

LIPOID PNEUMONIA IN THE RED FOX (*Vulpes vulpes*)

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Abstract - Eight red foxes (*Vulpes vulpes*) out of 182 examined animals, either found dead or shot in Cuneo and Vercelli provinces (Italy) during 1993, showed pulmonary lesions characteristic of lipid pneumonia. Histological examination showed an infiltration by vacuolated macrophages, containing sudanophilic material. Associated lesions were represented by bronchitis, localized emphysema and peribronchiolar aggregates of lymphoid nodules. The concomitant localized inflammatory process might be responsible for post-obstructive lipid pneumonia.

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1. Introduction

Although the red fox (*Vulpes vulpes*) is the most abundant and widely distributed predator species in Italy, little is known about its pulmonary pathology.

Lipoid pneumonia, a selected lesion of the lung, characterized by subpleural white plaques consisting of accumulations of vacuolated macrophages, has been recognized in cats (Dungworth 1985; Chalifoux and Morin 1987), dogs and mink (Corcoran *et al.* 1992), opossums (Brown 1988), rats (Beaver *et al.* 1963; Corrin and King 1969; Flodh *et al.* 1974; Weller 1985), mice (Emi *et al.* 1985) and man (Verbeken *et al.* 1989; Wolfson *et al.* 1989), though it does not appear to have been reported in foxes.

The cause of the disease is still unclear, but it is probably consequent to bronchial obstruction, inhalation of dust or disturbance of lipid metabolism (Brown, 1988).

Based on clinical and pathological findings, three types of lipid pneumonia have been described in wild and domestic species and in

man: exogenous, endogenous and post-obstructive lipid pneumonia (Corcoran *et al.* 1992).

This paper reports on several cases of lipid pneumonia found in red foxes, submitted during 1993 for pathological investigations at the Department of Animal Pathology of the University of Turin (Italy).

2. Materials and methods

Necropsies were performed on 182 red foxes either found dead or shot in Cuneo and Vercelli provinces (Italy) during 1993. Whole animals, usually refrigerated or frozen, were submitted. Sex, age, origin, date and diagnostic findings were recorded. Samples of tissues were fixed in 10% buffered formalin, paraffin embedded, cut at 4 μm , stained with haematoxylin and eosin, and observed by light microscopy. Part of the same tissues were snap frozen, cut at 4 μm and stained with Sudan IV for detection of lipids.

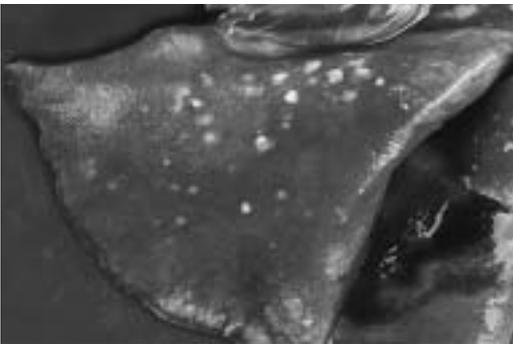


Fig. 1 - Red fox, lung, lipid pneumonia: subpleural plaques disseminated in the dorsal region of the diaphragmatic lobe

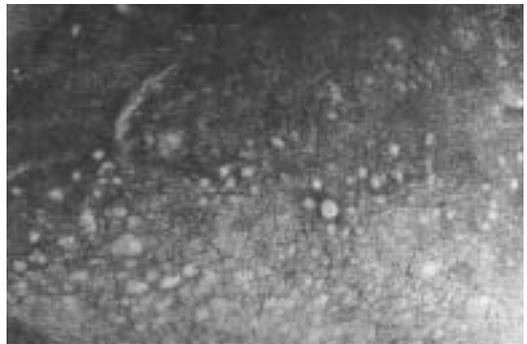


Fig. 2 - Red fox, lung: higher magnification of Fig. 1, showing multiple foci of lipid pneumonia and surrounding emphysema

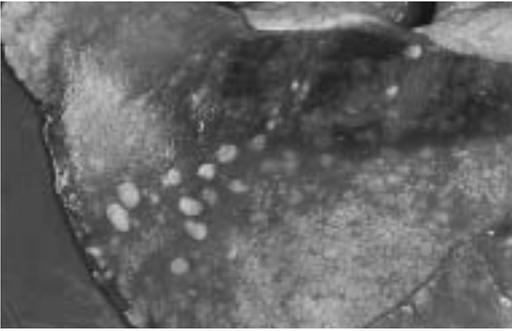


Fig. 3 - Red fox, lung, lipid pneumonia: subpleural plaques in the dorsal region of the diaphragmatic lobe, associated with local emphysema and inflammation of pulmonary parenchyma

3. Results

Out of 182 animals, 8 (4.4%) showed pulmonary lesions characterized by multiple subpleural plaques, disseminated or often coalescent, in the dorsal region of the diaphragmatic lobe (Fig. 1-3). The lesions appeared soft, raised under the pleural surface, white or grey in colour. The section surface of the plaques appeared solid, white or grey, surrounded by congested pulmonary parenchyma. Nodules, showing the same characteristics, were also disseminated in the cut surface of the diaphragmatic lobes in all specimens.

Histological examination of subpleural lesions showed a massive infiltration by vacuolated macrophages (Fig. 4, 5), often localized around bronchioles and vessels, causing disruption of the surrounding pulmonary parenchyma (Fig. 6, 7). Bronchiolar as well as alveolar lumina in affected areas appeared obliterated by the massive macrophagic infiltration. Lymphoid nodules and hypertrophy of vascular musculature were often present.

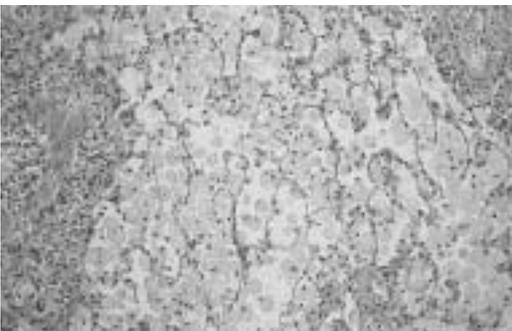


Fig. 5 - Red fox, lung, lipid pneumonia: accumulation of foamy macrophages in the deep pulmonary parenchyma (H. & E., x200)

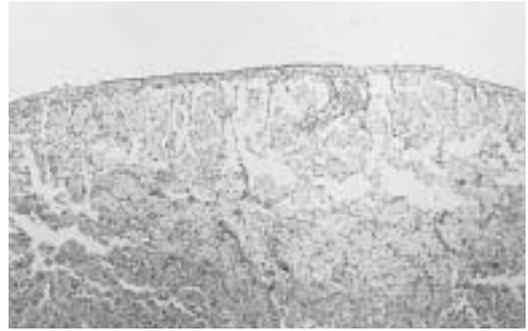


Fig. 4 - Red fox, lung, lipid pneumonia: subpleural plaque characterized by massive infiltration by foamy macrophages (H. & E., x100)

Frozen sections of the same pulmonary tissues, after staining with Sudan IV, showed sudanophilic material in macrophagic vacuoles (Fig. 8, 9). The surrounding pulmonary parenchyma showed foci of bronchitis, localized emphysema, and peribronchiolar and perivascular aggregates of lymphoid nodules associated with thickening of alveolar septa (Fig. 10, 11).

4. Conclusions

Lipoid pneumonia, previously reported in cat, dog, opossum, mink, rat, mouse and man, has never been observed in foxes; our report is therefore the first in this species.

Of the three types of lipid pneumonia until now described, only the exogenous form is consequent to inadvertent inhalation of lipidic substances (i.e. oil). In both endogenous and post-obstructive endogenous lipid pneumonia, the accumulated lipids are believed to be derived from endogenous sources, possibly in consequence of the obstruction of airways (e.g. by an inflammatory process) (Dungworth 1985;

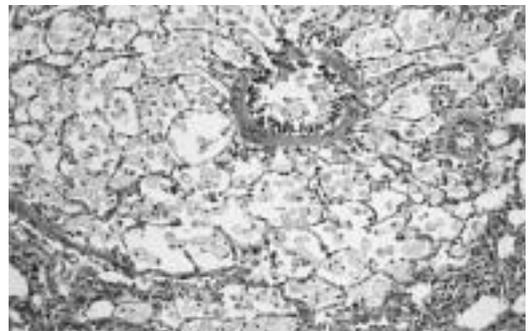


Fig. 6 - Red fox, lung, lipid pneumonia: peribronchiolar foamy macrophages (H. & E., x200)

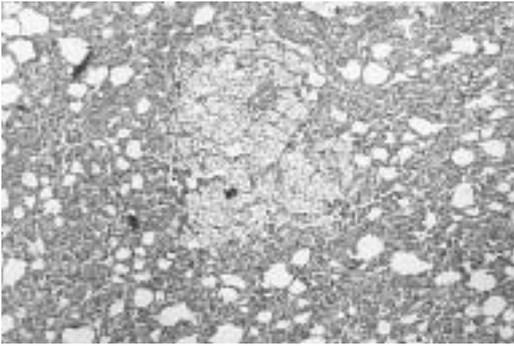


Fig. 7 - Red fox, lung, lipid pneumonia: a modest accumulation of macrophages surrounded by thickening of alveolar septa (H. & E., x100)

Weller 1985), exposure to dust (Corrin and King 1969; Lee *et al.* 1985) and vitamin deficient diets (Beaver *et al.* 1963).

Either production of large quantities of surfactant and subsequent phagocytosis by macrophages (Lee *et al.* 1985), or altered lipid metabolism (Flodh *et al.* 1974) may result in lipid accumulation in tissues.

The etiology of the cases described in the present study has not been fully elucidated, though a concomitant localized inflammatory process, which might be responsible for post-obstructive lipid pneumonia, has been observed. No signs of inhalation of fat substances were detected.

An interesting subject for further verification is represented by the rapid mobilization of body fat, as described in mink (Corcoran *et al.* 1992), where the lung could conceivably act as a route for excretion of lipids. Environmental factors (i.e. food availability) or concomitant diseases

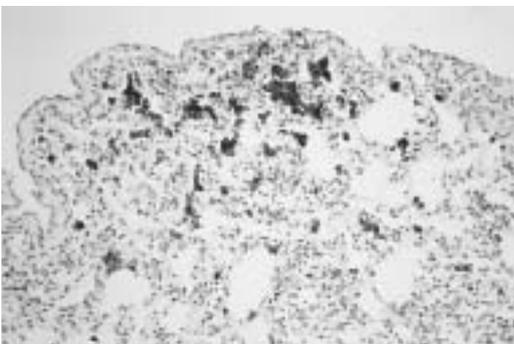


Fig. 8 - Red fox, lung, lipid pneumonia: sudanophilic material in macrophages of a subpleural lymphoid plaque (Sudan IV, x100).

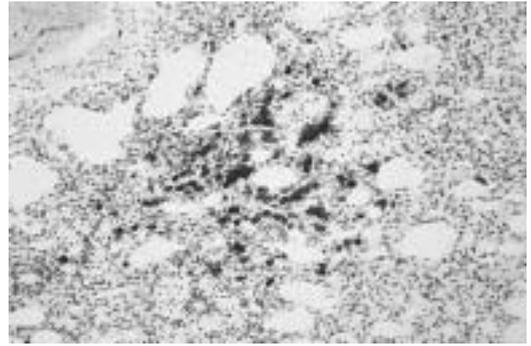


Fig. 9 - Red fox, lung, lipid pneumonia: sudanophilic material in macrophages of a deep nodule (Sudan IV, x100)

may therefore interact in the pathogenesis of the described lesions.

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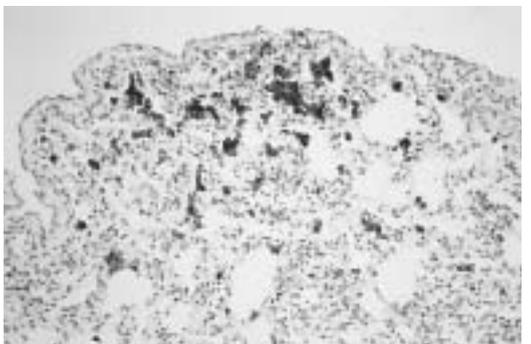


Fig. 10 - Red fox, lung: thickening of alveolar septa, lymphocytic infiltration and emphysema in the pulmonary parenchyma surrounding a focus of lipid pneumonia (H. & E., x100)

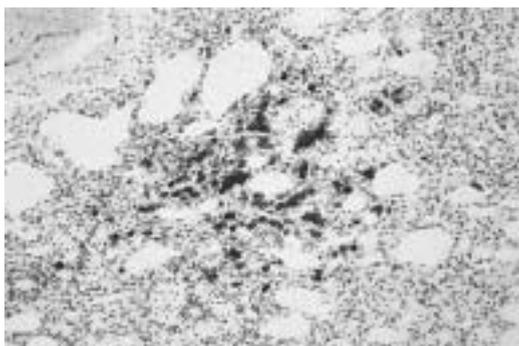


Fig. 11 - Red fox, lung: a bronchiolar lumen heavily infiltrated by inflammatory cells, surrounded by thickening of alveolar septa and emphysema, in the pulmonary parenchyma surrounding a focus of lipoid pneumonia (H. & E., x100).

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