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Right Middle Lobe Syndrome

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Abstract

Background: Right middle lobe syndrome (RMLS) is a clinical and radiological entity defined by recurrent or persistent atelectasis, inflammation, or infection of the right middle lobe. It results from the anatomically narrow and acutely angled middle lobe bronchus, which predisposes to impaired mucociliary clearance and mucus retention. Occupational exposure to metal fumes—common in boilermakers and welders—can further compromise pulmonary immune defences, increasing susceptibility to bacterial pneumonia. Underlying asthma adds another layer of vulnerability through chronic airway inflammation and epithelial dysfunction.

Case presentation: We report a 32-year-old male boilermaker with a 10-year history of mild persistent asthma (non-smoker, intermittent adherence to inhaled corticosteroids) who presented with lethargy, subjective fever, and a persistent dry cough for 7 days. He had worked in metal fabrication for 15 years with inconsistent use of respiratory protective equipment. Chest examination revealed crackles in the right mid-zone. Laboratory tests showed leukocytosis (WBC 10.6 ×10⁹/L) and elevated C-reactive protein (66 mg/L). Chest radiography and subsequent computed tomography demonstrated dense consolidation of the right middle lobe with reactive hilar and paratracheal lymphadenopathy. A respiratory viral panel was negative. He was admitted with a diagnosis of community-acquired pneumonia and treated with intravenous ceftriaxone (1 g bd) and oral doxycycline (100 mg bd) for 7 days.

Outcome: Clinical improvement (afebrile, reduced cough, normal appetite) occurred by day 5. However, repeat chest X-ray on day 5 showed persistent consolidation. The patient was discharged on oral cefuroxime 250 mg bd for an additional 5 days (total 12 days of antibiotics). Follow-up imaging was scheduled at 6–8 weeks, with bronchoscopy planned if radiological resolution was incomplete—to exclude an endobronchial lesion or foreign body.

Conclusion: This case illustrates the synergistic effect of anatomical predisposition (RMLS), occupational inhalations exposure (metal fumes), and underlying airway disease (asthma) in the development of pneumonia. It highlights the importance of occupational history-taking and the recognition that radiographic resolution in RMLS often lags clinical recovery.

Keywords: Right middle lobe syndrome; Community-acquired pneumonia; Occupational exposure; Metal fumes; Asthma; Boilermaker; Case report

Introduction

Community-acquired pneumonia (CAP) remains a leading cause of hospitalisation and mortality worldwide. In 2019, lower respiratory tract infections accounted for approximately 2.6 million deaths, with CAP contributing substantially to this burden [1]. Although microbial pathogens—most commonly Streptococcus pneumoniae, Haemophilus influenzae, and atypical organisms—directly initiate infection, host susceptibility factors and environmental exposures play pivotal roles in disease development and severity. Among the less commonly recognised host factors is right middle lobe syndrome (RMLS). First described by Graham et al. in 1948 [2], RMLS is not a single disease but a constellation of findings characterized by recurrent or persistent atelectasis, infection, or inflammation limited to the right middle lobe. The right middle lobe bronchus arises from the bronchus intermedius at an acute angle (30–45°) and has a relatively narrow lumen compared to other lobar bronchi. It is surrounded by four lymph node stations (pulmonary, hilar,

subcarinal, para-oesophageal), making it vulnerable to extrinsic compression. These anatomical features create a "choke point" that impairs mucociliary clearance, promotes mucus stasis, and creates a favourable environment for bacterial overgrowth [3,4]. RMLS can be classified as obstructive (intraluminal causes such as mucus plugs, foreign bodies, endobronchial tumours, or broncholithiasis; extraluminal causes such as enlarged lymph nodes from granulomatous disease or malignancy) or non-obstructive (associated with impaired mucociliary clearance as in asthma, primary ciliary dyskinesia, or bronchiectasis). In adults, non-obstructive RMLS is often linked to underlying airway diseases or recurrent aspiration [5]. Occupational inhalational exposure is increasingly recognised as an independent risk factor for respiratory infections. Workers in welding, metal fabrication, and boiler construction are routinely exposed to airborne metal fumes containing iron oxides, manganese, chromium, nickel, silica, and other industrial aerosols generated during high-temperature metal processing. These particles have been shown to impair alveolar macrophage function, induce oxidative stress, and suppress pulmonary immune defences [6,7]. Epidemiological studies have demonstrated increased mortality from infectious pneumonia and a higher incidence of invasive pneumococcal disease among welders and metalworkers [8,9]. Asthma, present in up to 10% of the adult population, further augments vulnerability to respiratory infections. Chronic airway inflammation, epithelial barrier disruption, impaired mucociliary clearance, and altered airway microbiome all predispose asthmatic individuals to bacterial superinfection [10]. When asthma coexists with RMLS and occupational metal fume exposure, the risk of clinically significant pneumonia may be magnified. We report a case of right middle lobe pneumonia in a boilermaker with long-term metal fume exposure and underlying asthma, highlighting the convergence of these three predisposing factors and the importance of follow-up imaging to confirm radiological resolution.

Case Presentation

A 32-year-old male presented to the emergency department with a 7-day history of lethargy, reduced appetite, subjective fevers (not measured at home), and a persistent, predominantly dry cough. He also reported mild pleuritic chest discomfort localised to the right anterior chest. There was no haemoptysis, night sweats, or weight loss. He denied recent travel, sick contacts, or exposure to tuberculosis.

Medical history

The patient had a history of asthma, diagnosed at age 18. He described intermittent wheezing and dyspnoea, typically triggered by upper respiratory tract infections and cold air. His maintenance therapy was inhaled salbutamol as needed, with low-dose inhaled beclomethasone prescribed but used inconsistently (approximately 2–3 times per week). He had no prior hospitalisations for asthma and no history of pneumonia.

Social history

He was a non-smoker (lifetime non-smoker, confirmed by urinary cotinine). He reported occasional alcohol consumption (2–3 standard drinks per week) and no illicit drug use. He lived with his partner in a suburban home with no known indoor mould or dampness.

Occupational history

The patient had worked as a boilermaker for 15 years (since age 17). His daily tasks included welding (shielded metal arc welding and gas metal arc welding), cutting, grinding, and assembling metal structures. He estimated being exposed to welding fumes for 6–8 hours per shift, 5 days per week, for most of his career. Respiratory protective equipment (P2 or N95 masks) was used inconsistently—approximately 30% of the time—and he had never undergone respirator fit-testing. He had no prior formal respiratory health surveillance.

Clinical examination

On presentation, vital signs were: temperature 37.8°C (subjective fever only), blood pressure 128/76 mmHg, heart rate 92 bpm, respiratory rate 18 breaths/min, oxygen saturation 96% on room air. He appeared tired but non-toxic. No acute respiratory distress was present. Chest auscultation revealed localised crackles in the right mid-zone (anterior and lateral chest), with no wheeze or bronchial breathing. The rest of the examination was unremarkable.

Laboratory investigations

Initial blood tests (day 1) are summarised in (Table 1). Notable findings included leukocytosis (WBC 10.6 $\times 10^9$/L) with neutrophilia (6.13 $\times 10^9$/L) and a markedly elevated C-reactive protein (66 mg/L). Eosinophil count was mildly elevated at 0.87 $\times 10^9$/L (reference <0.6). Renal and liver function tests were normal. A respiratory viral multiplex PCR panel (including SARS-CoV-2, influenza A/B, RSV, adenovirus, human metapneumovirus, rhinovirus) was negative. Sputum culture (spontaneous, good quality) grew normal respiratory flora; no S. pneumoniae, H. influenzae, or Legionella was isolated. Two sets of blood cultures were sterile. Urinary antigen tests for Legionella pneumophila and Streptococcus pneumoniae were negative.

Differential diagnosis

The differential diagnosis for persistent right middle lobe consolidation with reactive lymphadenopathy is broad (Table 2).

Based on the acute presentation with fever and elevated inflammatory markers, community-acquired pneumonia was the most likely diagnosis. However, the slow radiological resolution and occupational history raised consideration of alternative or contributing factors.

Differential diagnosis of persistent right middle lobe consolidation

Category Specific entities How excluded / assessed
Atypical pneumonia (Mycoplasma, Chlamydia) Doxycycline covered; serologies negative if done*
Tuberculosis / non-tuberculous mycobacteria No cavitation; negative IGRA (performed)
Fungal pneumonia (histoplasmosis, coccidioidomycosis) Not endemic in Queensland; serologies not done
Neoplastic Endobronchial tumour (carcinoid, squamous) CT did not show intraluminal mass; bronchoscopy if unresolved
Lymphoma / metastatic disease No systemic symptoms; lymph nodes small and reactive
Inflammatory Granulomatosis with polyangiitis (GPA) No upper respiratory or renal involvement; ANCA negative
Eosinophilic pneumonia Mild eosinophilia but no peripheral eosinophilia >10%
Anatomical Right middle lobe syndrome (non-obstructive) Supported by anatomy, asthma, delayed resolution
Occupational Metal fume fever / acute metal toxicity No systemic syndrome; presentation more consistent with infection
*Mycoplasma pneumoniae IgM was negative on day 4.

Management

The patient was admitted to the general medical ward. Given the absence of severe respiratory compromise (CURB-65 score 0), empiric antibiotic therapy for CAP was initiated:

- Ceftriaxone 1 g intravenously twice daily (providing coverage for S. pneumoniae, H. influenzae, and MSSA)
- Doxycycline 100 mg orally twice daily (for atypical pathogens)

The planned duration was 7 days. In addition, the patient received regular inhaled salbutamol as needed and was advised to restart his inhaled beclomethasone (two puffs twice daily) for asthma control. Chest physiotherapy (postural drainage and percussion over the right middle lobe) was initiated on day 2.

Outcome and follow-up

By day 5 of treatment, the patient was afebrile, his cough had reduced significantly, and his appetite returned. Vital signs remained stable. Repeat laboratory testing on day 5 showed a decline in CRP to 8 mg/L and normalisation of WCC (9.8 ×10⁹/L) – see Table 1.

However, a repeat chest X-ray on day 5 showed Table 1 persistent right middle lobe consolidation with minimal interval change. This discordance between clinical improvement and radiological persistence is well recognised in RMLS [11]. The patient completed 7 days of intravenous and oral antibiotics. He was discharged on oral cefuroxime 250 mg twice daily for an additional 5 days (total 12 days of therapy) as a step-down regimen. He was also prescribed regular inhaled beclomethasone and provided with a written asthma action plan (Figures 1-2).

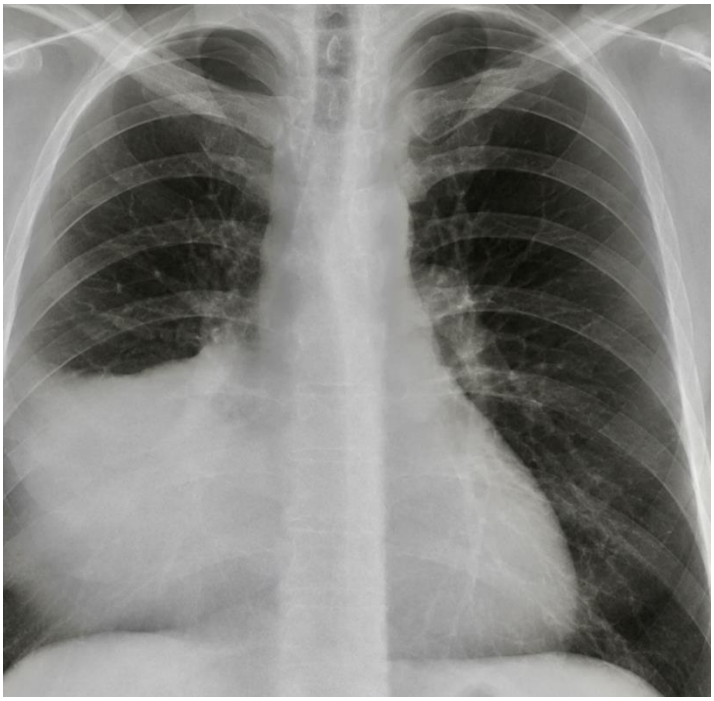


Figure 1: *Chest X-ray (PA view) showing right middle lobe consolidationloss of right heart border silhouette.*

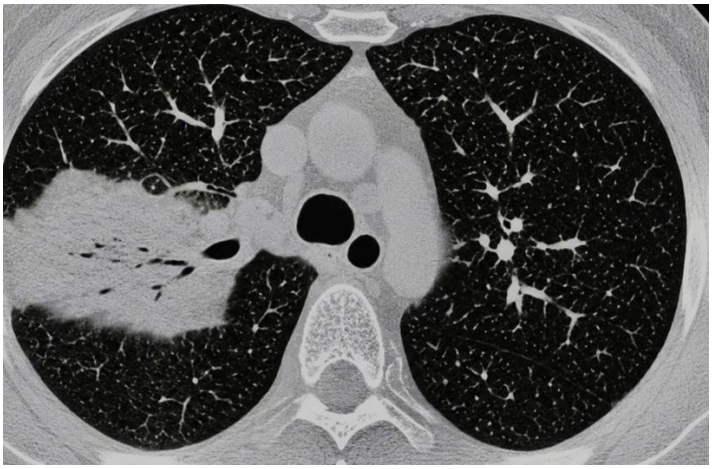


Figure 2*: CT chest (axial view) showing right middle lobe consolidation and reactive lymphadenopathy.*

Follow-up plan

- Outpatient respiratory review at 4 weeks
- Repeat chest X-ray and high-resolution CT chest at 6–8 weeks
- Bronchoscopy to be performed if complete radiological resolution does not occur by 8 weeks, or earlier if symptoms recur. Bronchoscopy would evaluate for an endobronchial lesion (e.g., mucus plug, foreign body, carcinoid tumour) or bronchial stenosis.

Time from treatment start Clinical findings Radiological findings (CXR/CT)

Day 0–2 Fever, cough, dyspnoea, malaise Consolidation clearly visible

Day 3–5 Reduced cough, improved oxygenation Usually no significant change

Day 5–7 Marked symptomatic improvement Consolidation still present

Week 2 Most patients clinically recovered Partial clearing of consolidation

Week 4 Asymptomatic Significant radiological improvement

Week 6–8 Normal clinical state Complete or near-complete resolution

***Table 1**: Laboratory findings at presentation and day 5.*

Parameter	Day 1	Day 5	Reference Range
Hemoglobin (g/L)	127	135	135–175
White cell count (×10⁹/L)	10.6	9.8	3.5–10.0
Neutrophils (×10⁹/L)	6.13	4.66	1.5–6.5
Eosinophils (×10⁹/L)	0.87	0.92	0–0.60
Platelets (×10⁹/L)	347	337	150–400
C-reactive protein (mg/L)	66	8	<5
Sodium (mmol/L)	139	139	135–145
Potassium (mmol/L)	4.3	4.4	3.5–5.5
Creatinine (µmol/L)	80	74	60–110
eGFR (mL/min/1.73 m²)	>90	>90	>59
Albumin (g/L)	38	38	23–43

Discussion

We present a case of right middle lobe pneumonia in a young boilermaker with three concurrent predisposing factors: (1) anatomical vulnerability (right middle lobe syndrome), (2) occupational exposure to metal fumes, and (3) underlying asthma. This combination likely explains the acute infection and the delayed radiographic resolution despite appropriate antibiotic therapy.

Right middle lobe syndrome – pathophysiology and clinical relevance

The right middle lobe bronchus has several unique features that render it prone to disease. Its length (approximately 1–2 cm) and acute angle of origin (30–45° from the bronchus intermedius) create a "trap" for mucus and debris. Furthermore, the bronchus is surrounded by a collar of four lymph node groups (pulmonary, hilar, subcarinal, para-oesophageal) that can compress it when enlarged [3,4]. The lumen diameter is also smaller than that of other lobar bronchi, so even mild mucosal oedema or mucus hypersecretion can cause critical narrowing. In non-obstructive RMLS, which is most likely in our patient (CT showed no visible endobronchial lesion or extrinsic compression), impaired mucociliary clearance is the dominant mechanism. Asthma is a classic cause of non-obstructive RMLS because of goblet cell hyperplasia, mucus hypersecretion, and ciliary dysfunction [10,13]. Our patient's inconsistent use of inhaled corticosteroids likely contributed to ongoing airway inflammation and mucus stasis.

Occupational metal fume exposure and pulmonary immunity

Welders and boilermakers inhale a complex mixture of metal oxides (iron, manganese, chromium, nickel, zinc) and gases (ozone, nitrogen oxides). These agents have been shown to suppress alveolar macrophage function, reduce bacterial phagocytosis, and impair neutrophil recruitment in animal models and human exposure studies [6,7]. A seminal epidemiological study by Toren [8] followed over 20,000 Swedish metal workers and found a significantly increased mortality from infectious pneumonia (standardised mortality ratio 1.9, 95% CI 1.3–2.7) among those exposed to inorganic dust and metal fumes. Similarly, Wong [9] reported a 2.5-fold increased risk of invasive pneumococcal disease in welders compared to the general population. Our patient had 15 years of heavy, unprotected exposure, placing him at high risk. His inconsistent use of

respiratory protection (only 30% of the time) further compounded this risk. We hypothesise that chronic metal fume inhalation induced a state of impaired pulmonary immune surveillance, allowing bacterial colonisation and rapid progression to pneumonia once a viral trigger or microaspiration occurred.

Interaction between asthma, RMLS, and metal fumes

These three factors likely acted synergistically. Asthma caused mucus hypersecretion and reduced ciliary clearance → mucus accumulated in the anatomically vulnerable middle lobe bronchus → metal fume-induced macrophage dysfunction allowed bacterial overgrowth → clinical pneumonia developed. The delayed radiographic resolution is explained by the persistence of mucus plugging and local inflammation even after bacterial killing—a phenomenon typical of RMLS [11]. In a study of 68 patients with RMLS, complete radiological resolution took a median of 8 weeks, and 15% had persistent atelectasis at 6 months [12].

Clinical implications and follow-up

This case underscores three key practice points:

- Take an occupational history in every patient with pneumonia, especially if recurrent or slow to resolve. A simple question— "What work do you do?"—can reveal significant risk factors.
- Do not equate clinical improvement with cure in RMLS. Persistent radiographic abnormalities are common and require follow-up imaging. Unnecessary repeated antibiotic courses can be avoided by explaining this natural history to the patient.
- Bronchoscopy has a defined role when radiological resolution is incomplete after 8 weeks. In adults, up to 20–30% of RMLS cases have an underlying endobronchial lesion, including carcinoid tumours, broncholiths, or foreign bodies [4,13]. Our patient will undergo bronchoscopy if his 8-week CT still shows consolidation.

Limitations

This is a single case report, so causal inference is limited. We did not perform baseline pulmonary function testing or bronchoscopy at the acute stage. Sputum culture did not identify a specific pathogen, which is common in CAP (up to 60% of cases have no microbiological diagnosis). We also could not measure occupational exposure levels retrospectively. Nevertheless, the combination of typical risk factors and clinical course is highly suggestive.

Learning Points / Take-Home Messages

- Right middle lobe syndrome (RMLS) should be suspected in patients with recurrent or persistent right middle lobe consolidation, especially those with asthma or other mucociliary disorders.
- Occupational exposure to metal fumes (boilermakers, welders, metal fabricators) increases the risk of bacterial pneumonia by impairing alveolar macrophage function. Always ask about work history.
- Radiographic resolution in RMLS often takes 6–8 weeks or longer, even after clinical recovery. Follow-up imaging is essential to confirm complete resolution and exclude an underlying endobronchial lesion.
- Bronchoscopy is indicated if consolidation persists beyond 8 weeks, to rule out a foreign body, mucus plug, or occult neoplasm.
- Management of acute pneumonia in patients with RMLS should include standard CAP antibiotics, chest physiotherapy directed to the right middle lobe, and optimisation of underlying asthma (inhaled corticosteroids).

Conclusion

This case demonstrates the convergence of anatomical predisposition (right middle lobe syndrome), environmental risk (occupational metal fume exposure), and host susceptibility (asthma) in the development of community-acquired pneumonia. Recognition of right middle lobe syndrome and its associated risk factors can improve diagnostic accuracy, guide appropriate follow-up, and prevent recurrent infections in vulnerable occupational groups. Clinicians should maintain a low threshold for occupational history-taking and follow-up imaging in similar patients.

References

1. GBD 2019 Diseases and Injuries Collaborators. Global burden of 369 diseases and injuries in 204 countries and territories, 1990–2019: a systematic analysis for the Global Burden of Disease Study 2019. Lancet. 2020; 396: 1204-1222.
2. Graham EA, Burford TH, Mayer JH. Middle lobe syndrome. Postgraduate Medicine. 1948; 4: 29-34.
3. Kwon KY, Myers JL, Swensen SJ, Colby TV. Middle lobe syndrome: a clinicopathological study of 21 patients. Human Pathology. 1995; 26: 302-307.
4. Gudbjartsson T, Gudmundsson G. Middle lobe syndrome: a review of the literature. Lung. 2012; 190: 359-363.
5. Einarsson JT, Einarsson JG, Isaksson H, Gudbjartsson T. Middle lobe syndrome: a nationwide study on clinicopathological features and surgical treatment. European J Cardio-Thoracic Surgery. 2021; 59: 1051-1058.
6. Antonini JM. Health effects of welding. Critical Reviews Toxicology. 2003; 33: 61-103.
7. Zeidler-Erdely PC, Erdely A, Antonini JM. Immunotoxicology of arc welding fume: influence of metal composition. J Immunotoxicol. 2012; 9: 225-236.

8. Toren K, Qvarfordt I, Bergdahl IA, Järvholm B. Increased mortality from infectious pneumonia after occupational exposure to inorganic dust and metal fumes. Thorax. 2011; 66: 992-996.
9. Wong A, Marrie TJ, Garg S, Kellner JD, Tyrrell GJ. Welders are at increased risk for invasive pneumococcal disease. Int J Infectious Diseases. 2010;14: e796-e799.
10. Jackson DJ, Johnston SL. The role of viruses in acute exacerbations of asthma. J Allergy Clinical Immunology. 2011; 128: 1160-1167.
11. Bruns AH, Oosterheert JJ, Hak E, Hoepelman AI. Pneumonia recovery: discrepancies in perspectives of the radiologist, physician and patient. J General Internal Medicine. 2010; 25: 203-206.
12. Metlay JP, Waterer GW, Long A. Diagnosis and treatment of adults with community-acquired pneumonia. American J Respiratory Critical Care Medicine. 2019; 200: e45-e67.
13. Priftis KN, Mermiri D, Papadopoulou A. Middle lobe syndrome in children: a retrospective study. Pediatric Pulmonology. 2005; 40: 332-336.