THE IMPACT OF HAZE ON THE LUNG OF HORSES

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Introduction

Although there has been claim that air pollution in any form may affect the respiratory system, conclusive evidence is lacking. Furthermore constituents of air pollutant in different parts of the world may vary and thus leading to different manifestation of clinical signs or lesions. Malaysia has experienced several episodes of haze and the 1997 episode was the worst. The aim of the study was to assess the effect of haze on the lung using the horse as a model with the following objectives:-to study the development of lesions in the lung during different stages of haze and to correlate the relationship between severity of lesions with the concentration of PM 10.

Materials and Methods

A total of 12, 5 and 12 culled horses killed in July , August and September 1997 respectively were used in this study. The period between July to August was designated as the "pre-haze" period while that of September as the "peak haze". The horses were killed by intravenous injection of pentobarbitone and sample of the lungs were collected immediately after death. The selected lobes were the primary bronchi of the left apical lobe (LAL), left diaphragmatic lobe (LDL), right apical lobe (RAL) and the right diaphragmatic lobe (RDL). The tissues taken were immediately fixed in 10% buffered formalin and processed in the routine manner and stained with Haematoxylin and Eosin (H&E). The lung sections were examined under a compound microscope at 100 magnification and the lesions observed were scored on a scale of + to ++++. The scoring was made based on the severity of the following lesions: congestion, oedema, alveolitis and bronchitis/bronchiolitis. The scores were then averaged upon the number of animals during the stipulated period and tabulated.

Results and Discussion

Lesions pertaining to oedema and congestion were not analysed (although scored) since they could be attributed by the effect of barbiturate. Lesions pertaining to alveolitis and bronchitis/bronchioliotis exhibited accumulation of cells mainly lymphocytes and macrophages. Some of the cells were in the from of aggregates (possibly bronchiole-associated lympoid tissues) while others in no specific manner. Only in one case, i.e., during peak haze there was also fibrinous exudate in the bronchioles and alveolar spaces. It is clearly evident that in most instances, the lesion scores of the lung during peak haze surpassed those before haze signifying that haze has great impact on the development of alveolitis and bronchitis/bronchiolitis. Particulates such as PM 10 or PM 2.5 have been known to cause or aggravate the development of the mentioned lesions and reduce the defence system to combat infections. Indirectly, as the haze worsen, the severity of the lesions increased indicating a direct correlation between severity and concentration of partiuclates. Furthermore, as for PM 2.5 or less, the ability of the lung to entrap and remove particulate of this size is minimal. The lobes chosen in this study (especially the apical lobes) are those whereby particles can settle directly by means of inertia, gravity and diffusion. As mentioned earlier, the particles themselves apart from being natural irritants can also incite immmunosuppresion, which in turn lead to a decrease in pulmonary defence mechanism. In short, the lung became more vulnerable to secondary infections, which under normal circumstances is well prevented.

Conclusions

The findings from this study showed that an increase in the concentration of particulate matter in air can lead to a decrease in pulmonary defence mechanism and an increase in the development of alveolitis and bronchitis/bronchiolitis.

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