Association of Avian Veterinarians Australasian Committee Ltd.
Annual Conference 2016 pp 46-48

Sarchocystis calchasi - a New Threat for Pigeons and Psittacine Birds

Michael Lierz
Clinic for Birds, Reptiles, Amphibians and Fish,
Faculty of Veterinary Medicine, Justus Liebig University,
Frankfurter Straße 91-93
35392 Gießen, Germany
michael.lierz@vetmed.uni-giessen.de

Sarcocystis calchasi is the causative agent of Pigeon Protozoal Encephalitis (PPE) a severe neurologic disease with high mortality rates in domestic pigeons. From 2006 to 2008, *S. calchasi* was recognized for the first time in Northern Germany in several pigeon flocks with neurologic signs (e.g. head tilt, ataxia, torticollis, paresis and paralysis) and losses of up to 25 (Olias et al., 2009b). Infections with PMV-1 and Salmonella spp. which represent the major differential diagnosis had been excluded. Sarcocysts were detected in the skeletal muscle of the affected pigeons and a granulomatous encephalitis was present (Olias et al., 2009a). Subsequently, European Accipiter hawks (Northern goshawks (*A. gentilis*) and sparrowhawks (*A. nisus*)) were identified as definitive hosts of *S. calchasi* (Olias et al., 2011). Dogs (*Canis lupus familiaris*), ferrets (*Mustela puterius furo*), rats (*Rattus norvegicus domesticus*), mice (*Mus musculus domesticus*) and Gyr-Saker hybrid falcons (*Falco rusticolus* x *Falco cherrug*) did not shed *S. calchasi* sporocysts after experimental infection (Olias et al., 2010a).

Since its description in 2009, *S. calchasi* has been reported also in pigeons with virtually identical PPE in the United States indicating a worldwide distribution of the parasite (Wünschmann et al., 2011; Olias et al., 2014). Recently, several psittacine species have been demonstrated as intermediate hosts of *S. calchasi* in natural and experimental infections (Rimoldi et al., 2013; Olias et al., 2014). The psittacine birds developed a disease that closely resembled PPE in pigeons. Despite the exclusion of chickens as intermediate hosts, *S. calchasi* might be capable of using a broad spectrum of intermediate hosts since psittacines and pigeons are only distantly related.

In the intermediate hosts (pigeons and psittacine birds) a biphasic disease occurred after experimental infection with *S. calchasi* sporocysts (Olias et al., 2010b; Olias et al., 2014). An acute phase with apathy and polyuria began at day 10 post infection (dpi) which was either followed by a complete recovery or death. This phase correlated with the first schizogonic phase of *S. calchasi* infection with asexual replication located mainly in liver and spleen. Central nervous signs like ataxia, head tilt and paresis developed around dpi 50. During this phase, mature sarcocysts were observed in the skeletal muscle and a granulomatous, necrotizing encephalitis was present. In pigeons, the development of clinical signs was dose-dependent. Pigeons with medium infectious doses (103 to 104 sporocysts) demonstrated both clinical phases. Pigeons with low infectious doses (102 sporocysts) developed only a chronic phase with neurologic signs and pigeons infected with high numbers of sporocysts (8 x 104 to 3 x 106) died during the acute phase (Olias et al., 2010b). In contrast, cockatiels (*Nymphicus hollandicus*) developed similar clinical signs of varying severity which was independent of the infectious dose (Olias et al., 2014). This difference between pigeons and psittacine birds was suggested to be the consequence of an evolutionary younger parasite-host relationship in psittacine birds (Maier et al., 2013).

Despite severe encephalitis during the chronic phase of PPE, parasite stages have rarely been identified in the CNS of some experimentally infected animals so far. These schizonts and sarcocysts were not associated with the inflammatory lesions (Olias et al., 2013). In naturally infected pigeons in the USA, however, intralesional schizonts were detected in some diseased pigeons (Olias et al., 2014). Since re-infection and strain differences cannot be ruled out in those natural infections, the direct role of *S. calchasi* in the development of CNS lesions remains unclear. Other Sarcocystis spp. causing encephalitis in avian species, including the extensively described *S. falcatula*, are usually associated with the schizogonic phase of the parasite's life cycle. In chickens (Mutalib et al., 1995) a cockatiel (Jacobson et al., 1984), a Northern gannet (Spalding et al., 2002), raptors (Olson et al., 2007; Wünschmann et al., 2009, 2010) and psittacine birds (Villar et al., 2008) numerous schizonts

were identified within the cerebral lesions. Few cases have been reported of Sarcocystis–related encephalitis without associated parasite stages in the neuronal tissue as was seen in *S. calchasi* infections (Munday et al., 1984; Foreyt et al., 1995). However, evidence on the pathogenesis of the encephalitis in absence of parasite stages is still lacking. It has been demonstrated for *S. calchasi* that the encephalitis appears already very early after infection and several weeks before the onset of neurologic signs (Maier et al., 2014). A transient or low-grade neuroinvasion of schizonts or a trigger located outside the CNS (either schizonts or immature sarcocysts) have been suggested to induce the encephalitis. An immune-mediated pathomechanism seems very likely since the amount of sarcocysts in the skeletal muscle did not correlate with the severity of the encephalitis (Maier et al., 2014). It was demonstrated, that *S. calchasi* is capable of modulating the cellular immune response as it is known from its closely related sister taxon *S. neurona* (Witonsky et al., 2008). During the acute, schizogonic phase Th1-related cytokines interleukin (IL)-12, IL-18 and IFN-γ were down-modulated and the second, neurologic phase was shown to be characterized by an extensive Th1-biased T-cell driven immune response suggesting a T-cell mediated delayed-type hypersensitivity reaction (Olias et al., 2013). However, a direct relation between the activated immune response and the development of the cerebral lesions has not been proven yet.

References

Foreyt, W. J., Baldwin, T. J. and Lagerquist, J. E. (1995): Experimental infections of *Sarcocystis* spp. in Rocky Mountain elk (*Cervus elaphus*) calves. *J. Wildl. Dis.* 31(4), 462-466.

Jacobson, E. R., Gardiner, C. H., Nicholson, A. and Page, C. D. (1984): *Sarcocystis* encephalitis in a cockatiel. Journal of the American Veterinary Medical Association. 185, 904-906.

Maier, K., Olias P., Enderlein D., Gruber, A. D. and Lierz, M. (2013): Pathogenesis and treatment of *Sarcocystis calchasi* infections in domestic pigeons. Proceedings of the First International Conference on Avian, Herpetological and Exotic Mammal Medicine 2013 (ISBN: 978-3-8359-6008-4), Wiesbaden, pp 384-385.

Maier, K., Olias, P., Enderlein, D, Klopfleisch, R., Mayr S. L., Gruber, A. D. and Lierz, M. (2014): Parasite distribution and early stage encephalitis in *Sarcocystis calchasi* infections in domestic pigeons (*Columba livia* f. *domestica*). Avian Pathology. 44, 5-12.

Munday, B. L. and Obendorf, D. L. (1984): Development and growth of *Sarcocystis gigantea* in experimentally-infected sheep. Veterinary Parasitology 15, 203-211.

Mutalib, A., Keirs, R., Maslin, W., Topper, M. and Dubey, J. P. (1995): *Sarcocystis*-associated encephalitis in chickens. Avian Diseases 39, 436-440.

Olias, P., Gruber, A. D., Kohls, A., Hafez, H. M., Heydorn, A. O., Mehlhorn, H. and Lierz, M. (2010a): *Sarcocystis* species lethal for domestic pigeons. Emerging Infectious Diseases 16, 497-499

Olias, P., Gruber, A. D., Heydorn, A. O., Kohls, A., Hafez, H. M. and Lierz, M. (2010b): Unusual biphasic disease in domestic pigeons (*Columba livia f. domestica*) following experimental infection with *Sarcocystis calchasi*. Avian Diseases 54, 1032-1037.

Olias P, Meyer A, Klopfleisch R, et al. Olias, P., Meyer, A., Klopfleisch, R., Lierz, M., Kaspers, B. and Gruber, A. D. (2013): Modulation of the host Th1 immune response in pigeon protozoal encephalitis caused by *Sarcocystis calchasi*. Veterinary Research 44, 10-19.

Olias, P., Gruber, A. D., Hafez, H. M., Heydorn, A. O., Mehlhorn, H. and Lierz, M. (2009a): *Sarcocystis calchasi* sp. nov. of the domestic pigeon (*Columba livia f. domestica*) and the Northern goshawk (*Accipiter gentilis*): light and electron microscopical characteristics. Parasitology Research 106, 577-585.

Olias, P., Gruber, A. D., Heydorn, A. O., Kohls, A., Mehlhorn, H., Hafez, H. M. and Lierz, M. (2009b): A novel *Sarcocystis*-associated encephalitis and myositis in racing pigeons. Avian Pathology 38, 121-128.

Olias, P., Maier, K., Wünschmann, A., Reed, L., Armién, A., Shaw, D. P., Gruber, A. D. and Lierz, M. (2014): *Sarcocystis calchasi* has an expanded host range and induces neurological disease in cockatiels (*Nymphicus hollandicus*) and North American rock pigeons (*Columba livia f. domestica*). Veterinary Parasitology 200, 59-65.

Olias, P., Olias, L., Krücken, J., Lierz, M. and Gruber, A. D. (2011): High prevalence of *Sarcocystis calchasi* sporocysts in European *Accipiter* hawks. Veterinary Parasitology. 175, 230-236.

Olson, E. J., Wünschmann, A. and Dubey, J.P. (2007): *Sarcocystis* sp.-associated meningoencephalitis in a bald eagle (*Haliaeetus leucocephalus*). Journal of Veterinary Diagnostic Investigation. 19, 564-8.

Rimoldi, G., Speer, B., Wellehan, J. F. X., Bradway, D. S., Wright, L., Reavill, D., Barr, B. C., Childress, A., Shivaprasad, H. L. and Chin, R. P. (2013): An outbreak of *Sarcocystis calchasi* encephalitis in multiple psittacine species within an enclosed zoological aviary Journal of Veterinary Diagnostic Investigation. 25,775-781.

Spalding, M. G., Yowell, C. A., Lindsay, D. S., Greiner, E. C. and Dame, J. B. (2002): *Sarcocystis* meningoencephalitis in a northern gannet (*Morus bassanus*). Journal of Wildlife Diseases. 38, 432-7.

Villar, D., Kramer, M., Howard, L., Hammond, E., Cray, C. and Latimer, K. (2008): Clinical Presentation and Pathology of Sarcocystosis in Psittaciform Birds: 11 Cases. Avian Diseases 52, 187-194.

Witonsky, S. G., Ellison, S., Yang, J., Gogal, R. M., Lawler, H., Suzuki, Y., Sriranganathan, N., Andrews, F., Ward, D. and Lindsay, D. S. (2008): Horses experimentally infected with *Sarcocystis neurona* develop altered immune responses in vitro. Journal of Parasitology 94, 1047-1054.

Wünschmann, A., Armien, A. G., Reed, L., Gruber, A. D. and Olias, P. (2011):. *Sarcocystis calchasi*-associated neurologic disease in a domestic pigeon in North America. *Transbound. Emerg. Dis.* 58(6), 526-530.

Wünschmann, A., Rejmanek, D., Conrad, P. A., Hall, N., Cruz-Martinez, L., Vaughn, S. B. and Barr, B. C. (2010): Natural fatal *Sarcocystis falcatula* infections in free-ranging eagles in North America. Journal of Veterinary Diagnostic Investigation. 22, 282-9.

Wünschmann, A., Rejmanek, D., Cruz-Martinez, L. and Barr, B. C. (2009): *Sarcocystis falcatula*-associated encephalitis in a free-ranging great horned owl (*Bubo virginianus*). Journal of Veterinary Diagnostic Investigation 21, 283-7.