Effect of hyperbaric oxygen on Zinc deficiency rats

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Summary

To examine the effect of hyperbaric oxygen on Zinc (Zn) deficient rats, we investigate the morphological changes and expression of inducible nitric oxide synthese (iNOS) and ED1 in lung of rats after hyperbaric oxygen exposure. Seven-weeks-old male Spreague-Dawley (SD) rats were fed on diets supplemented 0 or 0.02% Zn for 60 days. They were exposed to 2.8 ata for 60 minutes by 100% oxygen and examined pathohistological changes and expression of iNOS and ED1 in lung. In 0% Zn diet, sever morphological changes and strong expression of iNOS and ED1 were found. However, number of (WBC) did not increase in 0% Zn diet. These results suggest that the hyperbaric oxygen stress causes sever lung injury in Zn deficiency compared to normal condition.

Introduction

Hyperbaric oxygen therapy (HBO) is used in many diseases, for example, gas gangrene, carbon monoxide poisoning, cerebral edema and decompression sickness[1]. HBO is very effective method to raise partial oxygen pressure in the damaged tissues rapidly. However, HBO cause sometimes oxygen poisoning. There are two types of oxygen toxicity. One is pulmonary effects include damage to both capillaries and alveolar epithelium. Inhaling pure oxygen for long time at 1 ata (atmospheres absolute) causes edema and inflammation in lung.

Zn is a trace element essential for normal biological functions. In Zn deficiency, opportunistic infection and changes on the immune system are seen[2].

This study is to examine the effect of the exposure to hyperbaric oxygen on the Zn deficiency rat.

Methods

In the present study, two special diets were prepared (Oriental Kobo, Japan); a Zn deficient diet (0%Zn diet) and a standard diet (0.02%Zn diet, control). The 0% Zn feed, confirmed to contain 50 micro-g Zn/100g feed by atomic absorption spectrophotometry, was prepared by excluding Zn during the manufacturing process. The 0.02% Zn diet was prepared by adding

20mg Zn to 100g of the 0% Zn feed. As a result, the composition of these two special diets differed only in Zn content.

Seven week-old SD male rats had as libitum access to distilled water and were given either the 0% or 0.02%

Sixty days after initiating the special diet, rats were exposed to 2.8 ata for 60 minutes by 100% oxygen in a small hyperbaric chamber. And then they were decompressed to 1 ata. After decompression, the rat was removed from the chamber and observed their condition for 60 minutes. After 18 hours from surfacing, the excising lungs and

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blood sampling were carried out under light etherisation. The excised lungs were embedded on paraffin and sliced for examination of pathohistological changes (HE staining) and expression of inducible nitric oxide synthase (iNOS) and ED1 by immunohistochemistry

Results and Discussion

During the observation period, no abnormalities in appearance were noted on rats on the 0.02% Zn diet (n=3). On the other hand rats on the 0% Zn diet (n=2) could not move and gasped for breath. They recovered within 60 minutes form surfacing. Hypertrophy of alveolar and edema were seen in all cases. These changes were remarkably in the 0% more than0.02% Zn diet (fig.1). In 0%Zn diet, strong expression of iNOS and ED1 in epithelium of bronchioles were confirmed (fig2, 3). Number of white blood cells (WBC) was 10966.7/mm^3 (average) in 0.02% Zn diet and 5700/mm^3 (average) in 0% Zn diet.

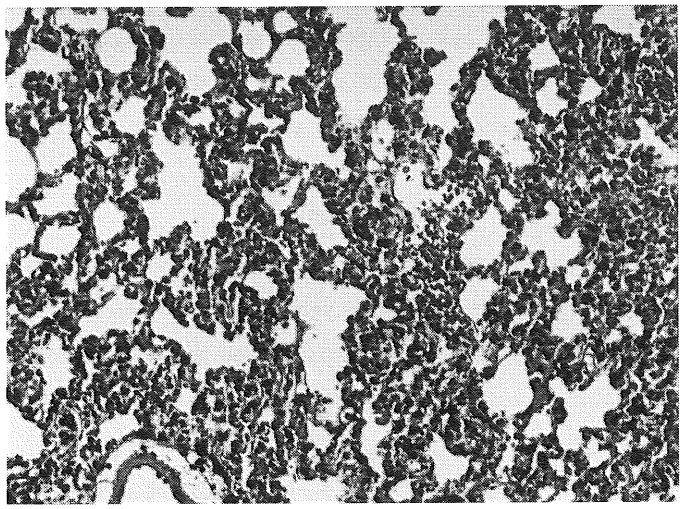

Fig. 1 Photomicrograph of the lung of Zn-deficient rat after surfacing from 2.8 ata oxygen.

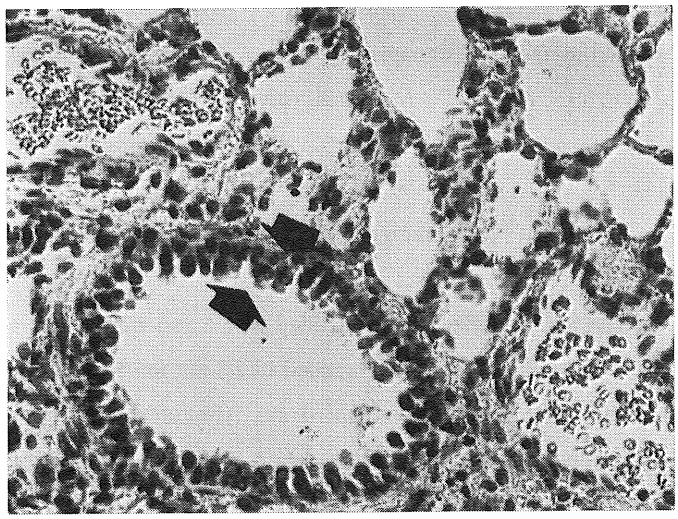

Fig. 2 Photomicrograph of the iNOS expression in the 0% Zn diet.

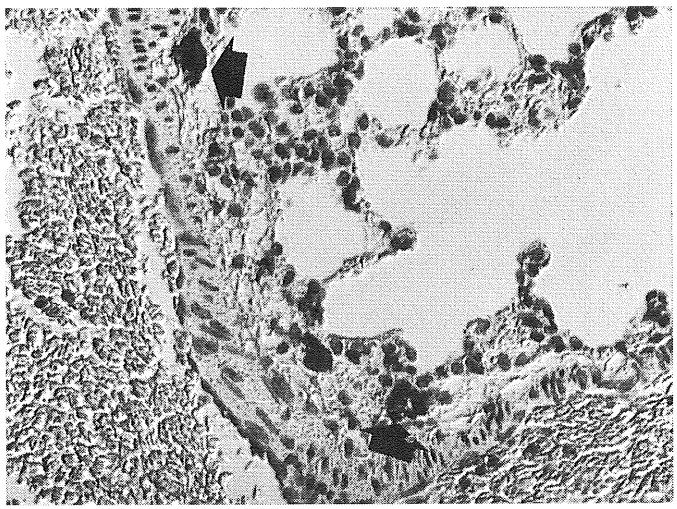

Fig. 3 Photomicrograph of the ED1 expression in the 0% Zn diet.

Pure oxygen breathing at 1 ata for long period causes inflammation of lung (Lorain- Smith effect). And inhalation of high partial pressure oxygen causes oxygen poisoning of the central nervous system (Paul Bert effect). We cannot decide the cause of these symptoms, which were found in 0% Zn diet after surfacing. However, this suggests that oxygen poisoning occurred easily in the rats fed on 0% Zn more than 0.02%. Thus our findings suggest that Zn deficiency reduce tolerance to the hyperbaric oxygen stress. This study indicated that the hyperbaric oxygen stress occurs sever lung injury in Zn deficiency.

The results of iNOS and ED1 expression indicates that lung inflammation in 0% Zn diet is severely compared to 0.02% Zn diet. However, WBC did not increase in 0% Zn diet. Many studies reported that Zn deficiency cause reduction of immune function associated with atrophy of thymus. Our previous study demonstrated that these changes were caused by acceleration of apoptosis[3]. Moreover, we also clarified Zn deficiency lead to increase of apoptosis in many other tissues. As a result, we assume that number of WBC could not increase in 0% Zn diet.

In conclusion, our results suggest that the exposure to hyperbaric oxygen have great influence on the body in Zn deficiency than normal condition and Zn deficiency effects the process of inflammation due to hyperbaric oxygen exposure. This point should pay attention when HBO is carried out to patients under Zn deficiency.

Acknowledgements

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