PEER REVIEWED CASE STUDY

Rabies in pigs: Clinical report and diagnostic challenges

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Abstract

This paper reports the occurrence of swine rabies in Brazil and discusses the difficulties of clinical and laboratory diagnosis in this species. Two pigs from different farms, were diagnosed with rabies and were raised where cattle had previously died from the disease. Affected pigs had hindlimb paralysis, lateral recumbency, and paddling movements. Histological examination of the brain revealed lymphoplasmacytic encephalitis, although intracytoplasmic inclusions were lacking. Immunohistochemistry revealed intense staining of rabies antigen in brainstem neurons, moderate immunoreactivity was observed in the cerebrum, and was absent in the cerebellum. Immunohistochemistry was effective in the definitive diagnosis of rabies encephalitis in swine.

Keywords: swine, viral encephalitis, rabies, immunohistochemistry, diagnosis

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Resumen - Rabia en cerdos: Reporte clínico y desafíos diagnósticos

Este artículo reporta la ocurrencia de la rabia porcina en Brasil y discute las dificultades del diagnóstico clínico y de laboratorio en esta especie. Dos cerdos de diferentes granjas fueron diagnosticados con rabia, éstos habían sido criados en una zona donde el ganado había muerto previamente a causa de la enfermedad. Los cerdos afectados presentaban parálisis de las extremidades traseras, decúbito lateral y movimientos de pataleo. El examen histológico del cerebro reveló encefalitis linfoplasmocítica, aunque faltaban las inclusiones intracitoplasmáticas. La inmunohistoquímica reveló una tinción intensa del antígeno de la rabia en las neuronas del tronco encefálico, se observó una inmunorreactividad moderada en el cerebro y ausente en el cerebelo. La inmunohistoquímica fue efectiva en el diagnóstico definitivo de encefalitis por rabia en cerdos.

Résumé – La rage chez les porcs: Rapport de cas et défis diagnostiques

Cet article rapporte l'occurrence de rage porcine au Brésil et mentionne les difficultés associées au diagnostic clinique et de laboratoire chez cette espèce. Deux porcs provenant de fermes différentes ont été diagnostiqués avec la rage et avaient été élevés à l'endroit où des bovins étaient auparavant décédés de cette maladie. Les porcs affectés présentaient une paralysie des membres postérieurs, un décubitus latéral et des mouvements de pédalage. L'examen histologique du cerveau a révélé une encéphalite lymphoplasmocytaire, bien que des inclusions intracytoplasmiques étaient absentes. Un examen par immunohistochimie a montré une coloration intense d'antigènes rabiques dans les neurones du tronc cérébral, une immunoréactivité modérée était observée dans le cerveau et était absente dans le cervelet. L'immunohistochimie a été utile pour établir le diagnostic définitif d'encéphalite rabique chez le porc.

Rabies is an acute viral disease caused by an RNA virus in the family *Rhabdoviridae*, which affects the central nervous system (CNS) causing encephalitis and death in all warm-blooded animals, including humans.[1] However, the diagnosis of rabies in pigs is less frequently encountered. A rabies retrospective study in Brazil showed that of 739 confirmed positive rabies samples, only 2 were from pigs.[2] A similar trend was observed in other parts of the world. In a US study examining rabies in domestic and wild animals, only 1 of 39 diagnosed cases was in swine.[3] In Canada, several diagnostic laboratories observed that the percentage of swine rabies ranged from 0.1% to 1.1% for all cases with positive diagnoses of the disease.[4] Only 13 of 814 (2%) rabies cases reported in Bhutan over a 14-year period were in pigs.[1] In Australia, a study was carried out with 252 brain samples from animals submitted for routine rabies diagnosis between April and November 2000, but none of them were from pigs.[5]

Because it is rarely observed in pigs, rabies has little economic significance in swine farming and is less commonly explored as a differential diagnosis related to porcine viral encephalitis. It is also a public health problem due to the risk of

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human exposure, therefore it is essential to understand the dynamics of the disease in swine. This study aimed to report the diagnosis of rabies in pigs from 2 different farms in Brazil and provide diagnosis information, indicating that rabies should always be considered a differential when diagnosing neurological diseases in swine, especially in endemic areas.

Case description

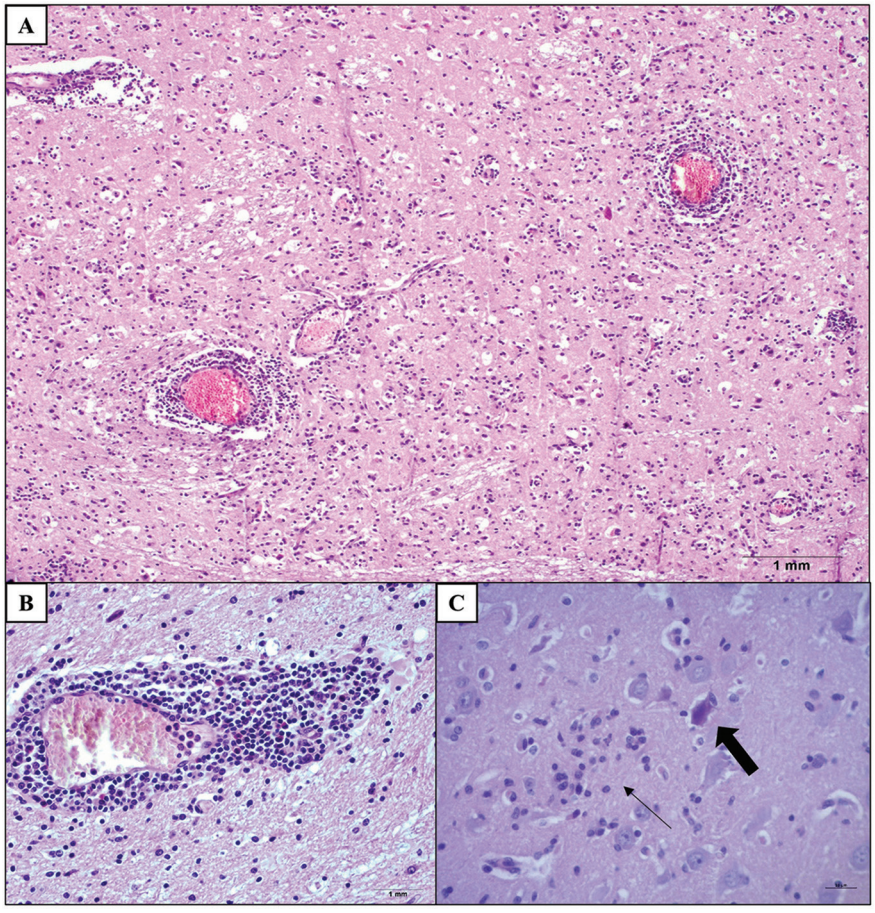
An autonomous veterinarian was called to the southern region of Brazil, in the municipalities of Veranópolis/RS (case 1) and Vidal Ramos/SC (case 2), in February 2009 and January 2013, respectively to examine pigs exhibiting CNS clinical signs. Both farms were primarily involved with dairy production and the affected pigs were raised for meat consumption and were the only swine on each farm. Southern Brazil is considered free of furious rabies (urban cycle) and classical swine fever (CSF). The country is free of porcine reproductive and respiratory syndrome virus and African swine fever (ASF).

The pig from case 1 was a barrow, about one year old from a commercial breed, that was housed with 2 calves. The farm did not vaccinate any animals against rabies, and there was evidence of bat exposure and bites observed in one calf and the pig. In the same week that the pig became ill, the State's Official Health Defence Service confirmed rabies in the suspected calf housed with the pig. The pig from case 2 was a gilt, approximately five months old from a Brazilian autochthonous breed (cross between Piau and Moura). The cattle located on this farm were prophylactically vaccinated for rabies and only the affected pig, that was not vaccinated, had neurological clinical signs. Neighboring farms also had cases of cattle rabies that had been diagnosed 3 weeks before the pig on the affected farm became ill. No bats (*Desmodum rotundus*) from any of the farms with pigs exhibiting clinical signs of rabies were captured or tested for rabies. In both pigs from case 1 and case 2, the clinical signs observed were inappetence, lateral recumbency, and paddling movements, which evolved in case 1 to hind limb paresis and progressive flaccid paralysis followed by death after four days of progressing clinical signs. The pig from case 2 was euthanized 2 days after the onset of clinical signs when paddling movements were observed.

Gross lesions were not observed during necropsy in either case 1 or 2. In case 1, fresh samples of cortex, hippocampus, and cerebellum from the CNS were submitted to a virology laboratory at Rio Grande do Sul state, which only performs direct fluorescent antibody testing (DFAT) for rabies. Sections of liver, kidney, lung, heart, and brain, preserved in 10% buffered formalin, were sent to the Pathology Laboratory from Santa Catarina State University for histopathologic examination seeking differential diagnoses. In case 2, the samples were only collected in 10% buffered formalin for histologic diagnosis. Due to the size of the formalin-fixed tissue samples submitted in both cases, about 3 × 5 cm each, the brain could only be differentiated into the cerebrum, cerebellum, and brainstem. The material was routinely processed for histopathology and stained with hematoxylin-eosin (H-E).

The type and distribution of histological lesions were similar in both pigs. However, the pig from case 1, which survived longer and died spontaneously, had more severe lesions compared to the pig from case 2. In case 1, perivascular lymphoplasmacytic infiltrates were observed around large numbers of blood vessels located in the grey and white matter of the cerebrum, with the intensity of the perivascular infiltrate more pronounced in the white matter of this region (Figure 1A and B). Individual necrosis of neurons, satellitosis, neuronophagia and multifocal microgliosis with astrocytosis were also observed (Figure 1C). In the brainstem, lesions were similar to those in the cerebrum, but inflammatory lesions were more pronounced and gliosis was diffuse. Cerebellar lesions were minimal and characterized by a mild infiltrate of lymphocytes in the meninges. In case 1, the

Figure 1: Histopathology of the brain from the pig in case 1. **A)** Perivascular lymphoplasmacytic infiltrate and diffuse gliosis in the brainstem of swine (hematoxylin-eosin [H-E], original magnification 100×). **B)** Higher magnification of the perivascular lymphoplasmacytic infiltrate with few eosinophils in the brainstem of swine (H-E, original magnification 400×). **C**) Necrosis of neurons (thick arrow) and microgliosis (thin arrow) in the cerebrum (H-E, original magnification 400×).



size of the perivascular infiltrate varied from two to four layers in many vessels, while in case 2 it was not more than two layers.[6] In addition, gliosis in case 1 was multifocal while gliosis in case 2 was diffuse without evidence of lymphocytic meningitis in the cerebellum. No intracytoplasmic inclusion bodies (Negri bodies) were observed within neuron cell bodies of either case. In addition to these lesions, case 1 had evident perivascular and perineuronal edema, a mild infiltrate of eosinophils associated with the lymphoplasmacytic component, areas with neuropil loss, and a mild infiltrate of Gitter cells.

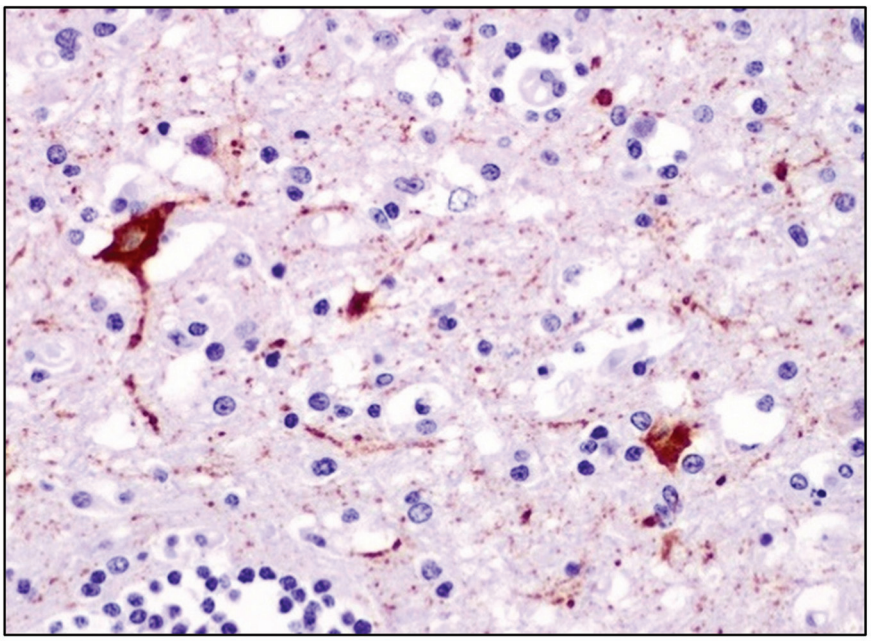
Based on the histological lesions and epidemiology, the brain tissues (cerebrum, cerebellum, and brainstem) were evaluated with immunohistochemistry (IHC) for rabies. The endogenous peroxidase activity was blocked by incubation of tissues in a 3% solution of hydrogen peroxide in methanol for 10 minutes, twice. The antigenic recovery by heat was made with citrate buffer for 5 minutes in a microwave (three cycles). Nonspecific reactions were blocked with 5% skimmed milk diluted in distilled water for 15 minutes. Rabies polyclonal antibody (#5199; Chemicon International, Inc) was used at a dilution 1:1000 in phosphate buffered saline and the slides incubated at 37°C for 1 hour in a humid chamber. After this step, the slides were incubated with secondary antibody linked to streptavidin-peroxidase (kit LSAB-HRP, K0690; Dako Cytomation) for 20 minutes. The chromogen used for development of signal was 3,3'Diaminobenzidine (DAB, K3468, Citomation Dako). For counterstaining, Harris hematoxylin was used. As a positive control, a sample of CNS tissue from cattle previously confirmed positive for rabies by IHC and DFAT was used. Immunohistochemistry revealed strong labelling for rabies antigen in the perikaryon and axons of neurons in the brainstem (Figure 2) and moderate labelling intensity in the cerebrum. Cerebellum samples in both cases did not show significant IHC staining.

Discussion

Rabies infections in pigs and cattle occurring on the same farm were previously described in Brazil.[7] Often, lesions suggestive of bat or dog bites cannot be identified at the time clinical signs are present due to the variable incubation period, which results in difficulties identifying the exact source of infection.[8] In Brazilian southern states, urban rabies has not been detected since the 1980s.[2] However, vampire bats of the species *Desmodus rotundus* remain a natural reservoir of the rabies virus. Vampire bats can only be caught by the State's Official Health Defense Service in cases of a disease epidemic. Puncture wounds reported in the pig from case 1 were evidence of bat bites and suggests that this was the most likely cause of infection. The incubation period of the rabies virus ranges from one to three months, therefore, bat bites are often not observed in the affected animal, as in case 2 described in this report. When available, cattle are considered the main food source for bats of the species *Desmodus rotundus*. In situations where there are few cattle, bats may choose to feed on another available species, such as swine.[7] Vaccination of cattle against rabies is not mandatory in Brazil but is routinely performed in regions of endemic rabies associated with *Desmodus rotundus*. Even in endemic regions, rabies vaccination of swine is not generally performed in Brazil but this practice should be considered in areas where cattle rabies epidemics are observed. In this clinical report, not vaccinating the affected pigs may have contributed to their susceptibility to infection and potential infection leading to clinical disease. In case 1 where vaccination was not carried out, both species (bovine and swine) became clinically affected in the same period. In case 2 where the cattle were vaccinated for rabies, only the swine demonstrated clinical signs of rabies.

Another factor that supports bats as the source of infection in both reported cases is the clinical manifestation of hind limb paralysis. In cases where pigs were infected with rabies from cats and dogs (urban cycle), their clinical signs presented differently from those described in this report and were characterized by aggression, vocalization, muscle spasms and tremors, kicking, hyperexcitation, continuous biting of other animals, and salivation.[9-11] In this case report, clinical signs in the affected pigs reported by their owners consisted of hind limb paralysis, lateral recumbency, and paddling movements. However, these same clinical signs can be observed in several other neurological diseases that occur in pigs raised in Brazil, including streptococcal meningitis, edema disease, salt or selenium poisoning, and porcine teschovirus (PTV). Pseudorabies virus (PRV) has not been reported in Southern Brazil since 2004,[12] and there is no clinical report of porcine sapelovirus A (PSV) or porcine astrovirus type 3 (PAstV3) in the country, but they are important differential diagnoses that must be considered where neurological disease occurs in swine. Porcine sapelovirus A was

Figure 2: Immunohistochemistry (IHC) of the brainstem from the pig in case 1. Rabies antigen was detected by IHC in the perikaryon and axons of neurons in the brainstem (Harris hematoxylin, original magnification 400×).



detected in pigs associated with an outbreak of PTV, but it was not possible to associate the role of these viruses in CNS lesions. In the same study, the presence of PAstV3 was investigated, but it was not detected.[13]

The two most frequent encephalitic diseases, streptococcal meningitis and edema disease, are easily distinguished by their distinct histopathology. Streptococcal meningitis is associated with suppurative encephalitis and in cases of edema disease, degeneration of vessels and absence of inflammatory cells is observed. When salt poisoning occurs, the intermittent convulsive episodes that repeat at regular intervals help diagnose the clinical suspicion. In histology, characteristic lesions of salt poisoning besides eosinophilic infiltrate includes laminar cortical necrosis, which does not occur in rabies cases. Selenium and *Aeschynomene indica* intoxications also present neurological clinical signs. However, the histological lesions are characterized mainly by symmetric and focal degeneration in the spinal cord in cases of selenium intoxication,[14] and in cerebellar and vestibular nuclei in cases of *A indica* intoxication.[15,16] Viral encephalitis by PTV, PRV, PSV and PAstV3, although presenting with lymphoplasmacytic infiltrates, are considered polyencephalomyelitis and present histological differences in the H-E staining that are used to choose if IHC is necessary.

In specific cases of PRV, the main lesions occur in the cerebral and cerebellar cortex, and when present, necrotic areas are associated with neutrophil infiltrates.[17] Histological lesions observed in tonsils, lung, liver, and kidney are not found in cases of rabies, which are restricted to the CNS. The age of the affected animals in both cases is also important as adult pigs rarely develop neurological conditions.[18] For PTV infections, lesions frequently occur in the brainstem, hypothalamus, and spinal cord regions. While the cerebral and cerebellar cortex are rarely affected, the cerebellar white matter is often severely involved[19] and differs from the absence of cerebellar lesions observed in this report. The spinal cord was not submitted for evaluation in this case, but polyencephalomyelitis is mainly observed in the ventral horn of the spinal cord in PTV infection, which is different from rabies infections that produce lesions in the white matter. Also, animals with PTV continue eating and drinking water, with adult animals being much less susceptible to the virus.[19] Collectively, confirming the presence of rabies virus by IHC is a highly effective method as an auxiliary tool in the formulation of the diagnosis.

Pigs typically show only one or two classic clinical signs of rabies or they are found dead with no premonitory signs,[4] which can occur in response to several other neurological diseases that affect swine. Veterinarians should also consider other epidemiological factors when making a presumptive diagnosis such as the animal's age, considering many viral diseases that cause encephalitis, like CSF, ASF, and PRV, affect mainly young piglets. The major challenge in diagnosing rabies in pigs is that it is rarely considered in the list of differential diagnosis of neurological diseases in swine. The samples submitted in buffered formalin for histological examination provided an effective method for creating a differential diagnosis and should be encouraged, especially in cases where there is acute death. Special attention should be given to pigs raised in regions where vectors are present, or which have concomitant cases of neurological diseases in other species, such as ruminants, that may also have rabies, streptococcal meningitis, or PRV. Especially in cases of neurological disease in locations where rabies is endemic, the collection of fresh samples should be included since DFAT is considered the gold-standard technique for rabies diagnosis, as in case 2 where only formalin-fixed tissues were submitted. Spinal cord was not collected in either of the cases in this report. Although it did not change the diagnosis, its sampling could have brought more information about rabies in pigs. High intensity of histological injury in the spinal cord has been reported in other rabies diagnoses in pigs.[20,21] Additional sampling of the spinal cord based on clinical signs should be considered, since it is an important differential diagnosis for PTV and selenium poisoning.

The histological findings described in this report were characterized by more pronounced lymphocytic meningoencephalitis in the brainstem compared to the cerebellum and cortex, which is similar to rabies lesions described by other authors.[20,22] The pig in case 1 had marked severe perivascular and perineuronal edema and mild eosinophilic infiltration associated with the lymphoplasmocytic component. Neurological clinical signs associated with histological lesions characterized by eosinophil infiltration and neuronal necrosis have been reported in pigs with salt poisoning or sodium ion intoxication, a condition that occurs due to excessive sodium chloride intake during water deprivation under normal salt intake conditions.[23] Pigs infected with rabies virus, although they do not reject water and food, are usually unable to physically swallow.[8] It is likely that the four-day clinical course of rabies in the pig from case 1 had hindered water consumption and perhaps if the animal survived longer, would have further progressed to corticolaminar necrosis commonly observed with salt poisoning. Convulsive episodes caused by salt intoxication may have been hidden due to flaccid paralysis caused by rabies.

The pig in case 1 survived longer, had spontaneous death, and had exhibited more severe lesions compared to the pig in case 2, which was euthanized for a diagnosis earlier in the progression of the disease. However, the IHC diagnosis in both animals was performed and showed similar immunoreactivity. In a study to diagnose bovine rabies, it was reported that IHC and DFAT were useful diagnostic techniques that could detect animals in the early stages of clinical evolution, with mild intensities of histological lesions.[24] This report established that early euthanasia during the course of clinical disease can be performed to reduce the suffering of animals without affecting the ability to diagnose the disease using IHC. As with clinical signs, microscopic lesions of nonsuppurative encephalitis are not definitive in rabies encephalitis and similar lesions can be found in other viral CNS infections. Intracytoplasmic viral inclusions in neurons are characteristic of rabies, and the presence of these structures is strongly suggestive of viral infection.[24] However, no inclusions consistent with rabies were observed in the brain of the pigs analysed in this study. The absence of Negri bodies in cases of swine rabies has also been reported in other studies.[7,9,20,22,25] The absence of intracytoplasmic viral inclusions in swine rabies represents a diagnostic challenge in this species, and the application of additional diagnostic methods such as IHC and DFAT technique is essential.

Direct florescent antibody testing is the gold standard for rabies diagnosis; however, a study conducted in Canada showed the sensitivity of DFAT for swine was 81.8% (18 of 22 cases were positive), which was much lower compared to other species tested within the same

laboratory.[4] False-negative DFAT results for swine rabies have also been reported by other authors.[20,22,25,26] These reports suggest that negative DFAT results in pigs should be interpreted with caution and other complementary diagnostic methods are important. Many factors may lead to the occurrence of false-negative DFAT, such as a low or irregular distribution of the virus in CNS tissue and the difficulties experienced with fluorescent antibody staining.[25] A study conducted to determine the regions of the brain where rabies antigen was found indicated that in most mammals, the most reliable part was the brainstem, particularly the thalamus. Other parts of the brain, including the hippocampus, cerebellum, and cerebrum, will occasionally give false-negative results for rabies antigen in DFAT diagnoses.[5]

The samples analysed in this report were fixed in 10% formalin with confirmatory diagnosis by IHC. This study showed that immunoreactivity was more intense in the areas of greater inflammation, with the most immunoreactivity observed in the brainstem, mild reactivity in the cerebrum, and no immunoreactivity observed in the cerebellum. Strong immunoreactivity in the brainstem has also been reported in other cases of rabies in pigs.[3,21,27] Also, no immunoreactivity in cerebellum samples was observed in horses and foxes.[3] Based on this, our recommendations are that the spinal cord and brainstem should routinely be sampled for histological analysis in cases where porcine rabies is a differential diagnosis, as sampling of only the cerebral cortex or cerebellum can increase the likelihood of a false-negative result. As the rabies virus does not have a homogeneous distribution throughout the CNS, it is possible that there are not enough viral particles to be marked in the IHC and DFAT techniques. The absence of Negri bodies,[7,9,20,22,25] and inconsistent detection of rabies virus in porcine CNS by DFAT highlight the complexities involved in the diagnosis of rabies in this species.

Implications

Under the conditions of this study:

- Rabies antigen was detected in the brainstem and cerebrum using histopathology and IHC.
- No rabies antigen was detected in the cerebellum by IHC.
- Immunohistochemistry was effective in confirming the rabies diagnosis.

Acknowledgments

Conflict of interest

None reported.

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References

1. Tenzin, Dhand NK, Ward MP. Patterns of rabies occurrence in Bhutan between 1996 and 2009. *Zoonoses Public Health*. 2011;58:463-471.**https://doi.org/10.1111/j.1863-2378.2011.01393.x**

2. Teixeira TF, Holz CL, Caxieta SPMB, Dezen D, Cibulski SP, da Silva JR, Rosa JCA, Schmidt E, Ferreira JC, Batista HBCR, Caldas E, Franco AC, Roehe PM. Rabies diagnosis in the state of Rio Grande do Sul, Brazil, from 1985 to 2007. *Pesq Vet Bras*. 2008;28(10):515-520. **https://doi.org/10.1590/S0100-736X2008001000012**

3. Stein LT, Rech RR, Harrison L, Brown CC. Immunohistochemical study of rabies virus within the central nervous system of domestic and wildlife species. *Vet Pathol*. 2010;47(4):630-633. **https://doi.org/10.1177/0300985810370013**

4. Yates WD, Rehmtulla AJ, McIntosh DW. Porcine rabies in Western Canada. *Can Vet J*. 1983;24:162-163.

5. Bingham J, van der Merwe M. Distribution of rabies antigen in infected brain material: Determining the reliability of different regions of the brain for the rabies fluorescent antibody test. *J Virol Methods*. 2002;101:85-94. **https://doi.org/10.1016/S0166-0934(01)00423-2**

6. Wisser CS, Neto AT, Batista HBCR, Mori E, Chierato ER, Fernandes MES, Traverso SD. Cattle rabies: the effect of clinical evolution, viral genetic lineage, and viral load on the severity of histological lesions. *Pesq Vet Bras.* 2020;40(4):227-233. **http://doi.org/10.1590/1678-5150-PVB-6438**

7. Nociti DLP, Junior JGC, Matta GCA, Aguiar DM. Rabies in a swine from Mato Grosso State, Brazil – Report of a bovine infection on the same farm. *Arq Inst Biol.* 2009;76(2):269-271. **https://doi.org/10.1590/1808-1657v76p2692009**

8. Lobato FCF, Silva ROS, Filho RBC, Pessoa LCD, Pires PS, Salvarani FM, Filho PM, de Assis RA. An outbreak of pig rabies in Tocantins State, Brazil. *Ci Vet Trop*. 2008;11(2-3):73-75.

9. Corner AH, Beauregard M, Mitchell HK. Rabies in piglets. *Can J Comp Med Vet Sci*. 1960;24(12):364-365.

10. Jiang Y, Yu X, Wang L, Lu Z, Liu H, Xuan H, Hu Z, Tu C. An outbreak of pig rabies in Hunan province, China. *Epidemiol Infect*. 2008;136(4):504-508. **https://doi.org/10.1017/S0950268807008874**

11. Tasiame W, Folitse RD, Emikpe BO, Adongo JA. First reported case of dog associated pig rabies in Ghana. *Afr J Infect Dis*. 2016;10(1):55-57. **https://doi.org/10.4314/ajid.v10i1.11**

12. Zanella JRC, Mores N, de Barcellos DESN. Main endemic health threats in the swine production chain in Brazil. *Pesq Agropec Bras*. 2016;51(5):443-453. **https://doi.org/10.1590/S0100-204X2016000500004**

13. Hammerschmitt ME, de Almeida PR, de Cecco BS, Lorenzett MP, Schwertz CI, da Cruz RAS, Caprioli RA, Schuh DT, Demoliner M, Eisen AKA, Spilki FR, Pavarini SP, Driemeier D. Swine polioencephalomyelitis in Brazil: identification of Teschovirus A, Sapelovirus A, and Enterovirus G in a farm from Southern Brazil. *Braz J Microbiol.* 2021;52:1617-1622. **http://doi.org/10.1007/s42770-021-00509-z**

14. Gomes DC, Souza SO, Juffo GD, Pavarini SP, Driemeier D. Selenium poisoning in swine in Southern Brazil. *Pesq Vet Bras.* 2014;34(12)1203-1209. **https://doi.org/10.1590/S0100-736X2014001200010**

15. Riet-Correa F, Timm CD, Barros SS, Summers BA. Symmetric focal degeneration in the cerebellar and vestibular nuclei in swine caused by ingestion of *Aeschynomene indica* seeds. *Vet Pathol*. 2003;40:311-316. **https://doi.org/10.1354/vp.40-3-311**

16. Oliveira FN, Rech RR, Rissi DR, Barros RR, Barros CSL. Poisoning in swine from ingestion of *Aeschynomene indica* (Leg.Papilionoideae) seeds. *Pesq Vet Bras.* 2005;25(3):135-142. **https://doi.org/10.1590/S0100-736X2005000300003**

17. Summers BA, Cummings JF, Lahunta A. Inflammatory diseases of the central nervous system. In: Summers BA, Cummings JF, Lahunta A, eds. *Veterinary Neuropathology*. Mosby-Year Book; 1995:99-136.

18. Swenson SL, Mead DG, Kinker DR. Rhabdoviruses. In: Zimmerman JJ, Karriker LA, Ramirez A, Schwartz KJ, Stevenson GW, Zhang J, eds. *Diseases of swine*. 11th ed. Wiley Blackwell; 2019:733-739. **https://doi.org/10.1002/9781119350927.ch45**

19. Cantile C, Youssef S. Nervous System. In: Grant MM, ed. *Jubb, Kennedy, and Palmer's Pathology of Domestic Animals.* 6th ed. Elsevier; 2016:350-396. **https://doi.org/10.1016/B978-0-7020-5317-7.00004-7**

20. Pessoa CRM, Silva MLCR, Gomes AAB, Garcia AIE, Ito FH, Brandão PE, Reit-Correa F. Paralytic rabies in swine. *Braz J Microbiol*. 2011;42:298-302. **https://doi.org/10.1590/S1517-83822011000100038**

21. Hazlett MJ, Koller MA. Porcine rabies in a closed feeder barn. *Can Vet J*. 1986;27(3):116-118.

22. Dhillon SS, Dhingra PN. A note on rabies in swine. *Vet Med Small Anim Clin*. 1973;68(9): 1044.

23. Sobestiansky J, Mores N, Souza MA, Moreno AM. Intoxication by minerals, chemicals plants and gases. In: Sobestiansky J, Barcellos D, eds. *Swine Disease*. 2nd ed. Cânone Editorial; 2012:553- 580.

24. Wisser CS, Fernandes MES, Melchioretto E, Ogliari D, Gava A, Batista HBCR, Mori E, Traverso SD. Influence of euthanasia, the intensity of inflammatory lesion and viral load in the laboratory diagnosis of rabies in cattle. *Pesq Vet Bras.* 2021;41:e06782. **https://doi.org/10.1590/1678-5150-PVB-6782**

25. Morehouse LG, Kintner LD, Nelson SL. Rabies in swine. *J Am Vet Med Assoc*. 1968;153(1):57-64.

26. Beauregard M, Boulanger P, Webster WA. The use of fluorescent antibody staining in the diagnosis of rabies. *Can J Comp Med Vet Sci*. 1965;29(6):141-147.

27. Siepker CL, Dalton MF, McHale BJ, Sakamoto K, Rissi DR. Neuropathology and diagnostic features of rabies in a litter of piglets, with a brief review of the literature. *J Vet Diagn Invest*. 2020;32(1):166-168. **http://doi.org/10.1177/1040638719898687**