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This is the author's manuscript

Original Citation:

Availability:
This version is available http://hdl.handle.net/2318/112767 since 2016-07-20T10:14:01Z

Published version:
DOI:10.1177/1040638712442881

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(Article begins on next page)
This is the author's final version of the contribution published as:


The publisher's version is available at:

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http://hdl.handle.net/2318/112767
Brief Research Reports

Endogenous lipid (cholesterol) pneumonia in three captive Siberian Tigers (*Panthera tigris altaica*)

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Abstract

During 2009 to 2011 seven Siberian Tigers (*Panthera tigris altaica*) aged between two and fourteen years from the Safaripark of Pombia were referred for necropsy to the Department of Animal Pathology of the University of Turin (Italy). Three tigers, aged ten (two animals) and fourteen years, had multifocal, irregularly distributed, white, soft, subpleural, 3 mm nodules scattered throughout the lungs. Histologically there were a marked infiltration of macrophages, with foamy cytoplasm, and multinucleate giant cells interspersed with numerous clusters of cholesterol clefts. A mild lymphocytic infiltration was localized around the lesion. These findings were consistent with endogenous lipid pneumonia. It was considered an incidental finding of no clinical significance.

Keywords: cholesterol pneumonia, endogenous lipid pneumonia, *Panthera tigris altaica*, Siberian Tiger
Lipid pneumonia is a term used to describe the presence of lipid in the lungs. Synonyms for this condition include cholesterol pneumonia, lipoid pneumonia, paraffinoma and alveolar histiocytosis.

Lipid pneumonia can be subdivided depending on the source of the lipids. Exogenous lipid pneumonia occurs following aspiration or inhalation of mineral, vegetable, or animal oils and has been reported in cattle and cats following forced administration of mineral oils.

Endogenous lipid pneumonia occurs when pulmonary cell membranes degenerate causing release of cholesterol and other lipids into the alveolar space. The suspected pathogenesis of endogenous lipid pneumonia is related to pulmonary injury which causes proliferation of alveolar type II cells, resulting in overproduction of cholesterol-containing surfactant that enters the alveoli and is phagocytosed by macrophages. These macrophages appear as clusters of foamy macrophages associated with cholesterol clefts.

The primary cause may be proximal airway obstruction, inhalation of irritating dust particles, pulmonary parasitism or disturbance of lipid metabolism.

Experimentally, laboratory animals on protein-deficient cirrhogenic and pantothenic-acid-deficient diets and those that have had hypophysectomy also have been shown to have an increased prevalence of endogenous lipid pneumonia.

The condition is reported frequently in rats and less frequently in other species such as mice, cats, human beings and dogs. It has also been described in wild animals such as Raccoons, Opossums, Genet, Foxes, Llama and Shrews.

Clinical signs, if present, are described as nonspecific, such as lethargy, anorexia, and weight loss, or signs of respiratory tract disease. No report is available in wild felids.

During the years 2009 to 2011 seven Siberian Tigers (Panthera tigris altaica) aged between 2 and 14 years were referred for necropsy examination to the Department of Animal
Pathology of the University of Turin (Italy) from the Safaripark of Pombia to ascertain the causes of death.

Tissue samples for histological examination were fixed in 10% neutral buffered formalin (pH7), wax-embedded sectioned at 4-µm using a microtome\(^a\), and stained with haematoxylin and eosin. Other samples were frozen in OCT, 4-µm section were cut using a cryostat\(^b\) and stained with Sudan III. All tissues were examined by light microscopy\(^c\).

At necropsy three out of seven tigers, whose cause of death was in two ten-year-old animals renal failure, and in one fourteen-year-old animal a pyloric obstruction, showed multifocal, irregularly distributed, white, soft, mainly subpleural, 3 mm nodules scattered throughout the lungs (fig. 1). They were most prominent on the dorsal regions. On cut section the plaques appeared to be solid and white, and located in the subpleural regions.

Histologically there was a marked infiltration of macrophages, with foamy cytoplasm, and multinucleate giant cells interspersed with numerous clusters of cytoplasmic clefts that were interpreted as outlines of cholesterol crystals (fig. 2). Localized around the lesions there was a mild lymphocytic infiltration. The majority of the lesions were located under the pleural surface. The same lesions were present to a lesser extension within the parenchyma, usually in the peribronchial or periarteriolar regions. Sudan III staining revealed red-stained fat-storing cells scattered in the lesions. The outlines and cholesterol crystals remained evident and not dyed (fig. 3). These findings were consistent with a diagnosis of endogenous lipid pneumonia as the necropsy of the animals included in the present study did not revealed any pulmonary obstruction or parasitism. Furthermore, aspiration or inhalation of mineral, vegetable, or animal oils due to oral administration of oil based drugs or substances were excluded. No pulmonary clinical signs or other macroscopic or histological pulmonary lesions were present. Moreover there were no cardiac changes compatible with a cor pulmonale. While the cause of the endogenous lipid pneumonia in the tigers reported here is
not known, it seems reasonable to suggest that it could be associated with the inhalation of
irritating dust particles in the animal enclosures open to vehicular traffic of visitors,
however in our cases no additional histologic findings were detected to support this
hypothesis. A genetic predisposition, as described in humans, cannot be completely ruled
out in these tigers.
Endogenous lipid pneumonia is often an incidental and not life-threatening finding, in
absence of clinical signs or with non-specific symptoms, radiographic features, and
haematologic and serum biochemical findings\(^\text{10}\).
To the author’s best knowledge this is the first report of endogenous lipid pneumonia in
tigers. The endogenous lipid pneumonia in these tigers was considered an incidental post-
mortem finding of no clinical significance.

Acknowledgements
The authors gratefully acknowledge the Centro di Referenza di Patologia Comparata “Bruno
Maria Zaini”, Italy

Declaration of conflicting interests
The authors declared no potential conflicts of interest with respect to the research
authorship, and/or publication of this article.

Funding
The authors received no financial support for the research, authorship, and/or publication of
this article.

\(^a\) Leica Microsystems, Wetzlar, Germany
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Legend for Figures

Fig. 1: lung, Siberian Tiger: multifocal, irregularly distributed, whitish, soft, subpleural nodules.
Fig. 2: lung, Siberian Tiger: infiltration of macrophages with foamy cytoplasm, and multinucleate giant cells, interspersed with numerous clusters of cytoplasmic cholesterol clefts (HE stain; 200X) Bar = 50 μm.

Fig. 3: lung, Siberian Tiger: diffusely red-stained fat-storing cells with outlines of cytoplasmic cholesterol crystals (Sudan III stain; 400X) Bar = 25 μm.