COVID-19 INFECTION AS A TRIGGER FOR SERONEGATIVE AUTOIMMUNE HEPATITIS: A CASE REPORT

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Introduction

Autoimmune hepatitis (AIH) is an immune-mediated liver disease that commonly develops in a genetically predisposed patient after exposure to an environmental trigger. These triggers include viruses, immunization and drugs. **Both COVID-19 infection and vaccination have been linked to the development of AIH.**

Case presentation

A 58-year-old male patient, known to have **vitiligo** was evaluated in the hepatology clinic for **elevation of liver function tests (LFTs) that was detected during his hospitalization for COVID-19 infection**. His initial laboratory investigations during his hospitalization were remarkable for ALT of 271 and AST of 175 U. During hospitalization, LFTs were trending down but remained elevated (Figure 1). The hepatitis workup (Table 1) was notable for an **elevated IgG level of 16.9 g/l** (normal range 7-16).

His radiological investigations included US abdomen that revealed mildly increased echotexture, and liver elastography that showed a stiffness average of 5.77 kPa indicating mild stiffness. The patient was offered a liver biopsy, but he was reluctant. During follow-up, his LFTs remained elevated but were fluctuating (Figure 1). On week 31, he finally agreed for liver biopsy that **showed moderate interface and portal tract inflammatory cell infiltrate composed mainly of lymphocytes with occasional eosinophils**. The histopathological findings were suggestive of AIH. On week 34, he was started on prednisone 40 mg. LFTs on week 36 were completely normal. The prednisone was tapered over the following two months.

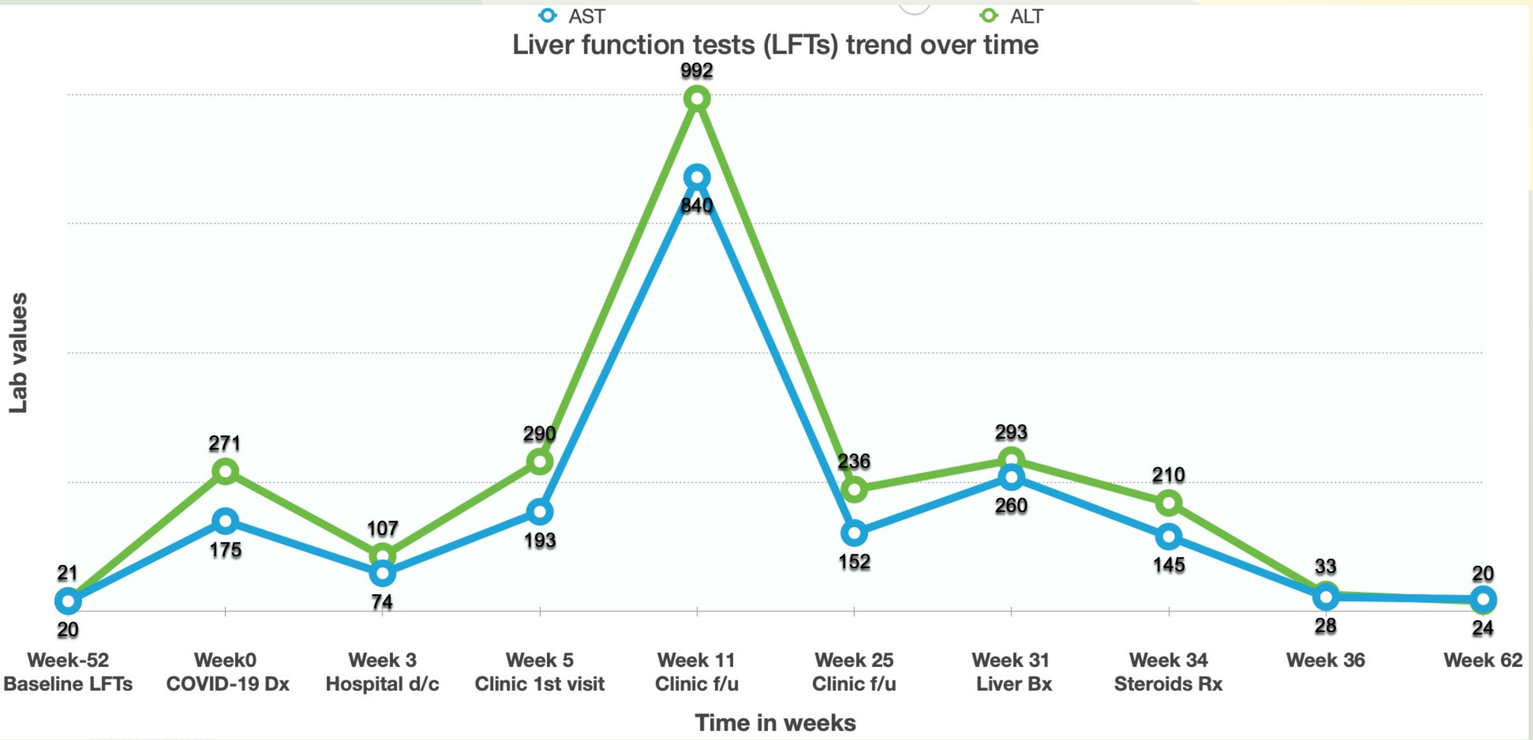

Figure 1. Trend of LFTs over time - Bx: biopsy - d/c: discharge - Dx: diagnosis - f/u: followup - Rx: treatment

Discussion & Conclusion

Our patient had an elevation of LFTs after COVID-19 infection that persisted for weeks and promptly responded to steroids therapy. His Revised Original Score for Autoimmune Hepatitis (AIH) was 17, indicating definite AIH.

One of the proposed mechanisms of AIH development after COVID-19 infection is the molecular mimicry between spike protein S1 and multiple human tissue proteins. More studies are needed to examine this association.

Contact

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Laboratory test	result
ALT	271 U/L
AST	175 U/L
ALP	38.9 U/L
Antinuclear Ab	Negative
Anti Mitochondrial Ab	Negative
Anti Mitochondrial M2 Ab	Negative
Anti Smooth Muscle Ab	Negative
Anti Liver Kidney Microsomes Ab	Negative
HBV Core Ab	**Reactive**
HBV Core Ab IgM	Negative
HBV Surface Antigen	Negative
HBV e Ab	Negative
HBV e Antigen	Negative
HAV Ab IgM	Negative
HCV Ab	Negative
HEV Ab IgG	**Reactive**
HEV Ab IgM	Negative
Herpes Simplex virus Ab IgM	Negative
Parvovirus Ab IgM	Negative
Alpha-1 anti-trypsin	37 (20-53 µmol/L)
Ceruloplasmin	33 (14 to 40 mg/dL)
CMV PCR	Negative
EBV PCR	Negative
HBV PCR	Negative

Table 1. Lab investigations – Ab: Antibody, (normal range)