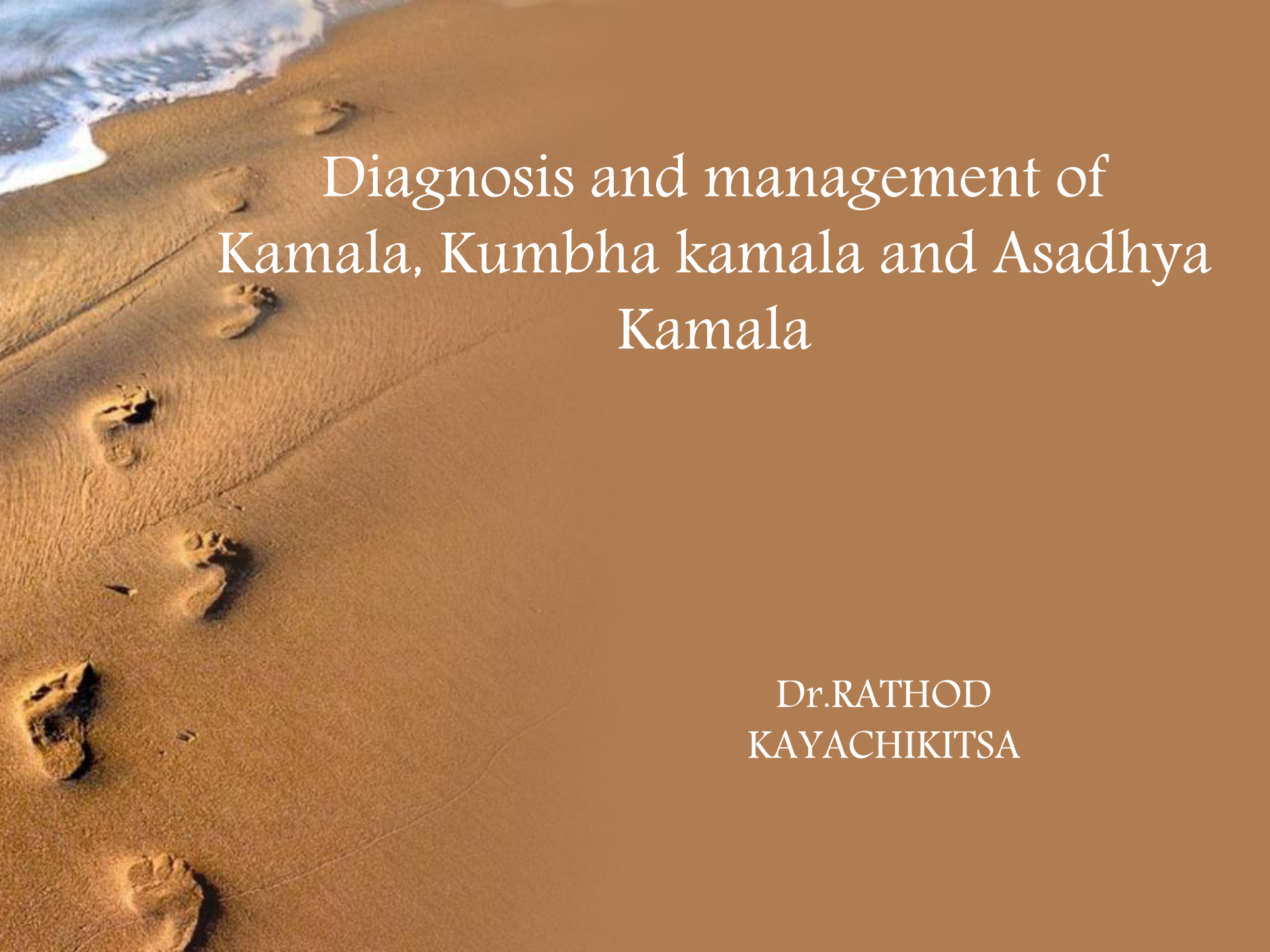


Welcome



An aerial photograph of a sandy beach. The sand is a warm, golden-brown color. In the upper left corner, the ocean waves are breaking, creating white foam. A series of footprints are visible, leading from the water towards the foreground. The overall scene is peaceful and natural.

Diagnosis and management of Kamala, Kumbha kamala and Asadhya Kamala

Dr.RATHOD
KAYACHIKITSA



Disorders of Liver

- *Acute Liver Disorder:*
 - *Viral, Drug, Gall stones, alcohol toxicity.*
- *Chronic Liver Disease:*
 - *Chronic hepatitis, Cirrhosis, viral, alcohol, congenital*
 - *Autoimmune hepatitis.*
- *Congenital Disorders:*
 - *Haemochromatosis, Wilsons.*
- *Tumors:*
 - *Benign: Adenoma, angioma, Nodular hyperplasia*
 - *Malignant: Hepatocellular carcinoma, Cholangiocarcinoma, Hepatoblastoma, Angiosarcoma.*
- *Cysts: Simple, Hydatid*

Clinical features & Pathogenesis

<i>Jaundice</i>	<i>Impaired conjugation or obstruction.</i>
<i>Dark urine</i>	<i>Conjugated hyperbilirubin</i>
<i>Pale stools</i>	<i>Biliary obstruction</i>
<i>Oedema</i>	<i>Low albumin – low oncotic pressure.</i>
<i>Steatorrhoea</i>	<i>Bile obstruction.</i>
<i>Pruritis</i>	<i>Bile obstruction → Bile salt in blood.</i>
<i>Ascitis</i>	<i>Portal hypertension , low albumin, hyper aldosterone</i>
<i>Bleeding</i>	<i>Coagulation factor synthesis</i>
<i>Haematemesis</i>	<i>Oesophageal varices. (hemorrhoids)</i>
<i>Encephalopathy</i>	<i>Toxic nitrogen products – gut bacteria.</i>
<i>Foetar hepaticus</i>	<i>Musty odor (mercaptans by gut bacteria)</i>

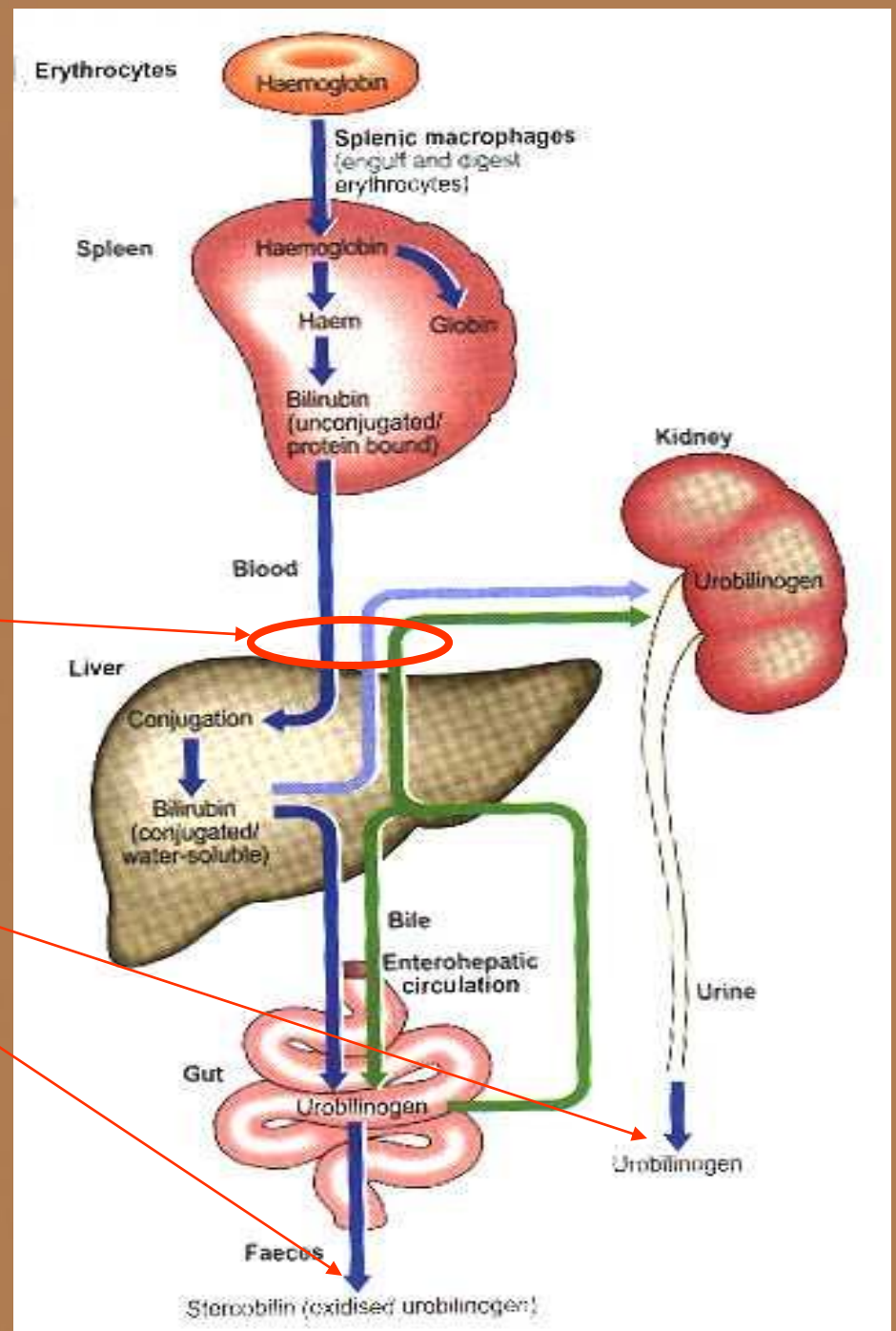
Jaundice



- *Yellow discoloration of skin & sclera due to excess serum bilirubin. $>40\mu\text{mol/l}$, (3mg/dl)*
- *Conjugated & Unconjugated types*
- *Obstructive & Non Obstructive (clinical)*
- *Pre-Hepatic, Hepatic & Post Hepatic types*
- *Jaundice - Not necessarily liver disease **

Bilirubin Metabolism

- Blood
- Conjugated & Unconjugated
- Urine – Urobilinogen
- Stool – Stercobilin



Bilirubin in the Liver Cell

1

- Hepatocyte (HC) uptake of UCB
- Alb+UCB dissociates and UCB enters HC
- By passive diffusion into HC – Ligandin bound
- Insoluble UCB is to be made soluble in HC

2

- Conjugation in ER of Hepatocyte (HC)
- Formation of mono and di glucuronides BMG, BDG
- UDP Glucuronosyl transferase is energy depend.
- Insoluble UCB made water soluble for excretion

3

- Excretion in into biliary canaliculi
- Rate limiting step in metabolism
- CB 50% is not protein bound – no loss of albumin
- Remaining 50% δ bilirubin – Irreversibly bound

Bilirubin in the Intestine

1. CB in bile is excreted into Duodenum

CB 10% diffuses in to blood

CB excreted is not reabsorbed

2. Conversion of CB into uro & stercobilinogen

starcobilinogen excreted in stool


Part of the UBG enters EHC

3. From gut, UBG but not CB enters EHC

Kidney excretes absorbed UBG

In biliary obst. UBG absent in urine

Bilirubin handling in Kidney



Conjugated Bilirubin	<ul style="list-style-type: none">• Bound (20 days)• Bilirubin in urine is conjugated
Unconjugated Bilirubin	<ul style="list-style-type: none">• Not filtered or secreted• Nil in urine
Urobilinogen in urine	<ul style="list-style-type: none">• Normally traces• ↑ in Cholestaiss



Common Causes of Jaundice

- *Pre Hepatic (Acholuric) - Hemolytic*
 - *Unconjugated/Indirect Bil, pale urine*
- *Hepatocellular – Viral, alcohol, drugs*
 - *Liver damage - unconjugated*
 - *Swelling, canalicular obstruction - Conjugated*
- *Post Hepatic (Obstructive) – Stone, tumor*
 - *Conjugated/Direct Bil, High colored urine.*



Viral hepatitis

- *Acute / chronic liver damage.*
- *A, E & B,C (Primary)*
- *EBV, Yellow fever, Herpes, CMV (secondary)*
- *Cytotoxic – hepatocyte injury – necrosis.*
- *Apoptosis, inflammation, Lymphocyte infiltr.*
- *Bridging fibrosis, Cirrhosis.*



Alcoholic Liver Injury:

- *Ethyl alcohol: Common cause of acute/Chronic liver disease*
- *Alcoholic Liver disease - Patterns*
 - *Fatty change,*
 - *Alcoholic hepatitis (Mallory Hyalin)*
 - *Alcoholic Hepatic fibrosis*
 - *Alcoholic Cirrhosis*
- *All reversible except cirrhosis stage.*



Pathogenesis of Alcoholic Liver Injury

- *Diversion of fat & carbohydrate metabolism to alcohol – fat storage.*
- *Acetaldehyde – metabolite – hepatotoxic*
- *Increased peripheral release of fatty acids.*
- *Alcohol stimulates collagen synthesis*
- *Inflammation, Portal bridging fibrosis*
- *Micronodular cirrhosis.*

Alcoholic Fatty Liver



Alcoholic Fatty Liver



Portal Hypertension

- *Portal hypertension may be defined as a portal pressure gradient of 12 mm Hg or greater.*
- *Many conditions are associated with portal hypertension, of which cirrhosis is the most common cause.*

Symptoms of portal hypertension

- *Hematemesis or melena (gastroesophageal variceal bleeding or bleeding from portal gastropathy)*
- *Mental status changes such as lethargy, increased irritability, and altered sleep patterns (presence of portosystemic encephalopathy)*
- *Increasing abdominal girth (ascites formation)*
- *Abdominal pain and fever (spontaneous bacterial peritonitis [SBP], which also presents without symptoms)*
- *Hematochezia (bleeding from portal colopathy)*

Cirrhosis

- ◆ *End stage complication of liver disease*
- ◆ *“Diffuse disorder of liver characterised by; Complete loss of architecture, Replaced by extensive fibrosis with, Regenerating parenchymal nodules.”*



Etiology of Cirrhosis

- *Alcoholic liver disease* 60-70%
- *Viral hepatitis* 10%
- *Biliary disease* 5-10%
- *Primary hemochromatosis* 5%
- *Cryptogenic cirrhosis* 10-15%
- *Wilson's, α 1AT deficiency* rare

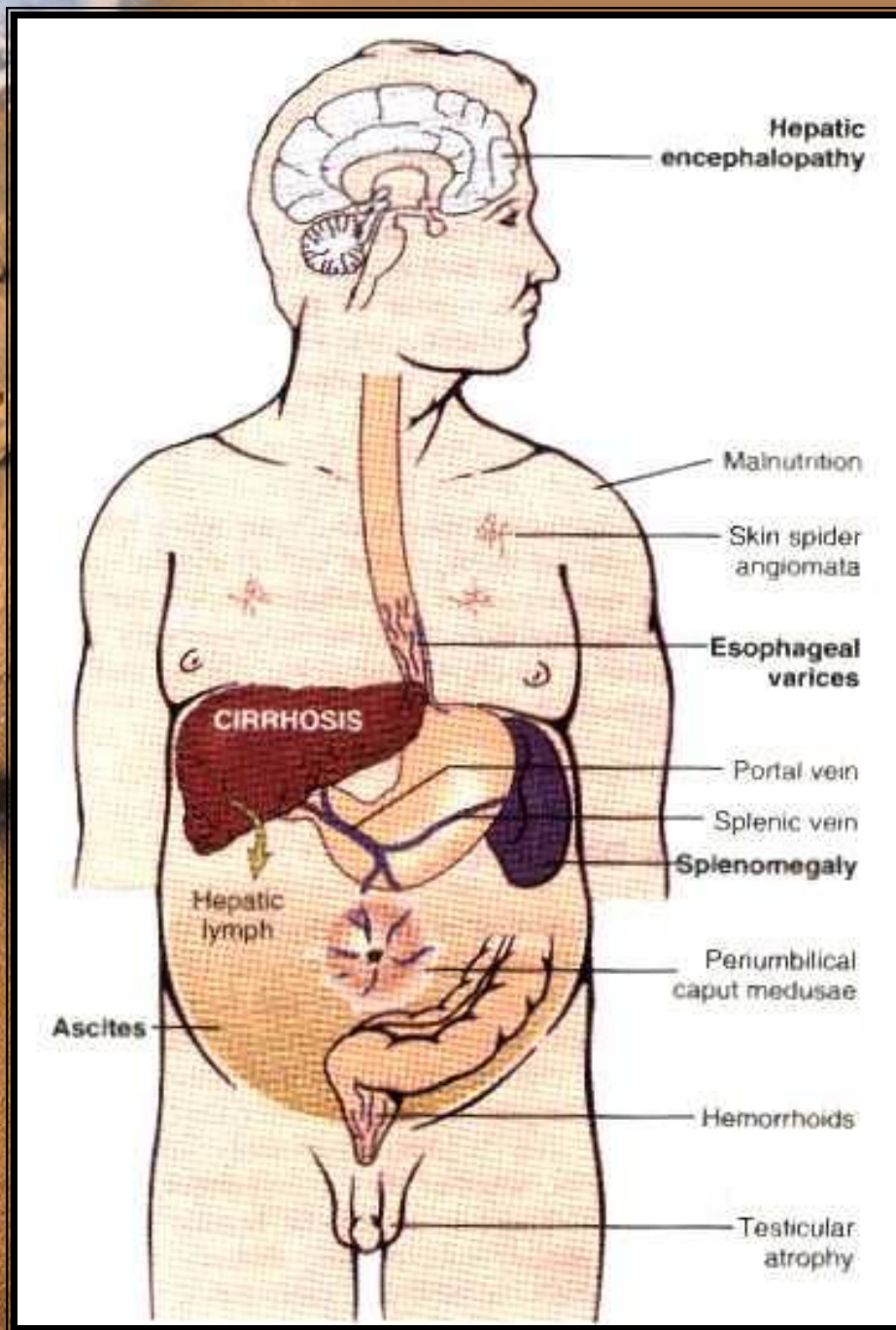
Pathogenesis of Cirrhosis

- *Diffuse liver injury leading to necrosis.*
 - *(Alcohol, virus, drugs, toxins, genetic etc.)*
- *Chronic inflammation & healing (hepatitis).*
- *Bridging fibrosis – loss of architecture.*
- *Regeneration → nodules.*
- *Obstruction to blood flow & shunts.*
- *Portal hypertension → spleen, varices*
- *Liver failure – Debilitation, Jaundice, Ascitis, edema, bleeding.*
- *Hormone imbalance – spider nevi, testes atrophy etc..*

Cirrhosis



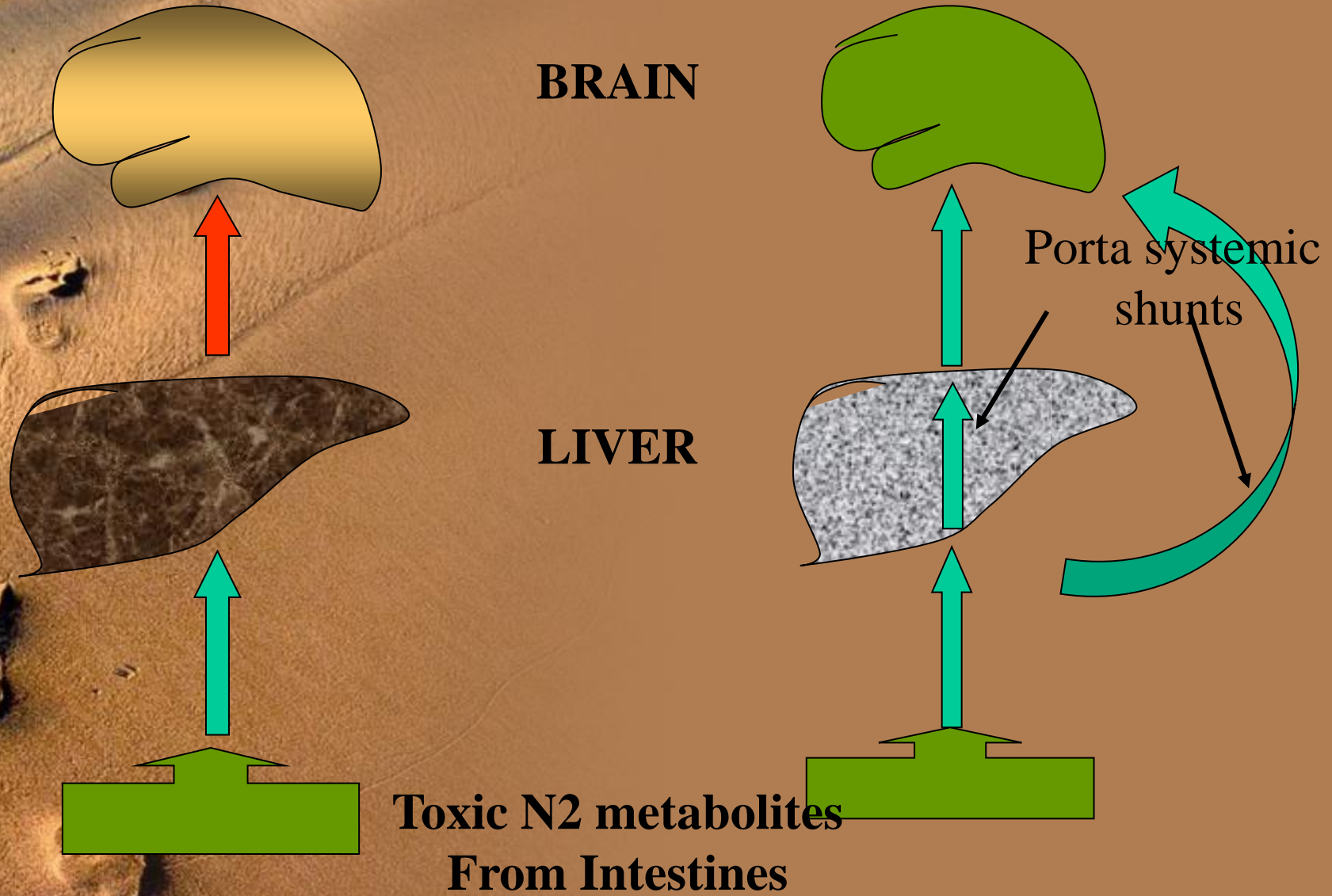
Cirrhosis Clinical Features



Ascitis in Cirrhosis



Pathogenesis of Hepatic Encephalopathy



NIDANA AND LAKSHANA OF KAMALA

पाण्डुरोगी तु योऽत्यर्थं पित्तलानि निषेवते ।

तस्यमसृग्मांसं दग्ध्वा रोगाय कल्पते ॥

हारिद्रानेत्रः स भृशं हारिद्रात्वङ्.नखाननः ।

रक्तपीतशकृमूत्रो भेकवर्णो हतेन्द्रियः ।

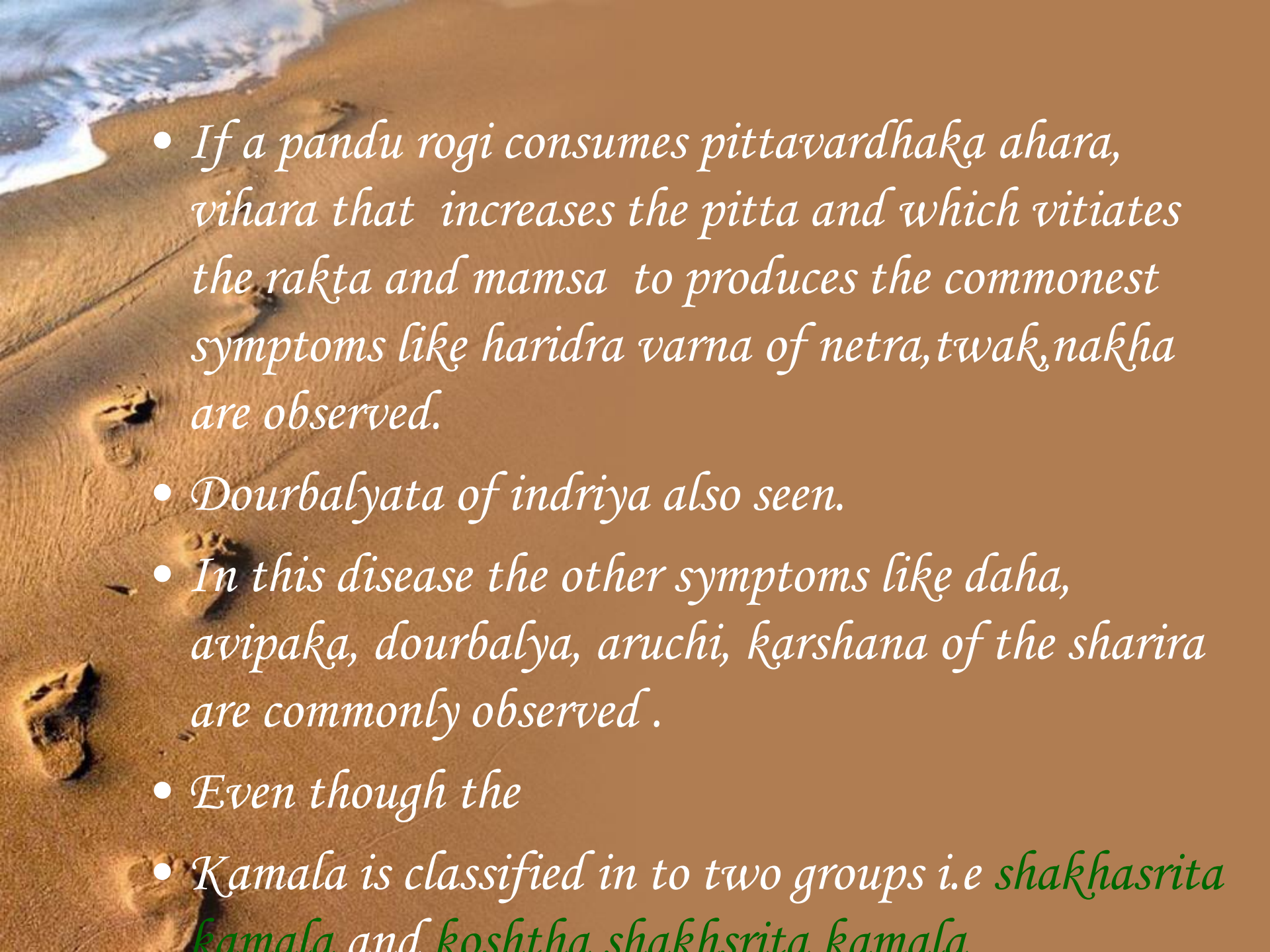
दाहाविपाकदौर्बल्यसदनारुचिकर्षितः ।

कामला बहुपित्तैषा कोष्ठशाखाश्रया मता ॥

च.चि.१६/३४-३६

भवेत्पित्तोल्बणस्यासौ पाण्डुरोगादृतेऽपि च ॥१७॥

अ.ह.नि.१३

- 
- If a pandu rogi consumes pittavardhaka ahara, vihara that increases the pitta and which vitiates the rakta and mamsa to produces the commonest symptoms like haridra varna of netra, twak, nakha are observed.
 - Dourbalyata of indriya also seen.
 - In this disease the other symptoms like daha, avipaka, dourbalya, aruchi, karshana of the sharira are commonly observed .
 - Even though the
 - Kamala is classified in to two groups i.e shakhsrita kamala and koshtha shakhsrita kamala.

Kumbha Kamala

कालान्तरात् खरिभुता कृच्छ्रा स्यात् कुम्भकामला । च.चि.१६/३६

उपेक्षया च शोफाढया सा कृच्छ्रा कुम्भकामला । अ.ह.नि १६

भेदस्तु तस्याः खलु कुम्भसाह्वः शोफो महांस्तत्र च पर्वभेदः ॥

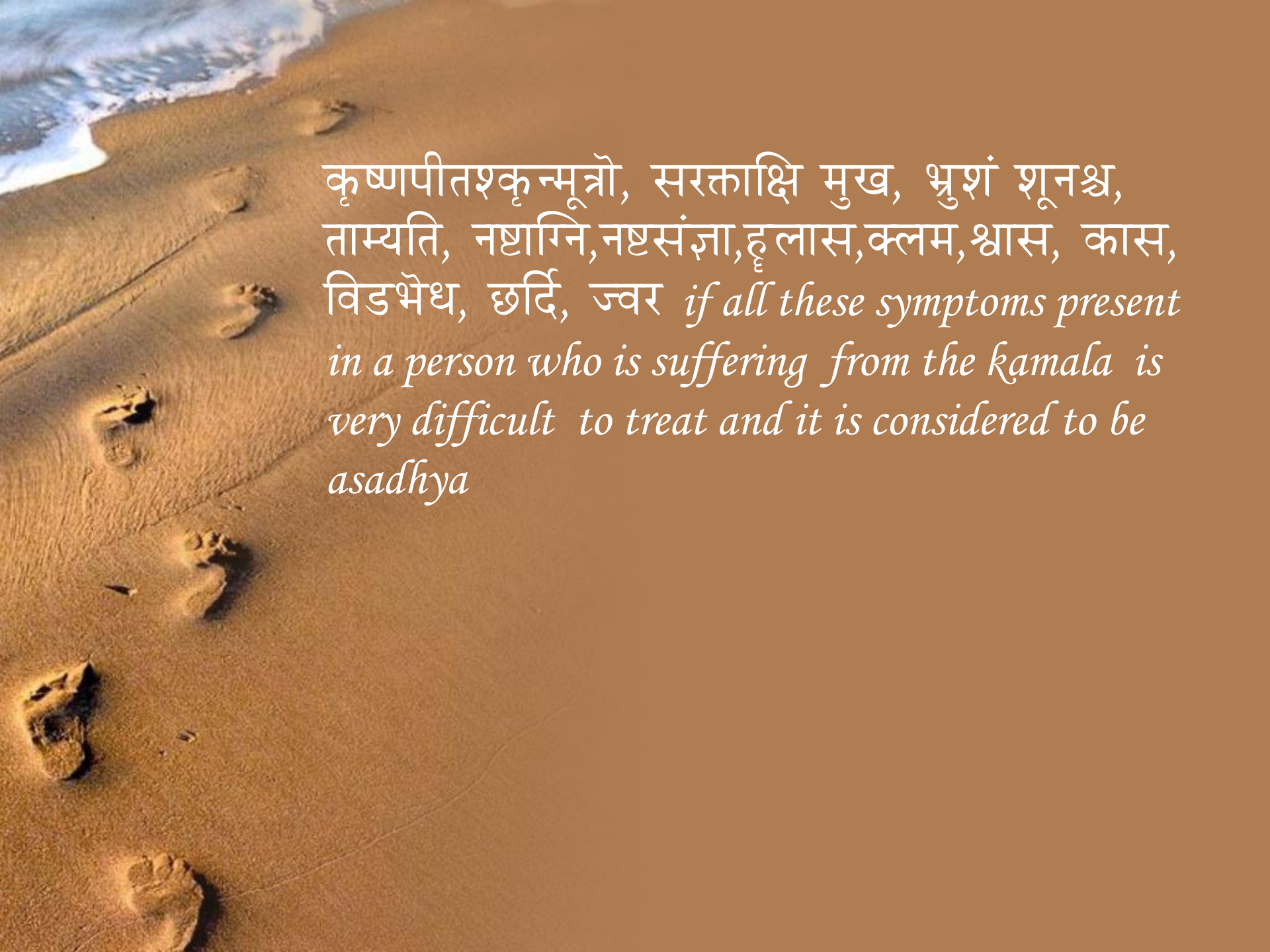
सु.उ.४४/११

Lakshana of kumbha kamala

- *Krushna peeta shkrut mootra – blackish yellow stool and urine*
- *Bhrushm shunah – severe edema of the body*
- *Sa rakṭakṣhi mukha chardi vit mutra – reddish sclera, mouth, vomitus, stool, urine*
- *Daha – burning sensation in the body*
- *Aruchi – anorexia*
- *Trushna – thirst*
- *Aanaha – constipation*
- *Tandra – drowsiness*

Asadhya Kamala

कृष्णापीतशकृन्मूत्रो भ्रुशं शूनश्च मानवः ।
सरक्ताक्षिमुखच्छर्दिविण्मूत्रो यश्च ताम्यति ।
दाहारुचितृषानाहतन्द्रामोहसमन्वितः ॥
नष्टाग्निसंज्ञः क्षिप्रं हि कामलावान् विपद्यते ।
साध्यानामितरेषां तु प्रवक्षामि चिकित्सितम् ॥
च.चि.१६/३७-३८



कृष्णापीतशकृन्मूत्रो, सरक्ताक्षि मुख, भ्रुशं शूनश्च,
ताम्यति, नष्टाग्नि, नष्टसंज्ञा, हृलास, क्लम, श्वास, कास,
विडभेध, छर्दि, ज्वर *if all these symptoms present
in a person who is suffering from the kamala is
very difficult to treat and it is considered to be
asadhya*

Chikitsa of kamala

विरेचन

In kamala virechana is considered to be a best line of treatment for pitta haranarthha

“विरेचनं पित्तहरणां श्रेष्ठः”

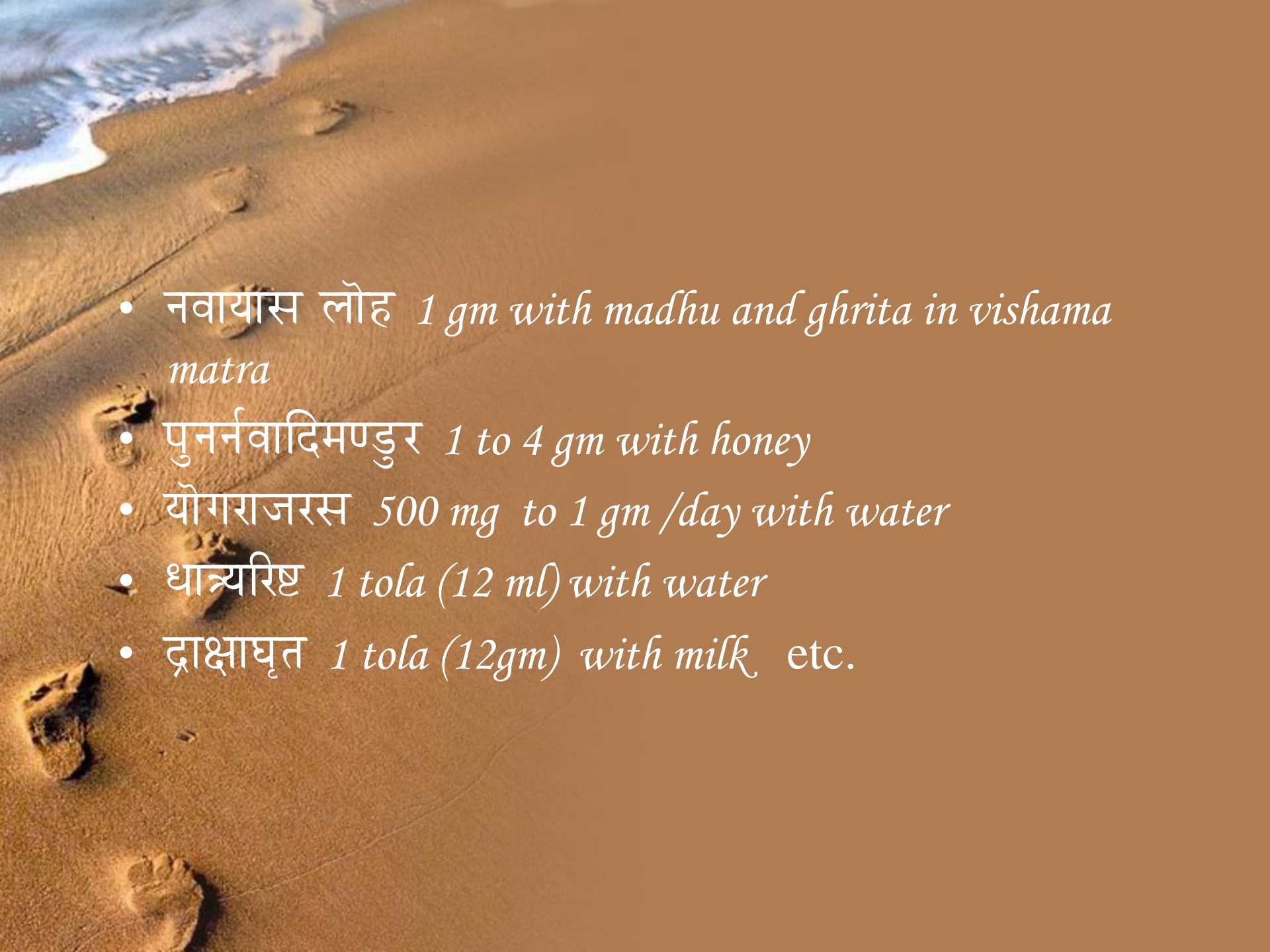
शमनौशधि

त्रिफलाया गुडुच्या वा दाव्या निम्बस्य वा रसः ।

प्रातर्माक्षिकसंयुक्तः शीलितः कामलापहः (चक्रदत्त)

त्रिफला क्वाथ
गुडूचि स्वरस
दारुहरिद्रा क्वाथ
निम्बत्वक क्वाथ

१ तौला *each one (10ml) wit madhu two times a day*

- 
- नवायास लोह 1 gm with madhu and ghrita in vishama matra
 - पुनर्नवादिमण्डुर 1 to 4 gm with honey
 - योगराजरस 500 mg to 1 gm /day with water
 - धान्त्र्यरिष्ट 1 tola (12 ml) with water
 - द्राक्षाघृत 1 tola (12gm) with milk etc.

Chikitsa of Kostha Shkhashrita Kamala

- १ कामलायां तु पित्तघ्नं पाण्डुरोगाविरोधि यत् । अ.ह.चि १६/४०
कमलायान्तु पित्तघ्नं
पाण्डुरोग अवरोधि यत्
- २ संशोध्यो म्रदुभिस्तिकैः कामली तु विरेचनैः। च.चि. १६/४०
- ३ रेचनं कामलार्तस्य स्निग्धस्यादौ प्रयोजयेत् ।
ततः प्रशमनी कार्य क्रिया वैध्येन जानता ॥ चक्रदत्त

स्नेहपान

पञ्च गव्य घृत

कल्याणक घृत

महातिकक घृत

Shakhasrita kamala chikita

तिलपिष्ट निभं यस्तु वर्चः स्रजति कामलि ।

श्लेष्मणा रुन्ध्य तत् पितं कफ हरैर्जयेत् ॥ च.चि.१६/१२४

कफ हरं पित्त वृधिकरं समं चिकित्सितमं ।

शाखाश्रय दोशस्य वृद्धि कोष्ठानयनार्थम् ॥

चक्रपाणि.च.चि.१६/१२८

There are some procedures to bring the shakhasraya dosha to
kosta i.e

वृद्ध्या विष्यन्दनात् पाकात् स्रोतोमुख विशोधनात् ।

शाखा मुक्त्वा मलाः कोष्ठं यान्ति वायोश्च निग्रहात् ॥

शाखाश्रय पितं स्वामाशयमेति याति (गन्गाधर च.चि.१६/१३१)

- *Shakhasrita pitta comes and settles in its sthan i.e swamashaya then above said line of treatment should be followed.*
- *Katuki, nimba, bhumyamalki, bhrungaraja, vasa, amruta, mulaka these drugs are very affective to treat the shakhasrita kamala.*

Yogas

- *Matulunga swarasa*
- *Amlavetas phala majja*
- *Apathya – avoid sneha dravyas and the food which is very heavy for digetion*
- *Pathya –yava, godhuma, mudga, adaki, masoor, shali, moolaka yusha, kulatha yush and laghu ahara*

Kumbha Kamala Chikitsa

- गौमूत्रेण पिबेत्कुम्भकामलायां शिलाजतु ॥५२॥
मासं माक्षिकधातुं वा किट्टं वाऽथ हिरण्यजम् ।

अ. हृ.चि.१६

- *The person who is suffering from kumbha kamala should have the shilajatu with gomutra or up to one month swarna makshika or roudya makshika with gomutra.*



Summary

- Kamala may be swatantra kamala or paratantra kamala.
- Kamala may be Koshta shakhasrita or shakhashrita.
- Clinical manifestation simulates liver disease injury.
- Symptoms of Kumbha kamala aching to symptoms of portal hypertension.
- Symptoms of Asadhya kamala tally's with cirrhosis of liver heading to complete liver failure.

An aerial photograph of a sandy beach. In the top left corner, the ocean waves are breaking onto the shore, creating white foam. A series of footprints are visible, starting from the water's edge and leading diagonally across the sand towards the right side of the frame. The sand is a warm, golden-brown color, and the overall lighting suggests a bright, sunny day.

Thanks for your attention!!