Integrated Traditional Chinese and Western Therapy for Common and Intractable Disease

常见疑难病症的中西医治疗

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内 容 简 介

本书总结了作者多年的临床经验，分20个部分，阐述了肝炎、腹泻、气喘等常见疑难病症的中西医治疗经验，有较强的实用性，英文译文精炼、准确、流畅。

本书可供各级临床中西医工作者、广大中西医院校学生及外国留学生使用。

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PREFACE

Two kinds of different medical managements are introduced to treat some diseases in this book, western medicine and traditional Chinese medicine. There are twenty parts in it. Each part is a treatise in which clinical description, pathologic information, pathophysiologic knowledge, diagnostic criteria and therapeutic measures both in western medicine and traditional Chinese medicine are well integrated, so that students and physicians consulting this book can secure the most useful information and find it in one place. And it also ushered critical evaluation of drugs, remedies, and nostrums in clinical medicine.

Authors
Xi'an

CONTENTS

PART ONE

VIRAL HEPATITIS

Hepatitis is applied to a broad category of clinicopathologic conditions that result from the damage produced by a viral, toxic, pharmacologic, or immunemediated attack on the liver. The common pathologic features of hepatitis are hepatocellular necrosis, which may be focal or extensive, and inflammatory cell infiltration of the liver, which may predominate in the portal areas or may extend into the parenchyma. Physical examination may show an enlarged tender liver and icteric mucous membranes. Laboratory evidence of hepatocellular damage is invariably found in the form of elevated transaminase levels. Independent of the cause of hepatitis, the clinical course may range from subclinical or mild to severe hepatocellular dysfunction with evidence of impairment of coagulation, marked jaundice, and disturbance of neurologic function.

Acute hepatitis implies a condition lasting less than 6 months, culminating either in complete resolution of the liver damage with return to normal liver function and structure or a rapid progression of the acute injury toward extensive necrosis and a fatal outcome(Table 1).

TABLE 1 Causes of Acute Hepatitis

Viral Hepatitis
Hepatitis A virus
Hepatitis B virus
Hepatitis C virus
Hepatitis D virus("delta agent")
Hepatitis E virus
Epstein-Barr virus
Cytomegalovirus
Alcohol
Toxins
Amanita phalloides mushroom poisoning
Carbon tetrachloride
Drugs
Acetaminophen
Isoniazid
Halothane
Chlorpromazine
Erythromycin
Other
Wilson's disease
Herbs

Chronic hepatitis is defined as a sustained inflammatory process in the liver lasting longer than 6 months and is often impossible to differentiate from acute hepatitis on histologic criteria alone. Inflammatory cell extending beyond the limits of the portal tracts surrounding isolated nests of hepatocytes (piecemeal necrosis) and portal and/or central areas of the hepatic lobules connected by inflammation, necrosis, and col-

lapse of architecture (bridging necrosis) are seen in severe forms of chronic hepatitis. However, these features may also be noted in uncomplicated acute hepatitis that ultimately resolves completely. A purely histologic diagnosis of chronic hepatitis usually requires evidence of progression toward cirrhosis, such as significant fibrous deposition and disruption of the hepatic lobular architecture.

Etiology

Viral hepatitis is caused by at least seven viruses. Hepatitis viruses A(HAV), B(HBV), C(HVC), D(HDV), E(HEV), and G(HGV) have been characterized at the molecular level. Hepatitis F virus is another potential infectious agent that has been experimentally transmitted from human stool to primates and is currently being characterized. Cytomegalovirus and Epstein-Barr virus occasionally cause hepatitis. HDV, and incomplete RNA virus, causes hepatitis only in patients with either acute (HDV coinfection) or chronic hepatitis B(HDV superinfection). HCV accounts for most cases of hepatitis previously designated "non-A, non-B". HBV has been extensively characterized. The complete HBV(Done particle) consists of several antigenically distinct components, including a surface coat[hepatitis B surface antigen (HBsAg)] and a core of circular DNA, DNA polymerase, hepatitis B core antigen (HBcAg), and hepatitis B e antigen (HBeAg). HBsAg may exist in serum either as part of the Dane particle or as free particles and rods. HBsAg, HBcAg, and HBeAg elicit distinct antibody responses from the host that are used to diagnose and characterize the state of virus

replication in the liver.

Transmission

HAV is excreted in feces during the incubation period and is transmitted by the fecal-oral route. It is thus implicated in most instances of waterborne and food-transmitted infection and in epidemics of viral hepatitis.

HBV is present in virtually all body fluids and excreta of carriers and is transmitted mainly by parenteral routes. Thus, transmission occurs most commonly by blood and blood products, contaminated needles, and sexual contact. High-risk transmission groups include the following: sexual partners of acutely as well as chronically infected persons, with male homosexuals being at particularly high risk; health professionals, particularly surgeons, dentists, and workers in clinical laboratories and dialysis units; intravenous drug abusers; and infants of infected mothers ("vertical transmission"). Patients with increased exposure to blood or blood products and/or with impaired immunity (e.g., patients undergoing dailysis, patients with leukemia or Down's syndrome) are also highly susceptible to HBV infection.

HCV, similar to HBV, is largely parenterally transmitted. HCV is the main cause of post-transfusion hepatitis, it is a common cause of hepatitis in intravenous drug users, and it accounts for at least 50% of cases of sporadic, community-acquired hepatitis. In these cases, the mode of virus transmission is unclear. HEV is the cause of an epidemic, waterborne hep-

atitis that has been associated with outbreaks, mainly in Asia and Africa.

Clinical and Laboratory Manifestations

Acute viral hepatits typically begins with a prodromal phase lasting several days and characterized by constitutional and gastrointestinal symptoms including malaise, fatigue, anorexia, nausea, vomiting, myalgia, and headache. A mild fever may be present. Symptoms suggestive of "flu" may be prominent; arthritis and urticaria, attributed to immune complex depostiton, may be present, particularly in hepatitis B. Smokers often describe and aversion to cigarettes. Jaundice soon appears with bilirubinuria and acholic (pale) stools, often accompanied by an improvement in the patient's sense of well-being. Jaundice may be absent (anicteric hepatitis), and in such cases medical attention is often not sought. The liver is usually tender and enlarged; splenomegaly is found in about one fifth of patients.

Transaminases (alanine transaminase and aspartate transaminase) are released from the acutely damaged hepatocytes, and serum transaminase levels rise, often to levels >20fold normal. An elevated serum bilirubin (>42.8 to 51.3 μmol/L) results in jaundice and defines icteric hepatitis. Values higher than 342 μmol/L are uncommon and approximately correlate with the severity of disease. Elevations in serum alkaline phosphatase are usually limited to three times normal levels, except in cases of cholestatic hepatits. A complete blood cell count most com-

monly shows mild leukopenia with atypical lymphocytes. The icteric phase of acute viral hepatitis may last days to weeks, followed by gradual resolution of symptoms and laboratory values.

Serodiagnosis

The ability to detect the presence of viral components in hepatitis B and C and antibodies to components of hepatitis A, B, C, and D has fostered progress in the epidemiology of viral hepatitis. These viral markers can be diagnostic of the cause of acute viral hepatitis. An etiologic diagnosis is of great importance in planning preventive and public health measures pertinent to the close contacts of infected patients and in evaluating prognosis. Epstein-Barr virus and cytomegalovirus hepatitis may also be diagnosed by the appearance of specific antibodies of the IgM class. In acute hepatitis B, HBsAg and HBeAg are present in serum. Both are usually cleared within 3 months, but HBsAg may persist in some patients with uncomplicated cases for 6 months to 1 year. Clearance of HBsAg is followed after a variable "window" period by emergence of anti-HBs, which confers long-term immunity. Anti-HBc and anti-HBe appear in the acute phase of the illness, but neither provides immunity. Uncommonly, during the serologic window period, anti-HBc may be the only evidence of hepatitis B infection, and IgM anti-HBc, a marker of active viral replication, suggests recent infection. HDV infection superimposed on HBV infection may be detected by specific antibody to this agent.

Acute hepatitis C can be detected using a sensitive polymerase chain reaction assay for HCV. RNA. Serum antibodies to HVC develop within 15 weeks of exposure or within 6 to 7 weeks after biochemical abnormalities are discovered.

Complications

Cholestatic Hepatitis In some patients, most commonly during HAV infection, a self-limited period of cholestatic jaundice may supervene that is characterized by marked conjugated hyperbilirubinemia, elevation of alkaline phosphatase, and pruritus. Investigation may be required to differentiate this condition from mechanical obstruction of the biliary tree.

Fulminant Hepatitis Massive hepatic necrosis occurs in $<1\%$ of patients with acute viral hepatitis and leads to a devastating and often fatal condition called fulminant hepatic failure.

Chronic Hepatitis Hepatitis A does not progress to chronic liver disease, although occasionally it has a relapsing course. Persistence of transaminase elevation beyond 6 months in patients with hepatitis B and C suggests evolution to chronic hepatitis, although slowly resolving acute hepatitis may occasionally lead to abnormal liver funcation tests for up to 12 months, with eventual complete resolution. HBV infection without evidence of any liver damage may persist, resulting in asymptomatic or "healthy" hepatitis B carriers. In Asia and Africa, many such carriers appear to have acquired the virus from infected mothers during infancy.

Rare Complication Acute viral hepatitis may be followed by aplastic anemia, which affects mostly male patients and results in a mortality of greater than 80%. Pancreatitis, myocarditis, and neurologic complications including Guillain-Barré syndrome, aseptic meningitis, and encephalitis have also been reported. Cryoglobulinemia, glomerulonephritis, and polyarteritis nodosa are associated with hepatitis B.

Treatment

No specific treatment exists for acute viral hepatitis. Management is largely supportive and includes rest, maintenance of hydration, and adequate dietary intake. Most patients show a preference for a low-fat, highcarbohydrate diet. Vitamin supplementation is of no proven value, although vitamin K may be indicated if prolonged cholestasis occurs. Activity is restricted to limit fatigue. Alcohol should be avoided until liver enzymes return to normal. Measures to combat nausea can include small doses of metoclopramide and hydroxyzine. Hospitalization is indicated in patients with severe nausea and vomiting or in those with evidence of deteriorating liver function, such as hepatic encephalopathy or prolongation of the prothrombin time. In general, hepatitis A may be regarded as noninfectious after 2 to 3 weeks, whereas hepatitis B is potentially infectious to sexual contacts throughout its course, although the risk is low once HBsAg has cleared. Although hepatitis C may also be transmitted to sexual contacts, the risk of this is considered less than for hepatitis B.

Diagnosis in Traditional Chinese Medicine

In traditional Chinese medicine, this disease belongs to the categories of "*huang dan*" (jaundice), "*gan yu*" (stagnation of liver-*qi*), "*xie tong*" (hypochondriac pain) and "*zheng ji*" (mass in the abdomen).

1. Epidemiologic information: The epidemic condition and a history of close contact with a hepatitis patient, or a history of blood transfusion or receiving blood preparations or immunization injections should be noticed.

2. Clinical features:

(1) The onset of the disease is insidious and slow. The patients often complain of fatigue and anorexia. Some have jaundice, but most of them belong to the types of non-icteric or mild hepatitis. Only ten percent of the patients have typical manifestations or jaundice.

(2) Patients with hepatitis A often manifest pyrexia, shorter course and rapid recovery; patients with hepatitis B usually have a chronic course and remain HB virus carriers for a long time. A few of them may progress to cirrhosis. The severity of the clinical features of Non-A Non-B hepatitis is between those of hepatitis A and hepatitis B. And its incubation period can be long or short.

3. Physical signs: The liver becomes enlarged, and tender on palpation and is painful on percussion. There is a mild change of liver texture. A small percentage of cases have splenomegaly. In the patients with icterohepatitis, jaundice may be found in the skin and sclera. Hepatic face, vascular

spiders and liver palms may be present in chronic active hepatitis. A few patients suffering from fulminant heptitis may have skin petechiae, epistaxis and ascites, or even hepatic coma, indicating poor prognosis.

4. Laboratory examination:

(1) Liver function: In patients with acute hepatitis, the SGPT is marked elevated up to several hundreds units, even more than one thousand units. In icterohepatitis, the icterus index and the one-minute bilirubin fixed quantity are increased. In severe and chronic active hepatitis, metabolism of protein is disturbed resulting in the change of ratio of serum albumin to globulin. The albumin level lowered but the globulin level elevated, even the ratio may be inverted. Signs of clotting disorder may be present.

(2) The detection of specific antigens and antibodies: It is available to detect the HAA in filtrate of stools and the anti-HAV of the IgG and IgM class in the serum in the diagnosis of hepatitis A. Three antigen and antibody systems, that is HBsAg, HBcAg and HBeAg with their antibodies, can be detected, which is valuable in the diagnosis and in predicting the severity, infectivity and prognosis of the hepatitis B. Non-A Non-B hepatitis can only be diagnosed by using the exclusive method.

(3)In chronic active hepatitis, tests for cellular immunity, humoral immunity and autoimmunity may be performed to evaluate the host immune mechanisms and severity of the disease so as to give a relevant treatment. Liver biopsy is only in-

dicated for those whose cases defy diagnosis through clinical and laboratory examination.

Differntiation and Treatment of Common Syndromes in Traditional Chinese Medicine

1. Icterohepatitis:

(1) *Yang* jaundice (Actue Icterohepatitis):

Clinical manifestations: Bright yellow coloration of the skin and sclera, fever, thirst, feeling of fullness and distension in the epigastrium, anorexia, fatigue, hypochondriac distension and pain, restlessness, nausea, scanty dark urine, dry stools, red tongue with yellow and greasy fur, taut and rapid pulse.

Therapeutic method: Removing pathogenic heat and dampness.

Recipe: Oriental Wormwood Decoction with additional ingredients.

Ingredients:	Herba Artemisiae Capillaris	30g
	Fructus Gardeniae	10g
	Radix et Rhizoma Rhei	6g (decocted later)
	Cortex Phellodendri	10g
	Flos Lonicerae	30g
	Fructus Forsythiae	15g
	Radix Isatidis	30g
	Rhizoma Imperatae	30g

Administration: All the above drugs are to be decocted in water to get 200～300ml of decoction. Take equal shares in the morning and in the evening.

Modification: In case of exhibiting more symptoms and signs of pathogenic heat, add

Folium Isatidis	30g
Herba Taraxaci	30g

In case of exhibiting more symptoms and signs of pathogenic damp, add

Rhizoma Atractylodis	10g
Cortex Magnoliae Officinalis	10g
Rhizoma Alismatis	10g

In case of nausea and vomiting, add

Caulis Bambusae in Taeniam	10g
Rhizoma Pinelliae	10g

In case of abdominal distension and anorexia, add

Fructus Crataegi	10g
Fructus Hordei Germinatus	10g
Massa Fermentata Medicinalis	10g

In case of cutaneous pruritus, add

Cortex Dictamni Radicis	15g
Fructus Kochiae	15g

(2) *Yin* Jaundice (Chronic icterohepatitis):

Clinical manifestations: Dark yellow coloration of the skin and sclera just like smoky colour, poor appetite, feeling of distension in the abdomen, loose stools, general debility tastelessness in the mouth, whitish thick and greasy fur of the tongue, deep thready and weak pulse.

Therapeutic method: Activating the function of the spleen, inducing diuresis and warming *yang*.