

FATAL DISSEMINATED TOXOPLASMOSIS IN CAPTIVE RING-TAILED LEMURS (*Lemur catta*)

Claudia Eleni (1), Caterina Raso (1), Valentina Galletta (1), Raffaella Parmigiani (1), Klaus G. Friedrich (2), Pilar Di Cerbo (2), Paolo Selleri (3), Fiorentino Stravino (1), Virginia Carfora (1), Cristiano Cocumelli (1)

(1) Istituto Zooprofilattico Sperimentale del Lazio e della Toscana “M. Aleandri”, Roma
(2) Fondazione Bioparco, Roma (3) Centro Veterinario Specialistico, Roma.

INTRODUCTION

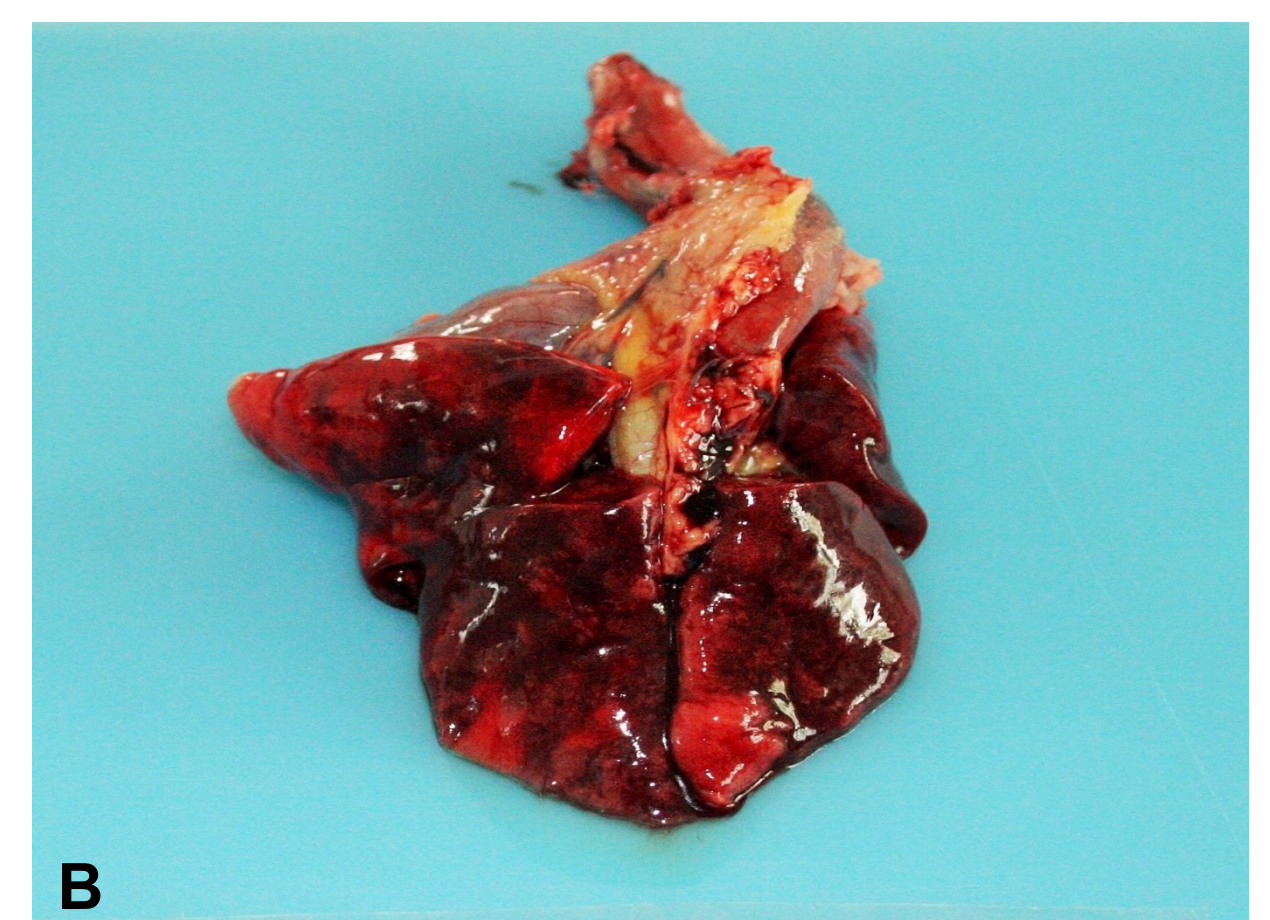
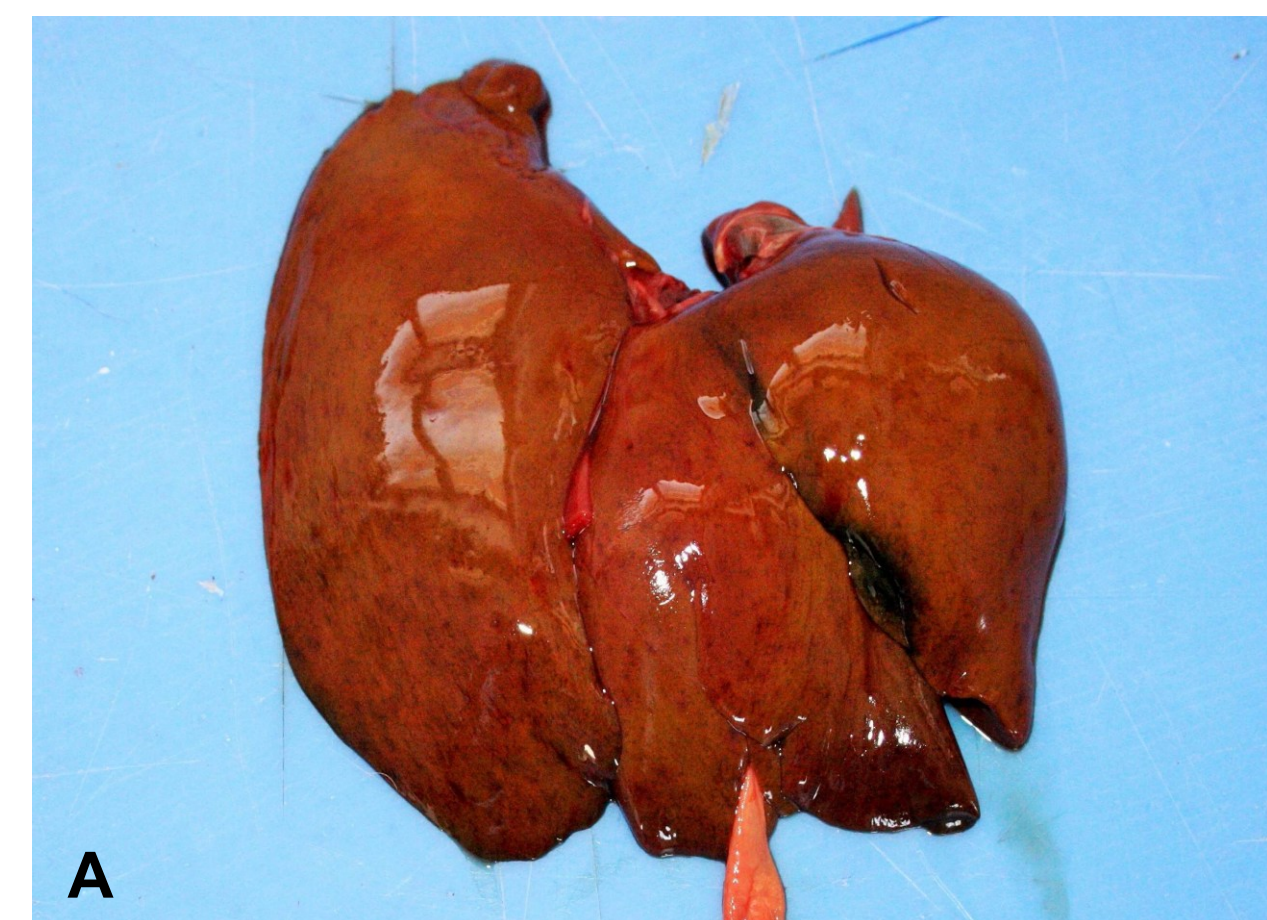
Toxoplasma gondii, the causative agent of toxoplasmosis, is an obligate intracellular protozoan parasite that infects a wide range of endothermic species, including humans. Among non-human primates, high susceptibility has been found in New World monkeys and prosimians (lemurs in particular), where the disease often results in an acute and fatal form [1]. Genetic diversity of *T. gondii* strains infecting primates is considered one of the factors involved in determining the different susceptibility [2]. We describe the pathological findings observed in captive ring-tailed lemurs (*Lemur catta*) died for toxoplasmosis.

MATERIALS AND METHODS

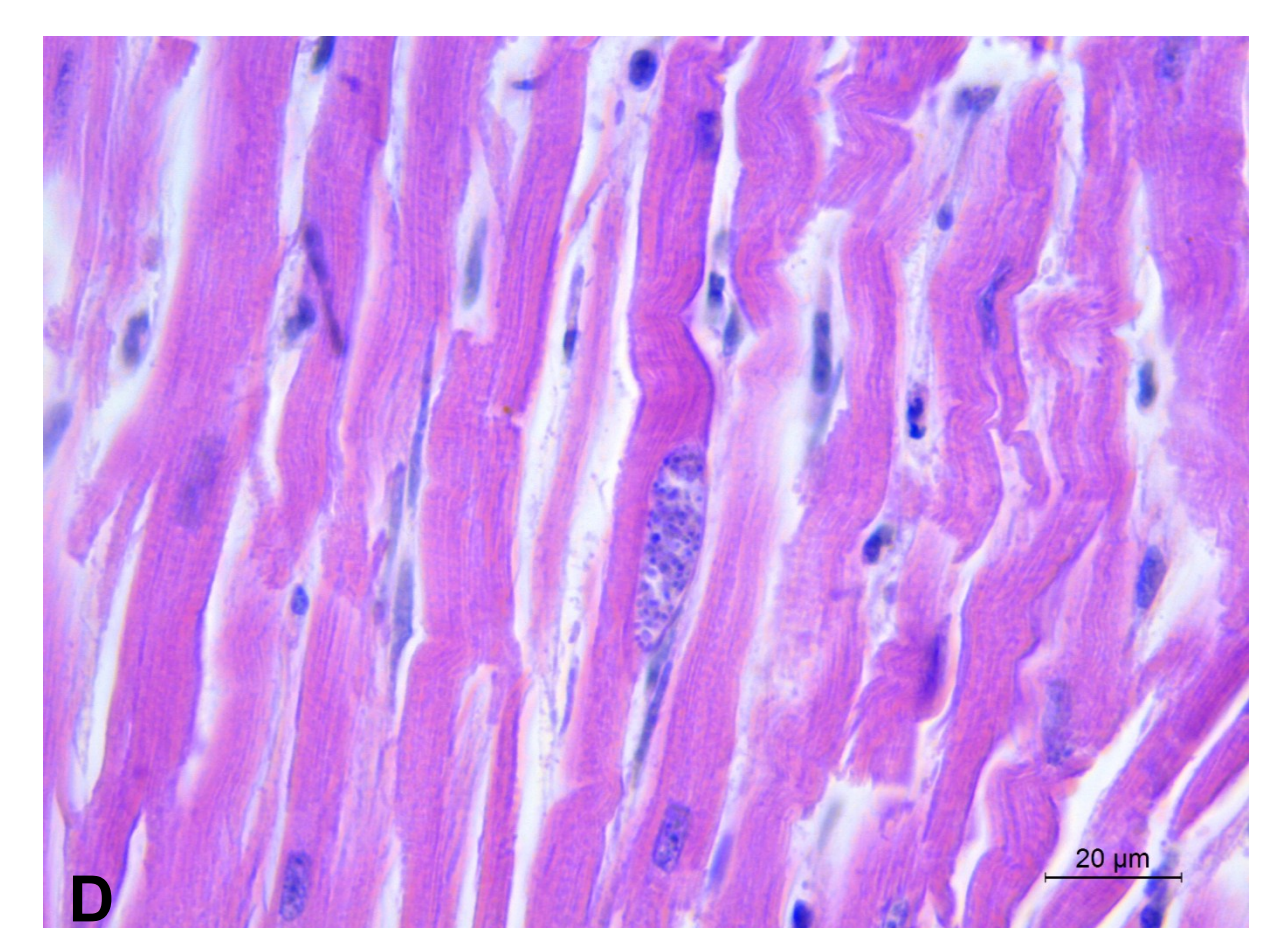
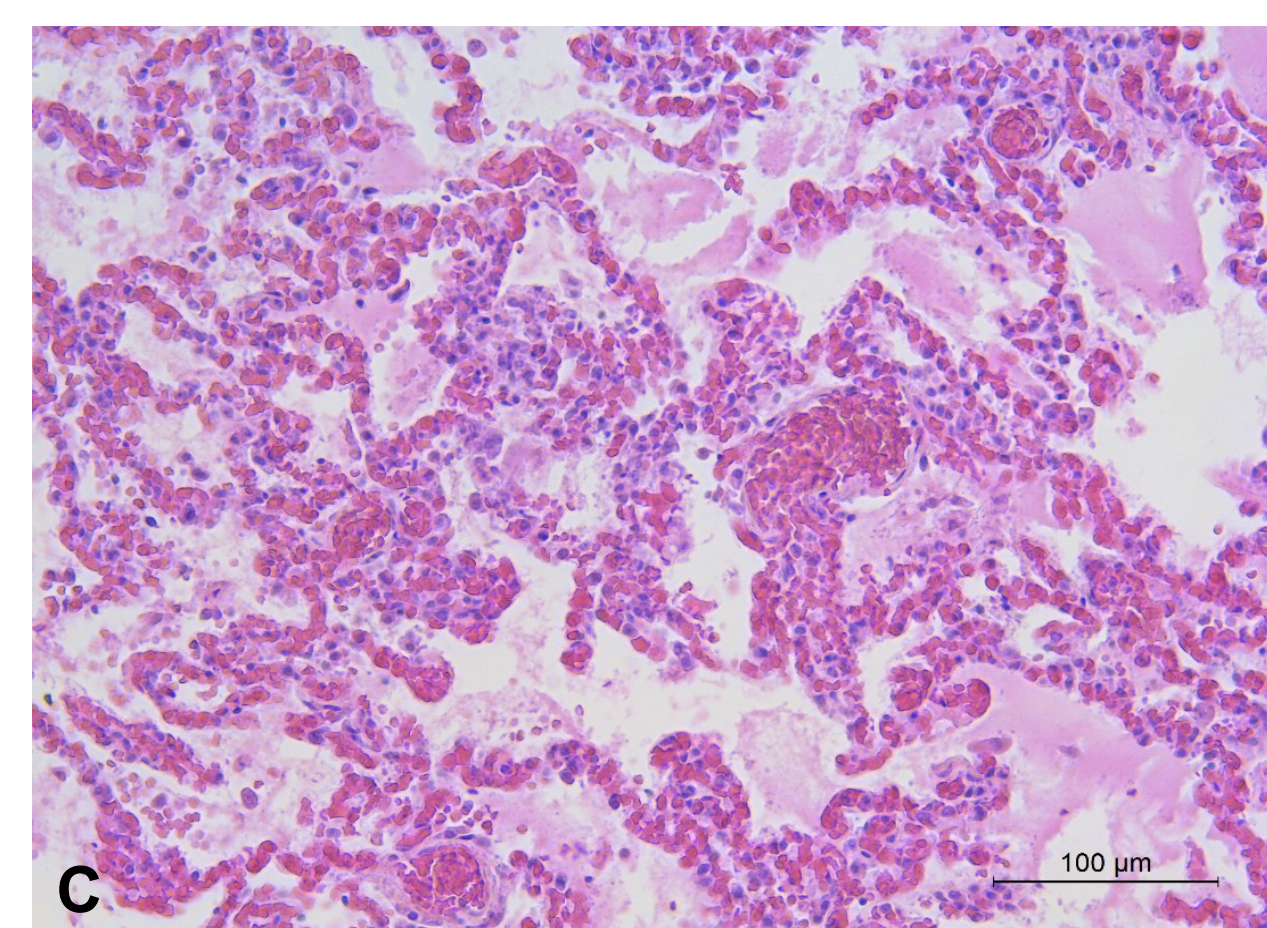
Two lemurs from two different zoological collection of Central Italy, were submitted to the Istituto Zooprofilattico Sperimentale del Lazio e della Toscana for post-mortem examination. Lemur 1, an adult female, was part of a group of four subjects who had all presented the clinical symptoms (malaise, anorexia and dyspnoea for 4-5 days) in the same period and rapidly died in a few days. Lemur 2 was a two-year-old male from a group of 15 subjects, who had showed lethargy, anorexia and progressive wasting for two weeks before death.

RESULTS

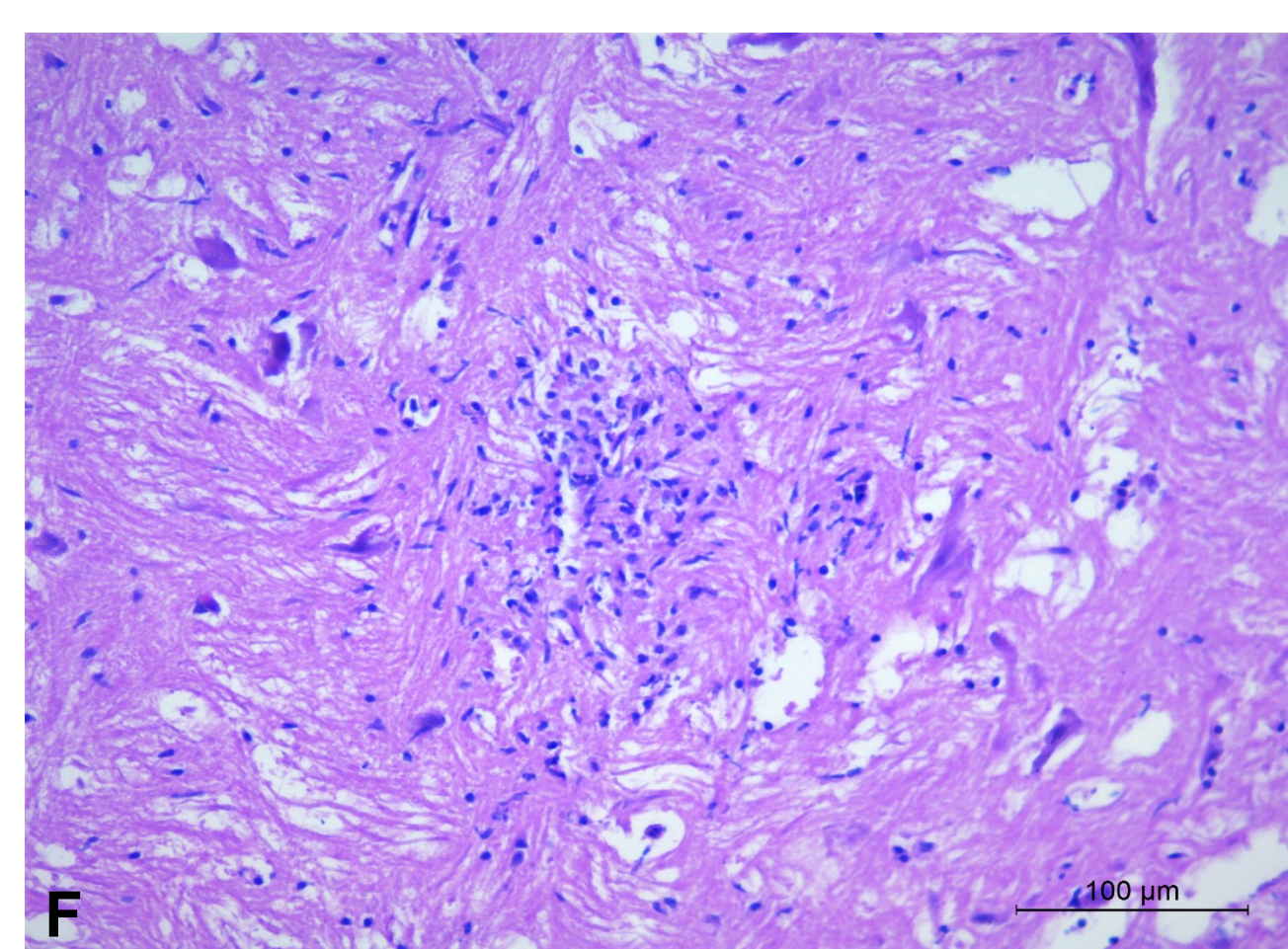
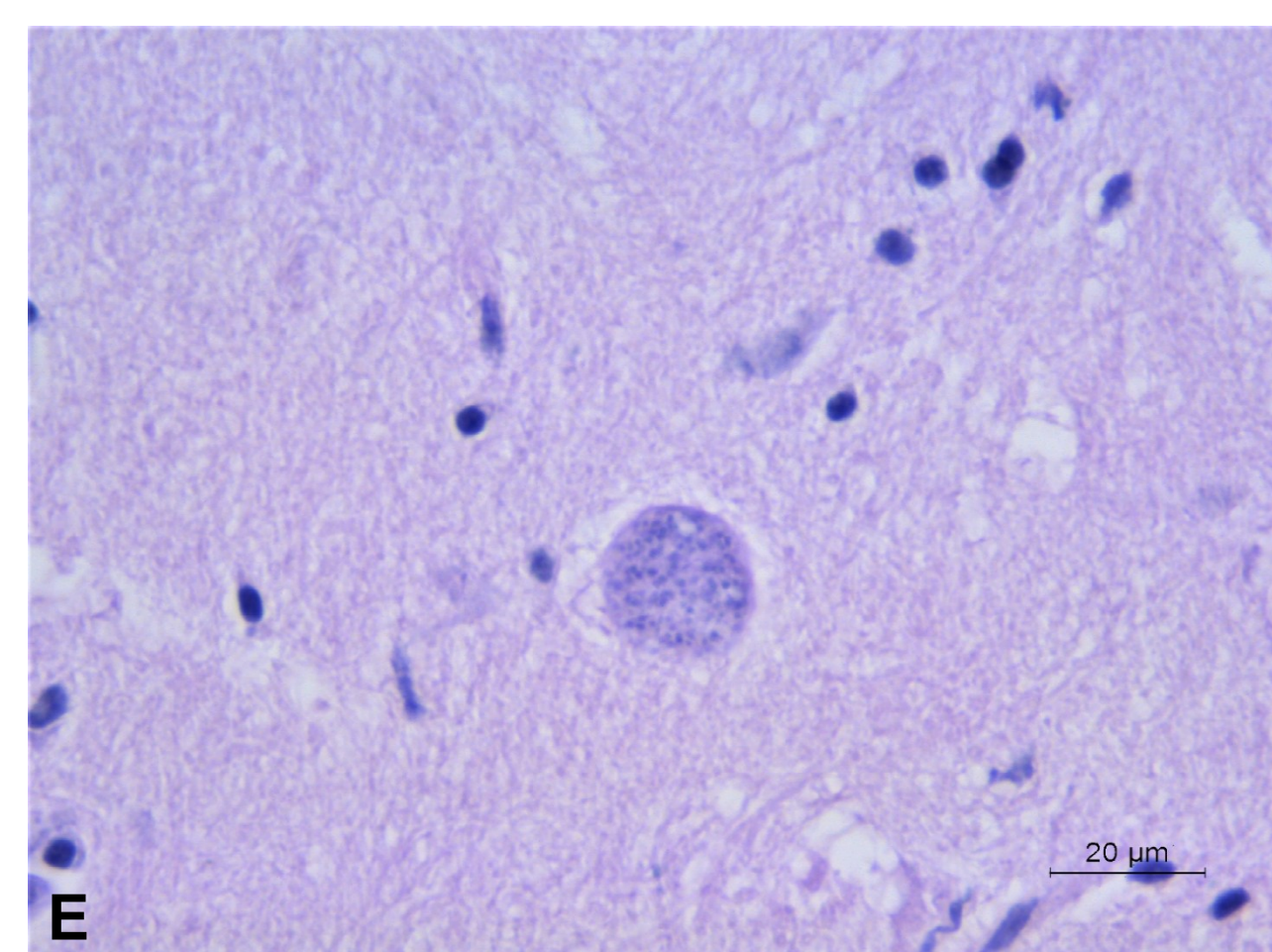
Post-mortem examination revealed in both lemurs hepatic degeneration and congestion, severe lung congestion and scattered haemorrhages of small intestine. Lemurs 2 showed also a sero-sanguinous nasal discharge and poor body condition. Specimens of the main organs were collected for histological, bacteriological, virological and molecular tests. Histologically, similar lesions were found, although more severe in lemur 1. Liver presented multifocal necrosis with fibrin deposition and degenerated neutrophils; severe multifocal steatosis was also detected. In lung, congestion and moderate alveolar oedema were observed, together with multifocal fibrinous bronchiolointerstitial pneumonia, particularly in lemur 1. Spleen, heart, mesenteric lymph node and intestine showed multifocal necrosis. In kidney, multifocal moderate membranous glomerulonephritis and lymphoplasmacytic interstitial nephritis were observed. Brain showed a moderate lymphocytic and neutrofilic meningoencephalitis, with scattered glial nodules and microhaemorrhages; in lemur 2 scattered *T. gondii* cysts were observed. Immunohistochemical labelling for *T. gondii* revealed numerous tachyzoites in several organs, especially in liver necrotic area. Real-Time PCR analysis for *T. gondii* was carried out as previously reported [3] on brain, lung, heart, liver and kidney samples and all tested positive. Bacteriological and virological tests performed on multiple organs were negative.



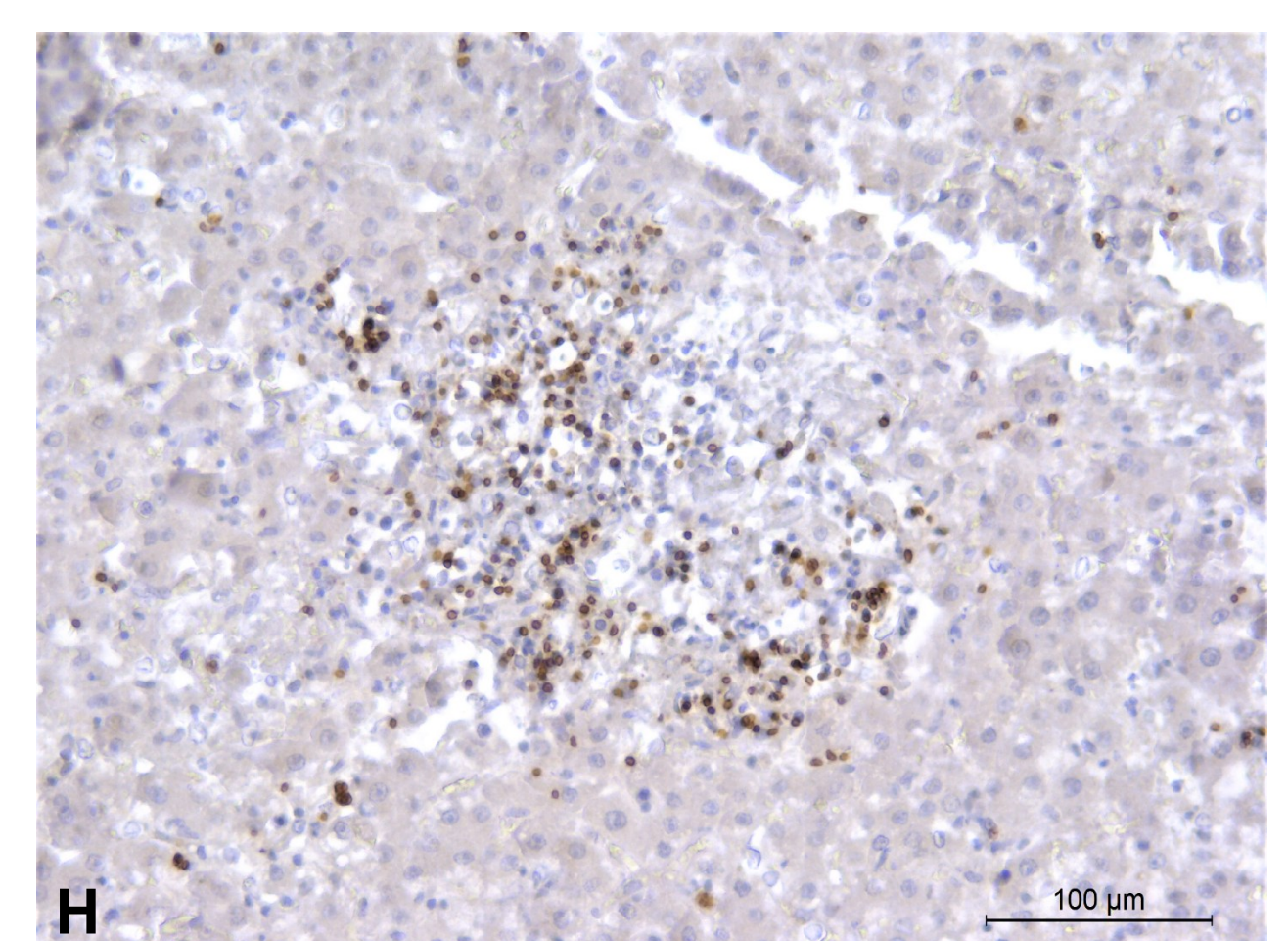
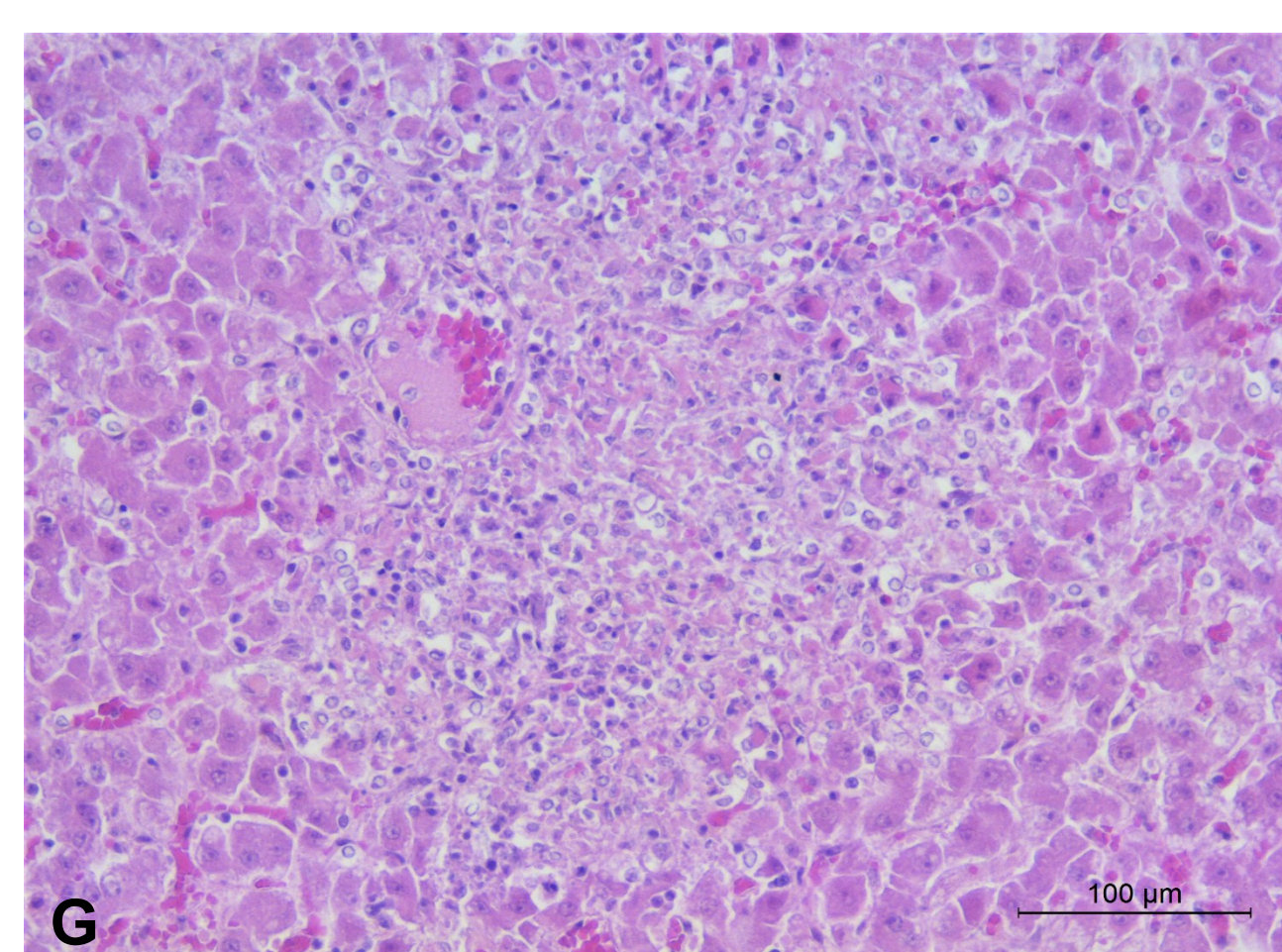
A. Lemur 2. Liver. Degeneration and congestion. **B.** Lemur 1. Lung. Sever congestion.



C. Lemur 2. Lung. Multifocal interstitial pneumonia with congestion and alveolar oedema. **D.** Lemur 1. Heart. A tissue cyst containing bradyzoites in the cytoplasm of a cardiomyocyte. Haematoxylin and eosin stain.



E and F. Lemur 2. Brain. A tissue cyst containing bradyzoites (E). Glial nodule and lymphocytic encephalitis. (F). Haematoxylin and eosin stain. **G and H.** Lemur 1. Liver. Necrotic area in the parenchyma (G). Haematoxylin and eosin stain. Numerous tachyzoites were detected by IHC in the necrotic area (H). Mab anti *T. gondii* p30, Invitrogen.



DISCUSSION AND CONCLUSION

Pathological findings observed are similar to those already reported in lemurs and New World primates [4,5]. These results confirm the high susceptibility of the lemurs to *T. gondii* infection and suggest including toxoplasmosis among the important diseases affecting these animals. Furthermore, despite we did not have had the opportunity to examine the other subjects cohabiting with the lemur 1, the presence of the same symptoms and the fatal outcome in all the animals, lead us to hypothesize that toxoplasmosis was the cause of death of the whole group of lemurs. Molecular in-depth studies to evaluate the genetic diversity of the two *T. gondii* positive samples, are still ongoing. Indeed, the results of genetic characterization could be useful to possibly understand the differences in the clinical course of the two cases described.

[1] Denk et al. Toxoplasmosis in Zoo Animals: A Retrospective Pathology Review of 126 Cases. *Animals*, 12, 619, 2022. [2] Dubey et al. Recent epidemiologic, clinical, and genetic diversity of *Toxoplasma gondii* infections in non-human primates. *Res Vet Sci*, 136, 631-641, 2022. [3] Lin et al. Real-time PCR for quantitative detection of *Toxoplasma gondii*. *J Clin Microbiol*. 11:4121-4125, 2000. [4] Siskos et al. Unique case of disseminated Toxoplasmosis and concurrent hepatic capillariasis in a ring-tailed lemur: first case description. *Primate Biol*. 2: 9-12, 2015.