

Postoperative polymyalgia rheumatica after lung cancer resection in the absence of immune checkpoint inhibitors: A case report and literature review

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Abstract. The present case report describes the development of polymyalgia rheumatica (PMR) in a 73-year-old male patient following lung cancer resection in the absence of prior exposure to immune checkpoint inhibitors (ICIs). Following a left upper lobectomy, the patient presented with a persistent fever and diffuse myalgia and arthralgia, which was unresponsive to antibiotic therapy. Subsequently, the patient was treated with non-steroidal anti-inflammatory therapy, which led to rapid clinical improvement and complete resolution of PMR-associated symptoms during the 6-month follow-up. Unlike the well-recognized ICI-associated PMR, the present case highlights a distinct postoperative presentation, suggesting that tumor-associated inflammation and surgical stress may act as independent triggers. Through a literature review, the present study further explores the potential mechanistic links between lung cancer and PMR, including the role of pro-inflammatory cytokines such as interleukin-6. These findings provide additional insight into PMR pathogenesis beyond immunotherapy-related mechanisms and may aid in the differential diagnosis of an unexplained fever and musculoskeletal symptoms in postoperative patients with cancer.

Introduction

Polymyalgia rheumatica (PMR) is a prevalent inflammatory rheumatic disease predominantly affecting individuals aged ≥ 50 years, characterized by pain in the muscles of the limbs and

joints, accompanied by a systemic inflammatory syndrome (1). Clinically, PMR presents with symmetrical pain and morning stiffness in the shoulder and pelvic girdle muscles, often accompanied by systemic symptoms and elevated acute-phase reactants. The condition is frequently associated with giant cell arteritis (GCA) (2-4). The incidence of PMR increases with age, and $\sim 9\%$ of patients exhibit subclinical GCA, further complicating its diagnosis and management (5).

The etiology and pathogenesis of PMR remain incompletely understood. Current evidence suggests a multifactorial origin involving genetic susceptibility, immune dysregulation (such as imbalances in T-cell subsets and activation of pro-inflammatory cytokines, including IL-6) and environmental triggers, such as infections or vaccinations (6,7). Regardless of the initiating factor, exposure to environmental stimuli activates both innate and adaptive immune responses, ultimately contributing to the development of PMR (8).

In recent years, the relationship between PMR and malignancy has become a focus of clinical research, although a definitive causal link remains controversial. A population-based study in Sweden demonstrated a markedly increased risk of malignancy within 1 year of PMR diagnosis, suggesting that PMR may act as a paraneoplastic syndrome or as an inflammation-driven comorbidity (9). By contrast, an analysis using the UK General Practice Database found no direct association between PMR and cancer risk (10). Notably, with the increasing use of immunotherapy, cases of PMR induced by immune checkpoint inhibitors (ICIs) [such as programmed cell death protein 1 (PD-1)/programmed death-ligand 1 (PD-L1) inhibitors] have been reported, likely resulting from autoimmune-like reactions mediated by T-cell overactivation (11). Outside the context of immunotherapy, secondary PMR following lung cancer surgery remains extremely rare, and the potential roles of surgical trauma and the tumor-associated inflammatory microenvironment in disease onset have not been systematically investigated.

Lung cancer is among the most prevalent malignancies worldwide (12), and the timely identification and management of postoperative complications are critical for patient outcomes. The present study reports the case of a 73-year-old man who developed PMR following lung cancer resection in the absence of immunotherapy. The diagnosis was established based on

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clinical features, laboratory findings, imaging characteristics and the American College of Rheumatology-European Alliance of Associations for Rheumatology (ACR-EULAR) 2012 classification criteria (13). Notably, the patient showed a favorable response to the selective cyclooxygenase-2 (COX-2) inhibitor celecoxib. The present report highlights a rare clinical presentation and may contribute to improved recognition and management of postoperative PMR, thereby reducing the risk of misdiagnosis or delayed diagnosis.

Case report

A 73-year-old man with a medical history of hypertension, type 2 diabetes mellitus and coronary artery disease underwent coronary stent implantation in August 2016. In March 2024, the patient was diagnosed with left upper lobe lung cancer (stage I; T1N0M0) according to the 8th edition of the TNM Classification of Lung Cancer (14) and underwent surgical resection at Shanghai Pulmonary Hospital (Shanghai, China). At 1 week after surgery, the patient developed fever, chills, generalized myalgia, fatigue and a mild cough with scant sputum.

Upon hospital admission, laboratory investigations revealed notable leukocytosis (white blood cell count, $16.79 \times 10^9/l$; reference range: $3.50\text{--}9.50 \times 10^9/l$), neutrophilia (86.7%; reference range: 40.0–75.0%) and monocytosis ($1.07 \times 10^9/l$; reference range: $0.10\text{--}0.60 \times 10^9/l$). Biochemical analyses indicated hypoalbuminemia (31.6 g/l; reference range: 40.0–55.0 g/l) and hyperglycemia (15.27 mmol/l; reference range: 3.89–6.11 mmol/l), alongside markedly elevated inflammatory markers, specifically C-reactive protein (CRP; 156.00 mg/l; reference range: <10.00 mg/l) and interleukin-6 (IL-6; 75.80 pg/ml; reference range: 0.00–7.00 pg/ml). A chest computed tomography (CT) scan demonstrated expected post-operative changes in the left lung, along with small bilateral pulmonary nodules, mild interstitial alterations and minimal pleural effusion (Fig. 1A). Initial microbiological cultures and comprehensive respiratory pathogen testing yielded negative results, effectively ruling out common infectious etiologies.

Due to the clinical presentation and elevated inflammatory markers, an infectious etiology was initially suspected. The patient received broad-spectrum antibiotic therapy with piperacillin/tazobactam (4.5 g every 12 h), followed by meropenem (1 g every 8 h) and linezolid (0.6 g every 12 h) due to persistent symptoms (Fig. 2). Despite antimicrobial therapy, the patient continued to experience intermittent afternoon fevers ranging from 37.1 to 38.4°C, and inflammatory markers remained persistently elevated (Fig. 2; Table I). Further investigations, including next-generation sequencing for atypical pathogens, failed to identify any causative infectious agents. Meanwhile, follow-up chest CT revealed no appreciable differences relative to the imaging performed at admission (Fig. 1B).

At ~4 weeks post-surgery, the patient experienced a persistent fever and severe myalgia and joint pain involving the shoulders, hips and knees, despite broad-spectrum antibiotic therapy and extensive negative infectious evaluations. However, no clear infectious focus was identified. Given that the musculoskeletal symptoms worsened with the fever, the patient was diagnosed with PMR according to the ACR-EULAR 2012 classification criteria (13). In the absence of ultrasound evaluation, the scoring

system ranges from 0 to 6 points; a score of ≥ 4 points is required for classification as PMR. The criteria include morning stiffness lasting ≥ 45 min (2 points), hip pain and/or limited range of motion (1 point), negative rheumatoid factor and/or anti-cyclic citrullinated peptide antibody results (2 points), and the absence of other joint involvement (1 point). In the present patient, morning stiffness lasting ≥ 45 min (2 point), hip pain with limited range of motion (1 point), and negative rheumatoid factor and anti-cyclic citrullinated peptide antibody results were observed (2 points), while no other joint involvement was identified. Accordingly, the patient obtained a total score of 5 points, meeting the classification criteria for PMR. Musculoskeletal ultrasound was not performed because the patient declined further imaging examinations. Although PMR is associated with an increased risk of GCA, the patient exhibited no clinical manifestations suggestive of concomitant GCA, such as a new-onset headache, visual disturbances or jaw claudication (15).

The patient was treated with non-steroidal anti-inflammatory therapy (200 mg celecoxib daily) for ~3 months, which led to rapid and marked improvement. The fever resolved and the muscle and joint pain gradually subsided. During a 6-month follow-up, the patient was evaluated monthly. The myalgia was noted to be nearly completely resolved, CRP and IL-6 levels had returned to normal, and quality of life was notably improved. No recurrence of PMR-related symptoms was observed during this period.

Discussion

The present study describes the case of a 73-year-old man with lung cancer who developed PMR 4 weeks after surgical resection, without prior exposure to ICIs. This report provides important insights into PMR pathogenesis in the tumor environment, beyond the well-documented ICI-related cases (16–20). By analyzing the unique triggering factors, potential mechanisms and therapeutic implications, the present report enhances the understanding of PMR as a complex interplay among malignant tumors, surgical stress and systemic inflammation, ultimately guiding more informed clinical decision-making.

PMR is generally considered an idiopathic inflammatory disease, with pathogenesis primarily linked to genetic susceptibility and immune dysregulation (21). Several studies have reported associations between PMR and viral infections (such as parvovirus B19 and parainfluenza virus) and bacterial infections (*Chlamydia* and *Mycoplasma*) (22,23). Additionally, PMR has been observed following vaccination, suggesting a potential link between immunization and disease onset (24,25). Recently, several cases of PMR following COVID-19 vaccination have been reported, with onset typically occurring within days to weeks after vaccination (26,27). In the present patient, although a COVID-19 vaccination was administered in 2021, no PMR-related symptoms occurred after the vaccination, and the interval between vaccination and PMR onset was ~3 years. Therefore, COVID-19 vaccination was considered unlikely to be the immediate trigger of the PMR in the present case.

Most recently, PMR has increasingly been recognized as an immune-related adverse event (irAE) in patients receiving ICIs, such as anti-PD-1/PD-L1 therapies (28). Pharmacovigilance data indicate that the risk of developing PMR in patients undergoing

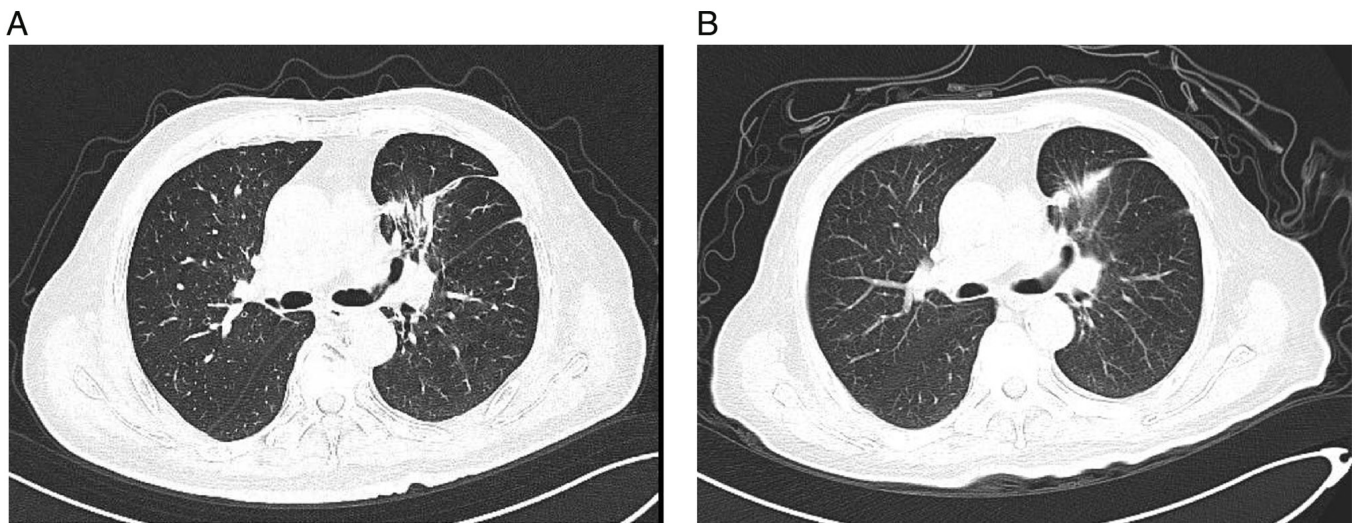


Figure 1. Chest computed tomography images of the patient upon admission and during follow-up. (A) Imaging on admission (March 2024). (B) Imaging at follow-up (April 2024).

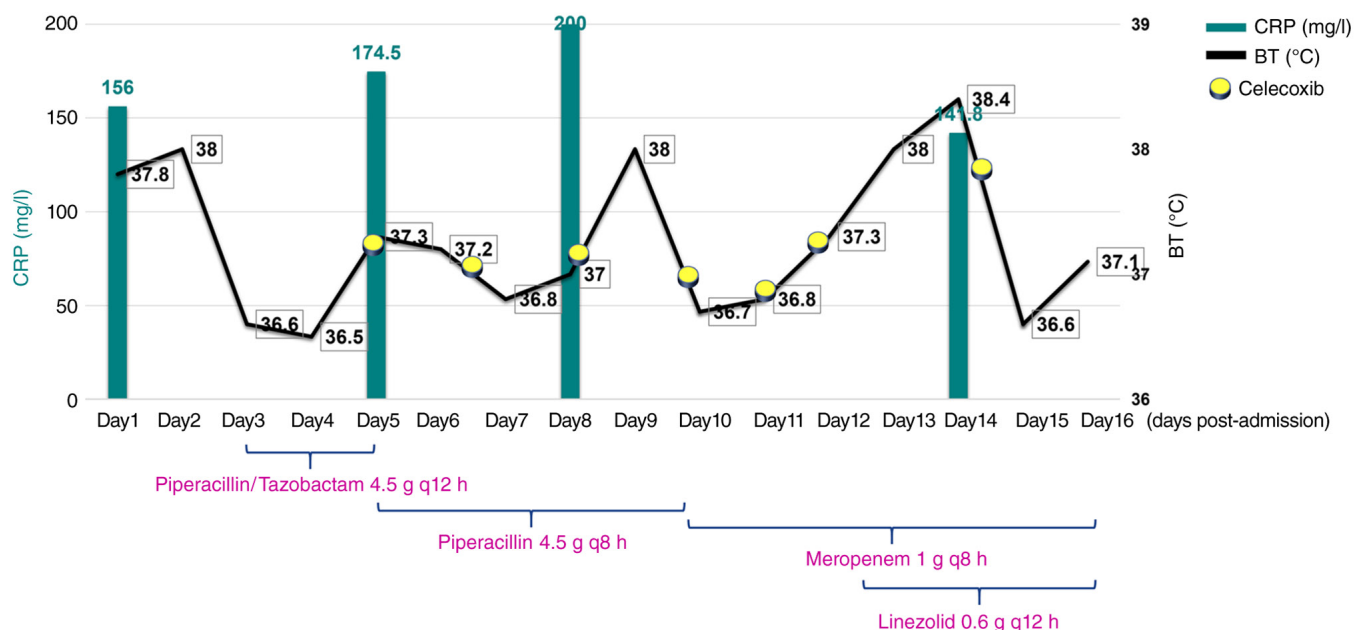


Figure 2. Dynamic changes in body temperature and CRP levels during hospitalization. Yellow circles indicate the initiation of celecoxib treatment (200 mg/day). Antibiotic treatment regimens are shown below the timeline. CRP, C-reactive protein; BT, body temperature.

ICI therapy is approximately five times higher than that in patients with cancer not receiving similar immunotherapy (29). Several case reports highlight ICI-associated PMR. For example, a patient with advanced non-small cell lung cancer, without pre-existing diabetes mellitus or autoimmune disease, received nivolumab as fifth-line therapy after disease progression following chemotherapy. After 3 years and 3 months of nivolumab treatment, the patient developed polydipsia, thirst and elevated blood glucose levels and was diagnosed with diabetes mellitus. Nivolumab treatment was continued; at 4 years and 5 months after initiation of therapy, the patient developed bilateral shoulder and knee pain, elevated C-reactive protein levels, normal creatine phosphokinase levels, and negative autoimmune antibody tests, leading to a diagnosis of PMR (11). Another case

involved a woman with lung adenocarcinoma who developed PMR as an irAE after nivolumab therapy (30). ICI-related PMR typically presents acutely soon after therapy initiation and responds well to glucocorticoids, reflecting the central role of adaptive immune dysregulation (31).

However, PMR is not exclusively observed as an irAE associated with ICI therapy. Previous reports have demonstrated that other anticancer treatments may also trigger PMR or PMR-like syndromes in patients with malignancies who have not received ICI therapy. For example, PMR accompanied by remitting seronegative symmetrical synovitis with pitting edema syndrome was reported in a patient with bladder cancer following transurethral tumor resection and subsequent intravesical *Bacillus Calmette-Guérin* instillation therapy (32).

Table I. Changes in inflammatory markers during hospitalization.

Variable	Day 1 post-admission	Day 5 post-admission	Day 8 post-admission	Day 14 post-admission	Reference range
WBC count, $\times 10^9/l$	16.79	14.18	15.83	14.11	3.50-9.50
Neutrophils, %	86.70	90.50	86.30	86.80	40.00-75.00
CRP, mg/l	156.00	174.50	>200.00	141.80	<10.00
IL-6, pg/ml	75.80	157.41	306.42	N/A	0.00~7.00
PCT, ng/ml	0.761	N/A	N/A	N/A	0.000~0.500
ESR, mm/h	N/A	N/A	N/A	84.18	0.00~20.00
Ferritin, ng/ml	N/A	N/A	2,095.47	N/A	23.90-336.20

WBC, white blood cell; CRP, C-reactive protein; IL-6, interleukin-6; PCT, procalcitonin; ESR, erythrocyte sedimentation rate; N/A, not available or not measured.

Table II. Differences between ICI-related and postoperative PMR.

Dimension	ICI-associated PMR	Lung cancer post-resection PMR (present case)
Immune dysregulation origin	Adaptive immunity	Innate immunity
Temporal association	Acute (weeks after ICI initiation)	Subacute (weeks after surgery)
Temporal association	Often concurrent autoimmunity (rash and thyroiditis)	Isolated PMR (no other autoimmune features)
Treatment sensitivity	Glucocorticoids	COX-2 inhibitors

ICI, immune checkpoint inhibitor; PMR, polymyalgia rheumatica; COX-2, cyclooxygenase-2.

In addition, cases of PMR have been reported during treatment with multikinase inhibitors (such as sorafenib and erlotinib) (33) and the PI3K δ inhibitor idelalisib (34). These observations suggest that, beyond ICI therapy, anticancer treatments may potentially induce PMR through mechanisms involving immune dysregulation.

Compared with drug-associated PMR, the development of PMR following major surgery is rarer. To date, a limited number of cases have been reported describing PMR onset after major surgical procedures, including cases following robot-assisted radical prostatectomy and cervical spine surgery (35-37). These patients developed typical shoulder and/or pelvic girdle pain accompanied by elevated inflammatory markers within several weeks of surgery and demonstrated favorable responses to glucocorticoid therapy. In the post-prostatectomy case, it was proposed that surgery-induced inflammatory responses may represent an important trigger for PMR development. It was further hypothesized that perioperative elevations in inflammatory cytokines, particularly IL-6, may contribute to the underlying pathogenesis (38). Furthermore, a case involving a patient with long-standing PMR who underwent ascending aortic aneurysm repair and aortic valve replacement revealed histopathological evidence of GCA after surgery. This finding further suggests a potential association between PMR-related large-vessel inflammation and major thoracic surgical stress. It also highlights the importance of careful perioperative assessment of inflammatory activity and vascular involvement in patients with PMR undergoing major surgical procedures (39).

In the present case, the patient neither received ICI therapy nor was exposed to other anticancer agents previously associated with PMR development. Instead, PMR developed 4 weeks after curative surgical resection for lung cancer. Together with the limited number of previously reported postoperative PMR cases, the present case further supports the hypothesis that major surgical procedures themselves may act as an independent immunological trigger for PMR development through mechanisms involving tissue injury, inflammatory cytokine release and postoperative immune remodeling. Therefore, distinguishing ICI-associated PMR from postoperative PMR is of clinical importance, and the key differences between these two conditions are summarized in Table II.

Further analysis of the potential mechanisms underlying postoperative PMR in the present patient suggests a 'predisposition and second-hit' model. The patient's advanced age and lung cancer-associated chronic inflammatory state may have created an immune-susceptible background. Previous studies have shown that lung cancer cells can produce pro-inflammatory cytokines, such as IL-6, contributing to a systemic inflammatory environment and potentially lowering the threshold for PMR development (40,41). In this context, curative lung cancer surgery may have served as a secondary trigger by inducing tissue injury, releasing damage-associated molecular patterns and promoting inflammatory cytokine production, including IL-6, IL-8 and VEGF (42). In the present patient, the postoperative IL-6 level peaked at 306.42 pg/ml. IL-6 is considered a key mediator in the pathogenesis of

PMR, as it contributes to systemic inflammation and promotes inflammatory responses in the synovial tissues, as well as the shoulder and pelvic girdle regions (43). Therefore, the postoperative inflammatory response may have acted as a 'second hit' in an immune system already primed by tumor-associated inflammation, ultimately triggering PMR. Moreover, potential inflammatory crosstalk may exist between PMR and malignancy. PMR-related inflammatory mediators, including IL-6, may influence tumor progression and immune regulation, while persistent tumor-associated inflammation may further sustain PMR-related inflammatory responses. Although these mechanisms require further validation, the present case highlights the potential contribution of tumor-associated inflammation, surgical stress and age-related immune alterations to postoperative PMR development.

The strong response of patients to celecoxib provides mechanisms and clinical insights. COX-2 mediates prostaglandin E2 (PGE2) synthesis (44), a key mediator of pain and inflammation in musculoskeletal tissues affected by PMR. By inhibiting COX-2, celecoxib directly targets PGE2-driven symptoms, explaining the rapid resolution of the fever and myalgia in the present patient. This contrasts with ICI-related PMR, which typically requires glucocorticoids to suppress adaptive immune overactivation (31,45). Although the present case supports celecoxib as an effective therapeutic option, larger studies are needed to validate its safety and efficacy in post-cancer resection PMR, particularly regarding long-term tumor outcomes. Additionally, the present case has several limitations. The patient declined musculoskeletal ultrasound examination, resulting in a lack of imaging-based supportive evidence, which may have limited further confirmation of the PMR diagnosis. In the future, with appropriate patient consent and clinical consideration, musculoskeletal ultrasound should be incorporated more systematically and comprehensively into the diagnostic evaluation of PMR to improve diagnostic accuracy and enhance disease characterization.

In summary, the present case extends beyond a mere clinical observation, highlighting the potential for PMR to arise from the interplay between tumor-associated inflammation and surgical trauma, independent of immunotherapy. Recognition of this pathogenic pathway may enhance diagnostic accuracy and therapeutic precision for clinicians, while providing researchers with a conceptual framework to study PMR as a dysregulated aseptic inflammatory disorder. The present report underscores that PMR should not be considered in isolation, but rather as part of a spectrum of age-related inflammatory processes influenced by malignancy and its treatment.

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Availability of data and materials

The data generated in the present study may be requested from the corresponding author.

Authors' contributions

YW and YZ performed imaging, analyzed data, wrote the manuscript and literature review. MW and PZ assisted with patient follow-up, laboratory data interpretation and manuscript revision. XJ conceived the study, provided clinical oversight for patient management and supervised the manuscript preparation. YW and XJ confirm the authenticity of all the raw data. All authors have read and approved the final version of the manuscript.

Ethics approval and consent to participate

This study was approved by the Ethics Committee of Jiangsu Province (Suqian) Hospital (Suqian, China; approval no. 2026-SR-0009). The requirement for informed consent was waived by the ethics committee.

Patient consent for publication

Written informed consent was obtained from the patient for publication of the present case report and the accompanying clinical images. All potentially identifiable information was removed, and the relevant data were anonymized to ensure the protection of patient privacy.

Competing interests

The authors declare that they have no competing interests.

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