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Case Report

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A Therapeutic Efficacy of Ursodeoxycholic Acid on Cholestasis after The Nutrition Therapy for Starvation Hepatitis

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1. Abstract

A 42-year-old woman with a history of anorexia nervosa visited to our hospital along with transaminitis of asparate aminotransferase (AST) and alanine aminotransferase (ALT) of 7700 U/mL and 5700 U/mL, respectively, which indicated starvation hepatitis. After refeeding of nutrition, AST/ALT decreased, transient elevated, decreased again, and maintained to 100-600 U/mL, and a liver biopsy was performed, which revealed cholestasis. AST/ALT rapidly improved with administration of ursodeoxycholic acid. Ursodeoxycholic acid was effective in a case of cholestasis due to nutritional treatment of starvation hepatitis in relation to malnutrition.

2. Introduction

Transaminitis may occur in lean patients with anorexia nervosa and other forms of malnutrition. Ischemic hepatitis, refeeding syndrome, and autophagy-related starvation hepatitis are thought to be the causes [1]. The primary treatment for these debilitated patients is nutritional therapy. We report a case of a debilitated patient with aspartate aminotransferase (AST) of 7770 U/L and alanine aminotransferase (ALT) of 5700 U/L. After initiation of nutritional therapy, AST/ALT gradually decreased to 100-600 U/L, but AST/ALT remained at this level. The patient was diagnosed with cholestasis due to starvation hepatitis caused by nutritional therapy, and after administration of ursodeoxycholic acid, AST/ALT rapidly normalized.

3. Case Presentaion

A 42-year-old unmarried female patient visited to the outpatient department of psychosomatic medicine at Fukuoka Tokushukai Hospital with her parents due to muscle weakness and weight loss. She had changed from anorexia nervosa to purging-type bulimia nervosa. Her alcohol intake had also increased to 50-100 ml of shochu. However, at the age of 35, she had a boyfriend. Thereafter, her mental condition stabilized, her pathological eating behavior stopped, and her eating disorder went into remission. Her weight was 45-46 kg. However, six months prior to her visit to our clinic, her relationship with her boyfriend broke down. Thereafter, she lost her appetite and her weight decreased. At the time of initial examination, his weight was 32 kg, height was 159 cm, and body mass index (BMI) was 12.7. Laboratory data showed mildly elevated aminotransferase levels (AST 78 U/L, ALT 76 U/L) and concomitant non-thyroidal illness (FT3 0.57pg/mL, FT4 0.70ng/dL, TSH 0.93 μIU/mL). Hospitalization was recommended due to malnutrition. However, she declined to be hospitalized. However, one week later, she was transported to our hospital by her parents due to mobility difficulties. Her weight had decreased to 30 kg (BMI 11.9). Blood pressure was 85/50 mmHg, pulse 60 bpm, and regular. She was awake. Muscle strength in the extremities was decreased. AST was 7770 U/L and ALT was 5700 U/L. Many other laboratory data were also abnormal, as shown in Table 1. After admission, hydration and nutritional support were initiated, and initial total calories were 500-600 kcal. AST and ALT gradually decreased, transient elevated, decreased again and maintained at 100-600 U/L (Figure 1). Anti-mitochondrial 2 and antinuclear antibodies were negative, and hepatitis C ribonucleic acid qualitative analysis was also negative. A liver biopsy was performed to clarify the cause of the high aminotransferase levels. The biopsy specimen showed bile

congestion (Figure 2). The diagnosis of cholestasis due to refeeding was made, and ursodeoxycholic acid was administered, after which AST and ALT rapidly normalized. After 100 days of hospitalization, the patient's weight had increased to 38 kg, AST was 16 U/L, and ALT was 19 U/L.

Table 1: Laboratory Finding on Admission.

Complete blood count		**Serology**	
WBC	3,930 /μL		
Neutrophils	79%	HBsAg	(-)
Lymphocytes	14%	HBsAb	(-)
Hemoglobin	14.3 g/dL	HCVAb	(-)
Platelet	11.6x10^4 /μL		
Coagulation			
PT-INR	1.34		
Biochemistry			
Total Protein	6.5 g/dL	UA	3.5 mg/dL
Albumin	4.3 g/dL	Na	138 mEq/L
T-Bil	5.3 mg/dL	K	3.7 mEq/L
AST	7770 U/L	Cl	97 mEq/L
ALT	5700 U/L	Ca	8.7 mg/dL
LD	3200 U/L	Mg	2.6 mg/dL
ALP	2750 U/L	P	3.0 mg/dL
γ-GTP	403 U/L	TG	41 mg/dL
BUN	35.0 mg/dL	HDL-CHO	108 mg/dL
Creatinine	0.59 mg/dL	LDL-CHO	39 mg/dL
eGFR	88.0 mL/min/L	β-HB	97 μmol/L
Glucose	78 mg/dL	prealbumin	12.0 mg/dL

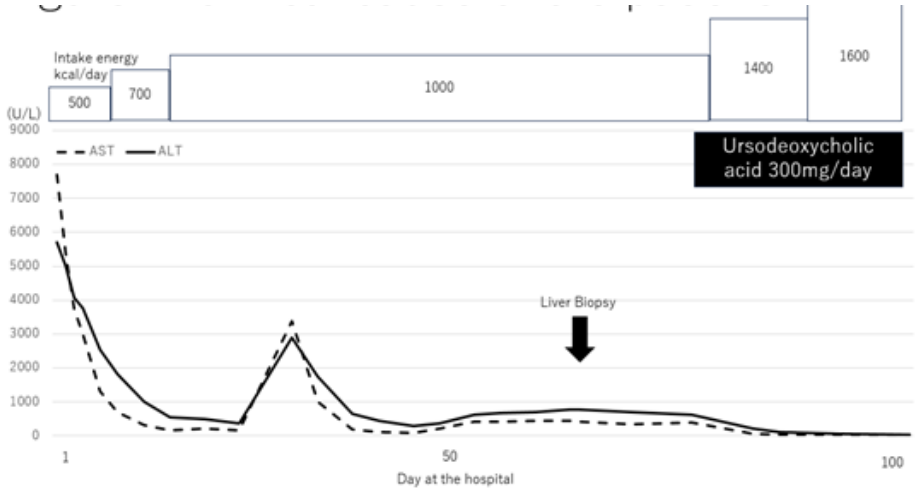

Figure 1. AST aspartate aminotransferase, ALT: alanine aminotransferase.

Figure 2-a

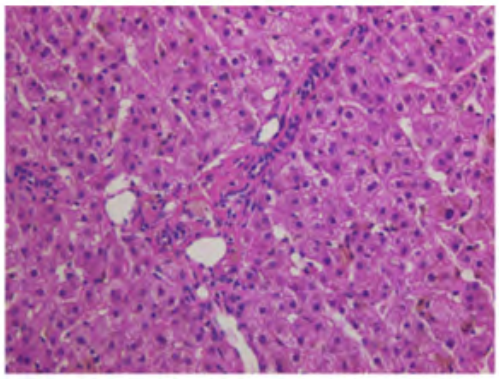

Figure 2-b

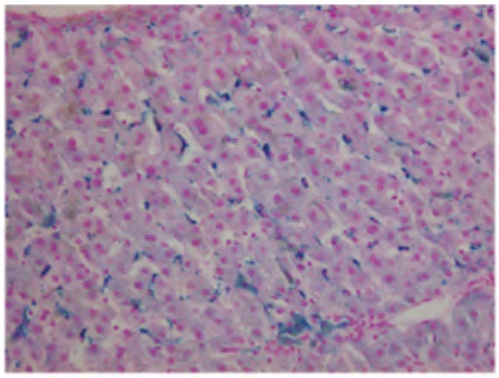

Figure 2. Liver specimen show well preserved lobular structure with no inflammatory cell infiltration and no portal fibrosis. Bile congestion is moderate in degree. Piecemeal necroses are not found, but single cell necrosis is found rarely. (a Hematoxylin and Eosin staining, b Berlin Blue Staining).

4. Discussion

Liver dysfunction due to malnutrition can be classified into starvation hepatitis and refeeding syndrome. In starvation hepatitis, adequate nutrition is important [1]. On the other hand, the initiation of avoid refeeding syndrome was recommended with fewer calories. Autophagy is associated with starvation hepatitis [1,2]. However, a recent study showed that initial refeeding of 2000 kcal is safe without refeeding syndrome in very underweight anorexic patients with close monitoring of electrolytes such as phosphate, potassium, and magnesium [3]. Thus, the method of nutritional therapy for malnutrition may be reevaluated in the future. In the present case, aminotransferase decreased to about 100-600 U/L after refeeding and persisted. Liver biopsy revealed cholestasis. Fatty liver and cholestasis have been reported with parenchymal nutrition [4,5] and enteral nutrition [6]. Hydrophobic bile acids have been reported to be cytotoxic to both hepatocytes and choledochal cells [6]. Urosdeoxycholic acids improves bile acid secretion and acts as anti-apoptosis of hepatocytes, is effective for cholestatic liver diseases, and usually medicated to primary biliary cholangitis [6]. The nutrition therapy is very important for starvation hepatitis. The elevation of aminotransferase during the treatment course for starvation hepatitis, cholestasis or fatty liver were occurred and ursodeoxycholic acids might be effective as our case and others [7]

5. Conclusion

Ursodeoxycholic acid is effective in a case of cholestasis due to nutritional treatment of starvation hepatitis in relation to malnutrition.

References

1. Nadelson AC, Babatunde VD, Yee EU, Patwardhan VR, Expanding the differential diagnosis for transaminitis in patients with anorexia nervosa. J Gen Intern Med. 2016; 32: 486-9.
2. Rautou P-E, Cazals-Hatem D, Moreau R, Francoz C, Feldmann G, Lebrec D, et al. Acute liver cell damage in patients with anorexia nervosa: a possible role of starvation-induced hepatocyte autophagy. Gastroenterology. 2008; 135: 840-848.
3. Cuntz U, Korner T, Voderholzer U. Rapid renutrition improves health status in severely malnourished inpatients with AN - score- based evaluation of a high caloric refeeding protocol in severely malnourished inpatients with anorexia nervosa in an intermediate care unit. Eur Eat Disorders Rev. 2022; 30: 178-189.
4. Btaiche IF, Khaldi N. Meatbolic complications of parenteral nutrition in adults, part 2. Am J Healt Syst Pharm. 2004; 61: 2050-2057.
5. Grau T, Bonet A, Rubio M, Mateo D, Farré M, Acosta JA, Blesa A, et al. the Working Group on Nutrition and Metabolism of the Spanish Society of Critical Care. Liver dysfunction associated with artificial nutrition in critically ill patients. Crit Care. 2007; 11.
6. Paumgartner G, Beuers U. Mechanisms of action and therapeutic efficacy of ursodeoxycholic acid in cholestatic liver disease. Clin Liver Dis. 2004; 8: 67-81.
7. El-Reshaid K. Refeeding syndrome in a patient with advanced kidney failure due to nephronophthisis. Saudi J Kidney Dis Transpl. 2013; 24: 1217-1222.