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(CASE REPORT)

Pulmonary Lymphomatous Granulomatosis: Case report of an uncommon tumor and a brief review of the literature

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Abstract

Pulmonary Lymphomatous Granulomatosis (PLYG) is a very rare condition induced by Epstein-Barr virus (EBV) that can present with nonspecific symptoms, leading to diagnostic delays. It has a low frequency and is heterogeneous with respect to clinical, radiological, and pathological features, which makes it a challenging diagnosis and often leads to misdiagnosis with more frequent inflammatory or neoplastic diseases. The case report illustrates the complexity of the path of a patient with high-grade PLYG and the importance of including it in the differential diagnosis of lung masses. Proper diagnosis requires a multidisciplinary approach, combining clinical presentation, high-quality imaging, careful histopathological analysis, and EBV in situ hybridization. The recognition of the various forms of PLYG and its changing grade-related treatment paradigms is the key to prompt intervention and better patient outcomes. As highlighted in this report, collaborative expertise in handling these rare entities is paramount and ultimately leads to increased awareness and improved patient care.

Keywords: Pulmonary Lymphomatoid Granulomatosis; Epstein-Barr Virus; Lymphoproliferative Disorder; Angiocentricity; T-Cell Immunodeficiency; Granulomatosis

1. Introduction

Lymphomatoid Granulomatosis (LYG) is an uncommon and challenging condition to diagnose. It is an EBV-related LPD, characterized by the infiltration of tissue by atypical lymphoid cells with an angiocentric distribution, causing angiodestruction. LYG disorder was first recognized in 1977 and has gone through various classification changes. [1] LYG is currently classified by the World Health Organization (WHO) as an EBV-positive B-cell lymphoproliferative disorder with its own grade system (Grades 1-3). The percentage of EBV-infected B-cells determines the grading of LYG. This classification is important because it determines the treatment plan, as low-grade disease is treated with immunomodulatory therapy, while high-grade disease is treated with aggressive chemotherapy. [2]

LYG is a disease predominantly affecting extranodal sites, with the lungs being the most frequently affected organ. It tends to present with nonspecific respiratory and constitutional manifestations, potentially causing delays in diagnosis. [3] [4] The central nervous system is also a common site and has serious prognostic consequences. [5] [6] LYG is a diagnostic and treatment enigma to most clinicians and pathologists because of its rarity and unpredictable

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presentation. We report this case to contribute to the limited literature on this rare condition, as it exemplifies an effective, modern management model for high-grade PLYG with subsequent low-grade recurrence. This is useful in understanding the changing clinical progression and treatment patterns in this entity.

2. Case Presentation

A 43-year-old female developed a 4-6-month history of progressive respiratory symptoms, which initially masqueraded as recurrent pneumonia. Her main complaints were chronic dry cough, gradually worsening dyspnea on exertion, and occasional fevers up to 101°F. The onset of constitutional symptoms occurred 2-3 months ago with 12-pound weight loss, drenching night sweats, and extreme fatigue. Neurological symptoms were present 6-8 weeks prior to diagnosis and included occasional confusion, slight left-sided weakness, bifrontal headaches, personality change, and word-finding problems. At this time, she was given several rounds of empirical antibiotics with little effect and presented twice to the emergency department with acute dyspnea.

On physical examination, the patient exhibited a low-grade fever (100.6°F), a rapid heart rate (104 bpm), and low oxygen saturation (91% on room air). Lung examination revealed crackles on both sides on inspiration and decreased breath sounds at the base of the right lung. Neurologically, there was a mild left-sided weakness, mild cognitive problems, and hyper-reflexes. Laboratory studies showed normocytic anemia with the hemoglobin level of 8.9 g/dL. She had very high inflammatory markers (ESR 82 mm/hr and CRP 52 mg/L), high LDH (620 U/L), and a slightly elevated calcium level. Chest CT revealed numerous nodules in both lungs, ranging in size from 0.5cm to 3.5cm. These nodules were found close to the blood vessels and airways, with cavitation of the larger ones. PET scan confirmed high activity of the lung nodules. Extensive white matter abnormalities were noted in the brain MRI. These abnormalities, combined with characteristic enhancement patterns, indicated a disease of an angioinvasive nature. These MRI findings led to the inclusion of LYG in the differential diagnosis. Other differentials included: primary CNS lymphoma, metastatic disease, inflammatory and autoimmune conditions, sarcoidosis, infections, and rare conditions such as Erdheim-Chester disease and amyloidosis.

A CT scan-guided lung biopsy of one of the largest nodules showed a combination of the typical features of LYG: the variety of lymphoid cells, tissue invasion, blood vessel damage, and foci of necrotic tissue with granulomas. Immunohistochemistry (IHC) studies showed CD20+, CD79a+, and BCL-6+ (Figure 1, A, B, C, D). In situ hybridization for EBER (Epstein-Barr-encoded RNA) demonstrated over 50 EBV-positive cells/hpf, consistent with a Grade 3 (high-grade) lymphomatoid granulomatosis. Monoclonal immunoglobulin heavy chain gene rearrangement was confirmed as B-cell clonality. The final diagnosis was pulmonary lymphomatoid granulomatosis (PLYG).

Six cycles of R-CHOP chemotherapy were used, and the patient responded well. The occasional side effects were managed successfully, and the patient experienced no trouble with the treatment. Complete metabolic recovery was observed, accompanied by improvements in respiratory, neurological, and imaging abnormalities. She stayed in full remission without EBV DNA being detected after 18 months.

Twenty months after treatment, surveillance images showed new bilateral pulmonary nodules, with mild respiratory symptoms. Repeat biopsy confirmed the recurrence of Grade 1 lymphomatoid granulomatosis with a greatly reduced EBV-positive B-cell burden (<20 cells per HPF), the most desirable recurrence pattern, rather than a high-grade recurrence. The low-grade recurrence was successfully treated with interferon alfa-2b (3 million units subcutaneously, three times per week) for 12 months without the necessity of another intensive chemotherapy course. This procedure relied on the immunomodulatory effects and T-cell responses directed against EBV using interferon.

The patient has been in a second complete remission for over three years following treatment of the recurrence. Mortality rates were high in the past, with 63-90 percent at 5 years, but, due to a low-grade recurrence profile, the present rituximab-based treatment has significantly reduced the prognosis. The patient has returned to normal functional status and enjoys a normal quality of life.

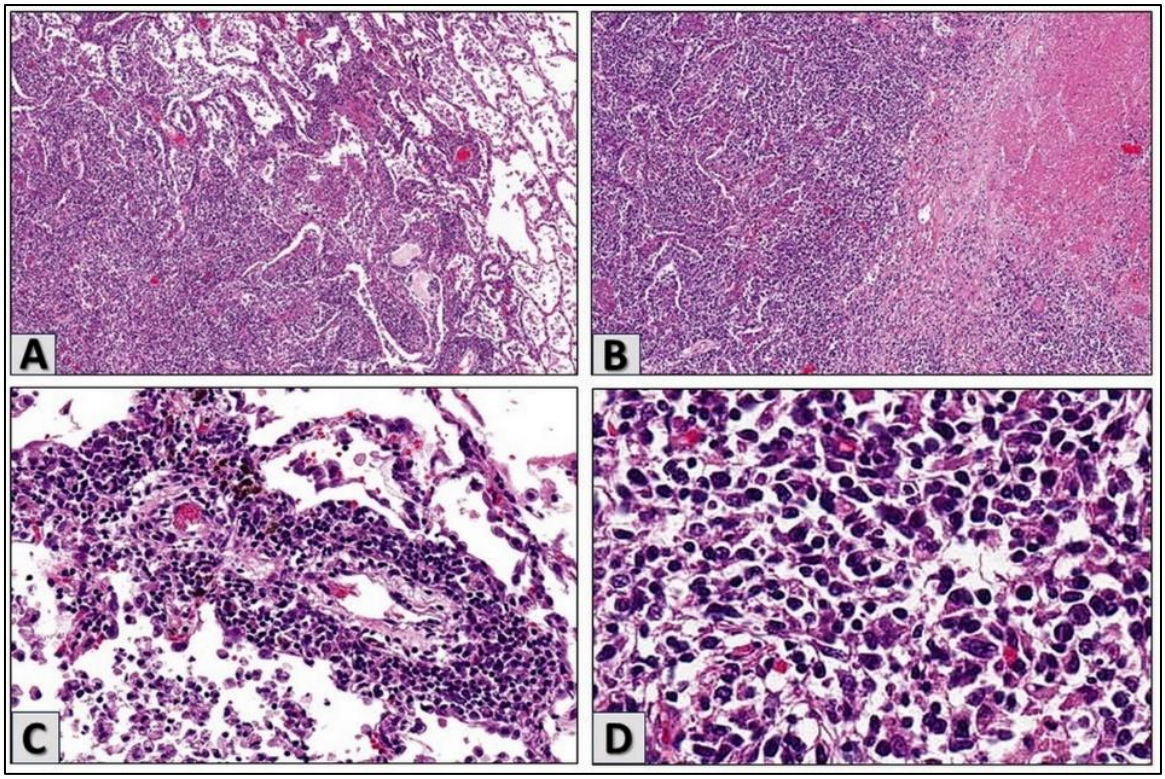

Figure 1 Histomorphology of pulmonary lymphomatous granulomatosis (PLYG)

- **1A:** Low power view showing diffuse solid infiltration of lung parenchyma (H&E stain X20)
- **1B:** low power view showing solid lymphoid infiltration with prominent necrosis (upper right corner) (H&E stain X20)
- **1C:** Intermediate power view showing infiltration of blood vessels by atypical lymphoid cells with vascular damage (H&E stain X40)
- **1D**: High power view showing diversity of atypical lymphoid cells infiltration of the lung tissue (H&E stain X60)

3. Discussion

3.1. History, Epidemiology, Risk Factors, and WHO Classification.

In 1972, Liebow et al. were the first to describe LYG as a clinicopathologic entity. [1] In the beginning, its classification was uncertain as it was regarded as a gray zone between a vasculitis and a lymphoblastic proliferative disease because of its typical angiocentric and angiodestructive features. Since the inflammatory infiltrate contains a large proportion of T-cells, it was initially believed to be a T-cell disorder. [1] Later studies verified that the cancerous cells are EBV-positive B-cells, resulting in their present classification. [7] [8] [9]

Currently, the WHO classifies LYG as a rare EBV-related B-cell lymphoproliferative disorder. It is described as an angiocentric and angiodestructive lymphoproliferative disease composed of EBV-infected B cells against a background of reactive T cells, typically affecting extranodal sites. [1] The WHO classification further presents a grading system (Grades 1, 2, and 3) according to the quantity of EBV-positive large atypical B-cells. Grade 1 contains a small number of EBV-positive cells, Grade 2 contains more, and Grade 3 contains many large B-cells, which is synonymous with an aggressive diffuse large B-cell lymphoma. [2] Our case presented initially as grade 3 and recurred in the form of a G1 tumor.

LYG is a rare disease, the prevalence of which is unknown; however, it is estimated that only approximately 600 cases have been reported in the literature. It occurs most commonly in adults between their fourth and sixth decades of life, with a pronounced male prevalence (a two-to-one male-to-female ratio). No racial predilection is known. [1] The ultimate concern for the development of LYG is an impaired or malfunctioning immune system. Although most patients have no history of known immunodeficiency, the disease is closely linked to conditions that compromise the immune surveillance of EBV. [10] Examples of these conditions are iatrogenic immunosuppression (e.g., due to methotrexate or

after transplantation), congenital immunodeficiencies (such as Wiskott-Aldrich syndrome), and acquired immunodeficiencies, such as HIV/AIDS. [10] [11] LYG has also been associated with autoimmune disorders such as rheumatoid arthritis and Sjogren syndrome. It has been postulated that the pathogenesis may be related to the immune system's inability to regulate B-cells infected with EBV, which leads to the appearance of lesions. [10] [12]

3.2. Pathogenesis and Pathophysiology of PLYG

The pathogenesis of pulmonary lymphomatoid granulomatosis (LYG) is thought to be both chemokine-dependent and EBV-related. Chemokines such as CXCL9 and CXCL10 (previously identified as IFN-γ-induced protein 10 [IP-10] and IFN-γ-inducible protein 10 [IP-10], respectively) tend to localize to the reactive cells in the tissue surrounding areas of necrosis secondary to tumor infiltration. [13] This leads to the assumption that the surviving cells along the tumor's perimeter are responsible for the chemokines that cause tissue damage and necrosis.

A separate study also highlighted the significance of Mig and IP-10 in lymphoproliferative disease tissues from EBV-positive LYG. They found that necrosis and vascular damage were the characteristic features of the lymphoproliferative lesions. [14] At the time of the study, the mechanism of chemokines was unknown, but under in vitro investigation, they proved to have potent T- and NK-cell chemoattractant activity. IP-10 also demonstrated an ability to increase the release of granule-derived serine esterases, which are part of the family of proteolytic enzymes stored in the cytotoxic granules of various immune cells, including cytotoxic T lymphocytes. [14] This article also includes a preclinical study that found an association between the interruption of IP-10 and Mig and the prevention of tumor growth, as well as reduced tumor growth, tumor necrosis, and limited metastasis. [14]

Other EBV-positive B-cell lymphoproliferative disorders (EBV+ B-LPDs) have also been found to undergo a related pathophysiological mechanism involving chemokines Mig and IP-10, as seen in EBV-related LYG. However, the mechanisms related to organ damage differ significantly between the two diseases. Most cases of EBV+ B-LPDs cause damage in tissues through the proliferation of cells infected with EBV, whereas LYG does not do this with EBV cells selectively, but with the host's immune system working against EBV. [13] This ultimately leads to a characteristic vasculitis that sets it apart from EBV+ B-LPDs. However, it is essential to note that the range of EBV+ B-LPDs is broad and encompasses various pathological mechanisms. [12]

A literature review on the carcinogenic mechanisms of virus-associated lymphoma found that EBNA1, a protein found to be the only consistently expressed viral protein during the latent phase of EBV, has been notably associated with the transformation of normal B lymphocytes infected with EBV into lymphoblastoid cell lines. [12] [19] Many studies reviewed point to the participation of this protein in the progression to malignancies. Mechanisms include the overexpression of oncogenes such as c-Myc, silencing of tumor suppressors, alterations to the cell cycle and migration, and regulation of adhesion. [12] This article states that previous studies have noted EBNA1 also leads to oxidative stress, induction of apoptosis, and DNA damage, as well as co-immunoprecipitation with Nm23-H1 in lymphocytes, leading to the spread of EBV-associated tumors. EBNA2 is also thought to be involved to a lesser degree in mechanisms such as the activation of transcriptional genes, like CD23, and the negative regulation of genes, like BCL6. [12] EBNA2 is also a functional homologue of activated Notch, a gene that regulates many developmental processes in tissues and is considered both an oncogene and a tumor suppressor. Further studies are needed to establish a direct correlation between EBNA2 and LYG development. EBNA3, LMP1, LMP2A, and LMP2B are other EBV proteins that have also been shown to play a role in the oncogenic process of EBV-related lymphomas. [19]

The EBV genome encodes mature miRNAs that are vital for regulating the viral life cycle, but also play an integral part in the progression of EBV-associated malignancies. They have different expression profiles depending on the cancer type and may also eventually interfere with the host miRNAs, which can lead to more severe complications. [12]

A study of tumor cells in LYG has demonstrated a functional defect in CD8+ cytotoxic T cells, which is thought to be a leading cause of disease development. [15] A review article found that there were moderately to severely decreased baseline CD3+ T cells. They also found that there was a more significant decrease in CD8+ T cells than in CD4+ T cells, and a less pronounced decrease in NK and B-cell subsets. [13] A comparison of the clonality of LYG tumors with their histologic grade, studied through immunoglobulin gene polymerase chain reaction, shows that there is increased clonality in grade 3 lesions (69% clonally rearranged). Low-grade LYG lesions were found to be infrequently clonal, with only 8% and 50% of lesions in grades 1 and 2, respectively, being clonally rearranged. They found that the shift from polyclonal to monoclonal disease is noted with increased histological grade and likely represents the selective transformation of EBV-infected B-cell clones. [13]

Although 95% of adults are infected with EBV at some point, most do not develop EBV-associated lymphomas. [12] Therefore, the virus does not act alone, and further investigations are needed to determine the mechanism by which malignancies, such as EBV+ PLYG, are produced.

3.3. Correlation with the Literature: Clinical Presentation, Diagnosis, and Treatment

Our patient exhibits a remarkable similarity in clinical course to the classical yet complicated presentation of LYG in the literature. LYG occurs mostly in the lungs, and more than 90 percent of patients have respiratory symptoms, including cough and dyspnea, which our patient experienced a few months ago. The pitfall of her initial misdiagnosis of recurrent pneumonia is also very common since these early symptoms are nonspecific. Constitutional B-symptoms (fever, night sweats, and abnormal weight loss) also reflect the archetypal description of a systemic LPD. [1]

A significant feature of the case is the early and extensive central nervous system (CNS) involvement, which is present in up to 50% of LYG patients and serves as a negative prognostic factor. Neurological problems exhibited in our patient (confusion, headache, and focal weakness) led to ordering advanced imaging, which was critical in the diagnosis. The brain MRI results, showing T1-hypointense and T2-hyperintense white matter lesions, implied the presence of an angioinvasive process, which is characteristic of LYG. [5] [6] The bilateral pulmonary nodules of a perivascular distribution and cavitation that she has on her chest CT are typical of LYG on imaging. High-grade disease is also congruent with the high metabolic activity on the PET scan. [16]

A CT-guided lung biopsy was the gold standard for making a definitive diagnosis. Our histopathological result showed the triad of characteristic features of LYG: a polymorphic lymphoid infiltration, angiocentricity and angiodestruction, and foci of necrosis. The atypical cells were identified as B-cells based on immunohistochemistry (CD20+, CD79a+, BCL-6+). [7] [17] Importantly, diagnosis and grading were established by the fact that EBER in situ hybridization showed a high count of EBV-infected cells, establishing Grade 3 disease. This result is critical because EBV-infected B-cells are the foundation of the WHO grading system, and the result is directly related to the choice of treatment. [2] Further investigation of the diagnosis of a malignant process was carried out by the confirmation of B-cell clonality by immunoglobulin heavy chain gene rearrangement.

The intervention plan used in our case is representative of the existing, grade-based therapeutic paradigm. Combination immunochemotherapy is the standard of care in high-grade (Grade 3) LYG, which is an aggressive lymphoma. [13] Our patient was treated with six rounds of R-CHOP, which is a common regimen in aggressive B-cell lymphomas, and demonstrated a fantastic complete metabolic remission. The latter is in line with reports that chemotherapy with rituximab can trigger lasting remissions in a substantial number of patients with high-grade disease. [1] [18]

The management of the recurrence is the most educational aspect of this case. It is described in the literature that patients who received treatment for high-grade disease may relapse, but with a lower-grade, immune-dependent type of LYG, believed to be caused by the preservation of defective immune surveillance against EBV. [13] [18] It is exactly what happened in our patient, whose recurrence was proved through biopsy to be Grade 1 disease. Instead of a repeat of intensive chemotherapy, she was treated successfully with interferon alfa-2b. The rationale behind this strategy is that low-grade LYG can be responsive to immunomodulatory therapies that boost the host's anti-EBV T-cell response. [14] [19] Recent long-term research has confirmed this approach, demonstrating that interferon can produce prolonged remission and significantly increase patient survival in low-grade LYG, as has occurred in our patient. [1] [14] Her three-year and continuing second remission, in which she returned to full functional function, demonstrates the efficacy of this sequential, customized treatment regimen and is a very promising outcome compared to the historically bleak prognosis of this disease.

3.4. Future Directions in Diagnosis and Treatment of PLYG

The current technological revolution is expected to advance even more in the management of rare diseases such as PLYG. More sensitive yet less invasive methods are the way forward in diagnostics for the future. The advent of more sophisticated liquid biopsies, namely circulating tumor DNA (ctDNA) and EBV DNA, will likely play a greater role, both in initial diagnosis and long-term surveillance. This may decrease the necessity of repetitive invasive tissue biopsies, enabling an earlier date of PLYG action of recurrence and real-time treatment-response surveillance. [20] Moreover, the analysis of radiological and pathological images with the use of artificial intelligence (AI) can offer more precise, faster, and standardized PLYG grading and reduce inter-observer variability in the near future. The combination of Artificial Intelligence (AI) and machine learning algorithms is expected to transform the diagnostics and prognostics of PLYG. In pathology, AI algorithms can be trained to process digital slide images, allowing fast, quantitative, and reproducible estimation of the count of EBV-positive cells, the basis of the WHO grading system. This can standardize the grading process across various institutions and pathologists, leading to more consistent treatment decisions. [21] Radiology AI

can add to the identification of small nodules on CT images and improve the measurement of tumor load changes over time with accuracy that outperforms the capability of human observers. [22] Not only that, but by combining large amounts of data, such as genomic, clinical, and imaging data, machine learning might discover new prognostic biomarkers and forecast how a particular patient will respond to a particular treatment, leading to a truly personalized treatment plan.

The future on the therapeutic front is towards very specific, personalized medicine. Though the existing treatments are effective, they are toxic. New treatments are being developed to target the distinctive biology of EBV-related malignancies. These involve adoptive T-cell therapies, in which the patient's T cells are modified to target and kill EBV-infected cells specifically. [23] Also, there is a bright perspective in creating new small molecule inhibitors that regulate pathways needed by EBV-transformed B-cells to survive. [24] The goal of these next-generation drugs is to give patients lasting remissions that cause fewer side effects and eventually change the long-term prognosis of those with this rare and complicated disease.

3.5. What Have We Learned from This Case?

The case PLYG provides several important learning lessons to clinicians. First, it highlights the relevance of having a high index of suspicion of LG in patients who come up with a combination of refractory respiratory symptoms, constitutional B-symptoms, and neurological impairments. The diagnosis of pneumonia is a very typical trap that not only postpones proper care but also initiates a false diagnosis.

Second, the case gives a striking example of the current, grade-adapted therapeutic model of LYG. A complex and effective treatment plan is illustrated in the successful management of the first high-grade disease with intensive immunochemotherapy (R-CHOP) and the second, low-grade recurrence with a less toxic, immunomodulatory regimen (interferon). The sequential method not only resulted in a lasting second remission but also helped preserve the patient's quality of life by preventing the cumulative toxicity of recurrent chemotherapy.

Lastly, the experience of this patient is a strong indicator of the dramatically better prognosis of LYG. Diseases with very high mortality can now be treated as chronic, curable diseases, with long-term survival and restoration of full functional status. We hope that reporting this and similar cases will encourage clinicians to engage in more aggressive diagnostics and personalized therapy for this rare but treatable cancer.

Abbreviations: Pulmonary Lymphomatoid Granulomatosis (PLYG); **Epstein-Barr virus (EBV); Lymphoproliferative disorder (LPD); Immunohistochemistry (IHC)**

4. Conclusion

We describe a case of high-grade Pulmonary Lymphomatoid Granulomatosis, which has been cured using immunochemotherapy, and a unique, low-grade recurrence that was completely cured using an immunomodulatory agent. The presented case is reported not only due to the rarity of the disease but also to emphasize the essential role of a grade-based, sequential treatment approach. It demonstrates that, despite a high-grade presentation, a low-grade relapse can be successfully treated using fewer intensive therapies, with excellent long-term results. Through this experience, we aim to raise clinicians' awareness of the evolving management of PLYG, solidifying the transition from a previously lethal diagnosis to a curable one and highlighting the possibility of achieving lasting remission and a normal quality of life.

Compliance with ethical standards

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Disclosure of conflict of interest

All authors make the following declarations

- **Payment/services information:** All authors have declared that they received no financial support from any organization for the submitted work.
- **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might be interested in the submitted work.

Statement of ethical approval

Ethical review and approval were not required for this study involving human participants. The paper has been sufficiently anonymized to maintain the patient's confidentiality.

Statement of informed consent

Consent for the publication of this case was obtained from the patient.

Data access statement

All relevant data are included in the paper.

Author contributions

All authors contributed equally to producing this manuscript.

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