STREPTOCOCCUS SUIS INFECTION (DIAGNOSIS, PREVENTION AND TREATMENT)

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Streptococcus suis (S. suis) is an important zoonotic pathogen, which can cause serious diseases such as meningitis, pneumonia, endocarditis, polyserositis, arthritis, septicemia and abortion in pigs. In recent years, the incidence rate of streptococcal meningitis has shown a significant upward trend. For humans, the threat of S. suis is also increasing. Therefore, strengthening the prevention and control of the disease has become an urgent task.

The premise of inducing meningitis is that S. suis invades the central nervous system and breaches the blood brain barrier (BBB). Due to the presence of the blood brain barrier, even though bacteria can enter the blood through the skin mucosa and other parts, a large number of bacteria in the blood cannot enter the brain through the blood brain barrier. The body relies on this barrier to protect the brain tissue from damage and maintain the homeostasis of the central nervous system. Brain microvascular endothelial cell (BMEC) is the basic component of the blood brain barrier, and a variety of neurological diseases are related to the dysfunction of the blood brain barrier, and S. suis can interact with brain microvascular endothelial cell and then cross the the blood brain barrier to cause central nervous system infection. However, the antibiotics used to treat the infection cannot pass through the barrier to reach the therapeutic target site, which is the key to the difficulty in the control of bacterial meningitis. Therefore, elucidating the mechanism of S. suis breaking through the blood brain barrier into central nervous system is an important breakthrough in developing S. suis meningitis control strategy.

Due to long-term unjustified use of antibiotics, bacterial resistance has increased, and antibiotic treatment disrupts the normal homeostasis of the body and intestinal flora. The problems caused by long-term, large-scale use of antibiotics are becoming more and more serious. Meningitis caused by streptococcus suis can no longer be treated with conventional antibiotics. Therefore, It is necessary to have a thorough understanding of the pathogenesis of meningitis.

This article reviews the discovery the clinical signs and symptoms of the disease, pathological changes, laboratory, measures of prevention and treatment for streptococcal infection of pigs in recent years.

Key words: streptococcus infection, suis, diagnosis, prevention, treatment

DOI https://doi.org/10.32782/bsnau.vet.2023.3.18

Introduction. *Streptococcus suis* is a zoonotic pathogen that is the etiological factor of streptococcal infection in pigs characterized by the development of sepsis, arthritis, endocarditis and meningitis (Zhou et al., 2020; Roy et al., 2018; Zhang et al., 2018; Dai et al., 2018; Okwumabua et al., 2020). There are 35 serotypes, with serotype 2 being the most virulent. At the same time, Streptococcus suis serotype 2 (S. suis 2) is also dangerous for humans (Deng et al., 2018; Guo et al., 2021; Hlebowicz et al., 2019; Xia et al., 2018; Xia et al., 2019). Streptococcus suis can induce meningitis in humans and pigs, which not only causes significant economic losses in pig industry, but also seriously threatens public health security. Although some progress has been made in the detection of S. suis 2, there are still many challenges in the research of diagnosis and treatment S. suis 2-induced infection, especially in the aspect of prevention (LeBel et al., 2018; Liu et al., 2018; Qian et al., 2018; Vötsch et al., 2019; Wang et al., 2020).

Clinical signs and symptoms. Septicemia. Septicemic streptococcus is mainly caused by group C streptococcus, group D streptococcus, and group E streptococcus. The

most acute cases often result in sudden death without any symptoms. The symptoms of the disease are more gradual and include a rise in body temperature above 41°C, muscle tremors, loss of appetite or paralysis, constipation, visual cyanosis of mucous membranes, flushing of the conjunctival membranes, and tears. The skin color in the ear root, neck, abdomen and other places is purple, and breathing difficulties appear in the later stage of the disease (Done, Williamson, & Strugnell, 2012). Death occurs within 3 days after the disease, and dark red blood flows from natural pores after death. Autopsy showed serous cavity effusion, cellulose attachment, nasal mucosa congestion and bleeding, larynx and trachea congestion, a large number of bubbles in the trachea, spleen enlargement of 1 ~ 3 times of normal, kidney swelling and bleeding, digestive tract mucosa with varying degrees of congestion and edema (G.-x. Zhang, 2012).

Meningitis. This type of suis streptococcus disease is caused by group R streptococcus and Group C streptococcus, with a small number of cases caused by L or S streptococcus infections. Diseased pigs present with

elevated body temperature, constipation, refusal to feed, and serous or mucous rhinorrhea. Soon after the disease appeared neurological symptoms, ataxia, empty chewing, spinning, hind limb paralysis, limbs were swimming. Post examination of diseased pigs showed meningeal congestion and bleeding, some of the diseased pigs had symptoms of submeningeal effusion, and white matter and gray matter were scattered in hemorrhagic spots under the section of the brain(Gottschalk & Segura, 2000).

Arthritis. This type of pig is mainly converted from the first two types of disease, the course of disease is slightly longer than the last two types, the symptoms are relatively mild, the body temperature sometimes increased and sometimes normal, mental and appetite instability, one or more joint enlargement, lameness, difficulty standing, and emaciation (Hedegaard, Zaccarin, & Lindberg, 2013). There are yellow jelly-like liquids in the joint capsule, and some are cellulose purulent substances.

Lymph node abscess. The main manifestations of infected pigs are suppurative lymphadenitis, the main lesions are mandibular lymph nodes, and sometimes the lymph nodes in the pharynx, ear and neck will also be damaged (Timoney, 2022). The lesions of lymph nodes are inflamed, swollen, palpated hard and hot pain, which will affect feeding, chewing and swallowing, and seriously affect breathing. When the purulent site ruptured, the systemic symptoms were relieved, the course of the disease was 3-5 weeks, and the mortality was low.

Pathological change. Liver. The central vein, interlobular vein and hepatic sinuses of the liver were dilated, microthrombus was formed in the cavity, fibrin was interwoven into a network, and there were a few red blood cells, neutrophils, mononuclear macrophages and lymphocytes in the network (Gelberg, 2017). Some hepatocytes showed coagulation necrosis, and some areas showed high levels of hemosiderin.

Kidney. The glomerular capillaries were dilated, there were red blood cells or cellulosic thrombosis in the lumen, the capillary endothelial cells were swollen, the glomerular volume was significantly increased, the coagulated necrosis of the renal tubular epithelium and the necrotic lesion had a large amount of neutrophils and macrophage infiltration, and the small veins around the necrotic lesion were significantly dilated and congested. There was extensive hemorrhagic necrosis in the localized renal tubules (Xu et al., 2010).

Lung. Alveolar wall thickened significantly, capillary dilatation and congestion or fibrinous thrombosis, pink serous fluid, cellulose, varying number of red blood cells and inflammatory cells can be seen in the alveolar, small vein dilatation and congestion or venous thrombosis, more Grade bronchi contained serous fluid, cellulose, red blood cells and other inflammatory products, interstitial edema widened, lymphatic vessels dilated (Lin et al., 2015).

Spleen. The volume of the white pulp was reduced, and the number of red blood cells was reduced. In the white pulp area, fragments of nuclei of necrotic cells were scattered, and white blood cells disappeared in part of the pulp area (S. Wang et al., 2022). In the red pulp area, the structure was blurred, the contour of capillaries was unclear, the

number of lymphocytes was reduced, and there was a large amount of cellulose dispersed in the spleen tissue, which contained neutrophils.

Lymph node. Fibrinous exudates and more brown hemosiderin were found in the medullary area of the dilated follicular germinal center of lymphatic follicles in the cortex.

Heart. There was no obvious lesion of myocardial fibers, but the intermuscular edema widened and the intermuscular veins were dilated and congested.

Bladder. There was swelling of the epithelial cells, edema in the submembrane, loose tissue structure, and fibrinous thrombosis in the capillaries in the submembrane.

Clinical diagnosis. According to the epidemic characteristics, clinical manifestations and pathological changes of *S. suis* disease, combined with the experience of technician, preliminary diagnosis can be made intuitively. After the disease, the skin of the sick pig will show purple indigo color, which generally appears in the eartips, abdomen and buttocks in the early stage, and will gradually extend to other parts as the disease worsens (Yang et al., 2009). Flushed skin and eye conjunctiva, purplish red spots spread to the lower ear, neck and inner extremities. These symptoms of pigs can be suspected to be *S. suis* disease.

Laboratory diagnosis. Staining Appropriate amount of diseased tissue was taken to make smear. If diseased tissue of dead pigs was collected, fresh diseased tissue had better be collected. After the smear is made, the flame is fixed for 1~2 times, and the slide is not hot when the flame is fixed. Then, the smear was dyed through four operations: primary dyeing (crystal violet staining for 1min), medium dyeing (iodine solution staining for 1min), decolorization (decolorization solution staining for 20~60s), and redyeing (casserine solution staining for 1min). Interpretation of positive results: The gram-stained pathogens were observed under a microscope, and purple round or oval pathogens were found. These pathogens were mainly arranged in pairs or short chains, and there were a few single round coccus.

Isolation culture. Disease materials were collected and inoculated into blood (including rabbit blood) AGAR medium, cultured at 37°C for (24±2) h, and then placed on a super clean platform to observe the solid plate culture. Interpretation of positive results: Round protrusions were found on the petri dish, indicating smooth and moist grayish-white pathogenic bacteria with obvious hemolytic rings around the colony (about 1mm in diameter). The rabbit plasma coagulase test was positive.

ELISA. Based on immunoassay technology, enzyme catalyzed reaction is used to enhance the sensitivity of specific antigen and antibody reaction, which is suitable for large-scale field diagnosis of *S.suis* infection(Xia, Wang, Wei, Jiang, & Hu, 2018). A variety of new ELISA technologies have been developed, and mature commercial kits are widely used.

PCR technique. It is used for rapid and sensitive detection of *S. suis* with multiple serotypes. Multiprimer PCR is a PCR amplification technique developed on the basis of traditional PCR. Multiple pairs of specific primers are used to simultaneously amplify different DNA fragments in the PCR

system, which greatly improves the detection efficiency (Z. Liu et al., 2013). Fluorescence quantitative PCR not only has the characteristics of high amplification efficiency of traditional PCR, but also has the characteristics of high specificity, high sensitivity and high precision of spectral technology. Moreover, fluorescence quantitative PCR can distinguish between current infection and previous infection, which is an aspect that immunological analysis cannot do.

Prevention and treatment. Prevention. Pig farms should develop a reasonable immunization plan, pregnant sows are usually vaccinated with inactivated *S. suis* vaccine 4 weeks before delivery; Piglets were inoculated once at 30 days of age and 45 days of age. Gilts are inoculated again before breeding and the protection period lasts for 6 months(Cloutier et al., 2003). It is important to note that the use of antimicrobials or the addition of antimicrobials to feed should be prohibited for approximately 15 days before and after swine vaccination. In addition, the vaccine dose can be appropriately increased for pigs at full age to prevent immune failure due to insufficient dose.

In the process of breeding pig farms adopt the way of self-breeding, combined with the model of all in and all out can reduce the incidence of pig herds (Dekker et al., 2013) . If it is necessary to introduce species from off-site, it is necessary to investigate the introduced place first to ensure that the introduced place is not an epidemic area, and then strictly quarantine the introduced pigs to ensure that there is no pathogen before introduction. In the process of introduction, transportation time and distance should be reduced to avoid contact with diseased animals on the road. After the introduction to the breeding farm, quarantine observation, no sick pigs were found, and the introduced pigs could be mixed for breeding.

The pig house needs to maintain good temperature and humidity, especially in different seasons to ensure that the temperature and humidity in the pig house change little, to avoid the temperature mutation caused by the pig house temperature rise and fall, resulting in the pig population is not adapted to. Especially in the hot summer and cold winter weather to pay attention to the pig house to maintain a relatively constant internal environment. The pig house should do a good job of ventilation, so that the harmful gases can be discharged in time. But in winter, to grasp the coordination of ventilation and insulation, to avoid excessive ventilation caused by too low temperature.

In the process of breeding, attention should be paid to the cleaning work of the internal and external environment of the pig house, and the feces and other excrement in the house should be cleaned in time. This can reduce the breeding of pathogens. In the process of breeding to develop a regular disinfection program and procedures, regular disinfection. Disinfection should be comprehensive and thorough to avoid the breeding and long-term existence of pathogens caused by lax disinfection. The disinfectant should be replaced regularly, and the same kind of disinfectant should not be used for a long time, so as to prevent pathogens from developing resistance to the disinfectant, resulting in poor disinfection effect.

Treatment. Isolation. Pig farms should formulate reasonable disease prevention and control system according to farm conditions and breeding scale (Andres & Davies, 2015). When pigs are infected with *S.suis* only, they should be isolated first, and their contaminated enclosures and feeds should be disinfected. 5% caustic soda can be selected as the disinfectant, and the pathogens should be completely and thoroughly sprayed to kill them. Contaminated areas should be disinfected continuously for more than 7 days, and pig farms should be empty for 30 days before use. The isolation house shall be disinfected twice a day. The sick pigs shall be disinfected with 0.1% negeramine, and the ground shall be washed and disinfected with 10% bleach powder to keep the ground dry to prevent the breeding of pathogens.

Local treatment. In pigs with lymph node abscess, tissue wounds will have ulceration and discharge of pus. These secretions also contain virulent *S.suis* (Hlebowicz, Jakubowski, & Smiatacz, 2019). Generally, after the abscess is mature, a sterile blade is used to cut open a wound, squeeze out pus, and then rinse with 3% hydrogen peroxide, and then rinse with 0.1% potassium permanganate water solution, and then smear 5% iodine tincture on the wound. If the abscess festers on its own, after rinsing can apply appropriate antibiotic ointment to inhibit bacterial infection. Clean the wound once a day until the lesion recovers.

Antibiotic therapy. Pig farm in the confirmed infection of *S. suis* disease, through the drug sensitivity test to determine the choice of antibiotics for treatment. According to the results of drug sensitivity, highly sensitive antibiotic drugs can be preferred (Seitz, Valentin-Weigand, & Willenborg, 2016). If weaned piglets are infected, chloramphenicol (25mg/kg body weight) can be injected intravenously, and 5% glucose and sodium chloride injection (200~300ml) can be injected intravenously twice /day for 3 to 5 days. If the sick pigs show neurological symptoms, they can be intramuscularly injected with chlorpromazine injection of 2mg/kg once per day-1 for 2~3 days; at the same time, they can be orally injected with vitamin C solution and intravenously injected with sulfadiazine for 50~100mg/kg once or twice per day for 2~3 days.

If there is no condition for drug sensitivity test, you can choose penicillin or streptomycin for treatment, in the treatment, but also for symptomatic treatment of symptoms. Anemone can be injected into the sick pig with fever, and for the sick pig with arthritis, turpentine can be applied to the affected area, which can play the effect of pain relief and swelling. Pigs with meningitis also need to be injected with VB1.

Conclusion. Streptococcus is one of the main zoonotic diseases of pigs, which is dangerous for humans. The most etiological factor of streptococcal infection in pigs is *S. suis* 2, which is usually isolated from clinically sick piglets and is considered the most virulent subtype of the pathogen. The main ways of transmission of infection are alimentary and respiratory infections. Infected animals often develop such symptoms assepticemia, meningitis, pneumonia, wet dermatitis, peritonitis, osteomyelitis, arthritis, pharyngitis, purulent pneumonia. For a long time, specialists managed

to control this infection quite successfully, but in the last few years, due to the problem of antibiotic resistance of bacteria

to medical drugs, this infection is becoming more and more widespread.

References:

- 1. Andres, V. M., & Davies, R. H. (2015). Biosecurity measures to control Salmonella and other infectious agents in pig farms: a review. *Comprehensive Reviews in Food Science and Food Safety, 14*(4), 317-335.
- 2. Arends, J., & Zanen, H. (1988). Meningitis caused by Streptococcus suis in humans. *Reviews of infectious diseases*, 10(1), 131-137.
- 3. Benga, L., Friedl, P., & Valentin-Weigand, P. (2005). Adherence of Streptococcus suis to porcine endothelial cells. *Journal of Veterinary Medicine, Series B*, *52*(9), 392-395.
- 4. Benga, L., Goethe, R., Rohde, M., & Valentin-Weigand, P. (2004). Non-encapsulated strains reveal novel insights in invasion and survival of Streptococcus suis in epithelial cells. *Cellular microbiology*, 6(9), 867-881.
- 5. Brassard, J., Gottschalk, M., & Quessy, S. (2001). Decrease of the adhesion of Streptococcus suis serotype 2 mutants to embryonic bovine tracheal cells and porcine tracheal rings. *Canadian Journal of Veterinary Research*, 65(3), 156.
- 6. Chang, P., Li, W., Shi, G., Li, H., Yang, X., Xia, Z., . . . Bei, W. (2018). The VraSR regulatory system contributes to virulence in Streptococcus suis via resistance to innate immune defenses. *Virulence*, *9*(1), 771-782.
- 7. Cloutier, G., D'allaire, S., Martinez, G., Surprenant, C., Lacouture, S., & Gottschalk, M. (2003). Epidemiology of Streptococcus suis serotype 5 infection in a pig herd with and without clinical disease. *Veterinary microbiology*, 97(1-2), 135-151.
- 8. Collin, M., & Ehlers, M. (2013). The carbohydrate switch between pathogenic and immunosuppressive antigen-specific antibodies. *Experimental dermatology*, 22(8), 511-514.
- 9. Dai, J., Lai, L., Tang, H., Wang, W., Wang, S., Lu, C., ... Wu, Z. (2018). Streptococcus suis synthesizes deoxyadenosine and adenosine by 5'-nucleotidase to dampen host immune responses. *Virulence*, *9*(1), 1509-1520.
- 10. de Greeff, A., Buys, H., Verhaar, R., Dijkstra, J., van Alphen, L., & Smith, H. E. (2002). Contribution of fibronectin-binding protein to pathogenesis of Streptococcus suis serotype 2. *Infection and immunity*, 70(3), 1319-1325.
- 11. Dekker, N., Bouma, A., Daemen, I., Klinkenberg, D., van Leengoed, L., Wagenaar, J. A., & Stegeman, A. (2013). Effect of spatial separation of pigs on spread of Streptococcus suis serotype 9. *PLoS One*, 8(4), e61339.
- 12. Deng, S., Xu, T., Fang, Q., Yu, L., Zhu, J., Chen, L., . . . Zhou, R. (2018). The surface-exposed protein SntA contributes to complement evasion in zoonotic Streptococcus suis. *Frontiers in Immunology*, *9*, 1063.
- 13. Done, S., Williamson, S. M., & Strugnell, B. W. (2012). Nervous and locomotor systems. *Diseases of swine, 10*, 303-305.
- 14. Feng, Y., Zhang, H., Wu, Z., Wang, S., Cao, M., Hu, D., & Wang, C. (2014). Streptococcus suis infection: an emerging/reemerging challenge of bacterial infectious diseases? *Virulence*, *5*(4), 477-497.
- 15. Gelberg, H. B. (2017). Alimentary system and the peritoneum, omentum, mesentery, and peritoneal cavity. *Pathologic basis of veterinary disease*, 324.
- 16. Gottschalk, M., & Segura, M. (2000). The pathogenesis of the meningitis caused by Streptococcus suis: the unresolved questions. *Veterinary microbiology*, 76(3), 259-272.
- 17. Gottschalk, M., Segura, M., & Xu, J. (2007). Streptococcus suis infections in humans: the Chinese experience and the situation in North America. *Animal health research reviews*, *8*(1), 29-45.
- 18. Guo, G., Du, D., Yu, Y., Zhang, Y., Qian, Y., & Zhang, W. (2021). Pan-genome analysis of Streptococcus suis serotype 2 revealed genomic diversity among strains of different virulence. *Transboundary and Emerging Diseases*, *68*(2), 637-647.
- 19. Haataja, S., Tikkanen, K., Hytönen, J., & Finne, J. (1996). The Galα1–4Gal-binding adhesin of Streptococcus suis, a Gram-positive meningitis-associated bacterium. *Toward Anti-Adhesion Therapy for Microbial Diseases*, 25-34.
- 20. Hedegaard, S. S., Zaccarin, M., & Lindberg, J. (2013). Septic arthritis caused by Streptococcus suis. *Ugeskrift for Laeger*, 175(22), 1574-1575.
- 21. Hlebowicz, M., Jakubowski, P., & Smiatacz, T. (2019). Streptococcus suis meningitis: epidemiology, clinical presentation and treatment. *Vector-Borne and Zoonotic Diseases*, *19*(8), 557-562.
- 22. Hughes, J. M., Wilson, M. E., Wertheim, H. F., Nghia, H. D. T., Taylor, W., & Schultsz, C. (2009). Streptococcus suis: an emerging human pathogen. *Clinical infectious diseases*, *48*(5), 617-625.
- 23. Jiang, X., Yang, Y., Zhou, J., Zhu, L., Gu, Y., Zhang, X., . . . Fang, W. (2016). Roles of the putative type IV-like secretion system key component VirD4 and PrsA in pathogenesis of Streptococcus suis type 2. *Frontiers in cellular and infection microbiology*, 6, 172.
- 24. LeBel, G., Vaillancourt, K., Yi, L., Gottschalk, M., & Grenier, D. (2018). Dipeptidylpeptidase IV of Streptococcus suis degrades the porcine antimicrobial peptide PR-39 and neutralizes its biological properties. *Microbial pathogenesis*, 122, 200-206.
- 25. Li, Q., Fu, Y., Ma, C., He, Y., Yu, Y., Du, D., . . . Zhang, W. (2017). The non-conserved region of MRP is involved in the virulence of Streptococcus suis serotype 2. *Virulence*, 8(7), 1274-1289.
- 26. Li, Q., Ma, C., Fu, Y., He, Y., Yu, Y., Du, D., . . . Zhang, W. (2017). Factor H specifically capture novel Factor H-binding proteins of Streptococcus suis and contribute to the virulence of the bacteria. *Microbiological research*, 196, 17-25.
 - 27. Li, T., & Yu, X. (2019). Isolation and identification of Streptococcus suis. Swine Production(3), 116-118.
- 28. Lin, X., Huang, C., Shi, J., Wang, R., Sun, X., Liu, X., . . . Jin, M. (2015). Investigation of pathogenesis of H1N1 influenza virus and swine Streptococcus suis serotype 2 co-infection in pigs by microarray analysis. *PLoS One, 10*(4), e0124086.

- 29. Liu, M., Xia, X., Liu, X., & Kasianenko, O. (2021). Research Progress on the pathogenic mechanism of Streptococcus suis 2. *Scientific Messenger of LNU of Veterinary Medicine and Biotechnologies. Series: Veterinary Sciences, 23*(104), 30-35.
- 30. Liu, W., Tan, M., Zhang, C., Xu, Z., Li, L., & Zhou, R. (2018). Functional characterization of murB-potABCD operon for polyamine uptake and peptidoglycan synthesis in Streptococcus suis. *Microbiological research*, 207, 177-187.
- 31. Liu, Z., Zheng, H., Gottschalk, M., Bai, X., Lan, R., Ji, S., . . . Xu, J. (2013). Development of multiplex PCR assays for the identification of the 33 serotypes of Streptococcus suis. *PLoS One*, *8*(8), e72070.
- 32. Lv, Q., Hao, H., Bi, L., Zheng, Y., Zhou, X., & Jiang, Y. (2014). Suilysin remodels the cytoskeletons of human brain microvascular endothelial cells by activating RhoA and Rac1 GTPase. *Protein & cell.* 5(4), 261-264.
- 33. Musyoki, A. M., Shi, Z., Xuan, C., Lu, G., Qi, J., Gao, F., . . . Haywood, J. (2016). Structural and functional analysis of an anchorless fibronectin-binding protein FBPS from Gram-positive bacterium Streptococcus suis. *Proceedings of the National Academy of Sciences*, 113(48), 13869-13874.
- 34. Norton, P. M., Rolph, C., Ward, P. N., Bentley, R. W., & Leigh, J. A. (1999). Epithelial invasion and cell lysis by virulent strains of Streptococcus suis is enhanced by the presence of suilysin. *FEMS Immunology & Medical Microbiology*, 26(1), 25-35.
- 35. Okwumabua, O., Williamson, C. H., Pearson, T. R., & Sahl, J. W. (2020). Draft Genome Sequence of a Streptococcus suis Isolate from a Case of Cattle Meningitis. *Microbiology Resource Announcements*, 9(19), e00153-00120.
- 36. Pian, Y., Gan, S., Wang, S., Guo, J., Wang, P., Zheng, Y., . . . Yuan, Y. (2012). Fhb, a novel factor H-binding surface protein, contributes to the antiphagocytic ability and virulence of Streptococcus suis. *Infection and immunity, 80*(7), 2402-2413.
- 37. Qian, Y., Zhang, Y., Yu, Y., Li, Q., Guo, G., Fu, Y., . . . Zhang, W. (2018). SBP1 is an adhesion-associated factor without the involvement of virulence in Streptococcus suis serotype 2. *Microbial pathogenesis*, 122, 90-97.
- 38. Quessy, S., Busque, P., Higgins, R., Jacques, M., & Dubreuil, J. D. (1997). Description of an albumin binding activity for Streptococcus suis serotype 2. *FEMS Microbiology Letters*, 147(2), 245-250.
- 39. Roy, D., Takamatsu, D., Okura, M., Goyette-Desjardins, G., Van Calsteren, M.-R., Dumesnil, A., Segura, M. (2018). Capsular sialyltransferase specificity mediates different phenotypes in Streptococcus suis and Group B Streptococcus. *Frontiers in microbiology, 9*, 545.
- 40. Segura, M., Calzas, C., Grenier, D., & Gottschalk, M. (2016). Initial steps of the pathogenesis of the infection caused by Streptococcus suis: fighting against nonspecific defenses. *FEBS letters*, *590*(21), 3772-3799.
- 41. Segura, M., Fittipaldi, N., Calzas, C., & Gottschalk, M. (2017). Critical Streptococcus suis virulence factors: are they all really critical? *Trends in microbiology*, *25*(7), 585-599.
- 42. Seitz, M., Valentin-Weigand, P., & Willenborg, J. (2016). Use of antibiotics and antimicrobial resistance in veterinary medicine as exemplified by the swine pathogen Streptococcus suis. *How to Overcome the Antibiotic Crisis: Facts, Challenges, Technologies and Future Perspectives*, 103-121.
- 43. Shi, J., Hu, D., Zhu, J., Zhang, X., Hou, T., Guo, J., . . . Wang, C. (2012). Capsular saliva acid of Streptococcus suis 2 influences virulence and host inflammatory responses. *Wei Sheng wu xue bao= Acta Microbiologica Sinica, 52*(4), 498-504.
- 44. Swildens, B., Stockhofe-Zurwieden, N., van der Meulen, J., Wisselink, H. J., Nielen, M., & Niewold, T. A. (2004). Intestinal translocation of Streptococcus suis type 2 EF+ in pigs. *Veterinary microbiology*, *103*(1-2), 29-33.
 - 45. Timoney, J. F. (2022). Streptococcus. Pathogenesis of bacterial infections in animals, 565-587.
- 46. Vadeboncoeur, N., Segura, M., Al-Numani, D., Vanier, G., & Gottschalk, M. (2003). Pro-inflammatory cytokine and chemokine release by human brain microvascular endothelial cells stimulated by Streptococcus suis serotype 2. *FEMS Immunology & Medical Microbiology*, 35(1), 49-58.
- 47. Vanier, G., Segura, M., Friedl, P., Lacouture, S., & Gottschalk, M. (2004). Invasion of porcine brain microvascular endothelial cells by Streptococcus suis serotype 2. *Infection and immunity*, 72(3), 1441-1449.
- 48. Vötsch, D., Willenborg, M., Oelemann, W. M., Brogden, G., & Valentin-Weigand, P. (2019). Membrane binding, cellular cholesterol content and resealing capacity contribute to epithelial cell damage induced by suilysin of Streptococcus suis. *Pathogens*, 9(1), 33.
- 49. Wang, J., Feng, Y., Wang, C., Zheng, F., Hassan, B., Zhi, L., . . . Jiang, S. (2017). Genome-wide analysis of a avirulent and reveal the strain induces pro-tective immunity against challenge with virulent Streptococcus suis Serotype 2. *BMC microbiology, 17*, 1-14.
- 50. Wang, S., Ma, M., Liang, Z., Zhu, X., Yao, H., Wang, L., & Wu, Z. (2022). Pathogenic investigations of Streptococcus pasteurianus, an underreported zoonotic pathogen, isolated from a diseased piglet with meningitis. *Transboundary and Emerging Diseases*, 69(5), 2609-2620.
- 51. Wang, Y., Gagnon, C. A., Savard, C., Music, N., Srednik, M., Segura, M., . . . Gottschalk, M. (2013). Capsular sialic acid of Streptococcus suis serotype 2 binds to swine influenza virus and enhances bacterial interactions with virus-infected tracheal epithelial cells. *Infection and immunity, 81*(12), 4498-4508.
- 52. Xia, X., Qin, W., Zhu, H., Wang, X., Jiang, J., & Hu, J. (2019). How Streptococcus suis serotype 2 attempts to avoid attack by host immune defenses. *Journal of Microbiology, Immunology and Infection*, *52*(4), 516-525.
- 53. Xia, X., Wang, X., Wei, X., Jiang, J., & Hu, J. (2018). Methods for the detection and characterization of Streptococcus suis: from conventional bacterial culture methods to immunosensors. *Antonie Van Leeuwenhoek, 111*, 2233-2247.
- 54. Xu, M., Wang, S., Li, L., Lei, L., Liu, Y., Shi, W., . . . Xu, M. (2010). Secondary infection with Streptococcus suis serotype 7 increases the virulence of highly pathogenic porcine reproductive and respiratory syndrome virus in pigs. *Virology journal*, 7(1), 1-9.

- 55. Yang, Q.-P., Liu, W.-P., Guo, L.-X., Jiang, Y., Li, G.-D., Bai, Y.-Q., . . . Jing, H.-Q. (2009). Autopsy report of four cases who died from Streptococcus suis infection, with a review of the literature. *European journal of clinical microbiology & infectious diseases*. 28, 447-453.
- 56. Yin, S., Daum, R. S., & Boyle-Vavra, S. (2006). VraSR two-component regulatory system and its role in induction of pbp2 and vraSR expression by cell wall antimicrobials in Staphylococcus aureus. *Antimicrobial agents and chemotherapy,* 50(1), 336-343.
- 57. Yu, Y., Qian, Y., Du, D., Xu, C., Dai, C., Li, Q., Zhang, W. (2016). SBP2 plays an important role in the virulence changes of different artificial mutants of Streptococcus suis. *Molecular bioSystems*, *12*(6), 1948-1962.
- 58. Zhang, C., Sun, W., Tan, M., Dong, M., Liu, W., Gao, T., Zhou, R. (2017). The eukaryote-like serine/threonine kinase STK regulates the growth and metabolism of zoonotic Streptococcus suis. *Frontiers in cellular and infection microbiology,* 7, 66
- 59. Zhang, G.-x. (2012). Diagnosis and Treatment of Swine Streptococcicosis. *Animal Husbandry and Feed Science*, 4(6), 243.
- 60. Zhang, S., Wang, J., Chen, S., Yin, J., Pan, Z., Liu, K., Jiang, Y. (2016). Effects of suilysin on Streptococcus suis-induced platelet aggregation. *Frontiers in cellular and infection microbiology, 6,* 128.
- 61. Zhang, Y., Lu, P., Pan, Z., Zhu, Y., Ma, J., Zhong, X., Yao, H. (2018). SssP1, a Streptococcus suis fimbria-like protein transported by the SecY2/A2 system, contributes to bacterial virulence. *Applied and Environmental Microbiology,* 84(18), e01385-01318.
- 62. Zhao, J., Pan, S., Lin, L., Fu, L., Yang, C., Xu, Z., Zhang, A. (2015). Streptococcus suis serotype 2 strains can induce the formation of neutrophil extracellular traps and evade trapping. *FEMS Microbiology Letters*, *362*(6).
- 63. Zhao, Y., Liu, G., Li, S., Wang, M., Song, J., Wang, J., Hu, F. (2011). Role of a type IV-like secretion system of Streptococcus suis 2 in the development of streptococcal toxic shock syndrome. *Journal of Infectious Diseases, 204*(2), 274-281.
- 64. Zheng, C., Ren, S., Xu, J., Zhao, X., Shi, G., Wu, J., Bei, W. (2017). Contribution of NADH oxidase to oxidative stress tolerance and virulence of Streptococcus suis serotype 2. *Virulence*, 8(1), 53-65.
- 65. Zheng, F., Shao, Z.-Q., Hao, X., Wu, Q., Li, C., Hou, H., Pan, X. (2018). Identification of oligopeptide-binding protein (OppA) and its role in the virulence of Streptococcus suis serotype 2. *Microbial pathogenesis*, *118*, 322-329.
- 66. Zhou, Z., He, H., Wang, K., Shi, X., Wang, Y., Su, Y., Zhang, Y. (2020). Granzyme A from cytotoxic lymphocytes cleaves GSDMB to trigger pyroptosis in target cells. *Science*, *368*(6494), eaaz7548. https://doi.org/10.1126/science.aaz7548
- 67. Zhou, Z., Zhu, X., Yin, R., Liu, T., Yang, S., Zhou, L., Ma, A. (2020). K63 ubiquitin chains target NLRP3 inflammasome for autophagic degradation in ox-LDL-stimulated THP-1 macrophages. *Aging*, *12*(2), 1747. https://doi.org/10.18632/aging.102710

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Стрептококова інфекція свиней (діагностика, профілактика, лікування)

Streptococcus suis (S. suis) є важливим зоонозним патогеном, який може спричинити серйозні захворювання, такі як менінгіт, пневмонія, ендокардит, полісерозит, артрит, септицемія та аборт у свиней. В останні роки рівень захворюваності на стрептококовий менінгіт демонструє значну тенденцію до зростання. Для людини S. suis також є небезпечним патогеном. Тому посилення профілактики та боротьби із захворюванням є актуальним завданням.

Передумовою індукції менінгіту є те, що S. suis проникає в центральну нервову систему та порушує гемато-енцефалічний бар'єр (ГЕБ). Через наявність гематоенцефалічного бар'єру, більшість бактерій з крові не може потрапити в мозок. Організм покладається на цей бар'єр, щоб захистити тканину мозку від пошкоджень і підтримувати гомеостаз центральної нервової системи. Мікросудинні ендотеліальні клітини мозку (ВМЕС) є основним компонентом гематоенцефалічного бар'єру, і різноманітні неврологічні захворювання пов'язані з дисфункцією гематоенцефалічного бар'єру, і S. suis може взаємодіяти з мікросудинними ендотеліальними клітинами головного мозку, а потім проникати через гематоенцефалічний бар'єр, щоб викликати інфекцію центральної нервової системи. Однак антибіотики, які використовуються для лікування інфекції, не можуть пройти через бар'єр, щоб досягти терапевтичної цільової ділянки, що є ключем до труднощів у контролі бактеріального менінгіту. Таким чином, з'ясування механізму проникнення S. suis через гематоенцефалічний бар'єр у центральну нервову систему є важливим проривом у розробці стратегії контролю менінгіту S. suis.

Внаслідок тривалого невиправданого застосування антибіотиків підвищується резистентність бактерій, лікування антибіотиками порушує нормальний гомеостаз організму та кишкову флору. Проблеми, спричинені тривалим, широкомасштабним використанням антибіотиків, стають дедалі серйознішими. Менінгіт, викликаний streptococcus suis, більше не можна лікувати звичайними антибіотиками. Тому необхідно мати глибоке розуміння патогенезу менінгіту. У цій статті розглядається дані щодо клінічних ознак і симптомів хвороби, патологічних змін, лабораторної діагностики, заходів профілактики та лікування стрептококової інфекції свиней за останні роки.

Ключові слова: стрептококова інфекція, свині, діагностика, профілактика, лікування.