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Anti-Ro-Positive Lupus-Like Multisystem Inflammatory Syndrome After BNT162b2 COVID-19 Vaccination: A Case Report

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Masood Shaikh [1], Sant Das [2], Sadhna Priya [3], Sana Khan [4]

1. Department of Respiratory Medicine, Queen Elizabeth Hospital, King's Lynn, GBR 2. Department of Internal Medicine, Diana Princess of Wales Hospital, Grimsby, GBR 3. Department of Acute Medicine, Diana Princess of Wales Hospital, Grimsby, GBR 4. Undergraduate Medical Education, Aga Khan University Hospital, Karachi, PAK

Corresponding author: Sana Khan, sanaarifkhan28@gmail.com

Abstract

Rare immune-mediated syndromes following COVID-19 vaccination have been described across a heterogeneous clinical spectrum. Anti-Ro/SSA-positive multisystem presentations with features of undifferentiated connective tissue disease (UCTD) or subacute cutaneous lupus erythematosus (SCLE) remain under-reported.

A 66-year-old Caucasian man with no prior autoimmune history developed a progressive multisystem inflammatory illness approximately 14 days after receiving his second dose of the BNT162b2 (Pfizer-BioNTech) COVID-19 vaccine. Clinical features included an erythematous malar and dorsal rash, pharyngitis, geographic tongue, constitutional upset with profound fatigue, night sweats, and unintentional weight loss of 12 kg. Investigations revealed normocytic anaemia, elevated inflammatory markers, and bilateral pleural effusions. Positron emission tomography-computed tomography (PET-CT) demonstrated metabolically active mediastinal, supraclavicular, and pelvic lymphadenopathy with increased bone marrow and splenic uptake. Autoimmune serology showed a positive antinuclear antibody (ANA) test and strongly positive anti-Ro/SSA antibodies, with negative anti-double-stranded DNA (anti-dsDNA) antibodies, normal complement levels, and negative antineutrophil cytoplasmic antibodies (ANCA). Extensive investigation excluded malignancy, infection, sarcoidosis, and classical systemic lupus erythematosus (SLE). A lower limb deep vein thrombosis developed during hospitalisation. Systemic corticosteroids produced clear and sustained clinical and radiological improvement.

The serological and clinical phenotype (anti-Ro-positive, anti-dsDNA-negative, complement-normal, without nephritis) is most consistent with UCTD or SCLE with systemic involvement rather than classical SLE. This case contributes to the emerging literature on post-vaccination autoimmunity, emphasises the importance of precise serological phenotyping, and highlights the need for structured long-term follow-up to determine whether such presentations represent transient immune dysregulation or the onset of persistent connective tissue disease.

Categories: Pulmonology, Allergy/Immunology
Keywords: anti-ro/ssa, asia syndrome, bnt162b2, corticosteroids, covid-19 vaccine, post-vaccination autoimmunity, subacute cutaneous lupus erythematosus, undifferentiated connective tissue disease

Introduction

Vaccination represents one of the most consequential public health interventions of modern medicine. The rapid global deployment of COVID-19 vaccines, beginning in late 2020 and accelerating throughout 2021, resulted in exceptionally large populations being vaccinated within a compressed timeframe. This created unprecedented opportunities for post-authorisation surveillance to identify rare adverse-event signals that conventional pre-licensure trials may have been insufficiently powered to detect [1]. Although new-onset rheumatic immune-mediated inflammatory conditions have been reported following COVID-19 vaccination, anti-Ro/SSA-positive multisystem presentations with features overlapping undifferentiated connective tissue disease or subacute cutaneous lupus erythematosus remain uncommon and are largely confined to individual case reports and small case series [2].

Rare immune-mediated responses to vaccination have been documented in the broader literature for decades. Shoenfeld and Agmon-Levin introduced the concept of autoimmune/inflammatory syndrome induced by adjuvants (ASIA) to describe immune-mediated disorders arising in genetically predisposed individuals following adjuvant exposure [3,4]. Proposed mechanistic pathways include molecular mimicry between vaccine antigens and self-proteins, bystander lymphocyte activation, polyclonal B-cell stimulation, and dysregulation of innate and adaptive immune responses, though precise pathophysiology remains incompletely characterised [5,6].

Early systematic reviews of post-COVID-19 vaccination autoimmunity have described a heterogeneous clinical spectrum encompassing immune thrombocytopenia, Guillain-Barré syndrome, autoimmune hepatitis, IgA nephropathy, Graves' disease, and SLE-like presentations [7]. However, descriptions of anti-Ro/SSA-positive multisystem syndromes with features overlapping undifferentiated connective tissue disease (UCTD) or subacute cutaneous lupus erythematosus (SCLE) remain absent from the published literature.

UCTD describes patients with clinical features suggestive of a systemic autoimmune disease, together with circulating autoantibodies, who do not fulfil classification criteria for a defined connective tissue disease such as systemic lupus erythematosus (SLE) [8]. SCLE is a distinct subset of lupus characterised by a non-scarring, photo-distributed annular or papulosquamous eruption, often accompanied by mild systemic features such as fatigue and arthralgia, but less commonly by severe renal or neurological involvement. Both entities are closely associated with antibodies against the Ro/SSA antigen. Unlike classical SLE, these anti-Ro-associated phenotypes are typically anti-dsDNA-negative and complement-normal [8], a distinction with practical implications for prognosis, organ monitoring, and treatment. We report such a case, discuss its serological and clinical implications, and propose a mechanistic framework grounded in current understanding of type I interferon biology.

Case Presentation

Patient background and vaccination history

A 66-year-old Caucasian man with no prior history of autoimmune or connective tissue disease, no significant chronic comorbidities, and no regular medications was referred for rheumatological evaluation of a progressive multisystem inflammatory illness. Symptoms began approximately 14 days after receiving his second dose of BNT162b2 (Pfizer-BioNTech) COVID-19 vaccine. No adverse reaction had been reported following the first dose. There was no family history of autoimmune disease. He was a non-smoker with only occasional alcohol use. No relevant occupational or environmental exposures were identified.

Clinical course

Weeks 2-4 Post-vaccination

Initial symptoms included right-sided otalgia with seropurulent discharge, pharyngitis, geographic tongue with surface fissuring, and a spreading erythematous maculopapular rash affecting the malar region of the face and upper back. The eruption was non-vesicular, non-ulcerative, and not associated with mucosal bleeding or targetoid lesions. He was managed in the community for a probable allergic or infective process, receiving topical corticosteroid cream for the rash alongside two sequential courses of broad-spectrum oral antibiotics. There was no clinical improvement following either course.

Months 1-3 Post-vaccination

Over the following weeks, the patient developed progressive constitutional symptoms comprising profound fatigue, exertional dyspnoea (NYHA class II-III), drenching night sweats, and unintentional weight loss of approximately 12 kg. He remained systemically unwell and was referred to secondary care for further evaluation.

Hospitalisation

The patient was admitted with worsening dyspnoea, profound fatigue, and ongoing constitutional symptoms. During the admission, he developed a symptomatic left lower limb deep vein thrombosis (DVT), confirmed on Doppler ultrasonography. Computed tomography pulmonary angiography excluded pulmonary embolism. He was anticoagulated with therapeutic enoxaparin and received a further course of broad-spectrum intravenous antibiotics without meaningful improvement.

Overall, symptom onset occurred approximately two weeks post-vaccination, with progressive systemic involvement over the following two to three months prior to hospitalisation (Table *1*).

Time	Event
Day 0	Second dose of BNT162b2
About day 14	Onset of otalgia, pharyngitis, geographic tongue, malar and upper back rash
Weeks 2-4	Topical steroid and two courses of oral antibiotics, no improvement
Months 1-3	Progressive fatigue, exertional dyspnoea, night sweats, 12 kg weight loss
Hospital admission	Left leg DVT, PE excluded on CTPA, enoxaparin, IV antibiotics, no improvement
Investigations	Serology, PET-CT, EBUS-TBNA, bone marrow biopsy and flow cytometry
After exclusion of alternatives	Systemic corticosteroids started, clinical and partial radiological improvement
Follow-up	Not available

TABLE 1: Timeline of clinical events

DVT: deep vein thrombosis; PE: pulmonary embolism; CTPA: computed tomography pulmonary angiography; IV: intravenous; PET-CT: positron emission tomography-computed tomography; EBUS-TBNA: endobronchial ultrasound-guided transbronchial needle aspiration.

Investigations

Full blood count demonstrated a normocytic anaemia (haemoglobin 98 g/L) without thrombocytopenia or leucopenia. The erythrocyte sedimentation rate was elevated at 89 mm/h. Serum biochemistry, including renal and hepatic function panels, was within normal limits. Urinalysis with microscopy showed no proteinuria, haematuria, or cellular casts. Complement levels were within the normal range (C3 1.66 g/L; C4 0.35 g/L). Serum immunoglobulins were not significantly elevated. HIV serology was negative. Serum angiotensin-converting enzyme (ACE) was mildly elevated at 52 U/L. Sputum culture and sensitivity testing for Mycobacterium tuberculosis and routine bacterial pathogens were negative. A short Synacthen test was normal.

Autoimmune serology demonstrated positive antinuclear antibody (ANA) and strongly positive anti-Ro/SSA antibodies. Anti-La/SSB, anti-dsDNA, anti-Sm, anti-RNP, anti-Jo-1, anti-Scl-70, and anti-centromere antibodies were all negative. ANCA testing was negative (PR3 ELISA 0.70, reference range 0.00-3.00; MPO ELISA negative). Lupus anticoagulant screening showed a mildly elevated lupus-sensitive APTT ratio of 1.17; anticardiolipin and anti-beta-2 glycoprotein I antibodies were within normal limits (Table *2*).

Antibody/Test	Result	Reference Range
Antinuclear antibody (ANA)	Positive (titre/pattern not available)	Negative
Anti-Ro/SSA	Strongly positive	Negative
Anti-La/SSB	Negative	Negative
Anti-dsDNA	Negative	Negative
Anti-Sm	Negative	Negative
Anti-RNP	Negative	Negative
Anti-Jo-1	Negative	Negative
Anti-Scl-70	Negative	Negative
Anti-centromere	Negative	Negative
PR3 ELISA	0.70	0.00-3.00 U/mL
MPO ELISA	Negative	Negative
Lupus-sensitive APTT ratio	1.17 (mildly elevated)	0.80–1.10
Anticardiolipin antibodies	Negative	<10 GPL/MPL U/mL
Anti-beta-2 glycoprotein I	Negative	<10 U/mL

TABLE 2: Autoimmune and thrombophilia serological profile

ANA: antinuclear antibody; APTT: activated partial thromboplastin time; dsDNA: double-stranded DNA; GPL/MPL: immunoglobulin G/immunoglobulin M phospholipid units; MPO: myeloperoxidase; PR3: proteinase 3; RNP: ribonucleoprotein; SSA/SSB: Sjögren syndrome antigen A/B.

PET-CT demonstrated metabolically active lymphadenopathy involving multiple mediastinal stations, the right supraclavicular region, and several pelvic nodes. Minimal bilateral pleural effusions were present (Figure *1*). Increased FDG uptake was additionally noted in the bone marrow and spleen in the absence of radiological splenomegaly. The reported differential diagnosis included sarcoidosis and lymphoma; tuberculosis and metastatic disease were considered less likely (Table *2*).

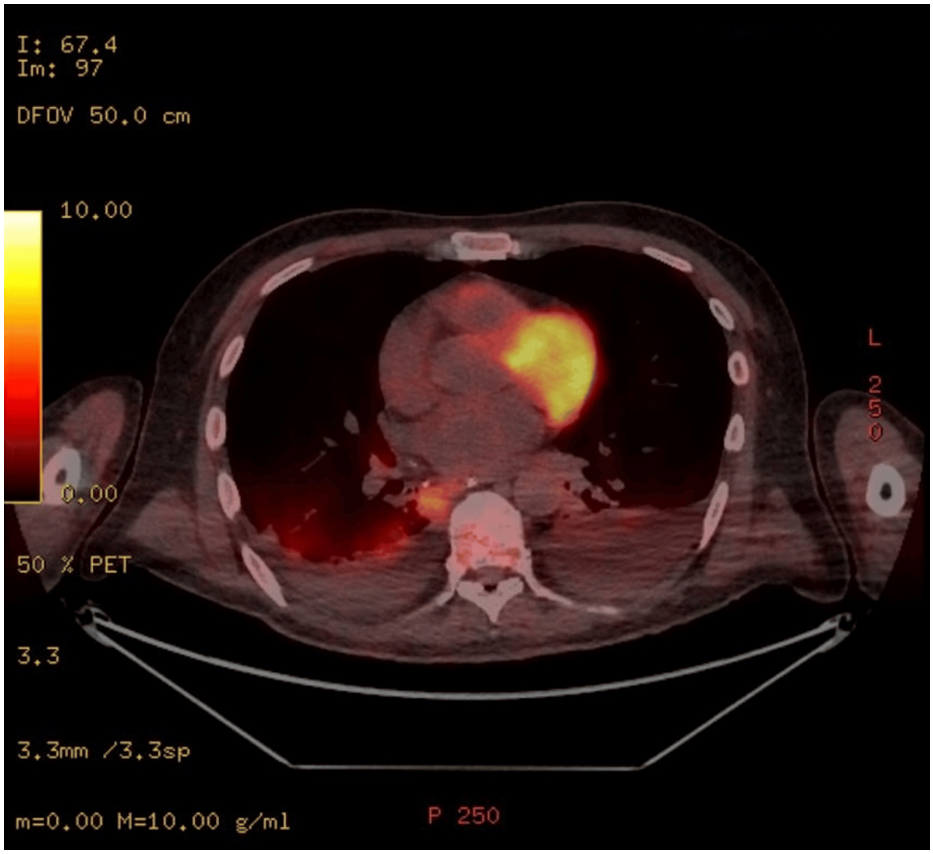


FIGURE 1: Fused PET-CT (axial view) demonstrating metabolically active mediastinal lymphadenopathy

Endobronchial ultrasound-guided transbronchial needle aspiration (EBUS-TBNA) of mediastinal lymph nodes demonstrated no granulomatous inflammation and no features of malignancy. Bone marrow aspirate and trephine biopsy showed only reactive changes without significant pathological infiltrate. Flow cytometry on peripheral blood and bone marrow identified no atypical lymphoid population.

Treatment and response

Following exclusion of infection, malignancy, sarcoidosis, and classical SLE, a trial of systemic corticosteroids was initiated; the specific agent, starting dose, and tapering schedule were not documented in the records available to the authors. The clinical response was progressive and unambiguous. Appetite returned, mobility improved, and the patient regained approximately 7 kg of body weight. Repeat imaging demonstrated near-complete resolution of bilateral pleural effusions and a reduction in paratracheal lymph node size, with residual stable mediastinal lymphadenopathy. Bilateral patchy ground-glass opacities persisted on high-resolution CT, but overall radiological appearances improved substantially. Follow-up data beyond the period of initial clinical and radiological response were not available to the authors at the time of writing. Accordingly, the current treatment status, the long-term clinical course, and repeat serological testing (including anti-Ro/SSA, ANA, and repeat antiphospholipid antibodies) could not be reported.

Discussion

Diagnostic framing

The serological profile in this case, characterised by strongly positive anti-Ro/SSA, negative anti-dsDNA, and normal complement levels, in the absence of nephritis, significant cytopenias beyond anaemia, or neuropsychiatric involvement, does not meet the 2019 ACR/EULAR classification criteria for SLE [8]. It is instead most consistent with undifferentiated connective tissue disease (UCTD) or subacute cutaneous lupus erythematosus (SCLE) with systemic inflammatory involvement. Anti-Ro/SSA positivity is the defining serological feature of SCLE and is the most prevalent antibody in UCTD; it is distinct from the anti-dsDNA-positive, complement-depleting biology that characterises classical SLE [8]. This distinction is not merely academic: it has direct implications for treatment selection, organ monitoring priorities, and long-term prognostication. Framing such cases within UCTD or SCLE rather than broadly as lupus-like disease reduces diagnostic imprecision and better guides clinical management. The phenotype in this case is most

consistent with an anti-Ro-positive UCTD/SCLE-spectrum inflammatory syndrome [8]. The anti-Ro-dominant serological profile serves as the central feature anchoring the phenotype within the SCLE/UCTD spectrum rather than classical SLE.

Temporal association and causality

Establishing causality in post-vaccination autoimmunity is inherently difficult, particularly when evidence is limited to a single case. In this patient, symptom onset approximately 14 days after the second vaccine dose establishes temporality and is compatible with the expected timeframe of an adaptive immune response; however, temporal association alone does not establish causation. A structured causality assessment reveals both supporting and limiting features: temporality is satisfied; biological plausibility is partial; consistency is limited by the scarcity of comparable published cases; specificity is weak because anti-Ro/SSA positivity may represent pre-existing subclinical autoimmunity unmasked by immunological activation; and a dose-response relationship cannot be assessed from a single observation. The clear response to corticosteroids supports an immune-mediated inflammatory process but does not independently establish vaccine causality [9].

Critically, pre-vaccination autoantibody status was unavailable. Anti-Ro/SSA antibodies are known to circulate subclinically for years prior to overt disease, and it remains plausible that vaccination precipitated clinical expression of pre-existing latent autoimmunity rather than inducing it de novo [10]. Furthermore, at the scale of billions of doses administered globally, coincidental development of UCTD or SCLE in the post-vaccination window is statistically expected regardless of any causal relationship [7]. Population-level pharmacovigilance data, not individual case reports, are required to determine whether BNT162b2 vaccination is associated with an excess risk of anti-Ro-positive autoimmune disease.

Proposed immunological mechanisms

Several non-mutually exclusive mechanisms may explain vaccine-associated autoimmunity in genetically predisposed individuals [5,6]. Molecular mimicry remains the most cited hypothesis: the SARS-CoV-2 spike protein shares peptide homology with several human proteins, and cross-reactive T- and B-cell responses have been experimentally demonstrated [11]. However, direct evidence linking spike-protein mimicry to anti-Ro/SSA induction is currently lacking. Bystander activation involves non-specific stimulation of autoreactive lymphocytes during a robust innate immune response without requiring antigen cross-reactivity [5,6]. In this context, the intense type I interferon (IFN-I) response induced by mRNA vaccines is of particular mechanistic relevance, as IFN-I signalling is directly implicated in the pathogenesis of both SCLE and SLE [12]. The anti-Ro-dominant serological profile in this case aligns with the IFN-I-driven biology of SCLE, making the vaccine's interferon-stimulating properties a biologically plausible, if unproven, link [12]. Polyclonal B-cell activation may additionally generate transient non-specific autoantibody production in individuals with underlying B-cell dysregulation [5,6].

The cutaneous component and Rowell syndrome

The malar and dorsal erythematous eruption with positive anti-Ro serology prompted consideration of Rowell syndrome, a rare entity characterised by lupus erythematosus associated with erythema multiforme-like lesions and specific immunological features, including ANA and anti-Ro/SSA positivity [13]. Rowell-like presentations following COVID-19 infection and BNT162b2 vaccination have been reported in small numbers [14,15]. However, in the absence of histopathological characterisation of the cutaneous eruption in this case, and given the breadth of systemic involvement that extends beyond the Rowell phenotype, this remains a descriptive consideration rather than a confirmed diagnosis [13-15]. Skin biopsy with direct immunofluorescence, notably absent in this case, would be definitive. Taken together, these distinctions emphasise that the present case is best interpreted through its anti-Ro-dominant phenotype rather than by alignment with classical SLE or isolated cutaneous lupus entities.

The thrombotic complication

The lower limb DVT merits careful interpretation. The mildly elevated lupus-sensitive APTT ratio of 1.17, in the context of acute systemic inflammation and concurrent anticoagulation with enoxaparin, is insufficient to diagnose antiphospholipid syndrome (APS). Established APS requires confirmed antiphospholipid antibody positivity on two occasions at least 12 weeks apart [8]. The anticardiolipin and anti-beta-2 glycoprotein I antibodies were negative on both occasions tested. The thrombotic event most plausibly reflects the convergence of prolonged immobility, a severe systemic inflammatory prothrombotic state, and possible transient immune-mediated coagulation perturbation. Repeat thrombophilia testing following clinical recovery and cessation of anticoagulation is strongly recommended.

Comparison with published post-vaccination SLE-spectrum cases

Several cases of new-onset SLE-spectrum disease following COVID-19 vaccination have been reported. Zavala-Miranda et al. described new-onset class V lupus nephritis following COVID-19 vaccination; Hidaka et al. reported Evans syndrome associated with SLE after BNT162b2; and Nune et al. described a case of new-onset SLE with high-titre ANA and elevated anti-dsDNA two weeks after the second Pfizer dose [16-18].

Our case is serologically and phenotypically distinct from all three: the absence of anti-dsDNA antibodies, normal complement levels, absence of nephritis, and the anti-Ro-dominant serology collectively differentiate it from classical SLE [8]. This distinction has been under-represented in the published literature, and we suggest that future case reports in this area employ precise serological phenotyping, including anti-Ro52 versus anti-Ro60 subtyping and ANA titre and pattern, to facilitate meaningful comparison across cases.

Limitations

Several important limitations constrain the conclusions that can be drawn from this report. The absence of pre-vaccination autoantibody data is the most critical: without a baseline, it is impossible to determine whether anti-Ro/SSA positivity was truly de novo or reflected pre-existing subclinical autoimmunity unmasked by vaccination [10]. The ANA titre and immunofluorescence pattern were unavailable, limiting serological characterisation. Histopathological confirmation of the cutaneous eruption was not obtained, precluding definitive classification of the rash. Treatment details, including the corticosteroid agent, starting dose, and tapering schedule, were not recorded, limiting the clinical utility of this case for physicians managing similar presentations. Serial autoantibody monitoring was not performed, meaning we cannot determine whether anti-Ro titres changed in parallel with clinical response [10]. Finally, follow-up beyond the initial treatment response was not available, so it is not possible to determine whether this represents transient vaccine-associated immune dysregulation or the onset of a persistent connective tissue disease requiring long-term immunosuppression [7]. Inflammatory markers were reported only at admission, and serial values were not available.

Conclusions

This case describes an anti-Ro-positive multisystem inflammatory syndrome with features most consistent with UCTD or SCLE, with systemic involvement arising in temporal association with BNT162b2 COVID-19 vaccination. Extensive investigation excluded malignancy, infection, sarcoidosis, and classical SLE. Systemic corticosteroids produced a clear and sustained clinical and radiological response. The case is distinguished from previously reported post-vaccination lupus presentations by its anti-Ro-dominant, anti-dsDNA-negative, complement-normal serological profile and adds to a growing body of literature characterising rare immune-mediated syndromes following COVID-19 vaccination. Follow-up beyond the initial response was not available, and the longer-term outcome remains unknown.

Clinicians evaluating unexplained systemic inflammatory illness following COVID-19 vaccination should include comprehensive autoimmune serology, including ANA titre and pattern, anti-Ro subtyping, and complement levels, as part of their assessment, and should consider skin biopsy where cutaneous features are present. Causality cannot be established from a single case, and the considerable public health benefit of COVID-19 vaccination remains undiminished by rare reports of this kind. Prospective research incorporating type I interferon gene signature assays, HLA typing, and serial autoantibody monitoring is needed to determine whether anti-Ro-positive autoimmune disease represents a genuine vaccine-attributable signal or an incidental coincidence in a large vaccinated population.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Acquisition, analysis, or interpretation of data: Sana Khan, Sant Das, Sadhna Priya

Drafting of the manuscript: Sana Khan

Concept and design: Masood Shaikh

Critical review of the manuscript for important intellectual content: Masood Shaikh, Sant Das, Sadhna Priya

Supervision: Masood Shaikh

Disclosures

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declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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