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FELINE INFECTIOUS PERITONITIS: PRESENT KNOWLEDGE

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Abstract—The purpose of this paper is to take stock of the knowledge gained over the last few years on Feline Infectious Peritonitis in a synthetic way.

Apart from the bibliographical study regarding the works of numerous authors, we state our results concerning the study of the experimental reproduction of the disease.

From all the cat infectious diseases, Feline Infectious Peritonitis is the clinical entity the most recently evidenced. In this paper we shall state the elements now clearly established on this disease but numerous points are still to be made clear. Contagious disease of viral origin, it affects domestic and wild Felidae. Clinically, in its most typical form, it is characterised by the progressive development of an exudative sero-fibrinous peritonitis and a febrile and gradual weakness of the general condition. It slowly and regularly develops into death.

Key words: Feline Infectious Peritonitis

PERITONITE INFECTIEUSE FELINE: CONNAISSANCES ACTUELLES

Résumé—L'objet de cette publication est de faire le point, de manière synthétique, des connaissances acquises au cours de ces dernières années sur la Péritonite infectieuse féline.

Outre, l'étude bibliographique se référant aux travaux de nombreux auteurs, nous faisons état de nos résultats relatifs à l'étude de la reproduction expérimentale de la maladie.

La péritonite infectieuse féline est l'entité clinique la plus récemment individualisée des maladies infectieuses du chat. Nous préciserons, au cours de cet exposé, les éléments la concernant qui sont désormais acquis, mais nous verrons que de nombreux points restent encore à préciser. Maladie contagieuse, d'origine virale, elle frappe les Felidae domestiques et sauvages. Elle est caractérisée cliniquement, dans sa forme la plus typique, par le développement progressif d'une péritonite exsudative séro-fibrineuse, accompagnée d'un affaiblissement fébrile et graduel de l'état général. Elle évolue lentement et régulièrement vers la mort.

Mots-cléfs: Péritonite infectieuse féline

EPIZOOTIOLOGY

Although clinically evidenced before 1962, it is at that time that Jean Holzworth [12] makes the first clinical and lesional descriptions under the name "Chronic fibrinous peritonitis". Wolfe and Griesemer [32] made the first complete study in 1966. Since then, the disease has been described in the five continents:

America	: United States [30, 23, 16, 17, 10, 7], Canada [25]
Africa	: South Africa and Senegal [3, and in 4]
Asia	: Japan [15]
Oceania	: Australia [31, 14]
Europe	: Great Britain [13], Ireland [11], Holland [18], Germany [27], Belgium [9], Switzerland [26] and France [1, 5].

Its incidence seems all the more high that it is better and better known of veterinary practitioners.

In the United States, Pedersen [21], referring to the autopsies carried out in the Veterinary School of Davis, California, notes that its incidence has increased from 1% in 1960 to 16% in 1973.

The disease affects more especially kittens of less than two years of age. It affects with an identical frequency males and females. There is no breed more especially subject to the disease than any other.

As we already told, this disease affects all the Felidae. It has been evidenced in black panthers [27], and lions, jaguars and leopards [4]. Although it is very easy to infect cats with the disease, it has been impossible to infect mice and newborn mice, rats, hamsters, guinea-pigs, ferrets and puppies.

Before tackling the clinical study of the disease, it is interesting to take into account the first serological survey recently carried out by Pedersen.

This author observes that:

- there are 87% of seropositivity in catteries having trouble with Feline Infectious Peritonitis (revealing up to 5% of clinical cases per annum);
- there are 20% of seropositivity in cats brought to a veterinarian for a visit (1.3% of clinical cases per annum).

This study shows the high proportion of sub-clinical and unapparent infections (14 cases out of 15).

Clinical and lesion study

The observation of the field disease makes it possible to recognise two forms of the disease: the wet form and the dry form.

(1) *Wet or exudative form.* The wet or exudative form is the classical form. It is characterised by an accumulation of fluid in the abdominal and/or thoracic cavities.

Fever, anorexia leading to a progressive loss of weight, depression and ascites are observed. In 15 to 30% of the cases, dyspnea due to a pleurorrhoea is also observed. A jaundice may also appear in the final stage of the disease.

(2) *Dry or parenchymatous form.* In the dry or parenchymatous form, there is no liquid effusion and it is more difficult to establish a diagnosis because only one organ may be affected: kidneys, eyes, liver, brain, abdominal lymphatic ganglia or lungs. Meningo-encephalitis [16, 24] were described, leading to convulsions, cerebellar and vestibular shortages bringing about a progressive ataxia, pareses of the hindquarters [17], panophthalmia [8, 2] including uveitis, iridocyclitis, choroiditis and retinitis.

Bullmore [2] considers that these forms represent 50% of the cases of Feline Infectious Peritonitis.

In hematology, a slight anemia is observed in all the forms of the disease. It may be serious when the disease is complicated by a leucosis or haemobartonellosis. On the opposite, a leukocytosis due to an increase of the neutrophils is observed. Toxicity vacuoles are sometimes observed in these neutrophils. But the number of lymphocytes is often very low. A relative increase of plasmatic proteins, a decrease of albumin and an increase of fibrinogens and gammaglobulins are observed in the serum [9, 21].

In the anatomico-pathological field, in the wet form of the disease, some fluid (sometimes in

large quantities: up to 1 l.) of straw-yellow to dark amber color is collected on opening the peritoneal cavity. This fluid is often transparent but sometimes curdled. It is slightly viscous and coagulated partially when exposed to the air. It has all the characteristics of a sero-fibrinous exudate.

The various peritoneum pleura are covered with white greyish fibrinous sediments with a granulated aspect. The epiploonomentum is thickened, opaque and infiltrated with a gelatinous oedema. Liver and spleen are compressed and deformed by a compact white fibrinous capsula. The abdominal viscera show lesions, more especially in the dry form of the disease. Points of sub-capsular and sub-serous necroses of 0.5–1 mm of diameter are sometimes observed on the liver, intestines, kidneys and bladder.

When the thoracic cavity is affected, the same granulous lesions as those observed on the peritoneal cavity are found on the pleura, diaphragm and lungs. When the nervous system is affected, meninges show whitish spots, often localised along vascular tracts.

Under the microscope, the initial lesion is from vascular origin. It is a *perivasculatitis*. These lesions are covered with a fibrinous sediment, later on overgrown by inflammatory cells: macrophages, lymphocytes and neutrophils [23].

The experimental reproduction of the disease is easy.

The inoculation of sensitive cats with a crushing of infected organ, fresh or stored at -70°C or with a fresh ascites fluid, carried out by the sub-peritoneal route, makes it possible to observe an acute form of the disease. The incubation lasts 2–3 days. There is an hyperthermia (39.5°C – 40°C). An anorexia is observed and death occurs between the 10th and the 15th day. The lesions are exactly the same as those described for the field disease, but these are sometimes more outspread. The amount of ascites varies according to the time of evolution of the disease. It is often less abundant than in the field exudative form.

The intravenous inoculation causes an over-acute evolution preventing the development of large macroscopic lesions, death occurring very quickly (7 days). The inoculation by ocular-nasal instillation causes no affection during the 2-month period of our observation. Ward observed the development of a pneumonia [30].

On the opposite of Pedersen [21], Hardy [10] showed that it was possible to reproduce the disease by the oral route. As a matter of fact, we reproduced it by the oral route but irregularly and not systematically.

The evolution is longer by the subcutaneous route. A lesion due to necrosis is observed at the point of inoculation, then the infection of the regional satellite ganglion and the spreading to the various organs [30].

The intrapleural inoculation causes pleuresia. The intracerebral inoculation causes encephalitis [30].

ETIOPATHOGENY

Viral etiology was suspected as soon as 1966. As a matter of fact, crushings of organs coming from infected cats filtered through 200 nm filters, made it possible to reproduce the disease. These crushings, treated with antibiotics with large spectrum, contained no bacterium, fungus, chlamydia, toxoplasma, nor mycoplasma.

As soon as 1968, Zook [33] observed particles with a viral aspect on sections coming from cats experimentally infected and observed under an electronic microscope. Since then, a lot of research workers have evidenced these viral particles, despite the low amount of infected cells (2%). We have observed such viral particles.

Ward, J. M. [29] has found the virus in various organs of 12 out of the 25 cats inoculated experimentally. The infected cells are macrophage cells, located at the periphery of the lesions due to necrosis.

The viral particles are observed near Golgi apparatus in the vesicles or budding from the smooth surface of the endoplasmic reticulum. No bud has been observed from the cytoplasmic membrane. Extra-cellular particles are rarely observed. The complete particles have a diameter of 70–80 nm. They are composed of a nucleoid of 50–55 nm, surrounded by a trilaminar envelope and topped by spiculae. This virus must be distinguished from the feline syncytial virus and from the feline leukosis virus.

Osterhaus [19] has showed that the virus has a sedimentation constant of 400 S and a density of 1.16 g/ml. Furthermore, the virus is sensitive to ether, formalin and heat. It is inactivated in 60 min at 56°C. The virus is extremely fragile. The morphogenesis, morphology and some physico-chemical properties, now known, are similar to those of Coronaviruses. But the most important problem remains the difficulty, for not saying the impossibility, of culturing it *in vitro*.

From all the cell cultures on which the virus multiplication has been attempted, Pedersen [20] has been able to evidence, by means of indirect immunofluorescence, the viral antigen only in the cytoplasm of peritoneal macrophages coming from inoculated cats and kept in survival. No cytopathic effect was observed. Only a quicker degeneration of the infected macrophages with regard to the control macrophages was observed. This difficulty to culture the virus remains the most important handicap in the study of this disease.

The source of virus in the field is unknown. It is tempting to consider its spreading through healthy carrier cats or in the state of sub-clinical infection.

The natural route is unknown though some authors succeed in reproducing it experimentally by the oral route and then we may suspect that it is one of the routes of infection.

We kept for 7 months a cat in permanent contact of animals experimentally infected. This contact cat remained sensitive to the disease. He died from Feline Infectious Peritonitis after the experimental infection. The viremia taking place during the infection, it is not excluded that hematophage parasites might be the possible vectors.

The neonatal or *in utero* transmission may be considered insofar as complete litters of kittens were decimated, the dam dying from the disease several months later [21].

DIAGNOSIS

The clinical diagnosis is easy when we have the exudative form owing to the presence of ascites fluid and/or pleural fluid. If the exudation is low or if it is a dry form, the diagnosis is more difficult. Apart from the depressive attitude and the animal's loss of weight, some other signs must be taken into consideration:

- Chronic fever of fluctuant intensity;
- Leukocytosis (neutrophilia, lymphopenia);
- Increase of plasmatic proteins (including the fibrogen);
- Nervous signs: incoordination, paresis, hyperesthesia, convulsions;
- Ocular lesions: fibrine precipitates adhering to the internal face of the cornea, retinal haemorrhages;
- Mesenteric nodes and swollen kidneys;
- Chronic hepatic and renal disorders.

Feline Infectious Peritonitis must be distinguished especially from:

- Leukosis (with which it may be combined);
- Tuberculosis;
- Toxoplasmosis (in the nervous and ocular forms);
- Cryptococcosis (in some ocular forms).

The chapters dealing with Treatment and Prophylaxis may be left out insofar as:

- the antibiotherapy and corticotherapy have only a relative effectiveness for not saying that it is null;
- it is difficult to contemplate a sanitary prophylaxis because of the epidemiological unknowns;
- it will be certainly possible to develop a medical prophylaxis according to the epidemiological impact of the disease when all the difficulties inherent to the culture of the virus are solved.

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