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Viral Pneumonitis Masquerading as Suspected Pulmonary Malignancy in a Patient with Significant Family History of Cancer: A Diagnostic Dilemma

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Abstract

Background: Community-acquired pneumonia (CAP) is a leading cause of hospital admission worldwide, with a broad differential diagnosis that frequently challenges clinicians.[1] Viral pneumonitis caused by pathogens such as Influenza, Adenovirus, and other respiratory viruses can convincingly mimic the radiological and clinical features of pulmonary malignancy, particularly in patients with a strong family history of cancer.[2,3]

The radiological overlap between multifocal consolidation with ground-glass opacities (GGOs) and primary or metastatic lung cancer poses a significant diagnostic challenge.[4]

Case Presentation: A patient in the fifth decade of life presented with a 10-day history of intermittent fever and

4-day history of productive cough with scanty non-foul-smelling white sputum, along with progressive breathlessness. On admission, the patient was ill-appearing, with RR 22 cpm, PR 110 bpm, and BP 120/80 mmHg. CURB-65 score was 1, placing the patient in the low-to-moderate risk CAP category.[6] Chest X-ray revealed bilateral lower-zone air-space opacities; auscultation demonstrated bilateral crepitations with bronchial breath sounds. Inflammatory markers showed CRP 119 mg/L and ESR 33 mm/hr at admission. Despite empirical antibiotic therapy, the patient deteriorated — CRP decreased to 77 mg/L by day 4 while ESR paradoxically rose to 63 mm/hr, suggesting a non-bacterial aetiology.[9,10] HRCT thorax revealed multiple patchy consolidations with surrounding GGOs across all lung fields. Given a striking family history — mother (uterine cancer), brother (laryngeal cancer), father (GIT cancer) — pulmonary malignancy was considered in the differential diagnosis.[5]CECT thorax showed nodular consolidations without post-contrast enhancement, and serial comparison demonstrated a slight reduction in nodule number, making malignancy less likely and supporting a diagnosis of viral pneumonitis.[13] The patient was treated with Oseltamivir 75 mg twice daily for 5 days with marked clinical improvement.

Follow-up HRCT performed 15 days after initiation of antiviral therapy demonstrated near-complete radiological resolution, and the patient was discharged in stable condition.[16]

Conclusion: This case underscores the critical importance of integrating clinical trajectory, serial inflammatory markers, and dynamic radiological comparison before attributing pulmonary findings to malignancy. Viral pneumonitis should remain an important differential diagnosis even when family history raises strong alarm. Early antiviral therapy with Oseltamivir may be life-saving in atypical viral pneumonitis, and clinicians must consciously guard against anchoring bias in complex diagnostic scenarios.[24]

Keywords: Viral pneumonitis, community-acquired pneumonia, ground-glass opacities, pulmonary consolidation, Influenza, Adenovirus, Oseltamivir, lung cancer mimic, HRCT thorax, CURB-65

Introduction

Community-acquired pneumonia (CAP) remains a leading cause of morbidity and mortality worldwide, with an estimated global incidence of 450 million cases annually resulting in approximately 4 million deaths.[1] Viral aetiologies, including Influenza A and B, Adenovirus, Respiratory Syncytial Virus (RSV), and more recently SARS-CoV-2, account for an increasingly recognised proportion of CAP, particularly in immunocompetent hosts.[2] Despite this, viral pneumonitis continues to be underdiagnosed due to the non-specific nature of symptoms, variable radiological appearances, and historical overemphasis on bacterial pathogens in clinical practice.[3]

The radiological manifestation of viral pneumonitis multifocal consolidations, bilateral GGOs, and peripheral nodular infiltrates overlaps significantly with several serious pulmonary conditions, including pulmonary metastases, primary bronchogenic carcinoma, organising pneumonia, fungal infections, and septic emboli.[4] This diagnostic overlap becomes particularly challenging when compounded by an extensive family history of cancer, which appropriately heightens clinical suspicion for underlying malignancy.[5]

Severity scoring tools such as CURB-65 and the Pneumonia Severity Index (PSI) have been validated to guide site-of-care decisions and risk stratification in

CAP.[6,8] However, they do not assist in distinguishing infectious from non-infectious or malignant aetiologies of pulmonary infiltrates. Consequently, complex cases with atypical presentations require a methodical, multi-modal approach integrating clinical, biochemical, microbiological, and radiological data.[7]

This case report describes a patient admitted with presumed viral pneumonitis, in whom pulmonary malignancy was initially considered in the differential diagnosis due to a strong family cancer history and complex bilateral radiological findings. We discuss the diagnostic reasoning, the role of serial imaging, application of severity scoring, and the clinical and radiological features that ultimately guided the most likely diagnosis and successful management with Oseltamivir.

Case Report

Patient Presentation

A patient in the fifth decade of life presented to the medicine outpatient department with a 10-day history of intermittent, low-to-moderate-grade fever (without chills or rigors), a 4-day history of productive cough with scanty non-foul-smelling white sputum, and progressive breathlessness at rest. There was no haemoptysis, no chest pain, and no documented significant weight loss at presentation. The patient had a striking and significant family history of malignancy: mother with uterine (endometrial) cancer, brother with laryngeal cancer, and father with gastrointestinal (GIT) cancer; no records of genetic testing or treatment details were available.[5,20]

Clinical Examination and Severity Scoring

On admission, the patient was ill-appearing with mild respiratory distress. Vital signs are summarised in Table 1. CURB-65 severity score was calculated as 1, placing the patient in the low-to-moderate risk CAP category; however, clinical illness warranted inpatient admission per British Thoracic Society guidelines.[6] Pneumonia Severity Index (PSI/PORT) class was estimated as Class II–III based on available parameters, supporting inpatient monitoring with active management.[8]

Table 1: Vital signs and severity scores on admission.

Parameter	Value	Interpretation
Respiratory Rate	22 cpm	Tachypnoea
Pulse Rate	110 bpm	Tachycardia
Blood Pressure	120/80 mmHg	Normal
Temperature	Febrile (on and off)	Systemic illness
CURB-65 Score	1	Low-moderate risk
PSI Class (est.)	II–III	Inpatient monitoring

Chest auscultation revealed bilateral fine-to-medium crepitations (predominantly basal) with bilateral bronchial breath sounds, clinically consistent with bilateral consolidation. No wheeze, no pleural rub, and no lymphadenopathy were noted.

Investigations

- **Laboratory Investigations**

Routine blood investigations were within normal limits: complete blood count (CBC), renal function tests (RFT), serum creatinine, liver function tests (LFT), and urine

routine microscopy were all unremarkable. Inflammatory markers are summarised in Table 2.

Table 2: Serial laboratory results. CRP = C-reactive protein; ESR = erythrocyte sedimentation rate.

Investigation	Day 1 Result	Day 4 Result	Reference Range
CBC	Normal	—	—
RFT / Creatinine	Normal	—	0.6–1.2 mg/dL
LFT	Normal	—	—
Urine Microscopy	Normal	—	—
CRP (mg/L)	119	77	<5 mg/L
ESR (mm/hr)	33	63	<20 mm/hr

An important clinical observation from this case is the pattern of serial inflammatory markers. CRP fell from 119 to 77 mg/L (partial reduction — suggesting some response to treatment or natural course), while ESR paradoxically rose from 33 to 63 mm/hr despite antibiotic therapy. ESR is a slower-responding acute-phase reactant that peaks later than CRP and may rise even as CRP begins to fall in early convalescence.[9] A dissociation between falling CRP and rising ESR in the setting of clinical deterioration on antibiotics is a recognised biochemical pattern in viral and atypical pneumonias, pointing towards a non-bacterial aetiology.[10] Sputum culture, blood cultures, and respiratory virus panel (Influenza A/B rapid antigen test, Adenovirus PCR) were not documented in this case but are strongly recommended in similar presentations.[3]

- **Radiological Investigations**

Chest X-ray (CXR) at admission revealed bilateral lower-zone air-space opacities consistent with bilateral consolidation or atelectasis. Combined with bilateral crepitations and bronchial breath sounds, this was initially interpreted as community-acquired bacterial pneumonia necessitating empirical antibiotic therapy.[11]

HRCT thorax was performed following failure to respond to broad-spectrum antibiotics. Findings revealed multiple patchy consolidations bilaterally with surrounding GGOs in all lung fields, without pleural effusion or mediastinal lymphadenopathy (Figures 1, 3, 4). The differential diagnoses raised by the radiological report were: fungal pulmonary opacities (e.g. invasive pulmonary aspergillosis), viral pneumonitis (Influenza, Adenovirus), and septic emboli.[4,23] Given the family history of cancer, pulmonary metastases or primary lung malignancy was additionally considered by the clinical team.[5,12]

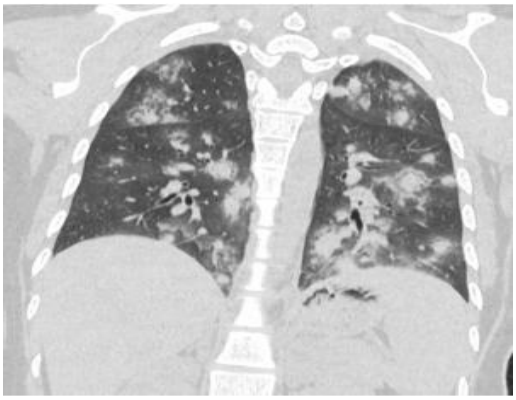

Figure 1: HRCT Thorax – Coronal view: bilateral multifocal consolidations with extensive surrounding

GGOs, prominently distributed across mid and lower lung zones.

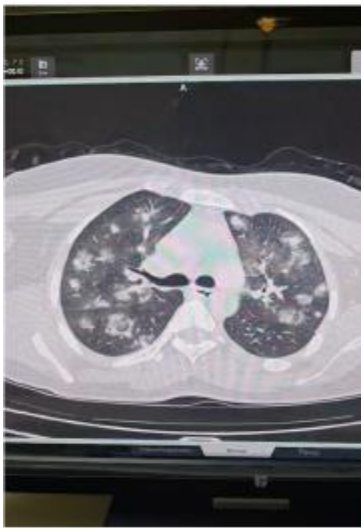

Figure 2: Axial HRCT Thorax: bilateral consolidation with peribronchial GGOs at the level of the carina.

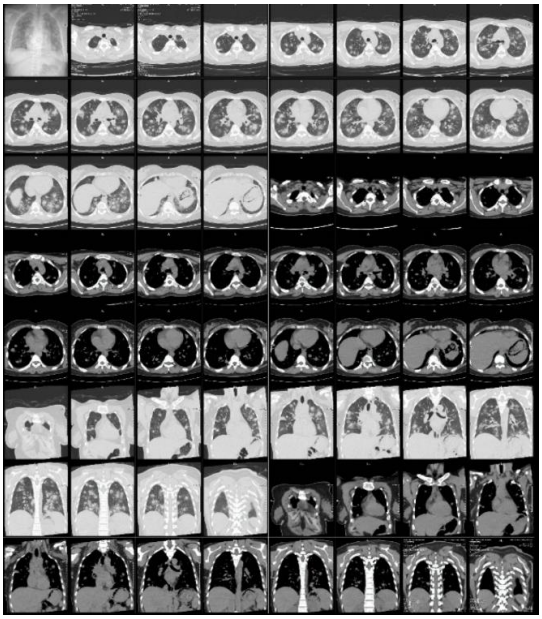

Figure 3: HRCT/CECT Thorax – Full radiological series montage including axial, coronal and lung-window reconstructions depicting the extent of bilateral pulmonary involvement with consolidative and ground-glass changes across all lobes.

CECT Thorax was subsequently performed to characterise the lesions and exclude malignancy. Findings showed multiple nodular consolidations with surrounding GGOs across all lung fields, with no abnormal post-contrast enhancement of the lesions (Figures 2, 5). Crucially, comparison with prior HRCT demonstrated a slight reduction in the number of nodules, indicating spontaneous partial resolution — a finding inconsistent with malignancy and highly consistent with an evolving infectious, self-limiting process.[13]

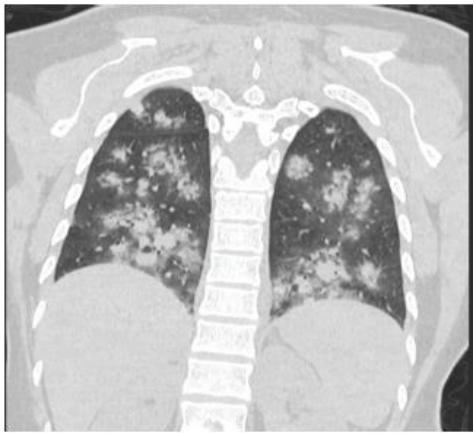

Figure 4: CECT Thorax – Coronal view: nodular consolidations with peripheral GGOs, no post-contrast enhancement, and reduced nodule count compared to prior HRCT, supportive of a viral aetiology.

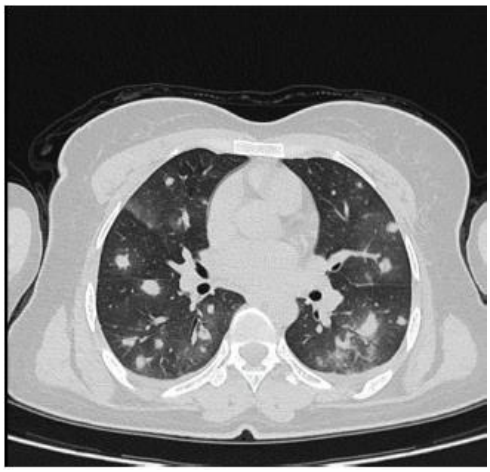

Figure 5: CECT Thorax – Axial section: bilateral nodular consolidations with surrounding halo-like GGOs, absence of enhancement distinguishing viral pneumonitis from pulmonary malignancy.

Management

- **Initial Antibiotic Therapy**

The patient was initially managed as CAP with empirical broad-spectrum antibiotic therapy per IDSA/ATS guidelines, likely comprising a beta-lactam with a macrolide or respiratory fluoroquinolone combination.[14]Despite 4 days of antibiotic therapy, the patient showed persistent fever, ongoing cough, and clinical deterioration with worsening breathlessness — a clinical trajectory highly suggestive of a non-bacterial aetiology.[15] The partial CRP reduction alongside rising ESR reinforced this clinical suspicion.[9,10]

- **Antiviral Therapy with Oseltamivir**

Following radiological re-evaluation suggestive of viral pneumonitis as the most likely diagnosis, Oseltamivir (Tamiflu) 75 mg orally twice daily for 5 days was initiated.[16] Oseltamivir is a neuraminidase inhibitor with established efficacy against Influenza A and B. The WHO and CDC recommend initiating antiviral therapy empirically in patients with severe or complicated influenza — including those with bilateral pneumonia — without awaiting confirmatory virological results, as early treatment is associated with better outcomes.[17] In hospitalised patients with severe influenza pneumonitis, late initiation (beyond 48 hours of symptom onset) has also been shown to confer clinical benefit.[18] A landmark meta-analysis by Dobson et al. (2015) in The Lancet demonstrated that Oseltamivir reduced the risk of lower respiratory tract complications requiring antibiotics by 44% in patients with confirmed influenza compared to placebo.[16] A further meta-analysis by Muthuri et al. (2014) reported significant mortality benefit and reduction in ICU admissions in hospitalised patients with influenza A H1N1pdm09 treated with neuraminidase inhibitors.[18]

In cases where Adenoviral pneumonitis is suspected, there is no established antiviral of proven efficacy in immunocompetent adults; supportive care is the mainstay, with Cidofovir considered only in severely immunocompromised patients.[19] The marked clinical improvement observed with Oseltamivir in this patient suggests an influenza-like viral illness as the probable aetiology, though definitive virology was unavailable.

- **Clinical Outcome and Follow-Up Imaging**

The patient showed marked clinical improvement within 3–4 days of initiating Oseltamivir defervescence, significant reduction in breathlessness, and improvement in overall condition — and was discharged in a stable state following completion of the 5-day antiviral course.

A follow-up HRCT thorax was performed 15 days after initiation of antiviral therapy to document radiological resolution and assess the benign, infectious nature of the previously noted lesions (Figure 6). This demonstrated near-complete clearance of the bilateral consolidations and GGOs, providing strong corroborative evidence against malignancy and supporting a diagnosis of probable resolved viral pneumonitis.[4]

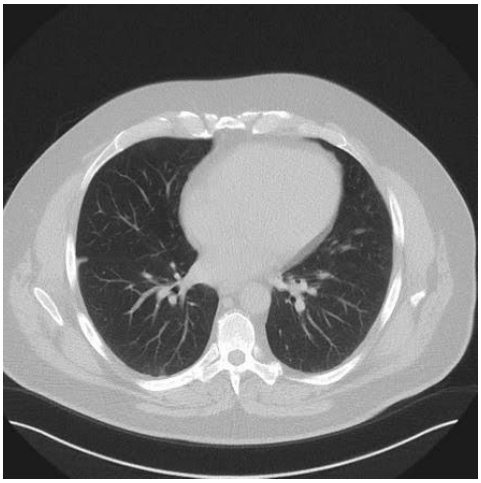

Figure 6: Follow-up HRCT Thorax (axial section) performed 15 days after initiation of Oseltamivir

therapy, showing near-complete radiological resolution consistent with resolving viral pneumonitis.

For completeness and optimal patient care, the following are additionally recommended in similar cases:

(1) Retrospective respiratory virus panel/PCR (Influenza A/B, Adenovirus, RSV, hMPV, Parainfluenza) if stored samples are available; (2) Pulmonology follow-up to ensure complete clinical and radiological resolution; and (3) Genetic counselling and age-appropriate cancer screening given the significant family history of malignancy across three first-degree relatives — Lynch syndrome and other hereditary cancer syndromes warrant evaluation.[20]

Discussion

- **Viral Pneumonitis: Epidemiology and Aetiology**

Viral pneumonitis accounts for approximately 20–30% of CAP cases in immunocompetent adults, though the true prevalence is likely higher due to systematic underdiagnosis.[2] The most common causative agents include Influenza A and B, Adenovirus, RSV, Parainfluenza virus, human Metapneumovirus (hMPV), and — since 2020 — SARS-CoV-2.3 Influenza pneumonitis in particular is well recognised to cause bilateral multifocal consolidations with GGOs that may be radiologically indistinguishable from other causes of acute pneumonitis.[21]

Adenoviral pneumonitis, classically described in military recruits and paediatric populations, is increasingly recognised in community-dwelling adults and can present with severe bilateral pneumonitis and a prolonged clinical course.[22] The clinical features in this patient — non-foul-smelling white sputum, bilateral crepitations, and GGOs — are consistent with both Influenza and Adenovirus aetiology. The absence of a documented serology or PCR panel remains a limitation of this case report, underscoring the importance of comprehensive virological testing in atypical pneumonitis.[3]

- **Radiological Overlap with Pulmonary Malignancy**

The radiological findings in this case — bilateral multifocal consolidations with surrounding GGOs — represent one of the most challenging diagnostic overlaps in pulmonary medicine.[4] These patterns may be seen in: viral pneumonitis (Influenza, Adenovirus, COVID-19), organising pneumonia (cryptogenic or secondary), pulmonary metastases (haematogenous spread), invasive mucinous adenocarcinoma (formerly bronchoalveolar carcinoma), lymphoma, invasive pulmonary aspergillosis (in immunocompromised patients), and septic emboli.[21,23]

Kim et al. noted that GGOs surrounding consolidation nodules — the 'halo sign' — while classically associated with invasive pulmonary aspergillosis, may also be seen in viral pneumonitis, organising pneumonia, and haemorrhagic metastases.[23] The absence of post-contrast enhancement and spontaneous reduction in nodule number on serial CECT imaging, followed by near-complete resolution on the 15-day follow-up HRCT, were the key radiological features that ultimately distinguished this case as infectious rather than malignant — a finding consistent with established radiology literature.[12,13]

- **Anchoring Bias and the Role of Family History**

This case illustrates the cognitive trap of premature diagnostic closure and anchoring bias — where an initial clinical impression disproportionately influences subsequent reasoning, leading clinicians to selectively interpret data in favour of a predetermined diagnosis.[24] The patient's family history of uterine cancer (mother),

laryngeal cancer (brother), and GIT cancer (father) across three first-degree relatives is clinically striking and warrants separate evaluation for hereditary cancer syndromes. Lynch syndrome (Hereditary Non-Polyposis Colorectal Cancer), which predisposes to colorectal, endometrial, and other extracolonic cancers — including rare cases of laryngeal and upper GI malignancies — should be considered and appropriate genetic counselling arranged.[20]

Croskerry's landmark work on cognitive errors in clinical diagnosis highlights anchoring bias as one of the most prevalent and dangerous diagnostic errors in medicine.[24] In this case, the combination of a highly persuasive family history, bilateral HRCT consolidations, and failure to respond to antibiotics created a misleading clinical narrative. It was the objective, dynamic radiological data (no post-contrast enhancement, interval reduction of nodules, and eventual near-complete resolution) and the response to antiviral therapy that corrected the clinical course.

- **Systematic Diagnostic Approach to Bilateral Multifocal Consolidations and GGOs**

A structured approach is essential when evaluating patients with bilateral multifocal consolidations and GGOs:

Table 3: Structured diagnostic approach for bilateral multifocal consolidations and GGOs.

Step	Action	Key Reference
1	Apply CURB-65 and PSI for severity stratification and site-of-care decisions	[6,8]
2	Baseline investigations: CBC, CRP, procalcitonin, blood cultures, sputum culture, urinary antigens, respiratory virus panel	[14]
3	Serial CRP/ESR monitoring — dissociation pattern suggests viral/atypical aetiology	[9,10]
4	HRCT thorax when atypical course, antibiotic failure, or discordant CXR findings	[7]
5	CECT with post-contrast characterisation to exclude malignancy; serial comparison imaging	[12,13]
6	CT-guided biopsy or bronchoscopy/BAL if radiological and clinical data remain inconclusive	[26]
7	Empirical antiviral therapy (Oseltamivir) if bilateral pneumonitis with viral features fails antibiotics	[17,18]
8	Follow-up imaging at 2 weeks to ~6-8 weeks to confirm radiological resolution and exclude residual/evolving disease	[20]

- **Oseltamivir in Viral Pneumonitis: Evidence Base**

Oseltamivir significantly reduces the duration of illness and risk of lower respiratory tract complications in influenza pneumonitis when initiated early.[16] Hospitalised patients with influenza pneumonitis — even when treatment is initiated beyond the conventional 48-hour window — demonstrate mortality benefit,

reduced ICU admissions, and shorter hospital stays with neuraminidase inhibitor therapy.[18]

For Adenoviral pneumonitis in immunocompetent adults, there are no randomised controlled trials of antiviral therapy, and management remains primarily supportive.[19] The clinical improvement observed with Oseltamivir in this patient, corroborated by near-complete resolution on the 15-day follow-up HRCT, most likely reflects an Influenza aetiology, although definitive virological confirmation was not available — a limitation that should be addressed in future similar cases through early comprehensive molecular respiratory diagnostics.[3]

- **Significance and Contribution to the Literature**

This case contributes to the literature for several reasons: (1) it demonstrates how viral pneumonitis can convincingly mimic pulmonary malignancy in both clinical and radiological presentation;[4] (2) it highlights the indispensable value of serial CT comparison, including a follow-up scan demonstrating resolution, in differentiating infectious from neoplastic pulmonary lesions;[13] (3) it illustrates anchoring bias in clinical practice and the cognitive risk of a strong family cancer history;[24] (4) it supports empirical antiviral therapy in atypical bilateral pneumonitis failing antibiotic management;[17] and (5) it raises awareness of hereditary cancer syndrome evaluation as a parallel clinical concern in patients with multi-generational family cancer histories.[20]

Conclusion

This case report presents an instructive diagnostic journey where clinical features initially raising strong suspicion for pulmonary malignancy — supported by an extensive family history of cancer, atypical clinical course, failure to respond to antibiotics, and complex bilateral HRCT findings ultimately revealed viral pneumonitis as the most likely underlying diagnosis. The critical turning points were the absence of post-contrast enhancement on CECT, the spontaneous partial resolution of nodules on serial imaging, and the near-complete radiological clearance on the 15-day follow-up HRCT, all of which are hallmarks of an infectious rather than malignant process.[13]

Several important lessons emerge: CURB-65 and PSI must be applied systematically at admission to guide initial management;[6,8] serial inflammatory marker dissociation (falling CRP, rising ESR) provides an early biochemical clue to non-bacterial, likely viral aetiology;[9,10] HRCT and CECT thorax with serial comparison imaging are indispensable in resolving diagnostic uncertainty;[7,12] empirical Oseltamivir is appropriate and effective in bilateral viral pneumonitis even beyond the conventional treatment window in hospitalised patients;[16,18] and anchoring bias remains an important source of diagnostic error that clinicians must consciously guard against.[24]

Finally, the family history of uterine, laryngeal, and GIT cancers across three first-degree relatives in this patient warrants a separate, structured evaluation for hereditary cancer syndromes, with appropriate genetic counselling and age-appropriate cancer surveillance — independent of the current presentation.[20] In conclusion, viral pneumonitis should remain an important differential diagnosis in bilateral multifocal pneumonitis, and a systematic, dynamic, and unbiased diagnostic approach culminating in follow-up imaging is the cornerstone of accurate and timely management.

Key Learning Points

1. Viral pneumonitis can present with bilateral multifocal consolidations and GGOs that are

radiologically indistinguishable from pulmonary metastases or primary lung malignancy.

2. Serial CT comparison demonstrating interval resolution of pulmonary lesions — including a follow-up scan — is a key radiological feature that differentiates infectious from malignant aetiology.

3. The dissociation between falling CRP and rising ESR on antibiotic therapy should prompt consideration of viral or atypical pneumonia.

4. Anchoring bias driven by a strong family history of cancer can lead to premature diagnostic closure; objective data must override clinical heuristics.

5. Empirical Oseltamivir therapy is appropriate and beneficial in hospitalised patients with bilateral viral pneumonitis, even when initiated beyond 48 hours.

6. Significant multi-generational family cancer histories warrant parallel evaluation for hereditary cancer syndromes regardless of the immediate presenting diagnosis.

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