Effect of Virechana in Alcoholic Liver Disease (ALD)

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Abstract

A 52 years old Male patient approached a health care center seeking an advice for general health improvement. On elucidating the history, the patient was known alcoholic since 20 years and consumed twice daily (250-300ml). Clinical examination revealed muddy conjunctiva and tremors in the hand. On palpation, tenderness was elucidated in right hypochondric region with three finger hepatic enlargement. Investigation revealed mild hepatomegaly (Yakruttodara) with diffuse fatty infiltration of liver, mild spleenomegaly (Pleehodara) and grade I prostatomegaly with elevated LFT levels. Urine examination presented with pale yellow color, slightly turbid with presence of Albumin, calcium oxalate crystals, bacteria and pus cells. The modality of treatment adopted was Virechana karma. Post-Virechana, there was a marked regression in hepatomegaly with normal LFT and urine routine.

Keywords: Yakruttodara; Hepatomegaly; Spleenomegaly.

Introduction

Alcoholic liver disease (ALD), which ranges from simple steatosis to cirrhosis and hepatocellular carcinoma (HCC), continues to represent a major health issue [1]. Alcoholic liver disease occurs after years of heavy drinking. Over time, scarring and cirrhosis can occur [2]. Cirrhosis is the final phase of alcoholic liver disease. Alcoholic liver disease may take the form of chronic illness (steatosis, steato-hepatitis, fibrosis and cirrhosis) or acute involvement (alcoholic hepatitis) [3].

Self-restraint from alcohol, Panchakarma therapy and proper nutrition would be the proper line of treatment. Steatosis is reversible upon withdrawal of alcohol, but alcoholic hepatitis can persist even with abstinence and may progress to cirrhosis. Virechana is one of modality of treatment in Udara roga. Hence, in the present case, we are presenting the result of Virechana in a case of 54 year male diagnosed as ALD.

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Case Report

A 54 year Old 65 kg moderately built male, belonging to middle class family approached the health care center seeking an advice for general health improvement. On elucidating the history, the patient was alcoholic since 20 years and consumed brandy daily (250-300ml) twice a day. On Clinical Examination, Muddy conjunctiva, tremors in hands were noticed. Tenderness in right hypochondric regions with three finger hepatic enlargement was found by palpation. Ultrasonography and Abdomen impression revealed mild hepatomegaly with diffuse fatty infiltration of liver, mild spleenomegaly and grade I prostatomegaly. Laboratory investigations revealed elevated LFT values of direct and serum bilirubin, serum globulin, serum alkaline phosphatase and SGOT. Urine examination presented with pale yellow color, slightly turbid with presence of albumin, calcium oxalate crystals, bacteria and pus cells.

Treatment

Arohana karma Snehapana was administered with Shatphala Ghrita. During Vishrama kala abhyanga with murchita tila taila and ushna jala snana was performed. On the final day of Vishramakala, Virechana dravya – Guda Haritaki

followed by Triphala Kashaya was administered. Patient had 8 vegas of Virechana.

Samsarjana Krama was advised after Virechana. After samsarjana Krama, the patient was prescribed the following medications. The patient was advised to avoid spicy, oily and salty food. He was also asked to taper the dose of alcohol thereby.

Observations and Results

All the values returned to normal limits with marked regression in hepatomegaly and spleenomegaly. Urine examination showed clear urine with absence of albumin, crystal, bacterial and pus reduction.

Table 1: Changes in laboratory parameters before and after treatment

Parameters	Before Treatment	After Treatment
Serum Bilirubin Total	1.3mg%	1.0mg%
Serum Bilirubin Direct	0.4mg%	0.2mg%
SGOT	54IU/L	40IU/L
S Globulin	3.5gm/%	3.3gm%
S. Alkaline phosphatase	127 IU/L	110 IU/L

Discussion

The development of ALD depends on several factors. Daily intake of alcohol for 10-12 years with doses in excess of 40-80 g/day for males and of 20-40 g/day for females generally causes ALD [4]. In the present case, the patient has a history of consuming Brandy (250-300ml) twice a day. The most common physical exam finding in patients with either steatosis or alcoholic hepatitis is hepatomegaly. Up to 70% of hospitalized patients with steatosis have hepatomegaly [5]. We found mild hepatomegaly with diffuse fatty infiltration of liver in the present case.

Virechana is one of the treatment modality in Udararoga [6]. Virechana aims at the elimination of Doshas trough Purisha-Dhara Kala (lower intestine) and Yakrita(liver). The vitiated Pitta Dosha present in entire body is alleviated and expelled out. Udara Vyadhi can be considered as one of the Udakavaha Sroto vikruti as Udakavaha Srotas is the main srotas what gets afflicted and Yakrut, Pleeha (Liver & Spleen) & the factors associated with them like Ranjak Pitta, Rakta Dhatu are involved in it. Excessive Dosha Sanchaya & Srotorodha are the two basic factors for the Samprapti of Udara. For the Samprapti Bhanga, Shodhan, especially Virechana, is very useful [7]. Due to Virechana, excessive amount of vitiated Doshas are eliminated thereby bringing marked regression in hepatomegaly and spleenomegaly with normal LFT and urine laboratory investigations.

Conclusion

The present case of ALD with mild hepatomegaly and spleenomegaly was treated with on the principle of Yakruttodara and Pleehodara chikitsa and Virechana was found effective bringing marked regression in hepatomegaly and spleenomegaly with normal LFT and urine laboratory investigations.

References

- 1. Thomas H. Frazier, Abigail M. Stocker, Nicole A. Kershner, Luis S. Marsano and Craig J. McClain, Treatment of alcoholic liver disease, Ther Adv Gastroenterol. 2011; 4(1): 63-81.
- 2. https://www.nlm.nih.gov/medlineplus/ency/article/000281.htm referred on 25th June 2016
- Bruha R, Dvorák K, Dousa M, Petrtýl J, Svestka T., Alcoholic liver disease, Prague Med Rep. 2009; 110(3): 181-90.
- Thun, M.J., Peto, R., Lopez, A.D., Monaco, J.H., Henley, S.J., Heath Jr, C.W. et al.Alcohol consumption and mortality among middle-aged and elderly U.S. adults. N Engl J Med. 1997; 337: 1705-1714
- Leevy, C.M. Fatty liver: a study of 270 patients with biopsy proven fatty liver and review of the literature. Medicine (Baltimore). 1962; 41: 249-276.
- 6. Charaka chikitsa sthana Udarachikitsa Adhyaya, 13/59, http://niimh.nic.in/ebooks/ecaraka/?mod=adhi assessed on 30th June 2016
- 7. http://sdlindia.com/media/Yakrut_Pleeha_ Visheshank_January_2011.pdf assessed on 30th June 2016.