DRUG INDUCED LIVER DISEASE

Pages with reference to book, From 32 To 33

Liver disease analogous to that found with acute viral hepatitis can be produced by several well tolerated therapeutic agents (Maddrey and Boitnott, 1977). Drugs may cause mild hepatic injury resulting in transient elevation of transaminases, non icteric and icteric hepatitis, fulminant hepatitis, chronic persistent and aggressive hepatitis, subacute hepatic necrosis and postnecrotic cirrhosis.

Elevation of serum transaminases occurs in 10% of patients treated with Isoniazid without any clinical evidence of liver disease (Schaper and Smith, 1969; Byrd et al, 1972). Clinically apparent hepatitis is infrequent in patients treated with this drug but when present it mimics acute viral hepatitis (Maddrey and Boitnott, 1973; Moss et al., 1972; Black et al., 1975; Martin and Arthaud, 1970). Fatal acute fulminant hepatitis (Maddrey and Boitnott, 1973; Black et al., 1975), submassive and massive necrosis and cirrhosis were also found in patients receiving Isoniazid. Liver disease of varying severity is more frequent in patients receiving Isoniazid longer than 2-3 months and in those who continue to take the drug even after the appearance of symptoms like nausea, malaise and anorexia (Maddrey and Boitnott, 1973; Black et al., 1975). Patients who recover show mild residual scarring without any evidence of ongoing necrosis.

Asymptomatic elevation of serum transaminases occur in 5% of patients receiving Methyl-dopa (Elkington et al., 1969; Irvine et al., 1962). Acute hepatitis, massive hepatic necrosis and bridging and/or multilobular necrosis and chronic aggressive hepatitis have been observed by Maddrey and Boitnott (1975) and Toghill et al (1974). Schweitzer and Peters (1974) reported a case of Aldomet induced hepatitis which progressed to cirrhosis. The LE cell and direct coomb's test were positive and smooth muscle antibodies were negative.

Halothane induced hepatic disease is rare (Klion et al., 1969; Peters et al, 1969; Sherlock, 1971) but a high mortality due to fulminant hepatocellular failure has been reported by various workers (Trey et al., 1968; Klion et al., 1969; Peters et al., 1969). Klatskin and Kimberg (1969) and Thomas (1974) have observed recur-rant hepatitis leading to cirrhosis and chronic aggressive hepatitis in patients exposed to this anaesthetic agent.

Oxyphemsatin induces an entire spectrum of hepatic damage ranging from asymptomatic transaminase increase to chronic aggressive hepatitis and cirrhosis of the liver (Reynolds et al., 1971; Cooksley et al., 1973; Willing and Hecker, 1971).

Phenothiazines produce cholestasis which occurs more frequently in females (Ishak and Irey, 1972) and subsides over an interval of 1-3 months after drug withdrawl.

Other commonly used drugs which are known to cause liver disease are sulphonamide, Nitrofurantoin and phenylbutazone (Lindberg et al., 1975).

Recognition that a drug is the probable cause of hepatic injury and its prompt withdrawl suffices in the management of drug hepatitis regardless of whether the injury is actue or chronic.

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