

ROLE OF CIRCULATING ANTIBODIES AND THYMUS-DEPENDENT LYMPHOCYTES IN PRODUCTION OF EFFUSIVE TYPE FELINE INFECTIOUS PERITONITIS AFTER ORAL INFECTION

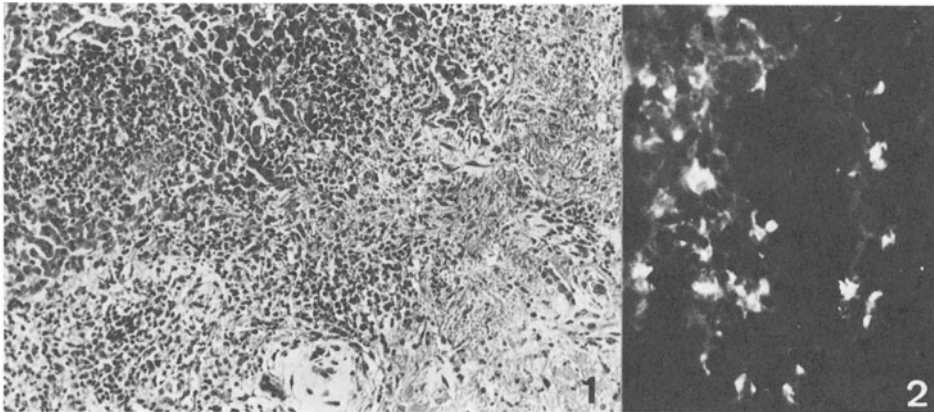
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Seropositive or antibody-transferred kittens have been reported to have an overt disease after parenteral challenge with virulent feline infectious peritonitis (FIP) virus, suggesting that the effusive type FIP^{2,3} might result from an interaction between the virus and host response. On the other hand, feline leukemia virus infection was suggested to enhance the infection of FIP virus¹. The present study deals with roles of circulating antibodies as well as thymus-dependent lymphocytes in pathogenesis after oral infection of FIP virus.

Fibrinous serositis was produced in 4 of 20 seropositive kittens or those having received transfer of anti-FIP cat antibody after intragastric inoculation with FIP virus, whereas 30 seronegative animals had no signs of illness but some enteritis. Lesions produced in the serosa and abdominal organs were characterized by fibrinous inflammation with necrotic and pyogranulomatous vasculitis as well as necrosis in lymphoreticular tissues(Fig.1). Viral antigen was detected within macrophages and enterocytes in those lesions by immunofluorescence assay(Fig.2).

In another experiment, 3 kittens were thymectomized (Tx). One of them received normal cat serum (Tx+Ab -); the other 2 remained non-treated(Nt). Another group of 5 kittens received anti-FIP cat antibody, and 2 of them were thymectomized(Tx+Ab) while 3 were sham-operated(Sh+Ab). Shortly after the operation all 8 kittens were challenged orally with FIP virus. All of Nt, Tx or Tx+Ab(-) cases were shown to have enteritis without serosal and visceral affections. Enteritis in Tx or Tx+Ab(-) cases was more profound than in Nt cases. Virus antigen was detected mostly within macrophages in the former whereas within enterocytes in the latter, suggesting that infection of macrophages in the tunica propria might be important in the subsequent



Antibody-transferred and intragastrically inoculated case dead on day 19 postinoculation.

Fig. 1. Severe coagulative necrosis, focal infiltration of inflammatory cells and proliferation of fibroblasts in the liver. HE stain x620.

Fig. 2. Viral antigen within the cytoplasm of macrophages accumulated in and around an interlobular artery.

production of lesions in abdominal organs. On the other hand, all cases of both Tx+Ab and Sh+Ab groups showed fibrinous serositis, which was developed more severe in Tx+Ab. Also in Tx+Ab cases virus antigen-positive cells were more numerous than in Sh+Ab.

These results suggest that the humoral antibody and T lymphocytes might play an important role in enhancing and suppressing, respectively, the production of serositis as well as parenchymatous lesions.

REFERENCES

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