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Ayurvedic management of *Ruddhapatha Kamala* (Obstructive Jaundice) - A Case Study

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ABSTRACT

Biliary obstruction[1] refers to blockage of any duct that carries bile from the liver to the gallbladder or from the gallbladder to the small intestine. The clinical setting of the failure of biliary flow may be due to obstruction by mechanical means or by metabolic factors in the hepatic cells. The discussion of metabolic causes of biliary obstruction is very complex; the pathogenesis is not always clearly defined. While on the other hand, the condition *Ruddha Patha Kamala* in *Ayurveda* is the nearest possible clinical correlation to biliary obstruction wherein the flow of *Pitta* is obstructed by *Kapha*.[2] A structural / physical complete blockage of biliary flow needs a surgical intervention while if the cause is due to metabolic compromise or a partial block, the condition can be reversed effectively through *Ayurvedic* medications. Here is a case of obstructive jaundice, with a h/o cholecystectomy 20y back and H/O ERCP 5y back, treated successfully with *Ayurvedic* medications and some diet modifications for about 42 days. A combination of *Avipatthikara Churna, Shweta Parpati, Yava Kshara* and *Katuki Churna* was the intervention given. *Mulaka Yusha* twice a day along with bland diet was advised as *Pathya*. By the end of 42 days, the LFT report turned completely normal and the patient felt very healthy subjectively. There exists a need in the current times to critically understand the treatment techniques and principles of traditional medicine systems and to make use of them in treating certain conditions where the other conventional medicine systems fail.

Key words: *Shakhashrita Kamala, Ruddhapatha Kamala, Obstructive jaundice, Biliary obstruction, Cholestasis, Ayurveda treatment*

INTRODUCTION

Jaundice is usually detectable when the plasma bilirubin exceeds 2.5mg/dl. The causes of jaundice overlap with variations in LFTs. In a patient with jaundice, it is important to consider whether the pathology is pre hepatic, hepatic or post hepatic. The treatment principles differ based on the type of pathology presented. The pre hepatic jaundice is caused either by hemolysis or by congenital hyperbilirubinemia and is characterized by the isolated elevation of bilirubin levels. The hepatic jaundice results from an inability of the liver cells to transport bilirubin into the bile, occurring as a consequence of parenchymal disease. In hepatic jaundice, the concentration of both conjugated and unconjugated bilirubin levels in blood increase. Obstructive jaundice/ cholestatic/ biliary obstruction may be caused by failure of hepatocytes to initiate bile flow or obstruction of the bile ducts or obstruction of bile flow in extra hepatic bile ducts.

In the absence of treatment, cholestatic jaundice tends to become progressively more severe because conjugated bilirubin, due to obstruction, passes back into the blood. The clinical symptoms of obstructive pathology are characteristically associated with pale

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stools and dark urine. Pruritis may be a dominant associated complaint.[3]

In *Ayurveda*, the concept of *Kamala* has been told under *Pandu Roga*. *Pandu* in *Ayurveda* is an umbrella term that includes all the blood related diseases and also the hepato - biliary tract related diseases. *Pitta Pradhana Tridoshas* when elevated, end up in *Dhatu Shaithilya*, *Gourava*, *Alpa Raktha, Alpa Meda, Nissarata* and *Shithilendriyata* completing the *Samprapthi* of *Pandu*.[4] The patient with *Pandu Roga* when indulges in furthermore *Pitta Kara Ahara Viharas*, the result is *Koshtashritha Kamala*. The same elevated *Pitta* if obstructed by *Kapha*, the movement of *Pitta* from the *Shakhas* towards *Koshta* is blocked leading to *Ruddhapatha Kamala* / *Shakhashrita Kamala*. In a broader sense, the pre hepatic / anemic type of jaundice can be seen as *Pandu*, the hepatic jaundice as *Koshtashrita Kamala* and the post hepatic / obstructive type as *Ruddhapatha Kamala*.

Investigations play an important role in understanding and managing liver diseases. Understanding the variations in LFT is the key to arrive at the correct diagnosis.

Bilirubin: The degree of elevation of bilirubin can reflect the degree of liver damage. A raised bilirubin often occurs earlier in the natural history of biliary disease than in the disease of liver parenchyma. Swelling of the liver within its capsule can impair bile flow and cause an elevation of bilirubin level that is disproportionate to the degree of liver injury.[5]

ALT and AST: Although both the transaminases are widely distributed, expression of ALT outside the liver is relatively low and this enzyme is therefore considered more specific for hepatocellular damage.

ALP and GGT: The pattern of modest increase in aminotransferase (ALT & AST) activity and large increases in ALP and GGT activity favors biliary obstruction and is commonly described as 'cholestatic' or 'obstructive'.[6]

Materials and Methods

A female patient aged 50y came with complaints of fatigue, myalgia, fever with chills on and off, loss of appetite, nausea, pain abdomen, dark yellow colored urine, mild bloating, oily stools, constipation, occasional body itching and weight gain since 1 month. Not a known case of DM2 and HTN, h/o cholecystectomy 20y back, h/o ERCP 5y back. On investigation, her LFT pointed towards obstructive pathology (AST, ALT and ALP raised. See Table 1, Image 1). And USG abdomen came with an impression of: obstructive biliary pathology secondary to CBD calculi, suggested ERCP, mild splenomegaly, bilateral renal cyst Rt > Lt with well-defined margins and with no echogenic effect (Image 2 and 3).

Table 1: LFT reports before and after treatment.

Parameter	Before treatment 23/11/2023	After treatment 04/01/2023	Reference range	Unit
Bilirubin total	**3.0**	0.74	0 to 1.1	mg/dL
Bilirubin direct	2.2	0.23	0 to 0.2	mg/dL
Bilirubin indirect	0.8	0.51	< 0.90	mg/dL
AST or SGOT	**300**	35	0 to 31	U/L
ALT or SGPT	**316**	24.9	0 to 33	U/L
AST/ALT ratio	0.95	1.41	< 1	
ALP	**437**	172	0 to 105	U/L
Total protein	7.6	7.41	6.4 to 8.3	g/dL
Albumin	4.4	4.34	4 to 4.9	g/dL
Globulin	3.2	0.74	1.9 to 3.7	g/dL

Intervention given:

Dry powders of *Avipatthikara Churna* 200g, *Shweta Parpati* 10g, *Yava Kshara* 50g, *Katuki Churna* 100g were made into a homogenous mixture and filled into size-2 capsules (approx. – 500mg). 2 capsules TID before food was the dosage given and *Mulaka Yusha* 50ml thrice a day before food for 42 days.

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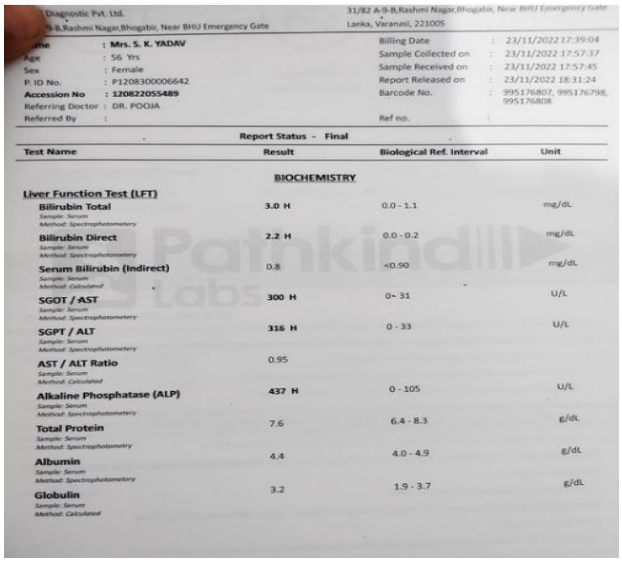
Image 1



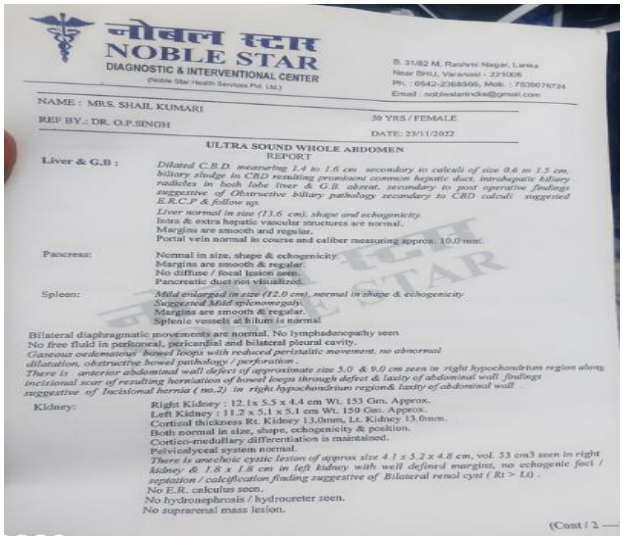
Diagnostic Pvt. Ltd.
31/82 A-9-B,Rashmi Nagar,Bhogabir, Near BHU Emergency Gate
Lanka, Varanasi, 221005

Name	: Mrs. S. K. YADAV	Billing Date	: 23/11/2022 17:39:04
Age	: 56 Yrs	Sample Collected on	: 23/11/2022 17:57:37
Sex	: Female	Sample Received on	: 23/11/2022 17:57:45
P. ID No.	: P12083000006642	Report Released on	: 23/11/2022 18:31:24
Accession No	: 120822055489	Barcode No.	: 995176807, 995176798, 995176808
Referring Doctor	: DR. POOJA		
Referred By	:	Ref no.	:

Report Status - Final

Test Name	Result	Biological Ref. Interval	Unit
BIOCHEMISTRY			
Liver Function Test (LFT)			
Bilirubin Total	3.0 H	0.0 - 1.1	mg/dL
Bilirubin Direct	2.2 H	0.0 - 0.2	mg/dL
Serum Bilirubin (Indirect)	0.8	<0.90	mg/dL
SGOT / AST	300 H	0- 31	U/L
SGPT / ALT	316 H	0 - 33	U/L
AST / ALT Ratio	0.95		
Alkaline Phosphatase (ALP)	437 H	0 - 105	U/L
Total Protein	7.6	6.4 - 8.3	g/dL
Albumin	4.4	4.0 - 4.9	g/dL
Globulin	3.2	1.9 - 3.7	g/dL

Image 2



नोबल स्टार
NOBLE STAR
DIAGNOSTIC & INTERVENTIONAL CENTER

NAME : MRS. SHAIL KUMARI
REF BY : DR. O.P.SINGH
50 YRS / FEMALE
DATE: 23/11/2022

ULTRA SOUND WHOLE ABDOMEN REPORT

Liver & G.B : Dilated C.B.D. measuring 1.4 to 1.6 cm secondary to calculi of size 0.6 to 1.3 cm. Biliary sludge in CBD resulting prominent common hepatic duct, intrahepatic biliary radicles in both lobe liver & G.B. absent, secondary to post operative findings suggestive of Obstructive biliary pathology secondary to CBD calculi suggested E.R.C.P & follow up.
Liver normal in size (13.6 cm), shape and echogenicity.
Intra & extra hepatic vascular structures are normal.
Margins are smooth and regular.
Portal vein normal in course and caliber measuring approx. 10.0 mm.

Pancreas: Normal in size, shape & echogenicity.
Margins are smooth & regular.
No diffuse / focal lesion seen.
Pancreatic duct not visualized.

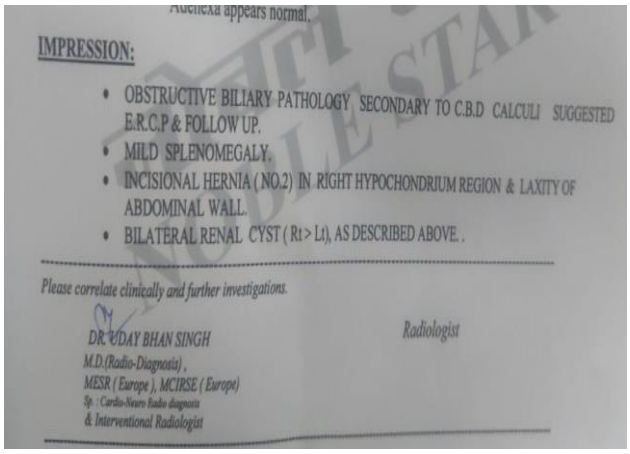
Spleen: Mild enlarged in size (12.0 cm), normal in shape & echogenicity.
Suggested Mild splenomegaly.
Margins are smooth & regular.
Splenic vessels at hilum is normal.

Bilateral diaphragmatic movements are normal. No lymphadenopathy seen.
No free fluid in peritoneal, pericardial and bilateral pleural cavity.
Gaseous oedematous bowel loops with reduced peristaltic movement, no abnormal dilatation, obstructive bowel pathology / perforation.
There is anterior abdominal wall defect of approximate size 5.0 & 9.0 cm seen in right hypochondrium region along incisional scar of resulting herniation of bowel loops through defect & laxity of abdominal wall findings suggestive of Incisional hernia (no.2) in right hypochondrium region& laxity of abdominal wall.

Kidney: Right Kidney : 12.1x 5.5 x 4.4 cm Wt. 153 Gm. Approx.
Left Kidney : 11.2 x 5.1 x 5.1 cm Wt. 150 Gm. Approx.
Cortical thickness Rt. Kidney 13.0mm, Lt. Kidney 13.0mm.
Both normal in size, shape, echogenicity & position.
Cortico-medullary differentiation is maintained.
Pelvicalyceal system normal.
There is anechoic cystic lesion of approx size 4.1 x 5.2 x 4.8 cm, vol. 53 cm3 seen in right Kidney & 1.8 x 1.8 cm in left kidney with well defined margins, no echogenic foci / septation / calcification finding suggestive of Bilateral renal cyst (Rt > Lt) .
No E.R. calculus seen.
No hydronephrosis / hydroureter seen.
No suprarenal mass lesion.

(Cont / 2 —

Image 3



Aorticxa appears normal.

IMPRESSION:

- OBSTRUCTIVE BILIARY PATHOLOGY SECONDARY TO C.B.D CALCULI SUGGESTED E.R.C.P & FOLLOW UP.
- MILD SPLENOMEGALY.
- INCISIONAL HERNIA (NO.2) IN RIGHT HYPOCHONDRIUM REGION & LAXITY OF ABDOMINAL WALL.
- BILATERAL RENAL CYST (Rt > Lt), AS DESCRIBED ABOVE .

Please correlate clinically and further investigations.

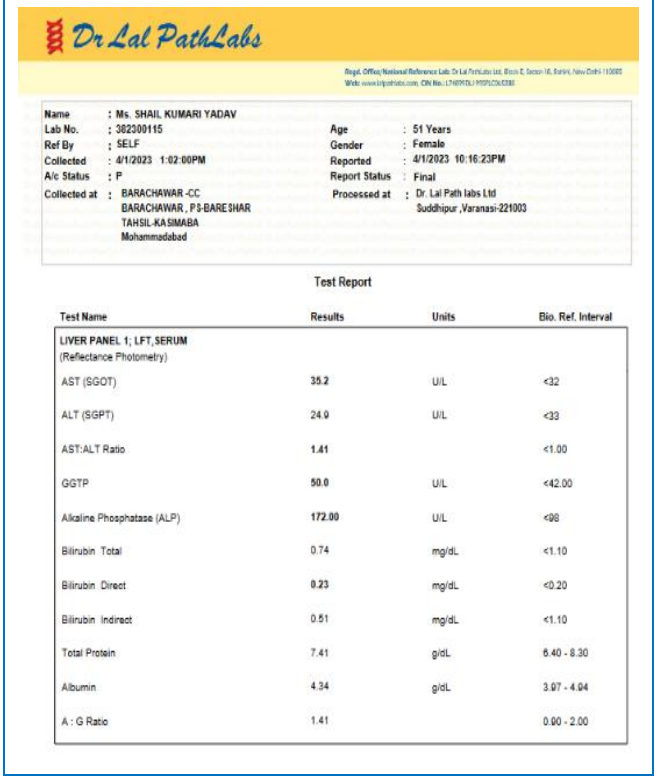
DR. UDAY BHAN SINGH
M.D.(Radio-Diagnosis),
MESR (Europe), MCIRSE (Europe)
Sp. : Cardio-Neuro Radio diagnosis
& Interventional Radiologist

Radiologist

RESULTS

The complaints - fatigue, myalgia, fever with chills on and off, loss of appetite, nausea, pain abdomen, dark yellow colored urine, mild bloating, oily stools and constipation, occasional body itching got completely reversed. And there was a remarkable improvement in LFT reports too (table 1 and image 4). However, USG abdomen was not repeated in spite of doctors' advice.

Image 4



Dr Lal PathLabs

Name : Ms. SHAIL KUMARI YADAV
Lab No. : 382300115
Ref By : SELF
Collected : 4/1/2023 1:02:00PM
A/c Status : P
Collected at : BARACHAWAR -CC BARACHAWAR , P.S-BARESHAR TAHSIL-KASIMABA Mohammadabad
Age : 51 Years
Gender : Female
Reported : 4/1/2023 10:16:23PM
Report Status : Final
Processed at : Dr. Lal Path labs Ltd Suddhipur ,Varanasi-221003

Test Report

Test Name	Results	Units	Bio. Ref. Interval
LIVER PANEL 1; LFT, SERUM (Reflectance Photometry)			
AST (SGOT)	35.2	U/L	<32
ALT (SGPT)	24.9	U/L	<33
AST:ALT Ratio	1.41		<1.00
GGTP	50.0	U/L	<42.00
Alkaline Phosphatase (ALP)	172.00	U/L	<98
Bilirubin Total	0.74	mg/dL	<1.10
Bilirubin Direct	0.23	mg/dL	<0.20
Bilirubin Indirect	0.51	mg/dL	<1.10
Total Protein	7.41	g/dL	6.40 - 8.30
Albumin	4.34	g/dL	3.97 - 4.94
A : G Ratio	1.41		0.90 - 2.00

DISCUSSION

The subject had a h/o cholecystectomy 20y back and h/o ERCP 5y back. She was not a known case of DM2 and HTN. The complaints like nausea, oily stools, bloating, constipation, mild generalized body itching etc., were suggestive of an obstructive hepatic pathology. And on investigations, the obstructive biliary pathology secondary to CBD calculi was confirmed. The treatment was started based on the lines of *Shakhashrita Kamala* i.e., '*Shleshmana Ruddhamargam Tam Pitttham Kapha Haraih Jayet*'.[7] Even though the *Kamala* is *Pitta Pradhana Vyadhi*, the *Shakhashritha* variety of *Kamala* has to be treated under the lines of anti *Kapha* drugs. So, the

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combination chosen was *Avipatthikara Churna, Shweta Parpati, Yava Kshara* and *Katuki Churna.* And *Mulaka Yusha* was advised as *Pathya*.

The *Avipatthikara Churna*[8], a *Ruksha Virechana Yoga* with *Trivruth* as main ingredient is from *Bhaishajya Ratnavali*, *Amlapittha Adhikara. Gunakarma - Katu Kashaya Tikta Rasa, Ushna Veerya, Pitthakapha Shamana, Anulomana, Deepana* and *Koshta Shodhana*. The *Shweta Parpati*[9], an *Ashmari Hara Yoga* with *Surya Kshara* as major ingredient is from *Siddha Yoga Sangraha*, *Ashmari-Mutrakricchra Adhyaya*. *Gunakarma - Katu Rasa, Ushna Veerya, Chedaka, Ashmari Hara, Kapha Hara.* The *Yava Kshara*[10], *a Shula Hara* drug with general *Kshara* properties is from *Rasatarangini*, *Kshara Trika Vijnaneeya Adhyaya*. *Gunakarma - Katu Rasa, Ushna Veerya, Kapha Vata Hara, Ama Shula Ashmari Dosha Hara*. The *Katuki Churna*[11] is a potent *Tiktha Rechaka, Kapha Pitta Hara* and *Yakruth Rasayana*. And the *Mulaka*[12,13] *Yusha* is *Katu Rasa, Ushna Veerya* and is *Kshara* in nature.

The logic behind the combination given was- *Avipatthikara Churna* as a *Sukha Ruksha Rechana*. *Shweta Parpati* and *Yava Kshara as Ashmari Hara*, *Shula Hara* and *Avruta Kapha Hara*. And *Katuki Churna* as a potent *Yakrith Rasayana*. Since the condition is *Ruddhapatha Kamala*, the usage of any *Sneha* and *Asavarishta* is not the appropriate choice. The drug or the combination of drugs chosen should be a *Ruksha Rechaka, Kapha Hara, Pitthavirodhi, Ashmari Hara, Shula Hara* and *Yakrit Rasayana*. And hence the combination was selected and administered.

For a *Pandu Rogi*, *Teekshna Shodhana* has been told as treatment. Whereas for a *Kamala Rogi*, only *Mrudu Ruksha Virechana* is advised.[14] The rationality behind avoiding *Teekshna Virechana* in *Kamala* may be due to the chronicity of the disease (if presented as the continuation of *Pandu - Paratantra*) as more chronicity leads to *Vata Vriddhi* leading to *Dourbalya* making the patient unfit for *Teekshna Shodhana*. And for a *Kamala* without the previous manifestation of *Pandu* (*Swatantra Kamala*), the logic of giving *Mrudu Virechana* may be - *Pitta Vriddhi* by itself causes *Balakshaya* unlike *Kaphavriddhi* (as increased *Kapha* will not hamper *Bala* of the patient usually). In that way, in a case of *Swatantra Kamala* also *Teekshna Virechana* has to be avoided. However, in *Balavan* patient, *Teekshna Shodhanas* can be carried out to achieve quicker results. The presented subject was a 50y old female with *Dourbalya* as one of the associated complaints. Hence, *Mrudu Ruskha Virechana* and *Kapha Hara* treatment was chosen and carried out. After a period of 42 days, good results were seen and the case was documented.

Conclusion

Today, in spite of researches, development of technology and newer drugs in modern medicine, treating the cases with hepatic compromise is still not up to the mark. A keen observation on the presenting complaints and correlating with investigations, a *Vaidya* can arrive at the nearest *Ayurvedic* diagnosis in a *Tridoshic* perspective and can plan the treatment accordingly. To conclude, whatever may be the condition, either *Ekadoshaja* or *Sannipataja*, when it comes to *Yakrith Roga*, *Ayurveda* has the potential to reverse or at least to manage even the last stage complications of hepatic compromise.

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