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editorial-office@sciformat.ca

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FROM A POSITIVE AUTOANTIBODY TO AUTOIMMUNE DISEASE: WHAT DOES THE EVIDENCE SHOW FOLLOWING VIRAL INFECTIONS?

Anna Gwóźdź-Broczkowska *(Corresponding Author, Email: agwozdz-broczkowska@wim.mil.pl)*
Military Institute of Medicine – National Research Institute, Szaserów 128, 04-141 Warsaw, Poland
ORCID ID: 0009-0005-4839-5261

Agata Kotłowska
Nicolaus Copernicus Hospital, Copernicus PL Sp. z o.o., Gdańsk, Poland
ORCID ID: 0000-0001-9665-8533

Julia Lenkiewicz
Nicolaus Copernicus Hospital, Copernicus PL Sp. z o.o., Gdańsk, Poland
ORCID ID: 0000-0002-6976-6990

Aleksandra Izabela Masłowska
Military Institute of Medicine – National Research Institute, Szaserów 128, 04-141 Warsaw, Poland
ORCID ID: 0009-0006-7634-1006

Wiktoria Napiórkowska
Military Institute of Medicine – National Research Institute, Szaserów 128, 04-141 Warsaw, Poland
ORCID ID: 0009-0007-9546-3356

Gabriela Luba
National Medical Institute of the Ministry of the Interior and Administration, Warsaw, Poland
ORCID ID: 0009-0007-4262-2093

Marcin Trzciński
Nicolaus Copernicus Hospital, Copernicus PL Sp. z o.o., Gdańsk, Poland
ORCID ID: 0000-0003-4817-7772

Oliwia Lenkiewicz
Nicolaus Copernicus Hospital, Copernicus PL Sp. z o.o., Gdańsk, Poland
ORCID ID: 0000-0002-4794-627X

ABSTRACT

Autoantibodies are commonly detected during or shortly after viral illness, sometimes in patients whose presentation already resembles a systemic or organ-specific autoimmune disorder. The difficulty is that the laboratory finding often arrives before the clinical meaning of that finding is clear. In acute parvovirus B19 and hepatitis E, ANA, RF or autoimmune liver serology may accompany the infection and then recede as the illness resolves. SARS-CoV-2 cohorts that include pre-infection samples show that genuine seroconversion can occur, but they also expose a separate problem: a newly detectable or persistent autoantibody is not the same endpoint as clinically defined autoimmune disease. HCV-associated cryoglobulinaemic vasculitis is different because infection, immune-complex formation and organ disease belong to the same pathogenic process. Across the other viral settings, interpretation is limited by referral bias, incomplete follow-up, assay heterogeneity and the frequent use of serological rather than clinical endpoints. Taken together, the recent literature supports post-viral autoreactivity far more consistently than it supports frequent progression to a new autoimmune disease. Diagnostic weight increases when the antibody is disease-concordant, persists beyond the acute infection and accompanies an objective phenotype that the infection no longer adequately explains.

KEYWORDS

Autoantibodies, Viral Infection, Seroconversion, Longitudinal Studies, Autoimmune Disease, Diagnostic Misclassification

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1. Introduction

A positive autoantibody can be clinically persuasive and still be misleading. This is especially true during acute infection, when fever, rash, arthritis, cytopenia, thrombosis, renal abnormalities or complement consumption may prompt rheumatological testing before the syndrome has declared itself. ANA, rheumatoid factor (RF), antiphospholipid antibodies (aPL) and other markers then enter the diagnostic discussion at a time when infection itself may account for much of the phenotype. Background ANA positivity is also common, and inappropriate or low-yield autoimmune testing remains frequent in referral pathways (Ahrari et al., 2020; Dinse et al., 2022). For that reason, the first positive sample obtained during infection is evidence that an antibody was detected at that moment. It is not evidence that the infection induced it.

Even a documented seroconversion does not settle the clinical question. Viral infections can perturb B-cell activation, expose intracellular antigens and promote cross-reactive responses, all of which make transient autoreactivity biologically plausible (Johnson & Jiang, 2023; Rojas et al., 2023). Pathogenicity, however, is a separate claim. The antibody may disappear, remain detectable without consequences, or accompany an inflammatory syndrome that ultimately proves unrelated to a conventional autoimmune disease. What matters clinically is the sequence over time: when the antibody appeared, whether the same reactivity persists, and whether an objective phenotype persists with it after the infection would ordinarily be expected to resolve.

Most clinical studies capture only part of that sequence. Cross-sectional datasets are useful for showing that an antibody and an infection coexist, but usually cannot establish induction. Referral cohorts tell us what difficult mimics look like in practice, although their denominators are already enriched for patients who appeared rheumatological enough to be tested. Follow-up is more informative, but it is often selective: patients who recover are less likely to return for repeat serology than those who remain symptomatic. Mechanistic studies answer yet another question, demonstrating binding, cross-reactivity or functional effects without necessarily showing a later autoimmune diagnosis. These designs are informative, but not interchangeable.

The question addressed here is therefore narrower than whether viruses are capable of eliciting autoreactivity. We ask when the available evidence permits a move from a positive autoantibody to a credible diagnosis of autoimmune disease. The principal endpoint is a clinically defined systemic or organ-specific disorder supported by objective inflammatory or organ findings; isolated seropositivity does not meet that

threshold. The viral examples were chosen because they stress different parts of the inference. B19 and HEV are acute mimics, chikungunya shows that persistent inflammation need not be accompanied by disease-specific seroconversion, and SARS-CoV-2 provides unusually strong temporal data because some cohorts have pre-infection samples. HCV-associated cryoglobulinaemic vasculitis and Zika-associated Guillain-Barré syndrome are kept as comparators rather than treated as equivalent pathways to systemic rheumatic disease.

2. Methodology and critical appraisal approach

We reviewed human studies published from 1 January 2016 to 20 August 2026. PubMed was searched separately for each viral setting using the virus name together with relevant autoantibodies and clinical outcomes; additional terms were used for autoimmune liver serology in HEV and for ganglioside or peripheral nerve antibodies in dengue and Zika. Reference lists of relevant original studies and reviews were also checked for papers published within the same period.

Study selection was purposive because the aim was not to derive a pooled prevalence estimate. We gave greatest weight to cohorts with a true pre-infection sample and subsequent clinical follow-up, since these can address both induction and disease. Planned longitudinal studies without a pre-infection baseline were useful for persistence or seroreversion, but not for proving when an antibody first appeared. Referral cohorts and cross-sectional studies were retained when they clarified a diagnostic mimic or a clinically important association. Mechanistic studies without a clinical endpoint were used only to support biological plausibility, and case reports were not used to estimate frequency.

During appraisal, we asked what link in the chain each study had actually measured. Particular attention was paid to first detection versus seroconversion, protocol-driven versus symptom-driven follow-up, laboratory persistence versus objective disease, and routine diagnostic assays versus broad research platforms. The studies were too heterogeneous in setting, assay, sampling schedule and follow-up for a pooled estimate to be meaningful, and no formal risk-of-bias score was applied. Table 1 summarises the selected studies in terms of the inference each design can support and the limitation that most restricts that inference.

Table 1. Selected studies and the strength of inference they support

Study / setting	Design feature that adds value	Main observation	Major limitation	Inference supported
D'Onofrio et al. (2024) Hassan et al. (2024) Negri et al. (2026) *Parvovirus B19*	Acute cohorts with clinical follow-up in different referral settings	Marked rheumatic mimicry. Most patients recovered, while a minority received later autoimmune diagnoses	No routine pre-infection serology. Strong selection effects and different testing pathways	Strong evidence for mimicry. Weak evidence for frequent transition to chronic autoimmune disease
Guillot et al. (2020) Hossain et al. (2022) *Chikungunya*	Long-term or longitudinal assessment of persistent joint disease	Chronic inflammatory manifestations can persist without consistent ACPA seroconversion	Selected rheumatology populations. Limited pre-infection serology	Persistent inflammation is well supported. RA-specific serological transition is not
Bonacci et al. (2017) Fayed et al. (2022) Lauletta et al. (2024) Ferri et al. (2026) *HCV*	Serial assessment before and after direct-acting antiviral therapy	Clinical and immunological responses improve with viral clearance but do not always move together	Baseline is pre-treatment rather than pre-infection. Tertiary-care selection	Strong evidence that viral activity and immune-complex disease are linked but not identical

Study / setting	Design feature that adds value	Main observation	Major limitation	Inference supported
Terziroli Beretta-Piccoli et al. (2018) Wu et al. (2020) Gui et al. (2021) *HEV*	Repeat autoimmune liver serology after acute HEV	ANA and SMA are common during acute infection and often disappear or fall in titre	Only subsets underwent repeat testing. Limited power for rare later autoimmune liver disease	Good evidence for transient autoimmune-like serology. Limited evidence for progression
Taeschler et al. (2022) Jernbom et al. (2024) *SARS-CoV-2*	Longitudinal sampling. Jernbom included true pre-infection samples	Some autoantibodies arise after infection and may persist	Research assays are not equivalent to routine diagnostic serology. Autoimmune diagnoses were not the primary endpoint	Strong temporal evidence for seroconversion, weaker evidence for clinical autoimmune transition
Ding et al. (2024) Dor et al. (2025) *EBV/CMV*	Contemporary clinical cohorts	ANA positivity can accompany acute infection	Cross-sectional or clinically triggered testing. No true baseline	Detection is supported. Induction and long-term meaning remain uncertain
Vo et al. (2020) Chatterjee et al. (2024) *Dengue*	Protein-array and routine ANA approaches	Broad autoreactivity and ANA positivity have been reported	Small or clinically selected cohorts. Assay-dependent results. Limited longitudinal follow-up	Biological or serological association, not proof of later systemic autoimmune disease
Rivera-Correa et al. (2019) Davies et al. (2023) *Zika*	Comparison of Zika-associated GBS with infected controls	Peripheral nerve and ganglioside-reactive antibodies are enriched in GBS	Organ-specific complication rather than systemic autoimmune disease	Supports immune-mediated neuropathy as a boundary case, not a general systemic pathway

3. From a positive autoantibody to disease: what can the evidence establish?

Chronology is the first obstacle. In many reports, the initial autoimmune sample is obtained only after infection has produced a rash, polyarthritis, thrombosis, hepatitis or another feature that prompts testing. If there is no earlier sample, the antibody may be new, pre-existing or intermittently detectable. This matters particularly for ANA, given its background prevalence in the general population (Dinse et al., 2022). A positive sample collected after infection has begun therefore establishes timing of detection, not timing of induction.

Who gets tested is equally important. Broad rheumatological panels are not routinely ordered in uncomplicated viral illness, so patients arriving in rheumatology clinics have already passed through a clinical filter. The B19 literature makes the effect visible. D'Onofrio et al. (2024) reported ANA in 21 of 38 tested patients referred to rheumatology, whereas Hassan et al. (2024) found ANA and RF in 13% of a small hospital cohort. Those percentages describe different clinical pathways and, in both studies, not every marker was measured in every participant. Treating them as directly comparable prevalence estimates would erase the selection process that produced them.

Follow-up does not automatically solve the problem. A prespecified repeat sample from both recovered and symptomatic participants can estimate persistence reasonably well; routine follow-up often cannot. In HEV, repeat serology was available for only a subset of initially positive patients (Terziroli Beretta-Piccoli et al., 2018; Gui et al., 2021). Chikungunya cohorts have a different but related problem: long-term rheumatology follow-up is excellent for describing chronic disease among those still symptomatic, yet it cannot by itself tell us how often chronic disease occurs in all infected individuals. Loss to follow-up is therefore not a neutral event.

There is also a tendency to let the laboratory endpoint stand in for the clinical one. The two may diverge. ANA can remain positive after complete recovery, while persistent synovitis may occur in a patient who never

develops ACPA. In HCV-associated cryoglobulinaemia, immune markers can remain abnormal after sustained virological response even when symptoms improve, and vasculitis may relapse despite viral clearance (Fayed et al., 2022; Ferri et al., 2026). Longitudinal studies are most informative when serology, symptoms, examination findings, organ injury and formal diagnosis are reported separately.

Assay choice further complicates comparison across studies. ANA by indirect immunofluorescence, antigen-specific immunoassays, multiplex platforms and proteome-wide discovery assays do not measure equivalent signals. Wang et al. (2021), for example, identified broad functional autoreactivity during COVID-19 using high-throughput methods, whereas Taeschler et al. (2022) examined autoantibodies closer to those used in routine systemic rheumatology. Both approaches are relevant, but a positive result on one cannot be interpreted as though it were the same event as a positive result on the other. Dengue studies illustrate the same issue from another direction: Chatterjee et al. (2024) found substantially different positivity rates by ANA immunofluorescence and line immunoassay in the same clinical setting.

Some apparently simple percentages are difficult to interpret because the denominator is unclear. 'Fifty per cent ANA positive' may mean half of an entire infected cohort, half of a symptomatic subgroup, or half of the patients in whom ANA happened to be ordered. B19 studies often have different denominators for different antibodies; HEV cohorts tend to have more complete initial serology but much less complete repeat testing. For a longitudinal question, both denominators matter: who entered the study, and who contributed a sample at the relevant time point. If retesting preferentially captures patients who remain unwell, persistence will be over-represented.

The cleanest design is uncommon but conceptually straightforward: a negative pre-infection sample, seroconversion on the same assay, repeat testing after recovery, and an independently documented clinical outcome. Such a study can address induction, persistence and disease in sequence. Most viral literatures do not contain all four elements. SARS-CoV-2 is unusual because biobanks and occupational cohorts occasionally provided genuine pre-infection samples; for many other infections, observation begins only after illness is established.

Classification adds another source of error. Weighted criteria are useful for constructing research cohorts, but several items that contribute to SLE or APS classification - fever, cytopenia, arthritis, low complement or thrombosis - can also occur during infection. A patient may therefore cross a numerical threshold during the acute illness and no longer do so a few weeks later. The issue is not that classification criteria are defective. It is that attribution and longitudinal reassessment matter when a competing explanation is present. B19 and infection-associated aPL positivity make this particularly obvious.

Time horizon is another source of inconsistency that is easy to miss when studies are compared side by side. A positive antibody at hospital discharge, at three months and at one year may represent very different biological situations, yet all are sometimes described simply as 'persistent'. The same applies to clinical outcomes. Arthritis lasting six weeks after B19 infection and a new rheumatological diagnosis confirmed a year later should not be treated as equivalent evidence of disease transition. For this review, longer follow-up is most informative when the same laboratory and clinical endpoints are reassessed rather than reconstructed from routine records.

4. What the evidence shows across viral infections

4.1. Parvovirus B19: convincing mimicry, uncertain transition

B19 is a useful place to start because the mimicry can be clinically convincing. Adults may develop abrupt symmetric polyarthritis together with rash, fever, cytopenia, low complement, ANA, RF and occasionally anti-dsDNA antibodies (Arvia et al., 2024). In the multicentre series reported by Dollat et al. (2018), 25 adults had extra-haematological manifestations and frequent immunological abnormalities. Repeat autoantibody testing was available in only a small subgroup, but most of those retested became negative within one or two months. The observation is compatible with transient autoreactivity; the denominator is too small to say how often persistence occurs.

The 2024 rheumatology-referred cohort reported by D'Onofrio et al. is more striking but also more vulnerable to selection. ANA was present in 21 of 38 tested patients and anti-dsDNA in 10 of 26, and many patients met SLE or RA classification thresholds during the acute episode. Most symptoms nevertheless resolved within six weeks. These data show how classification criteria can be misleading when applied to an illness that already generates fever, joint inflammation, cytopenia, and complement abnormalities. They do

not show that more than half of unselected adults with B19 infection develop ANA or that temporary classification equates to disease.

Hassan et al. (2024) found lower seropositivity in 23 hospitalised adults, with ANA and RF each reported in 13%. Most rheumatic manifestations resolved, although one patient developed SLE during 6-12 months of follow-up. Negri et al. (2026) subsequently studied 39 patients who presented to tertiary rheumatology or immunology care with immune-mediated manifestations during the 2024 outbreak. Autoantibodies were detected in 17 of 39 patients (44%); follow-up serology was available for 11 of those 17, and only two remained positive after a median of five months. Five of 39 patients (13%) received a new autoimmune rheumatic diagnosis that was confirmed on further follow-up. That proportion cannot be read as population incidence because the cohort was deliberately enriched for immune-mediated presentations and also included patients with pre-existing autoimmunity. Taken together, the contemporary studies provide strong evidence for short-term mimicry but much weaker evidence for frequent progression in unselected B19 infection. The initial antibody panel does not identify the clinically relevant minority by itself; persistent synovitis, active urinary abnormalities, recurrent cytopenia, serositis, or evolving organ disease changes the probability of an independent autoimmune process, whereas rapid spontaneous resolution lowers it.

The different ANA frequencies reported by D'Onofrio and Hassan are less contradictory than they first appear. One cohort began after referral to rheumatology because B19 resembled SLE or RA; the other started with hospitalised adults with acute infection. Negri et al. then selected yet another population - patients seen in tertiary rheumatology or immunology care for immune-mediated manifestations. Read in that order, the studies say more about referral context than about a single underlying ANA prevalence. They also leave the population risk of durable systemic autoimmune disease imprecise.

There is also an important asymmetry in what these cohorts can tell us. They are well suited to identifying the small number of patients whose illness remains clinically suspicious, because that is exactly who is referred and followed. They are poorly suited to estimating how often B19 causes autoimmune disease in the infected population as a whole. The 5/39 new diagnoses reported by Negri et al. (2026) are therefore clinically important but epidemiologically non-transferable. The result identifies a subgroup that deserves attention; it does not support a 13% post-B19 autoimmune disease risk outside a tertiary referral setting.

4.2. Chikungunya: persistent inflammation without a uniform RA pathway

Chikungunya is informative for a different reason. Here, the inflammatory phenotype may persist long after the acute infection even when a disease-specific antibody never appears. Meta-analyses from 2016 and 2018 reported substantial chronic musculoskeletal morbidity, although estimates varied markedly with case definition and follow-up (Rodríguez-Morales et al., 2016; Paixão et al., 2018). Hossain et al. (2022) later described a mixture of undifferentiated arthritis, spondyloarthritis and RA among patients with chronic manifestations, and a 2025 systematic review reached a similar conclusion about the heterogeneity of post-chikungunya inflammatory disease (de Souza et al., 2025).

The autoantibody data argue against a simple model in which persistent post-chikungunya inflammation routinely becomes seropositive RA. Guillot et al. (2020) reassessed a selected subgroup 13 years after the Reunion Island outbreak and observed persistent joint symptoms without ACPA seroconversion. Ansah-Dico et al. (2025) also found that RF, anti-CCP, ANA, and other autoantibodies did not explain relapse severity, pain, or disability. These studies do not make RA serology irrelevant in an individual patient; rather, they show that chronic chikungunya disease cannot be sorted reliably by a single antibody profile. Objective synovitis and its evolution remain more informative than persistent pain or an isolated weak positive test.

Outcome labels account for part of the apparent inconsistency in this literature. Chronic arthralgia, examination-confirmed synovitis and fulfilment of RA classification criteria overlap, but they are not the same endpoint. A paper that groups them under 'chronic inflammatory rheumatic disease' will appear to show more progression than a study that requires a specific RA phenotype. The 13-year Reunion Island follow-up is valuable precisely because it separates persistent symptoms from ACPA seroconversion, although its selected follow-up population prevents a population-level estimate.

This matters when RA is used as the endpoint. Persistent symmetric small-joint synovitis, a rheumatologist's diagnosis of RA and fulfilment of classification criteria are related observations, but they are not interchangeable, especially after an infection known to cause prolonged inflammatory arthritis. In a conventional RA cohort, ACPA has substantial diagnostic and prognostic value. After chikungunya, its absence does not make ongoing synovitis trivial, but it weakens the argument for a uniform transition to the usual seropositive RA pathway. The phenotype must be followed rather than inferred from the antibody panel.

4.3. EBV, CMV, and HIV: seropositivity with limited temporal resolution

The EBV and CMV data are much less informative about induction. Ding et al. (2024) found an association between EBV markers and ANA positivity in a cross-sectional hospital population, while Dor et al. (2025) described ANA positivity in selected patients admitted with acute EBV or CMV infection. Neither study knew whether ANA had been present beforehand. The appropriate conclusion is modest: ANA may be detected during these infections, but the incidence of new ANA and its long-term predictive value remain uncertain.

CMV-associated thrombosis illustrates a related problem. A 2018 systematic review found published cases of venous thrombosis in immunocompetent adults with acute CMV, and a later narrative review remained cautious about the rarity and heterogeneity of the presentation (Bertoni et al., 2018; Schattner, 2024). If aPL are present, they should not be used retrospectively to make the thrombosis 'autoimmune'. APS still depends on the antibody profile, persistence and the wider clinical history.

HIV is included mainly because it lowers the specificity of isolated rheumatological findings. Immune dysregulation, opportunistic infection, antiretroviral treatment and immune reconstitution can all contribute to musculoskeletal or vasculitic presentations (Akram et al., 2024; Gopal et al., 2025). Recent longitudinal data linking a newly detected autoantibody to a later systemic autoimmune diagnosis are sparse. It is therefore more useful here as a setting in which interpretation is difficult than as evidence for a well-defined infection-to-autoimmunity pathway.

4.4. HCV-associated cryoglobulinaemia: when infection and immune disease are part of the same process

HCV is not simply another example of transient seropositivity. In mixed cryoglobulinaemic vasculitis, infection, B-cell activation, immune-complex formation and organ disease are biologically connected. The clinically useful question is therefore different: not whether a positive RF or cryoglobulin is a false-positive consequence of infection, but how virological control, serological activity and clinical vasculitis relate to one another over time.

Within HCV-associated mixed cryoglobulinaemic vasculitis, mixed cryoglobulins, RF, low C4, purpura, arthralgia, neuropathy, and renal disease may form one coherent immune-complex syndrome (Moretti et al., 2024). RF derives meaning from the phenotype in which it occurs, not from positivity in isolation. Cryoglobulin testing is technically vulnerable, however, and pre-analytical handling can materially affect the result (Kolopp-Sarda & Miossec, 2022). An isolated low-level cryoglobulin without compatible organ disease is not equivalent to cryoglobulinaemic vasculitis.

The direct-acting antiviral era created a useful natural experiment by separating viral clearance from subsequent immune behaviour. Gragnani et al. (2016) and Bonacci et al. (2017) documented marked clinical improvement after viral eradication, while immunological recovery was less uniform. Later cohorts showed that sustained virological response does not guarantee permanent immunological remission. Fayed et al. (2022) reported relapse of cryoglobulinaemic vasculitis after viral cure, and Ferri et al. (2026) found persistent cryoglobulins to be associated with more severe complications during long-term follow-up. Lauletta et al. (2024) likewise observed that autoantibodies and cryoglobulins changed after treatment but did not all follow the same trajectory; autoimmune liver disease was newly recognised in a small number of patients. HCV demonstrates why virological, serological, and clinical endpoints need to be analysed separately even when a causal relationship between infection and immune disease is well established.

The antiviral studies are also a reminder that removal of a trigger is not a diagnostic test. Clinical improvement after viral eradication supports a causal role for HCV, but persistent cryoglobulinaemia does not necessarily mean active vasculitis, and incomplete immunological remission does not invalidate the original diagnosis. What makes the later cohorts informative is that they follow virological, serological and clinical trajectories separately. They still lack a pre-infection antibody baseline, but for this disease model that limitation is less important than it is in studies trying to prove de novo autoantibody induction.

4.5. Hepatitis E virus: a direct test of autoimmune-looking liver serology

HEV speaks directly to the diagnostic problem because acute viral hepatitis can look serologically like autoimmune hepatitis. Terziroli Beretta-Piccoli et al. (2018) found at least one autoantibody in 24 of 48 patients with acute HEV; ANA was present in 33%, SMA in 21% and ANCA in 14.6%. Follow-up serum was available for only seven autoantibody-positive patients. None developed AIH during the reported follow-up, but that small retested subgroup cannot exclude an uncommon later autoimmune liver disease.

Wu et al. (2020) provided a stronger prospective comparison. Among 198 patients with acute HEV, 50% had AIH-related autoantibodies; ANA was found in 37.4% and SMA in 22.7%. The titres and patterns were generally less typical of type 1 AIH than those observed in the AIH comparison group. Of 52 seropositive patients reassessed about a year later, 33 became negative and 19 remained positive at lower titres, with no new seropositivity emerging during follow-up. The predominant trajectory was transient or diminishing autoreactivity. The same study nevertheless reported two patients positive for ANA-H and SMA-AA who were ultimately diagnosed by the authors with type 1 AIH on the basis of serology and histopathology. In the detailed case, liver tests normalised with supportive hepatoprotective treatment rather than immunosuppression, and ANA/SMA titres fell during follow-up, which also illustrates how difficult attribution can be in acute HEV. The practical implication is not that HEV excludes AIH, but that acute infection substantially lowers the specificity of autoimmune liver serology at presentation.

Gui et al. (2021) reported autoimmune liver serology in 42.1% of 164 patients with acute HEV. Repeat testing was available for 27 of the 69 initially positive patients; ANA disappeared in 11 of 20 retested patients and AMA in four of five. One patient was subsequently diagnosed with primary biliary cholangitis after HEV clearance. This does not imply that HEV commonly causes chronic autoimmune liver disease. It does show why the minority with persistent cholestasis, hypergammaglobulinaemia, characteristic histology, or stable disease-specific serology cannot be dismissed simply because the illness began as viral hepatitis.

Across the three HEV cohorts, transient or diminishing autoimmune liver serology is the dominant pattern, but the follow-up is not complete enough to quantify rare outcomes with confidence. That distinction matters. These studies are strong evidence that acute HEV lowers the specificity of ANA and SMA for AIH at presentation; they are much less precise about the subsequent incidence of bona fide autoimmune liver disease.

A falling titre after viral clearance is reassuring, although not decisive by itself. Persistent high-titre, disease-concordant serology becomes more concerning when biochemical inflammation, hypergammaglobulinaemia or compatible histology persists as well. The rare diagnoses reported during follow-up are a useful counterweight to an overly simple 'infection-related false positive' interpretation. In practice, HEV changes the prior probability of AIH during the acute episode; it does not remove AIH from the differential diagnosis.

The two AIH diagnoses reported by Wu et al. (2020) deserve particular caution in this respect. They prevent the convenient conclusion that autoimmune serology during HEV is merely epiphenomenal, but they do not establish that HEV commonly initiates AIH. Attribution remains difficult because acute HEV itself can produce marked aminotransferase elevation and autoimmune-type serology, and in the detailed case the biochemical abnormalities resolved without immunosuppression while antibody titres fell. For a clinician, the useful signal is persistence of a coherent AIH phenotype - not the presence of ANA or SMA during the acute hepatitis alone.

4.6. SARS-CoV-2: what pre-infection samples add

SARS-CoV-2 generated a large autoantibody literature, but its main contribution to this question is not the number of reactivities reported. It is the availability, in some cohorts, of samples collected before infection. Wang et al. (2021) identified broad functional autoreactivity during acute COVID-19 using high-throughput methods. Taeschler et al. (2022), using autoantibody testing closer to routine clinical practice in 175 patients followed for up to one year, found overall ANA prevalence and patterns broadly similar to controls, although some paired results changed over time. The two studies measure different kinds of signal and should not be read as if they were interchangeable.

Jernbom et al. (2024) could address the baseline question more directly because pre-infection samples were available for part of the cohort and sampling continued for 16 months. New-onset autoantibodies were demonstrable, and some persisted, particularly after more severe disease. That is unusually strong evidence for infection-associated induction. It is weaker evidence for a subsequent systemic autoimmune diagnosis, because many reactivities came from broad research platforms and conventional rheumatological diseases were not the

primary clinical endpoint. In other words, the study closes the gap between detection and seroconversion more effectively than the gap between seroconversion and disease.

The aPL literature reaches a similar conclusion with more familiar assays. Ogrič et al. (2023) found aPL after SARS-CoV-2 infection, but levels, multiple positivity, and persistence were generally weaker than in APS, and only a minority remained positive at follow-up. Acute infection can reproduce part of an APS-like laboratory profile without reproducing the syndrome. This distinction is particularly important in patients with thrombosis, where the event itself requires standard assessment and aPL results gain diagnostic weight only when the clinical history, profile, and persistence support APS (Barbhaiya et al., 2023; Devreese et al., 2020; Smiyan et al., 2025).

Pre-infection sampling solves an important temporal problem, but not the question of pathogenicity. A newly detectable antibody may still have no validated disease threshold, especially when identified on a discovery platform. Persistence of such a signal is biologically interesting; it is not automatically equivalent to persistence of a clinical syndrome. This asymmetry in the SARS-CoV-2 data is important: induction is relatively well supported, routine progression to conventional systemic autoimmune disease is not.

These cohorts also highlight a problem that will become more common as discovery platforms become more sensitive. A study may convincingly show that infection changes the autoreactive repertoire without establishing that any individual reactivity has a validated diagnostic meaning. That is not a weakness of the laboratory work; it is a mismatch between a mechanistic endpoint and a clinical one. For the question posed here, the relevant downstream evidence would be prospective ascertainment of defined autoimmune diagnoses, ideally with the same participants followed after seroconversion. Such data remain far less developed than the serological literature itself.

4.7. Dengue and Zika as boundary cases

Dengue and Zika are deliberately kept at the margins of the review because they address a somewhat different biological problem. In dengue, experimental and clinical work has described autoreactivity against platelets, endothelial targets, coagulation proteins and other host antigens (Ghorai et al., 2024). Vo et al. (2020), using a protein-array approach, found increased IgM reactivity to numerous putative autoantigens and increased IgG reactivity to a smaller set, several involving coagulation or complement. That supports biological plausibility, but not a longitudinal transition to systemic autoimmune disease.

Routine serology can also be abnormal. Chatterjee et al. (2024) reported ANA by indirect immunofluorescence in 54.8% of 135 dengue-positive patients compared with 10.3% of dengue-negative controls, whereas line immunoassay positivity was much lower. The cohort lacked pre-infection samples and was clinically selected, so the difference does not establish incident connective tissue disease. Instead, the discordance between assays reinforces a broader point of this review: the diagnostic meaning of a positive autoantibody depends on method, context, and phenotype, not on binary positivity alone.

Zika provides stronger evidence for an organ-specific immune complication than for a systemic autoimmune pathway. Rivera-Correa et al. (2019) found broader anti-ganglioside reactivity in patients with Zika-associated Guillain-Barré syndrome (GBS) than in infected controls. Davies et al. (2023), in a larger comparison, likewise observed enrichment of peripheral nerve-reactive IgM and IgG among patients with Zika-associated GBS, although no single known antiglycolipid signature explained the syndrome. Reviews of Zika and dengue autoimmunity discuss molecular mimicry as a plausible mechanism (Zhou et al., 2025), but these data are best read as evidence of immune-mediated organ injury rather than as a route from viral infection to a conventional systemic rheumatic diagnosis.

For that reason, dengue and Zika are best interpreted as boundary cases rather than additional examples of the same pathway. They show that post-viral autoreactivity can accompany clinically important immune pathology without behaving like ANA-associated connective tissue disease or AIH-related serology. Treating dengue autoantibodies, Zika-associated nerve reactivity, B19 ANA and HCV cryoglobulins as one biological category would obscure more than it clarifies.

5. Discussion

5.1. When does a positive autoantibody support autoimmune disease?

No single percentage of autoantibody positivity answers the question posed in this review. What matters is whether the serological finding survives a series of increasingly demanding tests of interpretation. Was the antibody absent before infection? Does it remain detectable on a clinically interpretable assay? Does an objective inflammatory or organ phenotype persist, and is that phenotype still better explained by infection? B19 and HEV fail mainly at specificity during the acute illness; SARS-CoV-2 provides stronger evidence for true induction; HCV-associated cryoglobulinaemia is different again because the infection is already part of the disease mechanism.

The weakest claims come from studies that enter the story after the positive result has already been found. Cross-sectional EBV data, clinically triggered EBV/CMV testing and selected rheumatology cohorts can describe associations and mimics, but they cannot reconstruct a pre-infection baseline. HEV follow-up is more informative yet incomplete, and dengue or Zika studies often use mechanistic or organ-specific endpoints that are not equivalent to a new systemic autoimmune diagnosis. These differences in design matter more than the crude proportion of positive tests.

Classification criteria can compound the problem rather than resolve it. Acute infection may produce fever, cytopenia, arthritis, low complement or thrombosis - features that also contribute to SLE or APS classification. The 2019 EULAR/ACR SLE criteria include an attribution principle for this reason (Aringer et al., 2019), and current ACR methodology continues to distinguish classification from diagnosis (Johnson et al., 2026). A similar caution applies to APS: thrombosis during infection is clinically important, but one infection-associated aPL result is not enough to establish the syndrome (Barbhaiya et al., 2023; Devreese et al., 2020).

5.2. Implications for clinical interpretation and future studies

The clinical value of the same antibody changes over time. During an acute febrile illness or hepatitis, background positivity and infection-related immune activation reduce specificity. Weeks later, once the infection has resolved, persistent synovitis, nephritic urinary abnormalities, vasculitic neuropathy, inflammatory myopathy, continuing liver inflammation or recurrent thrombosis alter the pre-test probability. At that point, disease-concordant serology carries more weight. This is why a fixed schedule for 'repeat the panel in X weeks' is less useful than reassessment tied to the clinical course. If the patient has recovered, repeating broad panels rarely adds much. If objective disease persists, targeted retesting can be informative.

For future studies, temporal design is more valuable than simply adding more analytes. Pre-infection samples are ideal, but a prospective cohort recruited early in infection and retested at fixed intervals can still answer important questions. The same assay should be used throughout, reasons for testing should be recorded, and denominators should be reported for every marker at every time point. Matched non-infected controls are particularly useful for ANA, where background positivity is substantial.

Clinical outcomes also need to be separated more carefully. Persistent pain, a persistent antibody, examination-confirmed synovitis, organ injury and a formal autoimmune diagnosis are different endpoints. Combining them under labels such as 'persistent autoimmunity' makes the literature look more coherent than it is. Mechanistic findings require the same restraint. Molecular mimicry or host-antigen binding may explain how autoreactivity arises, but they do not by themselves establish a later clinical disease.

Several recent studies show that better inference is feasible even without a single standard design. DAA-era HCV cohorts follow immune disease after removal of the viral trigger; HEV cohorts repeat autoimmune liver serology after acute hepatitis; SARS-CoV-2 biobanks provide genuine pre-infection baselines; and B19 cohorts reveal how sharply referral setting changes the apparent frequency of autoantibodies. The useful question is not whether these studies are directly comparable, because they are not, but which link in the sequence each one actually measures.

One additional problem is incorporation bias. If the autoantibody being studied also contributes to the criteria used to define the later autoimmune outcome, the association between seropositivity and diagnosis can become partly circular. Longitudinal studies are more convincing when the endpoint includes evidence independent of the antibody itself - persistent examination findings, imaging, urinalysis, biopsy or treatment-requiring organ disease. This issue is particularly relevant when an acute infection temporarily fulfils SLE or RA classification criteria.

6. Conclusions

A positive autoantibody after viral infection is the beginning of an interpretation, not the end of one. In most contemporary studies, at least one step in the sequence remains uncertain: whether the antibody was genuinely new, whether it persisted, or whether a compatible clinical disease persisted with it. B19 and HEV show how convincing autoimmune-like serology may regress as the infection resolves. SARS-CoV-2 demonstrates that true seroconversion can be documented when pre-infection samples exist, yet that result does not by itself establish a parallel rise in conventional autoimmune disease. HCV-associated cryoglobulinaemia occupies a different category because the infection and immune disease are mechanistically connected. Overall, post-viral autoreactivity is well supported; progression from a positive autoantibody to a new autoimmune disease is much less consistently demonstrated. The diagnosis becomes credible when the serology and the clinical course remain concordant after the acute infection is no longer an adequate explanation.

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Author contributions:
Conceptualization: Anna Gwóźdź-Broczkowska, Agata Kotłowska
Methodology: Anna Gwóźdź-Broczkowska, Julia Lenkiewicz, Aleksandra Izabela Masłowska
Formal analysis: Anna Gwóźdź-Broczkowska, Aleksandra Izabela Masłowska, Wiktoria Napiórkowska
Resources, data curation: Agata Kotłowska, Julia Lenkiewicz, Marcin Trzciński
Investigation: Gabriela Luba, Marcin Trzciński, Oliwia Lenkiewicz
Writing – original draft: Anna Gwóźdź-Broczkowska, Marcin Trzciński
Writing – review & editing: Wiktoria Napiórkowska, Gabriela Luba

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