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EPIGENETIC TRANSMISSION OF FELINE INFECTIOUS PERITONITIS

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Abstract—Feline Infectious Peritonitis (F.I.P.) was diagnosed in the kittens of two successive litters born to a female presumed also infected.

At the same time, the two fathers and the other subjects of the cattery remained asymptomatic of F.I.P.

The clinical observations, supported by electrophoretic data, suggest the possibility of a direct transmission of the disease by the mother to her offspring, either by the transplacentary pathway (epigenetic transmission) or via the milk.

Key words: Epigenetic transmission, feline infectious peritonitis (F.I.P.), coronavirus, epidemiology

TRANSMISSION EPIGENETIQUE DE LA PERITONITE INFECTIEUSE FELINE

Résumé—La péritonite infectieuse féline a été diagnostiquée chez les chatons de deux portées successives issues d'une mère elle-même présumée atteinte.

Dans le même temps, les deux pères et les autres sujets de l'élevage sont restés indemnes de tout symptôme de péritonite infectieuse. Les observations cliniques étayées par des données électrophorétiques, suggèrent dans ce cas, la possibilité d'une transmission directe de la maladie par la mère à ses petits, soit par la voie transplacentaire (transmission épigénétique), soit par le lait.

La voie naturelle d'infection de la péritonite infectieuse reste encore mal connue et controversée; ceci d'autant plus que certaines caractéristiques du virus laissent à penser qu'il ne peut résister très longtemps dans le milieu extérieur; et que, d'autre part, les études expérimentales ont montré la difficulté de transmettre la maladie par les voies naturelles. Par contre, les voies d'inoculation sous-cutanée, intraveineuse et intrapéritonéale sont généralement suivies de succès. Dans l'état actuel de nos connaissances, la transmission épigénétique de la maladie paraît constituer une des voies que peut emprunter le virus pour se propager.

Mots-clefs: Transmission épigénétique, peritonite infectieuse féline (P.I.F.), coronavirus, epidémiologie

INTRODUCTION

The etiology of F.I.P. is slowly becoming understood ever since the experimental transmission of the disease by ultrafiltrates and the electron microscopic observation of viral particles in diseased tissues [1, 2].

Other authors have tried to specify the characteristics of the etiological agent [3, 4], or even to cultivate it [5].

The etiological agent seems to be a member of the *Coronavirus* group; it is sensitive to ether and to heat, and is normally inactivated within 24 hr at room temperature. Despite many attempts, it has not yet been isolated, so a serological test was difficult to set up. Nevertheless, Pedersen [6] recently performed serological studies which will undoubtedly help to clarify the pathogenesis and the epidemiology of the disease. The source of virus in the environment of the cat is not yet clearly known, although the virus seems to persist in asymptomatic carriers.

The natural route of infection is still under investigation. In effect, the virus is readily inactivated outside the host and experimental studies have shown that it is difficult to transmit

the disease by the usual routes [7]; on the other hand, the parenteral routes, such as subcutaneous, intravenous and intraperitoneal are ordinarily successful.

The epigenetic transmission (transplacental) of the disease seems to be one of the ways the virus may choose to propagate. Several clinical observations lead us to suppose this; this route of transmission may be invoked to explain the epidemiology of the cases reported in this paper.

There have been previous reports of multiple cases of feline infectious peritonitis in a household [8, 9], of neonatal feline infectious peritonitis [10], and multiple cases of feline infectious peritonitis among kittens produced by certain queens [7].

OBSERVATIONS

The owner of a small cattery breeding "Color Point" Persian cats sent, in succession, three kittens to the necropsy service of the Faculty. These kittens constituted the third litter of the female "Doechka", all of whose litters have been detailed on Table 1. Feline infectious peritonitis was diagnosed in all these cases based on macroscopic and microscopic lesions [11].

Table 1. Litters born to the cat "Doechka". Interval between litters: I and II: 8 months. II and III: 5 months. III and IV: 5 months

Litter No.	Father		Kittens
I	Victor	4	Males*
II	Ringo	4	2 Males* 2 Females*
III	Ringo	3	1 Male: dead of F.I.P. at the age of 4 months 2 Females: dead of F.I.P. at the age of 4½ months
IV	Juvenal	3	1 Male, Livius: dead of F.I.P. at the age of 2½ months 1 Male, Lucrèce 1 Female, Lucrecia

* Kittens for which present status is unknown.

Five months later, the same female gave birth to three kittens. One of them, "Livius", died at the age of two and a half months of F.I.P., identified by necropsic examination. We then proceeded with clinical and hematological examinations of the mother and the two other kittens. The mother presented minor weight loss and weakness but no significant hematological changes. The two kittens presented leucocytosis and modifications in the differential leucocyte count.

The same examinations were done again two months later, the mother and the kittens showing the same weight loss and weakness, accompanied in the latter by retarded growth. At this moment, electrophoresis revealed protein modifications in the mother's serum

compared with normal serum: an increase of an α_1 globulin (possibly orosomucoid), of a β_1 globulin (the transferrin) and of the γ globulins, and a decrease of albumin. During the last examination done five months later, the mother and her two kittens appeared clinically healthy. However, hematological anomalies were noted. Electrophoresis of the mother's serum proteins show the same modifications as previously noted, but accentuated; those of the kittens show the same particularities. The same examinations were done on the other cats of the same cattery, as no specific precautions were taken to avoid eventual contagion by direct or indirect contact between the animals. During our observation period, no other animal in the cattery besides the mother and the kittens of the third and fourth litters presented clinical or electrophoretic signs of F.I.P.

DISCUSSION

We have previously described [12] the important modifications of certain plasma proteins in cats suffering either from experimental or natural cases of F.I.P. These modifications are: an increase of fibrinogen, haptoglobin, transferrin, and of an α_1 glycoprotein which might be orosomucoid; in the natural disease, in addition to these modifications, a decrease of albumin appears, and the γ globulins are strongly increased, due to immunoglobulins of the IgG class (IgG_1 and IgG_2).

These modifications can be found in the plasma and in the ascitic fluid when such fluid is present.

The proteins which increase during the disease are some of the so-called inflammatory proteins. This increase of their plasmatic level is a feature relevant to nonspecific and general reactions of inflammation; thus it is not pathognomonic for F.I.P., even though it is always observed in cats suffering from the disease. The mother and two kittens of her last litter presented some of these electrophoretic modifications in their sera, which was not the case in the sera of the other subjects of the cattery. Although it is not proven, the modifications observed are compatible with an inactive or subclinical form of F.I.P.

The possibility of horizontal transmission of the disease either by the mother or by asymptomatic carriers in the immediate environment cannot be excluded, but the above facts suggest that in this case, epigenetic transmission of the disease may be incriminated. In effect, two successive litters born to the same mother sired by two different fathers, suffered from infectious peritonitis, whereas in the same cattery, promiscuity notwithstanding, no other kitten, nor other adult cat, have presented clinical signs or suspicion of infectious peritonitis.

Vertical transmission is also possible, as is transmission via the milk, but experimental studies have shown that it is difficult to transmit the disease by the oral route. The mother might well have passed on the genetic susceptibility to the disease without having passed on the disease itself. The intervention of the father can in all likelihood be excluded given that the two litters were sired by different fathers.

Epigenetic transmission of the disease seems plausible, but we need more experimental work to understand the natural route of infection.

This is essential for the general understanding of the disease and necessary for the development of a suitable prophylactic program.

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