Journal of Feline Medicine and Surgery (2004) **6**, 131–132

jfms

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Reviewed Letter

Human severe acute respiratory syndrome (SARS) and feline coronaviroses

A novel coronavirus (CoV) has been isolated from humans with severe acute respiratory syndrome (SARS) (Drosten et al., 2002; Ksiazek et al., 2003). Its pathogenic role, however, is debated, since SARS–CoVs have not been identified in all patients with SARS (Ksiazek et al., 2003). The SARS–CoV and the feline coronaviruses (FCoV) are phylogenetically quite distant (http://www.cdc.gov/ncidod/sars/pdf/proteinanalysis.pdf): nevertheless SARS and FCoV infections share many common features.

- Human CoVs and the feline enteric coronavirus (FECV) are low pathogenic and induce mild inflammations. In contrast, SARS and feline infectious peritonitis (FIP) are due to mutated and highly pathogenic CoVs (Vennema, 1999): the SARS–CoV probably arises from animal CoVs (Ksiazek et al., 2003); due to the above mentioned genetic differences between the SARS–CoV and the FCoVs it is very unlikely that cats might play a role in the SARS pathogenesis. However, also type II FCoVs probably originated by a recombination between type I FCoVs and canine CoVs (CcoV) (Herrewegh et al., 1998).
- SARS and FCoVs share a similar epidemiology (both the viruses rapidly spread within susceptible populations and the mortality rate is low) and similar clinico-pathological changes (lymphopenia and increased acute phase proteins are reported in both the diseases) (Drosten et al., 2002; Pedersen, 1995; Sparkes et al., 1994).
- Gross and microscopic pulmonary lesions are very similar: in particular alveolar damage, necrosis and macrophage infiltration can be detected both in SARS (Ksiazek et al., 2003) and FIP (Fig. 1).

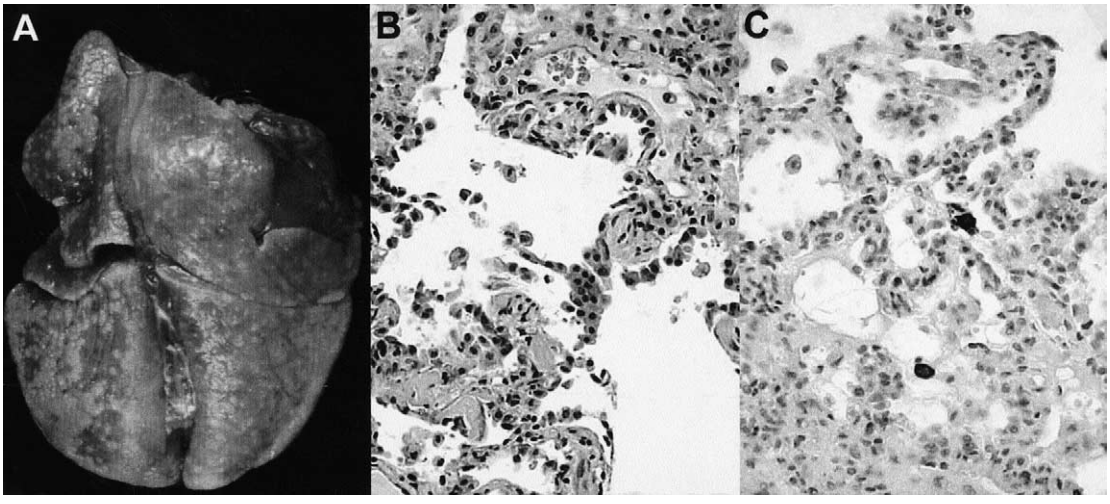

Figure 1 Lung from a cat with feline infectious peritonitis (FIP). A: multifocal to coalescing severe acute fibrinous pleuropneumonia; B: diffuse alveolar damage, with epithelial necrosis and sloughing and accumulation of macrophages and plasma cells. Hematoxylin and eosin, 100×; C: two FCoV-positive cells (macrophages). Immunohistochemistry with anti-FCoV monoclonal antibody, Mayer's hematoxylin counterstain, 100×.

1084-2756/04/$ - see front matter
doi:10.1016/j.jfms.2003.11.001

- Anti-FCoV antibodies identified SARS–CoVs in cell cultures, but no CoVs have been immunohistochemically detected within affected lung using different anti-CoV antibodies (Ksiazek et al., 2003). Interestingly, few FCoVs can be detected in large FIP lesions (Kipar et al., 1998; Paltrinieri et al., 1998).

Indeed, some differences between the two diseases are detectable, probably due to a different pathogenesis: although it cannot be excluded that the host reactions are responsible for SARS lesions, the SARS–CoV is probably directly cytopathic (Ksiazek et al., 2003). In contrast, FIP lesions are due to both type III and type IV hypersensitivity reactions (Paltrinieri et al., 1998): as a consequence, FIP is a systemic disease and usually affects young cats or cats with good body conditions, which have a more responsive immune system, while SARS is confined to the lung and is particularly severe in debilitated patients; during FIP the immune reaction induces typical electrophoretic changes (not reported during SARS) and a lymphoplasmocytic infiltration in the lesions (syncytial cells characterise SARS lesions); finally, no therapies are effective for FIP, while SARS can be treated with antiviral or supportive therapies. The similarities between FIP and SARS further support the hypothesis of the pathogenic role of the SARS–CoV. Moreover, FCoV biology has been extensively investigated and further studies on spontaneous cases of FIP might help the scientific community to draw useful information about the SARS–CoV.

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