hematological disturbances induced by lead administration, which could be attributed to either hyper stimulation of thrombopoiesis in the bone marrow as a response to peripheral loss of platelets caused by increased platelet aggregation and adhesion [4,10], or as response to decreased Hb concentration caused by lead acetate administration. This result is in agreement with the results of other researchers who found that accelerated platelet aggregation when there is lowered Hb concentration [23]. On the other hand, results of the present study reflected the protective role of methionine and vitamin E against changes induced by lead administration. There were no changes in platelet count in rabbits received vit.E and methionine (Table 2). This protective effect could be explained by the direct action of vit.E and methionine on reducing the aggregation and adhesion of platelets in peripheral blood vessels, keeping normal blood level of thrombocytes in rabbits. Further, Vit.E supplementation also proved to be effective in significantly decreasing the already raised values of platelets [24].

Although results in table 2 indicated a significant decrease in lymphocyte count, which was correlated with a significant increase in neutrophil count; however, the mean total WBC count was relatively unchanged. Despite that, not all researchers agree on the effects of lead on total and differential WBCs [18,25]. The damaging effects of the reactive oxygen species on living systems as a result of lead intoxication required more phagocytic functions faced by increase of neutrophils. These damaging effects were prevented by the protective action of vitamin E, thus, neutrophil count returned back to semi normal values in rabbits administered vit. E alone or mixed with methionine reflecting either the antioxidant property and/or the protective role of vit. E on bone marrow [12,22,24,25].

After 90 days of the experiment, results revealed a significant increase in serum iron faced by the significant decrease in serum ferritin in rabbits administering lead acetate as compared with the control and other groups (table-3). Lead inhibits ferrochelatase enzyme that catalyzes the incorporation of iron into the porphyrin ring causing reduced iron incorporation in Hb and elevation of serum iron [10,27]. Moreover, competition between lead and iron, since lead is similar to some other ions, enables lead to take the place of iron in heme molecule of Hb, leading to increased serum iron [28]. The decreased serum ferritin may be due to the accumulation of the ferritin in the form of the hemosiderin, resulting in hemosiderosis and hemochromatosis, both of which are associated with excessive levels of serum iron and % saturation of the transferrin with iron and decreased serum ferritin levels [29]. The hemosiderosis observed in the liver of rats administering lead acetate (Figure 2) may be due to increased serum iron released from the heme liberated from the premature hemolysed RBCs. This excess iron cannot be utilized for Hb synthesis, because of the toxic effects of lead. Increased intracellular iron, increased ferritin expression, deposed as hemosiderin in liver [5,20,30]. The severity of these findings were decreased in groups administered vit. E alone or mixed with methionine (Figures 3, 4 and 5), reflecting again the protective role of vit. E against lead toxic effects. These finding could be explained that prevention of the preliminary RBCs elimination decreases phagosytosis and consequently decreases iron deposition, as well as the antioxidant effect of vit. E in reducing liver parenchymal damage and ferritin release to the circulation. L-methionine produces an increase in ferritin protein expression, thus activation of endogenous iron sequestration could be an important mechanism by which methionine increases the cellular defense against oxidative injury [11,17].

In conclusion, decreased MCV, MCH, and MCHC and reticulocytosis demonstrate regenerative anemia in rabbits administering lead acetate; since reticulocytes are still synthesizing Hb and bone marrow tries to compensate these changes by an increase in the reticulocytes release while lead causes increased iron in circulation and intra cellular deposition, vit. E alone or mixed