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THE SGOT/SGPT RATIO IN ALCOHOLIC LIVER DISEASE

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SUMMARY

The SGOT/SGPT ratio has been estimated in 207 patients with alcoholic liver disease and in 3 control groups (43 viral hepatitis, 30 non-alcoholic chronic liver disease and 42 obstructive jaundice). The SGOT/SGTP ratio in alcoholic liver disease group was significantly higher (2.63 ± 1.82) than in any of the 3 control groups. If we consider the different alcoholic liver diseases the ratio is 1.82 ± 1.27 in Steatosis, lowest value of all; 3.00 ± 2.33 in Acute alcoholic hepatitis; 2.45 ± 1.62 in Cirrhosis; 2.19 ± 1.94 in Cirrhosis with Steatosis; 3.48 ± 1.63 in Cirrhosis with AAH. Values higher than 1.5 are suggestive of alcoholic liver disease and higher than 2.0 are almost diagnostic and found in 56% of the patients with alcoholic liver disease and only in 1 case of non-alcoholic liver disease.

Since alcohol abuse is one of the most common causes of hepatocellular diseases, a simple biochemical test characteristic of alcoholic liver disease would be very helpful as a screening procedure. Values of SGOT higher than SGPT have been frequently reported [1-3] but only recently Cohen and Kaplan [4] proposed the use of the SGOT/SGPT ratio higher than 2 as highly suggestive of alcoholic hepatitis or cirrhosis.

We studied a large group of alcoholics and divided them according to liver histology in order to determine the diagnostic usefulness of the SGOT/SGPT ratio

PATIENTS AND METHODS

Over a period of 5 years, alcoholic liver disease was diagnosed in 207 patients admitted to our department. There were 161 men and 46 women, ranging from 10 to 82 years of age (mean 49.36 ± 11.71 yr.). We reviewed their records and evaluated the SGOT/SGPT ratio. The results were compared with those obtained in 3 control groups: 43 patients with viral hepatitis, 30 with non-alcoholic chronic liver disease and 42 with obstructive jaundice (all proved extrahepatic malignant obstruction without liver metastases). All patients had been submitted to percutaneous liver biopsy (with the exception of 13 patients in the viral hepatitis group), and the slides were reviewed blindly by the pathologist.

Alcoholics were divided in 5 groups according to the morphological features: 1) steatosis, 30; 2) acute alcoholic hepatitis (AAH), 32; 3) cirrhosis, 71; 4) cirrhosis with steatosis, 29 and 5) cirrhosis with acute alcoholic hepatitis, 45.

SGOT/SGPT ratio was determined on the basis of the transaminases measurements in the first blood sample after admission, expressed in Karmen Units.

Statistical analysis was carried out by analysis of variance: method of multiple comparisons of Schéffé (5) and X^2 analysis. Values are expressed as mean $\pm$ 1 SD.

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RESULTS

Serum transaminases values were higher in patients with viral hepatitis: 74% had values in excess of 300 UK/ml compared with 13% of the non-alcoholic chronic liver disease, 5% of the alcoholics and 2% of those with obstructive jaundice.

In alcoholic liver disease the SGOT/SGPT ratio (2.63 $\pm$ 1.82) was significantly higher than in the 3 control groups: viral hepatitis, 0.88 $\pm$ 0.55 ($p < 0.001$), obstructive jaundice, 1.02 $\pm$ 0.38 ($p < 0.001$) and non-alcoholic chronic liver disease, 0.94 $\pm$ 0.41 ($p < 0.001$) (Fig. 1). There was no singnificant difference among the mean values of the 3 control groups, so we considered them for future analysis as a single non-alcoholic group: mean ratio 0.95 $\pm$ 0.47. Fig. 2 shows the distribution of the SGOT/SGPT ratios found in the alcoholics subdivided according to histology. The mean value of the ratio in each group is still higher than in the non-alcoholics, though the difference is less significant for the steatosis group ($p < 0.05$) than for any of the others ($p < 0.001$). In fatty liver the mean value of the SGOT/SGPT ratio (1.82 $\pm$ 1.27) was significantly lower than in AAH, 3.00 $\pm$ 2.33 ($p < 0.01$), cirrhosis, 2.45 $\pm$ 1.62 ($p < 0.01$), cirrhosis with steatosis, 2.19 $\pm$ 1.94 ($p < 0.05$) and cirrhosis with AAH, 3.48 $\pm$ 1.63 ($p < 0.001$).

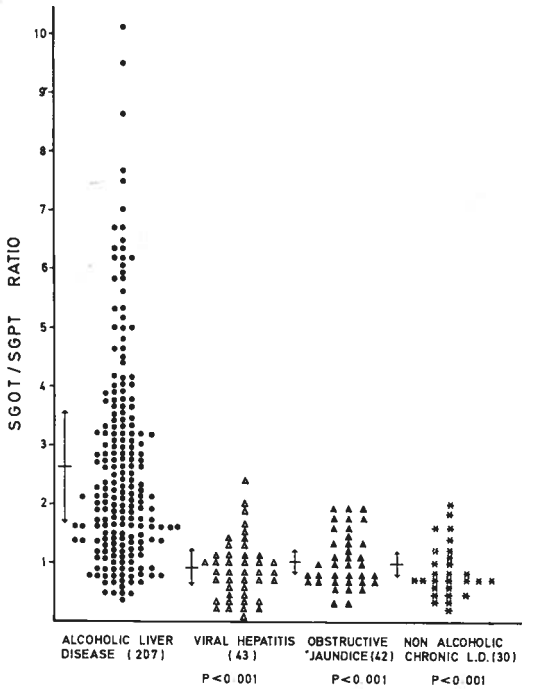

Fig. 1 — *SGOT/SGPT ratio in patients with liver disease.*

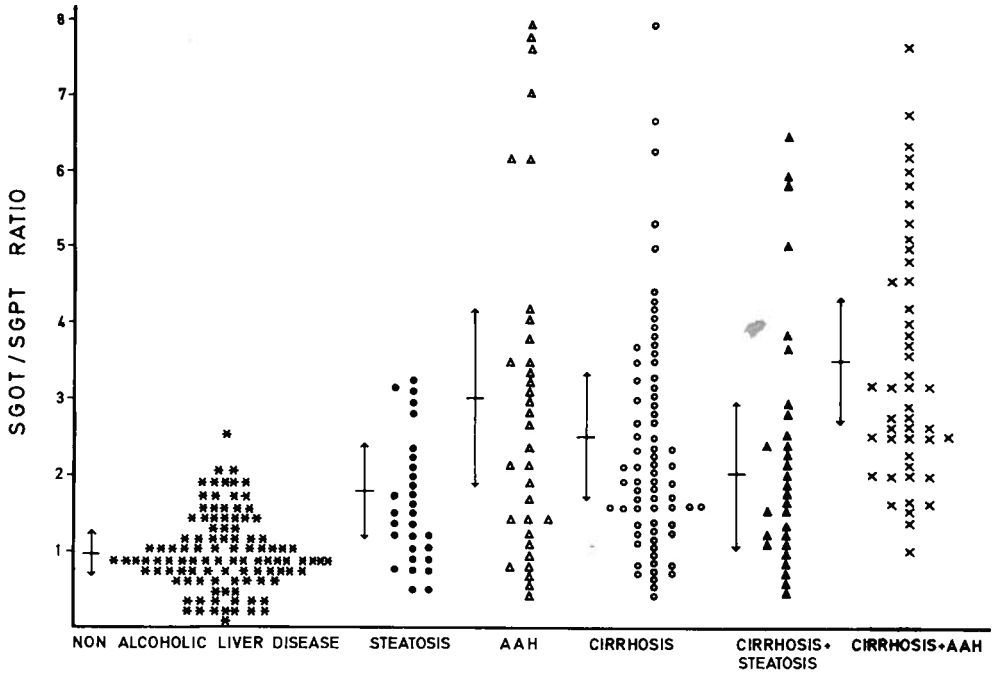

Fig. 2 — *SGOT/SGPT ratio in alcoholics divided according to liver histology.*

We have no information neither in alcoholics without liver disease nor in drug related hepatitis.

A SGOT/SGPT ratio > 1 was found in 180/207 patients with alcoholic liver disease (87%) compared with 44/115 (38%) of the non-alcoholics ($p < 0.001$). The difference is even more significant when we consider a ratio > 1.5 ($p < 0.001$). 56% (116/207) of patients with alcoholic liver disease presented a ratio > 2, compared with 0.9% (1/115) of those with non-alcoholic liver disease ($p < 0.001$). In cirrhosis + AAH, 84% of the patients had a SGOT/SGPT ratio > 2. The incidence is higher (though not significant) than in patients with AAH (60%), but is statistically different ($p < 0.001$) when compared with steatosis (30%), cirrhosis (49%) or cirrhosis + steatosis (52%) (Fig. 3).

We then evaluated which transaminase activity influenced the most the presence of a ratio > 2. This was mainly due to a significant decrease of SGPT ($p < 0.05$), though there was a slight increase in SGOT when compared with patients with a ratio < 2 (Fig. 4).

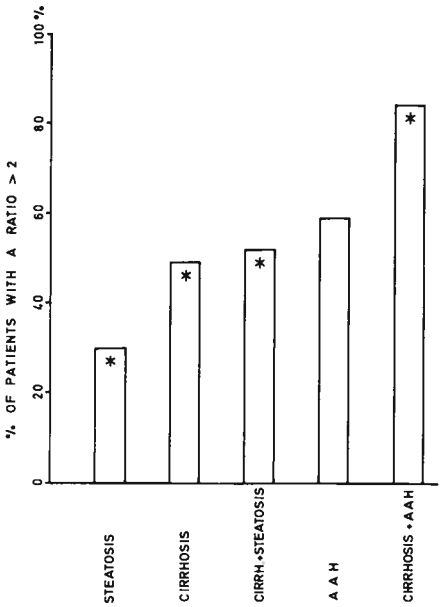

Fig. 3 — *Percentage of alcoholics with a SGOT/ /SGPT ratio greater than two (* $p < 0{,}001$).

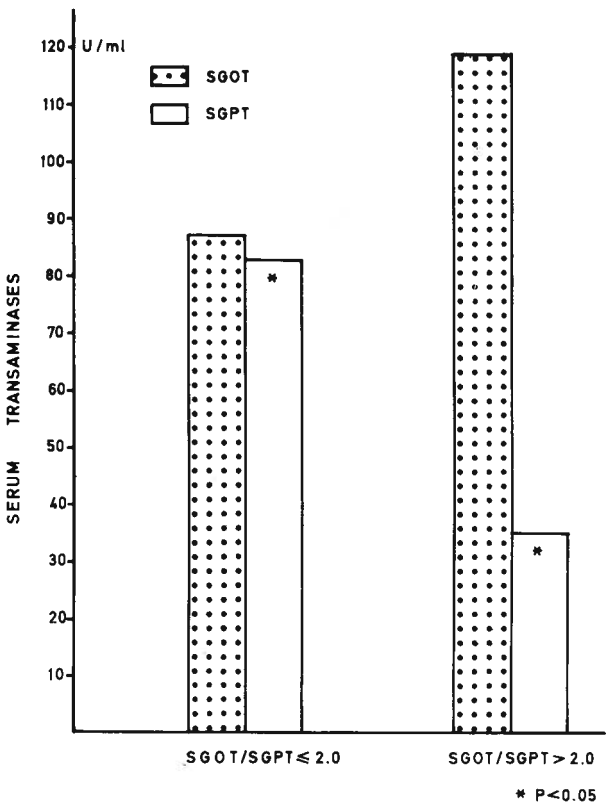

Fig. 4 — *Mean values of serum transaminases in patients with alcoholic liver disease when the SGOT/SGPT ratio is lower or greater than two (* $p < 0{,}05$).

DISCUSSION

Our results, similar to those obtained by Cohen and Kaplan [4] confirm the usefulness of the SGOT/SGPT ratio in distinguishing alcoholic liver disease from other aetiology, specially when serum transaminase activities are not very high. [3, 6, 7] A ratio higher than 1 is not characteristic, it represents the pattern in normal subjects. [3] Combined with an appropriate clinical setting, in our experience and in the hospital population a SGOT/SGPT ratio higher than 1.5 is highly suggestive of alcoholic liver disease. When superior to 2 is almost diagnostic. However it is not selective enough to grade severity of liver damage (jugded histologically), although it tends to be more frequent in AAH with or without cirrhosis. So far, transaminase changes seen in alcoholic liver disease are not yet fully understood; they are only partly caused by liver damage, as an increased SGOT activity following alcoholic *debauch* can be related to muscle damage. [8] We have shown that a SGOT/SGPT ratio > 2 is mainly due to a remarkable decrease of SGPT. This is probably specific of alcoholic liver disease as it is not observed in other hepatic diseases, it is not related to chronicity of liver disease or the presence of cirrhosis, and reflects a diminished hepatic GPT activity. [9, 10] Its cause has been recently enlightened by Ludwig and Kaplowitz [11] who demonstrated that the influence of pyridoxyne deficiency on the transaminase response to acute liver injury, while affecting both transaminases, results in a significantly greater reduction of GPT than GOT, both in liver cytosol and serum. Of particular interest is that patients with severe alcoholic fatty liver and cirrhosis have decreased hepatic Vit. B_6 content and low plasma pyridoxal phosphate levels (PLP — the active metabolite of Vit. B_6). Besides, there is a high incidence of deranged PLP metabolism in chronic alcohol abuse, even without evidence of liver disease. [12] If the effects of alcohol on pyridoxyne metabolism account, at least in part, for transaminase changes in alcoholic liver disease it is not surprising the high percentage of patients with a SGOT/SGPT ratio > 2 whatever the severity of liver damage.

RESUMO

A RELAÇÃO SGOT/SGPT NA DOENÇA HEPÁTICA ALCOÓLICA

Calculou-se o quociente TGO/TGP em 207 doentes com doença hepática alcoólica e em 3 grupos controlos: hepatite aguda viral (43 doentes), doença hepática crónica não alcoólica (30 doentes) e icterícia obstrutiva (42 doentes). O quociente TGO/TGP na doença hepática alcoólica é significativamente superior (2.63 ± 1.82) ao encontrado nos grupos controlos. Em relação aos diferentes tipos de lesão hepática alcoólica, verificou-se que os quocientes mais elevados se observam na Hepatite Aguda Alcoólica isolada (3.00 ± 2.33) ou associada a Cirrose Hepática (3.48 ± 1.63), e os mais baixos na Esteatose (1.82 ± 1.27), observando-se valores intermédios na Cirrose com esteatose (2.19 ± 1.94) ou sem esteatose (2.45 ± 1.62). Valores do quociente TGO/TGP superiores a 1.5 são sugestivos de doença hepática alcoólica, e superiores a 2 quase diagnósticos, uma vez que se observaram apenas em 1 dos 115 casos de doença hepática não alcoólica e em 56% dos doentes com doença hepática alcoólica.

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