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CASE REPORT

# A Fatal Case of Acute Steroid Responsive Meningitis Arteritis in a Dog

Kwiatkowska Miłosława*, Andrzej Pomianowski and Zbigniew Adamiak[1]

Internal Medicine Department; [1]Surgery and Roentgenology Department, Warmia and Mazury University in Olsztyn, Poland
*Corresponding author: miloslawa.kwiatkowska@uwm.edu.pl

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**ABSTRACT**

Nine years old female boxer was evaluated for progressive tetraparesis, cranial nerve deficits, lethargy and stupor. Serum biochemistry revealed elevated liver enzymes alanine aminotransferase (ALT) 379 u/l, asparaginian aminotransferase (AST) 55 u/l, alkaline phosphatase (ALP) 685 u/l and creatinine kinase (CK) 511 u/l, and elevated acute phase proteins C-reactive protein (CRP) 70 mg/ml. The cerebrospinal fluid examination revealed a mixed pleocytosis, protein content elevation (315 mg/dl) and positive Pandy reaction. Immunoglobulin A (IgA) in CSF was elevated (357 µg/ml), CRP was characteristic for a presentation period of disease (70 mg/ml). MRI examination revealed multifocal, diffused lesions in grey matter of frontal, occipital lobes. The white matter lesions were detected in cranial fossa region, thalamus, medulla oblongata and pons. The lesions were enhanced after contrast administration. Ventricular system was compressed; sediment was present in both of them. The biochemistry CSF results and MRI image were characteristic for steroid responsive meningitis arteritis (SRMA).



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## INTRODUCTION

Steroid responsive meningitis arteritis (SRMA) is an inflammatory disease occurring mainly in young, large breed of dogs such as béarnaise mountain dog, boxer (Wrzosek *et al*., 2009), border collie, Nova Scotia Retriever (Anifinsen, 2008). The disease was first recognized in beagles and was named "beagle pain syndrome" (Tipold and Jaggy, 1994). It is believed to be an immune mediated disease due to facts as: it responses to steroidal therapyassociated withelevated IgA levels in CSF and serum, and no infectious organism are detected (DeLahunta, 2008). The disease has two clinical forms: acute ("classic") and, chronic (protracted) (Tipold and Jaggy, 1994; Tipold and Schnatzberg, 2010). The most common symptoms are neck pain and rigidity, depression, pyrexia, stiff gait, ataxia and para/tetraplegia. Common hematobiochemical alterations may include mild leukocytosis with left shift, lymphopenia, eosinopenia, a mildly increase of ALP, AST activity and cholesterol, globulins concentration. Examination of CSF usually reveals mixed pleocytosis (neutrophils and mononuclear cells) and positive Pandy and None-Appelt reactions. Prognosis depends upon severity of the disease (clinical form) and response to treatment. The present report represents a fatal case of SRMA in a dog diagnosed by CSF analysis and magnetic resonance imaging (MRI) examination.

## HISTORY

Nine year old intact female boxer, weighing 25 kg was referred to the Teaching Hospital of Warmia and Mazury University, Olsztyn for the treatment of two weeks old progressive weakness, stiff gait and ataxia. Historically, the patient had been treated with cephalexin (Inj. Ceporex, Schering-Plough Animal Health, UK; @22 mg/BW s. c., q. 12h), and carprophen (Rimadyl, Pfizer Animal Health, UK; @ 4 mg/BW p. o., q. 24h) by a private veterinary practitioner. Administration of tramadol hydrochloride (Inj. Tramal, Grunenthal, Poland; @ 5 mg/ BW i. m., q. 8h) was ineffective to relieved the pain. In view of a transitory mild improvement in the condition of patient after a treatment with dexamethasone (0.2mg/Kg BW, s.c.; Inj. Rapidexon, Novartis Switzerland), the case was referred to Warmia and Mazury University Teaching Hospital, Olsztyn, Poland for diagnosis and treatment.

**Clinical examination, diagnosis and treatment:** Patient's rectal temperature was 101.3°F (38, 5). Physical examination revealed rigidity of neck with severe pain reflex, atrophy of temporal and front limbs muscles. Orthopedic examination was insignificant. The neurological examination revealed proprioceptive deficits and absence of deep pain sensation in all four limbs. The cranial nerve examination indicated multiple deficit and an absent menace response in left eye. Hopping and hemiwalking reactions were not examined owing to nonambulatory dog's state. Spinal cord reactions were mildly exaggerated on front limbs. Mentation was abnormal, a stupor was observed. The superficial sensation was diminished on the left head side, especially in the ear and nose region. The survey radiograph of cervical region did not reveal any abnormalities. An abdominal ultrasonogram revealed a slight hepatomegaly and thickened gall bladder wall with 2mm cholelith. Serum biochemical profile revealed elevated liver enzymes (ALT 379 u/l; AST 55 u/l; and ALP 685 u/l). A complete blood count revealed a mild leukocytosis with a left shift.

The CSF (cerebrospinal fluid) was collected through atlano-occipital puncture under general anesthesia. The sample was transparent and xanthochromic after centrifugation. Laboratory examination of CSF indicated elevated protein (315mg/dL; positive Pandy reaction). Cytological examination showed pleocytosis of mixed type (Fig. 1). The CSF was found negative for bacteria, *Neospora caninum, Toxoplasma gondii* and canine distemper virus. Based on the clinical and laboratory examinations, a presumptive diagnosis of SRMA was made. Elevated levels of IgA in CSF (357 µg/ml; reference range 0-0.2µg/ml) and serum (352 µg/ml; reference range), serum amyloid A (SAA) (2270 mg/mL; reference range 0-1 mg/mL), and C reactive protein (70mg/ml; reference range 0-1 mg/ml) confirmed the existing diagnosis as SRMA. Magnetic resonance imaging (MRI; Vet-MR Grande, 0.25 T, Milano Italy) examination of head (sequences FLAIR, T1, T2 and T1 post contras) showed multifocal and diffuse lesions in the cerebral grey matter of olfactory region, frontal and occipital lobes. In the T2 and FLAIR sequence, diffuse hyperintense lesions were detected in grey and white matter in cranial fossa

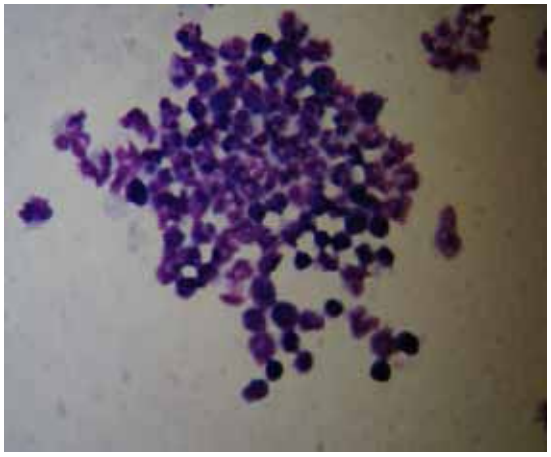

**Fig. 1:** A photomicrograph of Giemsa stained CSF smear indicates granulocytic infiltration with mononuclear cells.

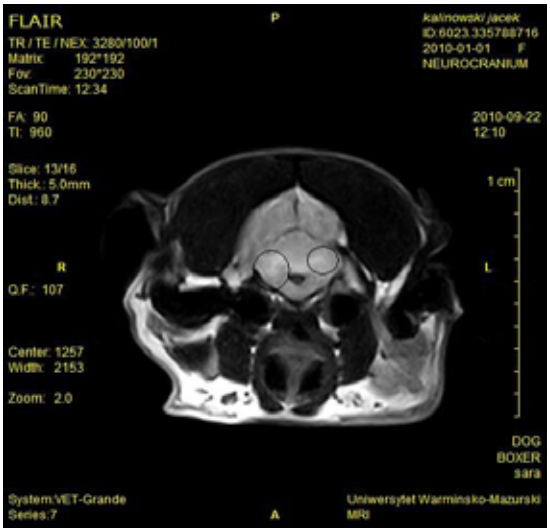


**Fig. 2:** FLAIR image, in transverse plane. Hyperintensive lesions in the pons region.

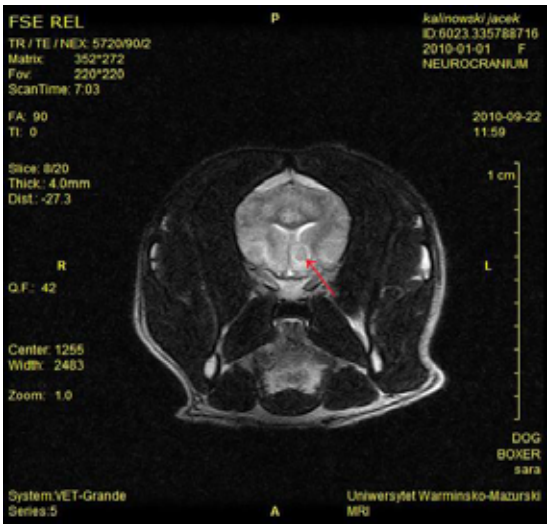


**Fig. 3:** T2 weighed image, in transverse plane. A ring shape enhancement visible in the optic chiasm region.

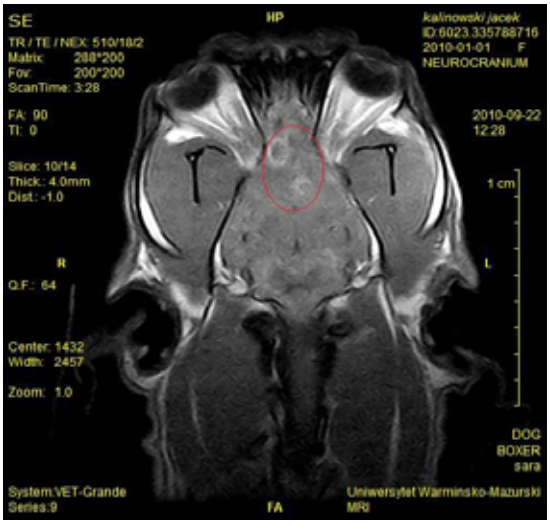


**Fig. 4:** TI image, in transverse plane after contrast administration. A ring shape lesions in the frontal region.

region, thalamus (Fig. 2), pons (Fig. 3), medulla oblongata. In T1 post contrast sequence, several hyper-intense ring shape lesions of varying sizes were noted. The largest focus had 9 mm diameter in the cranial fossa region (Fig. 4). The ventricles were found asymmetrical (right was smaller), compressed surrounded by the hyper-intense rim. The features of sediment presence were also found in right and left ventricle. The mass effect was noticed.

## DISCUSSION

The symptoms like intensive neck pain and rigidity, fever, lethargy and the course of the disease are very much characteristic for the SRMA, and are very much like those described in the literature (Tipold and Schnatzberg 2010; Lowrie, 2011). Also the pedigree (Boxer) is said to be predisposed to this disease (Cizinauskas and Jaggy 2000). To our knowledge the oldest dog described in literature was 7 years old whereas our patient was 9 years old. Information we obtained from anamnesis may possibly exclude former onsets of the disease; unless they had been mild enough to be missed. The fact that SRMA more often concerns young dogs, what strongly suggest that mechanism is immune-mediated, because the immune system is more easily activated in young dogs (DeLahunta 2008). The late onset in our patient may confirm boxers to be highly predisposed to SRMA. The other case report of SRMA in 7 year old boxer (Wrzosek *et al.* 2009) documented similarly MRI lesions and onset of the disease to our case. Maybe a conclusion that old boxers are more predisposed to steroid responsive meningo-encephalitis (SRME) than SRMA should be made. Cerebrospinal fluid examination is was indicative for an inflammatory central nervous system disease. The acute phase protein confirmed the SRMA suspicion; moreover they were characteristic for presentation phase of disease (Lowrie *et al*., 2009). The IgA was elevated in the serum (352 µg/mL) and CSF (357 µg/mL), as was CRP (70 mg/mL). SAA was very high, as described in the presentation phase of SRMA, 2770 mg/mL. This fact proves first disease onset in such an old dog. The relapse phase seems to be unlikely due to high 2770mg/mL SAA concentration (reference range for disease relapse phase 11, 6- 480, 5 mg/mL) (Lowrie *et al*., 2009). Cerebro-spinal fluid TNCC confirmed positive correlation with CRP serum concentration (Lowrie *et al*., 2009). MRI examination revealed diffused hyper-intense lesions in the thalamus, pons, and medulla oblongata cranial fossa region. In T1 post contrast images, the lesions were hyperintense that may be indicative of inflammatory disease. The asymmetry of ventricles may be due to its compression and nerve tissue malacio the hyper-intense. The sediment presence was in right and left ventricle is compatible with cerebral extension of steroid responsive meningitis arteritis (Wrzosek *et al*., 2009).

In comparison to other diagnostic techniques, MRI provides superior information about anatomy of the central nervous system; it may only indicate the type of anatomic alterations, pathological lesions. It must be remembered that some neurologic diseases result in microscopic lesions that may not be detected by MRI alone. Therefore cerebrospinal fluid examination has been historically used as a routine examination in inflammatory, infectious, neoplastic, traumatic, vascular, degenerative diseases nervous system diseases. Most CSF abnormalities are nonspecific and do strictly indicative for the diagnosis. Whereas in SRMA the examination has a higher diagnostic value in confirming suspicion of disease than MRI does. The conclusion we made, is that the CSF analysis was most helpful in detecting the inflammatory type of CNS disease, and excluding the neoplastic character of disease. It also needs to be considered that CNS inflammation may be a secondary finding, but then pleocytosis is usually not that high. (Anifinsen, 2009). High protein concentration may be indicative for neoplastic (26-1953 cells/ul) and inflammatory (27-1500 cells/ul) diseases, therefore an acute phase protein was evaluated and MRI examination was performed. The conclusion that CSF examination in SRMA is more specific and sensitive for the disease than MRI was made, but it also needs to be supplemented with acute phase protein examination in serum and CSF.

Administration of carprophen in dose 4 mg/kg of body weight, non-steroid anti-inflammatory drug, tramadol hydrochloride in dose 5 mg/kg of body weight did not help to diminish pain symptoms. A mild clinical improvement was achieved by a local vet next day with dexamethasone injection in the dose 4 mg/dog injected subcutaneously. When dog was referred to Clinical hospital of Warmia and Mazury, Olsztytn, Poland it was given 2 mg of prednisone per kg of body weight twice a day. The neurological status of a patient improved, but only after two days it deteriorated. The course of disease was acute (4 days), but strangely a mixed pleocytosis was detected, which is usually associated with a chronic, protracted form. The conclusion that severity of the neurological deficits and vast pathological lesions infiltrating the grey and white brain matter visible on MRI images confirm that only a quick diagnosis and treatment with glucocorticoids can improve the animal status.

The limitations in this case include lack of histopathological examination as owner disagreed to postmortem examination and lack of patient's parents/sibling history, what might have provided information for family predisposition to SRMA/ SRME.

## REFERENCES

Anifinsen KP, 2008. A retrospective epidemiological study of clinical signs and familial predisposition associated with aseptic meningitis in the Norwegian population of Nova Scotia duck tolling retrievers born 1994-2003. Can Vet J, 72: 350-355.

Cizinauskas S and A Jaggy, 2000. Long term treatment of dogs with steroid responsive meningitis-arteritis: clinical, laboratory and therapeutic results. J Small Anim Pract, 41: 295-301.

De Lahunta A and E Glass, 2008. Veterinary neuroanatomy and clinical neurology. 3rd Ed, Elsevier Science, Amsterdam, pp: 525.

Lowrie M, J Penderis, PD Eckersall, M McLaughlin, D Mellor and TJ Anderson, 2009. The role of acute phase proteins in diagnosis and management of steroid responsive meningitis arteritis in dogs. Vet J, 182: 125-130.

Tipold A and A Jaggy, 1994. Steroid responsive meningitis arteritis in dogs: long term study of 32 cases. J Small Anim Pract, 35: 311-316.

Tipold A and SJ Schatzberg, 2010. An update on steroid responsive meningitis-arteritis. J Small Anim Pract, 51: 150-154.

Wrzosek M, M Konar, M Vandevelde and A Oevermann, 2009. Cerebral extension of steroid-responsive meningitis arteritis in a boxer. J Small Anim Pract, 50: 35-37.