Magnetic Resonance Imaging and Histopathology of Encephalitis in a Pug

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ABSTRACT. A female 3-year-old Pug showing frequent epileptic fits during 8-months was shown on magnetic resonance imaging (MRI) to have dilated cerebral ventricles and inflammatory lesions in the whole cortex of the bilateral cerebral hemispheres as well as the lower left parietal and the middle right temporal lobes. Histopathology of the corresponding regions revealed meningoencephalitis characterized by wide distribution of degenerated neurons with glial satellitosis and neuronophagia and prominent perivascular cuffing with lymphoid cells.

— KEY WORDS: encephalitis, MRI, Pug


The pug encephalitis is characterized by chronic granulomatous encephalitis occurring in Pug breed dogs of both sexes aged 9 to 19 months [3, 6]. The disease has been reported in several countries including Japan [1, 2, 4], and similar lesions were recognized also in other canine breeds [5, 6]. Central nervous signs appear 1 to 6 months before death, showing shrill crying, epileptic fits, paralysis, incoordinate circling or movement, depression and coma. Neurologic examination reveals disorders in the posture adjustment and visual sense, while pupil reflection remains normal. Some affected animals show the central vestibular as well as cerebellar signs. No response to any antileptics and corticosteroids has been observed. Histopathologically granulomatous inflammation is seen in the cerebrum with lymphocytic meningitis. No etiologic agents have been isolated except for distemper virus from some vaccinated animals. This brief note is to describe a case of so-called Pug encephalitis successfully diagnosed by clinical signs and magnetic resonance imaging (MRI).

The patient was female 3-year-old Pug weighing 4.7 kg with frequent epileptic fits for the recent 8 months, in maximum, with seven fits occurring within a day one week before coming to the clinic. Distemper vaccination had been made once a year. Appetite and digestion were normal but there was some weakness and upper central nervous signs. She was subjected to MRI of the brain using an MRT-50GP (FLEXART: Toshiba-Medical, Tokyo) and then to postmortem pathology after euthanasia. For imaging blood vessels Gadolinium-DTPA (Gd-DTPA; 0.3 ml/kg, Schering, Tokyo) was injected intravenously. For imaging blood vessels Gadolinium-DTPA (Gd-DTPA; 0.3 ml/kg, Schering, Tokyo) was injected intravenously. The T1- and T2-weighted images were obtained at repetition time (TR) of 350 msec and echodelay time (TE) of 15 msec and at TR of 4,000 msec and TE of 120 msec, respectively.

The transversal (Fig. 1) and dorsal (Fig. 2) MRI of the brain showed irregularly dilated both ventricles, mostly at the right temporal regions. On T1- (Fig. 1a) and T2-weighted images (Fig. 2b), the low and high signals, respectively, were seen at the whole cortex of the both cerebral hemispheres as well as the lower left parietal and the middle right temporal lobes. Also at the middle- or lower-left and upper-right parietal lobes, there existed the T1-low (Fig. 2a) and T2-high (Fig. 2b) regions. In the midline area of the right frontal and occipital lobes, some T2-low and T2-high regions were present. The T1-lower or high and T2-high regions were observed also in another part of the left parietal lobe. Some vascularization was revealed within the cortical lesions with T1-low and T2-high signals on MRI of the patient after Gd-DTPA injection (Fig. 3).

On MRI of the right cerebral hemisphere, the cerebral midline as well as gyri were remarkably shifted to the right, suggesting that the cerebral lesions might have developed to a large extent from the middle cortex, resulting in more advanced atrophy of the right hemisphere than the left showing severe edema at autopsy.

At autopsy the animals was shown to have thick subcutaneous fat. No abnormal fluid accumulation was seen within the thoracic or peritoneal cavity. In the trachea and bronchi foamy exudate was present, and bloody fluid flew out from the cut surface of the lungs. The duodenal and colonic mucosa showed petechiae containing yellow viscous fluid. In the cerebral cortex there were several soybean-sized foci of malacia and calcification.

Histopathologically a number of degenerated pyknotic neurons were distributed in the temporal and dorsal occipital cortices of the right brain frequently with glial satellitosis and neuronophagia (Fig. 4). There was diffuse microglial proliferation and some rod-shaped cells. Perivascular cuffing with lymphoid cells were prominent (Fig. 5). Small malacic foci were seen in association with gemistocyte formation. A few submeningeal foci of calcification were present with perivascular infiltration of histiocytes and lymphocytes. There were no inflammatory lesions in the brain stem and around the fourth ventricle nor in the cerebellum. No inclusions were detected in any neurons and glia cells.

The heart showed some thrombi within the coronal venules, and many fat cells were distributed between myocardial fibers and under the pericardium. In the spleen lymphoid follicles were atrophied and histiocytes were markedly proliferated in the red pulp sinuses. The bronchial glands were hyperplastic and the space contained serous exudate, defoliated epithelial cells, neutrophiles, histiocytes and lymphocytes. The type II alveolar cells were proliferated and the congested interstitium showed some fibrosis and cellular infiltration.
Fig. 1. Transverse T1 (a)- and T2 (b)-weighted plane MRI of the brain, showing T1-slightly high and T2-high sporadic lesions as well as right-shifting of the midline due to their enlarged right ventricle.

Fig. 2. Dorsal T1 (a)- and T2(b)-weighted MRI of the brain, showing a T2-high area suggestively edematous and shifting the midline to right.

Fig. 3. Dorsal MRI after intravenous injection of Gd-DPTA (0.3 ml/kg), indicating sporadic granulomatous encephalitis with some vascularization and irregular-shaped dilatation of the lateral ventricles without enhanced images.
No significant changes were observed in the intestines. The congested liver showed dissociation of hepatocyte cords as well as dilatation of the sinusoids and spaces of Disse. Some hepatocytes were vacuolated containing prominent eosinophilic inclusions in the cytoplasm. No lesions were seen in the pancreas.

In the kidneys the glomerular basement membrane was thickened and the mesangial areas were extended. The proximal tubular epithelial cells were vacuolated with interstitial fibrosis, and the distal tubules contained PAS-positive cylinders. The adrenals showed no detectable changes.

The present case was clinically suggested by MRI to be of so called Pug dog encephalitis, and histopathology revealed characteristic non-purulent meningoencephalitis. Similar clinical signs might be recognized in necrotic encephalitis (NE) of Maltese and Yorkshire terriers, but the Pug encephalitis might be differentiated from NE in MRI by T1-weighted images with Gd-DTPA enhancement. The difference in the MRI seems to be clear though it depends on the duration of the sickness.

Virus isolation was negative in this case, and the cause of pug encephalitis remains unknown.

REFERENCES