

Atypical pathological presentation of *S. suis* serotype 2 induced cerebral abscess in a naturally infected buffalo calf

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Received: 20.8.2024; Accepted: 27.9.2024

ABSTRACT

The present study reports a rare case of cerebral abscess in a 10 month old male crossbred buffalo calf showing the clinical signs of lateral recumbency, torticollis, stiffness of neck, muscle twitching, paddling movement and opisthotonus. Grossly, the brain showed a large abscess in anterior part of the right cerebral hemisphere. Histopathologically, purulent meningitis was observed showing marked infiltration of inflammatory cells in the leptomeningeal space. The brain parenchyma showed purulent exudates lined by thick connective tissue capsule, marked perivascular cuffing, multi-focal areas of gliosis, vasculitis and neuronal degenerations. The DNA from the affected brain tissue showed the presence of *S. suis* by PCR targeting *gdh* gene and further confirmed as serotype 2 by multiplex PCR amplifying 498 bp base pair fragment of the capsular gene. Immunohistochemically, the inflammatory cells infiltrating the brain parenchyma and glial cells showed the abundant immunoreactivity for SS2 antigens confirming the etiology. The histopathological, immunohistochemical and molecular findings support the diagnosis of rare case of *S. suis* serotype 2 induced cerebral abscess in a buffalo calf.

Keywords: Buffalo calf, cerebral abscess, immunohistochemistry, neuropathology, PCR

Streptococcus suis is an emerging bacterial swine zoonotic pathogen responsible for causing huge economic loss to the pig production specially in intensive pig production systems¹. Recent report showed the average cost of *S. suis* per pig (summed across all production phases) was estimated to be 1.30 euros, 0.96 euros and 0.60 euros in Germany, Netherland and Spain respectively². This pathogen is frequently reported to be associated with various diseases such as meningitis, endocarditis, pneumonia, arthritis and septic shock in pigs and human^{1,2}. The prevalence of *S. suis* has been reported in Indian pigs^{3,4}. *S. suis* is among the pathogens for which scientific interest has increased faster in recent years, and it is currently included among the top ten swine pathogens worldwide. Besides pigs and humans, cross species transmission of *S. suis* infection in different species showing broad host range has been reported in previous reports^{5,6}. Among the different species reported, it has been isolated frequently from cattle showing diverse pathological cases of meningitis, bronchopneumonia and multifocal acute necrotizing hepatitis^{7,8}. Out of identified thirty-eight capsular serotypes of *S. suis* based on the co-agglutination and DNA sequencing methods, serotype 2 is the most common and virulent serotype frequently recovered from diseased swine and humans and predominant in Asia, North and South America¹. In the present investigation, we confirmed a rare case of *S. suis* infection in a buffalo calf showing the clinical signs of neurological manifestation.

A 10-month-old crossbred dead male buffalo calf was received to Postmortem Facility, IVRI, Izatnagar from experimental animal shed of Animal Nutrition division with the history of high fever, paddling movement, tremor, stiffness of neck, dysphasia, clonic convulsion and opisthotonus. The animal was under treatment after the immediate onset of clinical signs with an

How to cite this article : Babu, S., Murali, D., Patel, S., Thakor, J.C., Singh, R., Pasayat, M., Acharya, R., Tripathy, J.P., Sahoo, P.K., Sahoo, N.R. and Sahoo, M. 2025. Atypical pathological presentation of *S. suis* serotype 2 induced cerebral abscess in a naturally infected buffalo calf. Indian J. Vet. Pathol., 49(1) : 64-67.

antibiotic (cefazolin), neurobion and steroids. However, even with treatment, the calf died after undergoing treatment for 7 days.

The systemic necropsy was performed and thin representative tissues from the brain, lungs, heart, spleen, liver, kidneys and intestine were fixed in 10% neutral buffered formalin for pathological investigation. Fixed tissues were embedded in paraffin wax, sectioned at a thickness of approximately 5 µm and stained with routine hematoxylin and eosin (H&E). The immunolocalization of *S. suis*



Fig. 1. Cerebral abscess in a buffalo calf. Note the opened pus cavity at the anterior part of right cerebrum.

type 2 antigen was done on the paraffin embedded (FFPE) tissue sections of brain and lungs using commercially available polyclonal anti-rabbit *S. suis* type antibody (Cat orb7028, biorbyt). Another set of same tissues were kept in -20°C for molecular investigation. For the molecular detection, DNA was isolated from the affected brain, lungs and spleen tissues using DNAeasy blood and tissue kit (Qiagen, India) and screened for the probable bacterial agents such as *Listeria monocytogenes*⁹, *Streptococcus suis*¹⁰, *Pasteurella multocida*¹¹ and viral pathogens bovine Herpes virus¹² and Bovine viral diarrhoea virus¹³ were ruled out in complementary DNA (cDNA) prepared from brain tissue using published primers. For the serotype

identification of *S. suis*, multiplex PCR was used as per previous report¹⁴.

The neurological signs exhibited by the buffalo calf in the present study are consistent with those attributed to *S. suis* induced meningitis^{15,16}. Grossly, the brain showed abscess of 5.5 cm diameter raised firm abscess at the anterior part of right cerebral hemisphere (Fig. 1). The meningeal vessels covering the brain surface were markedly congested. On cutting the pus pocket, 20-30 ml of thick purulent creamy exudates oozed out. Besides brain, the remaining visceral organs did not show any appreciable gross lesions. Microscopically, the brain showed significant histopathological lesions. The leptomeningeal space was widened and filled with inflammatory cellular exudates along with congestion and oedema (Fig. 2a). The cerebrum and medulla showed predominant histopathological lesions as compared to cerebellum. The brain parenchyma showed marked presence of purulent exudates surrounded by thick connective capsule (Fig. 2b), perivascular cuffing (Fig. 2c), vasculitis (Fig. 2d), extensive areas of congestion, haemorrhages, oedema, multifocal areas of gliosis, increased microglial proliferation, astrogliosis, neuronophagia, swollen endothelial cells in capillaries, neuronal necrosis and degeneration. The purkinje neurons of cerebellum showed neuronal loss and neuronal degeneration with mild perivascular cuffing. The brain stem showed status spongiosus, gliosis, vasculitis, neuronal necrosis with severe perivascular cuffing. The neuropathological lesions observed in the present study

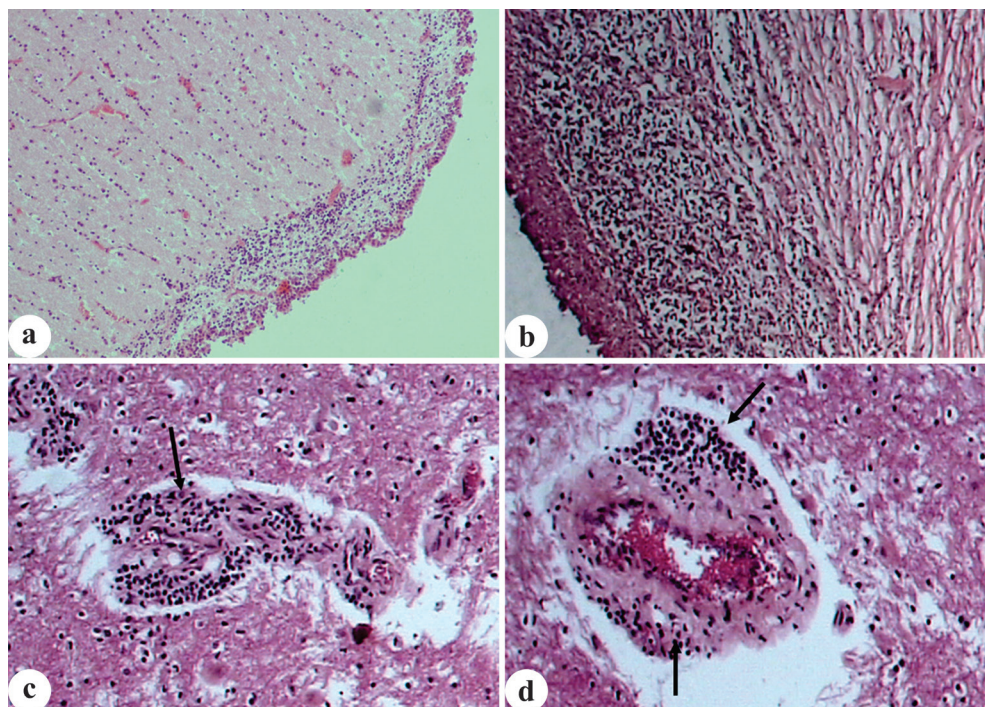


Fig. 2. Histopathological sections of brain of naturally infected buffalo calf showing **a.** Leptomeningitis (H&E X100); **b.** Purulent exudates lined by thick connective tissue capsule (H&E X100); **c.** Perivascular cuffing (H&E X200); **d.** Vasculitis (H&E X200).

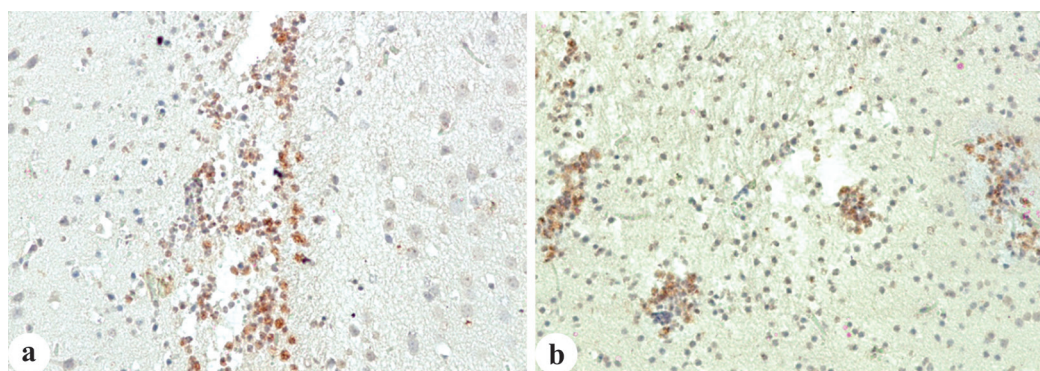


Fig. 3. Immunohistochemical stained sections of brain showing immunoreactivity **a.** Inflammatory cells, neocortex; **b.** Glial cells (IHC X200).

corroborated with the earlier findings of *S. suis* type 2 induced meningoencephalitis in natural and experimental cases^{17,18}. The microscopic lesions of meningoencephalitis with well encapsulated fibrous tissue was documented in a previous report¹⁹. The neuropathological lesions might be due to destruction of blood brain barrier by the actions of suilysin protein and other virulence factors of *S. suis*¹⁹. The immunohistochemical stained brain section showed the abundant immunoreactivity for *S. suis* type 2 antigen in the inflammatory cells infiltrating the brain parenchyma (Fig. 3a) and glial cells (Fig. 3b) correlating with neuropathological lesions. The similar findings were reported in previous reports^{17,18}. Besides brain, the visceral organs failed to show any appreciable gross or histopathological lesions. The absence of any systemic lesions in the present case ruled out the possibility of vascular dissemination from extraneural organs. The extensive lesions of meningoencephalitis with close proximity to right ear suggest the possibility of getting transient the infection from otitis interna¹⁹. Moreover, the colonization of *S. suis* within the nasal cavity

with transient rhinitis might lead to the subsequent dissemination of bacterial pathogens to the brain via cribiform plate might result in neuropathological lesion due to bacteraemia¹⁹. Earlier report showed the endocarditis associated brain lesion in slaughtered pigs²⁰. Further, brain lesions associated with endocarditis were mostly focal and disseminated to various organs. On contrary, in our case, neither endocarditis nor any other systemic disseminated lesions were recorded. Moreover, the distribution of the brain lesions were extensive rather than focal in the brain parenchyma. The pure involvement of brain without showing any systemic microscopic lesions was congruent with an earlier report describing *S. suis* type 2 induced cerebral abscess in a pig¹⁹. The genomic DNA isolated from the brain showed amplification for *S. suis* targeting partial fragment of *gdh* gene yielding 566 bp product (Fig. 4a) whereas other organs failed to show any amplification. Further, brain showed the amplification for partial fragment of capsular polysaccharide gene (*cps*) gene yielding 498 bp confirming the association of *S. suis* type 2 (Fig. 4b).

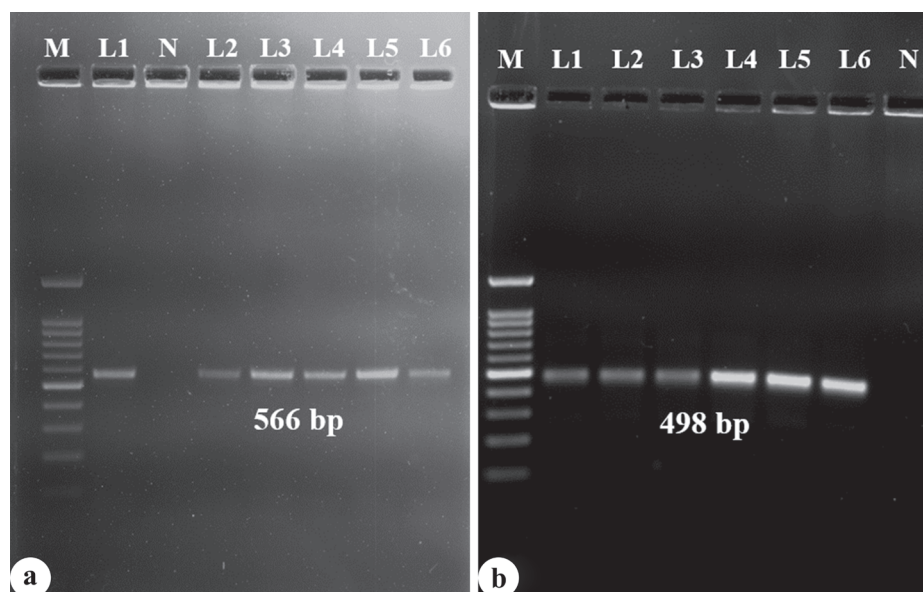


Fig. 4. Molecular detection of *S. suis* and its serotype in the brain showing **a.** M-100 bp marker, L1: Positive control, N: Negative control, L2-L6: Brain tissues positive for *S. suis* yielding 566 bp; **b.** M-Marker (100 bp), L1-L6: Positive for *S. suis* type 2 yielding 498 bp.

The brain failed to show amplification for other bacterial and viral pathogens. The detection of *S. suis* serotype 2 in the brain suggest the neurotropism of this pathogen. *S. suis* type 2 is reported worldwide associated with meningoencephalitis in pigs and human¹⁶.

Based on histopathological, immunohistochemical and molecular findings, the present case is confirmed to be a rare case of *S. suis* induced cerebral abscess in a buffalo calf. Therefore, the presence of *S. suis* has to be taken into consideration while dealing with cases of buffalo showing neurological manifestations.

ACKNOWLEDGEMENTS

The authors would like to acknowledge the financial support extended by the Director, Indian Veterinary Research Institute (IVRI), Izatnagar and Indian Council for Agricultural Research (ICAR), New Delhi are gratefully acknowledged.

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