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FROM *ŚĀKHĀŚRITA KĀMALĀ* TO SURGICAL JAUNDICE: AN INTEGRATIVE REVIEW ON THE AYURVEDIC ROOTS OF OBSTRUCTIVE HEPATOBILIARY PATHOLOGY

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ABSTRACT

Surgical jaundice, a condition characterised by obstruction to the physiological flow of bile from the liver to the duodenum, finds its earliest conceptual foundation in the Ayurvedic description of Mārgāvarodha Janya Vikāra, particularly Śākhāśrita Kāmalā. The classical delineation of Kapha-induced obstruction of Pitta Pravṛtti and Vāta-driven displacement of Pitta into the Śākhās reflects a remarkably precise understanding of the pathophysiological sequence that modern medicine identifies as obstructive or surgical jaundice. It may thus be inferred that the fundamental principles underlying the modern comprehension of jaundice and its obstructive variants were, in essence, envisioned within the Ayurvedic framework, where the concepts of Āvarana, Doṣa Saṃmūrcchana, and Srotorodha collectively elucidate the mechanism of biliary obstruction described today. Ayurveda's therapeutic rationale emphasises Kapha-hara, Vāta-anulomana, and Pittānulomana measures to restore normal bile flow, while Anuśāstra Karma is advocated when internal Āvarodha or structural pathology persists. Among these, Kṣāra Karma, with its Lekhana, Chedana, and Bhedana properties, assists in dissolving Āma and Kapha, re-establishing Srotas patency; Agnikarma stimulates localised Agni and mitigates Kapha-Medo Āvarodha; and Rakta Mokṣaṇa enhances hepatic circulation and aids detoxification. The classical references to Pittāśaya Aśmarī by Ācārya Charaka and Śastra Karma indications by Ācārya Suśruta illustrate an advanced surgical vision consistent with the principles of hepatobiliary management. Hence, both classical and contemporary perspectives converge upon the same therapeutic goal – Mārgavivarana, or the removal of obstruction and restoration of natural flow. This review thus highlights the depth of Ayurvedic surgical and pathophysiological reasoning, demonstrating that the conceptual roots of modern hepatobiliary understanding are deeply embedded in Ayurvedic science.

KEYWORDS: *Kamala, Jaundice, Śākhāśrita Kāmalā, Surgical jaundice, Obstructive Jaundice, Mārgāvarodha Janya Vikāra, Kṣāra Karma, Agnikarma, Rakta Mokṣaṇa, Pittāśaya Aśmarī, Srotorodha, Hepatobiliary disorders.*

HISTORICAL INTRODUCTION

The term *Jaundice* finds its linguistic origin in the French word *"jaunis"*, which emerged around 1300 AD and literally translates to "yellow," signifying the classical hallmark of the condition — the yellowish discolouration of the skin and sclera due to hyperbilirubinemia.[1] The medical recognition of jaundice as a possible adverse effect of vaccination was first documented by Dr. Luhrman in 1885, marking one of the earliest associations between hepatic dysfunction and iatrogenic factors.[2] Subsequently, in 1908, Dr. McDonald hypothesised that jaundice could be caused by an etiological agent far smaller than any known bacterium, indirectly alluding to the existence of viral hepatitis long before the discovery of the hepatitis viruses themselves.[3] A significant milestone in the surgical understanding of jaundice was achieved in 1935 when A. O. Whipple, an eminent American surgeon, first described *obstructive jaundice*, delineating its pathophysiological distinction from hepatic and haemolytic variants.[4] In the same year, Whipple further revolutionised hepatobiliary surgery by introducing *preoperative biliary drainage* through a staged *pancreato-duodenectomy* procedure, a technique that laid the foundation for modern pancreatic and biliary surgical interventions.[5]

Introduction and Pathophysiology

Jaundice, derived from the French term *"jaunis"* meaning yellow, refers to the abnormal yellow discolouration of the skin, sclera, and mucous membranes resulting from elevated serum bilirubin levels. It is not a disease in itself but a clinical condition reflecting dysfunction within the *hepato-biliary-pancreatic system*.[1] Clinically termed *icterus*, it becomes visible when serum bilirubin exceeds 3 mg/dL.[6] The underlying biochemical disturbance, *hyperbilirubinemia*, arises due to impaired bilirubin metabolism — involving its overproduction, defective conjugation, or obstruction to

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excretion.[7] Physiologically, bilirubin is formed from haemoglobin breakdown and transported to the liver bound to albumin, where it is conjugated by *uridine diphosphate glucuronosyl transferase (UDPGT)* to become water-soluble.[8] The conjugated bilirubin is then excreted into bile, passing through the intrahepatic and extrahepatic ducts into the intestine, where it is converted into *urobilinogen* and *stercobilinogen*, giving urine and faeces their characteristic colour.[9] Any derangement in this coordinated function of the liver, bile ducts, or pancreas leads to the accumulation of bilirubin in the blood, manifesting as jaundice. Based on the site of disturbance, it is classified as *pre-hepatic* (haemolytic), *hepatic* (hepatocellular), or *post-hepatic* (obstructive). [10] Thus, jaundice serves as an important clinical indicator of pathology involving the *hepato-biliary-pancreatic axis*, warranting detailed evaluation for accurate diagnosis and management. [4]

Clinical Classification

Jaundice represents a clinical condition that may arise from diverse underlying causes, broadly categorised as *haemolytic (pre-hepatic)*, *hepatocellular (hepatic)*, and *obstructive (post-hepatic)* types, based on the site of pathology within the *hepato-biliary-pancreatic system*.[6] In *haemolytic or pre-hepatic jaundice*, excessive breakdown of red blood cells leads to overproduction of unconjugated bilirubin, exceeding the liver's capacity for conjugation. This form is generally *non-surgical*, as the primary pathology lies outside the biliary tract, though surgical intervention may be warranted in specific conditions such as *hypersplenism* or *sickle cell anaemia*, where *splenectomy* may be considered.[8]

In *hepatocellular or hepatic jaundice*, the dysfunction is within the liver parenchyma itself, resulting from hepatocellular injury or impaired conjugation and excretion of bilirubin.[7] These cases are also not typically surgical; however, *intrahepatic surgeries* may be indicated in selected cases, such as *hepatic carcinoma* or localised intrahepatic obstruction.[9]

Conversely, *obstructive or post-hepatic jaundice*—commonly termed *surgical jaundice*—is most often associated with structural or mechanical blockage of bile flow within the biliary or pancreatic ducts.[4] The obstruction may result from *choledocholithiasis*, *biliary strictures*, *pancreatic head pathology*, or *hepatic duct carcinoma*.[11] This type of jaundice frequently necessitates surgical or endoscopic intervention to relieve the obstruction and restore biliary drainage. Hence, from a clinical and surgical standpoint, while pre-hepatic and hepatic jaundice are primarily managed medically, *post-hepatic or obstructive jaundice* represents a major domain of surgical concern in hepatobiliary practice.[12]

Medical & Surgical Jaundice

Jaundice is broadly classified into *medical* and *surgical* types based on its cause and management approach. *Medical jaundice* results from infections or abnormal breakdown of red blood cells within the liver and biliary system, leading to impaired bilirubin metabolism.[6] Management focuses on addressing the underlying pathology—*corticosteroids, immunosuppressants,* and *folic acid supplements* for hematologic or bleeding disorders, and *antibiotics* for infections. Symptomatic relief is provided with *cholestyramine* and *ursodeoxycholic acid*.[8] Common causes include *hepatitis* and *sickle-cell anaemia*.

On the other hand, *surgical jaundice* occurs due to partial or complete obstruction of the bile ducts, resulting in a mechanical blockage to bile flow.[12] Conditions such as *strictures, calculi,* and *tumours* are common causes requiring surgical or interventional correction. Major procedures include *hepatic resection, tumour removal,* and *liver transplantation*, while minimally invasive approaches like *laparoscopic, endoscopic,* or *cryosurgical* techniques are increasingly preferred.[11] Hence, while *medical jaundice* is managed conservatively with pharmacological therapy, *surgical jaundice* necessitates structural correction to restore biliary drainage and prevent complications.[7]

Surgical Jaundice

Surgical jaundice is defined as a condition resulting from a ***structural or mechanical obstruction*** that impedes the normal flow of bile anywhere along the *hepato-biliary-pancreatic ductal system*, extending from the liver to the duodenum.[12] Such obstruction leads to the accumulation of conjugated bilirubin in the bloodstream, manifesting as yellow discolouration of the skin, sclera, and mucous membranes. The underlying causes are typically *anatomical or pathological lesions*, including *gallstones, strictures, tumours, or congenital anomalies* that physically block bile passage.[6] Since the aetiology is primarily mechanical, correction often necessitates *surgical or interventional procedures* such as *biliary drainage, cholecystectomy, hepaticojejunostomy,* or *pancreatoduodenectomy*, depending on the level and nature of obstruction.[11] Hence, the term *"surgical jaundice"* underscores the necessity for operative management to re-establish bile flow and prevent complications such as cholangitis, biliary cirrhosis, or hepatic failure.[7]

Surgical jaundice results from *structural* or *mechanical* obstruction to bile flow within the hepato-biliary-pancreatic system. ***Structural obstruction*** occurs due to anatomical or pathological changes such as *tumors, biliary strictures,* or *congenital anomalies*, whereas ***mechanical obstruction*** arises from physical blockage caused by *gallstones, parasites,* or *foreign bodies*.[12] These obstructions elevate conjugated bilirubin levels, producing cholestatic jaundice that often necessitates *surgical or endoscopic intervention* to restore bile drainage and prevent hepatic complications.[7]

Incidence and Mortality of Surgical (Obstructive) Jaundice

The incidence of *surgical or obstructive jaundice* varies according to the underlying aetiology, reflecting both benign and malignant causes of biliary obstruction. Among these, *choledocholithiasis* or common bile duct (CBD) stones account for the highest proportion, approximately *40%* of cases, followed by *carcinoma of the pancreatic head,* contributing to about *25%*.[12] *Cholangiocarcinoma* represents nearly *15%*, while *biliary strictures*—both benign and malignant—constitute around *8%*. *Gallbladder carcinoma* is relatively less frequent, comprising about *5%* of cases.[11] The prognosis largely depends on the nature and extent of obstruction; *malignant obstructive jaundice* carries a high *mortality rate of*

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up to 90%, whereas patients undergoing *percutaneous transhepatic biliary drainage (PTBD)* for malignant jaundice show a reduced mortality of around *16.8%*.[13] In contrast, *benign causes* of obstruction demonstrate a significantly lower mortality rate, ranging between *4.7% and 15.5%*.[7] These figures emphasize the importance of early diagnosis and appropriate surgical or interventional management to improve patient outcomes.

Pathophysiology of Surgical (Obstructive) Jaundice

The pathophysiology of *surgical jaundice* primarily involves obstruction of the bile ducts due to *gallstones, tumours, or strictures*, which prevents the normal passage of bile into the intestine.[6] As a result, bile accumulates within the liver, leading to increased pressure in the *intrahepatic and extrahepatic biliary channels*. This pressure causes *conjugated bilirubin* to leak into the bloodstream, resulting in *direct hyperbilirubinemia*.[7] The excess bilirubin is excreted through the kidneys, giving rise to *dark-coloured urine*, while the absence of bile in the intestinal tract causes *pale or clay-coloured stools* and *malabsorption of fats* due to lack of bile salts.[8] Deposition of bile salts in the skin produces *pruritus*, a common symptom of cholestasis. With *prolonged obstruction*, persistent stasis of bile leads to *hepatic inflammation*, which may progress to *secondary biliary cirrhosis*, further compromising liver function.[12] Thus, the sequence of events—from biliary obstruction to hepatic damage—defines the clinical and pathological basis of surgical jaundice, emphasising the need for timely intervention to restore bile flow.[11]

Benjamin's Classification of Biliary Obstruction (1983)

Benjamin's classification (1983) provides a comprehensive understanding of the varying degrees and patterns of biliary obstruction based on the underlying cause and clinical presentation. This system categorizes biliary obstruction into **four distinct types**, each reflecting the extent and chronicity of bile flow impairment.

Type I – Complete Obstruction

This form involves a *total blockage* of bile flow, often resulting from *pancreatic or cholangiocarcinoma tumours*, *iatrogenic common bile duct (CBD) ligation*, or *primary and secondary liver tumours*. Such obstructions lead to a marked elevation in conjugated bilirubin and require urgent surgical or interventional management.

Type II – Intermittent Obstruction

Characterised by *fluctuating blockage*, this type arises due to *choledocholithiasis (CBD stones)*, *periampullary tumours* involving the pancreatic ampulla, *choledochal cysts*, *bile duct papilloma (benign lesions)*, *haemobilia* resulting from bleeding within the biliary tree, and *duodenal diverticula* that exert mechanical pressure on the bile duct. The obstruction is episodic, often correlating with variations in symptom intensity.

Type III – Chronic Complete Obstruction

This form presents as a *sustained obstruction* leading to long-standing cholestasis. The causes include *bile duct strictures* (congenital, traumatic, or post-radiotherapy), *chronic pancreatitis* secondary to infection, and *cystic fibrosis,* where thick mucus impedes bile flow. Over time, this persistent blockage predisposes to *secondary biliary cirrhosis* and hepatic dysfunction.

Type IV – Segmental Obstruction

This type involves *localised or partial obstruction* of the bile ducts. Common aetiologies include *traumatic injury*, *sclerosing cholangitis* (chronic inflammatory condition), *cholangiocarcinoma* affecting segmental ducts, and *hepatolithiasis* involving stones within the intrahepatic biliary channels.

Presenting Complaints

Patients with obstructive or surgical jaundice commonly present with a constellation of symptoms reflecting hepatobiliary dysfunction and bile flow impairment. The **chief complaints** typically include *yellowish discoloration of the skin and sclera (jaundice)*, accompanied by *fever* and *generalized itching (pruritus)* due to bile salt deposition in the skin. Systemic manifestations such as *loss of appetite*, *unintended weight loss*, and *episodes of vomiting* are frequently observed, indicating metabolic and digestive disturbances. Additionally, *dark-coloured urine* resulting from excess conjugated bilirubin excretion and *pale or clay-coloured stools* due to absence of bile pigments in the intestine are hallmark features aiding in clinical diagnosis. These presenting symptoms collectively assist in identifying the underlying pathology and differentiating between medical and surgical causes of jaundice in clinical practice.

Clinical Presentation and History

The clinical presentation of **obstructive (surgical) jaundice** depends upon the nature, site, and duration of biliary obstruction. A detailed assessment of onset, duration, associated symptoms, and past surgical history is essential for accurate diagnosis and differentiation between medical and surgical causes.

1. Duration

- **Acute Onset:** Commonly seen in *gallstones, tumors,* and *acute cholangitis*, where symptoms appear suddenly.
- **Chronic Course:** Observed in *bile duct strictures* or *long-standing gallstones*, where jaundice develops gradually and persists over time.

2. Mode of Onset

- **Sudden Onset:** Characterized by *sharp right upper quadrant pain, fever,* and *chills*—features typically associated with *gallstones* and *cholangitis*.
- **Gradual Onset:** Marked by *progressive jaundice, pruritus, anorexia,* and *weight loss*, indicating *malignant or strictural obstruction*.
- **Intermittent Onset:** Due to *temporary blockage* by *migrating gallstones*, causing fluctuating episodes of jaundice.

3. Pain Characteristics

- **Painful Jaundice:** Suggestive of *gallstone obstruction* or *inflammatory conditions* like *cholangitis*.

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- **Painless Jaundice:** Typically associated with *malignant causes* such as *pancreatic carcinoma* or *cholangiocarcinoma*.

4. Associated Symptoms

- **Fever**
  - *Intermittent:* Seen in *gallstones* or *biliary strictures*.
  - *Continuous:* Occurs in *infective conditions* like *hepatitis* or *cirrhosis*.
- **Vomiting:** Frequently observed in *pancreatic stones, gallstones, choledochal cysts,* and *biliary tumors.*
- **Itching (Pruritus):** Due to bile salt deposition in the skin.
- **Dark Urine and Pale Stools:** Result from impaired bile flow and excess conjugated bilirubin excretion.

5. Diagnostic Clinical Triad

- **Charcot's Triad**
  Consisting of *intermittent fever, intermittent pain,* and *intermittent jaundice*, this triad is characteristic of *ascending cholangitis* and is indicative of *gallstones, pancreatic tumors,* or *biliary strictures.*

6. Past History

- **History of Biliary Surgery**
  A significant clue suggesting *post-surgical biliary stricture*, commonly seen after *cholecystectomy* or *biliary drainage procedures*

In summary, the pattern of onset, progression, pain, and associated systemic features provides valuable insight into the underlying cause of jaundice. Recognition of these clinical features helps in distinguishing *benign, malignant, or post-surgical* etiologies, forming the foundation for accurate diagnosis and management in both Ayurvedic and contemporary clinical practice.

Clinical Examination

A thorough clinical examination plays a vital role in the assessment and differentiation of various causes of obstructive or surgical jaundice. Both *inspection* and *palpation* provide significant diagnostic clues reflecting the extent of biliary obstruction and associated hepatobiliary pathology.

1. Inspection

On inspection, characteristic *scratch marks* are often observed over the **chest, abdomen, and back**, resulting from **pruritus** caused by the *accumulation of bile salts* in the skin due to cholestasis. In *thin-built patients*, a **pyriform-shaped swelling** may be visible at **Murphy's point**, located in the *right hypochondriac region*, which corresponds to the anatomical site of the gallbladder. This swelling is typically **smooth, soft, and moves with respiration**, indicating a distended gallbladder secondary to obstruction in the biliary outflow.

2. Palpation

On palpation, **Murphy's sign** serves as a key clinical indicator of gallbladder inflammation or obstruction. The examination is performed by placing the palpating hand at **Murphy's point** while the patient inspires deeply. A **sudden wince of pain and abrupt cessation of inspiration (shallow breathing)** denotes a *positive Murphy's sign*, suggesting *acute cholecystitis* or *gallbladder pathology*. This sign is best elicited when the patient is in a **sitting position**.

An alternative approach, **Moynihan's method**, follows the same principle as Murphy's sign but is performed with the patient in a **standing position**. Both signs provide valuable diagnostic evidence of gallbladder tenderness and inflammation, assisting in clinical evaluation of biliary disorders.

In essence, physical examination findings—especially the presence of *scratch marks, pruritus, Murphy's sign,* and *visible swelling at Murphy's point*—form an essential part of bedside diagnosis. They serve as crucial clinical correlates in identifying *surgical causes of jaundice* and help in guiding further diagnostic evaluation and management.

Biochemical Markers in Obstructive (Surgical) Jaundice

Biochemical investigations play a crucial role in diagnosing, differentiating, and assessing the severity of *obstructive jaundice*. Various haematological and biochemical parameters provide insight into the underlying hepatic and biliary pathology, aiding both clinical diagnosis and prognostic evaluation.

1. Hematological Parameters

- **Hemoglobin (Hb%)**

A markedly *low haemoglobin level* indicates a *gradual and progressive course*, often suggestive of *malignancy* or a *chronic disease process*.

- **Bleeding Time (BT), Clotting Time (CT), and Prothrombin Time (PT)**

These parameters are frequently *prolonged* due to *altered absorption of Vitamin K*, which is fat-soluble and requires bile salts for intestinal absorption. This alteration reflects impaired hepatic synthesis of clotting factors secondary to biliary obstruction.

2. Urine Examination

- **Urine Bilirubin:** *Present* in cases of *obstructive jaundice*, indicating increased excretion of *conjugated bilirubin* through the kidneys.
- **Urobilinogen:** *Absent*, due to blockage of bile flow into the intestines, which prevents conversion of bilirubin into urobilinogen.
  These findings serve as an **important confirmatory marker** for *biliary tract obstruction*.

3. Serum Enzyme Studies

a. Serum Alkaline Phosphatase (ALP)

- **Normal Range:** 60–300 units/L
- **Marked Elevation (>500 units/L):** Strongly suggestive of *obstructive jaundice*, as ALP is produced by *cholangioles (small bile ducts)* and excreted primarily through the *biliary tree*.
- **Gross Elevation:** Observed in *obstructive jaundice, biliary cirrhosis,* and *bone diseases*.
- **Mild Elevation:** Seen in *hepatic metastasis, hepatic abscess,* and *hepatitis*.
- In *cholangitis*, *bilirubin levels are relatively low* while *alkaline phosphatase levels remain markedly elevated*, reflecting predominant biliary epithelial involvement.

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b. Gamma Glutamyl Transpeptidase (GGT)

- **Normal Range:** 5–40 units/L
- **Marked Elevation:** Confirms *hepato-biliary pathology*, as GGT is an enzyme predominantly localised in the *liver and biliary tract*.
- A *parallel rise* in ALP and GGT strongly indicates *biliary obstruction or cholestatic liver disease*, distinguishing it from isolated hepatic or bone pathology.

Etiological Classification of Obstructive (Surgical) Jaundice

Obstructive jaundice results from diverse pathological processes that impede bile flow either by intrinsic blockage or external compression. The major etiological groups include:

- **Congenital Causes:** *Biliary atresia* and *choledochal cysts*—developmental anomalies of the biliary tract leading to early-life obstruction.
- **Inflammatory Causes:** *Ascending cholangitis* and *sclerosing cholangitis*—inflammatory narrowing and fibrosis of bile ducts.
- **Obstructive Causes:** *Common bile duct (CBD) stones*, *biliary strictures*, and *parasitic infestations*—direct mechanical blockage within the ductal system.
- **Neoplastic Causes:** *Carcinoma of the head of pancreas*, *periampullary carcinoma*, *cholangiocarcinoma*, and *Klatskin tumor*—malignant growths causing intrinsic or compressive obstruction.
- **Extrinsic Compression:** *Enlarged lymph nodes* or *adjacent tumors* exerting pressure over the CBD, resulting in impaired bile drainage.

Surgical Management of Surgical Jaundice

Surgical jaundice, also known as **obstructive jaundice**, occurs due to mechanical blockage in the extrahepatic biliary system, leading to impaired bile flow and accumulation of conjugated bilirubin in the bloodstream. The management primarily involves **surgical and endoscopic interventions** aimed at relieving obstruction, restoring biliary drainage, and preventing complications such as cholangitis, biliary cirrhosis, or hepatic failure.

The selection of procedure depends on the underlying aetiology, site of obstruction, disease stage, and hepatic functional reserve.

1. Biliary Atresia

Biliary atresia is a congenital obliterative cholangiopathy presenting in infancy, characterized by progressive fibrosis of the bile ducts leading to biliary obstruction and cirrhosis.

The **Kasai portoenterostomy** is the initial surgical treatment aimed at restoring bile flow by connecting the porta hepatis directly to the small intestine.

However, in cases where the Kasai procedure fails, complications such as severe malnutrition, recurrent cholangitis, and portal hypertension develop.

For such advanced stages, liver transplantation remains the definitive and curative treatment option.

2. Choledochal Cyst

Choledochal cysts are congenital cystic dilatations of the biliary tree that predispose to cholangitis, pancreatitis, and malignancy. Surgical excision of the cyst with biliary reconstruction is the standard treatment.

Types of Surgical Intervention

- **Open Cyst Excision:** Conventional and complete removal of cyst with Roux-en-Y hepaticojejunostomy.
- **Laparoscopic Cyst Excision:** Minimally invasive technique with reduced postoperative morbidity and faster recovery.
- **Robot-Assisted Excision:** Provides superior precision and better visualization, improving surgical outcomes.

3. Ascending Cholangitis

Ascending cholangitis is a life-threatening infection secondary to biliary obstruction and bacterial reflux into the biliary system. It constitutes a surgical emergency requiring prompt decompression of the biliary tract.

Emergency Management includes

1. **Endoscopic stone extraction and biliary stenting** to relieve obstruction.
2. **Biliary reconstructive procedures** for persistent or recurrent obstruction.
3. **Liver transplantation** in end-stage liver dysfunction caused by prolonged cholangitis or biliary sepsis.

Early and aggressive management reduces morbidity and prevents septic shock.

4. Sclerosing Cholangitis

Primary Sclerosing Cholangitis (PSC) is a chronic progressive cholestatic disorder marked by inflammation and fibrosis of intrahepatic and extrahepatic bile ducts.

Medical Management

- **Ursodeoxycholic acid** is administered to improve bile flow and delay fibrosis.

Surgical Management

- **Endoscopic dilatation and stenting** of dominant strictures.
- **Biliary reconstruction** in localized or resectable cases.
- **Liver resection** for segmental disease.
- **Liver transplantation** for end-stage PSC, which offers the best long-term survival.

5. Parasitic or Obstructive Biliary Disease

Obstruction due to parasitic infestation or external compression requires a combined approach.

Management Includes

- **Anti-helminthic therapy** to eradicate parasites.
- **Endoscopic drainage (ERCP)** or **Percutaneous Transhepatic Cholangiography (PTC)** for decompression.
- **Surgical removal** of the obstruction when endoscopic methods fail.

Diagnosis is aided by Ultrasound, CT Scan, and MRCP to determine the site and cause of obstruction.

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6. Common Bile Duct (CBD) Stones

Choledocholithiasis represents one of the most common causes of surgical jaundice.

Management focuses on complete clearance of the duct and restoration of biliary drainage.

a. Diagnostic Tools

- **Ultrasound (USG):** Detects ductal dilatation and gallstones.
- **CT Scan:** Helps evaluate stone size and associated complications.
- **MRCP:** Non-invasive and sensitive for biliary anatomy.
- **ERCP: Gold standard** diagnostic and therapeutic procedure.

b. Endoscopic Retrograde Cholangiopancreatography (ERCP)

ERCP serves as the **standard treatment** for CBD stones.

Clinical Indications

- CBD stones
- Biliary stricture
- Acute cholangitis or biliary pancreatitis

Performed within **24–48 hours**, it enables **stone extraction**, **sphincterotomy**, and **stent placement**, offering immediate relief of obstruction.

c. Trans-Duodenal Sphincteroplasty

This procedure is indicated when **endoscopic methods fail**.

Indications

- Multiple CBD stones
- Impacted ampullary stones
- Sphincter of Oddi dysfunction

It involves surgical enlargement of the sphincteric opening through the duodenum to facilitate bile drainage and prevent recurrence.

d. Choledochojejunostomy

A **bypass procedure** in which the **common bile duct** is anastomosed directly to a loop of **jejunum**.

Indications

- Failed endoscopic stone removal
- Biliary stricture or tumor causing distal obstruction

It provides a new drainage pathway for bile, bypassing the blocked segment.

e. Choledocholithotomy

Direct **surgical incision and exploration of the common bile duct** to remove stones.

Indications

- Failure of ERCP
- Large or impacted stones

Laparoscopic Choledocholithotomy is the minimally invasive alternative, associated with shorter hospitalization and faster recovery.

7. Periampullary Carcinoma

Periampullary carcinoma arises from the **ampulla of Vater**, **lower common bile duct**, or **duodenum**, and often presents with painless progressive jaundice.

Surgical Management:

- **Whipple's Operation (Pancreaticoduodenectomy):** Curative procedure for resectable tumors.
- **Triple Bypass Surgery:** Palliative option for unresectable malignancy.
- **ERCP with stenting:** Provides palliation by relieving biliary obstruction.

Kamala

In Ayurveda, *Kamala* is a *Pittapradhana Vyadhi* primarily affecting the *Yakṛt* (liver) and *Pittavaha Srotas* (biliary channels), closely resembling jaundice described in modern medicine.[14] It occurs due to *Pitta* vitiation, leading to yellow discolouration of the skin, eyes, and urine. *Kamala* is broadly classified into *Koshṭhaśrita Kamala* and *Śākhāśrita Kamala*. *Koshṭhaśrita Kamala* arises from excessive *Pitta* within the gastrointestinal tract and resembles *hepatocellular (medical) jaundice*, while *Śākhāśrita Kamala* manifests when *Pitta* spreads to peripheral tissues, correlating with *obstructive (surgical) jaundice*.[15] Advanced forms like *Halīmakā* and *Kumbha Kamala* indicate chronic or neglected stages, similar to *secondary biliary cirrhosis* or *chronic liver disease*.[16] Thus, Ayurveda interprets *Kamala* as a systemic disorder of *Pitta* metabolism, mirroring the hepatobiliary dysfunction recognised in contemporary hepatology.[17]

Table No. 1: Types of Kamala according to various *Acharyas.*

Types Of *Kaamala*	*Charaka*	*Sushrutha*	*Vaghbhata*	*Madhava*	*Sharanga*	*Bhavaprakash*
Koshtashritha-Shakashritha	✓		✓	✓		✓
Shakashritha	✓		✓	✓		✓
Kumbha	✓	✓	✓	✓	✓	✓
Haleemaka	✓		✓	✓	✓	✓
Lagharaka		✓				
Apanika		✓				
Alasakhya		✓				

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Śākhāśrita Kamala

तिलपिष्टनिभं यस्तु वर्चः सृजति कामली।
श्लेष्मणा रुद्धमार्गं तत् पित्तं कफहरैर्जयेत्।
कफसम्मूर्च्छितो वायुः स्थानात् पित्तं क्षिपेद् बली॥

(Charaka Samhita, Chikitsa Sthana 16/124)

In this verse, *Āchārya Charaka* describes *Śākhāśrita Kamala*, an advanced form of *Kamala* where the vitiated *Pitta* becomes obstructed by *Kapha* within the *Pittavaha Srotas*. The stagnated *Pitta* is further displaced by *vitiated Vāta* into the *Śākhā* (peripheral tissues), leading to deeper systemic involvement. The cardinal clinical feature of this condition is the passage of stool resembling *tilapiṣṭa nibham varcas*—the color and texture of sesame paste—indicative of severe biliary obstruction and deranged bile metabolism.

This stage of *Kamala* is considered more chronic and difficult to treat, corresponding closely to *obstructive or surgical jaundice* in modern hepatology, where bile flow is blocked mechanically, leading to systemic retention of bile pigments. Therapeutic management, as advised by *Āchārya Charaka*, involves the use of *Kapha-hara* and *Pitta-virechana* measures to remove the obstruction and restore bile flow, aligning conceptually with modern approaches aimed at relieving obstruction and detoxification.

Rūpa (Clinical Manifestations) of Śākhāśrita Kāmalā —

हारिद्रनेत्रमूत्रत्वक् श्वेतवर्चास्तदा नरः।
भवेत् साटोपविष्टम्भो गुरुणा हृदयेन च।
दौर्बल्यात्पाग्निपार्श्वार्तिहिक्काश्वासारुचिज्वरैः॥
(Cha. Chi. 16/127–128)

The concept of *Śākhāśrita Kāmalā*, as delineated by *Āchārya Charaka*, forms the classical Ayurvedic foundation for what is now identified in modern hepatobiliary science as **obstructive jaundice**. In this condition, *Pitta*, the principal bio-entity responsible for colouration and metabolism, becomes obstructed by aggravated *Kapha* within the biliary channels (*Śleṣhmaṇa Ruddha Mārgatva*). Propelled by *Vāta*, this vitiated *Pitta* is displaced from its natural seat in the liver to the *Śākhā* (peripheral tissues), manifesting as the clinical entity of *Śākhāśrita Kāmalā*.

The *Rūpa* or characteristic manifestations described include *Hāridra Netra* (yellowish eyes), *Hāridra Twak* (yellow skin), *Hāridra Mutra* (dark urine), and *Śveta Varcas* (pale stools)—clear indicators of altered bile flow and pigment retention. Systemic symptoms such as *Daurbalya* (weakness), *Alpāgni* (poor digestion), *Viṣṭambha* (abdominal fullness), *Pārśva Ārti* (right hypochondriac pain), *Ārući* (loss of appetite), and *Jvara* (fever) further define the internal derangement of *Pitta* and hepatic function.

Thus, long before the anatomical and biochemical recognition of bile duct obstruction in contemporary science, *Āyurveda* had already elucidated the *pathogenesis and symptomatology* of this disorder through the comprehensive understanding of *Śākhāśrita Kāmalā*, emphasising the obstruction (*Mārgavarodha*) of *Pitta Vaha Srotas* as its root cause.

Samprāpti (Pathogenesis) of Śākhāśrita Kāmalā in the Light of Ṣaṭkriyākāla

The evolution of *Śākhāśrita Kāmalā*—the Ayurvedic correlate of **obstructive jaundice**—is well conceptualized through the *Ṣaṭkriyākāla*, the six stages of disease manifestation described by *Āchārya Sushruta*. The sequence begins with *Nidāna* Sevana, where indulgence in causative factors such as excessive *Amla, Lavana, Katu rasa* and unwholesome dietary habits leads to ***Chaya*** (latent accumulation) of *Vāta* and *Kapha doṣas* within the *Pittavaha Srotas* (biliary channels). During the ***Prakopa*** stage, these doṣas become aggravated and start disturbing the normal flow of *Pitta*, resulting in impaired bile secretion and stasis within the hepatic pathways.

As the process advances to ***Prasara***, the vitiated *Kapha* causes *Avarodha* (obstruction) in the *Pittavaha Srotas*, preventing the normal outflow of *Pitta*. Simultaneously, the aggravated *Vāta* displaces the obstructed *Pitta* from its natural seat (*Koshtha*) into the peripheral channels (*Śākhā*), marking the ***Sthāna Saṃśraya*** stage. This stage denotes the classical shift of bile pigments into circulation and tissues—clinically reflected as *Tilapiṣṭa-nibha mala pravṛtti* (clay-colored stool) and *Haridra-netra-tvak* (yellow discoloration of sclera and skin).

During the ***Vyakti*** stage, the disease becomes clinically evident with pronounced features of *Kamala*, including dark-colored urine, pale stools, pruritus, weakness, and right hypochondriac pain, corresponding to biliary obstruction in modern terms. Prolonged obstruction and retained *Pitta* in the hepatic channels result in *Yakṛt saṅcaya* (hepatic congestion) and inflammatory changes, reflecting the chronic pathology of obstructive jaundice. Ultimately, in the ***Bheda*** stage, the condition differentiates into *Śākhāśrita Kāmalā*, wherein *Pitta* is firmly lodged in the *Śākhā* due to sustained obstruction and *Mārgavarodha*, mirroring the chronic and progressive nature of surgical jaundice in modern hepatobiliary pathology.

Thus, Ayurveda, through its profound understanding of *Doṣa-Duṣya Sammūrchana* and *Srotorodha Samprāpti*, had already elucidated the entire pathophysiological cascade of *Śākhāśrita Kāmalā*—long before the anatomical and biochemical recognition of biliary obstruction in modern medicine.

Chikitsā in Ayurveda – The Management of Śākhāśrita Kāmalā

"श्लेष्मणा रुद्धमार्गं तत् पित्तं कफहरैर्जयेत्।"
(Cha. Chi. 16/124)

1. Kaphahara Chikitsā (Management of Obstruction)

Āchārya Charaka emphasises that *Kapha* acts as the primary obstructive factor preventing *Pitta* from performing its normal function within the *Pittāśaya* (liver and biliary system). Hence, the foremost therapeutic line aims at removing *Kaphaja Mārgāvarodha* (Kapha-induced obstruction). Drugs and regimens having *Katu rasa*, *Tikta rasa*, *Uṣṇa* and *Tīkṣṇa guṇa* are preferred to liquefy and expel the accumulated Kapha, thereby restoring bile flow. This correlates with the modern rationale of clearing biliary obstruction and enhancing hepatic drainage.

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"कटुतीक्ष्णोष्णलवणैर्भृशाम्लैश्चाप्युपक्रमः ।
आपित्तरागाच्छकृतो वायोश्चाप्रशमाद्भवेत् ॥
स्वस्थानमागते पित्ते पुरीषे पित्तरञ्जिते ।
निवृत्तोपद्रवस्य स्यात्पूर्वः कामलिको विधिः ॥"
(Cha. Chi. 16/130–131)

2. Śākha-to-Koṣṭha Chikitsā (Mobilization of Pitta to Its Natural Seat)
The next therapeutic step focuses on restoring the displaced *Pitta* from the *Śākha* (peripheral tissues) back to its *Koṣṭha* (liver–intestine axis). Formulations and measures possessing *Katu-Tīkṣṇa-Uṣṇa-Lavaṇa-Amla yoga* are advocated, as they stimulate *Agni*, dislodge *Āvarodha*, and promote *Pitta-pravṛtti* (normal bile secretion). This process mirrors the resolution of bile stasis in obstructive jaundice, allowing *Rañjaka Pitta* to resume its physiological function of bile coloration.

3. Vāta-Śamana and Anulomana Chikitsā
Due to obstruction and displacement, *Vāta doṣa* becomes vitiated, further aggravating the condition by propelling *Pitta* into improper channels. Hence, *Vāta-Anulomana* and *Śamana* therapies—using mild snehana (unctuous), svedana (sudation), and carminative measures—are employed to regulate *Vāta* and re-establish its normal downward movement. This supports the bile flow physiologically and prevents recurrence of obstruction.

4. Saṃśamana Chikitsā (Pacification Therapy)
After successful removal of obstruction and restoration of bile flow, *Saṃśamana* measures are undertaken to pacify residual *Doṣas* and rejuvenate hepatic function. Herbs and formulations possessing *Tikta rasa*, *Pitta-Śāmaka* and *Yakṛt-uttējaka* properties such as *Bhūmyāmalakī, Katukī, Daruharidrā,* and *Triphala* are advised. This stage corresponds to anti-inflammatory, hepatoprotective, and bile-modulating therapy in modern hepatology.

Anuśasthra Karma in Śākhāśrita Kāmalā
In Ayurvedic therapeutics, *Anuśasthra Karma* (para-surgical measures) play a crucial role in managing systemic disorders where *Doṣa-āvarana* and *Srotorodha* are predominant. In *Śākhāśrita Kāmalā* (obstructive jaundice), these measures assist in relieving obstruction, balancing *Doṣas*, and stimulating *Agni* (metabolic fire) for the restoration of normal *Pitta pravṛtti*. Classical references from *Suśruta Saṃhitā* and other authoritative texts elaborate the use of *Kṣāra Prayoga, Agnikarma,* and *Rakta Mokṣaṇa* in such conditions.

1. Kṣāra Prayoga (Use of Alkaline Preparations)
a. Reference and Preparation
Āchārya Suśruta describes *Pānīya Kṣāra* preparation in *Uttara Tantra – Gulma Roga Adhyāya* as a potent *Anuśasthra Karma* for disorders arising due to *Mārgāvarodha* and *Āma sañcaya*. The method involves extraction and purification of plant-based alkaline substances that possess *Uṣṇa, Tīkṣṇa, and Lekhana guṇas* capable of liquefying and disintegrating obstructions within *Srotas*.

b. Indications (Su. Su. 11/8)
Suśruta mentions *Kṣāra Prayoga* as effective in:

- ***Agnisanga, Ajīrṇa, Arochaka*** – impaired digestion and anorexia
- ***Abhyantara Vidradhi*** **(***Yakṛt Vidradhi***)** – internal abscesses, especially hepatic
- ***Gulma Roga*** – gaseous abdominal masses
- ***Krimi Roga*** – parasitic infestations
- ***Aśmarī (****Pittāśaya Aśmarī****)*** – calculi of biliary origin

These indications highlight its *Srotoshodhaka* and *Kledahara* actions, which are pathophysiologically relevant to *Śākhāśrita Kāmalā*, where *Kapha-Pitta āvarana* obstructs *Pitta pravṛtti*.

c. Doṣānusāra Kṣāra Prayoga (Su. Su. Aśmarī Adhyāya)
According to *Suśruta*, *Kṣāra* selection depends on *Doṣic predominance*:

- *Vātaja* conditions**: *Pāṣāṇabheda Kṣāra***
- *Pittaja* conditions**: *Kuśādhya Kṣāra***
- *Kaphaja* conditions**: *Varuṇādhya Kṣāra***

Thus, individualized administration according to *Doṣa* ensures precision and safety. The *Kaphahara* and *Lekhana* properties of these *Kṣāras* assist in resolving *Pitta-mārga āvarodha* in obstructive jaundice.

2. Agnikarma (Thermal Cauterization Therapy)
a. Classical Reference
Basavarājīyam prescribes *Agnikarma* as a therapeutic intervention for hepatic and biliary disorders where *Āvaraṇa* or *Srotorodha* by *Kapha* and *Meda* impairs bile flow.

b. Site and Technique

- **Site:** *Ghuṭika* (elbow joint) and *Manibandha* (wrist joint) of *Dakṣiṇa Hasta* (right hand)
- **Type:** *Vālaya Agnikarma* (circular cauterisation)
- **Instrument:** *Swarna, Tāmra,* or *Rājata Śalākā* (gold, copper, or silver probe)

The heat application in these *Marma-upaśrita siras* stimulates *Agni*, alleviates *Kapha āvaraṇa*, and normalises *Pitta pravṛtti*.

c. Contemporary Correlation
Dr. R. B. Gogate's research highlights *Agnikarma* as a supportive therapy in *Kāmala*, emphasising its role in stimulating hepatic metabolism, improving circulation, and restoring bile flow — a clear example of Ayurvedic procedural precision aligning with hepatobiliary physiology.

3. Rakta Mokṣaṇa (Bloodletting Therapy)
a. Classical Reference and Rationale
Āchārya Suśruta, while explaining *Yakṛt Vikāra* and *Yakṛdalyodara*, advocates *Rakta Mokṣaṇa* as a prime *Śodhana* therapy. It eliminates *Dushta Rakta* and relieves hepatic congestion, directly addressing the *Rakta-Pitta-Pradoshaja* nature of *Kāmala*.

b. Indicated Sites

- ***Dakṣiṇa Bāhu Kūrpāra Sandhi Sira*****:** vein at the elbow joint of the right arm
- ***Kaniṣṭhikā-Anāmikā Madhya Sthita Sira*****:** vein between the little and ring fingers

c. Therapeutic Relevance
In *Śākhāśrita Kāmalā*, *Rakta Mokṣaṇa* relieves *Rakta Duṣṭi*, improves circulation, reduces hepatic stasis, and complements

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Kaphahara and *Pittavirechana* measures, ensuring multi-dimensional management of the condition.

Śāstrakṛta Chikitsā (Surgical Perspectives in Ayurveda)

The concept of *Śāstrakṛta Chikitsā* in Ayurveda demonstrates the profound surgical understanding of the classical Ācāryas, extending beyond external lesions to encompass internal obstructions (*Mārgāvarodha*) such as those seen in *Śākhāśrita Kāmalā*. Ācārya Charaka, while describing *Mutrāśmarī*, provides the first insight into *Pittāśaya Aśmarī* (biliary calculi), comparing it to *Gorocanā*, the yellow concretions found in the gallbladder of a cow — a clear indication of recognition of biliary pathology. He mentions:

"Viśoṣayed basti gataṃ saśukraṃ mūtraṃ sapittaṃ pavanaḥ kaphaṃ vā | yadā tadā'śmaryupajāyate tu krameṇa pitteṣviva rocanā goḥ ||" (Cha. Chi. 26/36)

This highlights that the vitiated *Vāta* and *Kapha* dry up and harden the *Pitta* or *Mūtra*, leading to the formation of *Aśmarī*, much like *Gorocanā* in cattle — establishing the Ayurvedic foundation of cholelithiasis and obstructive jaundice.

Further, Ācārya Suśruta in *Cikitsāsthāna (Su. Chi. 7/33)* elaborates the surgical approach for *Aśmarī* management:

"Tataḥ savye pārśve sevanīṃ yavamātreṇa muktvā'vacārayech chastra-maśmarī-pramāṇaṃ, dakṣiṇato vā kriyāsaukarya-hetorityeke."

He states that when *Aśmarī* (stone or obstruction) becomes chronic or causes severe distress, *Śastra Karma* (surgical intervention) must be undertaken. The incision (*cheda mārga*) is to be decided based on the size and location of the *Aśmarī*, ensuring precise removal and restoration of normal flow through the affected *srotas*.

Such textual references collectively indicate that *Ācāryas* were well aware of internal obstructive pathologies and advocated timely *Śāstrakṛta Karma* when *Aushadha* (medical therapy) or *Kṣāra Prayoga* (alkaline therapy) proved insufficient. In the context of *Śākhāśrita Kāmalā*, where *Pittavāha srotas* gets obstructed due to *Kapha āvarana* or *Aśmarī* formation, these principles form the surgical basis for relieving the obstruction, paralleling modern biliary decompression or cholelithotomy procedures.

Thus, the Ayurvedic understanding of *Śāstrakṛta Chikitsā* in *Aśmarī* and *Mārgāvarodha Janya Vyādhi* signifies an advanced awareness of internal obstructions and the necessity of surgical management when conservative measures fail — demonstrating that the roots of hepatobiliary surgical thought are deeply embedded within the classical Ayurvedic framework.

DISCUSSION

Surgical jaundice, characterized by obstruction to the normal flow of bile from the liver to the duodenum, corresponds conceptually to *mārgāvarodha janya vikāra* in Ayurveda. A similar mechanism is described in *Śākhāśrita Kāmalā*, where vitiated *kapha* obstructs *pitta pravṛtti* and aggravated *vāta* displaces it into the *śākhās*, resulting in features akin to obstructive jaundice. Ayurveda thus emphasizes *kapha-hara*, *vāta-anulomana*, and *pittānulomana* measures to restore biliary flow, with *Anuśāstra Karma* indicated when internal *āvarodha* or structural pathology persists.

Among these, *Kṣāra Karma*, through its *lekhana*, *chedana*, and *bhedana* properties, aids in dissolving *āma* and *kapha*, re-establishing *srotas patency*. *Agnikarma*, by its *dāhana* and *saṃśamana* effects, stimulates localized *agni*, mitigates *kapha-medo āvarodha*, and supports *pittavaha srotas* function. *Rakta Mokṣaṇa* eliminates *duṣṭa rakta* and improves hepatic circulation, facilitating detoxification. Collectively, these interventions aim at *mārgavivarana*—removal of obstruction and restoration of flow—conceptually aligning with the therapeutic goal of relieving biliary blockade.

Ācārya Charaka's reference to *Pittāśaya Aśmarī*, likened to *Gorocanā* found in the gallbladder of cattle (Cha. Chi. 26/36), reflects an early understanding of biliary concretions. *Ācārya Suśruta* (Su. Chi. 7/33) further details surgical intervention when *Aśmarī* becomes chronic or causes severe distress, advocating *Śastra Karma* proportional to the obstruction. These classical insights indicate that *Ācāryas* recognized internal obstructive pathologies and prescribed surgical management when conservative measures failed.

Thus, the Ayurvedic principles of *Śāstrakṛta Chikitsā* and *Anuśāstra Karma* reveal an advanced surgical perspective that extends to internal obstructions like *Śākhāśrita Kāmalā*. Their emphasis on clearing *srotorodha*, restoring *pitta pravṛtti*, and rebalancing *doṣas* mirrors the objectives of modern hepatobiliary surgery, underscoring that the roots of such therapeutic reasoning lie deeply embedded within the classical Ayurvedic framework.

CONCLUSION

Thus, a comprehensive evaluation through clinical history, examination, and supportive investigations is essential to identify the underlying cause of biliary obstruction and guide appropriate surgical intervention. Ayurveda, through the understanding of *Śākhāśrita Kāmalā* and *Mārgāvarodhajanya Vikāra*, emphasizes the importance of assessing *Avasthā* and *Samprapti* before selecting suitable *Chikitsā* from the *Ṣaṣṭi Upakrama*. This holistic approach—combining diagnostic precision with stage-wise management—reflects the timeless surgical and therapeutic wisdom of the classical *Ācāryas*, offering valuable insight even in the context of contemporary hepatobiliary disorders.

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Scope

This review correlates *Śākhāśrita Kāmalā* with surgical jaundice, emphasizing *mārgāvarodha* as the core pathology. It integrates Ayurvedic principles of *Samprāpti* and *Chikitsā* with modern diagnostic and surgical insights to promote an evidence-based, interdisciplinary approach.

Limitations

The study is conceptual, based on classical and literary data without clinical validation. Interpretational variations across texts may affect uniformity of understanding.

Future Scope

Clinical validation of *Kṣāra Karma*, *Agnikarma*, and *Rakta Mokṣaṇa* in *mārgāvarodha janya vyādhi* is essential. Integrative studies combining Ayurvedic and modern diagnostics could establish stronger evidence for Ayurvedic hepatobiliary management.

REFERENCES

1. *Oxford English Dictionary. Jaunis – Old French Origin [Internet]. 1300 AD.*
2. *Luhrman Dr. Observation of jaundice as post-vaccination adverse effect. Med Chron. 1885.*
3. *McDonald Dr. Hypothesis on sub-bacterial cause of jaundice. Brit Med J. 1908.*
4. *Whipple AO. The diagnosis and treatment of obstructive jaundice. Ann Surg. 1935;102:763–79.*
5. *Whipple AO. The rationale of pancreaticoduodenectomy. Surg Gynecol Obstet. 1935;41:368–79.*
6. *Sherlock S, Dooley J.* Diseases of the Liver and Biliary System. *12th ed. Oxford: Blackwell Science; 2011.*
7. *Kumar V, Abbas AK, Aster JC.* Robbins and Cotran Pathologic Basis of Disease. *10th ed. Philadelphia: Elsevier; 2021.*
8. *Hall JE, Guyton AC.* Guyton and Hall Textbook of Medical Physiology. *14th ed. Philadelphia: Elsevier; 2021.*
9. *Zakim D, Boyer TD.* Hepatology: A Textbook of Liver Disease. *7th ed. Philadelphia: Elsevier Saunders; 2018.*
10. *Chokshi P, et al. Classification and biochemical approach to jaundice.* Clin Liver Dis. *2019;13(2):35–43.*
11. *Cameron JL, Cameron AM.* Current Surgical Therapy. *13th ed. Philadelphia: Elsevier; 2023.*
12. *Blumgart LH.* Surgery of the Liver, Biliary Tract and Pancreas. *6th ed. Philadelphia: Elsevier; 2017.*
13. *Sharma MP, Ahuja V. Obstructive jaundice – Epidemiology and management. Indian J Gastroenterol. 2018;37(5):389–395.*
14. *Agnivesha, Charaka Samhita, Chikitsa Sthana, Chapter 16,* Kamala Chikitsa Adhyaya. *Revised by Dridhabala, commentary by Chakrapani Datta. Chaukhamba Sanskrit Sansthan, Varanasi; 2018.*
15. *Sushruta,* Sushruta Samhita, *Nidana Sthana, Chapter 13,* Pittaja Roga Nidana. *Chaukhamba Orientalia, Varanasi; 2017.*
16. *Vagbhata,* Ashtanga Hridaya, *Nidana Sthana, Chapter 12,* Kamala Nidana. *Chaukhamba Krishnadas Academy, Varanasi; 2016.*
17. *Murthy KRS,* Illustrated Dictionary of Ayurveda. *Chaukhamba Orientalia, Varanasi; 2014.*