BRAIN LESIONS ASSOCIATED WITH INFECTIOUS KERATO-CONJUNCTIVITIS IN CHAMOIS AND ALPINE IBEX

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Abstract - Twenty-five Chamois (*Rupicapra rupicapra*) and 8 Alpine ibex (*Capra ibex ibex*) with infectious kerato-conjunctivitis were captured in the Gran Paradiso National Park. Only seven chamois and 4 alpine ibex showed signs of nervousness. The histopatological findings showed that 12 chamois and 4 ibex were affected by inflammatory alterations of the brain, represented by: foci of nono-purulent leptomeningitis as small accumulations of mononuclear cells; perivascular cuffings in the cerebral hemispheres; severe infiltration of lymphocytes in the choroid plexus fo the cerebral ventricles. The non-purulent morphology of the inflammation might be a consequence of the action of the primary pathogenic agent (*Mycoplasma, Chlamydia or Rickettsia*).

IBEX J.M.E. 2:17-22

1. Introduction

Infectious kerato-conjunctivitis in Chamois (*Rupicapra rupicapra*) and Alpine ibex (*Capra ibex*) appeared in the protected area of the Gran Paradiso National Park in May of 1981.
This primary development coincided with a peak in mortality in the Chamois population especially noted in the years between 1981 and 1985 and which may be attributed to lesions following ocular infection.
This disease, well known in Chamois, has been noted in all Alpine regions over the last decade: in France (Oudar *et al.*, 1982), Italy (Balbo *et al.*, 1982), Switzerland (Fellay, 1970; Nicolet, 1982), Austria (Steineck, 1982), Yugoslavia (Valentincic, 1982) and Spain (Sanchez Belda & Martinez Ferrando, 1982).
Thirteen years on from its appearance it is possible to state that it is a disease of a persistent nature which periodically re-appears, in isolated outbreaks.
The clinical and pathological aspects of the disease have been described in detail (Burgisser *et al.*, 1959; Lanfranchi *et al.*, 1982; Prave *et al.*, 1987; Gauthier *et al.*, 1988) while the etiology and epidemiology, despite a lengthy investigation (Nicolet *et al.*, 1974; Nicole *et al.*, 1975; Pairaudeau, 1977; Gilbert, 1979; Oudar *et al.*, 1982; Peruccio *et al.*, 1982; Bijlenga *et al.*, 1983; Blancou *et al.*, 1985; Costa, 1986; Gauthier *et al.*, 1988), are still uncertain.
One of the most interesting clinical aspects of this disease is the profound changes in behaviour noted in many subjects both in relation to space use (anomalous changes in altitude, use of marginal or extreme environments) and to social behaviour.
This symptomatology has been noticed not only in animals with severe ocular lesions but also in animals in an advanced stage of recovery, with *restitutio ad integrum* and re-establishment of visual functionality.
This work shows the histopathological findings in the central nervous system of Chamois and Alpine ibex affected by infectious kerato-conjunctivitis and how these inflammatory processes occur frequently even in subjects which do not show evident neurological signs.

Fig. 1 - Alpine ibex: ventral deviation of the head

Fig. 2 - Alpine ibex: lateral deviation of the head

2. Methods

Twenty-five chamois and 8 Alpine ibex with infectious kerato-conjunctivitis, were captured in the Gran Paradiso National Park. Seven chamois and four Alpine ibex showed signs of nervousness which, depending on the subject, was expressed by opisthotonus or with a lateral deviation of the head (Fig. 1 and 2).

Animals were euthanatized after sedation with Xylazine (60-100 mg per animal).

The brain was fixed with a 10% buffered formalin for at least three days; it was then cut transversally into parallel sections of approximately 0.5 cm thick. At least three portions from the caudate nucleus, the midbrain and the pons, including the cerebellum were then paraffin embedded. The sections obtained were then stained using common neuropathologycal techniques.

3. Results

The following findings were observed during necroscopic examination:

- denutrition;
- severe fibrinpurulent kerato-conjunctivitis with ophthalmitis;
- in some animals cysts of *Cysticercus tenuicollis* on the omentum or simil-caseous lesions in the bronchial lymph nodes were occasionally present;
- parasitic bronchopneumonia (Metastongyles);
- upon opening the skull the brain appeared normal both on the surface of the cortex and in structure.

The histopathological findings showed that 12 chamois and 5 ibex were affected by inflammatory alterations of the brain represented by:

- foci of non-purulent leptomeningitis as small accumulations of mononuclear cells, mostly lymphocytes and monocytes in the parietal leptomeninges (Fig. 3);
- mononuclear perivascular cuffings in the cerebral hemispheres (Fig. 4);
- severe infiltration of lymphocytes in the choroid plexus of the cerebral ventricles (Fig. 5), with deformation due to the accumulation of the inflammatory cells.

4. Conclusions

The neuropathology of wild animals in general and of small ruminants in particular is anything but well known. Apart from the work of Burgisser *et al.* (1959) involving 500 wild animals, 403 mammals and 97 birds little systematic research has been carried out. In the 44 chamois examined by these authors the following lesions were observed: one parasitic cyst, one meningoencephalitis, two proliferations of the cerebral vessels and one purulent encephalitis. No cerebral lesions were detected in the brains of the 7 ibex.

Montagout *et al.* (1981) did not find a single neuropathological case in 36 chamois. Sporadic nervous lesions are reported in literature. Oudar *et al.*, (1989) described a case of granulomatous encephalitis in a chamois affected by brucellosis. Histological lesions of meningo-encephalitis were found in chamois inoculated with affected eye material (Prave *et al.*, 1987). Parasites localized in the central nervous system, although well known, are not frequent in Chamois (Rossi *et al.*, 1988-89-90).

Only few data regarding the brain lesions associated with infectious kerato-conjunctivitis in wild ruminants can be found in literature. Lanfranchi *et al.* (1982) described brain lesions in Chamois with nervous symptoms and infectious kerato-conjunctivitis but did not provide further details. The authors interested in this disease have concentrated their studies mostly on the etiopathogenic and ophthalmological aspects, ignoring the neuropathological correlations.

In consideration of this, etiology plays an important role. Two or three hypotheses based on experimental data have been advanced. Some authors favour a *Mycoplasma conjunctivae* etiology (Nicolet & Freundt, 1975; Nicolet, 1982; Costa, 1986); while others underline the role played by the *Chlamydia psittaci* (Sanchez Belda & Martinez Ferrando, 1982; Tournut *et al.*, 1982), others feel that *Rickettsia* may be involved (Bijlenga *et al.*, 1983).

In domestic ruminants, nervous lesions induced by *Mycoplasma* spp. and *Chlamydia* are well known. Sporadic meningoencephalitis in cattle, described in several countries (Harshfield, 1970; Johnston *et al.*, 1962; Kalmar *et al.*, 1967) has been attributed to *Chlamydia* , while in Switzerland it was reported to be caused by *Mycoplasma* (Bestetti *et al.*, 1976). On the other hand Oudar *et al.* (1982), Gibert (1979), Pairaudeau *et al.* (1977) and Prave *et al.* (1987) assume that, on the basis of the epidemiological data, kerato-conjunctivitis in Chamois is a disease specific to this species and not connected, at least etiologically, with that of domestic ruminants.

The opportunistic microrganisms which can complicate the ocular lesions are numerous, for example *Corynebacterium* spp. (Nicolet *et al.*, 1974), *Staphylococcus aureus haemolyticus*

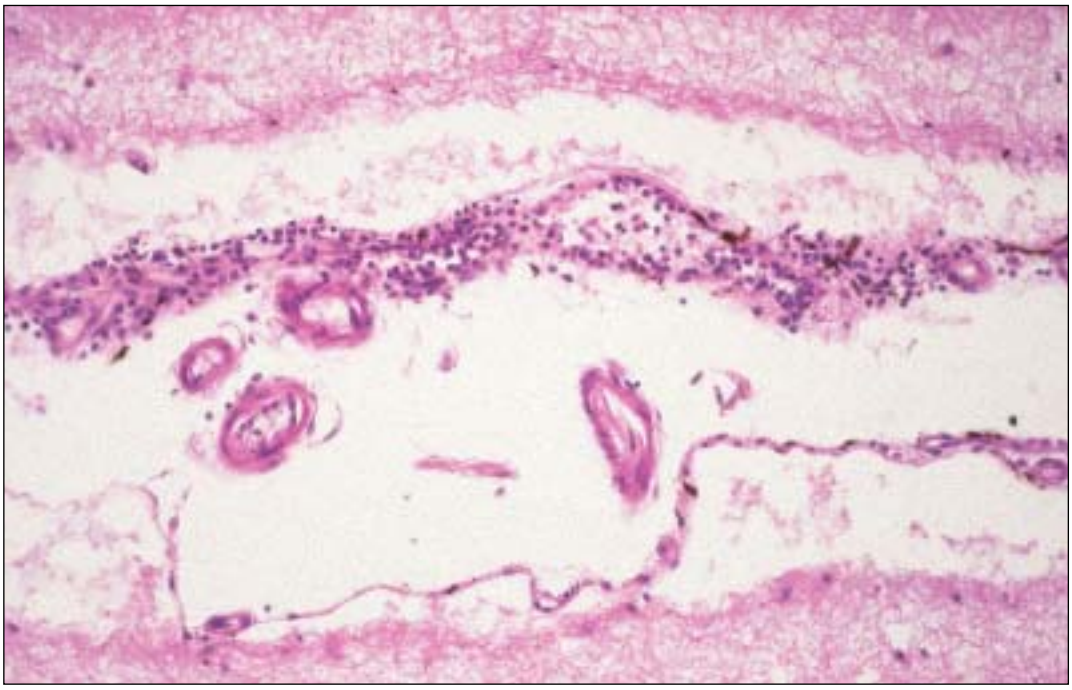

Fig. 3 - Parietal leptomeninges: foci of non-purulent leptomeningitis

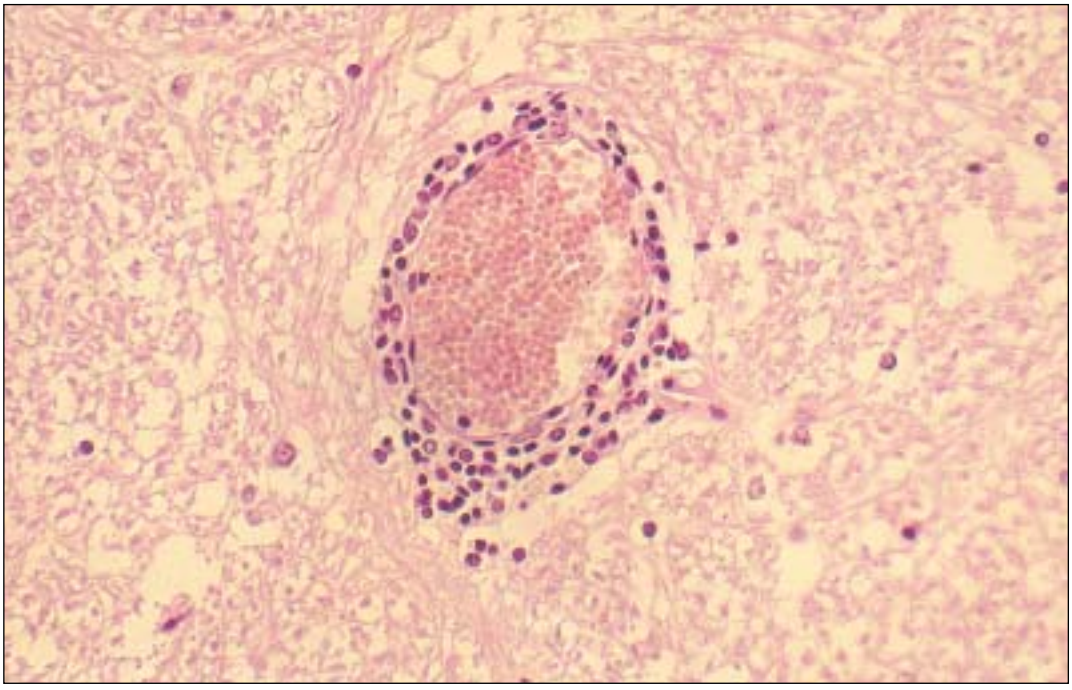

Fig. 4 - Cerebral hemispheres: perivascular cuffings

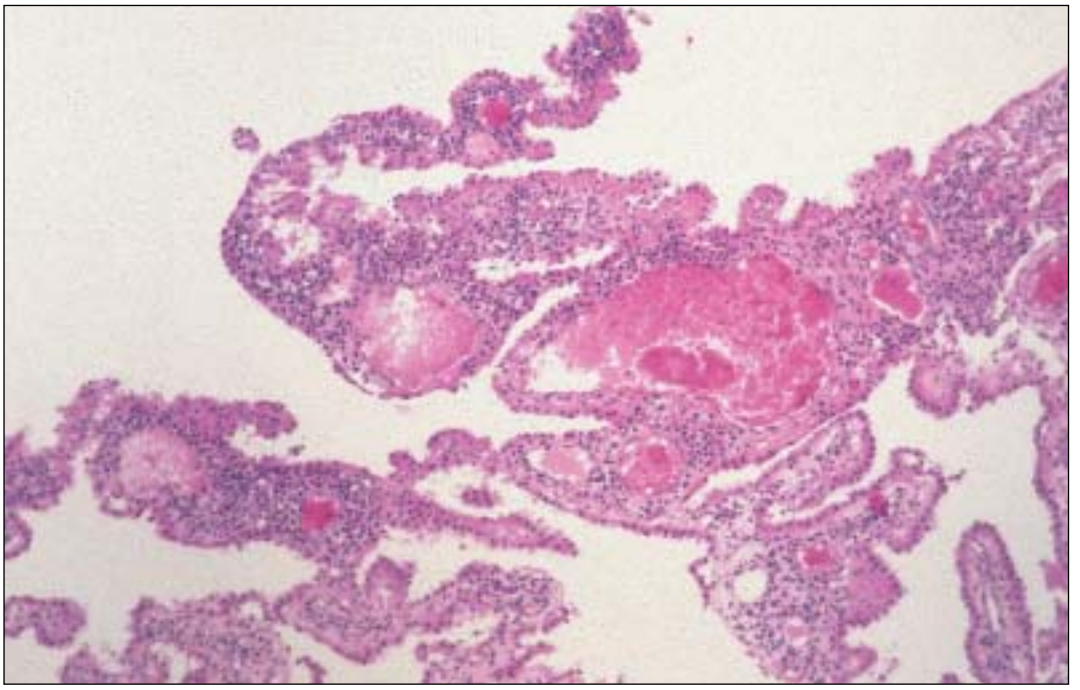

Fig. 5 - Cerebral ventricules: infiltrations of lymphocytes in the choroid plexus

(Vigliani & Rossi, 1982; Oudar *et al.*, 1982) but they should not be considered as primary agents.

The authors' observations allow the following considerations regarding the brain lesions:

a) the nervous symptoms might be attributed to the brain lesions;

b) the alterations can be considered as primary, *i.e.* induced by the infectious agent, or subsequent to secondary complications.

It must be pointed out that all the animals showing nervous symptoms observed by the authors revealed inflammatory lesions of the cerebellum. There is in fact a constant relationship between nervous symptomatology and lesions. Five chamois and one alpine ibex showed lesions of the central nervous system without showing any symptomatology. This could probably be explained by the fact that either the focal localization may enable a functional compensation or it does not damage the motor areas, leaving the functions unscathed. These processes could however be responsible of the behavioural modifications.

Regarding the second consideration, the non-purulent morphology of the inflammation would be a consequence of the action of the primary pathogenic agent, whether *Mycoplasma* or *Chlamydia*. If these lesions resulted from secondary complications, they would have to be purulent.

In conclusion our work shows the high incidence of brain lesions in wild ungulates affected by infectious kerato-conjunctivitis, and their presence also in subjects without nervous symptoms but with ocular lesions.

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