Toxic Effects of Crude Venom of a Desert Cobra, Walterinnesia aegyptia, on Liver, Abdominal Muscles and Brain of Male Albino Rats

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Abstract:

The toxic effect of an acute dose of Walterinnesia aegyptia crude venom was studied in male albino rats. Liver enzymes,alaninetransaminase (ALT), aspartate transaminase (AST) and gamma glutamyltransferase(-GT), total protein concentration and Alkaline phosphatase(ALP) enzyme activity in the liver, abdominal muscles and cerebrum brain were measured at timed intervals of 1, 3, 6, 12, 24, 72 h and 7 days post envenomation. The histological changes in the liver sections were simultaneously investigated. These parameters were found to be fluctuated with time, with a tendency to regain to normal control levels within the first 6 h. Histological changes induced by treatment with LD50 of W. aegyptia crude venom in liver 3 to 6 hours post envenomation showed inflammatory cellular infiltrations(ICI) around the hepatic vein, dilated blood sinusoids (S), hepatocyticvacuolations (HV) and prominent van kuffer cells. The 12 to 24 h period seems to be crucial for the process of physiological recovery. Histological changes induced by treatment with LD50 of W. aegyptia crude venom in liver 12, 24, 72 hrs to 7 days post envenomation showed hepatocytic-vacuolations, inflammatory cellular infiltration and dilated sinusoids. Under higher magnification, marginal chromatin (mc) patterns appear in some hepatocytes and clumped chromatin (cm) in others. With this same group, liver sections taken at 72 h and at 7 days showed increased inflammation and vacuolation as evidenced by an increase in inflammatory cells, some pyknotic cells (Pn), widened sinusoids and numerous van Kupffer cells. Fatty change or Steatosis(St) represents the intracytoplasmic accumulation of triglycerides (neutral fats) of parenchymal organs. Physiological adaptation and recovery from an LD50 venom dose seems to be achieved after one week, leaving the animal alive with several lesions especially in the liver (such as pyknotic nuclei, steatosis and clumped chromatin and disturbed physiological profile).

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