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Biochemical and histopathological study in rats intoxicated with carbon tetrachloride and treated *Vernonia amygdalina*

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Introduction: *Vernonia amygdalina* belongs to the family Asteraceae. *Vernonia amygdalina* contains active components or phytochemicals that can lead to liver regenerations in hepatotoxicity in hepatotoxicity.

Aim: The aim of this study is to assess the chemotherapeutic and hepatoprotective effect of leaf extract of *Vernonia amygdalina* in rats.

Method: A total number of 15 albino rats were fed on standard diet and divided into three groups. Rats of the first group were injected intraperitoneally with paraffin oil and tap water (control). Rats of the second and third groups were intraperitoneally injected with CCL₄, standard diet and tap water. The third group was treated with the leaf extract of *Vernonia amygdalina*. Blood and liver samples were collected for biochemical and histopathological analysis.

Results: Phytochemicals such as flavonoids, tannins were contained in the leaf extract of the plant. Levels of alanine ALT, GGT and AST were highest in the CCL₄ treated group with a mean and standard deviation of AST (88.25±14.22), ALT (89.25±8.99) and GGT (91.75±6.32) respectively as compared to the control and CCL₄ treated groups. Histopathologically, a greater amount of mononuclear cell infiltration, necrotic and few fibroblasts were observed in the liver of CCL₄ treated groups whiles liver regeneration was observed in the third group.

Conclusion: The results showed that the leaves of *Vernonia amygdalina* possessed hepatoprotective abilities.

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Medications in pregnancy: Potential pitfalls in the evaluation of postnatal effects in the offspring

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Fetal exposure to medications has been implicated in the development of childhood illnesses. Large cohort studies have reported increased risk of childhood asthma following maternal use of paracetamol, antibiotics and antacids in pregnancy. A greater risk for epilepsy in childhood has been attributed to fetal exposure to antibiotics; use of macrolides, in particular, was related to the development of cerebral palsy and epilepsy in the offspring. Prenatal exposure to antibiotics has been implicated in increased risk of offspring obesity. However, maternal consumption of antacids (proton pump inhibitors and H₂ blockers) 2 years after delivery (and not during pregnancy) was also related to the development of asthma in the offspring. Maternal use of paracetamol 30 days prior to the first menstrual period was reported to increase the risk of childhood asthma but this association was also found for the use not in pregnancy but one year after birth. Those contradictory reports may be explained by untested potential confounders; they stress the necessity more extensive pharmacoepidemiological investigations.

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