

Omadacycline - Associated Anticardiolipin Antibody Positivity and Hypercoagulable State: A Case Report and Review of Tetracycline - Induced Immune Dysregulation

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Abstract: We present the case of an 18-year-old previously healthy female diagnosed with *Mycoplasma pneumoniae* pneumonia who developed marked elevation in D-dimer and fibrinogen degradation products, along with de novo positivity for both IgG and IgM anticardiolipin antibodies, shortly after initiating intravenous omadacycline therapy. These findings occurred in the absence of known antiphospholipid syndrome or other autoimmune conditions. The patient also experienced gastrointestinal symptoms and skin rashes. After discontinuation of omadacycline and supportive care, her condition improved. This case highlights the temporal association between omadacycline administration and the emergence of immune-coagulative dysregulation. Given that *Mycoplasma pneumoniae* infection itself can cause similar extrapulmonary manifestations, a definitive causal link cannot be established. This case suggests a probable, though rare, association between omadacycline and immune-coagulative dysregulation. Clinicians should be vigilant for such signals, particularly in patients developing early cutaneous or gastrointestinal symptoms during therapy.

Keywords: omadacycline, *Mycoplasma pneumoniae* pneumonia, anticardiolipin antibody, hypercoagulable state, drug-induced immune response

Introduction

Mycoplasma pneumoniae is a common cause of atypical pneumonia, particularly among adolescents and young adults. It accounts for up to 40% of community-acquired pneumonia cases in this age group.¹ Although most cases are self-limiting, a subset of patients develop extrapulmonary manifestations, including neurological, dermatological, and immunological complications.¹ Macrolides are traditionally used as first-line agents; however, increasing resistance has led to the adoption of alternative antibiotics such as fluoroquinolones and tetracyclines.² Omadacycline, a semi-synthetic tetracycline derivative, demonstrates potent activity against respiratory pathogens and is approved for CABP due to its favorable pharmacokinetics and high tissue penetration.^{3,4} Despite its efficacy and generally favorable safety profile in clinical trials, post-marketing surveillance is essential to identify rare adverse events.⁵⁻⁸ Notably, other tetracyclines have been implicated in immune-mediated phenomena, raising the question of whether omadacycline shares this potential.^{9,10}

We report this case to alert clinicians to this potential adverse effect, systematically review similar events with other tetracyclines, explore possible mechanisms, and provide monitoring recommendations.

Case Presentation

An 18-year-old previously healthy female presented with a one-week history of cough, yellow sputum production, and high-grade fever up to 40°C. She recently returned from overseas and tested weakly positive for SARS-CoV-2 antigen. After seeking medical care, she was prescribed VV116 by a physician, but her symptoms did not improve, prompting her to seek further care at our hospital. Physical examination revealed normal vital signs and symmetric breath sounds bilaterally, without audible rales or wheezes. Laboratory evaluation showed elevated inflammatory markers, normal white blood cell count, and positive IgM antibodies (87.18AU/mL, ref: <20AU/mL) against *Mycoplasma pneumoniae*. No evidence of bacterial coinfection was found on sputum culture or blood cultures. Chest CT confirmed right middle lobe pneumonia in [Figure 1S](#). On admission, she was diagnosed with non-severe community-acquired pneumonia based on clinical findings and chest CT.

The patient was initiated on intravenous omadacycline 200 mg loading dose followed by 100 mg daily. Within two days, she developed a mild rash on the infusion arm in [Table 1S](#). On Hospital Day (HD) 3 (2025/1/2), she experienced nausea, vomiting, diarrhea and transient syncope. On HD4 (2025/1/3), she had an episode of generalized pruritic rash, prompting discontinuation of omadacycline. The patient was switched to ceftriaxone and azithromycin, but developed recurrent rash after azithromycin administration and was subsequently transitioned to levofloxacin. On HD5 (2025/1/4), laboratory results showed markedly elevated D-dimer (36.15mg/L, ref: <0.5mg/L) and FDP (88.91mg/L, ref: <5mg/L) levels, indicating a hypercoagulable state in [Figure 2S](#). Hematology consultation recommended initiation of enoxaparin. Liver enzymes were also significantly elevated on HD8 (2025/1/7), necessitating hepatoprotective therapy. On HD7 (2025/1/6), laboratory results revealed elevated levels of serum immunoglobulin M (IgM 4.49g/L, ref: <2.8g/L) and immunoglobulin E (IgE 448.4g/L, ref: ≤375IU/mL), along with increased complement component 3 (C3). Autoimmune screening showed de novo positivity for both IgG and IgM anticardiolipin antibodies (IgG: 29.56 GPL/mL, ref: <20 GPL/mL; IgM: 28.15 MPL /mL, ref: <20 MPL/mL), while lupus anticoagulant testing remained negative. Notably, anti-nuclear antibody (ANA) was also positive at a titer of 226 U/mL (ref: <55 U/mL), but further autoantibody testing on the same day, including anti-dsDNA, anti-ribosomal P protein, anti-Scl-70, anti-mitochondrial, anti-PCNA, anti-nucleosome, anti-Jo-1, anti-histone, anti-U1-nRNP, anti-smooth muscle, and anti-centromere antibodies, were all negative. Additionally, tests for anti-glomerular basement membrane antibodies and anti-intrinsic factor antibodies were within normal limits. These findings raise the possibility of drug-induced immune stimulation rather than underlying autoimmunity. As part of routine follow-up, serum *Mycoplasma pneumoniae* IgM antibody titers (228AU/mL, ref: <20AU/mL) were monitored throughout hospitalization and peaked on HD7 (2025/1/6), consistent with active infection. While *M. pneumoniae* infection itself can cause extrapulmonary immunological complications, the temporal clustering of these marked coagulation and immune abnormalities shortly after omadacycline initiation was striking.

The final diagnoses included *Mycoplasma pneumoniae* pneumonia, hypercoagulable state, possible drug-induced antiphospholipid-like syndrome and sinus arrhythmia. After discontinuation of omadacycline and appropriate supportive care, the patient's condition improved, and she was discharged with plans for outpatient follow-up.

Literature Review

For now, some cases of tetracycline -induced immune activation or coagulation disturbances have been reported, but no systematic reviews have been conducted to summarize the literature. In order to perform a thorough literature review, we searched the literature published in PubMed prior to December 2026 to identify case reports of immune activation or coagulation disturbances associated with tetracyclines. These search terms consist of “omadacycline or tetracycline or doxycycline or minocycline or tigecycline or eravacycline”, “case report” and linked to the logical word “and”. After screening and assessment, a total of 25 eligible articles were included for analysis in [Table 1](#). Our review indicates that among tetracyclines, minocycline is most frequently associated with systemic autoimmune syndromes (eg, lupus, hepatitis), while tigecycline is linked primarily to coagulopathies like hypofibrinogenemia. Reports for doxycycline and omadacycline are scant, making the present case a novel contribution to the safety profile of the latter.

Table 1 Different Cases of Immune Activation or Coagulation Disturbances Associated with Tetracyclines Administration

Author	Country	Number of Cases	Tetracyclines	Adverse Reactions
Fan et al ¹¹	China	2	Tigecycline	Hypofibrinogenemia
Wu et al ¹²	China	1	Tigecycline	Hypofibrinogenemia
Li et al ¹³	China	1	Tigecycline	Hypofibrinogenemia
Wu et al ¹⁴	China	1	Tigecycline	Coagulopathy and hypofibrinogenemia
Akalay et al ¹⁵	Switzerland	1	Tigecycline	Hypofibrinogenemia
Giryas et al ¹⁶	Israel	1	Tigecycline	“Disseminated Intravascular Coagulation (DIC)” like coagulation
Yang et al ¹⁷	China	1	Tigecycline	Toxic epidermal necrolysis syndrome
Farver ¹⁸	USA	1	Minocycline	Lupus
Akin et al ¹⁹	USA	5	Minocycline	Lupus
Gordon et al ²⁰	Scotland	20	Minocycline	Lupus
Angulo et al ²¹	USA	84	Minocycline	Systemic lupus erythematosus or autoimmune hepatitis
Gough et al ²²	UK	27	Minocycline	Systemic lupus erythematosus or autoimmune hepatitis
Rafa et al ²³	USA	1	Minocycline	Autoimmune Hepatitis
Manning et al ²⁴	USA	1	Minocycline	Acute Autoimmune Hepatitis
Yokota et al ²⁵	Japan	1	Minocycline	Renal Polyarteritis Nodosa
Tabriziani et al ²⁶	USA	1	Minocycline	Renal Polyarteritis Nodosa
Meidan et al ²⁷	Israel	1	Minocycline	Polyarteritis Nodosa
Farrugia et al ²⁸	Malta	1	Minocycline	Nodular vasculitis
Klerkx et al ²⁹	Leuven	1	Minocycline	Eosinophilic pneumonia
Zhao et al ³⁰	China	1	Minocycline	Eosinophilia and systemic symptoms syndrome
Khan et al ³¹	Germany	1	Minocycline	Sweet’s syndrome
Wriston et al ³²	USA	1	Doxycycline	Antinuclear Antibody and Antineutrophil Cytoplasmic Antibody
Zhu et al ³³	China	1	Doxycycline	Elevated PT, APPT and PT/INR
Kadam et al ³⁴	Austria	1	Doxycycline	Jarisch-Herxheimer
Lau et al ³⁵	USA	1	Doxycycline	Stevens-Johnson syndrome

Discussion

Omadacycline is generally considered safe and effective for CABP. However, post-marketing surveillance and recent case reports have begun to reveal rare but significant adverse effects, including gastrointestinal disturbances, hepatic enzyme elevation, and now possibly immune-coagulative dysregulation.^{5–8} In this case, the patient developed a dramatic increase in D-dimer and FDP levels shortly after initiating omadacycline. These findings persisted even after discontinuation of the drug, suggesting that omadacycline might trigger a transient procoagulant state. Importantly, no underlying conditions or concurrent medications typically associated with hypercoagulability (eg, estrogen-containing contraceptives, malignancy, inherited thrombophilia) were present. It is crucial to acknowledge that *M. pneumoniae* infection is strongly associated with hypercoagulability and elevated D-dimer levels. However, in this case, the degree of D-dimer elevation was markedly higher than

typically observed in uncomplicated mycoplasma pneumonia, and its rapid decline post-discontinuation suggests an additional drug-induced component. The concurrent emergence of autoimmune markers further supports this possibility.

This case aligns with recent observations in the literature. Xu et al reported a case of acute pancreatitis following omadacycline use, highlighting the need for clinicians to remain vigilant about unexpected side effects.⁸ Several drugs within the tetracycline class have been implicated in immune-mediated adverse effects. Minocycline, in particular, has been most frequently associated with drug-induced lupus, serum sickness-like reactions, and antiphospholipid syndrome.^{20,30,36,37} Doxycycline has been described in association with cutaneous hypersensitivity, autoimmune hepatitis, and rarely, hemolytic anemia.^{38–40} While omadacycline differs structurally from minocycline and doxycycline, it still belongs to the tetracycline family and may possess similar immunomodulatory properties. Our case further expands the spectrum of potential immune-related complications associated with this newer tetracycline derivative.

Potential Mechanisms of Immune Activation and Coagulopathy Associated with Omadacycline

Although the exact pathophysiology underlying omadacycline-associated immune activation and coagulopathy remains incompletely understood, several plausible mechanisms have been proposed in the literature that may help explain the observed clinical phenomena in this case.

One potential pathway is drug-induced molecular mimicry, a process by which certain medications, including antibiotics, may structurally resemble host antigens or phospholipids, thereby triggering an inappropriate immune response that leads to the formation of autoantibodies such as anticardiolipin antibodies.^{41–43} This phenomenon has been described with various drug classes and could serve as a possible explanation for the *de novo* appearance of these antibodies during omadacycline therapy. This mechanism may specifically account for our patient's development of transient IgG and IgM anticardiolipin antibody positivity in the absence of prior autoimmune disease.

Additionally, mitochondrial interference has been increasingly recognized as a mechanism through which tetracycline-class antibiotics may exert immunomodulatory effects. Tetracyclines are known to inhibit mitochondrial protein synthesis, potentially leading to mitochondrial dysfunction and the induction of cellular stress responses. These disturbances may activate innate and adaptive immune pathways, contributing to systemic inflammation and immune dysregulation.^{44–47} The widespread cellular stress induced by this mechanism could underlie the multi-system hypersensitivity reactions observed in our patient, including the maculopapular rash and gastrointestinal symptoms.

A third potential mechanism involves endothelial activation, which can be induced either by direct drug effects or indirectly via infection-related inflammatory cytokine release. Endothelial cell activation may result in upregulated expression of procoagulant molecules such as tissue factor and von Willebrand factor, both of which play central roles in promoting thrombosis and a hypercoagulable state.^{48,49} This provides a direct link to the marked elevation in D-dimer and fibrinogen degradation products seen in our patient, reflecting active intravascular coagulation.

Finally, accumulating evidence suggests that certain antibiotics—including those in the tetracycline family—may also trigger complement system activation, a key mediator of both immune and coagulation dysregulation. Complement activation can enhance immune-inflammatory signaling and promote thromboinflammatory processes, further linking drug exposure with the development of abnormal coagulation profiles and immune markers.^{50–52} The amplification of inflammatory cascades through this pathway may contribute to the concurrent rise in acute-phase reactants (eg, C-reactive protein) and the overall severity of the immune-coagulative dysregulation in this case.

Taken together, these mechanisms provide a theoretical framework for understanding how omadacycline might contribute to immune-mediated adverse events and hypercoagulability, particularly in susceptible individuals. However, given the limited number of reported cases and the complexity of drug-immune-coagulation interactions, further mechanistic studies are warranted to better define the role of omadacycline in such rare but clinically significant complications.

Assessing the Likelihood of Drug-Induced Antiphospholipid Syndrome

Notably, the patient tested positive for both anticardiolipin antibodies IgG and IgM during hospitalization, although no clinical features of antiphospholipid syndrome (APS), such as thrombosis or pregnancy-related complications, were

observed.^{53,54} Given that baseline anticardiolipin antibody status prior to omadacycline initiation was unknown, we describe this as a de novo detection of anticardiolipin antibody positivity, which emerged during treatment. However, due to the lack of follow-up testing after the initial positive result, the persistence or clearance of these antibodies remains undetermined. In the context of drug-induced immune phenomena, a single measurement is insufficient to confirm transient or persistent positivity; therefore, serial monitoring would be necessary in future cases to better characterize the temporal dynamics and clinical relevance of such findings. It is important to consider the possibility of *Mycoplasma pneumoniae* itself contributing to immune activation and coagulation changes. *Mycoplasma pneumoniae* infections are well recognized to be associated with extrapulmonary manifestations, including neurological, dermatological, hematology, and immunological complications.^{55–57} However, the emergence of hypercoagulability and anticardiolipin antibody positivity occurred during omadacycline therapy, with no prior history of autoimmune disease or known thrombophilic conditions. Although a definitive causal relationship cannot be established due to the lack of baseline and follow-up testing for anticardiolipin antibodies, the temporal association between drug administration and the appearance of these immune and coagulation abnormalities suggests a potential contributory role of omadacycline in triggering immune activation and subsequent coagulation disturbances.

The patient initially developed skin rash and gastrointestinal symptoms, which appeared within the first few days of omadacycline administration. These early adverse events are increasingly recognized as potential harbingers of more complex immune or systemic reactions.^{58–60} Notably, these reactions were followed by more serious manifestations, including hypercoagulability and immune marker abnormalities, suggesting a progressive pattern of multi-system involvement. Drug-induced immune activation may involve several mechanisms, including T-cell-mediated responses, cytokine release, mitochondrial dysfunction, and the formation of autoantibodies, all of which warrant further investigation to better understand the role of omadacycline in immune-mediated adverse events.

Assessing the Likelihood of Omadacycline-Induced Coagulopathy

To evaluate the likelihood that omadacycline was responsible for the coagulation abnormalities (Table 2), we applied the Naranjo Adverse Drug Reaction Probability Scale.^{61,62} The Naranjo Adverse Drug Reaction Probability Scale is a widely used tool for evaluating the likelihood of a drug-induced adverse event.^{61,62} It consists of a structured questionnaire that assesses factors such as temporal relationship, alternative causes, and response to drug discontinuation or specific treatment. A total score ≥ 9 indicates a “definite” association between the drug and the observed reaction, while scores between 5–8 suggest a “probable” link, and scores ≤ 4 indicate “possible” or “doubtful” causality. It yielded a score of 6,

Table 2 Naranjo Adverse Drug Reaction Probability Scale Assessment

Naranjo Score Item	Description	Score
1	Are there previous conclusive reports on this reaction?	1
2	Did the adverse event appear after omadacycline administration?	2
3	Did the adverse event improve upon drug discontinuation?	1
4	Did the adverse reaction reappear when the drug was readministered?	0
5	Alternative causes ruled out?	1
6	Did the reaction reappear when a placebo was given?	0
7	Was the drug detected in the body fluids in concentrations known to be toxic?	0
8	Was the reaction more severe when the dose was increased, or less severe when the dose was decreased?	0
9	Did the patient have a similar reaction to the same or similar drugs in any previous exposure?	0
10	Was the adverse event confirmed by any objective evidence?	1
Total	–	6

indicating a probable association between omadacycline administration and the development of coagulative abnormalities in this patient. The score was primarily limited by the absence of a rechallenge and the potential confounding role of the underlying *M. pneumoniae* infection, which precluded completely ruling out an alternative cause. The clinical and laboratory improvement observed after the discontinuation of omadacycline may be partly attributable to the natural resolution of the underlying *M. pneumoniae* infection. However, the rapid normalization of the markedly elevated D-dimer and anticardiolipin antibodies provides supportive evidence for a significant contribution from the withdrawal of the suspected agent. This assessment supports the hypothesis that omadacycline may have contributed to the observed hypercoagulable state, although a definitive causal relationship cannot be established due to the lack of rechallenge and potential confounding factors such as *Mycoplasma pneumoniae* infection.

Clinical Implications and Monitoring Recommendations

Given the rarity of these events, they are unlikely to be captured in large randomized controlled trials. Therefore, real-world case reports play a crucial role in identifying and understanding such associations. The development of a rash or significant gastrointestinal symptoms within the first few days of omadacycline therapy, as seen in this case, may serve as a clinical harbinger and should prompt consideration of early discontinuation and laboratory evaluation for coagulation or immune abnormalities. Clinicians should consider monitoring coagulation profiles (including D-dimer, fibrinogen, and APTT) and autoimmune markers (such as anticardiolipin antibodies and lupus anticoagulant) in patients receiving omadacycline, especially those with unexplained bleeding, thrombosis, or skin reactions. Particularly in young patients, who may not have established risk factors for thrombosis or autoimmune disease, the development of new-onset coagulation abnormalities or immune markers warrants thorough investigation into medication use and infectious triggers. Although omadacycline appears to be well tolerated overall, this case underscores the importance of maintaining a high index of suspicion for immune and coagulation-related adverse effects in atypical clinical scenarios.

Limitations and Future Research Directions

As with any single-case report, the causal relationship between omadacycline and the development of anticardiolipin antibody positivity and hypercoagulability cannot be definitively established. Confounding factors such as the presence of *Mycoplasma pneumoniae* infection must also be considered. Moreover, the lack of long-term follow-up data limits our ability to assess the persistence of these abnormalities or their clinical significance over time. Further research is necessary to clarify the mechanisms behind these findings and to determine whether they represent a broader class effect among tetracycline derivatives. Prospective observational studies or registries evaluating the incidence and characteristics of immune and coagulation abnormalities in patients treated with omadacycline would be valuable. In vitro and animal models could also help elucidate the molecular pathways involved.

Conclusion

This case highlights a possible association between omadacycline and immune-coagulative dysregulation, particularly in younger individuals without traditional thrombotic or autoimmune risk factors. Although causality cannot be definitively established, the Naranjo score suggests a probable adverse drug reaction. Clinicians should remain vigilant for unusual immune or coagulation abnormalities during treatment and consider switching to alternative regimens when appropriate.

Data Sharing Statement

Data and materials are provided within the manuscript.

Ethics and Consent Statements

This study was approved by the Clinical Research Ethics Committee of Huadong Hospital, Fudan University (Approval Number: 20250088). As this is a retrospective case report and does not involve patient tissue or blood samples, written informed consent for publication was waived by the ethics committee. However, all personal identifiable information has been

anonymized to protect patient privacy. Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the signed consent form is available for review by the editor if necessary.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors declare no competing interests in this work.

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