Skin manifestations of systemic disorders give a clue to the organ involved and help identify the possible disease-causing injury. Skin changes of liver cirrhosis are not specific, as they may be seen in disorders not involving the liver. Thus, a constellation of skin changes along with systemic features may help us to identify the disease-causing liver cirrhosis. Pruritus is one of the most common and distressful symptoms of liver cirrhosis, severely affecting the quality of life, which further necessitates understanding cutaneous manifestations of cirrhosis. Other nonspecific cutaneous manifestations include spider telangiectasia, palmar erythema, paper money skin, xanthomas, pigmentation changes, nutritional deficiencies, hair changes, and nail changes. This review discusses the nonspecific skin manifestations associated with liver cirrhosis followed by specific cutaneous findings seen in common diseases causing liver cirrhosis, such as viral infections, biliary tract disorders, chronic alcoholism, and metabolic disorders. Early recognition of cutaneous features can help prevent or delay the development of complications and end-stage disease, decreasing morbidity and mortality. (J CLIN EXP HEPATOL xxxx;xxx:xxx)

Pruritus

Pruritus is the commonest skin manifestation of liver cirrhosis and may appear before the diagnosis of liver disease. The pathophysiology of pruritus in cirrhosis is complex. Biochemicals such as histamine, substance P, bile acids, and endogenous opioids have been hypothesized to mediate itch in cirrhosis. Bile salts, along with other pruritogens, mediate cholestatic pruritus, but not every patient with increased bile salts or acids develop itching, and not every patient with raised levels respond to bile acid chelating agents. Blood levels of the μ-opioid receptor agonists methionine, enkephalin and β-endorphin are found to be raised in patients with cirrhosis. An excellent response has been observed to μ-opioid receptor antagonists naltrexone in patients with obstructive jaundice. These findings strongly suggest an indirect role of endogenous opioids in mediating cholestatic itching, but the severity of pruritus does not correlate with the levels of bile salts or endogenous opioids. Lyosphosphatidic acid (LPA) and autotaxin (an ectoenzyme involved in LPA production) are significantly increased in patients with cholestatic pruritus, suggesting they may be potential therapeutic targets. Typically, oral contraceptive pills intake in females increases autotaxin levels, and patients with bile obstruction commonly show increased pruritus during the progesterone phase of the menstrual cycle, late pregnancy, and hormone replacement therapy. Thus cirrhotic patients with reduced metabolism of androgens may find elevated autotaxin levels. Serum autotaxin levels have been reported to correlate with liver fibrosis. Therefore, they could be used to assess the progression of cirrhosis.

ITCHING IS MOST PROMINENT IN BILIARY OBSTRUCTIVE AND CHOLESTATIC DISEASES. IT IS MOST SEVERE AT ACRAL SITES AND...
Table 1  Diseases Causing Liver Cirrhosis.

- Alcoholic liver disease
- Chronic viral hepatitis (Hepatitis B and C)
- Autoimmune hepatitis (type 1, 2, 3)
- Nonalcoholic steatohepatitis
- Primary biliary cirrhosis (PBC)
- Primary sclerosing cholangitis (PSC)
- Autoimmune cholangiopathy
- Biliary atresia
- Cardiac cirrhosis
- Inherited metabolic diseases
  - Hemochromatosis
  - Wilson’s disease
  - Alpha-1 antitrypsin deficiency
  - Cystic fibrosis
- Inherited disease of sugar metabolism (galactosemia, glycogen storage disease)
- Inherited disease (Alagille syndrome)
- Infection
  - Syphilis
  - Brucellosis
  - Schistosomiasis
- Medications - methotrexate, isoniazid, acetaminophen, alpha methyl dopa
- Cryogenic cirrhosis
- Sarcoidosis
- Venous outflow obstruction - Budd Chiari syndrome
- Hepatotoxins - carbon tetrachloride

Table 2  Skin Lesions in Cirrhosis.

- Pruritus
- Jaundice
- Xerosis
- Excoriations marks
- Spider Nevi
- Palmar erythema
- Dilated abdominal and chest veins
- Periumbilical caput medusa
- Dollar paper skin
- Striae
- Purpura
- Cutaneous hyperpigmentation
- Loss of secondary sexual hair
- Gynaecomastia
- Testicular atrophy
- Dupuytren contracture
- Nail changes: clubbing pallor, Muehrcke’s bands, terry’s nail, watch glass deformity
- Muco-cutaneous features of vitamin deficiency

Associated lesions

- Xanthelasma
- Porphyria cutanea tarda
- Vasculitis
- Pyoderma gangrenosum
- Lichen planus
- Rosacea
- Rhinophyma
- Swelling of the parotid gland

“SKIN CHANGES IN CIRRHOSIS”  BHANDARI & MAHAJAN

Spider Angioma (Nevus Araneus)
A feature characteristically seen in nearly one-third of the patients with cirrhosis are spider angiomas, which have a small central arteriole resembling the body of a spider and tortuous thin radiating vessels resembling its legs. They are mostly seen on skin drained by superior vena cava. The lesion is blanchable and quickly refills when the pressure is released from the central arteriole. Its number correlates with the frequency of esophageal varices. Hyperestrogenemia is considered to be one of the responsible factors for the development of these vascular changes. Other biochemical substances such as vascular endothelial growth factor (VEGF), basic fibroblast growth factor (bFGF), substance P, and endogenous vasodilators have been implicated in the etiopathogenesis of spider telangiectasias. Multiple spider angiomas are typical of chronic liver disease with a specificity of 95% and act as a clinical marker for esophageal varices and hepatopulmonary syndrome. The nevi may disappear postliver transplant in patients with cirrhosis. It may also be noted in patients with rheumatoid arthritis, thyrotoxicosis, pregnancy, and severe malnutrition (Figure 1).

Palmar Erythema
Palmar erythema or liver palms is regarded as blanchable reddish discoloration of the palms, which may also involve the dorsal aspect of hands, fingertips, nail bed, and rarely soles of the feet. It is most predominant on the hypothenar eminences. Almost 23% of people with liver cirrhosis develop palmar erythema. It results from abnormal serum estradiol levels that activate the enzyme nitric oxide

follows a circadian rhythm, with the maximum intensity in the evening and early night. Primary cutaneous lesions are not observed in cholestatic liver cirrhosis, but vigorous scratching causes secondary changes such as erosions, prurigo nodularis, and lichen planus. They are mostly seen on skin drained by superior vena cava. The lesion is blanchable and quickly refills when the pressure is released from the central arteriole. Its number correlates with the frequency of esophageal varices. Hyperestrogenemia is considered to be one of the responsible factors for the development of these vascular changes. Other biochemical substances such as vascular endothelial growth factor (VEGF), basic fibroblast growth factor (bFGF), substance P, and endogenous vasodilators have been implicated in the etiopathogenesis of spider telangiectasias. Multiple spider angiomas are typical of chronic liver disease with a specificity of 95% and act as a clinical marker for esophageal varices and hepatopulmonary syndrome. The nevi may disappear postliver transplant in patients with cirrhosis. It may also be noted in patients with rheumatoid arthritis, thyrotoxicosis, pregnancy, and severe malnutrition (Figure 1).

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synthase to produce nitric oxide and induce vasodilation. It may also be seen in pregnancy, rheumatoid arthritis, diabetes, thyrotoxicosis, brain tumors, and liver diseases such as Wilson’s disease and hereditary hemochromatosis. Drugs like topiramate, albuterol, amiodarone gemfibrozil, and cholestyramine are known to cause palmar erythema. External causes such as smoking, alcoholism, and mercury poisoning also cause palmar erythema (Figure 2).¹⁸

**Xanthelasma**

Dyslipidemia is often a feature of cholestatic liver diseases. It presents as soft, yellow asymptomatic papules, plaques or nodules and may affect any body site. Primary biliary cirrhosis (PBC) is commonly associated with dyslipidemia and manifests as planar, tuberous, and tendinous xanthomas (Figure 3). Xanthoma striatum palmare is another finding reported in patients with PBC with hypercholesterolemia, which presents as multiple yellowish plaques over the creases of palms. Recurrent trauma increases vessels’ permeability, resulting in the seepage of lipids through these damaged vessel walls and deposition of lipids in pressure areas of the palm.

**Jaundice and Pigmentary Alterations**

Jaundice is the yellow pigmentation of skin, sclera, and mucous membranes resulting from the deposition of bilirubin and its metabolites in these tissues. Bilirubin levels...
beyond 2–2.5 mg/dL lead to clinically apparent jaundice. Blotchy or diffuse mud-grey pigmentation over the photo exposed areas may be noted in patients with cirrhosis, accentuated over the areola, palmar creases, perioral and periorbital areas. Melanosis is often seen in PBC and manifests early in the disease. Hemosiderin deposition leads to increased melanin synthesis, causing metallic brown pigmentation of skin in patients of hemochromatosis.19

Hair and Nail Changes
Patients with cirrhosis have thinning and partial loss of hairs. Males develop a female pattern of body hairs, especially pubic hairs, due to elevated estrogen levels. Nail changes in cirrhosis are nonspecific. Almost 15% of patients develop clubbing as a result of dilated digital arteriovenous anastomosis and increased blood flow. Other findings include thickening, longitudinal ridging, white transverse bands parallel to lunula (Muehrke’s bands), watch glass deformity, flattened brittle nails, or koilonychia due to micronutrient deficiency. In advanced cirrhosis, patients develop vascular nail bed changes secondary to overgrowth of connective tissue and present with proximal ground-glass opacification with a distal pink-brown color known as terry’s nails or leukonychia (Figure 4).20 In Wilson’s disease, the lunula turns blue and is named the Azure lunula. Spoon-shaped nails are seen in patients with hemochromatosis-induced cirrhosis. Hypertrophic osteopathy and splinter hemorrhages have been described in patients with cirrhosis.21

Mucosal changes - Patients with cirrhosis have decreased immunity and micronutrient deficiency. As a result, they commonly develop oral infections such as herpes and candidiasis. Angular cheilitis, aphthous ulcers,
atrophy, glossitis, mucosal hyperpigmentation, fissured tongue, and gingival hypertrophy can also be seen. Diuretic administration decreases the saliva causing dryness of the mouth and prone to decaying of the tooth. Those with viral hepatitis and cirrhosis develop leukoplakia, melanoplakia, lichen planus.22

BLEEDING AND CLOTTING DEFECTS
Cutaneous bleeding in the form of purpura and mucosal bleeding such as epistaxis and gingival bleeding may be noted in cirrhosis (Figure 5). Those with portal hypertension develop collaterals appearing as coiled varicose veins over the abdomen. The dilated radiating pattern of veins around the umbilicus is known as caput medusae or palm tree sign (Figure 6). Diffusely scattered tiny telangiectatic vessels resembling the silk threads of dollar bill are referred to as paper money skin.23

OTHER NONSPECIFIC CHANGES
Other vascular findings include corkscrew scleral vessels and bier spots.19 Bier spots are small irregular hypopigmented patches present commonly over arms and legs; they are considered an outcome of venous stasis and disappear on blanching.24

NUTRITIONAL DEFICIENCIES
The parenchyma of the cirrhotic liver is not able to store or metabolize micronutrients. Malabsorption secondary to steatorrhea further leads to decreased uptake of micronutrients. Table 3 enlists the mucocutaneous manifestation of micronutrient deficiency seen in liver cirrhosis.25

DISEASE-SPECIFIC CUTANEOUS MANIFESTATIONS
Viral Hepatitis
Hepatitis B Virus (HBV) can have a nonspecific erythematous rash or a serum sickness-like condition occurring in 20–30% of patients with hepatitis. Circulating immune complexes are responsible for the rash. Approximately 20% of patients suffering from polymyositis nodosa (PAN) have positive serology for hepatitis B, while approximately 8% of patients with acute HBV infection develop PAN that is negative for autoimmune antibodies.26 Antivirals, along with steroids, should be initiated in such cases. Similarly, 15% of patients who are Hepatitis B positive have raised Cryoglobulins levels and are mostly asymptomatic. Cryoglobulinemia may affect the cutaneous (purpura, necrotic ulcers), renal, and peripheral nervous systems. Immunofluorescence of affected skin shows deposits of IgM and C3 in the vessel wall.27 Gianotti Crosti Syndrome (papular acrodermatitis of childhood) is commonly seen with HBV infection, but after the vaccination program for hepatitis B, it is more commonly associated with other viral infections such as EBV. It presents clinically as small umbilicated papules affecting the limbs, buttocks, and cheeks. It is a self-resolving dermatosis with spontaneous resolution in 1–2 months and needs symptomatic treatment.28

Hepatitis C Virus (HCV) - Almost 70% of patients infected with the Hepatitis C virus develop chronic hepatitis, and 15–30% of chronic hepatitis develop liver cirrhosis. Similarly, 70% of patients with type II cryoglobulinemia (polyclonal IgG and monoclonal IgM rheumatoid factor) and a small portion of type III cryoglobulinemia (polyclonal IgG and polyclonal IgM rheumatoid factor) are HCV positive. Clinical presentation includes small vessel vasculitis commonly affecting lower extremities, acrocyanosis, livedo reticularis, glomerulonephritis, arthralgia, hepatosplenomegaly, and hypocomplementemia (Figure 7). HCV RNA has been identified from the involved organs due to cryoglobulinemia, mainly the skin and kidneys. Anti HCV therapy should be started as first-line therapy in such cases.29 Almost 5%–30% of patients with PAN are found positive for HCV infection, and they have reduced complement levels.30 There is a higher incidence of hepatitis C among patients with an erosive variant of oral lichen planus (Figure 8). A positive association of lichen planus has been found with PBC and chronic active hepatitis due to possible immune dysfunction.31,32 Porphyria Cutanea Tarda (PCT) is a blistering disorder affecting the photo-exposed areas. It is caused by the reduced activity of uroporphyrinogen decarboxylase (UROD) enzymes that can be acquired or inherited. Blisters resolve by scar formation, dyspigmentation, milia
formation, and hypertrichosis. HCV positivity may range from 10% to 90%. Sporadic form is the most common variant, and most of them are due to alcohol intake. Therefore, patients with alcoholic liver cirrhosis are more likely to have PCT. Necrolytic acral erythema is another marker of HCV infection, which can also be seen in zinc deficiency. Blister formation along with dusky red erythema may be noted over bilateral acral areas. Dusky reddish margins clearly demarcate the lesions. Chronic lesions develop hyperkeratotic psoriasiform changes. Clinical features mimic that of necrolytic migratory erythema and pseudo glucagonoma (Figure 9). Seropositivity for HCV in the presence of normal serum glucagon levels helps arrive at the diagnosis. Less well-corroborated associations with HCV infection include pyoderma gangrenosum, antiphospholipid syndrome, Behçet disease, vitiligo, amyloidosis, sarcoidosis and Sjögren syndrome. Table 4 describes the common cutaneous findings of viral hepatitis.

ALCOHOLIC LIVER CIRRHOSIS

It is recognized as the most common cause of cirrhosis in India. Patients with chronic alcohol intake develop pseudo-Cushingoid features like truncal obesity, moon facies, and proximal muscle wasting. The clinical features are nonspecific, but paper money skin and Dupuytren’s contracture are considered as strong clinical markers of alcohol abuse (Figure 10).

Facial lipodystrophy is commonly seen due to reduced caloric intake in patients with chronic alcoholism. Alcohol is a known trigger for PCT and even more in patients with concomitant HCV infection. There is a defective synthesis of clotting factors and cutaneous changes such as purpura, and other nonspecific changes like spider angiomas, palmar erythema may be noted in patients with alcoholic cirrhosis. Disseminated superficial periorificial erythema has been reported to be associated with alcoholic cirrhosis, which improves when liver function normalizes. Also, nutritional dermatosis may present in the form of crusted erosions over the periorificial and genital areas, glossitis, cheilitis, hair loss, and beau’s lines over nails.

PRIMARY BILIARY CIRRHOSIS (PBC)

It is an autoimmune disorder commonly affecting middle-aged females. Almost half of the patients with PBC have cutaneous involvement as the first symptom. Features of obstructive jaundice such as severe itching, secondary excretion, hyperpigmentation, and various cutaneous xanthomas due to dyslipidemia may be noted. The interscapular area, which is not accessible, is spared as noted in other cholestatic disorders, known as the butterfly sign. PBC has strong positivity for antimitochondrial antibodies. Other immune disorders associated include lichen planus, vitiligo, Raynaud’s disease, keratoconjunctivitis sicca, morphea, and systemic sclerosis.

OTHER BILIARY TRACT DISEASES

Primary Sclerosing cholangitis (PSC): Almost 66% of patients with PSC develop ulcerative colitis. These patients are more prone to develop cholangiocarcinoma and colorectal carcinoma. The associated cutaneous disorders with PSC are pyoderma gangrenosum and erythema nodosum. Pyoderma gangrenosum presents mainly in the form of a severely painful ulcer with a violaceous border, and erythema nodosum occurs as erythematous painful subcutaneous nodules commonly over legs.

Alagille syndrome is an autosomal dominant disease caused by mutations in the JAG1 (>88%) gene or the NOTCH2 gene (around 1%). It can involve multiple organ systems, and congenital biliary tract hypoplasia is a developmental anomaly in the liver. Features of cholestasis such as severe itching, raised bilirubins, and widespread xanthomas are seen.

METABOLIC DISEASE

Hemochromatosis - It is a disease resulting from increased iron absorption and subsequent deposition in
tissues and organs such as the liver, pancreas, heart, pituitary, and endocrine organs. This deposition leads to cutaneous pigmentation, diabetes, cirrhosis, and heart failure. Skin pigmentation occurs in up to 90% of patients. Greyish or bronze cutaneous pigmentation is often noted in the early course of the disease and is more profound over sun-exposed areas. Iron deposition stimulates melanocytes to produce melanin, which is further enhanced by UV-A rays. Ichthyosiform dryness of the skin, koilonychia, and loss of body hair also occur. Classic idiopathic hemochromatosis commonly affects men and becomes apparent after 40 years of age. The acquired form is seen secondary to hemosiderosis and alcohol abuse. Phlebotomy and iron chelating agents are the treatment modalities that also improve cutaneous pigmentation. Genetic screening of relatives is commonplace. The iron overload in hemochromatosis is another cause of acquired porphyria. Hence, PCT is an important clinical manifestation leading to the diagnosis of hemochromatosis.39

Wilson’s disease is an autosomal recessive disorder of copper metabolism. Progressive copper deposition in tissue such as the liver, brain, and eyes occurs. Azure lunulae (bluish nail lunulae) is one of the characteristic manifestations of the disease. Pretibial hyperpigmentation and Kayser-Fleischer rings in the corneal membrane can also be seen.40

Other metabolic causes of cirrhosis: The majority of patients with sarcoidosis involving the liver are asymptomatic. Almost 6% of these may develop cirrhosis secondary to sarcoidosis. Liver function derangement along with cutaneous lesions of sarcoidosis may be seen.41 Similarly, patients with porphyria cutanea tarda and erythropoietic protoporphyria may also involve the liver and develop chronic parenchymal liver disease and cirrhosis.42

Table 3 Mucocutaneous Manifestation of Micronutrient Deficiency Seen in Cirrhosis.

<table>
<thead>
<tr>
<th>Vitamin deficiency</th>
<th>Mucocutaneous manifestation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vitamin A</td>
<td>Xerosis</td>
</tr>
<tr>
<td></td>
<td>Deep skin fissures (dermomalacia)</td>
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<tr>
<td></td>
<td>Follicular hyperkeratosis (phrynoderma)</td>
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<tr>
<td></td>
<td>Xerophthalmia</td>
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<tr>
<td>Vitamin D</td>
<td>Alopecia</td>
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<tr>
<td>Vitamin E</td>
<td>Follicular hyperkeratosis</td>
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<tr>
<td>Vitamin K</td>
<td>Purpura</td>
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<tr>
<td></td>
<td>Ecchymosis</td>
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<tr>
<td></td>
<td>Gingival bleeding</td>
</tr>
<tr>
<td>Vitamin C</td>
<td>Follicular hyperkeratosis</td>
</tr>
<tr>
<td></td>
<td>Perifollicular hemorrhages</td>
</tr>
<tr>
<td></td>
<td>Stomatitis, epistaxis, bleeding gums (scurvy)</td>
</tr>
<tr>
<td>Vitamin B2 (riboflavin)</td>
<td>Seborrheic dermatitis</td>
</tr>
<tr>
<td></td>
<td>Stomatitis, gingivitis</td>
</tr>
<tr>
<td></td>
<td>Conjunctivitis, corneal vascularization</td>
</tr>
<tr>
<td>Vitamin B6 (pyridoxine)</td>
<td>Seborrheic dermatitis</td>
</tr>
<tr>
<td></td>
<td>Photodermatitis</td>
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<tr>
<td></td>
<td>Glossitis</td>
</tr>
<tr>
<td>Vitamin B12 (cyanocobalamin)</td>
<td>Hyperpigmentation of flexures, knuckles, palms, and fingers</td>
</tr>
<tr>
<td></td>
<td>Pigmented streaks over nails</td>
</tr>
<tr>
<td>Vitamin B3 (niacin)</td>
<td>Pellagra (dermatitis, dementia, and diarrhea)</td>
</tr>
<tr>
<td>Biotin</td>
<td>Alopecia</td>
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<tr>
<td></td>
<td>Eczema around nose and mouth</td>
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<tr>
<td></td>
<td>Conjunctivitis</td>
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<tr>
<td>Folic acid</td>
<td>Gray brown pigmentation on sun-exposed areas</td>
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<tr>
<td></td>
<td>Cheilitis</td>
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<tr>
<td></td>
<td>Glossitis</td>
</tr>
<tr>
<td></td>
<td>Mucosal erosions</td>
</tr>
</tbody>
</table>

Figure 7 Net-like pattern of livedo reticularis in HCV.

Figure 8 Oral lichen planus in HCV-induced cirrhosis.
SKIN CHANGES IN LIVER TRANSPLANT

Patients with liver transplants have compromised immunity, and the use of immunosuppressants further adds to it. They are prone to develop cutaneous infections and malignancies. Viral infections such as warts and herpes reactivation are commonly observed. Cytomegalovirus may cause vesicular eruptions, ulceration, and hemorrhagic crust formation over oral and genital mucosa. Fungal infections such as candida affect almost 20% of patients with liver transplants. Deep fungal infections such as aspergillosis, cryptococcosis, phaeohyphomycosis may develop over the skin. Bacterial infections due to Streptococcus and staphylococcus are commonly noted. Cutaneous manifestations of graft versus host disease may appear in varied presentations. Hypertrichosis, skin atrophy, xerosis, purpura, and acne are other cutaneous lesions. Porokeratosis may occur after 4–5 years of transplant. Cutaneous malignancies such as basal cell carcinoma, squamous cell carcinoma (over vulva and perineum), Kaposi’s sarcoma, and other lymphoproliferative disorders usually develop. Cutaneous lesions such as

Table 4  Cutaneous Manifestations of Viral Hepatitis.

<table>
<thead>
<tr>
<th>Common for Hepatitis B &gt; C</th>
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<tbody>
<tr>
<td>Urticaria</td>
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<tr>
<td>Serum sickness</td>
<td></td>
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<tr>
<td>Erythema Nodosum</td>
<td></td>
</tr>
<tr>
<td>Gianotti crosti (papular acrodermatitis of childhood)</td>
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<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>Common to both Hepatitis B and C</td>
<td></td>
</tr>
<tr>
<td>Small vessel vasculitis</td>
<td></td>
</tr>
<tr>
<td>Urticarial vasculitis</td>
<td></td>
</tr>
<tr>
<td>Erythema multiforme</td>
<td></td>
</tr>
<tr>
<td>Pruritus</td>
<td></td>
</tr>
</tbody>
</table>

Common for Hepatitis C > B

- Leukocytoclastic vasculitis
- Cryoglobulinemia
- Porphyria cutanea tarda
- Polymyositis nodosa

Hepatitis C

- Lichen planus
- Livedo reticularis
- Necrotic cutaneous erythema
- Red Finger Syndrome
- Autoimmune thrombocytopenic purpura,
- Behet’s disease
- vitiligo
- Sjogren’s syndrome

Figure 9 Necrolytic acral erythema in HCV.

Figure 10 Dermoscopic image of paper money skin in alcoholic cirrhosis.
palmar erythema, spider nevi, and pruritus improve post-liver transplant.13,14

Skin changes can aid in the early diagnosis of liver cirrhosis, and many of them can help us to suspect the etiological cause. Through the window of mucocutaneous manifestations, early detection of cirrhosis can help prevent or delay the development of complications and end-stage disease, improving the survival and quality of life.

CREDIT AUTHORSHIP CONTRIBUTION STATEMENT

Adhyatm Bhandari: Review of literature, Data curation, Writing – original draft. Rahul Mahajan: Conceptualization, Visualization, Supervision, Validation, Rewriting, Reviewing and Editing.

CONFLICTS OF INTEREST

The authors have none to declare.

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