The SGOT/SGPT Ratio—An Indicator of Alcoholic Liver Disease

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The SGOT/SGPT ratio is significantly elevated in patients with alcoholic hepatitis and cirrhosis (2.85 ± 0.2) compared with patients with postnecrotic cirrhosis (1.74 ± 0.2), chronic hepatitis (1.3 ± 0.17), obstructive jaundice (0.81 ± 0.06) and viral hepatitis (0.74 ± 0.07). An SGOT/SGPT ratio greater than 2 is highly suggestive of alcoholic hepatitis and cirrhosis. It occurs in 70% of these patients compared with 26% of patients with postnecrotic cirrhosis, 8% with chronic hepatitis, 4% with viral hepatitis and none with obstructive jaundice.

While no single biochemical liver function test is sufficiently specific to allow a definite diagnosis in patients with liver disease, we have been impressed by the consistency with which the serum glutamic oxaloacetic transaminase (SGOT) activity exceeds the serum glutamic pyruvic transaminase (SGPT) activity in patients with alcoholic liver disease. While this finding has been noted previously (1, 6), it does not appear to be widely recognized or generally accepted (7, 8). We therefore reviewed our experience to determine the diagnostic usefulness of the SGOT/SGPT ratio.

MATERIALS AND METHODS

Patient Selection. Data from 271 patients with histologically documented liver disease and elevated serum transaminases seen between July 1967 and June 1975 form the basis of this report. All patients met the following criteria: (1) unequivocal liver disease documented by either percutaneous needle biopsy or open biopsy of the liver, and (2) elevation of SGOT and/or SGPT, that is, values greater than 40 Karmen units. All SGOT and SGPT determinations were performed on the same blood sample. SGOT and SGPT were measured in the hospital clinical chemistry laboratory by the Karmen method. Where serial transaminase determinations were available, the set with the highest individual transaminase value, be it SGOT or SGPT, was used. All liver biopsies were reviewed by one of us (MMK) who had no knowledge of the transaminase values. Patients with the following diagnoses were included in the study: viral hepatitis (52 patients) (9), chronic active hepatitis (48 patients) (10), postnecrotic cirrhosis (30 patients) (11), alcoholic hepatitis and/or cirrhosis (104 patients) (12), and extrahepatic bile duct obstruction (37 patients). Statistical analysis was by unpaired analysis of variance.

RESULTS

The mean SGOT/SGPT ratio was significantly higher in patients with alcoholic liver disease (2.85 ± 0.2) than in each of the following groups: acute viral hepatitis, 0.74 ± 0.07 ($P < 0.001$), extrahepatic biliary obstruction, 0.81 ± 0.06 ($P < 0.001$), chronic active hepatitis, 1.3 ± 0.17 ($P < 0.001$), and inactive postnecrotic cirrhosis, 1.74 ± 0.20 ($P < 0.001$) (Figure 1). The great majority of patients with alcoholic liver disease (92%) and postnecrotic cirrhosis (70%) had SGOT/SGPT ratios greater than 1 (Figure 2). Only 12 percent of patients with viral hepatitis and 18 percent of patients with obstructive

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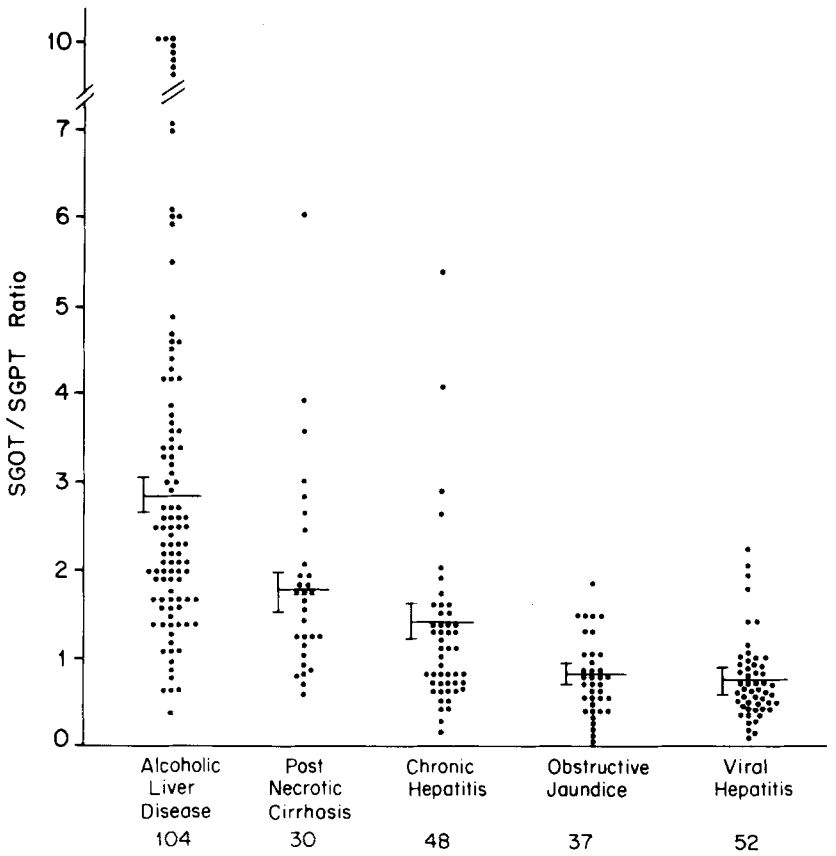

Fig 1. SGOT/SGPT ratio in patients with biopsy proven liver disease. The ratio was calculated from the serum sample that had the highest individual transaminase value, be it SGOT or SGPT.

jaundice had ratios greater than 1, while patients with chronic active liver disease were in the middle with 50%. When one looked at patients with an SGOT/SGPT ratio of 2 or greater, the ratio was surprisingly specific for patients with alcoholic liver disease (Figure 2). Seventy percent of these patients had ratios greater than 2, compared with only 26% of patients with postnecrotic cirrhosis, 8% with chronic active hepatitis, 4% with viral hepatitis, and none with obstructive jaundice.

The absolute SGPT determination was also of some use. The SGPT was less than 300 KU in 98% of patients with alcoholic liver disease, in 90% of patients with postnecrotic cirrhosis, in 70% of patients with obstructive jaundice, in 46% with chronic active hepatitis, and in only 21% of patients with viral hepatitis. That almost 80% of patients with viral hepatitis had SGPTs greater than 300 reflects the fact that liver biopsy was usually reserved for the sicker patients with viral hepatitis where transaminase values would be expected to be high.

While it is unlikely to find an SGPT level about 300 KU in patients with alcoholic liver disease, postnecrotic cirrhosis, or obstructive jaundice, any type of liver disease may be associated with an SGPT less than 300 KU. It was in this group of patients with moderately elevated transaminases and clinical presentations consistent with either mild viral hepatitis or early alcoholic liver disease where the SGOT/SGPT ratio was most valuable (Figure 3). When the SGPT was less than 300 KU, 91% of patients with alcoholic liver disease had an SGOT/SGPT ratio greater than 1, compared to only 4% of patients with viral hepatitis.

DISCUSSION

This study confirms the finding of Zimmerman and others (1, 6) and indicates that the SGOT/SGPT

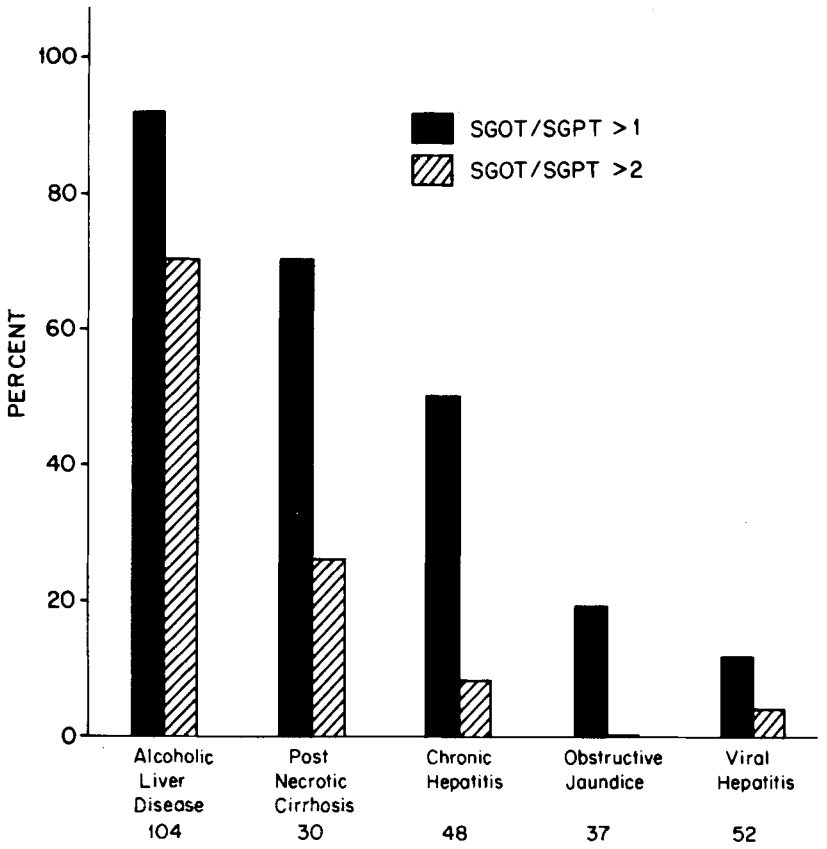

Fig 2. Percentage of patients with SGOT/SGPT ratios greater than one and greater than two.

ratio provides useful information in the diagnosis of liver disease. A ratio greater than 2 should make one suspect alcoholic liver disease. In addition, SGOT/SGPT ratios greater than 1 are unusual in patients with viral hepatitis and common in patients with postnecrotic cirrhosis as well as in those with alcoholic liver disease. The ratio is not particularly helpful in identifying patients with chronic active hepatitis or obstructive jaundice, although a ratio greater than 2 makes these diagnoses most unlikely.

We do not wish to imply that the transaminases should be used alone in diagnosis of liver disease. Obviously they must be used in conjunction with the other commonly employed liver function tests and considered in the context of the clinical setting. However, since both transaminases are often obtained, the information available should be used to its fullest extent. A high SGOT/SGPT ratio is particularly useful in suggesting alcoholic liver disease in patients with mild abnormalities of liver function tests in whom a history of alcohol excess is often hard to obtain.

We would agree with others (4, 5, 7) that in patients where the SGOT and SGPT exceed 300 KU, determination of the SGOT/SGPT ratio offers little additional useful information. In these individuals, a distinct minority of our own patients with liver disease, one is usually dealing with acute hepatocellular diseases such as hepatitis, heart failure, or toxic exposures. Other laboratory tests are usually abnormal and the likelihood of cirrhosis or alcoholic liver disease is low. However, when the transaminases are less than 300 KU, the diagnostic possibilities are broad and the SGOT/SGPT ratio may be helpful.

The pathophysiologic basis of the elevated SGOT/SGPT ratio in patients with alcoholic liver disease is not known. However, preliminary results from our laboratory show that hepatic GPT activity is significantly lower in patients with alcoholic liver

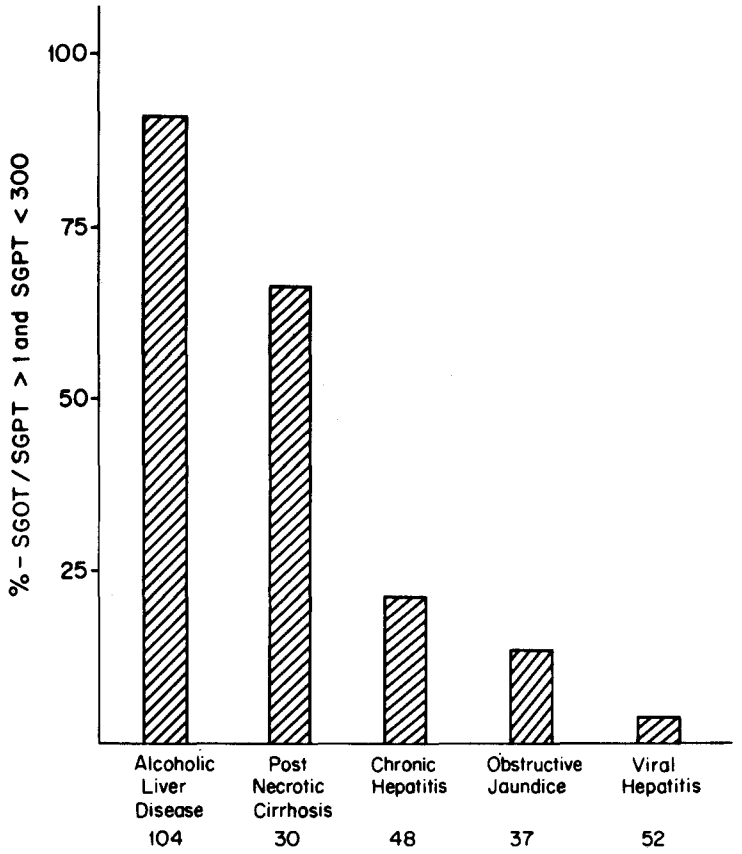

Fig 3. Percentage of patients with an SGOT/SGPT ratio greater than one and with the SGPT less than 300 Karmen units.

disease compared to both normal individuals and those with acute viral hepatitis (13). Thus there may be less GPT available to leak into serum in patients with alcoholic liver disease.

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