of glucocorticoid across the plasma membrane and influences the intracellular entrance and transport to the nucleus of steroid bound to protein [8].

Glucocorticoids inhibit glucose and amino acid uptake in many instances and enhance lipolysis in adipose tissue [9]. In the liver, these steroids stimulate a number of enzymes and increase protein and glycogen content. There is an enhanced hepatic capacity for gluconeogenesis; which, with substrate from catabolism elsewhere, results in increased glucose production.

The integrated effects of glucocorticoids thus result in hyperglycemia, negative nitrogen balance and fat loss [9]. The general stimulatory glucocorticoid effect on the liver is in pattern of hypertrophy of hepatocytes, since the total protein content in the liver cell is increased [10].

The objective of this study was to explore the effects of dexamethasone sodium phosphate on the rabbit liver as a model for human liver, by a light microscope, using two extreme of doses and two durations to confirm the dose and duration dependency.

Methods

Healthy white New Zealand female rabbits weighing between 1000-1250 grams were kept in separate plastic cages, fed *ad-libitum* and used for scientific research from January to march 2012 in Al-Mustansiriyia College of Medicine Laboratories.

The animals were divided into six groups, seven animals in each. The first group was treated daily for 10 days with (0.5 mg/kg of body weight (b.w.) equal to 0.1 ml/kg b.w.) intramuscular injection of dexamethasone sodium phosphate as single injection every 24 hours (ZMC import-export GmbH Germany as 8 mg/2 ml ampoules) in the thigh muscle. The second group was treated with (1.5 mg/kg b.w. equal to 0.4 ml/kg b.w.) of the same reagent for 10 days. The third group was received (0.5 mg/kg b.w.) of dexamthasone for 15 days. The fourth one was treated with (1.5 mg/kg b.w.) of dexamethasone sodium phosphate for 15 days. The fifth group was considered as a control (1) animals, they received equal amounts of 0.9% saline solution as intramuscular injections for 10 days. The sixth group was received also 0.9% saline solution for 15 days and considered as the control (2) group [11].

Twenty- four hours after the last injection, the animals were anaesthetized with chloroform. After dissection of the abdomen, the liver were removed and they were fixed in 10% formaline solution for 24 hrs., dehydrated, cleared, and embedded in paraffin and the blocks obtained were sectioned and stained by:

1. Haematoxyllin and Eosin stain (H&E): routine slandered stains for general structure of liver [12].
2. Alcoholic periodic acid- Schiff's stain (PAS): for carbohydrates including glycogen, mucin, and most basement membranes [13]. Staining methods and techniques were done on the basis of Humason and Luna [12, 13].

Results

H&E sections show vacuolation and ballooning of hepatic cells, which started to appear more clearly in the second and fourth groups (as the dose and duration of treatment were increased) as seen in fig. 1.

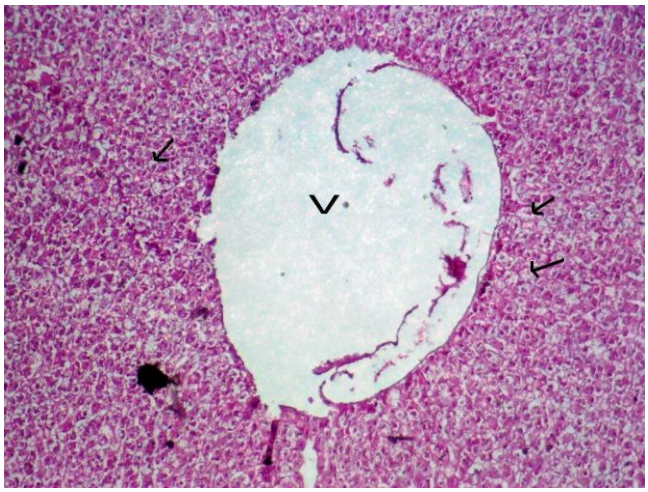

Fig. 1: Photomicrograph of liver cells of treated groups showing dilated and congested central vein (V), hepatic cells (arrows) H&E X100.

Degenerative changes of liver cells were noticed including; distortion of nuclei with distortion of