Japanese Journal of Gastroenterology and Hepatology

ISSN: 2435-1210 | Volume 8 Case Report

Complete Remission of Protein-Losing Enteropathy with Chronic Hepatitis B by Traditional Chinese Medicine in an Adult: An Unusual Case Report

Ting Xiang¹, Haoxuan Luo¹, Baoguo Sun2, Jun Meng², and Shijun Zhang^{1*}

Department of Traditional Chinese Medicine, The First Affiliated Hospital of Sun Yat-Sen University, Guangzhou, Guangdong 510080, P.R. China

²Department of Nephrology, Chongqing Hospital of Chinese Medicine, Chongqing 400000, P.R. China

*Corresponding author:

Shijun Zhang,

Department of Traditional Chinese Medicine, The First Affiliated Hospital of Sun Yat-Sen University, 2th Zhongshan Road, Guangzhou, Guangdong 510080, P.R. China,

E-mail: zhshjun@mail.sysu.edu.cn

Keyword:

Protein-losing enteropathy; Intestinal lymphangiectasia; Chronic hepatitis B; Traditional Chinese Medicine

Received: 04 May 2022 Accepted: 23 May 2022

Published: 27 May 2022 J Short Name: JJGH

Copyright:

©2022 Shijun Zhang, This is an open access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and build upon your work non-commercially.

Citation:

Shijun Zhang. Complete Remission of Protein-Losing Enteropathy with Chronic Hepatitis B by Traditional Chinese Medicine in an Adult: An Unusual Case Report.

J Gstro Hepato. V8(17): 1-4

Abbreviations:

PLE: Protein Losing Enteropathy; CHB: Chronic Hepatitis B; IL: lymphangiectasia; TCM: Traditional Chinese Medicine

1. Abstract

We herein report a 22-year-old man with the history of chronic hepatitis B and serum HBV DNA level was high suffering from systemic edema due to protein-losing enteropathy(PLE), which subsequently proved to be intestinal lymphangiectasia (IL). His symptoms aggravated with the therapy of liver-protecting, prednisolone in 3 hospitals, furthermore, he was in ICU by phlegmonous inflammation result of severe edema. However, his symptoms complete remission with the use of proved recipe of TCM in 3 months at hometown. He is on good with only adefovir dipivoxil and HBV DNA at undetectable levels for a 5 years follow-up period. The onset of PLE maybe related to active CHB. TCM therapy should be considered as possible effective treatment strategies for PLE.

2. Introduction

Protein-losing enteropathy (PLE) is a disease that induces abnormally serum proteins loss into the intestinal lumen and it is related to many diseases [1,2,3]. Intestinal lymphangiectasia (IL), a rare kind of PLGE firstly reported by Waldmann et al, was defined as congenial malformation or obstruction of intestinal lymphatic drainage resulted in the leakage of lymph with extra protein [4]. This is often a primary disorder has been recorded in children, but initial definition in adults has become increasingly more common [5,6,7]. Recently, although

several reports have concerned about the liver cirrhosis associated IL, there is no published information regarding Chronic Hepatitis B (CHB) associated IL [8,9,10]. Similarly, there have been controversies over the treatment of IL on adult. Therefore, we herein report a rare case that an adult who was diagnosed as the IL accompany with CHB according to the symptom of sever edema afterwards completed recovered by the treatment of a proved recipe of Traditional Chinese Medicine (TCM) on 5 years of follow-up.

3. Case Report

3.1. Medical History

A 22-year-old male was admitted to locally for insidious onset of abdominal distention and mild pitting edema in lower limbs for several months. He has the medical history of CHB infection since he was 10 years old. Computed tomography (CT) of abdomen and pelvis showed retroperitoneal lymphadenopathy but no abnormalities of the liver and bowel, while the further biopsy revealed a reactive hyperplasia of lymph nodes and dilatation of lymph sinus. Meanwhile, a 1.7 g/dl albumin, an abnormal hepatic function (AST 198 U/L and ALT 58 U/L) and high serum HBV DNA level (>1 million copies/mL) by laboratory examination, in association with a negative result of urinalysis indicated a diagnosis of active CHB induced hypoproteinemia. Therefore, the treatment of diuretic combine with

interferon was applied for 3 months, which was suspended due to the poor effects.

3.2. Treatment in our Hospital

He was subsequently transferred to our hospital 6 months later due to the aggravated symptoms such as severe pitting edema, tightness of chest and shortness of breath. 28 days before his edema aggravated the blood examination showed significant liver damage (Figure 1 -28d). The further examinations were performed and listed in Table 1 in details.

Several therapeutic protocols were carried out such as human serum albumin(HSA), diuretic, adefoir and liver-protecting. Although the liver function improved two weeks later (AST 105 U/L, ALT 105

U/L), continuous hypoalbuminemia (Serum Albumin <1.7 g/dl and Total Serum Protein <4.0 g/dl) was observed. (Figure 1 10d to 25d).

3.3. Treatment in Other Hospitals

He was transferred to two hospitals for further alternative treatment (Figure 1 25d to 63d). The lymphatic imaging decreased in the right part of hypogastrium was detected by the 99mTc-abeled sulphur colloid (99mTc-SC) scintigraphy. Predonisolone (PSL starting dose of 50 mg/day) was administered, but his edema did not improve. However, he was transferred to the Intensive Care Unit immediately for the severe phlegmonous inflammation appeared on the lower limbs consequently owing to the edema-induced malnutrition. Finally, the patient requested to discharge from the hospital and returned home after the phlegmonous inflammation relieved.

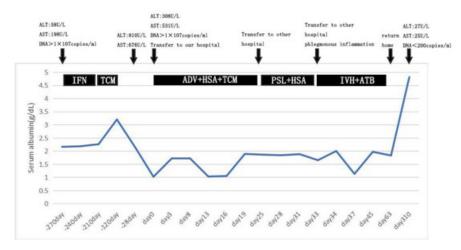


Figure 1: Clinical course along with the serum albumin levels. IFN: interferon, ADV:adefovir dipivoxil, HSA: human serum albumin, PSL: prednisolone, ATB: antibiotics.

Table 1. The examination results of the patient in our Hospital.

Examination Type	Result	
Physical Examination	Palpation	Not palpable for liver and spleen.
		Pitting edema from facial to foot.
	Auscultation	Negative for cardiac murmur.
	Inspection	Negative for jaundice of sclera and skin.
		Total Protein:3.2g/dl(normal range:6.4-8.7g/dl);
	Blood Routine	Albumin:1.0g/dl(3.5-5.5g/dl); AST:531U/L and ALT:
		308U/L(1-40U/L); ChE:1990(5300-12900U/L); AFP:
		136.47μg/L(0-20μg/L); Lymphocyte(LY): 0.34 (0.19-0.47); TBIL & PT: Normal a
Laboratory	Urinalysis	Quantitative Urinary Protein: 7.9 mg/dl; Negative for urinary protein ^b .
Examination		
	Immunological	Lower for Serum Immunoglobulin & C3 Complement; Negative for Immune Rheumatic.
	Examination	
	Thyroid & Renal Function	Normal.

	Abdominal	Good echogenicity without local damage or increased diameter for the intrahepatic biliary vessels; normal range for the diameter of the portal vein; normal for the spleen, pancreas and kidneys.
	Renal Color	Pericardial effusion.
Imaging	Ultrasound	
Examination	X-ray	Both-sided pleural effusion.
	Gastroscopy	A mild swelling of the entire gastric mucosa without any tumor or erosive ulcerative lesions or giant folds; no gastroesophageal varices detected.
Endoscopy		
Examination	Colonoscopy	A mild edema; no tumor tissues or erosive ulcerative lesion detected.
	Capsule Endoscopy	An edematous but no impairment in entire gastrointestinal tract.
Pathological	Biopsy	A chronic inflammatory (Four biopsies taken from stomach).
Examination		

Note: a. The Blood Routine indicated an active CHB (AST: Aspartate Aminotransferase; ALT: Alanine Aminotransferase; ChE: Cholinesterase; AFP: Alpha Fetal Protein; TBIL: Total Bilirubin; PT: Prothrombin Time);

- b. The Urinalysis reflected that the levels of edema and hepatic lesion were discordant, which indicated a prognosis of PLGE;
- c. The Endoscopy Examination showed negative results of entire gastrointestinal mucosa, but the number of lymphocytes in the serum was significantly reduced, which supported the diagnosis of IL induced PLGE.

3.4. Treatment at Home by a Recipe of TCM

His father received a recipe of TCM, by which another patient was cured completely. He try this recipe along with adefovir dipivoxil and HSA administration. The bowel movement of the patient increased and edema almost eliminate one month later. Then, the HSA was suspended while the frequency administration of this recipe was dropped to once in one week gradually in three months. Fortunately, the edema was never relapsed again by administrating the recipe occasionally without the diet control. The reexamination results of the patient one year later, which showed a normal serum protein values (Serum Albumin: 4.0 mg/dl; Total Serum Protein: 6.2 mg/dl) and an undetectable level of HBV DNA. The TCM recipe consists of Dendrobium 20g, White Peony Root 20g, Fructus Aurantii 20g, Lonicera japonica 20g, Akebia quinata Decne 20g, Bupleurum chinense 20g, Squama Manis 20g, Areca Catechu 20g, Glycyrrhiza 15g, Gardenia Jasminoides 20g, Eclipta Prostrata 20g, Rhubarb 20g and Folium Sennae 20g.

4. Discussion

We herein present a rare case of CHB with PLE. Reviewed the entire diagnosis procedure, the characteristic features such as hypoalbuminemia and lymphopenia, the dilatation of lymph sinus revealed by biopsy of retroperitoneal lymphadenopathy and an abnormal lymphatic drainage imaging in hypogastrium detected by 99mTc-SC, we finally diagnose PLE in this case may caused by IL. IL along with the cirrhosis might result from the portal hypertension induced inflammation and straitness of lymph vessel,9 nevertheless, the exact

mechanism of obstruction in lymphatic drainage in this patient complicating CHB have to be clarified. However, it was observed in this case that the onset of IL was accompanied by liver damage and high level of HBV DNA. When the edema subsided after the treatment, the liver function was normal and HBV DNA was undetectable. This case may implies that the IL is correlated with the activity of the CHB. Furthermore, there is still much to learn about the pathogenesis of the IL together with the CHB.

Since it have to be reinforced, the protocol for the IL generally aims at its protopathy. The protocols of antiplasmin, corticosteroids, octreotide, small intestine resection, peritoneovenous shunt and intestinal transplant were defined to be variable effective for the IL.9,11,12 Also, the lifelong dietary modification with high protein, fat restriction and substitution with the Medium Chain Triglyceride are advocated in the management of IL.7 As for this case, the severe hypogammaglobulinemia and lymphopenia had resulted in the recurrent-IL and opportunistic infections which had threatened the life of the patient. The critical state lead the patient cannot but choose this recipe of TCM therapy. Surprisingly, the five years follow-up verified the certain effects of this recipe since the re-examinations showed a normal data in association with an unrestricted diet and no abnormal symptoms, which implied that the TCM was an alternative approach for the IL. To our knowledge, there is very little publish information regarding the TCM therapy for the IL, especially for the severe cases. All in all, this case demonstrates a novel idea on the therapeutic protocol of the IL by TCM, further studies are needed to confirm the curative effect and mechanism of this recipe.

References

- Ito S, Higashiyama M, Horiuchi K, Mizoguchi A, Soga S, Tanemoto R, et al. Atypical Clinical Presentation of Crohn's Disease with Superior Mesenteric Vein Obstruction and Protein-losing Enteropathy. Intern Med. 2019; 58: 369-374.
- Udink Ten Cate FE, Hannes T, Germund I, Khalil M, Huntgeburth M, Apitz C, et al. Towards a proposal for a universal diagnostic definition of protein-losing enteropathy in Fontan patients: a systematic review. Heart. 2016; 102: 1115-1119.
- Umar SB, DiBaise JK. Protein-losing enteropathy: case illustrations and clinical review. Am J Gastroenterol. 2010; 105: 43-49.
- Waldmann TA. Gastrointestinal protein loss demonstrated by Cr-51labelled albumin. Lancet. 1961; 2: 121-123.
- Borzutzky A, Espino A, Alberti G, Torres J, Harris PR. Primary Intestinal Lymphangiectasia (Waldmann's Disease). Am J Gastroenterol. 2019; 114: 197.
- Lopez RN, Day AS. Primary intestinal lymphangiectasia in children: A review. J Paediatr Child Health. 2020; 56: 1719-1723.
- Freeman HJ, Nimmo M. Intestinal lymphangiectasia in adults. World J Gastrointest Oncol, 2011; 3: 19-23.
- Dahlqvist GE, Jamar F, Zech F, Geubel AP. In-111 transferrin scintigraphy in cirrhosis with hypoalbuminemia: evidence for protein-losing enteropathy in a small group of selected cases. Scand J Gastroenterol. 2012; 47: 1247-1252.
- Lee HL, Han DS, Kim JB, Jeon YC, Sohn JH, Hahm JS. Successful treatment of protein-losing enteropathy induced by intestinal lymphangiectasia in a liver cirrhosis patient with octreotide: a case report. J Korean Med Sci. 2004; 19: 466-469.
- Stanley AJ, Gilmour HM, Ghosh S, Ferguson A, McGilchrist AJ. Transjugular intrahepatic portosystemic shunt as a treatment for protein-losing enteropathy caused by portal hypertension. Gastroenterology. 1996; 111: 1679-1682.
- Balaban VD, Popp A, Grasu M, Vasilescu F, Jinga M. Severe Refractory Anemia in Primary Intestinal Lymphangiectasia. A Case Report. J Gastrointestin Liver Dis. 2015; 24: 369-73.
- Ersoy O, Akin E, Demirezer A, Yilmaz E, Solakoglu T, Irkkan C, et al. Evaluation of primary intestinal lymphangiectasia by capsule endoscopy. ENDOSCOPY. 2013; 45: E61-2.