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**A CONTRIBUTION TO THE NEUROPATHOLOGY
OF THE RED-NECKED OSTRICH (STRUTHIO CAMELUS)
- SPONGIFORM ENCEPHALOPATHY -**

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Introduction

Since the first appearance of BSE in Great Britain in 1985 (review in TRUYEN & KAADEN, 1990), research into the incidence, diagnosis, differential diagnosis and epidemiology of spongiform encephalopathies in humans and animals has been a focus of medical and public interest. In view of the growing number of reports of "new" spontaneously or experimentally susceptible species (cats: WYATT et al, 1990; pigs: DAWSON et al, 1990), and of the associated questions with regard to the causal agent and in particular its transmissibility, it seems essential that agnopathogenetic individual cases should also be described. We therefore report below the preliminary findings of morphological examinations of three red-necked ostriches in 1986, 1988 and 1989, taking account of differential diagnostic factors.

History/subjects

The three ostriches (Flock A: Ostrich 1, female, adult, 150 kg; Flock B: Ostrich 2, female, adult, 80 kg; Ostrich 3: male, juvenile, 60 kg) came from two zoos in North West Germany and were euthanised because of their hopeless prognosis. Preliminary reports indicated that all three birds had presented protracted central nervous symptoms with ataxia, disturbance of balance and discoordinated feeding behaviour. Ostrich 2 had also exhibited pronounced lameness of the left lower limbs and the juvenile bird was suffering from perosis. The birds were fed on vegetable material, supplemented by commercial compound poultry feed and raw meat, some of which was obtained from local small emergency slaughterers. Comparable clinical pictures with fatal outcome in individual birds had occurred in both flocks: in a male bird at the same time (Flock A) and in several ostriches over recent years (Flock B).

Methods

Autopsy was followed in all three cases by histopathological examination of the following tissues: heart (several locations including coronary arteries and aorta), right and left pulmonary lobes, liver, kidneys, limb musculature, peripheral nerves (brachial plexus, sciatic nerve, in each case both left and right) and brain (left and right cerebral hemispheres, two samples each from the cranial/caudal third, two sagittal sections of the cerebellum, two cross-sections of the brain stem at the level of the optical lobes, four cross-sections from the medulla oblongata). The tissue material was fixed in formalin and embedded in Paraplast by the conventional method and the sections were evaluated using the following staining techniques and histochemical reactions: all organs: haematoxylin eosin staining; brain: PAS reaction (McManus), Ziehl/Neelsen staining (mod. Pearse), iron method (Lillie) for detection of neuromelanin, Turnbull's reaction (Bancroft & Stevens), alkaline Congo red method (Puchtler) (cf SCOON & SCHINKEL, 1986), myelin sheath staining (Spielmeyer) (ROMEIS, 1968). In addition, unstained sections were examined by fluorescence microscopy (to detect autofluorescing lipofuscin granula) and the following lipid stains were applied to cryostat sections of liver, and of heart and skeletal musculature: Sudan III, Sudan black, oil red.

Findings

Ostrich 1

Brain: Whilst only middle grade oedematization of the neuropil was noted in the cerebral and cerebellar region, major changes were detected in the brain stem and medulla oblongata (Figures 1-3): in addition to pronounced vacuolation of the grey matter, optically vacant, ovoid to spherical vacuoles of differing sizes occurred bilaterally symmetrically in numerous neurons of the brain centres nucleus ruber, vestibular nucleus and reticular formation, in certain cases compressing the Nissl substance into a narrow fringe. In addition, fine granular pigments were found in the perikaryon of the neurons (with and without vacuoles), which showed a golden brown coloration in the haematoxylin eosin specimen, gave positive reactions to both PAS and Ziehl-Neelsen and also exhibited a yellowish-green spontaneous autofluorescence. Lillie staining to detect neuromelanin gave a negative result. The pigments thus exhibited the characteristics of lipofuscin (SCHOON & SCHINKEL, 1986). Ferruginous pigments and histochemically detectable amyloids were absent. Mild gliosis, isolated necrotic neurons and neuronophagia were observed only in the cranial locations of the brain stem.

Other findings: The ostrich exhibited marked adiposity and multiple pressure sores of both lower limbs. Moderate steatosis was found in the heart and skeletal musculature and in the liver. Multifocal arteriosclerotic plaques were also noted in the coronary and limb arteries.

Ostrich 2

Brain: Histopathological changes in the brain of this ostrich were limited to the medulla oblongata and were qualitatively consistent with those found in Ostrich 1, although confined, bilaterally symmetrically, to small localised areas and affecting only individual neurons. Gliosis reaction was almost entirely absent.

Other findings: The carcass was moderately well nourished and exhibited multifocal dermal and muscular necroses on both lower limbs in conjunction with lateral chronically destructive tarsitis and coxitis. In the internal organs, parenchymatous degeneration of the liver and kidneys and multifocal arteriosclerotic plaques in the coronary arteries were noted.

Ostrich 3

Brain: Whilst no histopathological changes were found in the cerebrum and cerebellum of this ostrich, a high grade spongy dispersion of the neuropil existed in all locations examined in the brain stem and medulla oblongata (status spongiosus, Figure 4). Individual neurons contained optically vacant vacuoles of varying size, whilst numerous nerve cells exhibited clear signs of nuclear degeneration, in particular in the form of nuclear pyknosis. Low grade gliosis was also noted in all locations.

Other findings: The left lower limb of this bird exhibited defective positioning of the tarsal joint resulting from axial distortion of the long bones with appplanation of the lateral [Rollkamm - word not found] and resultant instability of the tendons and inward turning of the tarsus.

Discussion

Although ostriches are widely kept in zoos, there are virtually no detailed descriptions of central nervous disorders with associated locomotor disfunction in this species. Neurological symptoms have been reported in connection with an outbreak of Newcastle Disease (KLÖPPEL, 1969) and bacterial meningitis has been described (GRZIMEK, 1953), whilst other, sporadic cases have remained etiologically unexplained (ZUKOWSKY, 1959; LANDOWSKI, 1965). Disfunctions of the locomotor system of extracerebral origin occur predominantly in juvenile ostriches, emus and rheas in connection with muscular disease, perosis and trauma (FROIKA, 1982, 1983; MIHALIK & SRANK, 1982; SCHRÖDER & SEIDEL, 1989). One of the ostriches we examined was suffering from perosis, another from unilateral tarsitis and coxitis. All three, however, exhibited neuropathological findings consisting of a gradual, bilaterally symmetrical, spongiform encephalopathy of varying degree in the brain stem and medulla oblongata. No descriptions of such findings in this species appear in any of the literature we have been able to obtain.

These histopathologically confirmed brain changes are not consistent either with those caused by the classic viral infections in domesticated and wild birds or with those described by GRATZL & KÖHLER (1957) and CHEVILLE (1966) as typical of Vitamin E deficiency-related encephalopathy in chicks. Instead, at the light microscopy level, both in qualitative terms and in the pattern of distribution in the central nervous system, there is a high degree of coincidence with findings which occur in transmissible spongiform encephalopathies in mammals (scrapie, BSE, transmissible mink encephalopathy, chronic wasting disease of captive mule deer and elk) (HADLOW, 1961; BURGER & HARTSOUGH, 1965; HARTSOUGH & BURGER, 1965; WILLIAMS & YOUNG, 1980; WELLS et al, 1987, 1989).

The sporadic occurrence of vacuoles in individual neurons of the nucleus ruber in cattle was interpreted species-specifically as an artefact by FRANKHAUSER et al (1972). We are unable to judge whether a similar conclusion is also appropriate in the case of the ostrich, since our experience is based on only a small number of neuropathologically investigated cases. However, examination of the brains of twelve other ostriches which came to autopsy after death from extracerebral causes did not reveal any such findings. FRANKENHAUSER et al (1972) also emphasise that none were observed by them either in small ruminants or in the horse or the dog.

It is not possible at this time to determine whether and to what extent our neuropathological findings in an omnivorous bird, the ostrich, are etiopathogenetically consistent with those of the spongiform encephalopathies of mammals. There are no indications whatever in the relevant literature of even a hypothetical susceptibility in birds, although it must be said by way of qualification that clinical manifestations would be most unlikely in short-lived farm poultry, given the long incubation period. Moreover, Germany was officially free of scrapie and BSE at the time the condition appeared in the ostriches. The question of possible contamination of carcass meal is discussed in the work of TRUYEN & KAADEN (1990).

Conclusive diagnosis, especially in these cases, and in spite of the certainty ascribed by WELLS et al (1989) to histopathological diagnosis in cattle, also requires electron microscopic detection of so-called scrapie-associated fibrils (SCOTT et al, 1987; HOPE et al, 1988) and attempts, by inoculation of suspect brain material, to transmit the disease to the mouse (TRUYEN & KAADEN, 1990). Both of these procedures are normally carried out using fresh material, whereas we now have only tissue fixed in formalin and embedded in Paraplast.

Etiological consideration must also be given retrospectively to unidentified toxic influences, unknown species-specific deficiency diseases and unexplained predisposing metabolic conditions.

The etiologically unexplained neuropathological findings reported here, together with the multitude of unanswered questions in this connection, underline the need for further, systematic, standardised studies in this species, based on a larger sample of birds.

Summary and Literature

[Not translated]

Figures

Figure 1: Spongiform encephalopathy with oedematisation and vacuolation of the neuropil and "ballooning" degeneration of virtually all neurons in this area of the brain - brain stem. (H.-E.-Frgb., magnification x 120)

Figure 2: Detail of Figure 1. In addition to oedematisation of the neuropil, numerous, optically vacant vacuoles in the neurons, with partial displacement of the Nissl substance - brain stem. (H.-E.-Frgb., magnification x 480)

Figure 3: Medulla oblongata with high grade spongiform dissociation of the neuropil. (H.-E.-Frgb., magnification x 300)

Figure 4: Medulla oblongata. Status spongiosus with neuron degeneration. (H.-E.-Frgb., magnification x 300).