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Case Report

COVID-19-associated Autoimmune Hepatitis: A Case Report and Literature Review

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Abstract

Background: Autoimmune hepatitis (AIH) is a chronic inflammatory disease with unclear etiology. Various vaccines have been reported as triggers of AIH. Recently, with the ongoing COVID-19 pandemic and widespread vaccination worldwide, several cases of COVID-19 vaccination-associated (CA) AIH, occurring with or without COVID-19 infection, have been reported. ***Case presentation:*** In this report, we describe a 66-year-old female who developed biopsy-proven acute-onset autoimmune hepatitis after receiving four doses of the COVID-19 vaccine and experiencing one COVID-19 infection in 2022. The patient was immediately treated with prednisone. Her liver enzymes gradually decreased to the normal range after treatment. In addition, we reviewed 20 cases of CA-AIH reported from multiple countries. The summarized data showed that CA-AIH and classical AIH share some clinical, serological, and histopathological features, such as female predominance and a middle-aged distribution. All patients had some positive circulating autoantibodies, including anti-nuclear antibody and/or positive anti-smooth muscle antibody. Histologically, CA-AIH showed a more acute onset compared to classical AIH, which typically presents with more chronic hepatitis. ***Conclusions:*** This case report provides additional evidence supporting an association between COVID-19 vaccination and/or infection and AIH, suggesting more causality than coincidence.

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Introduction

Autoimmune hepatitis (AIH) is a complex disease with unclear etiology. Multiple factors are believed to be involved in the etiology, including immunologic, genetic, infectious, and environmental factors. AIH is considered an autoimmune-mediated chronic progressive liver disease affecting all ages, genders, and ethnic populations.[1] Commonly, middle-aged females have the highest risk for developing AIH, with a male-to-female ratio of 1:4 to 1:6. The current concept of the immunopathogenesis of AIH is that autoreactive T cells break through self-tolerance to hepatic autoantigens, triggering an immune response that results in liver damage such as necroinflammation and fibrogenesis.[2] Therefore, detectable autoantibodies such as anti-nuclear antibody (ANA), anti-smooth muscle antibody (ASMA), or anti-liver kidney microsome antibody, and elevation of serum immunoglobulin G, are all characteristic serological features of AIH. Histologically, the key features of AIH are portal inflammation with mainly lymphoplasmacytic or lymphocytic infiltration and interface hepatitis.[3]

The clinical presentation of AIH is heterogeneous, ranging from mild subclinical symptoms to severe acute-onset fulminant type. The vague symptoms, plus the lack of signature diagnostic markers, make early definitive diagnosis of AIH challenging. The current diagnostic criteria are based on combined laboratory, histologic, and clinical findings, which often lead to a delayed diagnosis of AIH. Acute-onset AIH does occur, but it is often overlooked. Therefore, timely diagnosis of AIH is crucial and life-saving, because early treatment with immunosuppressive therapy can prevent the progression to liver failure and cirrhosis.[4]

Recently, emerging acute-onset autoimmune hepatitis-like cases have been reported worldwide following a COVID-19 vaccination and/or a COVID-19 infection.[5,6] With the broad application of the COVID-19 vaccine and the high incidence of COVID-19 infections, the relationship between AIH and COVID-19 vaccination with/without infection has emerged as a new topic of interest. In this study, we present a case of a 66-year-old female patient with acute-onset AIH after receiving four COVID-19 vaccines and a COVID-19 infection in 2022. Meanwhile, we also reviewed 20 cases collected from 14 countries and summarized the clinicopathological features. The purpose of this case report and literature review is to explore the relationship between COVID-19 with/without vaccination and autoimmune hepatitis.

Case presentation

The patient is a 66-year-old female with a past medical history of diverticulitis and gastroesophageal reflux disease. She had markedly elevated alkaline phosphatase (ALP), alanine transaminase (ALT), and aspartate transaminase (AST), along with increased bilirubin and international normalized ratio, after an asymptomatic COVID-19 infection in October 2022 (Table 1). In addition, she had received four injections

Keywords: Autoimmune hepatitis; COVID-19; Vaccination; Liver enzymes; Acute onset; Antinuclear antibody.

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Table 1. Laboratory parameters of the patient before and after treatment (one month vs six months)

Parameters	Pre-treatment		Post-treatment		Reference range
	1st	2nd	1 m	6m	
ALT (U/L)	1,600	1,116	53	25	7 - 35 U/L
AST (U/L)	1,500	972	28	23	10 - 40 U/L
ALP (U/L)	220	230	82	75	30 - 110 U/L
Bilirubin (total, mg/dL)	7.6	N/A	1.3	0.6	0.2 - 1.4 MG/DL
ANA	+ (1:320)		N/A	N/A	<1: 40 Negative

ALP, alkaline phosphatase; ALT, alanine aminotransferase; ANA, antinuclear antibody; AST, aspartate aminotransferase.

of the COVID-19 vaccine (Moderna 100 mcg/0.5 mL) on January 8, 2021; February 25, 2021; November 1, 2021; and April 1, 2022, respectively. The computed tomography image of her abdomen and pelvis was unremarkable, other than cysts on her liver. Meanwhile, a liver biopsy was performed and showed florid acute hepatitis with preserved architecture. Portal tracts revealed mixed inflammatory infiltration composed of lymphocytes, plasma cells, some histiocytes, neutrophils, and a few eosinophils. The interlobular bile duct showed no evidence of cholangiocytic injury. The lobular tissue revealed an inflammatory infiltrate of lymphocytes and histiocytes with a few neutrophils. Trichrome stain showed no significant portal fibrosis. Immunohistochemical stains for Herpes simplex viruses 1 & 2, cytomegalovirus, Epstein-Barr virus, COVID-19, adenovirus, and hepatitis B surface and core antigens were all negative. Copper stain was also negative. The findings were nonspecific, with possible etiologies including infection, drug, medication, toxin-induced injury, and immune-mediated injury. The patient did not receive any treatment because her total bilirubin gradually declined and eventually normalized.

However, on March 8, 2023, routine follow-up blood tests showed elevated liver function tests (LFTs) (Table 1). A second liver biopsy was performed and showed severe portal and lobular inflammation with dense and diffuse mixed inflammation, focal bridging necrosis, and interface hepatitis. The inflammatory cells included reactive lymphocytes, plasma cells, neutrophils, and rare eosinophils. Mild portal tract injury with occasional intraepithelial lymphocytes was seen. Zone 3 perivenular inflammation and hepatocyte dropout with endothelial cell damage were also present, along with scattered acidophilic bodies. A trichrome stain showed no significant increase in fibrosis. The predominant T-cell infiltration in both the sinusoidal and portal tracts was highlighted by a CD3 marker. Only small clusters of B cells were found in the portal tracts by the CD20 marker (Figs. 1 and 2). Immunostains for adenovirus, cytomegalovirus, Herpes simplex virus, and Epstein-Barr virus (EBER ISH) were negative. The findings were consistent with an acute hepatitis pattern, with autoimmune hepatitis at the top of the differential diagnosis, despite the serologic workup initially showing negative ANA and ASMA. A later workup revealed positive ANA (1:320) with a homogeneous nuclear pattern, confirming the diagnosis of autoimmune hepatitis. The patient is doing well with normalized LFTs after being immediately prescribed immunosuppression with prednisone (Table 1).

Discussion

The first case report regarding COVID-19 vaccination-associated AIH (CA-AIH) was published by Dr. Bril in 2021.[5] With the COVID-19 pandemic peak in 2021–2022 and the massive application of the COVID-19 vaccination, robust similar cases had been reported.[6,7] Thus, the association between AIH and COVID-19 vaccination/infection has elicited much attention. Dr. Bril raised a question about the causality or casualty between the links. In order to clarify this relationship, we collected and analyzed 20 cases reported during 2021–2022 from 14 countries (references see Table 2).[5–24] The detailed clinical and pathological features of 20 cases are listed in Table 2.[5–24] In this series, most patients received COVID-19 vaccines, including both activated mRNA vaccines and inactivated types, except for one unvaccinated patient with a COVID-19 infection (Case 20). Four patients had both a COVID-19 vaccination and infection, including our case. Among the total 22 patients from 20 case reports, the female patients were predominant, with a mean age of 55.3 ± 19.3 years (ranging from 20 to 85 years old). The latency time after vaccination (1st, 2nd, or 3rd dose) ranged from two to 56 days, with the most common latency time of seven days and a mean time of 16 days. Patients exhibited a range of symptoms, from no significant symptoms to common gastrointestinal symptoms such as jaundice, abdominal pain, dark urine, pruritus, or fatigue, etc. For other diseases present in these patients, five patients (23%) had thyroid disease (Hashimoto thyroiditis or hypothyroidism), five patients (23%) had hypertension, two patients (9%) had diabetes, two (9%) had suffered previous liver diseases (hepatitis C or primary biliary cholangitis), one had Sjögren disease. Positive ANA was detected in 68% of patients, mostly with homogeneous or speckled patterns, which are similar to the classical AIH (c-AIH) ANA patterns, and ASMA was found in 32% of patients. Both ALT and AST were significantly elevated (ALT: 1248 ± 627 U/L; AST: 1133 ± 486 U/L), and ALP and gamma-glutamyltransferase were mildly elevated (ALP: 185 ± 101 U/L; gamma-glutamyltransferase: 298 ± 135 U/L). Other positive antibodies were occasionally detected, such as anti-dsDNA, anti-actin antibody, anti-neutrophil cytoplasmic antibody, anti-soluble liver antigen, anti-liver cytosol antibody, HLA-DR4, and DR3, etc. A liver biopsy was performed on all patients due to the presence of markedly increased liver enzymes. Different from the c-AIH, which presents mostly with chronic hepatitis and advanced fibrosis, the majority of CA-AIH cases presented with an acute-onset hepatitis pattern with minimal to mild fibrosis. The histologic findings were compatible with AIH with severe acute hepatitis presentation in 19 patients. The liver biopsy showed marked portal and lobular inflammation with prominent lymphoplasmacytic infiltration. One study showed that the infiltrative lymphocytes were predominantly CD8-positive T cells.[13] Prominent interface hepatitis and/or rosette formation were seen in 90% of the cases. Necrosis was identified from scattered acidophilic bodies, spotty necrosis, to diffuse necroinflammatory foci and even bridging necrosis. Centrilobular necrosis was seen in seven cases. Among the 22 patients, 18 cases (82%) had minimal to mild fibrosis,

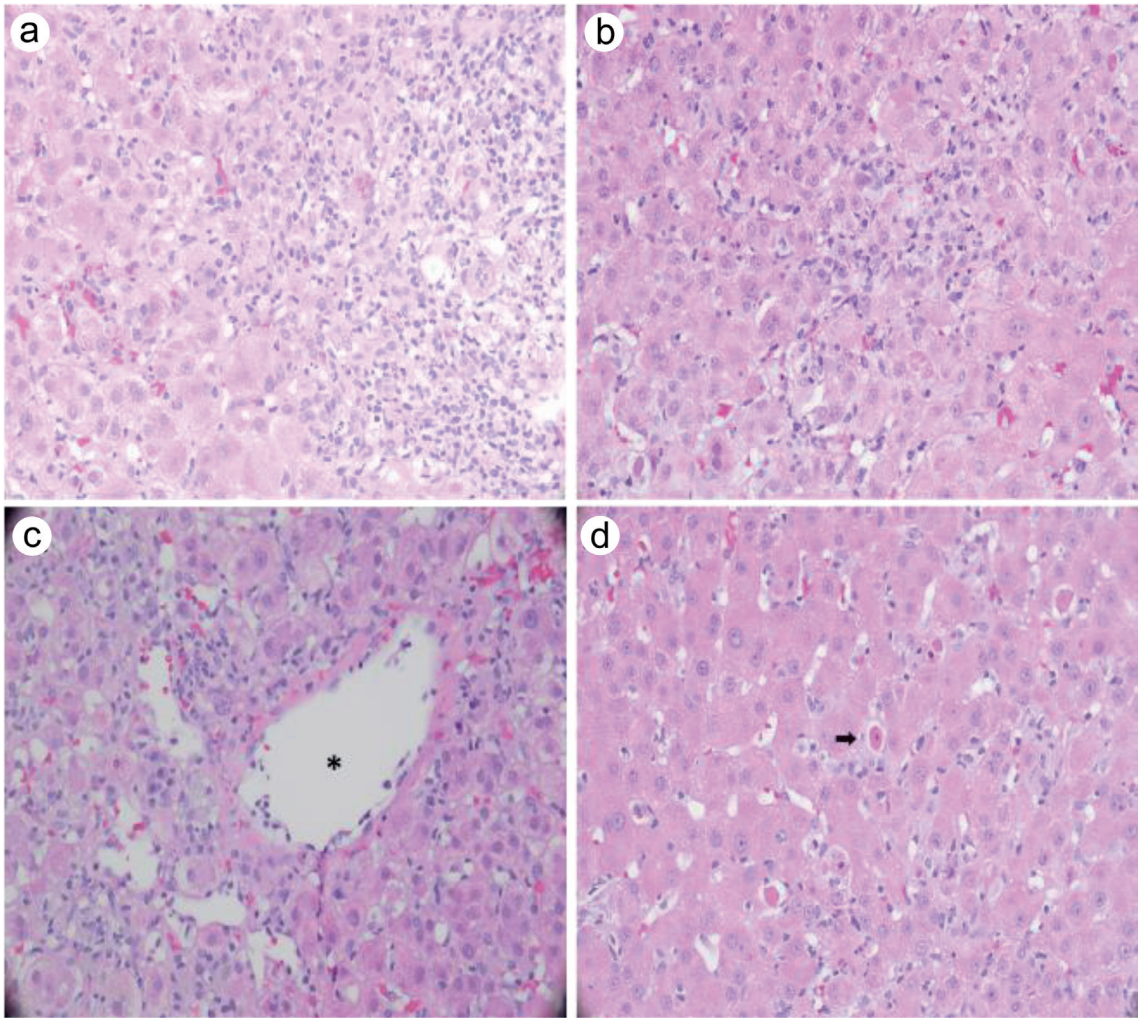

Fig. 1. Histological morphological features of liver biopsy. (a) Portal inflammation with prominent lymphoplasmacytic infiltration and interface hepatitis (×200). (b) Lobular inflammation with lymphoplasmacytic infiltration (×200). (c) Centrilobular (*) inflammation and necrosis (×200). (d) Acidophilic bodies (arrow) in the lobules (×200).

consistent with an acute-onset pattern. Only four cases (18%) showed chronic features, ranging from focal bridging fibrosis to advanced fibrosis (cirrhosis). After diagnosis, most patients were immediately treated with steroids and responded well; however, two patients developed fulminant liver failure and eventually died.

Based on the analyzed data collected from 20 cases, CA-AIH shares common features with c-AIH as follows: female predominance with mostly middle-aged distribution, positive ANA and/or positive ASMA. The serological tests showed a predominantly hepatocellular injury pattern. Histological features demonstrated an acute hepatitis pattern with prominent portal and lobular inflammation, interface hepatitis, and necrosis to varying extents—ranging from acidophilic bodies to spotty necrosis and even bridging necrosis. Centrilobular necrosis was also present. In contrast to c-AIH, which presents with chronic hepatitis, most CA-AIH cases in this series were discovered at an early stage. In the current case, the patient had an asymptomatic COVID-19 infection in 2022 and received four COVID-19 vaccinations in 2021–2022. Her elevated LFTs were detected after the COVID-19 infection in 2022. After that, her LFTs trended down to normal limits without any treatment. The liver biopsies showed an acute hepatitis pattern featuring prominent lymphoplasmacytic infiltration and interface activity. The histological features suggested autoimmune hepatitis and were confirmed with a later positive ANA test. Her liver enzymes returned to a normal range with prompt steroid treatment. Throughout the entire period, she had minimal symptoms. As shown in the 20 cases, the symptoms varied from none to fever, nausea, abdominal pain, and jaundice. Since ANA and ASMA markers are usually negative in the early stage, this may cause a delayed definitive diagnosis and necessary effective treatment in CA-AIH.

Both the 20 cases and our case described an acute-onset presentation of autoimmune hepatitis. Autoimmune hepatitis is a complex autoimmune disease with an uncertain mechanism. It is thought to be a T-cell–mediated autoimmune disease secondary to a failure of immune tolerance triggered by environmental factors and genetic predisposition. The various environmental factors include infection, medications, toxins, etc. In particular, vaccines are also considered potential triggers of AIH. Vaccine-induced autoimmune liver disease has been reported in the past, including hepatitis B, influenza,

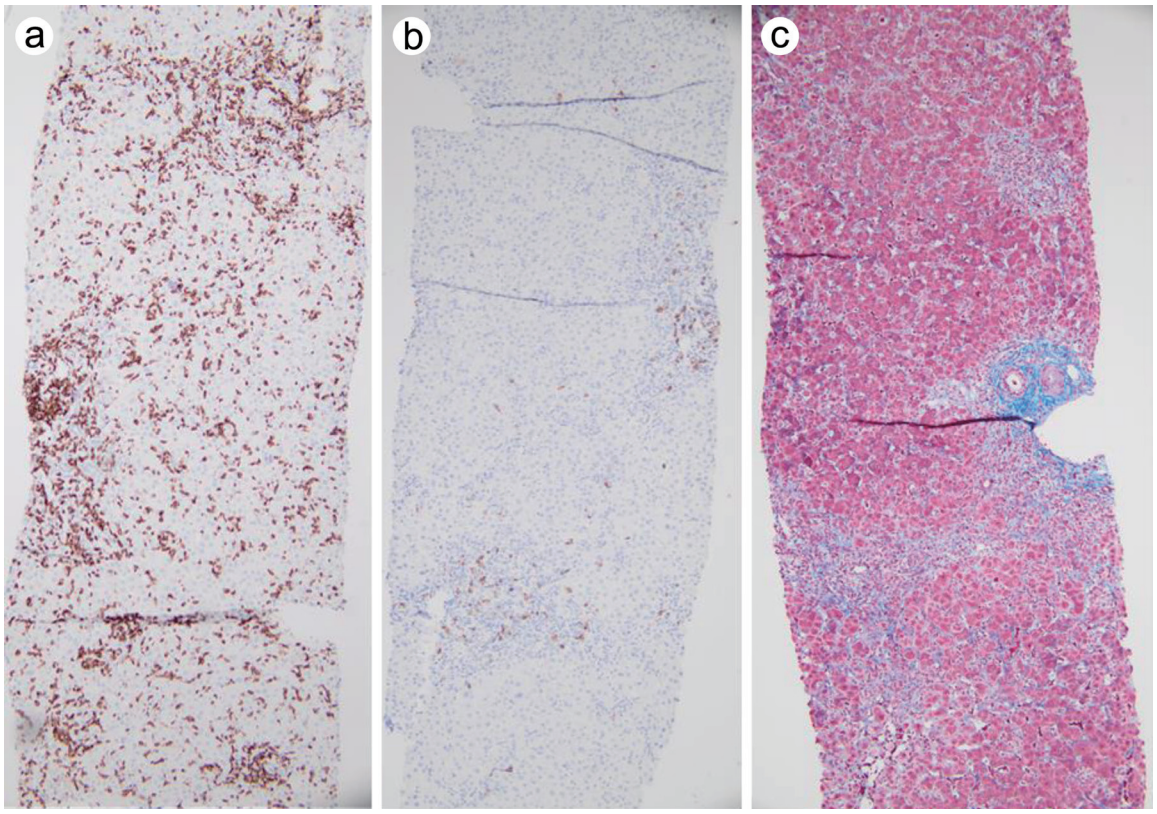

Fig. 2. Immunostains and trichrome stain of liver biopsy. (a) CD3 (×100); (b) CD20 (×100); (c) Trichrome stain highlights portal fibrosis and collagen deposition in the bridging necrotic area. No significantly increased fibrosis.

and HPV vaccines. The key mechanism of vaccine-induced AIH is believed to be molecular mimicry, which is caused by immune cross-reactivity due to structural similarity between vaccine and host antigens. The second mechanism considered in vaccine-induced AIH is called bystander activation, which is intense stimulation of the innate immune system caused by adjuvants. The third mechanism is due to epitope spreading, which is an immune response to endogenous epitopes by release of self-antigens during the inflammatory process.[25,26] The immune response is mediated through activated cytotoxic CD8-positive T cells, including spike-specific CD8-positive T cells.[11] In addition, studies showed that COVID-19 infection may also have a triggering effect on various autoimmune diseases through molecular mimicry and hyperstimulation of the immune system.[27]

In another study of 87 patients collected from 18 countries, SARS-CoV-2 vaccination-induced liver injury showed female dominance, middle age, a hepatocellular injury pattern, and 67% of patients with positive ANA and 18% with positive ASMA. The overall clinical and serological features were similar to our analysis of 20 cases.[28] In addition, AIH should be considered even without significant positive ANA, because the antibody was positive in only 68% of patients in our study, had delayed expression in our case, and was even negative in another report.[29] As the first autoantibodies to be associated with c-AIH, studies found that the common immunofluorescence patterns of ANA in AIH are speckled or homogeneous.[30,31] These patterns were also demonstrated in the majority of the reported cases and in our case. Although not specific, these ANA patterns provide more evidence of COVID-19–induced AIH.

Since the first case report of AIH following COVID-19 vaccination, the link between AIH and COVID-19 has been questioned. Although 20 cases comprise a small-scale study, it sheds light on a potentially novel clinical entity. As we know, the diagnosis of AIH is complex and challenging. Due to variable symptoms and possibly delayed ANA levels, the accurate diagnosis of CA-AIH is even more difficult. With the ongoing increase in COVID-19 cases internationally and the widespread use of the vaccine, it is necessary for clinicians to consider CA-AIH in the differential diagnosis of patients with nonspecific acute hepatitis, especially in the middle-aged female population. Even without a definitive diagnosis, long-term monitoring of this population is needed in order to avoid a worse disease prognosis. In addition, early diagnosis of CA-AIH will most benefit patients due to their positive response to steroid therapy. The benefits of vaccination are significant in fighting the pandemic. Although adverse effects of vaccination are rare, addressing them with caution will be best for future patient care.

Conclusions

In the current report, we presented a case of a 66-year-old female patient with acute-onset AIH, confirmed by histological and serological features following COVID-19 vaccination and infection. Taken together with the analysis of 20 similar cases from other reports, we provide stronger evidence to support the relationship between autoimmune hepatitis and COVID-19 infection and/or vaccination. More importantly, our patient responded well to steroid treatment administered immediately after diagnosis, which helped avoid a worse

Table 2. Summary of clinicopathological parameters from 20 case reports

Author	Year, Country	Age (y)	Gender	Vaccine (interval)	Infection	LFT	Autoantibodies	Liver biopsy: Inflam/Necrosis/Fibrosis
Bril *et al*.[5]	2021, USA	35	F	Pfizer (13 d)	No	ALT: 2,001; AST: 754; ALP:170	ANA: 1:1,280 (homogenous); dsDNA: 1:80; ASMA: –	Yes/Yes/No
Vuille-Lessard *et al*.[6]	2021, Switzerland	76	F	mRNA-1273 SARS-CoV-2 (2–3 d)	Yes	ALT: 579; AST: 811; ALP:124	ANA: 1:1,280 (homogeneous); ASMA: 1:1,280; ANCA: >1:1,280	Yes/Yes/No
Ghielmetti *et al*.[7]	2021, Switzerland	63	M	mRNA-1273 SARS-CoV-2 (7d)	No	ALT: 1,038; AST: 1,127; ALP: 192; GGT: 536	ANA: 1: 640 (speckled); AMA+	Yes/Yes/No
Londoño *et al*.[8]	2021, Spain	41	F	mRNA-1273 SARS-CoV-2 (7d)	No	ALT: 1,312; AST: 993; ALP: 190; GGT: 209	ANA: 1:80; ASMA: 1:40; ASLA: +; ALC: +	Yes/Yes/No
Clayton-Chubb *et al*.[9]	2021, Australia	36	M	Oxford-AstraZeneca (26 d)	No	ALT: 1,774; AST: 633; ALP: 118; GGT: 136	ANA: 1:160 (Speckle)	Yes/Yes/No
Garrido *et al*.[10]	2021, Portugal	65	F	Moderna-COVID-19 vaccine (14 d)	No	ALT: 1,092; AST: 1,056; ALP: 24; GGT: 329	ANA: 1:100 (speckled pattern)	Yes/Yes/No
Avci *et al*.[11]	2021, Turkey	61	F	BNT162b2 mRNA (30 d)	Yes	ALT: 455; AST: 913; ALP: 436; GGT: 292	ANA: 1:100 (weakly granular); ASMA: 1:100	Yes/Yes/No
Mathew *et al*.[12]	2022, India	20s	F	AstraZeneca (8 d)	Yes	ALT: 1,790; AST: 1,855	ANA: 1:100	Yes/No/Yes
Boettler *et al*.[13]	2022, Germany	52	M	BNT162b2 (10 d 1st; 51 d 2nd)	No	ALT: 2,130; AST: –; ALP: 142; GGT: 217	ANA: 1:200	Yes/Yes/No
Rela *et al*.[14]	2021, India	38	F	Covishield (20 d)	No	ALT: 1,025; AST: 1,101	ANA: 1: 80; IgG: 16.5 mg/L	Yes/Yes/No
		62	M	Covishield (16 d)	No	ALT: 1,094; AST: 1,361	Negative	Yes/Yes/No
Hasegawa *et al*.[15]	2022, Japan	82	F	Pfzer-BioNTech (7d)	No/HCV+	ALT: 1,097; AST: 1,682; ALP: 77; GGT: 104	IgG: 1,809	Yes/Yes/No
Mekritthikrai *et al*.[16]	2022, Thailand	52	F	Inactivated (7d)	No	ALT: 180; AST: 566; ALP: 228	ANA: 1:160 (fine speckled); ASMA: 1:400	Yes/Yes/Yes
Ueno *et al*.[17]	2022, Japan	54	F	Pfzer- (X2); Moderna (7d after the third dose)	No	ALT: 2,472; AST: 2,001; ALP: 352; GGT: 416	ASMA: 1:40; HLA-DR4+	Yes/Yes/No
Camacho-Domínguez *et al*.[18]	2022, Colombia	79	M	ChAdOx1 nCoV-19 (15 d)	No	AST: 2,003; ALT: 1,994	ASMA: 71.0 U; ANA: 1:80	Yes/Yes/No
Pinazo-Bandera *et al*.[19]	2022, Spain	77	F	BioNTech BNT162b2 and Spikevax ARNm-1,273 (2 d after the 2nd dose)	No	ALT: 552; AST: 474; ALP: 159	IgG: nl; ANA: 1: 60; AMA: 1:40; HLA-DR4 +	compatible with autoimmune hepatitis
		23	M	ChAdOx1nCoV-19 Vaxzevria/Covishield	No	ALT 587; AST 702; ALP 202	IgG: 1,647; ANA: –; AMA: –; HLA-DR3: +	compatible with AIH
Fimiano *et al*.[20]	2021, Italy	63	F	BNT162b2 mRNA (Pfizer/BioNTech) (37 d)	No	ALT: 1,778; AST: 1,625; ALP: 273; GGT: 419	anti-Sars-CoV 2 spike protein: 35,000 BAU/mL (>1,000 times UNL). Other antibodies are negative	Yes/Yes/No
Romero-Salazar *et al*.[21]	2022, Spain	76	M	Pfizer (~30 d)	No	Elevation	ANA: + (1:160)	Yes/Yes/Yes
Durazo *et al*.[22]	2022, USA	49	F	Unvaccinated	Yes	ALT: 851; AST: 614; ALP: 70	IgG: 2,093. Others negative	Yes/Yes/No
Kang *et al*.[23]	2022, Korea	27	F	Pfizer/BioNTech COVID-19 (14 d)	No	TB: 8.6; AST: 1,004; ALT:1,478; ALP: 182	ANA: 1:80; Mixed pattern; IgG: 1,641	Yes/Yes/Yes
Yoshida *et al*. [24]	2022, Japan	85	F	Pfizer/BioNTech, 56 d after 2nd dose	No	AST: 1,383; ALT: 925; ALP: 207; GGT: 326	IgG: 2,126; ANA: 1:1,280, speckled pattern. ASMA: 1:20 (marginally +)	Yes/Yes/No

ALC, anti-liver cytosol; ALP, alkaline phosphatase; ALT, alanine aminotransferase; AMA, anti-mitochondrial antibody; ANA, antinuclear antibody; ASLA, anti-soluble liver antigen; ASMA, anti-smooth muscle antibody; AST, aspartate aminotransferase; BAU, binding antibody units; dsDNA, double-stranded DNA; GGT, gamma-glutamyl transferase; HLA-DR, human leukocyte antigen; IgG, immunoglobulin G; LFT, liver function test; mRNA, messenger RNA; TB, total bilirubin; UNL, upper normal limit.

prognosis, including the need for liver transplantation. Early diagnosis of AIH is critical for patients with COVID-19-related conditions.

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Conflict of interest

The authors declare no conflict of interest.

Author contributions

Supervising the literature search and writing the majority of the paper (YW), providing the reported cases, guiding and editing throughout the writing process (YW), evaluating the histopathological images, and preparing the figures (YW, XC, AA, XD). All authors have read and approved the final manuscript.

Ethical statement

This study was performed in accordance with the Declaration of Helsinki (as revised in 2024). This case report does not include any identifiable patient information. According to institutional policy, this case report is exempt from Institutional Review Board approval.

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