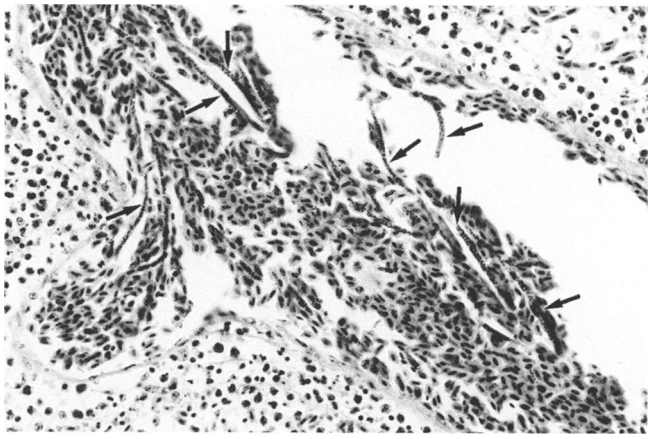

FIG 2: Pulmonary blood vessel showing large numbers of microfilariae (arrowed). Haematoxylin and eosin x 297

1908. Plimmer (1912) described infection in 74 species of birds and Markbreiter (1923) found microfilariae in 21 species. In most of these cases little or no attempt was made to establish whether the infection was associated with pathological changes, and Plimmer (1912) commented 'microfilariae are the least harmful to their host of any of the blood parasites, and all the pathological effects we know of filariae are due to the adult worms, which have been found in only a small proportion of the cases here'. Nevertheless, it is of particular interest to note that in these reports, red billed blue magpies (referred to by the synonym occipital blue pie [*U melanocephala occipitalis*]) are repeatedly mentioned as dying with microfilarial infections and there is also a case of infection in a yellow billed blue pie (*U flavirostris*) (Plimmer 1912, 1913, 1915, Markbreiter 1923, Hamerton 1931, 1934, 1935, 1938, 1939, 1943, Hamerton and Rewell 1948, Rewell 1948). Furthermore, in one such report an occipital blue pie is said to have died with a 'mass infection' of microfilariae (Hamerton 1931) and in another report microfilariae were recorded in the blood of two occipital blue pies, both of which also had filarial worms in their lungs (Rewell 1948).

The filarial worms seen by Rewell (1948) in association with microfilariae in the blood were not identified, although on three other occasions nematodes found in the body cavities of blue pies were identified as *Diplotriaena* species (Markbreiter 1923, Hamerton 1934, Hamerton and Rewell 1948). In the present case it was not possible to identify the nematodes seen in the histological sections of lung, although they were very slender and quite unlike those of the family Syngamidae, which are occasionally seen in parabronchi. In the authors' experience the presence of such nematodes is unusual and it seems likely that they were associated with the microfilarial infection. Filarioid worms of the genus *Chandlerella* have been recorded in red billed blue magpies (referred to by the synonym *U sinensis*) in China and in various other corvids, including large billed crow (*Corvus macrorhynchus*) in India (Anderson and Freeman 1969) and black billed magpie (*Pica pica hudsonia*) in America (Anderson 1992).

Despite the dearth of recent evidence of microfilarial infection in captive birds the historic records, together with the present case, suggest that such infection may not be uncommon, particularly within the genus *Urocissa*. Where such birds become ill it is recommended that blood samples are taken and stained films are checked for the presence of microfilariae, as it is likely that one of the modern anthelmintics may well prove effective in the treatment of the condition. Further investigations are also needed to determine the identity of the parasite. The origin of the infection in the present case is unknown, but the birds had been purchased from a dealer 12 months prior to being moved to Cornwall, and it is likely that they had been imported. In that case the infection could have been acquired overseas, but the possibility of a reservoir of infection in British wild birds cannot be ignored. Although the magpie (*P pica*) would seem a likely reservoir host, blood samples from 21 birds, taken over a two year period, all proved negative (V. R. Simpson, unpublished data).

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Meningeal carcinomatosis in a dog

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MENINGEAL carcinomatosis or carcinomatous meningitis is an uncommon pathological condition defined as a difuse leptomeningeal infiltration by extraneural solid tumour cells that have metastasised to the central nervous system (Grossman and Moynihan 1991). Most of the primary tumours causing meningeal carcinomatosis are adenocarcinomas, mainly of pulmonary (40 per cent) and mammary (30 per cent) origin (Pérez de Colosía and others 1994). Meningeal carcinomatosis has been described as developing in 5 per cent of mammary tumours (Yap and others 1978) and 18 per cent of oat-cell carcinomas in the lung (Aroney and others 1981).

In man, the real frequency of meningeal carcinomatosis is unknown, but is estimated to be a complication of 5 to 8 per cent of solid tumours, in which case it would represent the third most frequent neurological complication of malignant tumours, following cerebral metastasis and spinal cord compression (Pérez de Colosía and others 1994).

In veterinary medicine to date, only one reference exists about the occurrence of this entity, related to a case of hydrocephalus (Jubb and Huxtable 1993).

A 10-year-old female crossbreed dog was presented with anorexia, polydipsia, polyuria, left eye mydriasis and disminished left pupilar light response. Four days later mydriasis was complete, the pupilar light response had disappeared in both eyes and the dog had become ataxic. Three days later the dog developed a vestibular syndrome showing constant horizontal nystagmus. The animal did not respond to treatment and, due to the poor prognosis and evolution of the disease, was euthanased.

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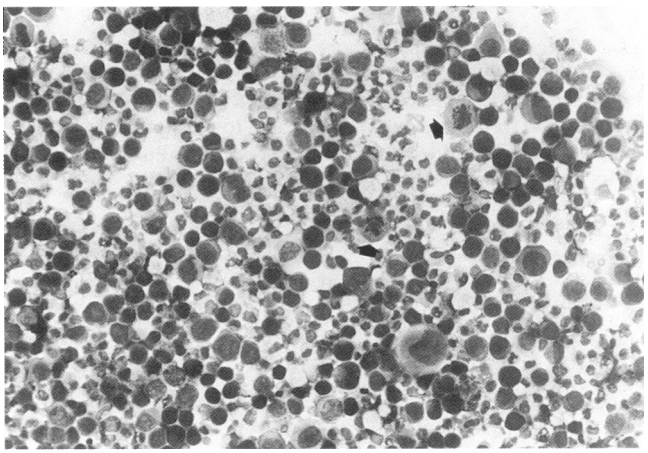

FIG 1: Cerebrospinal fluid cytology showing a mixed population of epithelial neoplastic cells and imflamatory cells. Note mitosis in neoplastic cells (arrows). Diff Quick stain x 29

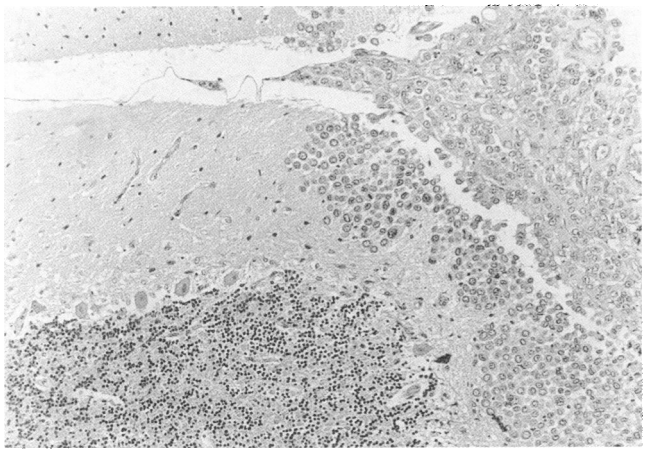

FIG 2: Presence of neoplastic cells in the cerebellar leptomeningeal space showing an invasive pattern towards the neural parenchyma. Haematoxylin and eosin x 14

Necropsy was performed immediately after euthanasia as was a cytospin cerebrospinal fluid cytology test (Fig 1). An intense pleocytosis of a mixed cell population was observed. Most of the cells were neoplastic epithelial-like round cells, with round nuclei containing one or more nucleolar forms and basophilic cytoplasm; numerous mitotic figures were observed. The second population consisted of mononuclear cells, mainly lymphocytes.

Macroscopically, the only evident lesions were several well encapsulated nodes up to 3 cm in diameter in the mammary gland. Tissue samples were fixed in 10 per cent buffered formalin for routine histopathological analysis.

Microscopically, the mammary nodes corresponded to glandular epithelium proliferation showing a variable growth pattern from hyperplastic to papillar, alveolar or solid, in different areas. In solid areas cells were anaplastic and undifferentiated, with a polyhedrical shape and a highly variable size. Cells showed a basophilic cytoplasm and pale nuclei with marginated chromatin and evident nucleolar forms in a variable number. Multinucleated cells were frequent and numerous mitotic and apoptotic cells were observed. Several foci of mononuclear inflammatory cells surrounded the tumour. An anaplastic solid adenocarcinoma was diagnosed. Inguinal lymph nodes had been invaded by these cells which had disrupted the structure of the nodes. From this location cells had metastasised to the lung, adrenal gland, uterus and central nervous system including the eye. In the lung, several foci of neoplastic cellular proliferation were seen; the cells were located at alveolar walls and in some areas grew in a solid form. Some were seen inside capillary vessels. In the adrenal gland, neoplastic cells were located at the fascicular zone of the cortex. In the uterus, neoplastic cells infiltrated the endometrium and produced endometrial gland dilatation. In the eye, the cells surrounded the optical nerve in between the perineurium and the leptomeninges, producing compression of the nerve. The neoplastic cells did not infiltrate any eye structure apart from the iris where they were located in the caudal border, close to the choroid coat, in a highly vascularised area. In the central nervous system the neoplastic cells were mainly located at the leptomeninges surrounding the brain. In several locations the cells proliferated, even showing alveolar-like structures, and invaded the encephalic parenchyma (Fig 2). In the mesencephalon, thalamus and cerebellum small foci of neoplastic cellular proliferation were seen in white and grey matter, usually associated with capillary vessels.

Due to the major presence of neoplastic cells in the leptomeninges, a meningeal carcinomatosis was diagnosed. The similarity between the neoplastic cells located in the mammary gland and in the leptomeninges suggested that the primary source of the meningeal carcinomatosis was the mammary tumour.

It is not known how the neoplastic cells reached the leptomeninges. There are several hypotheses about the pathological mechanism of meningeal carcinomatosis (Russell and Rubinstein 1989). The most accepted explanation is that encephalic metastasis occurs first followed by subsequent cerebrospinal dissemination. In the present case this would be the most feasible explanation, since several metastatic foci were found in the encephalic parenchyma, always close to a blood vessel. However, the absence of metastatic foci in several cases cited in the literature indicates that metastasis can involve the leptomeninges directly and spread from there (Russell and Rubinstein 1989). Another source of spread could be metastasis in the choroid plexuses; again, in the described case, metastatic foci were found very close to the fourth ventricle choroid plexus. Finally, another route that has been proposed as a way of entry to the central nervous system is via the perineural lymphatics of spinal nerves (Russell and Rubinstein 1989).

In man, the clinical diagnosis of meningeal carcinomatosis is made through cerebrospinal fluid examination; only 3 per cent of patients suffering from meningeal carcinomatosis show a normal cerebrospinal fluid analysis, which includes cytology and biochemistry. Despite no neoplastic cells being found on cytological examination, an increase in the fluid pressure and in the values of glucose and proteins are constant features (Grossman and Moynihan 1991).

A reverse transcriptase-polymerase chain reaction analysis has been described to detect mRNA for keratin 19, for diagnosing occult mammary tumours (Datta and others 1994). To confirm the origin of the meningeal carcinomatosis a PCR of cerebrospinal fluid should be run.

This study provides evidence of the occurrence of meningeal carcinomatosis in the dog. For its diagnosis cerebrospinal fluid cytology is needed. It has to be emphasised that the absence of such cytology in the diagnostic procedure for a central nervous system disorder prevented an in vivo diagnosis in this case. Further use of this simple technique should be promoted, since it provides useful information for the differential diagnosis of central nervous system diseases.

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