Cirrhosis

This article discusses various aspects of cirrhosis, focusing on it's relationship to hepatitis C. This piece originally appeared in the HCV Advocate.

October 19, 2015 By Alan Franciscus

This column is about cirrhosis—the causes, how it develops, the symptoms and consequences and issues about HCV treatment related to cirrhosis.

1. What are the Causes of Cirrhosis?

Cirrhosis is caused by many substances (alcohol), viruses (hepatitis B, C and D), and even by metabolic disorders (diabetes). Currently, the most common reason for liver transplantation in the United States is from complications from the hepatitis C virus. Cirrhosis caused by hepatitis C is responsible for more than 19,000 deaths every year. Prior to the emergence of hepatitis C, the most common cause of cirrhosis was alcohol consumption. Fatty liver is also a common cause of cirrhosis, and it is expected to surpass the hepatitis C virus as the most common cause of cirrhosis and liver transplantation in the next two decades.

2. How Does Cirrhosis Develop?

In the case of hepatitis C, the development of cirrhosis is a complex process of liver cells becoming damaged or destroyed by the hepatitis C virus. Furthermore, the body's immune system seeks out and identifies the hepatitis C virus (and the destroyed liver cell) as a foreign pathogen—attacks it and kills it. As a result, scar tissue develops. Usually, the liver can repair itself, but as the hepatitis C virus causes more and more damage, it overwhelms the body and the damage builds and builds. As more scar tissue develops the damaged cells start to connect, and fibrosis develops.

Over time, the scar tissue can be so extensive that it can interfere with the functioning of the liver. This is called cirrhosis. Cirrhosis is classified into two types: compensated and decompensated. Compensated means that the liver is extensively scarred but can still perform most of its functions; decompensated means that the liver is extensively scarred and unable to perform many of the functions that keep the body healthy.

3. What are the Tests to Identify Cirrhosis?

There are many types of tests to find out if someone has cirrhosis. In the past, the most common test was a liver biopsy. The procedure requires a medical person to insert a needle through the skin to extract a piece of liver tissue and examine it under a microscope.The liver biopsy is still being used, but it is also being replaced by other procedures such as a Fibroscan (an imaging

test), Fibrometer (combination of blood tests), and other blood tests to gauge the degree of liver damage.

There are many models used to grade and stage the degree of liver damage. The most common is the Metavir. The Metavir has an inflammation and fibrosis scoring stage—in this article I am just listing the fibrosis stages:

- Stage F0 = no fibrosisStage F1 = mild fibrosis
- Stage 2 = moderate fibrosis
- Stage 3 = bridging fibrosis
- Stage 4 = cirrhosis

Note: This is important to know because many insurance companies are using this system to approve or deny insurance for HCV treatment claims.

4. What are the Symptoms and Consequences of Cirrhosis?
In the early stages of extensive scarring—called compensated cirrhosis—the symptoms may be similar to hepatitis C—fatigue, loss of appetite, muscle and joint pain, flu-like symptoms, nausea, indigestion, headaches and many other symptoms. As cirrhosis develops and reaches the later stages—called decompensated cirrhosis—the symptoms become more pronounced and can become life-threatening.In addition to the symptoms described above I have listed some of the more common serious conditions below:

- Portal Hypertension: blood cannot flow through the liver because of the extensive scarring.
- Encephalopathy: the liver is not able to remove toxins such as ammonia, and the result is that these toxins invade the brain. Symptoms include personality changes, and changes in sleep patterns (sleep reversal—awake all night, sleep all day).
- Ascites: accumulation of fluids in the abdominal cavity.
- Edema: accumulation of fluid in the extremities— usually in the feet and legs.
- Coagulopathy: the liver is not able to produce clotting factors that stop the blood from bleeding.
- Male and Female Hormone Regulation: the liver may not be able to regulate female and male hormones.
- Severe Itching: the impairment of bile flow that can cause severe and at times debilitating itching.
- Wasting Syndrome: the liver is not able to process nutrients so people can have severe muscle wasting and weight loss.

Most of these conditions can be managed effectively with lifestyle changes, medications and medical procedures—at least in the short term. The most important step is to be medically monitored and managed on a regular basis. At this point, a person should be evaluated for a liver transplant. The problem is that there are only an estimated 6,000 available livers for the estimated 15,000 livers needed every year for transplantation in the U.S.

5. HCV Treatment

Hepatitis C treatment can now cure most people, the treatment duration is shorter, and treatment side effects are lower than ever. However, once people develop cirrhosis, it becomes more difficult especially for those who are infected with genotype 3 and who have cirrhosis— the second most prevalent genotype in the United States. Unfortunately, we also know that many insurance companies are denying coverage of hepatitis C medications to only those who are in the early stages of HCV infections (F0, F1, F2). Many insurance companies are only covering F3 and F4 unless there are other severe complications. Here's the problem—if you wait until stage F3 or F4 and are cured you will have to be medically followed for the rest of your life since there is a possibility that you could still have liver disease progression. **However,** if you are treated early (F0, F1, F2), and cured you are free of future complications. Does this scenario make any sense to you? It does not make any sense to me either!

This article originally appeared in the mid-month September HCV Advocate; permission to reprint given by Alan Franciscus.
