

**C H A P T E R - V**

**GENERAL DISCUSSION**

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The observations of liver histology and kidney histology are the conclusive in curative effects of mandur bhasma since the normal histology is the reflection of normal physiology or indicator of normal physiology. Inverse is also true.

The acute hepatic injury induced by  $\text{CCl}_4$  alone,  $\text{CCl}_4$  + liquid paraffin or even liquid paraffin alone had shown minimum partial spontaneous recovery on cessation of the treatment for 7 days. There is hardly any event observed which can show the regeneration of liver. Therefore, there are two possible reasons that indicate the cure. One the recovery of hepatic cells from toxic effects and second is that within the 7 days of mandur bhasma treatment the regenerative cycle may have passed so that there are no signs of regeneration. Since the hepatoprotection offered by Mandur bhasma in earlier work is through regenerative events in the liver (Devarshi et al, 1986).

Not only  $\text{CCl}_4$  but liquid paraffin also is injurious to liver and comparatively more injurious to kidney.

The alterations in histological elements are dominated by centrolobular necrosis in  $\text{CCl}_4$  treated rats. Different types of necrosis in the hepatocytes in liquid paraffin and  $\text{CCl}_4$  + liquid paraffin treated livers are observed which are described in detail in Chapter III. The entry of trafficking elements Kupffer cells and sinusoidal cells during acute hepatotoxicity is conspicuously blocked

but in curation period their number and distribution in the liver is restored which indicated hardly any infiltration of sinusoidal cells which is significant sign in the recovery or cure of liver.

The other parameter used is to study cure is lipid peroxidation which is well studied in  $\text{CCl}_4$  induced hepatic injury. The lipid peroxidation is coupled with the microsomal membrane damage (Mead, 1976; Henriksen et al,1976; Mehendale, 1985). With the microsomal membrane damage glucose-6-phosphatase a membrane bound enzyme also shows loss (Mehendale, 1985, Lehninger, 1982; Nordlie et al,1982). Therefore to evaluate the membrane damage and recovery glucose-6-phosphatase activity is used along with alterations in lipids and proteins content.

The treatment of mandur bhasma leading to 60 to 75 % recovery of liver is expressed through histological recovery the alterations in biochemical parameters can be evaluated to suggest probable path of hepatocurative processes.

The data indicates that lipid peroxidation is coupled with membrane and hence enzyme damage which is at equilibrium during active times of recovery leading to suppression of lipid peroxidation and normal maintenance of the enzyme glucose-6-phosphatase enzyme activity.

The simultaneous renal stress due to hepatic injury is also

partially recovered which is also observed in our earlier studies of hepatoprotection by Mandur bhasma from  $\text{CCl}_4$  induced hepatic injury (Devarshi et al, 1986).

Thus mandur bhasma is not only hepatoprotective but hepatocurative also. The curative effects are possibly achieved through invoking the in vivo systems to cure the liver from the damage occurred due to lipid peroxidation. The systems may be biological antioxidative agents (Fridovich, 1975; Jappel, 1968) or the protection through structural separation by isolating the peroxidation catalyst from the unsaturated lipids and lipids are appear to be protected by the masking of double bonds (Barber and Bernheim, 1967) or through lowering the  $\text{O}_2$  tension locally (Philpott et al, 1974).

Any one of the above systems or integral use of three may be involved in curation.

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