Dear Editor,

We present the case of a 60-year-old woman with no drug allergies or toxic habits, with hypothyroidism and receiving treatment with levothyroxine. She was admitted in February 2021, and presented with choluria of 72 hours progression, no abdominal or respiratory clinical symptoms and no related fever. Findings of the medical examination highlighted mucocutaneous jaundice and recorded oxygen saturation of 97% in ambient air. There was a notable pattern of cytolysis compatible with acute hepatitis, and no history hepatotoxic drugs. We performed screening for acute hepatitis in addition to serology testing, determination of autoantibodies, immunoglobulins, a PCR test for COVID and a radiologic study (Table 1).

She presented a positive PCR test for Covid-19, (SARS-2 RNA: positive CT=10), in addition to abnormal radiological findings in the lungs, without development of respiratory failure in the progression. Due to the Covid 19 infection and the suspicion of the onset of autoimmune hepatitis (AIH){a score of 19 using International Autoimmune Hepatitis Group criteria (without a liver biopsy and a value of +3 on the negative viral markers item)}. Treatment began with corticosteroids, achieving a positive response. The study was completed as an outpatient with a liver biopsy (with a COVID 19 RNA: positive CT of 35), and the exhaustive study no gave findings compatible with direct cytopathic injury due to COVID. After responding to induction therapy, she is currently in remission on maintenance therapy with azathioprine.
Discussion

The infection of SARS-CoV-2 presents as a respiratory illness, but may manifest with complications that are gastrointestinal, neurological, thromboembolic and cardiovascular, and immune-related. Ampuero describe how patients with liver involvement are at risk of developing complications from Covid-19.

In individuals with a genetic predisposition, virus and drugs may be trigger agents for AIH. We present a case triggered by COVID-19. Possible mechanisms may be an excessive immune response to the infection leading to a cytokine storm which, due to molecular mimicry between the virus and human proteins, causes the production of autoantibodies.

We found two descriptions of cases in the literature.

Table 1: Radiology parameters and biopsy

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Reference values</th>
<th>At onset (4/02/2020)</th>
<th>Follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>GOT/AST (IU/L)</td>
<td>1-31 (UI/L)</td>
<td>1830</td>
<td>29</td>
</tr>
<tr>
<td>GPT/ALT (IU/L)</td>
<td>1-31 (UI/L)</td>
<td>1422</td>
<td>30</td>
</tr>
<tr>
<td>GGT</td>
<td>1-38(UI/L)</td>
<td>224</td>
<td>35</td>
</tr>
<tr>
<td>ALP</td>
<td>30-120 (mU/mL)</td>
<td>116</td>
<td>116</td>
</tr>
<tr>
<td>INR</td>
<td>0.850-1.50</td>
<td>1.55</td>
<td>1.30</td>
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<tr>
<td>Platelets</td>
<td>140-400x10⁹/L</td>
<td>101000</td>
<td>155000</td>
</tr>
<tr>
<td>BR (mg/dL)</td>
<td>0.1-1 mg/dL</td>
<td>11.73</td>
<td>1</td>
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<tr>
<td>Ferritin (ng/mL)</td>
<td>10-120 mg/mL</td>
<td>4065</td>
<td>119</td>
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<tr>
<td>IL6 (pg/ml)</td>
<td>0.00-7pg/ml</td>
<td>14,23</td>
<td>6</td>
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<tr>
<td>Alb (g/L)</td>
<td>3.5-5 g/dL</td>
<td>3.2</td>
<td>3.6</td>
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<tr>
<td>SED (mm)</td>
<td>1-20</td>
<td>53</td>
<td>15</td>
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<tr>
<td>D-dimer (ng/ml)</td>
<td>&lt;500</td>
<td>576</td>
<td>460</td>
</tr>
<tr>
<td>LDH (mg/dL)</td>
<td>240-480</td>
<td>738</td>
<td>86</td>
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<tr>
<td>HAV</td>
<td>Negative</td>
<td>Negative</td>
<td>Immunization</td>
</tr>
<tr>
<td>Test</td>
<td>Result</td>
<td>Core +</td>
<td>Immunization</td>
</tr>
<tr>
<td>--------------</td>
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</tr>
<tr>
<td>HBV</td>
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<tr>
<td>EBV</td>
<td>Negative</td>
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<tr>
<td>CMV</td>
<td>Negative</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HSV</td>
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<tr>
<td>ANA</td>
<td>-</td>
<td>1/320</td>
<td>ND</td>
</tr>
<tr>
<td>A SMA</td>
<td>-</td>
<td>1/80</td>
<td>ND</td>
</tr>
<tr>
<td>Actin</td>
<td>-</td>
<td>+</td>
<td>ND</td>
</tr>
<tr>
<td>Immunoglobulin G (mg/dL)</td>
<td>700-1200 mg/dL</td>
<td>2775</td>
<td>900</td>
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<tr>
<td>Gamma globulin (gr/dL)</td>
<td>0.7-1.2 g/dL</td>
<td>1.9</td>
<td>1.2</td>
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<tr>
<td>Echography</td>
<td>No pathological findings</td>
<td></td>
<td>No abnormalities due to gas interposition.</td>
</tr>
<tr>
<td>Abdominal chest X-ray CT</td>
<td>No pathological findings</td>
<td></td>
<td>Without dilation of access, with normal liver parenchyma and small bilateral sub-pleural ground-glass opacities.</td>
</tr>
<tr>
<td>Liver biopsy</td>
<td>Hepatic cylinder where it was possible to assess 10 portal spaces, observing an increase of inflammatory infiltration consisting of lymphocytes and plasma cells. Infiltrate extended to the interface, with focal necrosis of hepatocytes and ballooning. Lobular lymphocyte aggregates were likewise observed. No significant steatosis observed. Perls Prussian blue showed granular iron deposits (confluent, visible at 10x) predominantly in portal macrophages. Fibrous portal expansion is noted with some fine bridging.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

REFERENCES


