Case Report

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EFFECTIVENESS OF YASHTIMADHU CHURNA, AMALAKI RASAYANA AND PHALATRIKADI KWATH IN HEPATOSTEATOSIS: A CASE REPORT

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ABSTRACT

Hepatic steatosis is defined as the accumulation of intrahepatic fat of at least 5% of liver weight, which may lead to metabolic dysfunction and inflammation in the liver. It is a common condition in 7% to 30% of people and gets worse over time into liver fibrosis, cirrhosis, and hepatocellular carcinoma. Modern science illustrates that there is no specific treatment protocol for fatty liver disease (FLD) except a healthy diet and exercise. In this case report, an individual was diagnosed with hepatosteatosis, having symptoms- pain in the abdomen with heartburn, burning in the throat, excessive thirst, belching, lassitude, constipation, nausea, and a yellowish appearance in the eyes. Fibroscan shows 77% of fat deposition with fibrosis (Grade F1). Based on the cardinal features of Udarashula, Chardi, and Peetanetra, the patient was assigned as a case of Parinam Shula and treated according to the line of management of Shula Chikitsa. After 3 months of drug intervention, there is a significant reduction in symptoms. Fibroscan shows a significant reduction in fat deposition (77% to 24%) with changes in fibrosis grading (from F1 to F0).

Keywords: Hepatosteatosis (NAFLD), *Glycyrrhiza glabra* L., Amalaki Rasayana, Parinam Shula.

INTRODUCTION

Non-alcoholic fatty liver disease is a broad spectrum of damage to the liver that can be due to hepatocyte injury, inflammatory processes, or fibrosis. It can range from milder forms to more severe forms. A hepatic steatosis is a moderate form of NAFLD defined as the accumulation of intra-hepatic fat of at least 5% of liver weight, which may lead to metabolic dysfunction, ballooning, and lobular inflammation.[1] Previous studies demonstrate the prevalence of the disease worldwide is 33.6%, out of which 24% of patients were diagnosed through fibrosis or CT scan.[2] Current studies believe that NAFLD develops in two ways. The first type of NAFLD has a narrow relationship with metabolic syndromes like diabetes mellitus, hypertension, and dyslipidaemia. The second type of NAFLD has a relationship with infections like viral hepatitis.[3] Initially, it is asymptomatic, but with the progression of the disease, symptoms arise, such as fatigue, pain in the upper right abdomen, and yellowing of the skin and eyes. In NAFLD, insulin resistance is the primary pathological mechanism that changes the enzyme levels in the liver.[4] In this case, an individual presents with clinical presentation pain in the abdomen: mild, dull, intermittent, occurring in the right upper abdomen with a burning sensation for 4 months, aggravates after meals, causes excessive thirst, heartburn and burning in the throat, lassitude, constipation, indigestion, nausea, and a yellowish appearance in the eyes. A fibroscan reveals 77% of fat deposition with fibrosis (Grade I), and endoscopy shows mild erosions in the ileocecal junction. Based on the cardinal feature (Pratyatm lakshan), udarashula, chardi, peetanetra case was assigned as parinam shula, and the patient was treated on the line of treatment of shula chikitsa.[5]

This case report illustrates that cases of NAFLD can be treated on the line of management of Parinam Shula Chikitsa. After the intervention, there is a significant reduction in deposited fat (from 77% to 24%) and grading of fibrosis (FI to F0).

Case Report

Patient Information

A 35-year-old male patient from Kanpur, Uttar Pradesh, who works as a shopkeeper, was diagnosed with 'hepatosteatosis' based on his fibroscan and USG report (21.09.2022). He has ongoing nausea and infrequent morning vomiting, loss of appetite, and discomfort in faeces (once within three days) over the past week, as well as a slight dull abdominal pain that lasts 24 hours but becomes worse after meals and is better after vomiting for 4 months. Heartburn, throat burning, excessive thirst, belching, lassitude, and a yellowish hue in his eyes have been bothering him for the past eight months.

History of Present Illness

The patient is suffering from mild pain in the right hypochondriac region, which persists 24 hours with a burning sensation, aggravated after meals and relieved after vomiting for the last 4 months. He was initially treated with allopathic medicines, viz.Cap. Omeprazole 1 BD, Cap. Amoxicillin 1 BD, Syp. Zinc (20 mg) 1 teaspoon TDS, by a contemporary physician for abdominal pain and acidity for 8 months, but he didn't get relief. He was advised for liver function test (LFT), fibroscan and upper GI endoscopy. The reports of fibroscan revealed 77% fat deposition with fibrosis Grade I. Endoscopy reports also show mild erosions in the ileocecal wall.

Past History: There is no history of fever, infective illness, lifestyle diseases, autoimmune diseases, endocrine or metabolic

disorders or any major surgery. But he was a chain smoker up to 10 years.
H/o Dietary habits: Vegetarian
Addictions: Tea thrice a day, Smoking 10 sticks/day for 10 years
Personal History: Appetite is reduced (food intake once a day)
Bowel: Severely constipated (once in 3 days)
Micturition: Normal (8-10 times/24 hours)
Family history: No family history

Physical Examination
General physical Examination: The patient was dull, weak and thin with icterus in the eyes. Vitals are stable. There is no paleness cyanosis seen. BMI was 24.09 kg/m^2. Blood pressure was 110/80 mmHg, pulse rate 74/min.

Per abdomen examination, there is mild tenderness in the right hypochondriac region. Hardening felt in the right hypochondriac region pointed towards the umbilicus. Bowel sounds were present.

Clinical Findings: liver fibroscan shows abnormal CAP and E (k pa) values, illustrating 66-70% hepatosteatosis with fibrosis (Grade I). Endoscopy reveals mild erosions in the ileocecal wall.

Diagnostic criteria

Table 1: Resemblance between Clinical features of NAFLD and Parinam Shula

Clinical features in favour of NAFLD	Clinical features in favour of Parinam shula
Pain in the abdomen is mild; intermittent type aggravates after a meal.	Udarashula
Heartburn and burning sensation in the throat	Hrit-kantha Daha
Loss of appetite, Abdomen distension with feeling of fullness	Aruchi, Adhman , Atopa
Nausea and vomiting	Chardi
Constipation	Vibandh
Yellowish appearance in eyes	Peetanetra

Table 2: Timeline

Time frame	Intervention
18-9-22	Diagnosed as Hepatosteatosis (Parinam shula) 1 year ago, came for the first time in Kayachikitsa OPD based on cardinal feature abdominal pain, vomiting and heartburn confirmed by investigations Fibroscan show fat deposition with fibrosis Grade 1.
22-12-22	The first time came in Kayachikitsa OPD for better treatment. Screening, history and physical examination were done. Based on the cardinal feature of 'Udarashula', the patient was diagnosed as a case of 'Parinam shula'.
22-12-22	The drug was selected on a line of management of Parinam shula; during drug intervention, a specific diet and lifestyle were advised.
23-3-23	After 1st follow-up improvement in the grading of symptoms mentioned in Figures 1-5
26-7-23	After 2nd follow-up with significant clinical findings mentioned in Table 3.

The rationale for drug selection

After the complete screening of the patient and consent was taken, based on cardinal features, Udarshula's patient was diagnosed as a case of Parinam shula and hence treated on the line of management of shula chikitsa. The drugs selected for treatment were Madhu-Amalaki Rasayana and Phalatrikadi kwatha, indicated as a drug of choice for shodhan. Simultaneously, the tablet Hepano is used as a hepatoprotective supplement for 20 days.

Drug Intervention
Madhuyashti churna, 3 gm with honey, BD, 1/2 hour before lunch and dinner.
Amalaki churna, 3 gm with honey, BD, 1/2 hour before lunch and dinner.
Phalatrikadi kwatha, 30 ml at night.
Tablet Hepano, 2 tablets BD after breakfast and lunch for 20 days.

RESULTS AND DISCUSSION

After drug intervention, there is a significant result in fibroscan investigation.

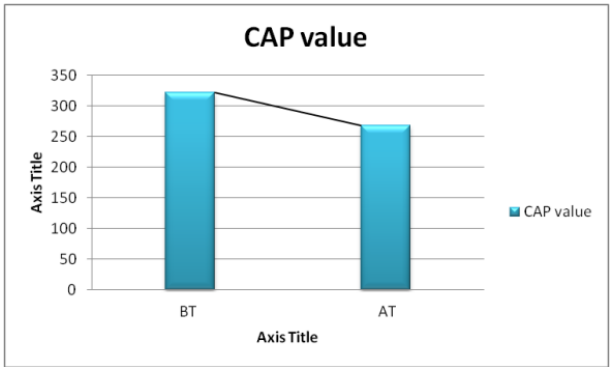


Figure 1: CAP value before and after treatment

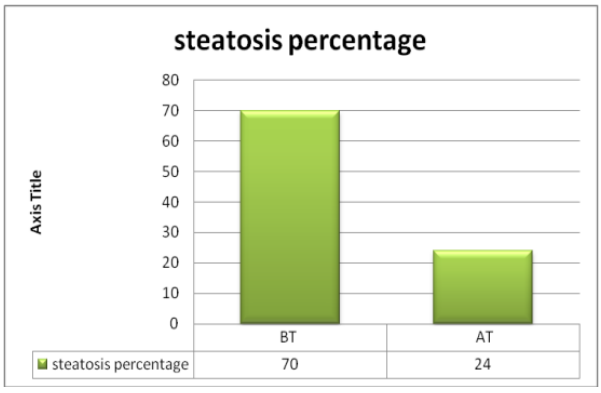
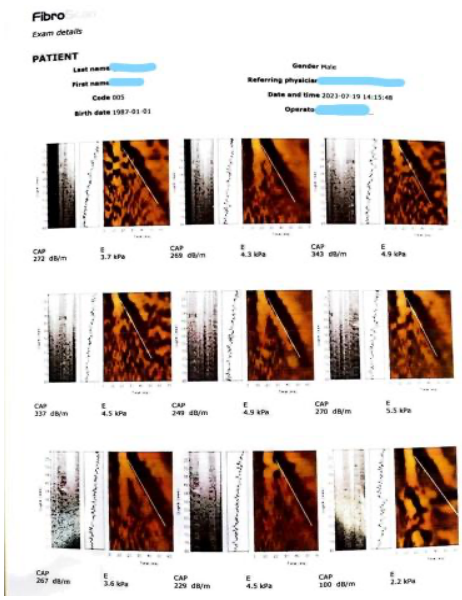


Figure 2: E (kpa) value before and after treatment



Figures 3 and 4: Reports of Fibroscan before treatment

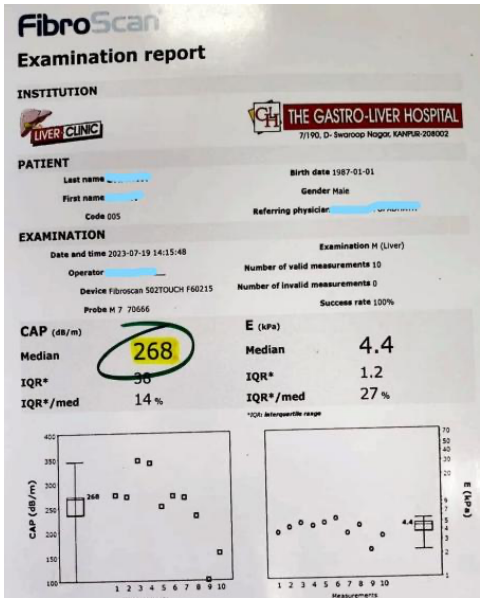


Figure 5: Reports of Fibroscan after treatment

Table 3: Assessment of Symptoms and Signs during intervention

Symptoms	18-9-22	22-12-22	23-3-23	26-7-23
Abdominal Pain	3+(24 hours aggravates after meal with burning sensation)	2+ (occur after a meal with a burning sensation)	Absent	Absent
Infrequent vomiting in the morning	Every morning	Once or twice a week	Absent	Absent
Loss of Appetite	Once a day	Once a day	Twice a day	Twice a day
Constipation	Grade III (once within three days)	Grade I (Regular with hard stool)	Regular evacuation	Regular evacuation

DISCUSSION

NAFLD and its advanced stages, which significantly impact global health, are of exploding interest. According to recent studies, the number of people with NAFLD will increase from 2015 to 2030, from 83.1 million to 100.9 million.[2] The severity of the damage to the liver cells and the ensuing fibrosis determines the range of abnormal liver conditions referred to as NAFLD. Hepatic steatosis (also known as fatty liver), one of these, is a milder condition marked by the buildup of intrahepatic fat that accounts for at least 5% of the weight of the liver and may cause metabolic dysfunction, ballooning, and lobular inflammation.[6] The most vital risk factor for NAFLD is metabolic syndrome, which is often associated with obesity, hyperglycaemia, hyperlipidaemia, cardiovascular disease, pollutants, smoking and genetic factors. Using genomic-wide association, a variant gene encoding a variant gene that reduces AST and ALT levels and raises the risk of hepatocellular inflammation was discovered by genome-wide association studies. These metabolites cause hepatocellular injury, mortality, and stress, which results in fibrogenesis.[3] Parinam shula is a type of Udarashula explained in Sushruta Samhita. The cardinal feature of the disease is Bhukt Jeeryate yat Shula," i.e., pain in the abdomen after digestion of food.[7] Ayurvedic classics illustrate that Ahara rasa is converted into Rasa dhatu after Paka with the help of Jatharagni, and the conjugation of ahara rasa through Bhutagni takes place in adhamashaya (yakrit). But faulty diet and lifestyle aggravate Tridosha (Samana vayu, Pachaka Pitta, and Kledaka Kapha) and hamper the Jatharagni, leading to aam medas and blocking (sanga) the Raktvaha srotas (mainly yakrit).

In this instance, a person exhibits a clinical presentation of mild, dull abdominal discomfort in the right upper belly, causing a burning feeling. The pain worsens after meals. Additionally, he reported experiencing extreme thirst, heartburn, throat burning, laziness, constipation, indigestion, nausea, and eyes that appeared yellow. His liver fibroscan exhibits aberrant CAP and E (k pa) values and displays grade-I hepatosteatosis in 66-70% of the liver. According to endoscopy, the ileocecal wall has minor erosions. According to the Ayurvedic diagnosis, the patient was designated as a case of Parinam Shula and managed according to shula chikitsa. The treatments chosen are Phalatrikadi kwatha for shodhana, Yashtimadhu churna, and Amalaki rasayana for shaman.

Phalatrikadi kwatha has the following properties: Pittahara, Pittarechaka, Yakrituttejaka, Deepan, and Tridoshahara. Phalatrikadi kwatha excrete Tridosha's from Pitta sthana to correct Agni. Previous studies have shown that Phalatrikadi kwatha is primarily effective in the treatment of liver cirrhosis, alcoholic hepatitis, fatty liver disease, and, more likewise, conditions of the liver. The combination of Madhuyashti churna and Amalaki churna is for shamana chikitsa. Yashtimadhu and Amalaki churna are madhur rasa, guru (heavy) snigdha guna (slimy), sheeta virya (cool), and madhur vipaka, with properties like balya (strength), Pittahara (antacid) raktprasadak (blood cleansing action), chardighna (anti-emetic), and vishaghna (anti-toxic).[8,9] As per modern studies, Yashtimadhu have antioxidant, anti-ulcer, anti-viral activities, and an immunomodulator effect. Amalaki, also called Amrita phala (life-giving fruit), has antioxidant, adaptogenic (rasayana), cell migration and binding properties (sandhaniya).[10] As per modern science, Amalaki is an excellent hepatoprotective agent.[11] There is a significant reduction in fibrosis in the liver. Simultaneously, remarkable changes were seen in the symptoms of the disease. According to this study, a fibroscan of the liver should be used to rule out yakrit vikriti in the case of parinam shula since the places of shula are jathar, parshva, and nabhi. After three months of medication

treatment, there is a considerable reduction in hepatic fat deposition (77% to 24%), and fibrosis Grade I changes to Grade 0. The use of therapeutic intervention, together with nidana parivarjana (avoid risk factor) and pathya-apathya (healthy diet), halts the progression of the illness and restores normal liver function.

CONCLUSION

The case study reveals the efficacy of Ayurvedic formulations in hepatosteatosis based on Ayurvedic principles applied in the treatment protocol of shula chikitsa. The prognosis of a disease is uncertain. There is a significant relief in symptoms within 2 weeks, and after 3 months, there is a substantial reduction in fat deposition in the liver (fat reduces from 77% to 24%), and fibrosis Grade I converts to Grade 0.

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We would like to thank the patient, who consented to have his case presented and published.

Patient perspective

The patient was delighted as symptoms were relieved soon after the initiation of Ayurveda treatment. The patient followed all the instructions and responded well to the prescribed treatment by healing the scars quickly.

REFERENCES

1. Nassir F, Rector RS, Hammoud GM, Ibdah JA. Pathogenesis and Prevention of Hepatic Steatosis. Gastroenterol Hepatol (N Y). 2015;11(3):167-175.
2. Browning JD, Szczepaniak LS, Dobbins R, *et al.* Prevalence of hepatic steatosis in an urban population in the United States: Impact of ethnicity. Hepatology. 2004;40(6):1387-1395. DOI:10.1002/hep.20466.
3. Nassir F, Rector RS, Hammoud GM, Ibdah JA. Pathogenesis and Prevention of Hepatic Steatosis. Gastroenterol Hepatol (N Y). 2015;11(3):167-175.
4. Kristina M, Utzschneier, Steven E. Kahn. The role of insulin resistance in non-alcoholic fatty liver disease. The Journal of Clinical Endocrinology & Metabolism. 2006;91(12): 4753-61.
5. Shastri Ambikadutta, Sushruta, Ayurveda tattvasandipika, Uttar tantra, edition 2016, Chaukhamba Sanskrit Sansthan, Varanasi, P 351-P352.
6. Kitade H, Chen G, Ni Y, Ota T. Non-alcoholic Fatty Liver Disease and Insulin Resistance: New Insights and Potential New Treatments. Nutrients. 2017;9(4):387. Published 2017 Apr 14. DOI:10.3390/nu9040387
7. Tripathi Brahmanand Madhavkar, Madhukosa, Shulanidanam,15th edition 2020, Chaukhamba Surbharati Prakashan, Varanasi. P 584-P585.
8. Punchihewa TKG, Uyeanege PP, Keerthirathne DHGANA. Systematic Review on Phalatrikadi Kashaya. Journal of Trend in Scientific Research and Development. 2021; 5 (4): 179-185.
9. Anagha Korhalkar, Manasi Deshpande, Priya Lele, Meera Modak. Pharmacological studies of Yashtimadhu (*Glycyrrhiza glabra* L.) in various animal models - A review. Global Journal of Research on Medicinal Plants & Indigenous Medicine; Koppa, 2013; 2 (3): 152-164.
10. Anti-ulcer Indian Herbal Pharmacopoeia, vol. I, II Regional Research Laboratory, Jammu, and Indian Drug Manufacturers Association, Mumbai, 1998.
11. Thilakachand K, Mathai R, Simon P, Ravi R, and Baliga-Rao M., Hepatoprotective Activities of the Indian Gooseberry (*Emblica officinalis*): A Review, Journal Food and Function, Royal Society of Chemistry, 2013; 4(10):1431.

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