Clinical Aspects of Liver Diseases

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► Aretaeus (2nd century AD) coined the term "skirros", because he thought that inflammation of the liver led to its **hardening (= skirros)**. • In 1543 A. Vesal described the granulation of the liver surface as being responsible for the compression of the small hepatic vessels. Even at that time, he associated these changes, which were thought to accompany a shrinking of the liver, with alcohol consumption. • When J. Posthius (1590) described ascites, he said that the changed liver was "all granulated inside". A drawing by J. Brown (1685) shows coarse nodular liver cirrhosis. G. B. Morgagni (1761) also wrote a treatise on cirrhosis, in which he described the small vessels as being compressed due to a shrinking and hardened liver. M. Baillie (1818) wrote an excellent description of the morphology of liver cirrhosis and, like A. Vesal, he also postulated a causal connection with excessive alcohol consumption. • The first accurate report on atrophic, portal cirrhosis was given by R. T. H. Laennec (1819) as an incidental inclusion in his book «Traité d'auscultation». *Because of the* **yellow colour** *of the liver* **(= kirros)**, *he coined the term* **cirrhosis.** • The first microscopic examinations were carried out by F. Kiernan (1833), R. Carswell (1838) and E. Hallmann (1839). F. Th. Frerichs differentiated between two stages of the cirrhotic course: the stage of inflammation and the stage of shrinking with formation of nodes. In 1911 F. B. Mallory defined cirrhosis as a "chronic, destructive, progressive process" with regeneration, accompanied by scarring and shrinking of the connective tissue. A. Ghon (1928) recognized the transformational processes in the liver as being an essential feature of cirrhosis. • In 1930 R. Rössle provided a morphological definition of cirrhosis by stating **three criteria:** (*1.*) destruction of liver parenchyma, (*2.*) connective tissue proliferation, and (*3.*) nodular compensatory hyperplasia together with regeneration of liver parenchyma. In the following years, a **fourth criterion** was added: (*4.*) disturbance of the intrahepatic vascular system with consecutive formation of arteriovenous and portovenous anastomoses (H. Thaler, 1952, 1957, 1968; H. Popper et al., 1958; P. P. Anthony et al., 1977; A. M. Rappaport, 1980).

1 Definition

Cirrhosis is a gradually developing, chronic disease of the liver which always involves the organ as a whole. It is the irreversible consequence and final stage of various chronic liver diseases of different aetiology or the result of long-term exposure to various noxae. • The extent of the morphological changes depends on the cause and stage of cirrhosis. Accordingly, there is a wide spectrum of morphological findings and clinical symptoms. The variations of this disease range from symptom-free conditions, non-characteristic complaints and different laboratory findings through to life-threatening complications. Since, in most cases, no clear dividing line can be drawn between cirrhosis and the preceding liver disease, it is very difficult to determine the point where the cirrhotic stage begins; as a rule, the transition is fluent.

► Localized transformation processes such as those observed in **scarred liver** (s. p. 405) are not considered to be cirrhosis. The loss of parenchyma in scarred liver is generally the result of reduced blood supply in the respective area. Deep-set scars create the picture of a **funnel-shaped liver** (s. p. 406). Similarly, pronounced **liver fibrosis** (s. p. 405) does not fulfil the criteria of cirrhosis, since the lobular architecture as well as the intrahepatic and intra-acinar vascular supply are uncompromised. While fibrosis constitutes a precirrhotic stage, it does not necessarily progress to cirrhosis itself. Fibrosis can regress! • *Thus,* **liver cirrhosis** *is characterized by the following* **five criteria:**

1. Pronounced, insufficiently repaired necroses of the parenchyma (with or without inflammatory processes)
2. Diffuse connective tissue proliferation
3. Varying degrees of nodular parenchymal regeneration
4. *Loss and transformation of the lobular structure within the liver as a whole*
5. *Impaired intrahepatic and intra-acinar vascular supply*

2 Classification

Definitive classification of cirrhosis is difficult. It can be categorized according to its (*1.*) aetiology, (*2.*) morphology, (*3.*) pathogenetic development, and (*4.*) clinical features.

2.1 Aetiological classification

Classification of cirrhosis according to its **aetiology** would be desirable, as this approach may help determine prophylactic and therapeutic measures as well as prognosis. If all diagnostic options are employed and the patient cooperates optimally, an aetiological identification of cirrhosis is possible in almost all cases today. • Due to improved detailed diagnostics, the group of so-called *cryptogenic cirrhoses* has been consistently reduced (< 10% of cases). (s. tab. 35.2)

However, classification of cirrhosis based on aetiology is limited for several reasons: (*1.*) the cause of cirrhosis cannot be determined in many cases, (*2.*) a certain cause gives rise to different morphological forms in individual patients, (*3.*) there may be several causes for the same form of cirrhosis, and (*4.*) various causes may frequently coincide in diverse combinations.