (1974): 311-312.

7. Mert M., *et al*. "A hyperparathyroid case with pulmonary edema: can hypercalcemia trigger pulmonary edema?" *American Journal of Emergency Medicine* 30 (2012): 512.

8. Chen HI., *et al*. "The detrimental role of inducible nitric oxide synthase in the pulmonary edema caused by hypercalcemia in conscious rats and isolated lungs". *Journal of Biomedical Science* 15 (2008): 227-238.

9. Ueno K., *et al*. "Fulminant respiratory failure due to progressive metastatic pulmonary calcification with no predisposing factors after successful renal transplantation: A case report". *Pediatric Transplant* 20.8 (2016): 1152-1156.

10. Liang Z., *et al*. "Metastatic pulmonary calcification misdiagnosed as a fungal infection: A case report". *Molecular and Clinical Oncology* 4.3 (2016): 409-412.

11. Sun HM., *et al*. "Rapid development of metastatic pulmonary calcifications in primary hyperparathyroidism: a case report and literature review". *Diagnostic Pathology* 12.1 (2017): 38.

12. Phitayakorn R and McHenry CR. "Hyperparathyroid crisis: Use of bisphosphonates as a bridge to parathyroidectomy". *Journal of the American College of Surgeons* 206 (2008): 1106-1115.

13. M Nakamura., *et al*. "Adult T-cell leukemia with hypercalcemia-induced metastatic calcification in the lungs due to production of parathyroid hormone-related protein". *Internal Medicine Tokyo Japan* 40 (2001): 409-413.

14. SA Peter and JF Cervantes. "Hypercalcemia associated with adult T-cell leukemia/lymphoma (ATL)". *Journal of the National Medical Association* 87 (1995): 746-748.