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Case Report

Hodgkin Lymphoma - A Rare Neoplasm with Diagnostic and Therapeutic Challenges

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Abstract: The Classical Hodgkin Lymphoma (CHL) had four subgroups: nodular sclerosis, mixed cellularity, lymphocyte-rich and lymphocyte-depleted. Clinical diagnosis can be difficult because the symptoms are like other diseases. Lymphadenopathies and B symptoms may allow a diagnosis of suspicion, which must be confirmed by lymph node biopsy. One of its key features is the presence of mononucleated and multinucleated cells, the Hodgkin and Reed Sternberg cells. Chemotherapy (QT) ABVD (adriamycin, bleomycin, vinblastine, dacarbazine) is considered the standard therapy. The follow-up is very important due to the relapse risk and the potential late toxicity. The authors report a rare case of cHL nodular sclerosis IV-A, International Prognostic Score (IPS) 5 diagnosed by excisional biopsy of adenomegaly of the left armpit. The 18F-fluorodeoxyglucose positron emission tomography (18F-FDG PET) revealed a good response after QT. A rare bleomycin pulmonary toxicity was diagnosed, and he died. This paper aims to review the current knowledge concerning CHL.

Keywords: Hodgkin Lymphoma; Chemotherapy; ABVD; Positron Emission Tomography Bleomycin; Pulmonary Toxicity.

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1. Introduction

In lymphomas, there is an accumulation of lymphocytes in the lymph nodes, which may extend to the bloodstream (leukemic phase) or infiltrate organs. Lymphomas are divided into Hodgkin Lymphoma (HL) and Non-Hodgkin Lymphoma (NHL). Classic Hodgkin Lymphoma (cHL) is a rare B-cell neoplasm, with the nodular sclerosis subtype (NSCHL) being the most common. In Portugal, HL accounts for less than 1% of annual cancer diagnoses. The recommended therapy for HL is ABVD chemotherapy. Late-onset pulmonary toxicity induced by bleomycin is rare [1-5].

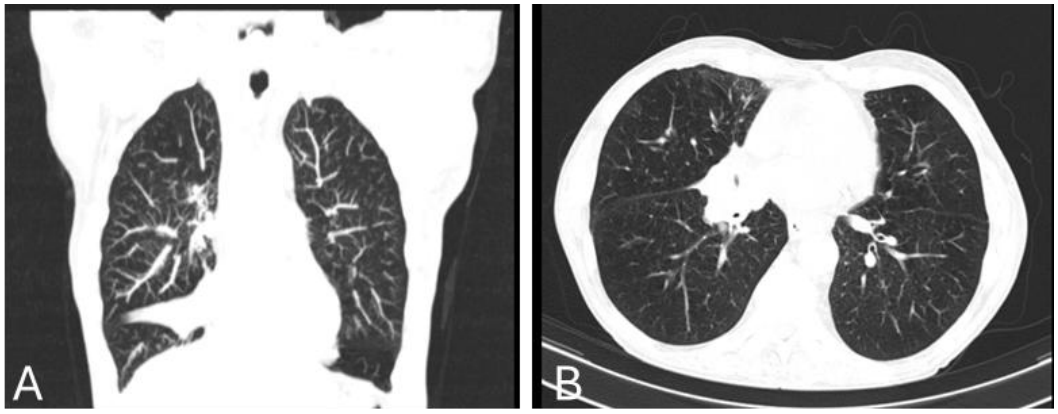
The authors present the case of a 65-year-old male smoker with chronic obstructive pulmonary disease who presented with weight loss, asthenia, anorexia, dyspnea, pleuritic chest pain, macrocytic anemia, leukocytosis, hyponatremia, generalized lymphadenopathy (cervical, axillary, mediastinal, retroperitoneal, mesenteric, inguinal), an infiltrative mass in the right lung hilum, pleural effusion, pericardial effusion, and splenic hypodensities. An excisional biopsy of a left axillary lymph node diagnosed advanced stage IV-A NSCHL, with an International Prognostic Score (IPS) of 5. The 18F-FDG PET scan showed a good response after six cycles of ABVD chemotherapy. Over the following three years, the patient experienced worsening dyspnea with minimal exertion, desaturation on the six-minute walk test, and spirometry with reduced carbon monoxide diffusion capacity due to bleomycin-induced toxicity, ultimately leading to death.

2. Case Report

A 65-year-old male, chronic smoker, with a known history of chronic obstructive pulmonary disease (COPD), classified as mMRC grade 2 and GOLD stage 3E, under regular treatment with Fluticasone + Formoterol (125 + 5 μg), was admitted to the Emergency Department with complaints of weight loss (4 kg over the past two months), asthenia, anorexia, dyspnea, and pleuritic chest pain for the past three days. He reported noticing a swelling in the left axilla for approximately one month. On examination, he appeared emaciated, weighing 45 kg, with a palpable, hard, painless mass in the left axilla measuring 10 × 8 cm. He was afebrile, hemodynamically stable, with an oxygen saturation of 91% on room air (FiO_2 0.21). Pulmonary auscultation revealed rhonchi.

Laboratory tests showed macrocytic anemia (hemoglobin 12.8 g/dL), leukocytosis (12.64 × 10^9/L) with neutrophilia (88.6%), elevated C-reactive protein (16.9 mg/dL), hyponatremia (sodium 125 mmol/L), elevated lactate dehydrogenase (533 U/L), increased beta-2 microglobulin (3.27 mg/L), and folic acid deficiency. Arterial blood gas analysis on room air revealed acute-on-chronic respiratory failure. Cervico-thoraco-abdomino-pelvic computed tomography (CT) showed extensive supra- and infra-diaphragmatic lymphadenopathy (cervical, axillary, mediastinal, retroperitoneal, mesenteric, and inguinal), pulmonary emphysema, right pleural effusion, and a small pericardial effusion. A 40 mm infiltrative mass in the right lung hilum was associated with middle lobe atelectasis (Figures 1A, 1B).

Figure 1. Chest CT scan revealed a 40 mm infiltrative mass in the right pulmonary hilum (A and B).



Notably, there were enlarged lymph nodes in the left infraclavicular region (50 mm), extending to the left anterosuperior axillary region (46 × 32 mm) and the inferior axilla (37 × 26 mm) (Figure 2A). Subcentimetric hypodense lesions were also observed in the spleen (Figure 2B). The most likely working diagnosis was a lymphoproliferative disorder. The patient was admitted to the hospital. Due to euvolemic hyponatremia, intravenous fluid therapy was initiated. Given the suspicion of acute exacerbation of chronic respiratory failure due to bacterial superinfection, treatment was started with amoxicillin/clavulanic acid 1.2 g every 8 hours for 8 days and azithromycin 500 mg once daily intravenously for 5 days, with clinical improvement. Autoimmunity screening, serologies, and microbiological tests were negative (Table 1). Serum protein electrophoresis was normal. Immunosuppression was ruled out based on negative HIV testing and normal serum immunoglobulin levels.

Spirometry revealed severe airflow obstruction (FEV_1 48%) and reduced alveolar-capillary diffusion of carbon monoxide. The pulmonology team adjusted the inhaled therapy to budesonide, formoterol, and glycopyrronium (5/7.2/160 μg) twice daily. Bronchofibroscopy showed stenosis of the right upper lobe bronchus due to extrinsic compression

by the hilar infiltrative mass. Bronchial biopsies ruled out malignancy, and microbiological analysis of the bronchial aspirate excluded tuberculosis. The excisional biopsy of the left axillary lymph node revealed a diagnosis of nodular sclerosis classical Hodgkin lymphoma (NSCHL).

Figure 2. A. Cervico-thoracic CT revealed a 50 mm left infraclavicular lymphadenopathy extending into the left axilla, along with multiple enlarged lymph nodes in both axillary regions. B. Abdominal CT revealed hypodense lesions in the spleen consistent with infiltration by lymphoproliferative disease.

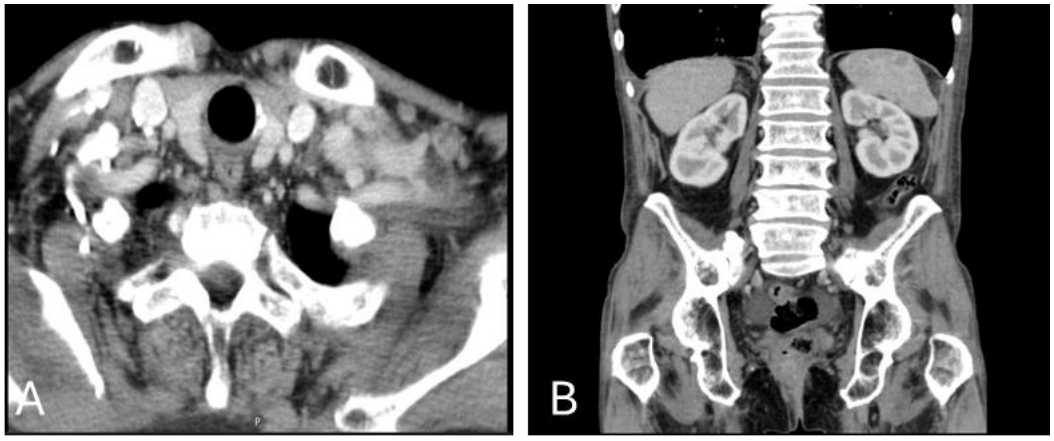


Table 1. Serologies and Microbiological Tests.

Result	Interpretation
Nasopharyngeal SARS-CoV-2 PCR	Negative
Nasopharyngeal Influenza/Respiratory Syncytial Virus PCR	Negative
Urinary Legionella/Pneumococcal Antigens	Negative
HIV	Negative
Hepatitis A/C/E Viruses	IgM/IgG negative
Hepatitis B Virus	HBsAg, Anti-HBs, Anti-HBc negative
Widal Test	Negative
Wright Test	Negative
Rickettsia	IgM/IgG negative
Cytomegalovirus / Epstein-Barr Virus / Toxoplasma gondii	IgM negative / IgG positive
Parvovirus / Treponema pallidum / Leptospira	IgM/IgG negative
Blood cultures / Urine culture	Negative

Histological examination showed effaced architecture with a multinodular pattern, fibrous septa, areas of necrosis, and the presence of Reed-Sternberg cells along with eosinophils, neutrophils, histiocytes, lymphocytes, and plasma cells. Immunohistochemistry demonstrated positivity of Reed-Sternberg cells for CD30 (clone BER-H2) and CD15 (clone MMA), with CD15 also staining polymorphonuclear cells. CD20 (clone L26) was negative in Reed-Sternberg cells, while CD3 (clone 2GV6) was positive in background lymphocytes. Hematology performed a right iliac bone marrow biopsy, which showed no evidence of neoplastic infiltration. The patient started corticosteroid therapy with prednisolone at 1 mg/kg/day. A final diagnosis of NSCHL, clinical stage IV-A (asymptomatic, with nodal, mediastinal, and splenic involvement), was established. The International

Prognostic Score (IPS) was 5, based on male sex, age ≥45 years, serum albumin <4 g/dL, hemoglobin <10.5 g/dL, and stage IV disease.

Colonoscopy revealed a 15 mm ulcer in the ileocecal valve (biopsy ruled out malignancy) and a 17 mm sessile polyp in the transverse colon, which was histologically confirmed as a tubulovillous adenoma with low-grade dysplasia. On the fourth day of hospitalization, the patient developed atrial fibrillation with rapid ventricular response. Sinus rhythm was restored after amiodarone administration, and carvedilol 6.25 mg every 12 hours was initiated. Transthoracic echocardiogram showed preserved left ventricular systolic function, grade I diastolic dysfunction of the left ventricle, and a small pericardial effusion. On the 21st day of hospitalization, the patient was discharged on prednisolone 50 mg/day, pantoprazole 40 mg, budesonide/formoterol/glycopyrronium 5/7.2/160 μg twice daily, carvedilol 6.25 mg every 12 hours, and supplementation with cyanocobalamin/pyridoxine/thiamine (0.2 mg/200 mg/100 mg) and folic acid 5 mg once daily. Serial 18F-FDG PET scans were performed. The baseline PET scan prior to chemotherapy demonstrated lymphoproliferative involvement in supradiaphragmatic and infradiaphragmatic lymph nodes, mediastinum, and spleen (Tables 2 and 3).

Table 2. 18F-FDG PET Scan Using the 5-Point Scale (Deauville Criteria).

Score	PET/CT Findings	Interpretation
1	No uptake	Negative
2	Uptake ⩽ mediastinum	Negative
3	Uptake > mediastinum but ⩽ liver	Negative
4	Uptake moderately > liver and visually higher than adjacent previous disease	Positive
5	Uptake markedly > liver and/or new lesions	Positive
Xa	New areas of uptake unlikely to be related to lymphoma	Positive

Table 3. Serial 18F-FDG PET Scans Before and After Initiation of ABVD Chemotherapy.

Findings	1st PET (Before Chemotherapy)	2nd PET (After 2 Cycles)	3rd PET (After 4 Cycles)
Uptake	Splenic, pulmonary	Reduced metabolic activity in left axillary lymph nodes	Faint uptake in left axillary lymph nodes
Lymph node involvement	Supradiaphragmatic / Infradiaphragmatic	Persistent mediastinal-hilar lymphoma	Moderate uptake in:
	Supraclavicular		Lower paratracheal
	Axillary		Left tracheobronchial
	Mediastinal		Pulmonary hila (especially right)
	Right perihilar		Mediastinal-hilar persistence
	Right pericardiac		
	Right paravertebral		
	Right costophrenic		
	Lumboaortic		
	Para-aortocaval		
	Peripancreatic		
	Mesenteric		
	Right obturator		
	Inguinal		

Deauville Score	5	4	3 / X → 2

The patient was proposed for ABVD chemotherapy. Prophylactic treatment was initiated with acyclovir 400 mg twice daily, cotrimoxazole 960 mg three times per week, metoclopramide 10 mg, and as-needed laxatives. He received influenza, pneumococcal, and SARS-CoV-2 vaccinations. A central venous catheter (Implantofix) was placed, and ABVD chemotherapy was initiated. After two cycles, a second PET scan showed partial improvement. Following four cycles, a third PET scan demonstrated significant metabolic response (Table 3). The patient completed six cycles of ABVD over six months, with good tolerance and remained asymptomatic.

He continued clinical, laboratory, and imaging follow-up every six months. Thoraco-abdomino-pelvic CT showed radiological improvement of lymphadenopathy and the pulmonary mass. Myocardial scintigraphy demonstrated normal ejection fraction. At the two-year follow-up, thyroid ultrasound was normal. However, he developed progressive dyspnea on mild exertion. Three years after exposure to bleomycin, a six-minute walk test revealed desaturation during exertion, and spirometry showed very severe airway obstruction. Bronchodilator response testing was negative, and diffusion capacity for carbon monoxide (DLCO) decreased by more than 25% compared to baseline spirometry. These findings were interpreted as pulmonary toxicity secondary to bleomycin. Ambulatory oxygen therapy and pulmonary rehabilitation were initiated. Four years after completing chemotherapy, the patient passed away.

3. Discussion and Conclusion

In developed countries, Hodgkin lymphoma (HL) is more common in males between the ages of 15–35 and after 55 years [4,5]. Risk factors for HL include Epstein-Barr virus (EBV) infection, immunodeficiency, organ transplantation, use of immunosuppressants, autoimmune diseases, and genetic predisposition [3,5–12]. In classical Hodgkin lymphoma (cHL), the presence of Hodgkin and Reed-Sternberg (HRS) cells is a hallmark [13,14]. The authors reflect on the differential diagnoses associated with HL. Other lymphoproliferative disorders are excluded by lymph node biopsy, such as Primary Mediastinal Large B-Cell Lymphoma, Diffuse Large B-Cell Lymphoma, and Anaplastic Large Cell Lymphoma [15]. Pulmonary neoplasia, tuberculosis, and fungal infections are excluded through bronchoscopy [1–5].

In HL, most patients present with painless supradiaphragmatic lymphadenopathy, most frequently in the cervical region. Mediastinal involvement may be present with cough, chest pain, dyspnea, or imaging abnormalities. HL characteristically spreads through lymphatic pathways. Although uncommon at presentation, extranodal dissemination may occur. Local spread by direct invasion or through lymphatic vessels typically involves structures contiguous with the affected lymph nodes. Hematogenous dissemination, which usually follows splenic involvement, most frequently affects the liver, bone marrow, lungs, and bones. Other sites such as skin, central nervous system, gastrointestinal tract, and musculoskeletal system are rarely involved. Patients may present with B symptoms (fever, night sweats, unexplained weight loss >10% over the previous 6 months). Pruritus and lymph node pain after alcohol intake are less common. Rare presentations in advanced HL include mesenteric, Waldeyer's ring, popliteal, or epitrochlear lymph node involvement, superior vena cava syndrome, acute spinal cord compression, CNS involvement, glomerulonephritis, testicular masses, and intestinal obstruction. In elderly patients, systemic symptoms and infradiaphragmatic disease are more common. Cytopenias, hypoalbuminemia, elevated erythrocyte sedimentation rate, C-reactive protein, and lactate dehydrogenase are frequent findings [1–5,13,15–17].

Nodular sclerosis classical Hodgkin lymphoma (NSCHL) is most common in individuals aged 10–24, shows a female predominance (in contrast to other subtypes), and is

histologically characterized by nodules separated by collagen bands, with typical green birefringence under polarized light. Mediastinal involvement occurs in 80% of cases, bulky disease in 50%, and B symptoms in 40% [5,13,16]. A definitive diagnosis requires histology and immunohistochemistry from an excisional lymph node biopsy, which reveals large multinucleated Reed-Sternberg cells, mononuclear Hodgkin cells, and an inflammatory background. Immunohistochemical markers for cHL include CD3, CD15, CD20, CD30, and CD45 [18]. Nearly all HRS cells are CD30+, and the majority (85%) are CD15+, though these markers are not specific. These cells do not express CD3 or the common leukocyte marker CD45. There is often a loss of B-cell–specific gene expression, although 30–40% of cHL cases express CD20 and 10% are CD79a+ [1–5,12,18,19].

Chest radiography can detect mediastinal masses. Cervico-thoraco-abdomino-pelvic CT provides superior anatomical disease staging [3,13]. 18F-FDG PET/CT is more sensitive than CT in detecting nodal and extranodal involvement and is recommended for initial staging in HL. Caution is advised, as PET scans are frequently positive in areas of infection or inflammation unrelated to HL. When uptake occurs in atypical or previously unidentified areas, further clinical and pathological evaluation is recommended [1–5,18,20]. In this case, NSCHL presented in an atypical and rare manner: a 65-year-old male smoker with chronic obstructive pulmonary disease, weight loss, dyspnea, chest pain, lymphadenopathy, macrocytic anemia, leukocytosis, hyponatremia, elevated C-reactive protein and beta-2 microglobulin, an infiltrative hilar mass in the right lung, pleural and pericardial effusions, and splenic hypodensities, all supporting the hypothesis of a lymphoproliferative disease with nodal, mediastinal, and splenic involvement. A left axillary lymph node biopsy confirmed NSCHL (presence of Reed-Sternberg cells).

Bone marrow biopsy excluded medullary involvement. Corticosteroid therapy with prednisolone 1 mg/kg/day was initiated. Bronchoscopy excluded lung cancer and tuberculosis. Immunosuppression was ruled out (negative HIV, normal serum immunoglobulins). 18F-FDG PET showed supra- and infradiaphragmatic involvement. Given the advanced stage IV-A (stage IV indicating diffuse or disseminated extranodal involvement with nodal disease, A indicating absence of B symptoms) and an International Prognostic Score (IPS) of 5 (male sex, age ≥45 years, serum albumin <4 g/dL, hemoglobin <10.5 g/dL, clinical stage IV), the patient underwent six cycles of ABVD chemotherapy. Serial PET scans demonstrated a favorable response to treatment.

Clinical staging assesses the extent of disease and is important for both prognosis and therapeutic planning. The Ann Arbor staging system (Cotswolds modification) comprises four stages. The letter A denotes absence of symptoms, while B includes fever >38°C for three consecutive days, night sweats, or unexplained weight loss greater than 10% of body weight over the previous six months. The designation X refers to bulky disease (mass with maximum diameter ≥10 cm or mediastinal mass with a maximum diameter ≥1/3 of the maximum intrathoracic diameter at the T5–T6 intervertebral space on chest X-ray), and E indicates localized involvement of a single extranodal site contiguous with or proximal to the involved nodal region. Stage I refers to involvement of a single lymph node region or lymphoid structure (spleen, thymus, or Waldeyer's ring), or a single extranodal site (IE).

Stage II includes involvement of two or more lymph node regions or lymphoid structures on the same side of the diaphragm (II), or localized involvement of a single extranodal site (IIE). The number of anatomical regions involved should be specified (e.g., II3, since mediastinal lymph nodes constitute a single nodal region). Stage III includes lymph node regions or lymphoid structures on both sides of the diaphragm (III), or localized involvement of a single extranodal site (IIIE), or splenic involvement (IIIS), or both (IIIES). Subclassification into stage III1 includes splenic, hilar, celiac, or portal node involvement, whereas stage III2 includes para-aortic, iliac, and/or mesenteric node involvement. Stage IV corresponds to diffuse or disseminated involvement of one or more extranodal organs, with or without associated nodal involvement, or localized involvement of the liver or bone marrow [1,2,3,4,5]. Staging by bone marrow biopsy is not required in most cases of

HL, as bone marrow involvement is rare in stage I or II in the absence of adverse prognostic factors. Bone marrow biopsy is recommended in patients with B symptoms, peripheral cytopenias, and/or stage III–IV disease, as occurred in this case [18].

The Ann Arbor clinical stage and the presence of adverse prognostic factors stratify patients into three risk groups according to the European Organization for Research and Treatment of Cancer (EORTC): early-stage favorable, early-stage unfavorable (or intermediate), and advanced-stage. Early-stage disease has an excellent prognosis with overall survival (OS) greater than 90%, whereas advanced-stage disease presents an OS of 75–90%. In advanced-stage HL, the International Prognostic Score (IPS) is used to identify patients who may benefit from individualized therapy, as it predicts five-year progression-free survival (PFS) and OS. The IPS is based on seven factors (age ≥45 years, male sex, serum albumin <4 g/dL, hemoglobin <10.5 g/dL, clinical stage IV, leukocyte count ≥15,000, and lymphocyte count <600 or <8% of the total leukocyte count), each of which has been shown to have a similar independent prognostic impact, with the presence of each factor reducing five-year PFS by 8%. The presence of three or more adverse prognostic factors is associated with a five-year PFS of 55% and OS of 70% [1-5,16,20,21].

The decision to initiate treatment for HL must be individualized, considering risk-benefit analysis. The economic impact of treatment includes short-, medium-, and long-term complications and improvements in survival. Treatment is determined based on histology and whether the patient has early-stage favorable, early-stage unfavorable, or advanced-stage disease. This stratification allows the intensity of therapy to be adjusted according to the risk of relapse and potential long-term toxicity [1-5,22]. The standard treatment for advanced-stage HL is ABVD chemotherapy, as it offers better outcomes and lower toxicity (including lower incidence of infertility, myelodysplastic syndromes, and secondary acute myeloid leukemia). The standard therapeutic regimen for advanced-stage HL consists of six to eight cycles of ABVD, with reported five-year PFS rates of 70% and OS between 82% and 90% [1-5,20,23,24]. The role of radiotherapy (RT) in advanced-stage disease remains undefined, but irradiation of initially bulky masses or residual masses may be considered after first-line ABVD chemotherapy [2,16,18,25,26].

It is essential to evaluate the response to initial treatment, as this determines the need for additional therapy. Restaging should include all tests that were abnormal at initial staging. Response assessment in HL is complicated by the frequent persistence of residual masses (particularly in the mediastinum), which are often due to fibrosis rather than active disease or relapse risk. CT evaluates anatomical remission but cannot distinguish active disease from fibrosis. 18F-FDG PET is more reliable in identifying persistent active disease, as it predicts treatment outcomes early on and serves as an indicator of chemosensitivity after two cycles of ABVD. A negative 18F-FDG PET after two cycles of ABVD indicates an excellent prognosis, whereas residual or non-modifiable uptake is associated with poorer outcomes. Predicting final therapeutic response based on early 18F-FDG PET assessment may be useful for implementing response-adapted treatment strategies [1-5,13,18,27-29].

The late effects of chemotherapy are major determinants of morbidity, mortality, and quality of life, requiring long-term follow-up. During the first ten years after treatment, lymphoma progression is the leading cause of death; however, beyond that period, mortality is primarily related to late effects. Secondary neoplasms most commonly include solid tumors (lung, breast, skin, gastrointestinal tract) and hematological malignancies (leukemia, myelodysplastic syndromes, secondary lymphomas). Radiotherapy (RT) is associated with an increased risk of cancer in irradiated areas, while chemotherapy is linked to leukemia, non-Hodgkin lymphoma, and lung cancer. Influenza, pneumococcal, and meningococcal vaccinations are recommended in patients who receive splenic RT. Following treatment for HL, there is an increased incidence of myocardial infarction, heart failure, coronary artery disease, and valvular heart disease. Left ventricular ejection fraction should be assessed both before and after doxorubicin-based chemotherapy. The risk of cardiovascular morbidity and mortality is associated with supradiaphragmatic RT and

anthracycline-based chemotherapy. Neck irradiation is linked to hypothyroidism and an increased risk of thyroid neoplasms. ABVD chemotherapy is associated with azoospermia. In women, alkylating agents or pelvic RT can lead to ovarian failure, premature menopause, and infertility. Other complications include myelosuppression after chemotherapy, increasing the risk of infections [1-5,20,30,31].

Bleomycin-induced pulmonary toxicity can vary in presentation, including interstitial fibrosis, interstitial pneumonia, organizing pneumonia, eosinophilic pneumonia, diffuse alveolar damage, and pneumonitis. Most patients present with a subacute course (symptoms within 1 to 6 months after exposure), although chronic progression (after 6 months) may occur, as seen in this patient. Clinical manifestations include cough, pleuritic pain, dyspnea, hypoxemia, pulmonary opacities, or an asymptomatic decline in carbon monoxide diffusion capacity. Risk factors include age, smoking, underlying pulmonary disease, cisplatin, gemcitabine, prior radiotherapy, use of filgrastim, renal insufficiency, and cumulative dose. Pulmonary function testing with carbon monoxide diffusion measurement is recommended before and after bleomycin treatment. Management includes permanent discontinuation of bleomycin, switching to the AVD regimen, and initiating systemic corticosteroids for 4–6 weeks in acute cases [1-5,18,32-35]. In this case, the risk factors for bleomycin-induced pulmonary toxicity were age (65 years), smoking, and underlying chronic obstructive pulmonary disease. The patient developed progressively worsening dyspnea on exertion. Three years after bleomycin exposure, the six-minute walk test showed oxygen desaturation, and spirometry revealed a more than 25% reduction in carbon monoxide diffusion compared to baseline, culminating in death.

Post-treatment follow-up should be individualized based on clinical context (age, disease stage, and initial therapy). Clinical and laboratory evaluations are recommended every 2–4 months during the first two years after treatment, every 3–6 months during the subsequent three years, and annually thereafter. All patients should receive annual influenza vaccination. Chest X-ray or CT is recommended every 6–12 months during the first 2–5 years. Abdominal and pelvic CT is recommended every 6–12 months during the first 2 to 3 years. Routine use of 18F-FDG PET for follow-up is not recommended due to a high incidence of false positives. An echocardiogram is advised 10 years after completing therapy, even in asymptomatic patients [1-5,18].

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Conflicts of Interest: None.

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