Toxicity effects of nanosilver on liver enzymes, liver and lung tissues

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ABSTRACT

In past handkerchief soaked in silver was used to treat soldiers' wound. Currently nanosilver is used widely in health and medicine to deterge germs.

It is well proven that nanomaterial has characteristics which are totally different in comparison to similar compounds with bigger dimension. Size, shape and surface area are criteria causing these differences. Understanding physical and chemical aspects of nanomaterial is important in environmental and human toxicology. In this study nanosilver with 70 nanometer size was consumed by mice with 25, 50, 100 and 200 milligram in 28 days. The control group consumes physiologic serum instead. After 28 days to assess morphological and pathological, blood samples were obtained from ocular eye, then autopsy was done in order to evaluate enzymatic change rate of BILI ALP -SGOT-SGPT and histopathologic effects of nanosilver on lung and liver tissue. In 100 and 200 milligram doses significant changes observed in hepatocytes and lung cells. The lung tissue in experimental group was morphologically more massive and more achromic in comparison to control group which showed emphysema. From histopathology point of view, emphysema occurs in alveoli. It means that the membrane between cells disappeared. Interstitial Pneumonia was observed in some parts of lung. Biochemical tests revealed that BILI rate had no significant change. However the increase of aminotransferases and alkaline phosphatase due to nanosilver treatment determined lung cells and hepatocyte damage because of the usage of nanosilver.

KEYWORDS:

Histopathology, liver tissue, lung tissue, alkaline phosphatase, toxicology

INTRODUCTION

It is well proven that nanomaterial has characteristics which are totally different in comparison to similar compounds with bigger dameters. Size, shape and surface area are criteria causing these differences [1]. Although, the tiny size of nanosilver is the main cause of its characteristics; it can jeopardize the health of living organisms. In other words, size and surface changes of nanomaterial has a critical role in its toxicity [2]. Understanding physical and chemical aspects of nanomaterial is important in environmental and human toxicology [3]. Evaluating and assessing dangerous and toxic effects of nanomaterial can assist us to enface them.

Nowadays nanosilver agents frequently use because of their hydroxide ion production ability (which in turn is due to catalytic reaction) and Ag-S band production in bacterial membrane [4,5]. Hence, nanomaterial used in medical and hygiene equipment and also in packaging industry can enter into lung, liver and blood [6]. Current study was done to evaluate the effect of nanomaterial on rats by gavage feeding. In addition, the aggregation of nanosilver in liver and lung tissues and also changes in SGOT-SGPT-ALP-BILI enzymes were assessed. After feeding rats by nanomaterial, samples were obtained in determined times and enzymatic and cellular changes were assessed. Mitochondria are the most sensitive cell organelles against nanomaterial. Main place for aggregation of nanosilver is liver.

Materials and Methods

In this research 25, 50,100,200 milligram dozes of nanosilver with 70 nanometer size were given to rats during 28 days by oral consumption. 32 rats in 4 groups each consists of 8 members were our experimental groups and 1 group was used as control group feeded by physiological serum. After day 28, blood samples were taken from the ocular of eyes and autopsy was done.

Results and Discussion

In liver tissue (see figure 1) concentration of 25 milligram (N2) caused the existence of inflammation neutrophil and hepatocyte inflation around central vein; however, observed fibrosis was less than higher doses. In 50 milligram concentration (N3) fatty change was observed in liver tissue. In port space and in parenchyma lymphocytes were observed. Fibrosis also observed which consisted of sedimented collagen and cellular inflation existed in hepatocytes surrounding central vein. In concentration of 100 milligram (N4), again there was little fibrosis. More lymphocytes were observed in port space. 200 milligram concentration (N5) showed more fibrosis than the other concentrations. Lung tissue (see figure 2) was morphologically more massive and more achromic than control group which showed lung emphysema. From histopathological point, emphysema observed in lung alveoli which means that the cell membranes were ruined. N2 concentration caused interstitial pneumonia in some parts of lung. N3concentration emphysema led to lung alveoli. N4 and N5 concentration resulted in alveoli emphysema and pneumonia. Statistical analysis of biochemical markers (figure 3 and 4) showed BILI rate had no significant changes. In current research increase of aminotransferases and alkaline phosphatase level revealed hepatocyte damage by nanosilver, because these are cellular enzymes and they are traceable in serum when the cells were injured [7]. In addition to be a toxin against P450 in liver and kidney, nanosilver is also a cancerogenic material in long-term which is metabolized by enzymes in liver microzomes. This leads to damage of hepatocytes, apoptosis and eventually necrosis. Long-time contamination with nanosilver causes liverfibrosis or liver cancer.

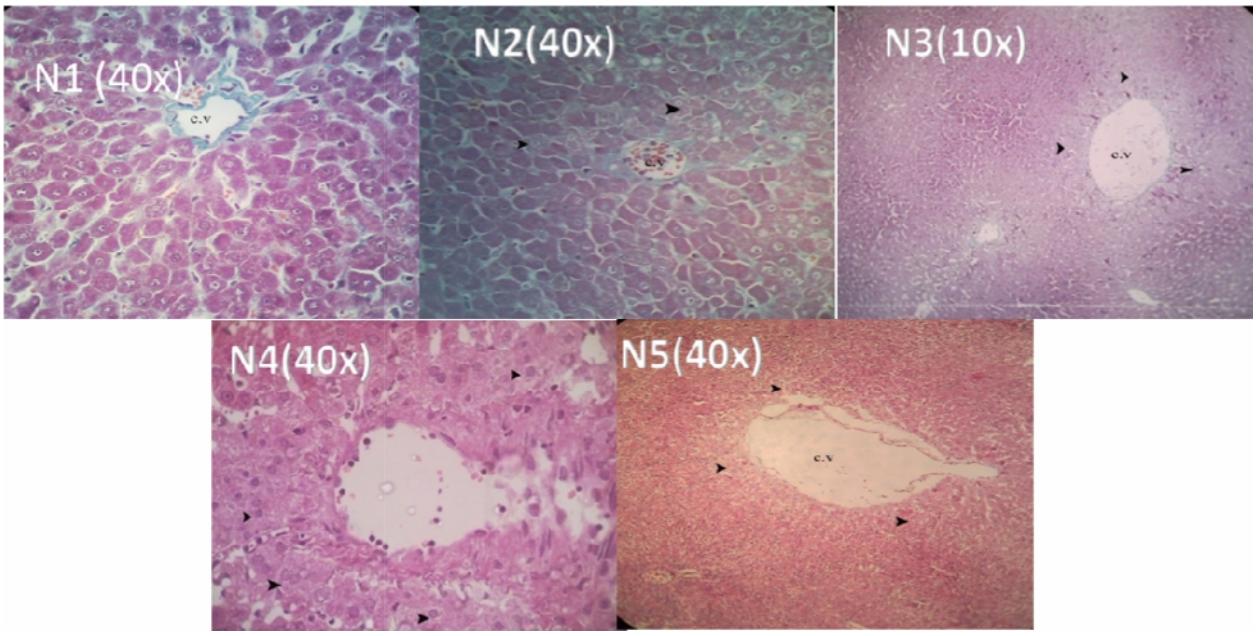

Figure 1. N1: control group's liver tissue; N2: chronic cellular inflation around central vein; N3: chronic hepatocyte inflation around central vein; N4, N5: acute hepatocyte inflation around central vein

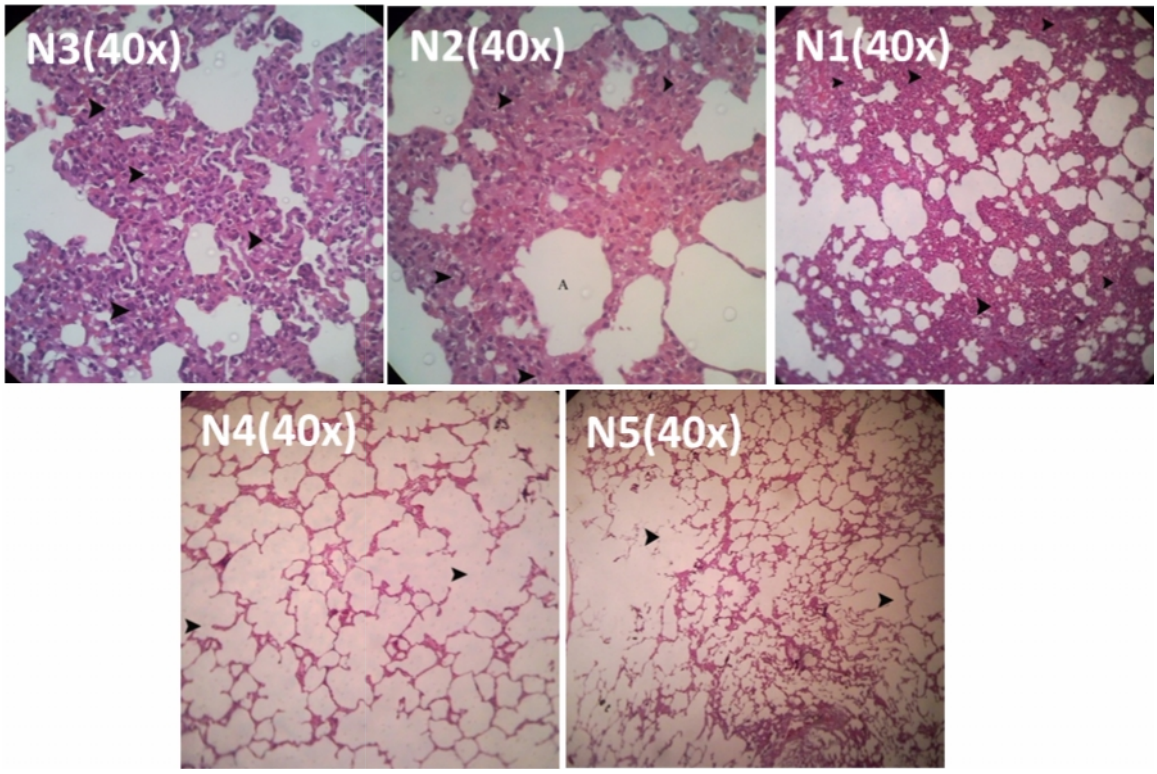

Figure2. Lung tissue. N1: control group's lung tissue; N2, N3: interstitial pneumonia; N4, N5: emphysema in alveolar duct

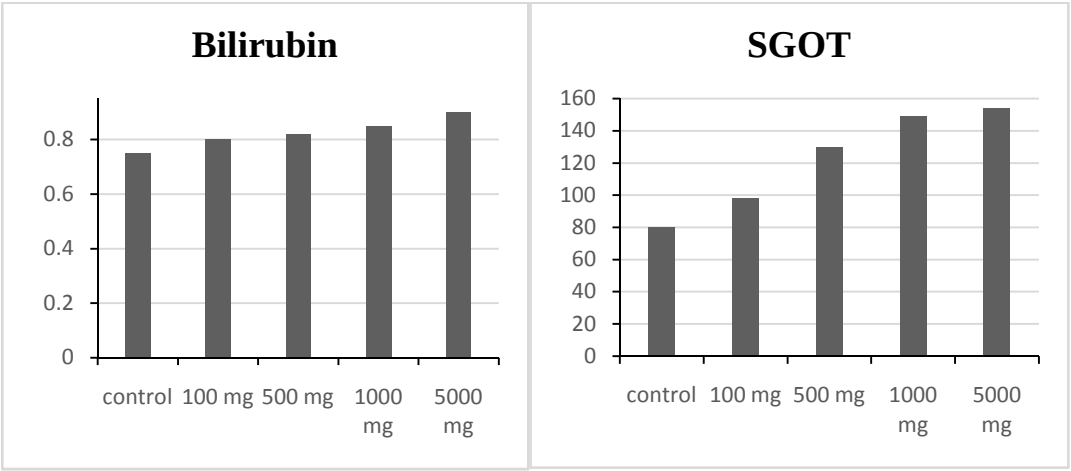

Figure 3. Alterations in BILI-SGOT

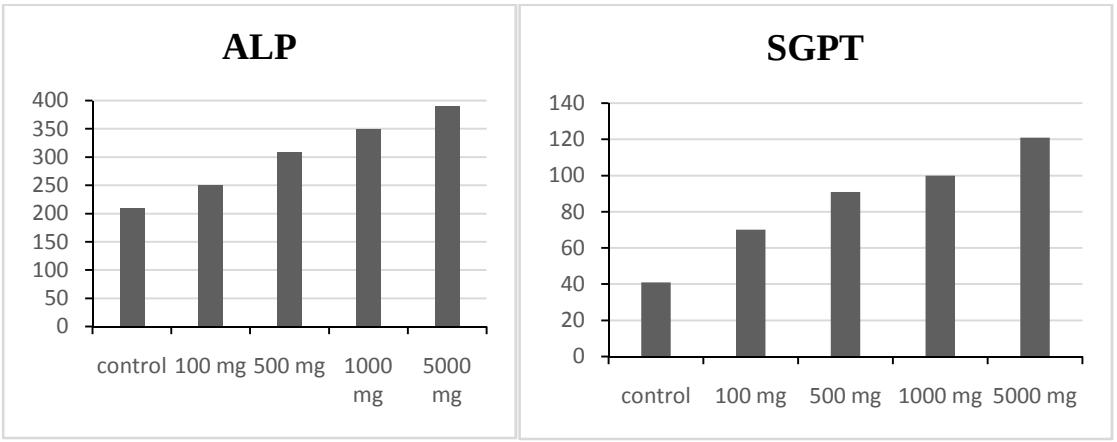

Figure 4. Changes in ALP and SGPT

Conclusion

Nanomaterial enters the body from 3 ways. In digestive path, nanomaterial was intake along the intestine and aggregates in liver, spleen and testicles. Aggregation in tissues leads to cellular intake. In this case final aggregation site is lysosome or cytoplasm. Elimination of nanomaterial by hepatic macrophages causes the production of free radical due to oxidative steps of phagocytosis [8]. Low dose caused the present of neutrophil inflammation and hepatocyte inflation around central vein, but observed fibrosis is less than higher doses. In high concentration fatty change and in port space in parenchyma lymphocytes were observed and also fibrosis which was the sedimentation of collagen and cellular inflammation of hepatocytes were exhibited around the central vein. Lung tissue in nanosilver feeded rats was morphologically more massive and more achromic than control group which showed lung emphysema. Histopathologically emphysema

observed in lung alveoli which means that the cell membranes were ruined. In some parts of lung interstitial pneumonia was seen. Biochemically the results of experiments showed that the rate of BILI had no significant change. On the other hand, increase of aminotransferases and alkaline phosphatases due to treatment with nanosilver characterizes lung and liver cell damages, because these enzymes are located within cells and they enter the serum from injured cells. Nanosilver is a toxin for liver and lung tissues and also during long time it is cancerogenic. Because of its antibacterial characteristics, nanosilver is currently used widely in pharmaceutical, hygiene and nutritional industries. Therefore we recommend that more research should be carried out on the safty of nanosilver.

References

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