Case Report

High level increase in liver enzymes and severe thrombocytopenia in a male case of anorexia nervosa

Mojgan Karahmadi¹, Elmira Layegh², Samira Layegh³, Maryam Kepour³

Abstract

BACKGROUND: Anorexia nervosa (AN) is a difficult-to-treat psychosomatic disease. Very few cases of acute liver failure associated with AN have been described. We describe one patient who was affected by AN and presented high level increase of serum liver enzymes, along with severe thrombocytopenia. Then, we discuss the possible etiopathogenic factors.

METHODS: A 14-year-old boy with AN was admitted in the pediatric psychiatric emergency department of Alzahra Hospital with impaired electrolyte levels, bradycardia, hypotension, liver dysfunction, and thrombocytopenia.

RESULTS: A ten-time increase in liver enzymes and thrombocytopenia were observed on admission. After two months of treatment, the levels were within the normal range.

CONCLUSIONS: Improvement of initial clinical symptoms and recovery of liver enzymes and thrombocytopenia after the treatment suggested that liver dysfunction and thrombocytopenia may be observed in AN patients and should be taken care of by physicians.

KEYWORDS: Anorexia Nervosa, Liver Dysfunction, Thrombocytopenia.

Anorexia nervosa (AN) is a difficult-to-treat psychosomatic disease.¹ Due to malnutrition, various physical problems have been reported to occur in patients with AN including diminished bone mineral density, increased QT interval, cardiomyopathy, and liver dysfunction. Thrombocytopenia is also known to occur in cases of low body weight accompanied by AN.²,³

Several studies have described an increase in serum liver enzyme in severely malnourished patients affected by AN,⁴⁻⁶ as well as during the refeeding phase of therapeutic intervention.⁷ In the case of anorectic patients, the slight to moderate increase in liver enzyme is expected to reflect a fatty liver that is typical of several protein-energy malnutrition states.⁸,⁹ Nevertheless, very few cases of acute liver failure associated with AN have been described.¹⁰,¹¹ Aside from malnutrition, several pathogenetic factors have been considered to justify the shift from normal to slightly increased serum liver enzymes. These factors include acute complications such as hepatotropic viruses (hepatitis A and B, cytomegalovirus, Epstein–Barr virus), acute liver hypoperfusion, hypothermia, alcohol, cannabis, and cocaine abuse, and hepatotoxic drugs (paroxetine, amphetamines, benzodiazepines, methadone, opiates, etc.).¹² In this study, we describe a patient who was affected by AN and presented high level increase in serum liver enzymes and severe thrombocytopenia. Then we discuss the possible etiopathogenic factors and describe our
medical intervention.

**Case presentation**

A 14-year-old boy with an 8-month history of AN referred to our outpatient clinic (Department of Pediatrics, Alzahra Hospital). Because of his physical condition (weight loss, bradycardia, imbalanced electrolytes, hypothermia, hypotension, and lung and liver dysfunction) we decided to hospitalize him (Figure 1).

On admission to the psychiatric unit, his body weight and body mass index (BMI) were 31 kg and 13.2, respectively. In addition, his liver enzymes were high, and his platelet decreased as shown in Table 1. Other causes of acute liver dysfunction such as hepatotoxic drugs, alcohol, cocaine and viral hepatotoxic diseases were evaluated and then ruled out.

We started to treat the patient with conservative management including stabilizing electrolytes by serum therapy and nourishment using a high calorie diet. A marked decrease in serum transaminase and an increase in platelet count were observed with a gradual normalization of all the biochemical parameters within one months of hospitalization. He started eating and his electrolytes got balanced and his family became happy about his recovery. Therefore, he was discharged after one months of hospitalization and successfully continued his rehabilitation program on an outpatient basis.

![Figure 1](image_url). A male patient with anorexia nervosa.
Table 1. Changes in liver enzymes and platelet numbers during the hospitalization period.

<table>
<thead>
<tr>
<th></th>
<th>The first day</th>
<th>During the first week</th>
<th>After two weeks</th>
<th>At discharge time</th>
</tr>
</thead>
<tbody>
<tr>
<td>AST (IU/L)</td>
<td>170</td>
<td>236</td>
<td>523</td>
<td>40</td>
</tr>
<tr>
<td>ALT (IU/L)</td>
<td>160</td>
<td>281</td>
<td>585</td>
<td>110</td>
</tr>
<tr>
<td>Platelet (/mm$^3$)</td>
<td>133,000</td>
<td>50,000</td>
<td>63,000</td>
<td>283,000</td>
</tr>
</tbody>
</table>

AST: Aspartate aminotransferase (normal range ≤ 42 IU/L)
ALT: Alanine aminotransferase (normal range ≤ 48 IU/L)
Plat: Platelet (normal range: 150000–450000)

Discussion
This was one of the uncommon cases of AN with a high level increase (10 times more) of liver enzymes and sever thrombocytopenia. Minor degrees of liver injury have been reported in up to 40% of patients with AN.\textsuperscript{13} Although the mechanism of liver injury in AN has thought to be due to protein-calorie malnutrition of the kwashiorkor type accompanied with fatty changes, this has not been rigorously demonstrated and the mechanism is still unknown.\textsuperscript{13, 14} In AN, malnutrition has been reported to cause thrombocytopenia along with liver dysfunction.\textsuperscript{2}

One report has described temporal changes in liver enzyme levels, platelet count, and thyroid peroxidase (TPO) levels in a patient with AN.\textsuperscript{3} Acute liver failure with a massive increase of serum liver enzyme may be a severe, though relatively uncommon, complication caused by severe malnutrition such as that secondary to AN.\textsuperscript{4-6} As reported in the literature, serum transaminases do not reach such high values during the refeeding syndrome.\textsuperscript{7} To avoid complications of refeeding syndromes,\textsuperscript{7} nutritional rehabilitation is started with a low-energy oral and parental nutrition (25 kcal/kg body weight).

Therefore, follow up of serum liver enzymes should be part of our evaluation to prevent the uncommon but serious risk of acute liver failure. We should also examine the patient for thrombocytopenia.

Conclusion
Our case report showed an AN patient with increased level of liver enzymes about 10 times more than normal range, as well as severe thrombocytopenia, which is uncommon. Therefore, it is concluded that physicians should be aware of high level increase of liver enzymes and severe thrombocytopenia which may occur in AN.

Conflict of Interests
Authors have no conflict of interests.

Authors’ Contributions
MK introduced the case and managed the process. SL found pediatric references and provided information on anorexia nervosa. MK provided up-to-date data regarding the case.

References


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