

A Rare Case of Mediastinal NUT (Nuclear Protein in Testis) Carcinoma Presenting with Acute Respiratory Failure: A Case Report

Hafiz Javed¹, Arfa Ahmad¹, Abdul Rehman¹, Michael W Mikula²

¹Department of Medicine, TidalHealth Peninsula Regional, Salisbury, MD, USA; ²Department of Pathology, John Hopkins Hospital, Baltimore, MD, USA

Correspondence: Abdul Rehman, Department of Medicine, TidalHealth Peninsula Regional, 100 E Carroll St, Salisbury, MD, 21801, USA, Tel +1 410 543 7536, Fax +1 410 543 7272, Email abdul.rehman_aku@yahoo.com



Background: NUT (nuclear protein in testis) carcinoma is a rare but aggressive cancer, which was first described in 1991. Given the rarity of this enigmatic entity, risk factors are unknown, treatment options are limited, and the prognosis is dismal. We present the case of a 30-year-old male who presented with acute respiratory failure secondary to a rapidly enlarging mediastinal mass and was subsequently diagnosed with NUT carcinoma.

Case Report: A 30-year-old male presented with symptoms of rapidly progressive shortness of breath and chest pain. CT chest revealed mediastinal lymphadenopathy with extrinsic compression of the left lower lobe bronchus leading to lobar atelectasis. Bronchoscopy with endobronchial ultrasonography was performed to obtain a fine-needle aspirate of the subcarinal lymph node, which was consistent with poorly differentiated carcinoma. An ultrasound-guided percutaneous right supraclavicular core-needle biopsy confirmed NUT carcinoma. Despite prompt initiation of mechanical ventilation as well as chemotherapy, the patient's condition continued to deteriorate and he passed away after being hospitalized for 127 days.

Conclusion: Mediastinal NUT carcinoma is a rare cause of acute respiratory failure. Despite the rarity of this cancer, its aggressive and serious nature necessitates all physicians to be aware of this clinical entity and maintain a high index of suspicion. Prompt diagnosis and initiation of chemotherapy is essential. The prognosis remains dismal given that the cancer is resistant to known antineoplastic therapies. Further research should focus on new treatment paradigms for this rare cancer and patients should be encouraged to participate in clinical trials.

Keywords: NUT midline carcinoma, nuclear protein in testis, BRD4-NUT fusion, t(15;19) translocation, bromodomain-containing protein 4, respiratory distress

Introduction

NUT (nuclear protein in testis) carcinoma is a rare but aggressive malignancy driven by the formation of NUT fusion oncoproteins.¹ This malignancy was first described in two separate case reports in 1991^{2,3} and was initially thought to be a subset of thymic carcinoma and only affect structures in the midline. However, as our understanding of this malignancy has evolved, this cancer of epithelial origin (carcinoma) is now believed to represent a distinct subtype of squamous cell carcinoma of head and neck or lung origin—although this is not universally accepted.^{4,5} The report of the First International Symposium on NUT carcinoma suggests that NUT carcinoma is substantially underdiagnosed and its true incidence may surpass that of Ewing sarcoma.^{6,7} Little is known about the risk factors for this malignancy and median overall survival is only 6 months.

The primary molecular pathogenic event in NUT carcinoma is a chromosomal translocation resulting in fusion of *NUTM1* (NUT carcinoma family member 1) gene with a gene encoding a BET (bromodomain and extraterminal domain)-associated protein, such as *BRD4*, *BRD2*, *BRD3*, *NSD3*, *ZNF592*, or *ZNF532*.^{8–12} The fusion oncoproteins resulting from this chromosomal translocation, such as the BRD4-NUT fusion oncoprotein, form chromatin complexes

that play key roles in upregulating expression of proto-oncogene transcription factors like *SOX2*, *TP63*, and *MYC*.^{10–14} The most common fusion oncoprotein is BRD4-NUT, resulting from the chromosomal translocation t(15;19).^{10–14} Diagnosis of NUT carcinoma relies on the detection of these fusion oncoproteins, either by immunohistochemistry (staining for NUT fusion oncoprotein), cytogenetics (fluorescent in situ hybridization) or genetic sequencing (such as reverse transcriptase-polymerase chain reaction).^{12–15}

Histologically, NUT carcinoma demonstrates sheets of poorly differentiated epithelial cells with variable squamous differentiation, and immunohistochemical staining for NUT protein is diagnostic.^{4–6,11} The increasing availability of NUT-specific immunohistochemistry and next-generation sequencing has improved diagnostic accuracy; however, optimal therapeutic strategies remain undefined.¹⁵ Conventional cytotoxic chemotherapy often results in transient responses, while novel targeted therapies, including bromodomain and extra-terminal (BET) inhibitors, have shown promise but remain largely investigational.^{16–19}

Based on published case series, primary pulmonary, sinonasal and salivary gland NUT carcinoma are the most common sites of origin.^{12–15} Sinonasal carcinoma frequently presents with symptoms of epistaxis, headache, or nasal congestion. Salivary gland NUT carcinoma is often diagnosed as a rapidly enlarging parotid or submandibular gland mass. Primary pulmonary carcinoma usually results in cough, hemoptysis, shortness of breath, or chest pain. The male-to-female ratio is nearly equal.^{14,15} NUT carcinoma of pulmonary origin frequently presents as a mediastinal mass with respiratory symptoms related to airway compression or post-obstructive pneumonia.^{7,14} Radiographically, these tumors may mimic lymphoma or germ cell malignancies, leading to diagnostic delays that can be fatal given the fulminant disease course.¹³

Initial presentation of pulmonary NUT carcinoma with acute respiratory failure is exceedingly rare. Here, we report the case of a 30-year-old male who presented with acute respiratory failure and was found to have mediastinal NUT carcinoma. Despite aggressive treatment with chemotherapy as well as mechanical ventilation, the patient's clinical condition rapidly declined and his cancer continued to progress. Endobronchial stent placement was performed to alleviate airway obstruction, which was complicated by early malignant occlusion of the stent leading to refractory hypoxic respiratory failure and eventual death. Written informed consent was obtained from the patient's wife for the publication of this tragic case report.

Case Report

Initial Presentation

A 30-year-old male with a past medical history of childhood pertussis and essential hypertension presented to the emergency department of our institution with complaints of shortness of breath. He had been having some cough with sputum production for almost a month. He was initially diagnosed with a viral upper respiratory tract infection. Subsequently, as his symptoms did not improve, his primary care physician prescribed a course of azithromycin. He completed this course without any improvement in symptoms. He was evaluated again and underwent a chest X-ray, which was reportedly normal. Subsequently, he was evaluated in an urgent care center and prescribed oral amoxicillin-clavulanate. He took this medication without any improvement in symptoms and got evaluated by another provider, who prescribed a course of oral levofloxacin. However, by this time, the patient began to develop symptoms of shortness of breath, which prompted him to seek emergency medical care. The patient denied any known allergies and he had no prior history of any surgical procedures. His regular medications included lisinopril 20 mg by mouth daily, amlodipine 5 mg by mouth daily, and hydrochlorothiazide 12.5 mg by mouth daily. His family history was remarkable for bladder cancer in a maternal grandmother. He had a history of cigarette smoking in the past with an exposure of 8 pack-years and vaped daily. He denied any history of illicit drug use or alcohol use. He was married but had no children.

On initial evaluation, he was noted to be tachycardiac (pulse rate 125 beats per minute) and tachypneic (respiratory rate 24 breaths per minute). His physical examination was notable tachypnea, increased work of breathing and decreased air entry at left lung base. No cervical, axillary, or inguinal lymphadenopathy was appreciated. No hepatosplenomegaly was appreciated. No testicular masses could be appreciated. His chest X-ray showed left lower lobe collapse with obliteration of the left hemidiaphragm (Figure 1). His laboratory investigations were notable for neutrophilic leukocytosis

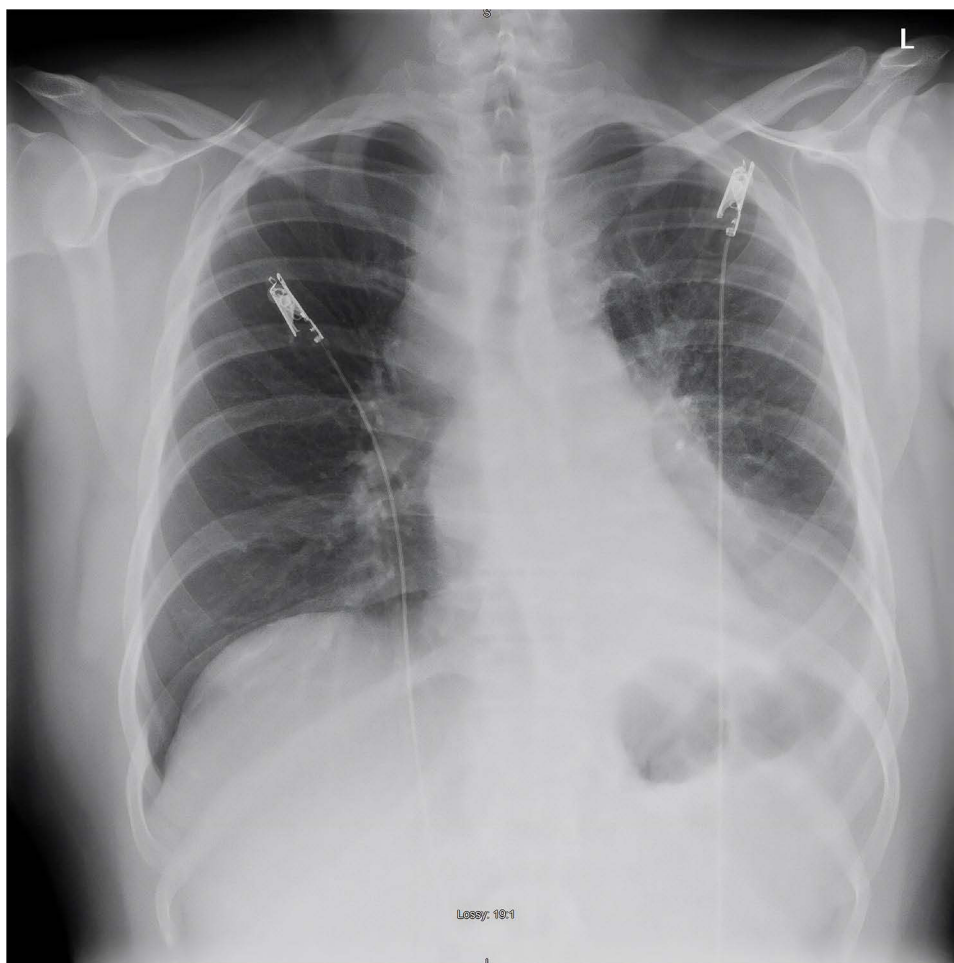


Figure 1 Chest X-ray on initial presentation. Anteroposterior plain chest radiograph demonstrating volume loss of the left hemithorax along with left lower lung zone haziness and obliteration of the left hemidiaphragm. These radiographic findings are suggestive of left lower lobe collapse.

(total leukocyte count 15,900 cells/mm³ with 89% neutrophils), normochromic normocytic anemia (hemoglobin 11.2 g/dl with mean corpuscular volume 92 fl) and hyponatremia (serum sodium 128 mmol/l). Blood cultures were sent and the patient was empirically started on broad-spectrum antibiotic therapy for possible pneumonia. A computed tomography (CT) scan of the chest without contrast was obtained (Figure 2), which showed a mediastinal mass with extrinsic compression of the left lower lobe bronchus leading to left lower lobe collapse. Paratracheal, hilar, and subcarinal lymphadenopathy were also noted. The patient was admitted to the hospital for further diagnostic work-up and management. At this time, he was saturating 96% on 2 L/min oxygen by nasal cannula.

Diagnostic Work-Up and Tissue Diagnosis

After admission to the hospital, pulmonary and thoracic surgery teams were consulted. Differential diagnosis at this time included mediastinal germ cell tumor, lymphoma, and other malignancies. Tumor markers were checked, which were notable for a markedly elevated lactate dehydrogenase (1441 IU/L) along with normal levels of alpha-fetoprotein and beta-human chorionic gonadotropin. Peripheral blood lymphocyte flow cytometry did not reveal any monoclonal populations of T- or B-cells. CT abdomen and pelvis scans with contrast were performed, which showed bulky retroperitoneal lymphadenopathy (Figure 3). Ultrasonography of the scrotum was performed, which did not reveal any testicular masses. The patient then underwent bronchoscopy with endobronchial ultrasound (EBUS), which revealed large contiguous masses of lymph nodes involving stations 7, 4L, and 4R. Multiple fine needle aspirate (FNA) samples were obtained from the lymph nodes for cytology as well as flow cytometry. Bronchoscopy with a regular scope was also

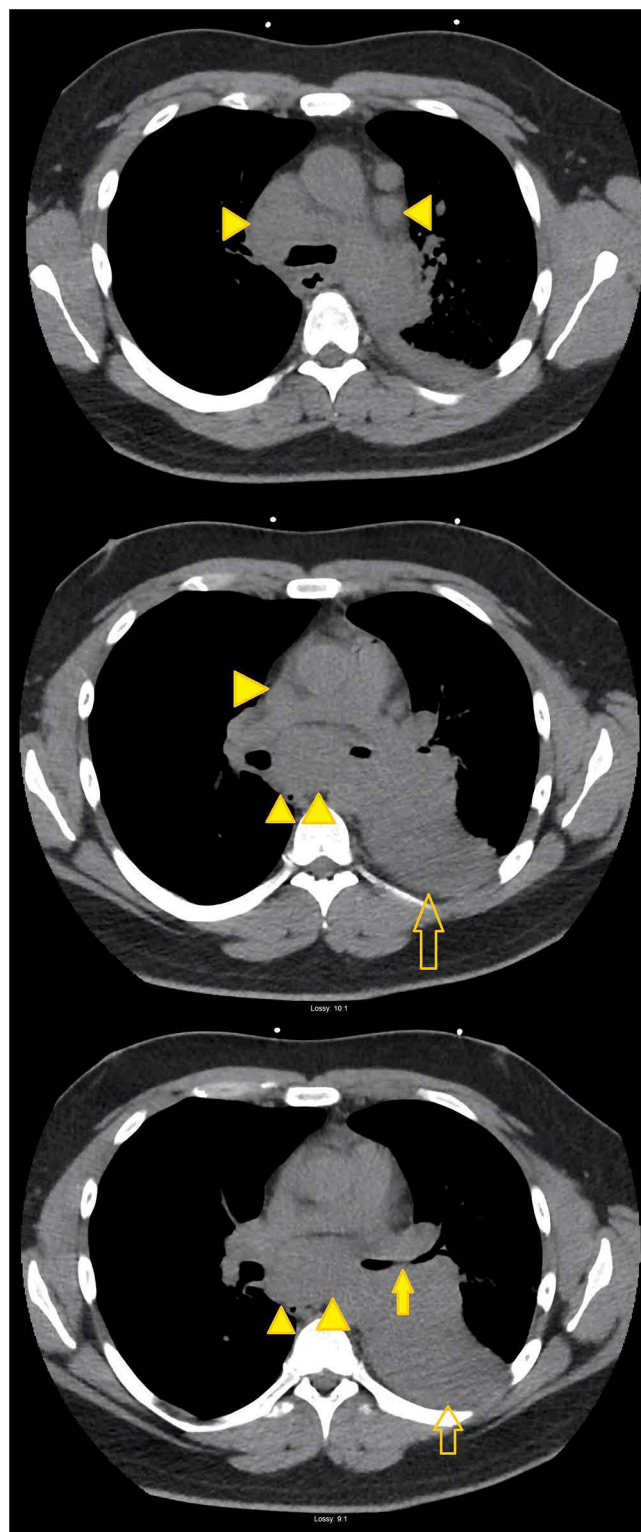


Figure 2 Computed tomography of the chest without contrast on initial presentation. Axial slices of computed tomography of the chest demonstrating bulky mediastinal lymphadenopathy (arrowheads) along with narrowing of the left mainstem bronchus (solid arrow) and left lower lobe collapse (hollow arrow).

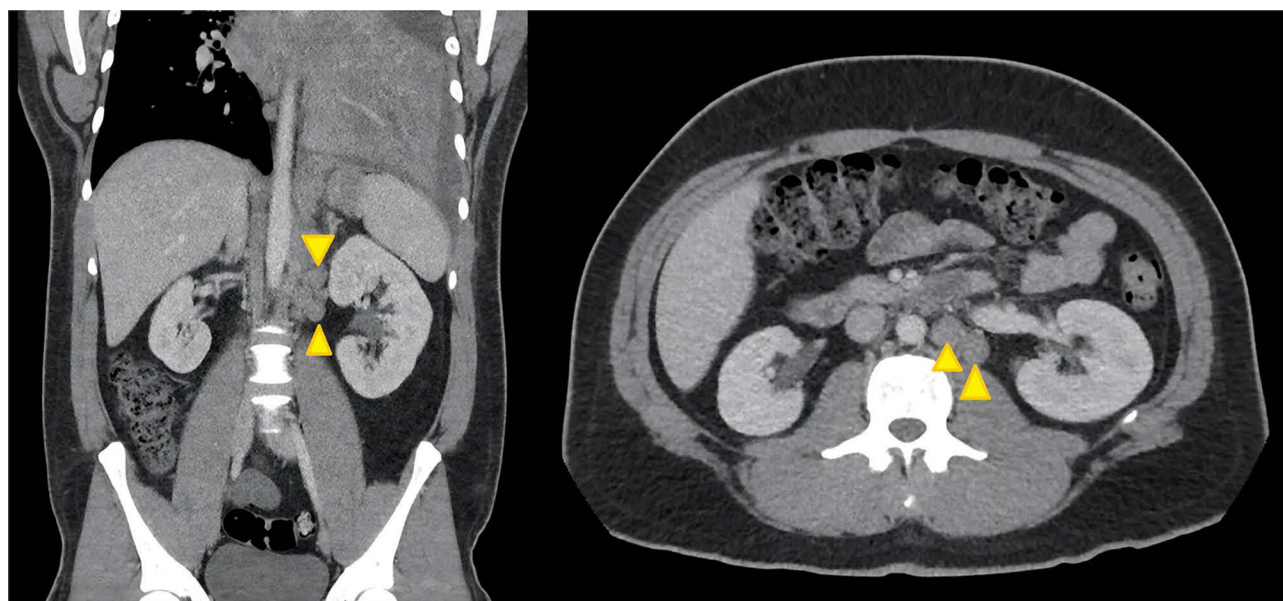


Figure 3 Contrast-enhanced computed tomography of the abdomen. Axial and coronal slices of computed tomography of the abdomen demonstrating bulky retroperitoneal lymphadenopathy (arrowheads).

performed to examine the left lower lobe bronchus, which showed significant narrowing involving all of the small airways. At this time, endobronchial stent insertion was not pursued.

Post-procedure, the patient was noted to become more hypoxic and have hoarseness of voice. A repeat CT chest with contrast was performed, which showed worsening atelectasis throughout the left lung secondary to extrinsic compression of left-sided bronchi from extensive mediastinal and hilar lymphadenopathy. The patient subsequently began to complain of blurring of vision and a neurology team was consulted. A brain MRI without and with contrast was performed, which was negative for any intracranial pathology. Lumbar puncture was also performed, which revealed normal cerebrospinal fluid (CSF) glucose and protein. No CSF pleocytosis was appreciated. At this time, the patient's lymph node FNA cytology results came back positive for poorly differentiated carcinoma. Flow cytometry from the FNA sample as well as cerebrospinal fluid did not reveal any monoclonal lymphocyte populations. The patient's respiratory status continued to decline during this time and he required endotracheal intubation. After a multidisciplinary discussion between the pulmonary, oncology, and thoracic surgery teams, the patient was recommended to be transferred to a higher center of excellence for further management.

After being transferred to the oncologic center of excellence, the patient underwent rigid bronchoscopy, which showed complete collapse of the left mainstem bronchus along with extrinsic compression of the mid-trachea. A 12 mm × 30 mm endobronchial stent was deployed in the left mainstem bronchus. In consultation with oncology and interventional radiology, a percutaneous ultrasound-guided core needle biopsy was performed from an enlarged right supraclavicular lymph node ([Supplementary Figure 1](#)). The patient's respiratory status remained tenuous and he continued to require mechanical ventilation. A repeat CT scan of the chest showed debris filling the left endobronchial stent with resultant collapse of the entire left lung and a large left-sided pleural effusion. A left-sided 14-Fr chest tube was placed and 1.35 L of serosanguineous fluid was drained. Despite this, a repeat chest radiograph showed complete opacification of the left hemithorax secondary to a combination of atelectasis and pleural effusion. A repeat rigid bronchoscopy was performed, which showed tumor tissue completely occluding the distal end of the endobronchial stent with mostly patent proximal portions ([Supplementary Figure 2](#)). The prior endobronchial stent was grasped, revised slightly proximally and a new 10 mm × 30 mm endobronchial stent was inserted in the left upper lobe bronchus to intentionally jail the left lower lobe bronchus (which was occluded by tumor tissue) but maintain the patency of the left upper lobe and lingula. During this procedure, the tracheal mucosa was noted

to be edematous and studded in appearance. By this time, the patient's supraclavicular lymph node biopsy results came back, which revealed tumor cells diffusely positive for AE1/3, NUT1, p40, and INSM1, focally positive for synaptophysin, and negative for OCT-3/4, TTF1, Napsin-A, c-Kit, and CD5 (see Figure 4). These findings were consistent with NUT carcinoma.

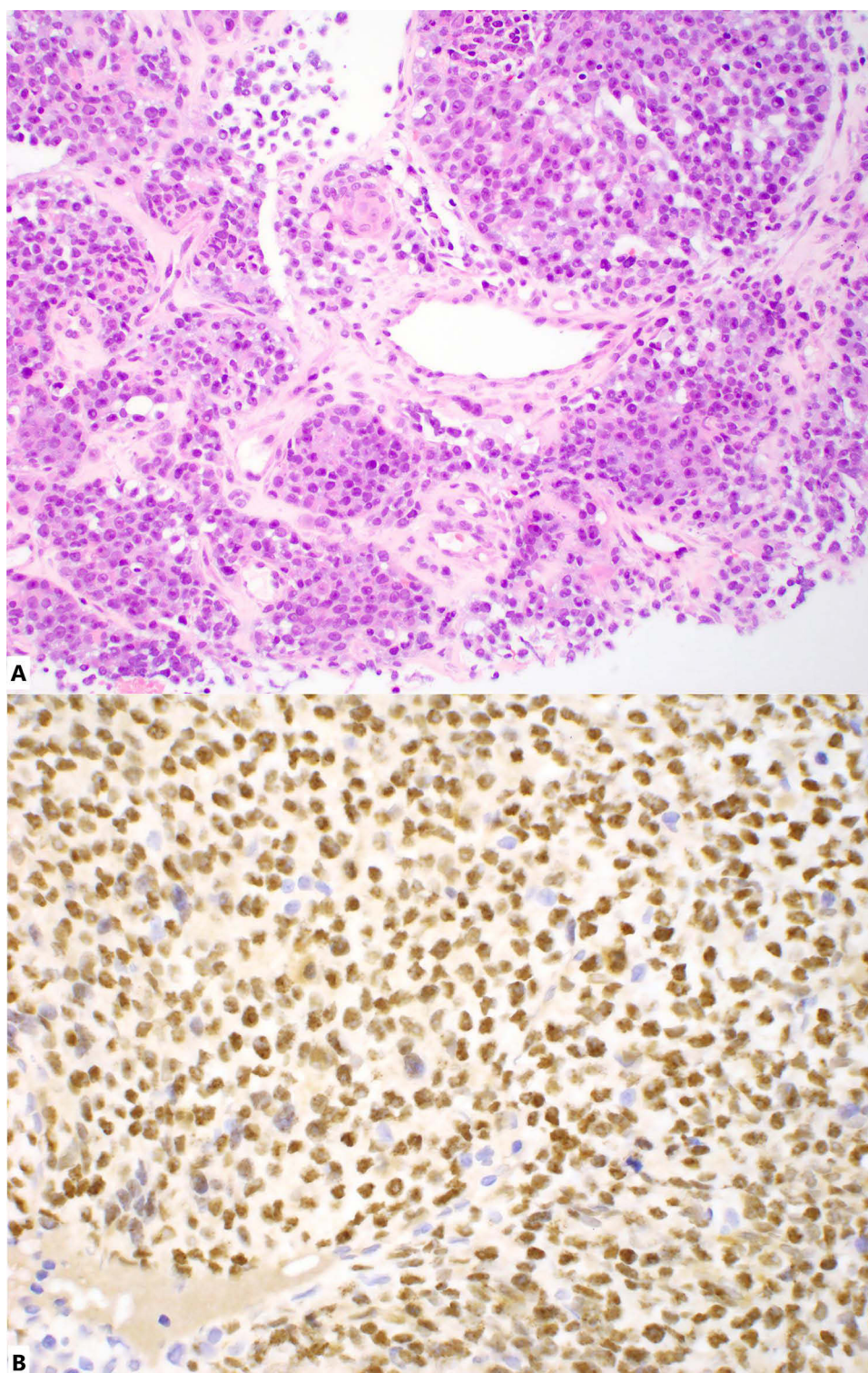


Figure 4 Photomicrographs of core needle biopsy of right supraclavicular lymph node. **(A)** Hematoxylin and eosin (H&E) stain demonstrating poorly differentiated cancer cells. **(B)** Immunohistochemical staining for NUT protein showing diffuse positivity in tumor cells.

Chemotherapy and Subsequent Outcome

After a multidisciplinary discussion of pulmonary, thoracic surgery, and oncology teams with the patient's family, open tracheostomy and percutaneous endoscopic gastrostomy were performed. Ultrasound-guided testicular extraction for sperm cryopreservation was also performed. Chemotherapy with carboplatin and paclitaxel was initiated. Patient's subsequent hospital course was complicated by recurrent fevers attributed to RSV (respiratory syncytial virus) infection, hospital-acquired pneumonia, and drug-induced fever. The patient developed significant bone marrow toxicities after receiving three cycles of carboplatin and paclitaxel, which precluded any further administration of chemotherapy. He also had persistently poor oxygenation and high ventilatory requirements despite undergoing multiple bronchoscopies with bronchoalveolar lavage and dilatation of left-sided bronchi. Tumor was deemed refractory to all treatments and progressive recurrent airway occlusion ([Supplementary Figure 3](#)) led to persistent, refractory respiratory failure. After a detailed discussion with the patient's family, goals of care were transitioned to comfort care and palliative extubation was performed. The patient died after a prolonged hospitalization spanning over 127 days.

Discussion

NUT carcinoma, previously known as midline NUT carcinoma, is a rare but aggressive malignancy characterized by the presence of NUT fusion oncoproteins.¹ The present case documents the case of an unfortunate 30-year-old male who developed rapidly progressive respiratory failure secondary to mediastinal NUT carcinoma. Despite receiving three cycles of carboplatin and paclitaxel while being mechanically ventilated, the patient had no improvement in his clinical condition and died within 6 months of his initial presentation. The dismal outcome in this case was driven by the cancer's rapid progression and refractoriness to chemotherapeutic agents, which led to recurrent endobronchial stent occlusion and refractory respiratory failure.

Lessons Learnt from This Case

This case illustrates several defining and clinically important features of pulmonary NUT carcinoma: aggressive local invasion, progressive airway compromise, diagnostic ambiguity, and limited responsiveness to systemic chemotherapy. The patient's initial presentation with cough and progressive dyspnea – initially treated as pneumonia – reflects the nonspecific symptomatology commonly reported in pulmonary NUT carcinoma and contributes to frequent diagnostic delay.^{6,9} Additionally, the differential diagnosis of a large mediastinal mass in a young adult typically includes lymphoma, germ cell tumors, and thymic malignancies. In this case, normal alpha-fetoprotein and beta-human chorionic gonadotropin levels, combined with markedly elevated lactate dehydrogenase, further obscured the diagnosis. Fine needle aspiration cytology revealed a poorly differentiated carcinoma, underscoring the limitation of small biopsy samples in diagnosing NUT carcinoma without specific immunohistochemical staining.^{12–15} Definitive diagnosis was ultimately established through core biopsy with diffuse nuclear positivity for NUT protein, emphasizing the importance of maintaining clinical suspicion and pursuing adequate tissue sampling.

Airway compromise is a particularly devastating feature of pulmonary NUT carcinoma.^{14,20} In this patient, extrinsic compression progressed rapidly to complete left mainstem bronchial obstruction despite early intervention. Recurrent endobronchial stent occlusion by tumor tissue highlights the limitations of mechanical airway stabilization in the setting of unchecked tumor proliferation. Similar cases in the literature describe repeated bronchoscopic interventions with diminishing returns, often serving as temporizing rather than definitive measures.^{20,21} The decision to intentionally jail the left lower lobe bronchus in order to preserve left upper lobe ventilation reflects the complex risk–benefit calculations required in such cases. While interventional pulmonology can be lifesaving, its effectiveness is ultimately constrained by tumor biology.

Synthesis of the Available Literature

Since its first description in 1991, literature regarding patients with NUT carcinoma has been limited to a handful of case series and some case reports. The NUT Carcinoma Registry was established in 2010 and has been documenting more than 50 cases of NUT carcinoma annually.⁷ The report of the First International Symposium on NUT carcinoma

emphasizes the fact that NUT carcinoma is substantially underdiagnosed and its true incidence may surpass that of Ewing sarcoma.⁶ It is believed that cases of NUT carcinoma are sometimes misdiagnosed and this observation is supported by the fact that more than half of all cases of NUT carcinoma were diagnosed after the year 2010.^{8,9} Based on crude estimates, the true incidence of NUT carcinoma is believed to be 1,400 cases per year.⁵ The diagnostic delays and challenges seen in our case can attest to the fact that NUT carcinoma are likely misdiagnosed and underdiagnosed.

NUT carcinoma is a poorly differentiated epithelial neoplasm (carcinoma) characterized by the formation of NUT fusion oncoproteins.⁵ Prior to 2023, NUT carcinoma was misclassified as a thymic neoplasm due to difficulty in identifying the exact tissue of origin. However, genetically engineered mouse models of NUT carcinoma confirmed the tissue of origin to be epithelial tissue.^{10,11} This explains the virtually ubiquitous expression of common epithelial markers, AE1/AE3 and CK5, in biopsy specimens of NUT carcinoma.¹² The most common fusion oncoprotein is BRD4-NUT resulting from the chromosomal translocation t(15;19). However, *NUTM1* fusion partners can include *BRD2*, *BRD3*, *NSD3*, *ZNF592*, or *ZNF532* – all of which can modulate expression of key proto-oncogene transcription factors. Molecular techniques can be used to identify specific *NUTM1* fusion chaperones, which may have potential prognostic and therapeutic implications.¹² Chau et al¹⁴ identified three statistically distinct risk groups of NUT carcinoma based on a retrospective analysis of 124 cases: (i) group A included non-thymic primary, BRD3-NUT or NSD3-NUT with a median survival of 36.5 months; (ii) group B included non-thymic primary, BRD4-NUT with a median survival of 10 months; and (iii) group C included patients with primary thoracic NUT carcinoma, regardless of the actual NUTM1 fusion partner, with a median survival of 4.4 months. Our case being a pulmonary NUT carcinoma fits into group C and had a time from initial presentation to death of less than 5 months.

Diagnosis of NUT carcinoma relies on the detection of *NUTM1* fusion oncoproteins, either by immunohistochemistry (staining for NUT fusion oncoprotein), cytogenetics (fluorescent in situ hybridization) or genetic sequencing (such as reverse transcriptase-polymerase chain reaction). Determination of NUTM1 fusion oncoproteins has therapeutic implications as some patients may be candidates for experimental targeted therapy with BET bromodomain inhibitors.^{17–19} Since most cases are locally advanced and/or metastasized on presentation, curative surgery is not usually feasible.^{12,13} Concurrent chemotherapy and radiation therapy is often employed as first-line therapy, although most cancers are resistant to these antineoplastic treatments – as demonstrated by our case.^{4,11} Due to this reason, prognosis is dismal with a median survival of less than 6 months.^{6,12–14} Conventional chemotherapeutic agents used in prior reports included ifosfamide-etoposide, etoposide-platinum, platinum-paclitaxel, and ifosfamide-etoposide-vorinostat.⁶ Experimental trials for NUT carcinoma are exploring BET bromodomain inhibitors, histone deacetylase inhibitors, cyclin-dependent kinase inhibitors, and immunotherapy-based regimens.⁶ The First International Symposium on NUT Carcinoma strongly encouraged patients with NUT carcinoma to participate in clinical trials given the immense unmet need for newer and effective therapies.⁶ Moreover, all physicians taking care of patients with NUT carcinoma should register their patients on the NUT Carcinoma registry to grow this repository of invaluable data.

Conclusion

NUT carcinoma is a highly aggressive cancer with dismal prognosis. Although NUT carcinoma is considered rare, it is frequently misdiagnosed and underdiagnosed. The present case documents the case of a patient with mediastinal NUT carcinoma and rapidly progressive respiratory failure. Despite maximal supportive care and multidisciplinary management, the patient experienced relentless disease progression culminating in prolonged mechanical ventilation and eventual transition to comfort-focused care. This outcome was unfortunately consistent with published survival data, which report median survival times of 6–9 months for thoracic NUT carcinoma. Due to this reason, NUT carcinoma should be considered in all young patients presenting with progressive mediastinal masses and airway compromise. Prompt immunohistochemical testing for NUT protein should be pursued in cases of poorly differentiated carcinoma of unclear origin. Additionally, early referral to specialized oncologic centers may facilitate access to clinical trials and experimental therapies. All physicians taking care of patients with NUT carcinoma should consider registering their patients on the NUT Carcinoma Registry. Patients are encouraged to participate in clinical trials as there is an urgent unmet need for novel therapies for this highly aggressive and resistant cancer.

Ethical Considerations

Written informed consent was obtained from the patient's wife for the publication of this case report. Institutional approval was not required for the publication of this case report.

Acknowledgments

The authors of this case report would like to acknowledge all the doctors, nurses, and staff of TidalHealth Peninsula Regional (Salisbury, MD) as well as John Hopkins Hospital (Baltimore, MD), who were instrumental in taking care of this challenging case.

Disclosure

The authors report no conflicts of interest.

References

1. Kroening G, Luo J, Evans MG, et al. Multiomic characterization and molecular profiling of nuclear protein in testis carcinoma. *JCO Precis Oncol.* **2024**;8(8):e2400334. doi:10.1200/PO.24.00334
2. Kees UR, Mulcahy MT, Willoughby ML. Intrathoracic carcinoma in an 11-year-old girl showing a translocation t(15;19). *Am J Pediatr Hematol Oncol.* **1991**;13(4):459–464.
3. Kubonishi I, Takehara N, Iwata J, et al. Novel t(15;19)(Q15; P13) chromosome abnormality in a thymic carcinoma. *Cancer Res.* **1991**;51(51):3327–3328.
4. French CA, Miyoshi I, Kubonishi I, Grier HE, Perez-Atayde AR, Fletcher JA. BRD4-NUT fusion oncogene: a novel mechanism in aggressive carcinoma. *Cancer Res.* **2003**;63(2):304–307.
5. Luo J, Bishop J, DuBois SG, et al. Hiding in plain sight: NUT carcinoma is an unrecognized subtype of squamous cell carcinoma of the lungs and head and neck. *Nat Rev Clin Oncol.* **2025**;22(4):292–306. doi:10.1038/s41571-025-00986-3
6. French CA, Cheng ML, Hanna GJ, et al. Report of the First International Symposium on NUT Carcinoma. *Clin Cancer Res.* **2022**;28(12):2493–2505. doi:10.1158/1078-0432.CCR-22-0591
7. NUT Carcinoma Registry. Basic facts about NUT carcinoma [Internet]. Boston (MA): Dana-Farber Cancer Institute (US); **2025**. Available from: https://nc-registry.org/?page_id=67. Accessed July 13, 2025.
8. Li W, Chastain K. NUT midline carcinoma with leukemic presentation mimicking CD34-positive acute leukemia. *Blood.* **2018**;132(4):456. doi:10.1182/blood-2017-07-796268
9. What is NUT carcinoma? [Internet]. Boston (MA): Dana-Farber Cancer Institute (US); **2025**. Available from: <https://www.dana-farber.org/cancer-care/types/nut-carcinoma>. Accessed July 13, 2025.
10. Durall RT, Huang J, Wojenski L, et al. The BRD4-NUT fusion alone drives malignant transformation of NUT carcinoma. *Cancer Res.* **2023**;83(23):3846–3860. doi:10.1158/0008-5472.CAN-23-2545
11. Zheng D, Elnegiry AA, Luo C, et al. Brd4::Nutm1 fusion gene initiates NUT carcinoma in vivo. *Life Sci Alliance.* **2024**;7(7):e202402602. doi:10.26508/lsa.202402602
12. Chen J, Li M, Lu H. Nuclear protein in testis carcinoma of the lung. *Transl Oncol.* **2023**;30:101640. doi:10.1016/j.tranon.2023.101640
13. Albrecht T, Harms A, Roessler S, Goepfert B. NUT carcinoma in a nutshell: a diagnosis to be considered more frequently. *Pathol Res Pract.* **2019**;215(6):152347. doi:10.1016/j.prp.2019.01.043
14. Chau NG, Ma C, Danga K, et al. An anatomical site and genetic-based prognostic model for patients with nuclear protein in testis (NUT) midline carcinoma: analysis of 124 patients. *JNCI Cancer Spectr.* **2019**;4(2):pkz094. doi:10.1093/jncics/pkz094
15. Jung M, Kim S, Lee JK, et al. Clinicopathological and preclinical findings of NUT carcinoma: a multicenter study. *Oncologist.* **2019**;24(8):e740–e748. doi:10.1634/theoncologist.2018-0477
16. Luo J, Sanchez M, Lee E, et al. Initial chemotherapy for locally advanced and metastatic NUT carcinoma. *J Thorac Oncol.* **2024**;19(5):829–838. doi:10.1016/j.jtho.2023.12.022
17. Piha-Paul SA, Hann CL, French CA, et al. Phase I study of Molibresib (GSK525762), a bromodomain and extra-terminal domain protein inhibitor, in NUT carcinoma and other solid tumors. *JNCI Cancer Spectr.* **2019**;4(2):pkz093. doi:10.1093/jncics/pkz093
18. Wang ZQ, Zhang ZC, Wu YY, et al. Bromodomain and extraterminal (BET) proteins: biological functions, diseases and targeted therapy. *Sig Transduct Target Ther.* **2023**;8:420. doi:10.1038/s41392-023-01647-6
19. Sun Y, Han J, Wang Z, Li X, Sun Y, Hu Z. Safety and efficacy of bromodomain and extra-terminal inhibitors for the treatment of hematological malignancies and solid tumors: a systematic study of clinical trials. *Front Pharmacol.* **2020**;11:621093. doi:10.3389/fphar.2020.621093
20. Reddy R, Woods TR, Allan RW, et al. NUT (nuclear protein in testis) carcinoma: a report of two cases with different histopathologic features. *Int J Surg Pathol.* **2019**;27(2):225–229. doi:10.1177/1066896918796606
21. Shenoy KD, Stanzione N, Caron JE, et al. Midline carcinoma expressing NUT in malignant effusion cytology. *Diagn Cytopathol.* **2019**;47(6):594–598. doi:10.1002/dc.24150

International Medical Case Reports Journal**Publish your work in this journal**

The International Medical Case Reports Journal is an international, peer-reviewed open-access journal publishing original case reports from all medical specialties. Previously unpublished medical posters are also accepted relating to any area of clinical or preclinical science. Submissions should not normally exceed 2,000 words or 4 published pages including figures, diagrams and references. The manuscript management system is completely online and includes a very quick and fair peer-review system, which is all easy to use. Visit <http://www.dovepress.com/testimonials.php> to read real quotes from published authors.

Submit your manuscript here: <https://www.dovepress.com/international-medical-case-reports-journal-journal>

Dovepress

Taylor & Francis Group