Heart Disease and the Liver

Pathologic Evaluation

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KEYWORDS

• Liver • Congestive hepatopathy • Cardiac cirrhosis • Fontan
• Acute cardiogenic liver injury

KEY POINTS

- Congestive hepatopathy is progressive liver dysfunction resulting from chronic heart failure.
- MELD-XI scores and liver histology may be used in conjunction to risk-stratify patients for cardiac transplantation.
- After surgical repair of congenital cardiac abnormalities, sequelae of congestive hepatopathy include an increased risk for hepatocellular carcinoma.
- Cardiac medications, including amiodarone and calcium-channel blockers, have been implicated in progressive liver dysfunction.

HISTORICAL PERSPECTIVE

The structural and functional changes that develop in the liver in patients with cardiac disease have intrigued clinicians and pathologists for at least 2 centuries. The microscopic description of the "nutmeg," congested liver in heart failure (**Fig. 1**) is attributed to Kiernan in 1833.[1] Once Kiernan's "lobule" had become firmly entrenched in the conceptual microanatomy of the liver, by the early twentieth century more complete morphologic studies of centrilobular congestion began to emerge,[2] and in 1901, Mallory[3] (of Mallory-Denk body fame) described the features of centrilobular necrosis in autopsy specimens (see **Fig. 1**C). By the 1950s, during the formative years of modern hepatology, the liver in cardiac disease was addressed in publications by 2 of the giants in the field, Dame Professor Sheila Sherlock (The Royal Free Hospital, London, UK) and Professor Hans Popper (The Mount Sinai Medical Center, New York City) (**Fig. 2**),.[4,5] Wallach and Popper[5] examined centrilobular necrosis in individuals with cardiac disease, whereas Sherlock's seminal paper was a correlative study of

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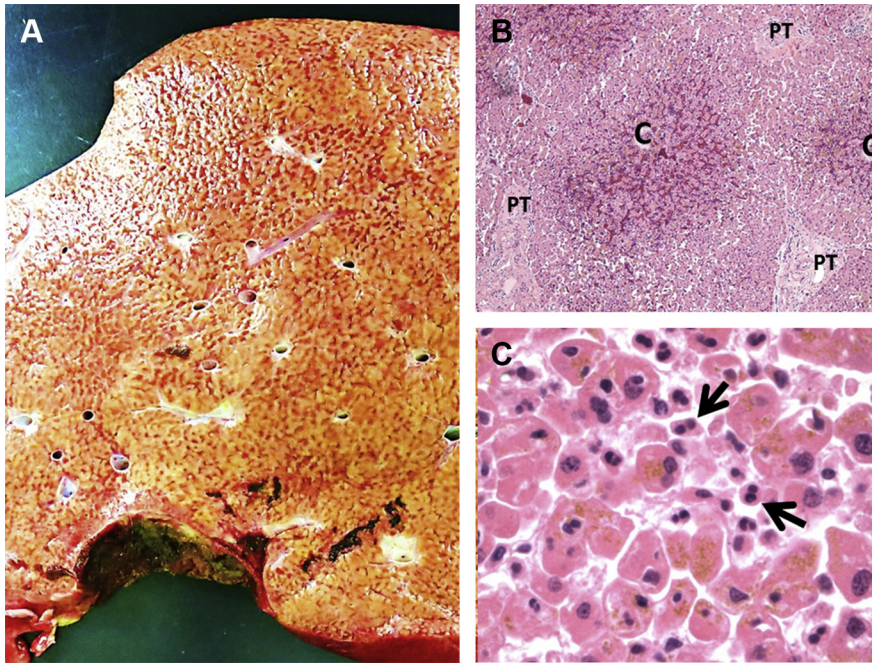

Fig. 1. (*A*) Postmortem example of the classical "nutmeg" liver with centrilobular congestion in CH. (*B*) Centrilobular regions (C) show congestion. The liver parenchyma around the portal tracts (PT) is spared (hematoxylin-eosin, original magnification ×25). (*C*) Many cases of CH at postmortem show evidence of associated acute left ventricular dysfunction and centrilobular coagulative necrosis, as seen here. If the patient was sustained for several days with pressor agents, there may also be a neutrophil infiltrate (*arrows*) near the hepatocytes with coagulative necrosis (hematoxylin-eosin, original magnification ×400).

serum liver tests, cardiac catheterization results, and hepatic abnormality in a cohort of individuals with cardiac disease of multifactorial cause. Sherlock's clinical cases reflected the general state of medicine in that period: cases of rheumatic heart disease were abundant, and there was a comparatively high number of cases of constrictive pericarditis. Hypertensive heart disease and atherosclerotic cardiovascular disease were, as today, also represented in the heart failure study subjects. The hepatic lesions shown in photomicrographs in Professor Sherlock's study remain, even now, some 65 years after publication, representative of the histologic spectrum of "congestive hepatopathy," (**Box 1**) including centrilobular congestion and sinusoidal dilatation, atrophy of liver-cell plates and fibrosis involving centrilobular regions and, later, even portal tracts (**Figs. 3–5**). "Reversed lobulation" was also described in her treatise, the process whereby relatively uninvolved portal tracts come to lie at the centers of hepatic parenchymal units circumscribed peripherally by fibrosis linking central veins. As with other publications of that period and later, the term "cardiac cirrhosis" was often used (while, in fact, a true cirrhosis was not present morphologically), chiefly because the criteria for cirrhosis were less stringent than those of today. In many instances, the term "cardiac cirrhosis" was used to describe combinations of central-to-central or central-to-portal bridging fibrosis with nodularity due to periportal regenerative hyperplasia or in cases of perivenular fibrosis (cardiac *sclerosis*) with frank nodular regenerative hyperplasia (NRH)[6]