

AYURVEDIC MANAGEMENT OF UDARA ROGA: A CASE STUDY

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India.

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ABSTRACT

Udara Roga, one among the *Ashtamahagada*, is a *Tridoshaja Vyadhi* predominantly caused by *Agnimandya*, leading to *Mala Sanchaya* and distension of the abdomen. Among its eight types, *Jalodara* closely resembles ascites and is characterized by *Kukshi Adhmana* (abdominal enlargement), *Karapada Shopha* (pedal edema), *Mandagni* or *Atyanta Nastagni*, and *Krushagatra* (emaciation). The pathology involves vitiation of *Udakavaha Srotas*, with *Talu* and *Kloma* described as its *Moola Sthana* in classical texts such as *Charaka Samhita*, *Sushruta Samhita*, and *Ashtanga Hridaya*. Ascites, commonly associated with liver cirrhosis and other systemic disorders, presents with fluid accumulation in the peritoneal cavity and has a high recurrence rate with conventional management. *Ayurveda*

emphasizes correction of the underlying pathogenesis through *Agni Deepana*, *Nitya Virechana* for *Dosha-Dushya Sammurchana Vighatana*, and strict *Pathya Ahara*. This case study of a 34-year-old male diagnosed with ascites demonstrated significant clinical improvement following *Ayurvedic* management including daily therapeutic purgation, digestive stimulants, and regulated diet. The approach addressed the root cause by restoring *Agni*, regulating *Srotasa*, and reducing fluid accumulation, highlighting the potential of *Ayurvedic* intervention in the management of *Udara Roga*.

KEYWORDS: *Udara Roga*, Ascitis, *Nitya Virechana*, *Pathya*.

INTRODUCTION

Udara Roga refers to generalized abdominal enlargement arising from various etiological factors and is enumerated among the *Ashtamahagada* in *Ayurveda*. It is considered difficult

to manage due to its deep-rooted origin in *Agnidushti*.^[1] When a person with *Mandagni*, or limited digestive capacity, engages in *Malina Ahara*, or *Viruddha Ahara*, *Pap Karma* which causes the vitiation of *Dosha*, there will be a buildup of *Dosha* because of the impaired digestion.^[2] Impairment of *Agni* along with *Mala Vriddhi* leads to vitiation of *Prana*, *Agni*, and *Apana Vayu*, resulting in obstruction of the upward and downward channels of circulation. The aggravated *Doshas* localize between *Twak* and *Mamsa*, producing progressive abdominal distension.^[3] The principal clinical manifestations include *Kukshi Adhmana* (abdominal enlargement), *Karapada Shopha* (pedal edema), *Mandagni* or *Atyanta Nastagni*, and *Krushagatra* (emaciation).^[3]

Udara Roga is classified into eight types—*Vatodara*, *Pittodara*, *Kaphodara*, *Sannipatodara*, *Plihodara*, *Baddhagudodara*, *Kshatodara*, and *Jalodara (Udakodara)*.^[4] *Yakritodara* is often considered under *Plihodara* due to similarity in etiology and management. Among these, *Jalodara* closely correlates with ascites, characterized by pathological accumulation of fluid within the peritoneal cavity.

In contemporary medicine, ascites commonly develops secondary to chronic liver disease and is associated with significant morbidity and mortality due to complications such as spontaneous bacterial peritonitis and hepato-renal syndrome. Although modern interventions like diuretics and transjugular intrahepatic portosystemic shunt provide symptomatic relief, recurrence of fluid accumulation is common. In contrast, *Ayurvedic* management aims at correcting *Agnidushti*, removing *Srotorodha*, and addressing the root pathology, thereby offering a holistic and sustained therapeutic approach.

In *Ayurveda*, the management of *Udara Roga* includes *Snehana*, *Swedana*, *Virechana Karma*, *Anuvasana Basti*, and *Siravyadha*, administered according to the patient's condition and stage of the disease.^[3] These therapies aim to eliminate vitiated *Doshas*, relieve obstruction, and restore impaired *Agni*.

Dietary regulation plays a crucial role in treatment. Recommended foods include *Rakta Shali* (*Oryza sativa*), *Yava* (*Hordeum vulgare*), and *Mudga* (*Vigna radiata*), along with meat soup of animals from arid regions, milk, cow's urine, and honey. Preparations such as *Yavagu* (rice gruel) and *Odana* (rice) are advised with cereal or meat soups processed with mildly sour substances, small quantities of ghee, pungent drugs, and *Panchamula*—*Bilva* (*Aegle*

marmelos), *Agnimantha* (*Clerodendrum phlomidis*), *Shyonaka* (*Oroxylum indicum*), *Kashmari* (*Gmelina arborea*), and *Patala* (*Stereospermum suaveolens*).^[3,4]

Additionally, *Takra* (buttermilk) and milk are specifically indicated in the management of *Udara* due to their therapeutic benefits in correcting *Agni* and reducing fluid accumulation.

Shodhana, *Shamana*, and *Nidana Parivarjana* constitute the main lines of treatment for any disease. In the present study, *Nitya Virechana Karma* was selected as the *Shodhana Chikitsa*.

Along with this, the patient was administered *Arogyavardhini Rasa* tablets and Tab. Liv, with *Sukhoshna Jala* as *Anupana*. *Kumari Asava* 20 ml was given twice daily with 20 ml of warm water. Additionally, a *Kwatha* was prepared from the *Churnas* of *Patola*, *Kutaki*, *Nimba*, *Haritaki*, and *Daruharidra* (2 gm each), and 25 ml of the prepared *Kwatha* was administered.

Nidana Parivarjana and appropriate *Pathyapathya* were advised to the patient. *Arka Patra Pattabandhana* was performed as a local measure.

Assessment of the treatment was carried out by comparing pre- and post-treatment laboratory investigations and by measuring the patient's abdominal girth.

CASE PRESENTATION

HISTORY OF PRESENT ILLNESS

A male patient 34 yrs old, residing in Hotagi road, Solapur visited Kayachikitsa OPD, Seth Sakharam Nemchand Jain Rugnalaya, Solapur on 29/10/2025 presented with complaints of swelling in foot and abdomen since 1 month ass with difficulty in breathing in supine position, disturbed sleep and reduced appetite, abdominal pain and abdominal distension.

1. Main Complaints

- *Udara vridhi* (increased abdominal girth), from 1 month
- *Kshudhamandhya* (decreased appetite), from 1- 2 months
- *Dourbalya* (general weakness), from 1 month
- *Ubhayapadashotha* from 1 month and
- *Udar Adhman* (abdominal distention) from 1 month
- *Pitvarniya mutrapravrutti* (yellowish urine)

Medical History

No history of Hypertension, Diabetes Mellitus, Malaria, Typhoid, Tuberculosis etc.

Surgical History

No any major surgical illness.

Family History

No evidence of this type of disease in the family.

Addiction

Alcohol consumption since 8 year.

PERSONAL HISTORY

- Bowel : irregular bowel
- Bladder: 2-3 times/day
- Sleep- disturbed
- Diet: mixed

Physical Examination

BP – 110/70 mmHg

P – 84/min

SPO2 – 98 %

O2 Respiratory rate – 20/min

O/E

Pallor – +++

Icterus – ++

Bilateral pedal edema – ++++

Systemic Examination

- ❖ Respiratory system - AEBE
- ❖ Cardiovascular system – S1 S2 normal
- ❖ Central nervous system - Patient was conscious and well oriented.
- ❖ Per abdomen

Inspection- Distended abdomen with the everted umbilicus.

Palpation – Tenderness over right hypochondric and epigastric region.

Percussion – Shifting dullness and fluid thrill were present.

ASTHAVIDHA PARIKSHA

- *Nadi* - 84/min
- *Mala* - unsatisfactory
- *Mutra* - 2-3times/day
- *Jivha* - *Saama*
- *Shabda* - *Spashta*
- *Sparsha* – *Ushna*
- *Druka* – *Pitavarni*
- *Akriti* - *Madhyam*

DASHAVIDHA PARIKSHA

- *Prakruti* - *Kapha Pitta*
- *Vikruti* - *Saman Vata, Apan Vata and Pachak Pitta, Kledak Kapha*
- *Sara* - *Madhyam*
- *Samhanana* – *Madhyam*
- *Pramana* - **Height :- 167 cm Weight :- 59 kg BMI :- 21.2 kg/m²**
- *Satva* – *Madhyam*
- *Satmya*– *Madhyam*
- *Ahara shakti*– *Madhyam*
- *Vyayam shakti*– *Madhyam*
- *Vaya*– *Madhyam*

MATERIAL AND METHOD

Centre of Study- This study was carried out in Seth Sakharam Nemchand Jain Ayurveda Rughalay, Solapur, Maharashtra.

Single Case study

Hetu (Causative factor) - Chronic alcoholism

SAMPRAPTI GHATAKA

- *Dosha*: *Tridosha (Pitta- Vata Pradhana)*
- *Dushya*: *Rasa, Sweda, Udaka vaha Srotas*
- *Adhishthana*: *Udara Pradesh*

INVESTIGATIONS

Sr. No.	Name of Investigation	Readings
1	BSL (R)	75
2	Sr. Creatinine	0.6
3	LFT	Bilirubin Total – 8.5 mg/dl Direct – 6.8 mg/dl Indirect – 1.70 mg/dl SGOT – 83 U/L SGPT – 44 U/L Alk Phosphatase – 318 IU/L
4	Urine Examination	Bile pigment, Bile salt present
5	S. Amylase	124.6
6	S. Lipase	119.7

USG**IMPRESSION**

Mild Hepatomegaly

Liver shows raised reflectivity with coarse parenchyma with surface nodularity

Liver shows persistently increased median values at elastography up to $Cs=2.83$ m/se(Normal range $Cs=1$ to 1.4 m/sec) (Values above 2.4m/sec suggests liver Cirrhosis).

Portal Vein (9.7mm) - Splenic vein (5.8MM) not dilates

Elo Mild Inter-loop Ascitis in abdomen & in pelvis –

Mild to moderate Splenomegaly-137mm shows normal reflectivity, Portal flow-8.7cm/sec is reduced than SV flow-40cm/sec.

Hepatic Artery Mildly dilated -shows increased flow --PSV-225cm/sec.

F/S/O

MILD HEPATOMEGALY, MILD TO MODERATE SPLEENOMEGALY WITH SIGNS OF LIVER CIRRHOSIS WITH PORTAL HYPERTENSION WITH MILD INTER-LOOP ASCITIS IN ABDOMEN & PELVIS -- NO SIGNS OF PV THROMBOSIS. NO E/O FOCAL MASS LESION/NEOPLASM SEEN IN LIVER. NO E/O PL EFFUSION.

Gall Bladder-partially distended - No e/o GB calculus or GB mass seen- No e/o GB wall thickening -No e/o GB wall odema. No e/o Acute or Chr. Cholecystitis or Pancreatitis. No e/o calculus in visualised CBD CBD/IHBR/PD are not dilated.

REST OF THE SCANNED ORGANS APPEAR NORMAL

TREATMENT PROTOCOL

INTERNAL MEDICINES (30 - 10 – 2025 to 18 – 11 – 2025)	
1.	<i>Arogyavardhini Rasa</i> 500mg bd with <i>Koshna Jal</i>
2.	<i>Kumari Asava</i> 10ml bd with <i>Koshna Jal</i>
3.	Tab. LIV 52 1bd with <i>Koshna Jal</i>
4.	<i>Shankhavati</i> 250mg bd with <i>Koshna Jal</i>
5.	<i>Trivruttavaleha</i> 10gm <i>nishakali</i> with <i>Koshna Jal</i>
6.	<i>Gokshuradi Guggulu</i> 3bd with <i>Koshna Jal</i>
7.	<i>Patol, Kutaki, Nimb, Haritaki, Daruharidra</i> each 2gm 5gm + 100 ml water – Heat – 25ml <i>Kashaya</i> bd

PANCHAKARMA (30 - 10 – 2025 to 18 – 11 – 2025)	
1.	<i>Arkapatta patra bandhan (Udarpradeshi)</i>

Pathya–Apathya

Strict dietary regulations were implemented for the patient.

He was maintained exclusively on *Godugdha* (cow's milk).

No other food items or water were administered during the two-month treatment period.

ASSESSMENT**Symptomatic relief**

Symptoms which were observed before and during the treatment such as abdominal distension, heaviness, nausea, bipedal edema, anorexia, icterus, pallor, weakness, muscle cramps, dyspnea on exertion were not observed at the end of therapy.

Sign/Symptom	Before Treatment	After Treatment
Icterus	++	No
Pedal Edema	Rt. 30 cm Lf. 31 cm	Rt. 28 cm Lf. 28 cm
Abdominal Girth	96 cm	90 cm

INVESTIGATION REPORT

SR. NO.	NAME OF INVESTIGATION	READINGS BEFORE TREATMENT	READINGS AFTER TREATMENT
1	CBC	HB – 13.5 g/dl WBC – 6200/mm ³ PLT – 119000/mm ³	HB – 12.2 g/dl WBC – 5000/mm ³ PLT – 234000/mm ³
2	LFT	Bilirubin Total – 8.5 mg/dl Direct – 6.8 mg/dl Indirect – 1.70 mg/dl SGOT – 83 U/L SGPT – 44 U/L Alk Phosphatase – 318 IU/L	Bilirubin Total – 3.9 mg/dl Direct – 2.4 mg/dl Indirect – 1.50 mg/dl SGOT – 74 U/L SGPT – 41 U/L Alk Phosphatase – 169 IU/L
3	S. Amylase	124.6 U/L	40.03 U/L
4	S. Lipase	119.7 U/L	43.73 U/L

DEPARTMENT

- USG
- ECG



S.S.N.J.A. Trust's S.G.R. Ayurved College attached,
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OPD NO. : 2003 DATE : 10/11/25

PATIENT'S NAME : Mr. DNYANESHWAR GHUMARE AGE : 34 Yr SEX : Male

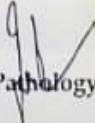
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LIVER FUNCTION TEST

INVESTIGATIONS	PATIENT'S VALUE	REFERENCE RANGE
Bilirubin Total :	3.9 mg/dl	0.2 - 1 mg/dl
Direct :	2.4 mg/dl	0.0 - 0.2 mg/dl
Indirect :	1.50 mg/dl	0.1 - 1 mg/dl
S.G.O.T.	74 U/L (37°C)	8 - 40 U/L (37°C)
S.G.P.T.	41 U/L (37°C)	0 - 49 U/L (37°C)
Total Proteins :	7.9 g/dl	6 - 8 g/dl
Albumin :	3.8 g/dl	3.5 - 5.5 g/dl
Globulin :	4.10 g/dl	1.7 - 3.0 g/dl
A:G ratio :	0.93	1.5 - 2.5
Alk.Phosphatase :	169 IU/L	35 - 123 IU/L

- Get Well Soon -


 Medical Technologist


 Incharge Pathology Dept.

Dr. Nitant Vora, M.D.[Path]










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PATIENT'S NAME : Mr. DNYANESHWAR GHUMARE
AGE : 34 Yr SEX : Male
REFERRED BY : S.S.N.J. Ayurvedic Rugnalaya. DATE : 30/10/25

BIOCHEMISTRY EXAMINATION

INVESTIGATIONS	PATIENT'S VALUE	NORMAL RANGE
S.Amylase	124.6 U/L	< 110
S.Lipase	119.7 U/L	Upto 70 U/L


Dr. Nitant Vora
M.D. [Path.]

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PATIENT NAME: == MR. DNYANESHWAR GHUMARE 34Y/M 10 pm | Sunday Closed DATE: 30/10/2025
 REFERENCE BY: == DR. S. S. AYURVED HOSPITAL—SOLAPUR.

USG ABDOMEN & PELVIS PORATL DOPPLER

Liver—Mild Hepatomegaly -Rt. Lobe—14.79mm, LL—6.33mm—Liver shows raised reflectivity with coarse parenchyma with minimal surface nodularity - Liver shows persistently increased median values at elastography up to Cs==2.83 m/sec (Normal range Cs==1 to 1.4 m/sec) (Values above 2.4m/sec suggests liver Cirrhosis)- No e/o Focal mass lesion or Neoplasm seen in Liver. No signs of obstructive jaundice. Portal Vein (9.7mm) - Splenic vein (5.8MM) not dilated - No e/o Portal vein / SV or Hepatic Vein / IVC thrombosis -CBD & IHBR/PD are not dilated. No e/o Dilated Collaterals seen in gastric bed or at porta or at Splenic hilum- E/o Mild Inter-loop Ascitis in abdomen & in pelvis - No e/o pleural effusion -Mild to moderate Spleenomegaly—137mm shows normal reflectivity. Portal flow—8.7cm/sec is reduced than SV flow -40cm/sec. Hepatic Artery Mildly dilated -shows increased flow --PSV -225cm/sec.

Gall Bladder — partially distended -- No e/o GB calculus or GB mass seen-- No e/o GB wall thickening --No e/o GB wall odema. No e/o Acute cholecystitis. No e/o calculus in visualised CBD.

Pancreas & Spleen - Mild to Moderate Spleenomegaly --137mm- Pancreas well seen shows normal size & reflectivity - No e/o focal mass in spleen or Pancreas. No e/o Acute or Chr. Pancreatitis.

Both Kidneys— Both Kidneys normal in size & reflectivity. No e/o calculus or Hydronephrosis or Hydropelvis or mass or scarring in Both Kidneys-- Renal Cortical thickness of Both Kidneys appear normal. No e/o Hydroureter on either sides, **RK—97MM X36MM, LK—94MMX48MM**

Urinary bladder—appears empty --normal. No e/o calculus or mass in Urinary Bladder. Bladder wall is not thickened. No e/o calculus at U-V Junction. Lower Ureters are not dilated. Prostate appears normal-14gms.

E/o Mild Inter-loop Ascitis in abdomen & pelvis No e/o Pleural Effusion. No e/o mass in abdomen or pelvic cavity. No e/o enlarged mesenteric or para-aortic Lymph or RIF nodes. Small Bowel loops in abdomen & pelvis are not dilated. No e/o Bowel wall thickening. No e/o mass seen in abdomen or in pelvic cavity. No e/o **dilated Appendix seen in RIF --No e/o inflammatory changes in RIF. No e/o Thickening of Colonic wall.** --- ON PAGE II

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PAGE II

IMPRESSION:== Mild Hepatomegaly -Liver shows raised reflectivity with coarse parenchyma with minimal surface nodularity - Liver shows persistently increased median values at elastography up to Cs==2.83 m/sec (Normal range Cs==1 to 1.4 m/sec) (Values above 2.4m/sec suggests liver Cirrhosis)- No e/o Focal mass lesion or Neoplasm seen in Liver. No signs of obstructive jaundice. Portal Vein (9.7mm) - Splenic vein (5.8MM) not dilated. No e/o Portal vein / SV or Hepatic Vein / IVC thrombosis -CBD & IHBR/PD are not dilated. No e/o Dilated Collaterals seen in gastric bed or at porta or at Splenic hilum- E/o Mild Inter-loop Ascitis in abdomen & in pelvis - No e/o pleural effusion -Mild to moderate Spleenomegaly—137mm shows normal reflectivity. Portal flow—8.7cm/sec is reduced than SV flow -40cm/sec. Hepatic Artery Mildly dilated -shows increased flow --PSV -225cm/sec. =F/S/O MILD HEPATOMEGALY—MILD TO MODERATE SPLEENOMEGALY WITH SIGNS OF LIVER CIRRHOSIS WITH PORTAL HYPERTENSION WITH MILD INTER-LOOP ASCITIS IN ABDOMEN & PELVIS. - NO SIGNS OF PV THROMBOSIS. NO E/O FOCAL MASS LESION / NEOPLASM SEEN IN LIVER. NO E/O PL. EFFUSION.

Gall Bladder -- partially distended -- No e/o GB calculus or GB mass seen-- No e/o GB wall thickening -- No e/o GB wall odema. No e/o Acute or Chr. Cholecystitis or Pancreatitis. No e/o calculus in visualised CBD. CBD/IHBR/PD are not dilated.

REST OF THE SCANNED ORGANS APPEAR NORMAL. NO E/O HYDRONEPHROSIS CALCULUS OR MASS IN BOTH KIDNEYS -NO E/O HYDROURETER ON EITHER SIDES. URINARY BLADDER (EMPTY) & PROSTATE APPEAR -NORMAL.

SMALL BOWEL LOOPS ARE NOT DILATED. SHOW NO WALL THICKENING. NO E/O PARA-AORTIC OR MESENTERIC OR RIF ADENOPATHY. NO E/O MASS IN ABDOMEN OR PELVIC CAVITY. NO E/O APPENDICITIS.

ADV: CLINICAL & LAB CORRELATION.

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DR. Mrs. P. S. DESHMUKH M.B.B.S., DMRE (Mum.)

Time : 11.00 am to 08.00 pm | Sunday Closed

PATIENT NAME: == MR. DNYANESHWAR GHUMARE 34Y/M DATE:-20/11/2025
 REFERENCE BY: ==DR. S. S. N. AYU. COLLEGE SOLAPUR.

USG ABDOMEN & PELVIS PORTAL DOPPLER-

Liver—Rt. Lobe—124mm, LL—39mm—Liver normal in size & shows raised reflectivity with coarse parenchyma with Minimal surface nodularity – Caudate Lobe normal -- Liver shows persistently grossly increased values at elastography up to Vs==3.79 m/sec (Normal range Vs==1 to 1.4 m/sec) (Values above 2.4m/sec suggests liver Cirrhosis)- No e/o Focal mass lesion or Neoplasm seen in Liver. No signs of obstructive jaundice. Portal Vein (8.3mm) not dilated - Splenic vein (4.6MM) not dilated - No e/o Portal vein / SV or Hepatic Vein / IVC thrombosis --CBD & IHBR/PD are not dilated. No e/o Dilated Collaterals seen porta or at Splenic hilum- Very minimal Inter-loop Ascitis in RIF and around Liver up to 2cc to 4cc- No e/o Pl. effusion on either sides - Mild Spleenomegaly—126mm shows normal in reflectivity. Portal flow—40cm/sec is similar to SV flow -38cm/sec. Hepatic Artery mildly dilated shows increased flow --PSV -140cm/sec.

Gall Bladder- contracted normal --shows Minimal GB wall odema—3.7mm—due to ascitis - No e/o GB calculi seen— No e/o GB mass seen -- No e/o Cholecystitis.

Pancreas & Spleen --Mild Spleenomegaly --126mm normal reflectivity --Pancreas well seen -normal size & reflectivity -- No e/o focal mass in spleen or Pancreas. No e/o Acute or Chr. Pancreatitis.

Both Kidneys—normal in size & parenchymal reflectivity. No e/o calculus or Hydronephrosis or Hydropelvis or mass or scarring in Both Kidneys. No e/o perinephric fluid around Kidneys. Cortical thickness & Reflectivity of Both Kidneys appear normal. No e/o Hydroureter on either sides. RK—93MM X33MM, LK—102MMX45MM.

Urinary bladder—Partially filled --appears normal. No e/o calculus or mass in Urinary Bladder. Bladder wall is not thickened. No e/o calculus at U-V Junction. Lower Ureters are not dilated. Prostate appears normal—11.7gms.

Very minimal Inter-loop Ascitis in RIF and around Liver up to 2cc to 4cc. —No e/o Pl. effusion - No e/o mass in abdomen or pelvic cavity. No e/o enlarged mesenteric or para-aortic Lymph or RIF nodes. Small Bowel loops in abdomen & pelvis are not dilated. No e/o Bowel wall thickening. No e/o mass seen in abdomen or in pelvic cavity. No e/o dilated Appendix seen in RIF --No e/o inflammatory changes in RIF. No e/o Thickening of Colonic wall.

--- ON PAGE II

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PAGE II

IMPRESSION:== F/S/O LIVER NORMAL IN SIZE SHOWS RAISED REFLECTIVITY AND COARSE PARENCHYMA WITH MINIMAL SURFACE NODULARITY WITH MILD SPLEENOMEGALY WITH EARLY SIGNS OF LIVER CIRRHOSIS WITH PORTAL HYPERTENSION WITH VERY MINIMAL INTER-LOOP ASCITIS IN RIF AND AROUND LIVER UP TO 2CC TO 4CC — PV & SV ARE NOT DILATED. NO E/O PL. EFFUSION - NO SIGNS OF PV THROMBOSIS. NO E/O FOCAL MASS / NEOPLASM SEEN IN LIVER.

GALL BLADDER- CONTRACTED NORMAL -E/O MINIMAL GB WALL ODEMA—3.7MM—DUE TO ASCITIS-NO E/O GB CALCULI SEEN—NO E/O GB MASS SEEN -- NO E/O CHOLECYSTITIS.

REST OF THE SCANNED ORGANS APPEAR NORMAL.

NO E/O HYDRONEPHROSIS CALCULUS OR MASS IN BOTH KIDNEYS—NO E/O HYDROURETER ON EITHER SIDES.

URINARY BLADDER & PROSTATE APPEAR — NORMAL—11.7GMS.

SMALL BOWEL LOOPS ARE NOT DILATED SHOW NO WALL THICKENING. NO E/O PARA-AORTIC OR MESENTERIC OR RIF ADENOPATHY. NO E/O MASS IN ABDOMEN OR PELVIC CAVITY. NO E/O APPENDICITIS.

ADV: CLINICAL & LAB CORRELATION.

DR. PRADEEP DESHMUKH, MD, DMRD.

DISCUSSION

In *Charaka Samhita*, *Acharya Charaka* describes various etiological factors of *Udara Roga*. In the present case, excessive intake of spicy and salty food along with *Mandagni* played a significant role in disease manifestation. From an *Ayurvedic* perspective, *Jalodara* involves vitiation of *Udakavaha Srotas* and *Kloma*, which are considered the *Moola* (root) of *Udakavaha Srotas* as described by *Charaka* and *Sushruta Samhita*. *Kapha*-induced obstruction leads to dysfunction of *Kloma* and associated structures, resulting in abnormal fluid accumulation.

This pathological process can be correlated with fluid collection in the peritoneal cavity, resembling ascites, which may be further aggravated by systemic factors. Fundamentally, *Udara Roga* originates from *Agnimandya* and *Tridosha* vitiation. Therefore, the principle of “*Agni Samrakshana*” is central to management. Restoration and maintenance of *Agni* according to the patient’s physiological and pathological state remain essential for effective *Ayurvedic* management of *Udara Roga*.

The patient was strictly restricted from taking any *Ahara* or *Jalapana*^[5], as the *Doṣhas* were *Sanghatita* in *Koshtha*, leading to *Agnimandya*.^[6,7] In this condition, administration of *Godugdha* was advised.^[8] Owing to its *Laghu*, and *Mṛudu Virechana* properties, it helped to stimulate and strengthen the patient’s *Jatharagni* while providing adequate *Bala*. These qualities supported the restoration of digestive capacity and promoted *Kshudha prachiti* (return of proper appetite).

The *Chikitsa Sutra* of *Jalodara* is called “*Nitya Virechana*.” Regular therapeutic purgation is indicated to eliminate the Sanga of vitiated *Doṣhas* and accumulated fluid from the *Koṣhṭha*. The *Yakrut* (liver) is considered the *Mula Sthana* of *Rakta*, and due to the *Ashraya–Ashrayi Sambandha* between *Rakta* and *Pitta*, *Virechana* is regarded as the most appropriate therapy for aggravated *Pitta Doṣha*. By facilitating systematic expulsion of vitiated *Doṣhas* and excess fluid, *Virechana* helps reduce abdominal distension and edema. In the present case, initiation of *Nitya Virechana* resulted in marked improvement in the cardinal symptoms of *Jalodara*. *Trivrutavaleha* functioned as a *Mridu Virechaka* here⁹, facilitating controlled elimination of *Doshas*.

Arogyavardhini Rasa acted as *Deepana-Pachana* and *Yakrit-uttejaka*, correcting *Agnimandya* and supporting hepatic metabolism. *Kumari Asava* enhanced *Jatharagni* and exhibited mild

Virechaka action, aiding in *Pitta* regulation. Liv-52 provided hepatoprotective support and improved appetite. *Shankhavati* corrected *Ajeerna* and reduced *Ama* formation through its potent *Deepana* effect. *Gokshuradi Guggulu* contributed through its *Mutrala* and *Shothahara* properties, thereby reducing fluid retention and edema. The *Kashaya* prepared from *Patola*, *Kutaki*, *Nimba*, *Haritaki*, and *Daruharidra* exerted *Tikta-Kashaya* predominance, promoting *Pitta-Kapha Shamana*, *Rakta Shodhana*, and *Kledahara* action.

Udara Pattara Bandhana with *Arka Patra* was performed every day to stop the *Vata* from further expanding the abdomen.^[10,11]

Clinically, there was significant improvement in all assessment parameters. Abdominal girth reduced from 96 cm to 90 cm, bilateral pedal edema decreased markedly, and icterus resolved after completion of therapy. Symptoms such as *Udara Adhmana*, *Kshudhamandya*, *Dourbalya*, dyspnea, and anorexia subsided completely. Laboratory investigations also showed considerable improvement, particularly in serum bilirubin, liver enzymes, and platelet count, indicating restoration of hepatic function.

The sequential therapeutic approach of *Agni Deepana*, *Nitya Virechana* for *Dosha Nirharana*, *Mutrala* and *Kledahara* measures for fluid reduction, strict *Pathya Palana*, and *Yakrit-Poshaka Shamana Aushadhi* effectively addressed the underlying *Samprapti* rather than providing only symptomatic relief. By correcting *Agnidushti*, removing *Srotorodha*, and restoring *Udakavaha Strotas* function, the treatment achieved sustained clinical remission. The absence of recurrence and marked symptomatic as well as biochemical improvement at the end of therapy suggest that early, well-planned *Ayurvedic* intervention can play a significant role in the effective management of *Udara Roga (Jalodara)*, even in cases associated with hepatic pathology.

CONCLUSION

Udara Roga, particularly *Jalodara*, is a serious condition arising primarily from *Agnimandya* and *Tridosha* vitiation, leading to *Srotorodha* and pathological fluid accumulation. The present case demonstrates that management based on classical principles described in the *Charaka Samhita*—with emphasis on *Agni Deepana*, *Nitya Virechana*, *Shamana Aushadhi*, and strict *Pathya Palana*—can effectively address the root pathology rather than merely providing symptomatic relief.

The therapeutic approach corrected *Agnidushti*, eliminated vitiated *Doshas*, reduced *Kleda*, restored *Udakavaha Srotas* function, and supported *Yakrit* health. Significant clinical improvement in abdominal girth, pedal edema, icterus, appetite, and laboratory parameters substantiates the efficacy of the treatment protocol.

This case highlights that early diagnosis, proper *Nidana Parivarjana*, and a systematically planned *Ayurvedic* intervention can yield encouraging outcomes in *Udara Roga (Jalodara)*, even in cases associated with hepatic involvement. Further clinical studies with larger sample sizes are recommended to validate these findings and establish standardized treatment guidelines.

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