Austin Journal of Veterinary Science & Animal Husbandry

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Austin Publishing Group

Research Article

Pneumonia in a Snail *Cornu aspersum* (Gastropoda, Pulmonata)

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Received: May 07, 2015; **Accepted:** December 28, 2015; **Published:** December 31, 2015

Abstract

The larger part of the shell in a garden snail (*Cornu aspersum*) had been eaten away by mice, thus exposing the lung. This has most probably, affected the main pulmonary blood vessels running over the dorsal surface of the lung. The alveolar walls had atrophied, mimicking alveolar emphysema. Many blood capillaries were open. In consequence blood had floated in the alveolar spaces. Gas exchange was severely hampered. Inflammatory reactions were minimal.

Introduction

The garden snail (*Cornu aspersum*; earlier *Helix aspersa*) has a spherical shape with the larger diameter of the shell to range from 25 to 40mm and the height from 25 to 35mm. It originates from the Mediterranean region where the snails have been cultivated as food by the romans. During the conquest of Europe roman soldiers carried the snails as a start for their helicicultures. It is a very adaptive species and has since spontaneously invaded the Western part of Europe up to Sweden and even into Finland [1]. In addition deliberate and accidental anthropochorus activity has led to a worldwide spread. It feeds on a wide variety of crops and ornamentals and therefore is locally considered as a pest.

The bigger variety of the garden snail (*C. a. magna*) is almost equal in size to the edible snail (*H. pomatia*) in addition it lays more eggs. These two characteristics make the garden snail a preferred object for cultivation as a delicacy. Research has revealed a number of bacteria and helminths inhabiting the intestine and the hepatopancreas [2]. In breeding systems pathogenic bacteria have been isolated [3]. The lung mite *Riccardoella limacum* is widely spread and may damage the lungs [4]. The garden snail is also known as an intermediate host for Trematodes and Nematodes which have zoonotic potencies [5].

To the best of my knowledge there is no information about pathology of the respiratory system in the garden snail. This prompted me to communicate a case of pneumonia in a garden snail.

Materials and Methods

The animal was humanely euthanized by intrahemocoelic injection of 0,1ml T61 (Bayer). It was fixed in toto in 10% neutral formalin over 72hours. Decalcification was in formic acid (Kristensen's solution). The procedure reduced the shell to a thin film. The specimen was cut longitudinally into 4 slides of ± 3mm thickness. These were embedded in paraffin. For histology slides were cut at 4µm, stained with H&E and mounted in Eukitt (Sigma). For comparison, a normal lung was derived from an archived *C. aspersum*, which had been processed identically. The nomenclature of anatomical structures was found to be incomplete. Thus I choose to use names derived from comparable structures and pathologies in mammals.

The patient

The specimen was found around noon, fully exposed in a private garden in the Netherlands. It was seriously damaged. The periostacum (the shell) failed from the top down to about halfway. The exposed surface was smooth, without signs of bites of ruptures. Respiration was laborious. At expiration greyish mucous fluid appeared in the pneumostoma (the respiratory opening). It made an impression as though the animal was in major problems.

Histopathology

The lung of the patient showed extensive lesions. Blood vessels bordering the dorsal periphery of the lung were dilated and filled with blood. The alveolar walls carrying the capillaries often were markedly reduced in number, leaving larger spaces. The remaining alveolar walls appeared as widely separated, small, short projections originating from the periphery of the lung. Only a few crossed over to the other side of the lung. A loss of at least 80 % of the alveolar walls was estimated. The alveoli were filled with vaguely eosinophilic clouds of proteinaceous material. This material revealed a fine network of short threads. Knobs, indicating fibrin fibres, were not recognized. Widely scattered globules surrounded the clouds. The larger proportion of the globules had a roundish shape, were homogenous and markedly eosinophilic. The diameter varied between ± 3 and 12µm. Some globules were larger. The latter showed an interrupted rim and a fragmented centre as though they were disintegrating. Small groups of eosinophilic haemocytes had accumulated between the propria of the alveolar walls and the blood capillaries

Blood vessels along the periphery of the lung were dilated. The alveolar walls were seriously damaged. In comparison to the normal situation Figure 1, they were locally broadened by a central

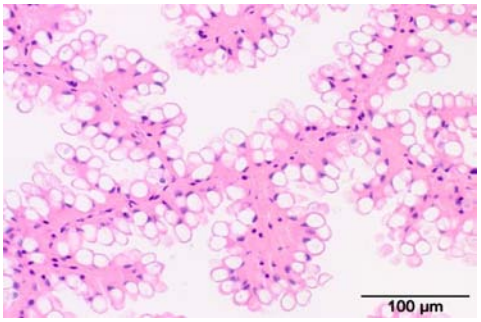

Figure 1: *Cornu aspersum* Lung normal H&E.

Citation: Zwart P. Pneumonia in a Snail *Cornu aspersum* (Gastropoda, Pulmonata). Austin J Vet Sci & Anim Husb. 2015; 2(3): 1018.

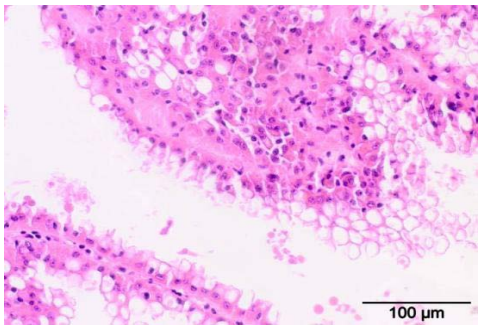

Figure 2: *Cornu aspersum*. Infiltrate of eosinophilic haemocytes H&E.

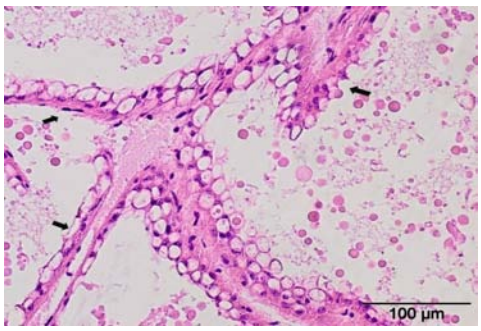

Figure 3: *Cornu aspersum*, Lung. Alveolar walls thickened. Proteinaceous material in alveolus plus many proteinaceous globules. Respiratory blood capillaries damaged (arrows) H&E.

accumulation of homogenous eosinophilic material identical to the blood in the atrium of the heart. Most probably these were dilatations of blood vessels. Locally, the blood capillaries were lifted from the alveolar wall by accumulations of round to oval haemocytes (Figure 2). Necrosis was absent in or around these accumulations.

The blood capillaries in the lung were seriously damaged. Often, the walls at the alveolar side were open. Eosinophilic globules, identical to these in the alveoli, were often present in the remains of the blood capillaries (Figure 3).

Discussion

The lung in Pulmonates is described as a bag-like cavity with blood vessels disposed as a network in the roof ((Figure 4), [6]). In origin the pulmonary sac is formed by the fusion of the mantle edges along the animal's back. Air enters or leaves the sac through a contractile opening termed the pneumostome. The roof of the sac contains a network of blood sinuses. Alveolar walls, implanted on the periphery of the lung sac, form a network. Respiratory blood capillaries line the alveolar walls [7]. In my experience, the blood capillaries appear as being empty. This is in agreement with the fact that the haemocyanin – binding the oxygen - is blue when it is oxygenated and colourless when it is not [8].

The specimen endured an extensive trauma of the periostacum. Such traumata are said to be the result of gnawing by mice (*Mus musculus*) or shrew-mice (*Crocidura sp*). These animals act very precisely and restrict themselves to the calcified shell [9]. It may be surmised that removing a larger part of the shell has resulted in drying of the exposed surface of the lung. In consequence, this may

Figure 4: *Cornu aspersum* Anatomical situation of the lung.

have had a direct effect on the blood vessels running over the dorsal surface of the lung. In addition a surmised loss of fluid by evaporation will have had effect on the homeostasis of the patient.

The changes in the lung of the patient i.e. a marked atrophy of the walls of alveoli resulted in wide (alveolar) spaces. This could eventually be indicated as extensive emphysema. However, in this case, it was combined with an accumulation of a protein containing fluid. It may be suggested that the fluid was blood, floating from the damaged, open respiratory capillaries. This process is quite different from a non-cardiogenic pulmonary edema in mammals where a transudate accumulates in the alveoli. It may be related here that the function of the heart in the edible snail (*Helix pomatia*) is independent of the oxygen consumption [10]. This implies that the changes in the lung may not have interacted with the heart-beats and the circulation of the blood.

Inspiration and expiration is by contraction and relaxation of ventrally situated arched diaphragm [11]. The laborious respiratory movements of the patient indicate that this mechanism was intact.

Conclusion

The pathogenesis of the pulmonary changes in this snail may be regarded a cascade of events.

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Austin J Vet Sci & Anim Husb - Volume 2 Issue 3 - 2015
ISSN: 2472-3371 | www.austinpublishinggroup.com

Citation: Zwart P. Pneumonia in a Snail *Cornu aspersum* (Gastropoda, Pulmonata). Austin J Vet Sci & Anim Husb. 2015; 2(3): 1018.