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Changes in Serum Transaminases due to Hepatotoxicity and the Role of an Indigenous Hepatotonic Liv.52

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ABSTRACT

The beneficial role of Liv.52 against CCl_4 induced hepatotoxicity has been studied histologically in albino rats and also by assessing the alterations in the serum transaminases (SGPT and SGOT). It has also been found that pre- and post-treatment with Liv.52 prevents any notable hepatic damage and the serum transaminase levels tend to reach the normal range. It has been found that CCl_4 induced hepatotoxicity resulted in a significant elevation of the SGPT level. The complete safety of Liv.52 as a hepatotonic in the prevention and correction of hepatic damage has been emphasized.

INTRODUCTION

Experimental poisoning with carbon tetrachloride (CCl_4) has an ancient and varied history and its hepatotoxic action has been reviewed by several workers (Himsworth 1950; Drill 1952; Popper and Schaffner 1957). Yet the views on the mechanism of genesis of hepatotoxicity are still controversial.

The use of serum enzyme activities for human clinical diagnosis has increased greatly during the past several years (Bergmeyer 1963; Tietz 1970; Wilkinson 1970). In the past it has also been reported that serum transaminase activity has been found to be altered in certain pathological conditions which were associated with necrosis or other type of cellular damage of cardiac, hepatic or skeletal muscle tissues (Molander *et al.* 1955; Steinberg and Ostrow 1955; Mason and Wroblewski 1957). The activity of glutamic pyruvic transaminase (SGPT) was found to be relatively greater in liver than in other tissues as compared to the activity of glutamic oxaloacetic transaminase (SGOT) (Wroblewski and La Due 1956; Henry 1959). This might perhaps suggest that the serum glutamic pyruvic transaminase (SGPT) might possibly be a more specific index of liver cell damage than the SGOT, because of its relative concentration in the hepatic tissue. The assessment of liver function and the use of serum enzyme changes in toxicity studies has been reviewed (Cornish 1971; Grice 1972; Plaa 1968). Grice *et al.* (1971) examined the correlation between serum enzymes, isoenzymes and histologically detectable organ damage due to carbon tetrachloride, mercuric chloride, diethanolamine and thioacetamide.

The present investigation was undertaken to find out the protective role of the indigenous drug, Liv.52 (The Himalaya Drug Co.) on experimentally-induced hepatic damage with carbon tetrachloride by assessing the alterations in the serum transaminase activity as a criterion of hepato-cellular integrity.

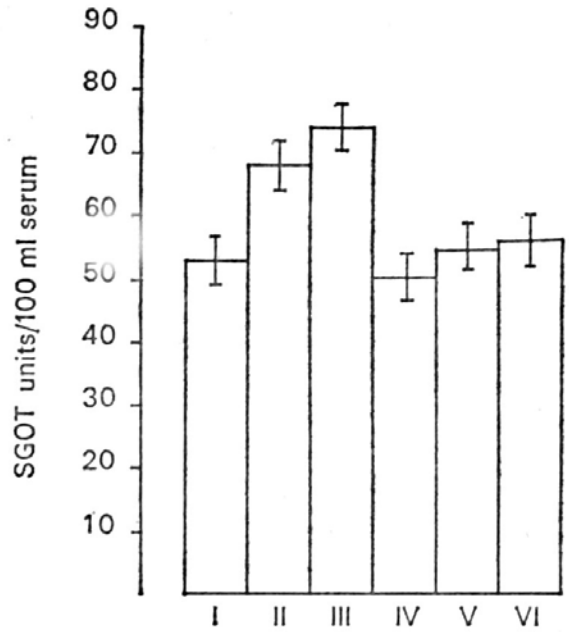
MATERIALS AND METHODS

Experiments were performed on 60 male albino rats weighing between 100 to 130 g. They were divided into the following six groups of ten each. Group I: Control, Group II: received CCl_4 (0.2 ml/g) orally and was sacrificed after 24 hours. Group III: received CCl_4 (0.2 ml/g) orally and was sacrificed after 48 hours. Group IV: received Liv.52, 2 ml daily for 10 days. Group V: was administered Liv.52, 2 ml daily for 10 days also CCl_4 (0.2 ml/g) and Liv.52, and sacrificed after 24 hours of CCl_4 administration. Group VI: was administered Liv.52 as the above group and CCl_4 and Liv.52 and was sacrificed after 48 hours of CCl_4 administration.

The serum of control and drug-treated groups was collected and the transaminases (SGPT and SGOT) were estimated according to the method of Reitman and Franket (1957).

A piece of liver was also examined histologically to ascertain the degree of liver damage.

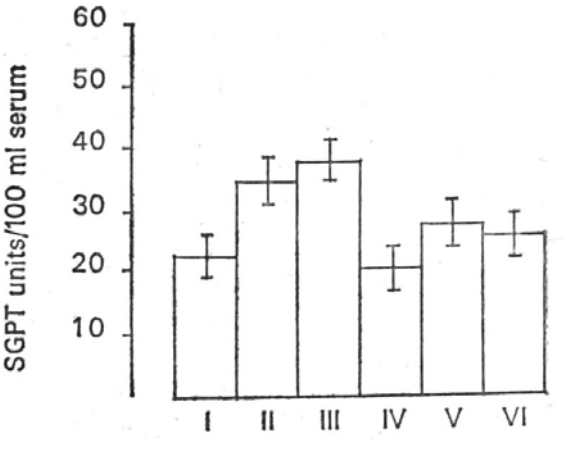
Fig. 1: Effect of CCl_4 and Liv.52 on serum glutamic oxaloacetic transaminase (SGOT). I = Control; II = 24 hours after CCl_4; III = 48 hours after CCl_4; IV = Liv.52 for 10 days; V = Liv.52 + CCl_4 after 24 hours, later for 10 days; VI = Liv.52 + CCl_4 + Liv.52, 48 hours, later for 10 days. Vertical bars = S.E.



RESULTS

The activities of SGOT and SGPT of the CCl_4 treated groups showed a significant elevation (Figs. 1 and 1a) when compared to the groups, which received Liv.52 before and with CCl_4 administration.

Fig. 1a: Effect of CCl_4 and Liv.52 on serum glutamic pyruvic transaminase (SGPT). Description as in Fig. 1.



DISCUSSION

The results of the present study revealed a significant increase of the serum transaminases with CCl_4. This elevation could be explained on the basis that CCl_4 caused necrosis of liver cells. It is known that as a result of necrosis of the hepatic cells caused by acute infection or chronic liver disease, these enzymes are released

into the circulation with consequent rise in the serum levels (Wroblewski and La Due 1955; Molander *et al.* 1957; Zelman *et al.* 1959) and a persistent rise in serum transaminase levels is presumably an indication of continuing liver cell damage. The elevation of the serum transaminase, especially the SGPT activity, could be explained on the basis of damage due to CCl_4. Administration of Liv.52 before and after CCl_4 poisoning prevented liver damage which was also confirmed histologically and the serum transaminase levels also remained within the normal range. This finding indicates that probably Liv.2 conditioned the hepatic cells and prevented any further damage to the liver parenchyma by virtue of which the leakage of these enzymes into the circulation was prevented. It could also be emphasized that Liv.52 aided quicker regeneration of hepatic parenchyma and also its stimulating action markedly increased the functional efficiency of liver. This could be substantiated with the observation of Molander *et al.* (1957), who also have reported that the serum levels of the transaminases return to normal as the liver parenchyma heals and the liver cells regenerates. This is what happened with the Liv.52 treated groups in this study. Therefore, it could be concluded from this investigation that Liv.52 has proved to be a good hepatotonic and could be used to rectify hepatic damage.

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