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Abnormal Indicators of Autoantibodies and Liver Fibrosis in Patients with Covid-19

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1. Abstract

The authors describe a 48-year-old man with Covid-19 diagnosed in January 2020 in Huangshi, Hubei Province in China, along with two other critically ill patients with Covid-19 who were also seen in the same intensive care unit. Mild liver dysfunction combined with abnormal indicators of liver fibrosis and autoantibodies were seen in all three patients.

2. Case Report

We report three patients with mild liver dysfunction combined with abnormal indicators of liver fibrosis and autoantibodies. Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection was diagnosed in all three patients. These patients were admitted to the intensive care unit which was managed by a multidisciplinary team from the Affiliated Sir Run Run Hospital of Nanjing Medical University in the Huangshi Central Hospital in Huangshi, Hubei Province in China.

Fever, chills, and fatigue lasting one week developed in a previously healthy 48-year-old man (patient 1) (Table 1). Covid-19 was diagnosed by reverse- transcriptase polymerase-chain-reaction on January 25, 2020. The initial treatment was antibiotics, antivirus and supportive therapy; however, he then had progressive hypoxemic respiratory failure warranting the initiation of invasive mechanical ventilation. The Acute Physiology and Chronic Health Evaluation II (APACHE II) score during the first 24 hours of intensive care unit stay was 25 (scores range from 0 to 71, with higher numbers indicating greater risk of hospital death).

Laboratory results on admission to the intensive care unit are summarized in (Table 1). The patient had mild liver dysfunction as evidenced by elevated levels of alanine aminotransferase, aspartate aminotransferase, lactate dehydrogenase, gamma-glutamyl transferase, and serum bilirubin. Subsequent serologic testing showed elevated liver fibrosis indicators, including serum hyaluronic acid, procollagen type III, laminin and collagen type IV. Autoantibodies test showed significant positive results for antinuclear antibodies (ANA) and anti-ribonucleoprotein/Sjögren's-syndrome A antigen (anti-Ro/SSA), along with decreased concentration of complement 3. The patient received methylprednisolone treatment for 27 days. It was noteworthy that the level of ANA gradually decreased, and liver function returned to normal. The patient was finally extubated from mechanical ventilation on February 21, 2020 and transferred to rehabilitation ward.

Two other male patients with similar findings were seen at the same intensive care unit from January 15 to March 20, 2020. Serologic tests in these patients showed mild liver dysfunction combined with positive results of ANA and anti-Ro/SSA, as well as elevated levels of liver fibrosis indicators and decreased level of complement 3 or complement 4. Further clinical details are summarized in (Table 1).

Patients with severe Covid-19 seem to have higher rates of liver dysfunction [1]. Presence of ANA and anti-Ro/SSA often related to autoimmune dysfunction including autoimmunity hepatitis. ANA is the most common autoantibody detected in type I autoimmune hepatitis, with a prevalence of 80%-100% in many studies [2]. Decreased levels of complement 3 and/or complement 4 and elevated levels of liver fibrosis indicators are also associated with the severity of autoimmunity hepatitis [2]. Due to the impaired immune status in SARS-CoV-2 infection [3], the association between COVID-19 and autoimmune liver injury requires further investigation.

Table 1: Clinical Characteristics of three patients with abnormal indicators of liver fibrosis and autoantibodies.

Characteristics	Patient 1	Patient 2	Patient 3
Demographic Characteristics			
Age-yr	48	71	56
Sex	Male	Male	Male
Body mass index	20.76	24.22	20.28
Initial findings			
Medical history	None	Valvular heart disease	None
Symptoms at disease onset	Fever, chills, fatigue	Fever, fatigue, inappetence, cough	Fever, fatigue, cough, dyspnea
Imaging features	Ground-glass opacity, bilateral pulmonary infiltrates	Ground-glass opacity, bilateral pulmonary infiltrates	Ground-glass opacity, bilateral pulmonary infiltrates
Treatment before admission to ICU	Antibiotics, antivirus	Antibiotics, antivirus	Antibiotics, antivirus, intravenous immune globulin
Findings on admission to ICU			
Days since disease onset	6	60	23
Disease severity	Critical	Critical	Critical
APACHE II score	25	12	22
Laboratory findings			
White-cell count (per mm3)	7890	11,620	4200
Total neutrophils (per mm3)	6420	9760	3670
Total lymphocytes (per mm3)	860	490	280
Total monocytes (per mm3)	600	990	170
Platelet count (per mm3)	94,000	170,000	105,000
Hemoglobin (g/liter)	120	90	70
Albumin (g/liter)	35.4	35	34.6
Globulin (g/liter)	26.1	32.5	14.3
Alanine aminotransferase (U/liter)	66	60	14
Aspartate aminotransferase (U/liter)	80	65	28
Lactic dehydrogenase (U/liter)	480	534	217
Total Bilirubin (μmol/liter)	32.2	22.7	22.1
Direct Bilirubin (μmol/liter)	13.1	10.6	11.3
Indirect Bilirubin (μmol/liter)	19.1	12.1	10.8
gamma-glutamyl transferase (U/liter)	91	52	24
Creatinine (μmol/liter)	59	58	44
Creatine kinase(U/liter)	92	43	205
Procalcitonin (ng/ml)	0.3	0.33	0.5
C-reactive protein (mg/liter)	66.17	28.69	160.64
Interleukin-6 (pg/ml)	8.23	35.7	517.8
Immunoglobulin G (g/liter)	16.98	22.03	8.56
Antinuclear antibodies (AU/ml)	>500	>500	76.6
SSA/Ro60KD (AU/ml)	87.16	26.35	3.32
SSA/Ro52KD (AU/ml)	44.55	17.89	3.47
Complement 3 (g/liter)	0.61	0.6	0.47
Complement 4 (g/liter)	0.23	0.14	0.14
Serum hyaluronic acid (ng/ml)	1044.5	920.7	>2000
Procollagen type III (ng/ml)	390.1	691.4	904
Laminin (ng/ml)	197	227.7	98.24
Collagen type IV (ng/ml)	52.18	142	80.87

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