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CASE STUDY

Management of Udar Vyadhi with Ayurveda with Special Reference to Ascites

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ABSTRACT

Udara roga means any etiology-related generalized abdominal distension or hypertrophy. Ascites is the most prevalent sign of liver malfunction, and despite the use of sophisticated medical equipment, there is still no proven method of curing ascites in patients as mentioned in Acharya if it is not treated the patient will die soon. Modern therapies only offer temporary relief with time-dependent recurrence, yet fluid continues to build up in the abdominal cavity. *Jalodara* is one of the eight types of *Udar roga* described in *Ayurveda.* It is mentioned in all three *brihatrayee* texts (*Charaka Samhita, Susruta Samhita, and Ashtanga Hridaya*). *In Ayurveda, Udara roga* covers conditions such as gaseous distension, hepatosplenomegaly of various etiologies, intestinal blockage, and intestinal perforation in addition to ascites and fluid buildup in the peritoneal (common presentation is abdominal distension throughout). Its pathophysiology is thought to be primarily caused by mandagni. *Udakavaha srotas* and *Ambu vaha srotas* are mentioned in relation to *Jalodara* pathology in all three sources. *Talu and kloma* make up the *moola* (*base*) *of Udakavaha srotas*. In *Ayurveda, kloma* is a contentious subject. Some writers liken it to the pancreas. *Jalodara* is said to be caused by the dusti (fault) of *udakavaha srotas or (kloma). Ayurvedic m*edicine offers relief in such circumstances without causing any negative side effects. Ayurvedic care of *Udar* (ascites) with medications such as provocation of digestion daily therapeutic purging, stimulants for hepatic function, and only milk diet that operates on the basis of the pathophysiology of ascites and by splitting down of pathogenesis produces good results in management. Cirrhosis of the liver is the most frequent cause of ascites in the developed world. Other causes include pancreatitis, TB, cancer, heart failure, and hepatic vein blockage. The highlighted mechanism in cirrhosis involved elevated portal blood pressure and blood vessel dysfunction. The true cause and severity of ascites have a significant impact on a person's prognosis. This article discusses several *Acharya* treatments from an Ayurvedic perspective.

1. INTRODUCTION

One of the main illnesses brought on by *Agni dushti* is *udara*.[1] When a person with *mandagni,* or limited digestive capacity, engages in *malina ahara, or viruddha ahara, pap karma,*[2] which causes the vitiation of *dosha,* there will be a buildup of *dosha* because of the impaired digestion. As a result, the upper and descending channels of circulation get blocked and *Prana, Agni, and Apana* become vitiated. Therefore, the *doshas* become trapped between the skin and muscle, resulting in a significant expansion of the belly and *Udara*.[3,4] *Talu and kloma* make up the *moola* (base) of *Udakavaha srotas*. *In Ayurveda, kloma* is a contentious subject. Some writers liken it to the pancreas.

This is the *samanya samprapti of Udara* as described in the classical literature, which may vary in various ways from person to person. It is crucial to interpret the *samprapti* in each patient by analyzing the *hetus*, vitiation of the *dosha* by *vikalpa samprapti*[5] (which *guna* of the *dosha* is primarily responsible for its vitiation), and its *sammurchana* with the *dushyas* further leading to the manifestation of disease, that is, the journey of a *hetu* up to disease manifestation should be well understood).[6] Once the *samprapti* is seen, it is simple to treat as necessary.

This essay examines a method for doing *Udara chikitsa* in which the *samprapti* was visualized and the *chikitsa* was performed in accordance

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with that visualization employing a variety of *siddhanta* and *shodhana* and *shamana aushadhis*. *Charak Samhita* and *Pran-Apan-Agni dushti* are the main pathology of *Udar* which is described.[6]

According to *Ayurveda*, the *udar roga* is one of the eight major ailments (*ashta maha gada*). The most important part to perform in its development is *Mandagni*. There are eight different sorts of abdominal diseases known as *udar* roga that are listed in texts: *Vadodara, pittodara, kaphodara, sannipatodara, pleehodara, baddhodara, kshatodara, and udakodara* or *jalodara*.[6] In general, *jalodara* is understood to be a condition in which the *udara* (abdomen) becomes filled *with jaliya ansh or jal* (body fluid). It is known to be an illness that is challenging to treat. Ascites, or free fluid inside the peritoneal cavity of the belly, is what it is called in modern times. It is the most typical liver characteristic.

1.1. Case

A female patient of age 40 years was having complaints of abdominal distension, heaviness of the abdomen, breathlessness, nausea, facial and periorbital edema, dyspnea on exertion, loss of appetite, and oliguria for the past 8 days. Earlier patient was taking treatment for liver cirrhosis with acute onset of ascites, she got hospitalized and also did tapping for 2 times within a month. As was suffering from severe breathlessness complained at that span of time 1 and 1/2 L of fluid drained through tapping, she relived from symptoms but after some time, she relapsed with all symptoms. After 5 months of all treatment patterns adopted, she came to our institute for further treatment.

1.2. Main Complaint

- *Udara vridhi* (increased abdominal girth), from 6 months
- *Kshudhamandhya* (decreased appetite), from 7–8 months
- *Dourbalya* (general weakness), from 7–8 months
- *Ubhayapadashotha* from 7–8 months and
- *Krishnavarna* (bilateral pedal edema and discoloration) from 6 months.

2. CASE REPORT

2.1. History

- N/H/O - Malaria, typhoid, Koch's, HTN, DM, etc.
  - Left kidney reveals gross hydronephrosis with calculus measuring 19 mm noted within the left PUJ.
  - Esophageal candidiasis
  - Low-grade esophageal varices

2.2. Surgical History

- Left forearm operated in view of fracture (6 months ago) (January 2022).

2.3. Family History

No evidence of this type of disease in the family.

2.4. Addiction

- Alcohol consumption – No.

2.5. Physical Examination

- BP – 110/70 mmHg P – 84/min
- SPO_2 – 97 % O_2
- Respiratory rate – 20/min
  - Temperature
- Pallor – +++
- icterus – ++
- Bilateral pedal edema – ++++
  - Facial and periorbital edema ++
  - Mild pallor and icterus +.

2.6. Systemic Examination

- Inspection: Distended abdomen.
- Palpation: Tenderness in the right hypochondriac region. *Hepatomegaly* – 3 cm below right costal margin.
- Percussion: Fluid thrill – present shifting dullness – present.

2.7. Systemic Examination

1. Respiratory system - Air entry was reduced on both sides with crepitations bilaterally.
2. Cardiovascular system - Regurgitation sound was present over aortic, pulmonary, tricuspid, and mitral areas
3. Central nervous system - Patient was conscious and well oriented.
4. Per abdomen
   - Inspection- Distended abdomen with the everted umbilicus.
   - Palpation – Hepatomegaly of three fingers was present.
   - Percussion – Shifting dullness and fluid thrill were present.

2.8. Investigations

Table 1 shows laboratory investigations.

Table 2 shows USG. The investigation reports before and after treatment are cited from Figures 1-12 below.

3. MATERIALS AND METHODS

3.1. Treatment

1. Diet – Patient was advised to take only *Shunthi, trikatu siddha godugdha* on *kshudhaprachiti* for an initial 1 month where diet and salt were prohibited. *Laghu ahara such as laja* and *krushara* was started after 2 weeks. *Lavana varjit mansrasa* was advised after 1 month of therapy.
2. Castor oil 10 ML with *Triphala churna* (before drinking milk earlier or before food later on) for up to 2 weeks.
3. *Kutaki churna* 5 g at night time was given for 20 days and *Shunthi kwatha* 20 ml
4. Tablet calcimax forte 1 OD was started after 12 days.
5. *Udara pattabandhan* with *Hingvastak churna* and *Eranda patra* was done throughout the therapy.
6. Nebulization with duoline twice a day for 15 days and then with NS was carried out for 8 days and was kept SOS thereafter.
7. Previous allopathic treatment for heart diseases was continued as it is.
8. All vital parameters such as BP, RR, SpO_2, temperature, BSL (R), weight, input and urine output, stool color, and abdominal girth were monitored regularly.
9. Treatment schedule – Tables 3 and 4.
10. Symptomatic relief: Symptoms which were observed before and during the treatment such as abdominal distension, heaviness, nausea, facial and periorbital edema, anorexia, oliguria, icterus, pallor, weakness, muscle cramps, giddiness, dyspnea on exertion were not observed at the end of therapy.

11. Systemic examination: Air entry was almost equal bilaterally and crepitations were reduced. Grade of murmurs was reduced to Grade 3. Abdominal distension was not noted and shifting dullness and fluid thrill were absent after treatment.
    - First follow-up was taken at 26 May 2023 after 10 days of discharge from the hospital 26 May 2023 to 14 June 2023 same treatment was continued and
    - Second follow-up was taken on 14 June 2023
    - Third and last follow-up was taken on 10 July 2023.

3.2. *Pathya-Apathya*

Diet was restricted to the patient.

She was kept on only cow milk (*trikatu siddha godugdha).*

No food items and water were given to the patient for 2 months.

4. RESULTS

Significant results were found in all symptoms such as abdominal girth, icterus, pallor, bipedal edema, and general weakness [Tables 5 and 6].

5. DISCUSSION

In the *Charaka Samhita*, *Acharya Charaka* lists numerous causes of *udara roga*. The patient in the present situation was overindulging in spicy and salty foods and had a low digestive fire (*mandagni*).

We note that when characterizing the pathophysiology of *jalodara* from an *Ayurvedic* perspective, *Udakavahi srota and kloma* are frequently cited. The *Udakavahi srotas* defect is brought on by *kapha* blockage, which is causing dysfunction in the *kloma* and surrounding structures. *Kloma* is a controversial but significant subject in *Ayurveda*, and *Charaka and Susruta* have referred to it as the *mool (basis) of udakavaha srotas.*

As a result, hepatic lymph begins to drain into the peritoneal cavity. Ascites develops as ascites when additional factors such as hypoalbuminemia and hyperaldosteronism augment and intensify them.

Alterations to the *Agni* state, which are influenced by vitiated *Tridosha,* are the basic pathophysiology of *Udara Roga,* and thus, to treat any illness, it is imperative to adhere to the *"Agni Samrakshana"* principle. To return *Agni* to normalcy from an altered state, attention should be paid to *Agni* in relation to their healthy state, disease state, and diagnostic status. The lifestyle has a balanced and healthy quantum. Clinicians must concentrate clinically on the *Agni s*tates of their patients.

The patient previously had a history of *Udara,* which was treated at the time with allopathic medicine and eventually went away, but some *doshas* were still there, or *kinchit avashishta dosharupa moola*. Due to early menopause, IUD, MTP, and *Jwara itihasa satatya* (history of recurrent febrile sickness), the patient had *dhatu kshay*. Because the *vyadhi ghatka bhava* (which prevents the occurrence of disease) such as *vyayama* and *vidhiyukta ahara vihara* was absent, further *hetusevana* caused the *kinchit avashishta dosharup moola,* which in turn caused *Udara* to reoccur.[7] In addition, all of these circumstances cause the *khavaigunya* of *Udaka, Prana, Rasa,* and *Pranavaha srotas* to grow, which causes vitiated *doshas* to lodge there and manifest as *Udara.*[8]

Since *Nidana parivarjana* is the fundamental *siddhanta* for *samrapti vighatan*. Hetus are *santarpanjanya* which leads to gross in *lakshana*.[9] The patient was forbidden to consume any *ahar* or *jalapana*.[10] Because the *doshas* are sanghatita in koshta, causing *agnimandya*[11,12] only *Shunth*i and *trikatu siddha godugdha*[13] with *deepana, laghu, mrudu virechana,* will give *bala* to *rogi's jhatragni*.[14-16] Qualities were provided on *kshudhaprachiti* for the first two weeks. *Triphala kwatha*,[17] which has the properties to remove extra water, was supplied. It also has *deepana, laghu, ruksha,* and *mrudu anulomana.*

Nitya virechana should be administered because *Srotas avarodha* and *dosha atimatra upchaya*, or an excessive buildup of *dosha in Udara,* exist.[13,18,19] However, because the patient had *durbala*, *mrudu virechana* was given daily[20] and in smaller amounts (*alpasha*), along with *kutaki churna*[21] (*ruksha and deepana*), for a period of 1 month. Throughout the course of the therapy, *Udara Pattara Bandhana with Hingvastak and Eranda Patra* was performed every day to stop the *Vata* from further expanding the abdomen.[22-24]

However, the patient was experiencing *dourbalya, bhramaprachiti, ubhaya pad pindik tod veshtanam*, and *grathit mala pravrutti*, which show a change in *vyadhi avastha*, which is how *chikitsa* should be changed, that is, when enough *Rukshana* and *drava shoshana* are attained.[25] As a result, after a serum electrolyte assessment, *mrudu Sneha virechana* was initiated as *bruhana chikitsa*, followed by *laghu ahara, lavana varjit mansarasa, Shastika shali pinda sweda* over both extremities, tablet calcimax forte, and *mrudu Sneha virechana.*

With the aid of the aforementioned treatment, the *sara kitta vimochana* – a function of *prakrut agni* – was restored, leading to an increase in urine output and the normalization of bowel habits. With the aid of the aforementioned treatment, the obstruction in the circulating channels was also removed, which allowed the bodily function of *uttarotar dhatuposhana* to resume. Therefore, an increase in RBC and Hb was noted. Nebulization and vasa *patra swarasa* were administered to relieve the chest congestion. All crucial variables were regularly checked.

The patient received *Ayurvedic* treatment using an integrative strategy. Treatment for *udara* involves the external application of *pattbandhan* (abdomen belt), *nitya virechana* (purgative), *agnideepan* (raise appetite), *balaprapti* (increase strength), and *yakrituttejjak* (stimulant for hepatic function). Significant improvements were seen in the form of reduced pedal edema, reduced belly girth, increased hunger, and increased strength. *Chikitsa siddhanta* is *"nityameve virechayet"* for *udarvyadhi*. When the *virechana jatharagni* and *dhatvagni* rise, *Virechana* checks the incorrect *jatharagni* and *dhatvagni.*[26]

Due to persistent constipation in ascites, it has a laxative effect that aids in removing toxins from the body.[27] It has cholagogue, hepatoprotective, and stimulating effects on the liver. As a result, because it has a laxative and diuretic activity that aids in eliminating extra fluid from the body, it is helpful in cases of generalized edema and ascites. Hepatoprotective activity known as *yakrituttejak* is performed by *arogyavardhini* vati.[28]

Virechana activity is prominent in castor oil with *triphala churna,* which is used in situations of ascites. It regulates the bowels in cases of chronic constipation and, through the action of its *ushnatikshnavyavayi gunas*, promotes therapeutic *mutral* (a diuretic), *shothaghna* (which lessens edema), and purgation. *Punarnavasava* helps the kidneys function better. Using *mridu swedan, patrapatta bandhan* avoids *vata prakop* and supports diuretic activity. The patient gains strength from cow milk without the body's fluid level rising. According to Ayurveda*, Udar is asadhya vyadhi* (incurable), yet we can provide the patient

with symptomatic relief, a decrease in fluid, and an improvement in quality of life.

The patient received *Gomutra*[29] for 2 weeks, *Cow's Mutra* (urine) *Tikshna* and *Ushna Guna* strengthens *Agni* (digestive force). It removes *Strotosanga* (channel obstruction) and aids in *Samprapti Vighatana* (pathogenesis breakdown) thanks to its *Ushna* (hot), *Tikshna* (sharp), and *Ruksha* (dry) *Guna*. The elimination of *Apya Dosha* (water retention) occurred concurrently.

Nitya Virechana – The *Chikitsa Sutra of Jalodara* is called *"Nitya Virechana." Virechana* is required to disperse the *Sanga* of all *Dosha* and retained fluid and separate them. The *Mula Sthana* (central location) of *Rakta* is the liver *(Yakrita*). Because of the reciprocal reliance between *Rakta* and *Pitta (Ashraya* and *Ashrayi Sambandha*), purgation is the greatest cure for a vitiated *Pitta Dosha*. By reducing fluid in the abdominal cavity*, virechana* also reduces belly girth and edema.[30] More results were achieved in all the symptoms after starting daily therapeutic purgation.

Arogyavardhini Vati is esteemed for its advantages, particularly for the liver. *Arogyavardhini* supports equilibrium, a healthy digestive system, and preserves the function of the liver. *Katuki* (Picrorhiza kurroa Royle ex Benth.), which functions as *Pitta Virechana* and acts on *Yakrita,* is its major component.[31,32] Ascites can result from any pathology of the liver, heart, kidney, etc.; however, ascites from liver illness is challenging to cure, necessitating the necessity to address the pathology from its underlying source. These medications were given because the patient in the current instance also has hepatomegaly. It makes the liver work better.

4.1. *Punarnavadi Kwatha* and *Punarnavadi Mandura*

Udara Roga should be treated with *Punarnavadi Kwatha,* which also lessens *shotha* (swelling). It also corrects *Shwasa* and *Pandu*. The *Kwatha* was prescribed since the patient was on *Jalodara* and experienced all of these symptoms, which had a major impact on all of the symptoms. In addition, *Mandura* is recommended for *Pandu* (anemia), *Shotha* (edema), and *Shwasa* (bronchial asthma), all of which resulted in a notable improvement for *Pandu*.[33,34]

Gandharva Haritaki was given for *Vatanulomana* purpose. *Apana Vayu* is also included in *Samprapti of Jalodara.* Because of *Gandharva Haritaki, Apana Vayu* moves toward its normal path and it helps counteracting pathology. It also possesses a laxative effect.

6. CONCLUSION

All of the *Jalodara* symptoms have improved as a result of daily therapeutic purging, diet restrictions, and *Ayurvedic* medications. In this case, the abdominal girth, pedal edema, and all of the aforesaid symptoms were greatly improved with no adverse effects. Despite the fact that the patient was only on a milk diet, no negative effects were observed during or after treatment. In this example, *Arogyavardhini Vati* was administered for 45 days constantly, but no negative effects were observed; hence, it can also be stated that metallic preparations are not damaging to the body if given in appropriate doses but rather provide additional benefits. As a consequence, it may be concluded that *Ayurvedic* medications including *Nitya Virechana* provide better results in ascites with no side effects.

Udara is mostly influenced by *Agnidushti, Doshasanchaya, and Srotorodha.* The visualization of *samprapti* in each patient using *Hetu vinishchay, anshansh kalpana* of *dosha prakopa,* and *dosha* leading to further vitiation of *dushya* should be properly understood. If done correctly, *Samprapti vighatana* based on *Nidana parivarjana*, *Agnideepana, Srotas shodhana,* and *Nitya virechana* can treat *Udara* if done in line with the *Vyadhi avastha, Rugna bala*, *Aushadhi matra,* and *Kala*.

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11. CONFLICTS OF INTEREST

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REFERENCES

1. Acharya YT. Charak Samhita of Charaka, Chikitsa Sthan. 2017th ed., Ch. 13., Verse no 9. Varanasi: Choukhamba Surbharati Prakashan; 2017. p. 491.
2. Ganesh G. Sartha Vagbhat Rajesh Prakashan Chikitsa Sthan. Ch. 15. Shlok no.2. Varanasi: Choukhamba Surbharati Prakashan; 2018. p. 300.
3. Acharya YT. Charak Samhita of Charaka, Chikitsa Sthan. 2017th ed., Ch. 13., Verse no 11. Varanasi: Choukhamba Surbharati Prakashan; 2017. p. 491.
4. Garde GK. Sartha Vagbhat, Nidana Sthan. Reprint 2012 ed., Ch. 12., Verse no 2. Varanasi: Choukhamba Surbharati Prakashan; 2017. p. 197.
5. Acharya YT. Chakrapani Teeka, Charak Samhita of Charaka, Nidan Sthan. 2017th ed., Ch. 1. Verse no 11(5). Varanasi: Choukhamba Surbharati Prakashan; 2017. p. 197.
6. Acharya YT. Chakrapani Teeka, Charak Samhita of Charaka, Nidan Sthan. 2017th ed., Ch. 1. Verse no 11. Varanasi: Choukhamba Surbharati Prakashan; 2017. p. 196.
7. 7Acharya YT. Charak Samhita of Charaka, Chikitsa Sthan. 2017th ed., Ch. 30. Verse no 327. Varanasi: Choukhamba Surbharati Prakashan; 2017. p. 649.
8. Acharya YT, editor. Sushruta Samhita of Sushruta, Sutra Sthan. 1992 Reprint edition., Ch. 24. Verse no 10. Varanasi: Choukhamba Publications; 1992. p. 10.
9. Acharya YT. Sushruta Samhita of Sushruta, Uttara Tantra. 1992 Reprint edition., Ch. 1. Verse no 25. Varanasi: Choukhamba Publications; 1992. p. 597.

10. Acharya YT. Charak Samhita of Charaka, Chikitsa Sthan. 2017th ed., Ch. 13. Verse no 100. Varanasi: Choukhamba Surbharati Prakashan; 2017. p. 496.
11. Acharya YT. Charak Samhita of Charaka, Chikitsa Sthan. 2017th ed., Ch. 13. Verse no 96. Varanasi: Choukhamba Surbharati Prakashan; 2017. p. 496.
12. Garde GK. Sartha Vagbhat, Chikitsa Sthan. Reprint 2012 ed., Ch. 15. Verse no 121. Varanasi: Choukhamba Surbharati Prakashan; 2017. p. 306.
13. Shastri AD. Sushruta Samhita of Sushruta, Chikitsa Sthan. 2014 Reprint edition., Ch. 14. Verse no 9. Varanasi: Choukhamba Publications; 2014. p. 87.
14. Acharya YT. Charak Samhita of Charaka. Chikitsa Sthan. 2017th ed., Ch. 13. Verse no 194. Varanasi: Choukhamba Surbharati Prakashan; 2017. p. 500.
15. Shastri AD. Sushruta Samhita of Sushruta, Chikitsa Sthan. 2014 Reprint edition., Ch. 14. Verse no 19. Varanasi: Choukhamba Publications; 2014. p. 90.
16. Garde GK. Sartha Vagbhat, Chikitsa Sthan. Reprint 2012 ed., Ch. 15. Verse no 131. Varanasi: Choukhamba Surbharati Prakashan; 2017. p. 307.
17. Tripathi B. Bhavamishra, Bhavprakash Nighantu, Haritakyadi Varga. Reprint 2010. Verse no 42-43. Varanasi: Choukhamba Bharati Academy; 2010. p. 12.
18. Shastri AD. Charak Samhita of Charaka, Chikitsa Sthan. 2017th ed., Ch. 13. Verse no 61. Varanasi: Choukhamba Surbharati Prakashan; 2017. p. 495.
19. Garde GK. Sartha Vagbhat, Chikitsa Sthan. Reprint 2012 ed., Ch. 15. Verse no 1. Varanasi: Choukhamba Surbharati Prakashan; 2017. p. 300.
20. Acharya YT. Charak Samhita of Charaka, Kalpa Sthan. 2017th ed., Ch. 12. Verse no 69. Varanasi: Choukhamba Surbharati Prakashan; 2017. p. 674.
21. Tripathi B. Bhavamishra, Bhavprakash Nighantu, Haritakyadi Varga. Reprint 2010. Verse no 151-152. Varanasi: Choukhamba Bharati Academy; 2010. p. 67.
22. Acharya YT. Charak Samhita of Charaka, Chikitsa Sthan. 2017th ed., Ch. 13. Verse no 60. Varanasi: Choukhamba Surbharati Prakashan; 2017. p. 495.
23. Shastri AD. Sushruta Samhita of Sushruta, Chikitsa Sthan. 2014 Reprint edition., Ch. 14. Verse no 18. Varanasi: Choukhamba Publications; 2014. p. 90.
24. Garde GK. Sartha Vagbhat, Chikitsa Sthan. Reprint 2012 edition., Ch. 15. Verse no 51. Varanasi: Choukhamba Surbharati Prakashan; 2017. p. 303.
25. Acharya YT. Charak Samhita of Charaka, Nidan Sthan. 2017th ed., Ch. 8. Verse no 37. Varanasi: Choukhamba Surbharati Prakashan; 2017. p. 229.
26. Tripathi R, editor. Charaka Samhita. Siddhi Sthana 1/17,879. Varanasi: Chaukhamba Prakashana; 2007.
27. Kajaria D, Tripathi JS, Tiwari SK. Utilization of panchakarma in health care: Preventive, nutritive and curative treatment of disease. J Pharm Sci Innov 2013;2:1-5.
28. Antarkar DS, Vaidya AB, Joshi JC. A double blind clinical trial of Arogya-vardhini--an ayurvedic drug--in acute viral Hepatitis. Indian J Med Res 1980;72:588-93.
29. Pandey GS, editor. Bhavprakash Nighantu of Shri Bhavamisra, Mutra Varga. Ch. 18. Ver. 1 6. Varanasi: Chaukhambha Bharati Academy; 2010. p. 761.
30. Jadhav DK. An ayurvedic approach in the management of Jalodara (Ascites): A case study. Int J Ayurveda Res 2016;1:90-1.
31. Pandey GS, editor. Bhavprakash Nighantu of Shri Bhavamisra, Guduchyadi Varga. Ch. 3, Ver. 210, Varanasi: Chaukhambha Bharati Academy; 2010. p. 393.
32. The Ayurvedic Formulary of India. Part 1. 2nd Revised English edition. Sec 20. Rasayoga 20:4 Arogyavardhini Gutika. New Delhi: Government of India, Ministry of Health and Family Welfare; 2003. p. 258.
33. The Ayurvedic Formulary of India. Part 1, 2nd Revised English Edition. Sec 20. Kvatha Curna 4:21 Punarnavadi Kvatha Curna. New Delhi: Government of India, Ministry of Health and Family Welfare; 2003. p. 58.
34. The Ayurvedic Formulary of India. Part 1, 2nd Revised English Edition. Sec 19. Mandura 19: 1. Punarnavadi Mandura. New Delhi: Government of India, Ministry of Health and Family Welfare; 2003. p. 251.

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Table 1: Laboratory investigations

Parameters	7 July 2022	1 August 2022	4 August 2022	Before treatment	After treatment	24 June 2023
Hb	8.1 mg/DL	6.3 mg/DL	6.3 mg/DL	9.6 mg/DL	10.9 mg/dl	11.4 mg/dl
WBC	22,280/cumm	21600/cumm	7800/cumm	11,600/cumm	11300/cumm	11.3/cumm
RBC	2.91 mill./cumm	2.27 mill./cumm	2.16 mill./cumm	3.15mill./cumm	3.86 mill./cumm	3.9 mill./cumm
Total bilirubin	2.4 mg/dl	1.4 mg/dl	-	4.6 mg/dl	1.0 mg/dl	1.2 mg/dl
Direct bilirubin	1.8 mg/dl	0.7 mg/dl	-	1.4 mg/dl	0.2 mg/dl	0.082 mg/dl
Indirect bilirubin	0.6 mg/dl	0.7 mg/dl	-	3.2 mg/dl	0.8 mg/dl	0.0842 mg/dl
SR creatinine	3.9 mg/dl	2.3 mg/dl	1.9 mg/dl	2.1 mg/dl	0.6 mg/dl	0.782 mg/dl
Blood urea	200 mg/dl	200 mg/dl	200 mg/dl	56 mg/dl	38 mg/dl	19.32 mg/dl
ESR		80				76

WBC: White blood cells, RBC: Red blood cells, ESR: Erythrocyte sedimentation rate

Table 2: USG

USG	Before treatment	After treatment
Coarse echotexture of the liver with surface nodularity – cirrhotic changes. Few small periportal and perisplenic collaterals and perisplenic collaterals. Prominent portal vein (12 mm) with a sent color uptake – thrombosis. Gross ascites Bilateral grade 1 renal parenchymal changes. Mild splenomegaly.	Mild *hepatomegaly* Moderate *ascites*. Grade-1 fatty liver. Tapping done (13/07/2022) about 2.5 lit clear fluid drained.	N/O – *Hepato* and *splenomegaly* – Minimal with normal hepatic vein-free fluid in perihepatic area. Grade – 1 fatty liver. Both kidneys show normal corticomedullary ratio and both kidneys show no calculus. No active lesion on chest, heart, and arota is normal, pleural space is also appears normal.
X-Ray chest	Left pleural effusion (1/8/2022)	

USG: Ultra Sonography

Table 3: Treatment schedule

Date	Medicine	Dose	*Anupana*	Times
30/8/2022	*Chandraprabha vati*	250 mg	Lukewarm water	2 times a day
	Guduchi ghan vati	500 mg	Lukewarm water	3 times a day
	Adulsa ghan vati	500 mg	Lukewarm water	3 times a day
	Punarnava mandur	500 mg	Lukewarm water	3 times a day
	Arogyavardhini vati	500 mg	Lukewarm water	2 times a day
	Punarnavashtak kwath	20 ml	Lukewarm water	2 times a day
	Castor oil	30 ml	Milk	Morning
8/11/2022	*Gokshur ghan*	500 mg	Lukewarm water	3 times a day
	Chandraprabha vati	250 mg	Lukewarm water	2 times a day
	Punarnava ghan	500 mg	Lukewarm water	3 times a day
	Madhu Malini Vasant	500 mg	Lukewarm water	3 times a day
	Punarnava mandur	500 mg	Lukewarm water	3 times a day
	Arogyavardhini vati	500 mg	Lukewarm water	2 times a day
	Punarnavashtak kwath	20 ml	Lukewarm water	2 times a day
	Asthiposhak vati	2 g	Lukewarm water	2 times a day
	Castor oil	30 ml	Milk	Morning
9/12/2022 Bp-110/70 mmHg Wt- 48.9 kg	*Vatvajradi vati*	500 mg	Lukewarm water	2 times a day
	Punarnava ghan	500 mg	Lukewarm water	3 times a day
	Gokshur ghan	500 mg	Lukewarm water	3 times a day
	Arogyavardhini vati	500 mg	Lukewarm water	2 times a day
	Punarnavashtak kwath	20 ml	Lukewarm water	2 times a day
	Punarnava mandur	2 g	Lukewarm water	3 times a day
	Castor oil	30 ml	Milk	Morning

(Contd...)

Table 3: *(Continued)*

Date	Medicine	Dose	*Anupana*	Times
10/3/2023 Bp - 110/70 mmhg Wt - 49 kg	*Punarnavadi mandur*	500 mg	Lukewarm water	3 times a day
	Laghu malini vasant	500 mg	Lukewarm water	3 times a day
	Punarnava ghan	500 mg	Lukewarm water	2 times a day
	Arogyavardhini vati	500 mg	Lukewarm water	2 times a day
	Punanarvashtak kwath	20 ml	Lukewarm water	2 times a day
	Asthiposhak vati	2 g	Lukewarm water	2 times a day
	Castor oil	30 ml	Milk	Morning

Table 4: Treatment after discharge from hospital

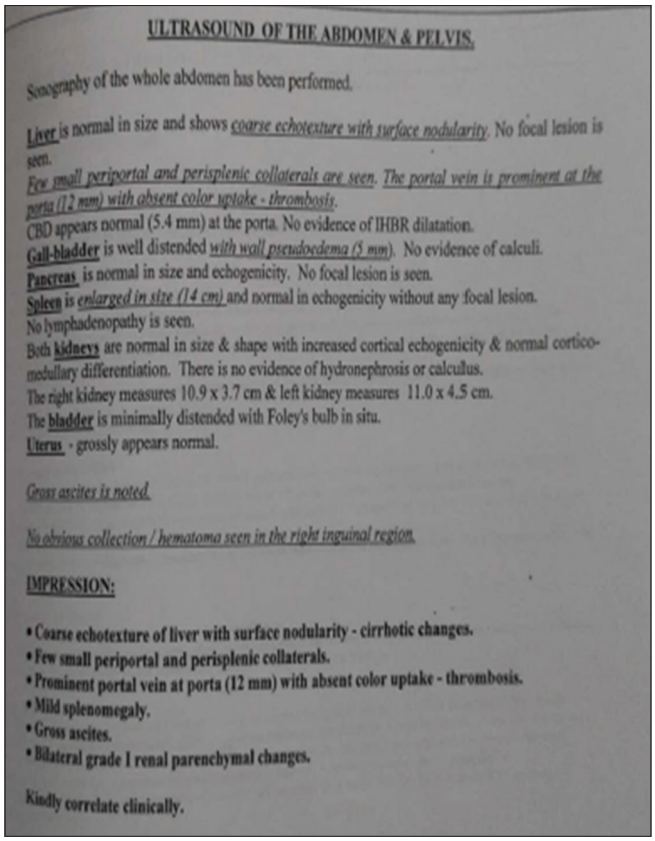
Date	Medicine	Dose	Anupana	Times
26/5/2023 BP-126/70mm hg Wt-52kg	Shankh vati	250 g	Lukewarm water	3 times a day
	Madhumalini vasant	500 g	Lukewarm water	3 times a day
	Punarnava ashtaka	20 ml	Lukewarm water	2 times a day
	Punarnava ghan	500 g	Lukewarm water	2 times a day
	Sukshma triphala	500 mg	Lukewarm water	2 times a day
	Gandhak rasayan	500 mg	Lukewarm water	2 times a day
	Arogyavardhini vati	2 g	Lukewarm water	2 times a day
	Punarnavadi mandur	30 ml	Milk	Morning
10/7/2023 Wt -64.5 kg Bp-125/80 mmhg	*Tribhuvan kirti ras*	250 mg	Lukewarm water	3 times a day
	Laxmivilas ras	500 mg	Lukewarm water	3 times a day
	Punarnavadi mandur	500 mg	Lukewarm water	3 times a day
	Kanchanar guggulu	500 mg	Lukewarm water	2 times a day
	Pathyadi kwath ghan	250 mg	Lukewarm water	2 times a day
	Hingwashtaka churna	500 mg	Lukewarm water	2 times a day
	Punanarvashtak kwath	20 ml	Lukewarm water	2 times a day

Table 5: Relief in symptoms

Date	Anorexia	Abdominal distension	Bipedal edema	Icterus	Pallar	General weakness
30/8/2022	+++	++	++	++	++	+++
8/11/2022	++	++	++	++	++	+++
9/12/2022	++	++	++	+	++	++
12/1/2023	+	++	+	-	+	+
10/3/2023	-	+	+	-	+	-
First follow-up 26th May 23	-	+	-	-	+	-
2nd follow-up 14th June 23	-	+	-	-	-	-

Table 6: Measurement of girth of abdomen

Date	4 cm below umbilicus	At umbilicus	4 cm above umbilicus
30/8/2022	83 cm	80 cm	75 cm
8/11/2022	82.5 cm	80 cm	74.5 cm
9/12/2022	82 cm	80 cm	74.5 cm
12/1/2023	81 cm	79.5 cm	74 cm
10/3/2023	80.5 cm	79 cm	73 cm
First follow up 26th May 23	77 cm	75.5 cm	69.5 cm
2nd follow up 14th June 23	74.5 cm	72 cm	65.5 cm



ULTRASOUND OF THE ABDOMEN & PELVIS.

Sonography of the whole abdomen has been performed.

Liver is normal in size and shows coarse echotexture with surface nodularity. No focal lesion is seen.
Few small periportal and perisplenic collaterals are seen. The portal vein is prominent at the porta (12 mm) with absent color uptake - thrombosis.
CBD appears normal (5.4 mm) at the porta. No evidence of IHBR dilatation.
Gall-bladder is well distended with wall pseudoedema (5 mm). No evidence of calculi.
Pancreas is normal in size and echogenicity. No focal lesion is seen.
Spleen is enlarged in size (14 cm) and normal in echogenicity without any focal lesion.
No lymphadenopathy is seen.
Both kidneys are normal in size & shape with increased cortical echogenicity & normal cortico-medullary differentiation. There is no evidence of hydronephrosis or calculus.
The right kidney measures 10.9 x 3.7 cm & left kidney measures 11.0 x 4.5 cm.
The bladder is minimally distended with Foley's bulb in situ.
Uterus - grossly appears normal.

Gross ascites is noted.

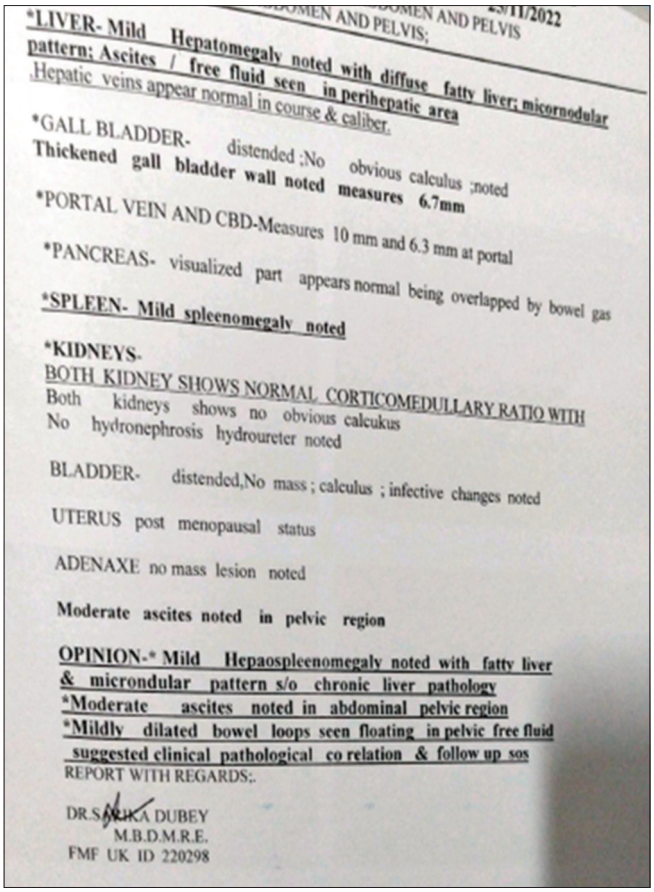
No obvious collection / hematoma seen in the right inguinal region.

IMPRESSION:

- Coarse echotexture of liver with surface nodularity - cirrhotic changes.
- Few small periportal and perisplenic collaterals.
- Prominent portal vein at porta (12 mm) with absent color uptake - thrombosis.
- Mild splenomegaly.
- Gross ascites.
- Bilateral grade I renal parenchymal changes.

Kindly correlate clinically.

Figure 1: Before treatment



...OMEN AND PELVIS 25/11/2022
...OMEN AND PELVIS;

*LIVER- Mild Hepatomegaly noted with diffuse fatty liver; micornodular pattern; Ascites / free fluid seen in perihepatic area
Hepatic veins appear normal in course & caliber.

*GALL BLADDER- distended ;No obvious calculus ;noted
Thickened gall bladder wall noted measures 6.7mm

*PORTAL VEIN AND CBD-Measures 10 mm and 6.3 mm at portal

*PANCREAS- visualized part appears normal being overlapped by bowel gas

*SPLEEN- Mild spleenomegaly noted

*KIDNEYS-
BOTH KIDNEY SHOWS NORMAL CORTICOMEDULLARY RATIO WITH
Both kidneys shows no obvious calcukus
No hydronephrosis hydroureter noted

BLADDER- distended,No mass ; calculus ; infective changes noted

UTERUS post menopausal status

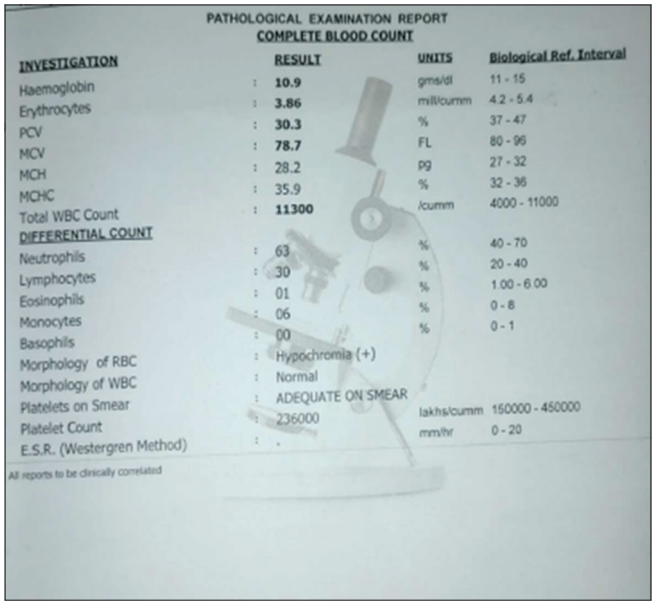
ADENAXE no mass lesion noted

Moderate ascites noted in pelvic region

OPINION-* Mild Hepaospleenomegaly noted with fatty liver & microndular pattern s/o chronic liver pathology
*Moderate ascites noted in abdominal pelvic region
*Mildly dilated bowel loops seen floating in pelvic free fluid
suggested clinical pathological co relation & follow up sos
REPORT WITH REGARDS;.

DR.S[illegible]KA DUBEY
M.B.D.M.R.E.
FMF UK ID 220298

Figure 2: After treatment

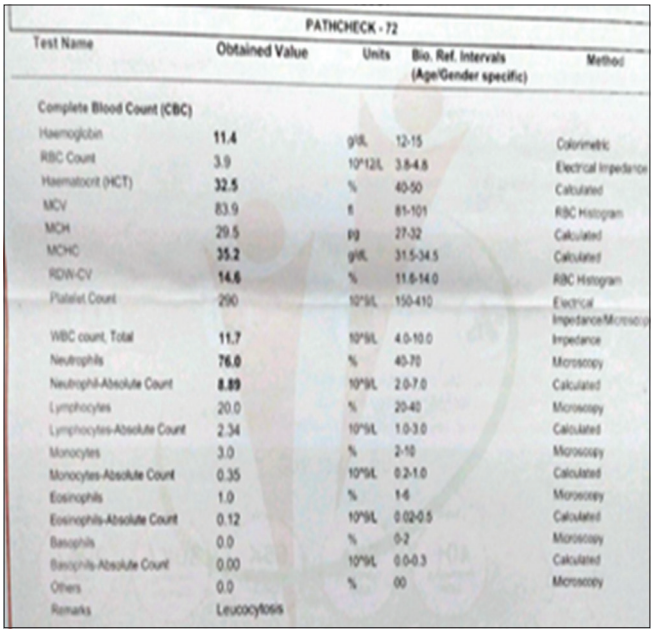


PATHOLOGICAL EXAMINATION REPORT
COMPLETE BLOOD COUNT

INVESTIGATION		RESULT	UNITS	Biological Ref. Interval
Haemoglobin	:	10.9	gms/dl	11 - 15
Erythrocytes	:	3.86	mill/cumm	4.2 - 5.4
PCV	:	30.3	%	37 - 47
MCV	:	78.7	FL	80 - 96
MCH	:	28.2	pg	27 - 32
MCHC	:	35.9	%	32 - 36
Total WBC Count	:	11300	/cumm	4000 - 11000
DIFFERENTIAL COUNT				
Neutrophils	:	63	%	40 - 70
Lymphocytes	:	30	%	20 - 40
Eosinophils	:	01	%	1.00 - 6.00
Monocytes	:	06	%	0 - 8
Basophils	:	00	%	0 - 1
Morphology of RBC	:	Hypochromia (+)		
Morphology of WBC	:	Normal		
Platelets on Smear	:	ADEQUATE ON SMEAR		
Platelet Count	:	236000	lakhs/cumm	150000 - 450000
E.S.R. (Westergren Method)	:	-	mm/hr	0 - 20

All reports to be clinically correlated

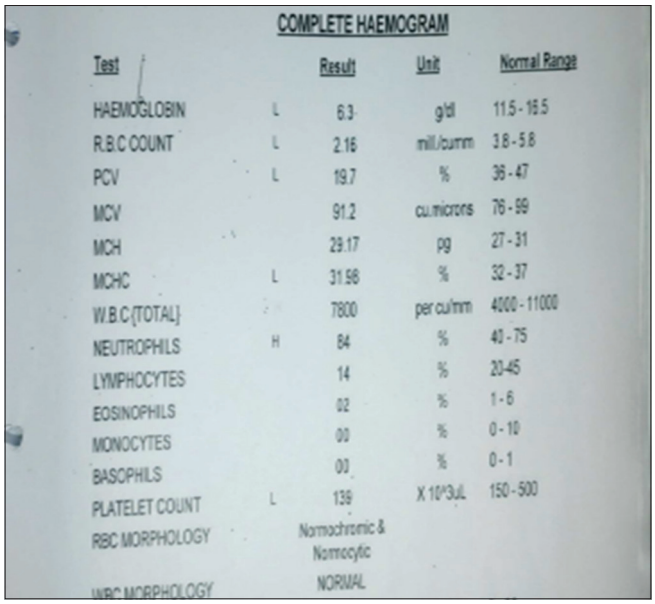
Figure 3: Before treatment



PATHCHECK - 72

Test Name	Obtained Value	Units	Bio. Ref. Intervals (Age/Gender specific)	Method
Complete Blood Count (CBC)				
Haemoglobin	11.4	g/dL	12-15	Colorimetric
RBC Count	3.9	10^12/L	3.8-4.8	Electrical Impedance
Haematocrit (HCT)	32.5	%	40-50	Calculated
MCV	83.9	fl	81-101	RBC Histogram
MCH	29.5	pg	27-32	Calculated
MCHC	35.2	g/dL	31.5-34.5	Calculated
RDW-CV	14.6	%	11.6-14.0	RBC Histogram
Platelet Count	290	10^9/L	150-410	Electrical Impedance/Microscopy
WBC count, Total	11.7	10^9/L	4.0-10.0	Impedance
Neutrophils	76.0	%	40-70	Microscopy
Neutrophil-Absolute Count	8.89	10^9/L	2.0-7.0	Calculated
Lymphocytes	20.0	%	20-40	Microscopy
Lymphocytes-Absolute Count	2.34	10^9/L	1.0-3.0	Calculated
Monocytes	3.0	%	2-10	Microscopy
Monocytes-Absolute Count	0.35	10^9/L	0.2-1.0	Calculated
Eosinophils	1.0	%	1-6	Microscopy
Eosinophils-Absolute Count	0.12	10^9/L	0.02-0.5	Calculated
Basophils	0.0	%	0-2	Microscopy
Basophils-Absolute Count	0.00	10^9/L	0.0-0.3	Calculated
Others	0.0	%	00	Microscopy
Remarks	Leucocytosis			

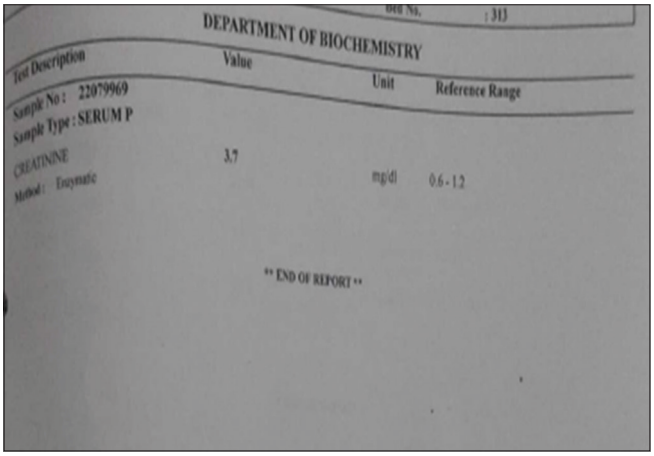
Figure 5: After treatment



COMPLETE HAEMOGRAM

Test		Result	Unit	Normal Range
HAEMOGLOBIN	L	6.3	g/dl	11.5 - 16.5
R.B.C COUNT	L	2.16	mill/cumm	3.8 - 5.8
PCV	L	19.7	%	36 - 47
MCV		91.2	cu.microns	76 - 99
MCH		29.17	pg	27 - 31
MCHC	L	31.98	%	32 - 37
W.B.C.(TOTAL)		7800	per cu/mm	4000 - 11000
NEUTROPHILS	H	84	%	40 - 75
LYMPHOCYTES		14	%	20-45
EOSINOPHILS		02	%	1 - 6
MONOCYTES		00	%	0 - 10
BASOPHILS		00	%	0 - 1
PLATELET COUNT	L	139	X 10^3/L	150 - 500
RBC MORPHOLOGY		Normochromic & Normocytic		
WBC MORPHOLOGY		NORMAL		

Figure 4: Before treatment

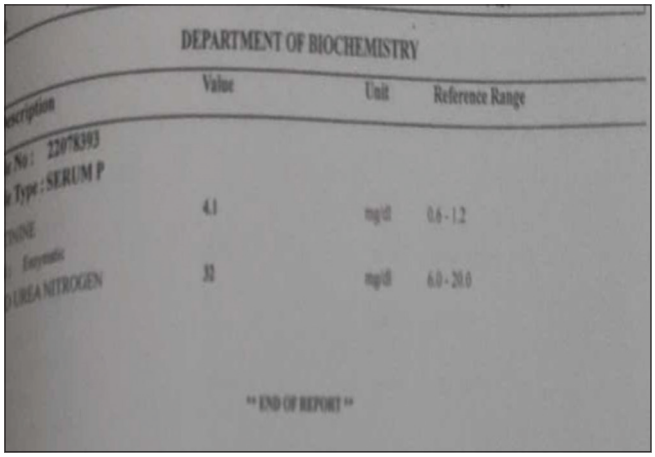


DEPARTMENT OF BIOCHEMISTRY

Test Description	Value	Unit	Reference Range
Sample No : 2207969			
Sample Type : SERUM P			
CREATININE	3.7	mg/dl	0.6 - 1.2
Method : Enzymatic			

** END OF REPORT **

Figure 6: Before treatment

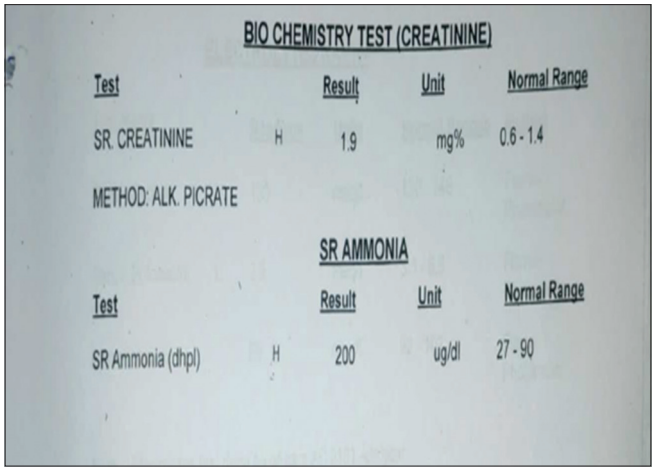


DEPARTMENT OF BIOCHEMISTRY

Value	Unit	Reference Range
4.1	mg/dl	0.6 - 1.2
33	mg/dl	6.0 - 20.0

** END OF REPORT **

Figure 7: After treatment



BIO CHEMISTRY TEST (CREATININE)

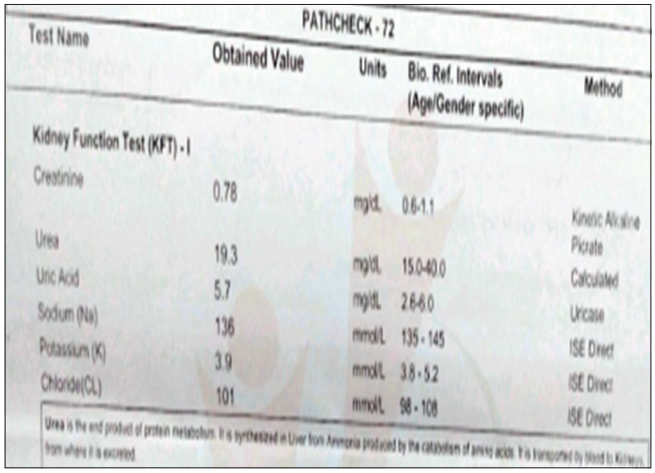
Test		Result	Unit	Normal Range
SR. CREATININE	H	1.9	mg%	0.6 - 1.4

METHOD: ALK. PICRATE

SR AMMONIA

Test		Result	Unit	Normal Range
SR Ammonia (dhpl)	H	200	ug/dl	27 - 90

Figure 8: After treatment

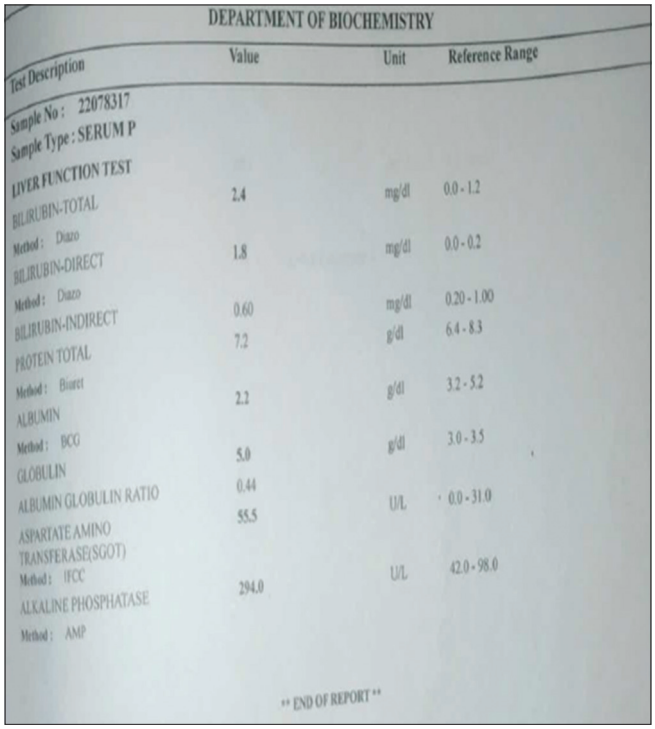


PATHCHECK - 72

Test Name	Obtained Value	Units	Bio. Ref. Intervals (Age/Gender specific)	Method
Kidney Function Test (KFT) - I				
Creatinine	0.78	mg/dL	0.6-1.1	Kinetic Alkaline Picrate
Urea	19.3	mg/dL	15.0-40.0	Calculated
Uric Acid	5.7	mg/dL	2.6-6.0	Uricase
Sodium (Na)	136	mmol/L	135 - 145	ISE Direct
Potassium (K)	3.9	mmol/L	3.8 - 5.2	ISE Direct
Chloride(CL)	101	mmol/L	98 - 108	ISE Direct

Urea is the end product of protein metabolism. It is synthesized in Liver from Ammonia produced by the catabolism of amino acids. It is transported by blood to Kidneys from where it is excreted

Figure 9: After treatment

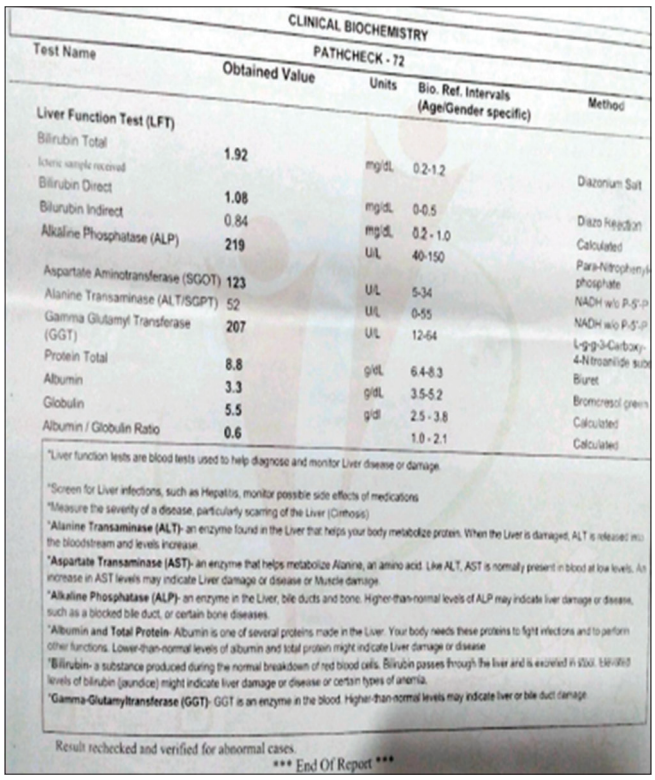


DEPARTMENT OF BIOCHEMISTRY

Test Description	Value	Unit	Reference Range
Sample No : 22078317			
Sample Type : SERUM P			
LIVER FUNCTION TEST			
BILIRUBIN-TOTAL	2.4	mg/dl	0.0 - 1.2
Method : Diazo			
BILIRUBIN-DIRECT	1.8	mg/dl	0.0 - 0.2
Method : Diazo			
BILIRUBIN-INDIRECT	0.60	mg/dl	0.20 - 1.00
PROTEIN TOTAL	7.2	g/dl	6.4 - 8.3
Method : Biuret			
ALBUMIN	2.2	g/dl	3.2 - 5.2
Method : BCG			
GLOBULIN	5.0	g/dl	3.0 - 3.5
ALBUMIN GLOBULIN RATIO	0.44		
ASPARTATE AMINO TRANSFERASE(SGOT)	55.5	U/L	0.0 - 31.0
Method : IFCC			
ALKALINE PHOSPHATASE	294.0	U/L	42.0 - 98.0
Method : AMP			

** END OF REPORT **

Figure 10: Before treatment



CLINICAL BIOCHEMISTRY

PATHCHECK - 72

Test Name	Obtained Value	Units	Bio. Ref. Intervals (Age/Gender specific)	Method
Liver Function Test (LFT)				
Bilirubin Total	1.92	mg/dL	0.2-1.2	Diazonium Salt
Icteric sample received				
Bilirubin Direct	1.08	mg/dL	0-0.5	Diazo Reaction
Bilirubin Indirect	0.84	mg/dL	0.2 - 1.0	Calculated
Alkaline Phosphatase (ALP)	219	U/L	40-150	Para-Nitrophenyl phosphate
Aspartate Aminotransferase (SGOT)	123	U/L	5-34	NADH w/o P-5'-P
Alanine Transaminase (ALT/SGPT)	52	U/L	0-55	NADH w/o P-5'-P
Gamma Glutamyl Transferase (GGT)	207	U/L	12-64	L-g-g-3-Carboxy-4-Nitroanilide
Protein Total	8.8	g/dL	6.4-8.3	Biuret
Albumin	3.3	g/dL	3.5-5.2	Bromcresol green
Globulin	5.5	g/dl	2.5 - 3.8	Calculated
Albumin / Globulin Ratio	0.6		1.0 - 2.1	Calculated

*Liver function tests are blood tests used to help diagnose and monitor Liver disease or damage.

*Screen for Liver infections, such as Hepatitis, monitor possible side effects of medications

*Measure the severity of a disease, particularly scarring of the Liver (Cirrhosis)

*Alanine Transaminase (ALT)- an enzyme found in the Liver that helps your body metabolize protein. When the Liver is damaged, ALT is released into the bloodstream and levels increase.

*Aspartate Transaminase (AST)- an enzyme that helps metabolize Alanine, an amino acid. Like ALT, AST is normally present in blood at low levels. An increase in AST levels may indicate Liver damage or disease or Muscle damage.

*Alkaline Phosphatase (ALP)- an enzyme in the Liver, bile ducts and bone. Higher-than-normal levels of ALP may indicate liver damage or disease, such as a blocked bile duct, or certain bone diseases.

*Albumin and Total Protein- Albumin is one of several proteins made in the Liver. Your body needs these proteins to fight infections and to perform other functions. Lower-than-normal levels of albumin and total protein might indicate Liver damage or disease

*Bilirubin- a substance produced during the normal breakdown of red blood cells. Bilirubin passes through the liver and is excreted in stool. Elevated levels of bilirubin (jaundice) might indicate liver damage or disease or certain types of anemia.

*Gamma-Glutamyltransferase (GGT)- GGT is an enzyme in the blood. Higher-than-normal levels may indicate liver or bile duct damage

Result rechecked and verified for abnormal cases.

*** End Of Report ***

Figure 11: After treatment

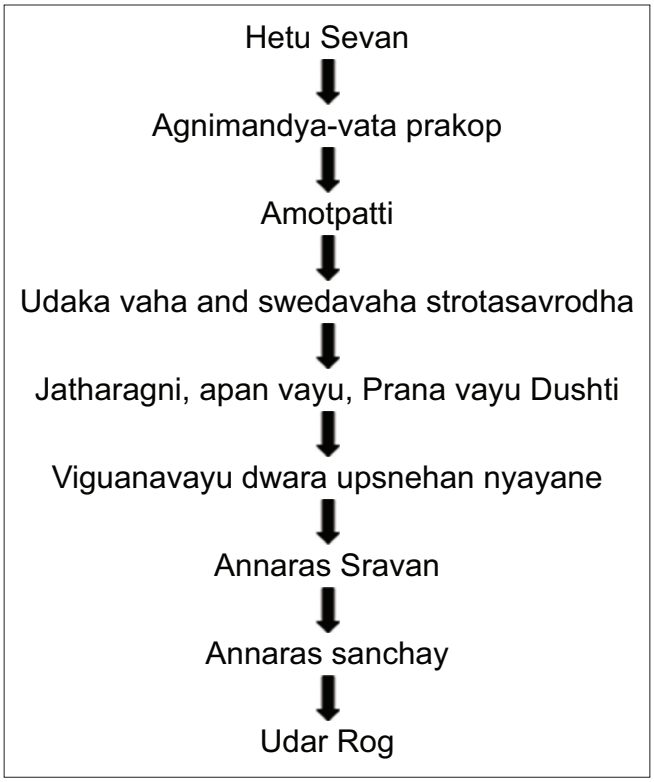


Figure 12: Samprapti of ascites