Exudative epidermitis in pigs / Greasy pig disease

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Abstract

Exudative epidermitis caused by *Staphylococcus hyicus* is an acute, sporadic or enzootic disease in newborn piglets and newly weaned pigs. It is characterized by abundant secretion of the sebaceous glands of the skin and excoriation of the epidermis, leading to dehydration, necrosis and death of the affected animals. The spread of the disease in traditional and industrial farms is closely related to the conditions under which pigs are raised.

The article explains the epizootological features, the pathogenesis, the clinical and pathomorphological manifestations of the disease in the different forms of the course (peracute, acute, subacute and chronic). Out of laboratory and laboratory methods of diagnosis are indicated. It is focused on the possibilities of controlling the disease – such as therapy, metaphylaxis with medicated premixes and immunoprophylaxis, as well as general measures for prevention and control.

Key words: pigs, *Staphylococcus hyicus*, exudative epidermitis, control

Exudative epidermitis (EE), also knwn as "greasy pig disease", is an acute, sporadic or enzootic infectious diseases, mainly affecting neonatal and newly weaned piglets, which is characterized by hyperfunction of the sebaceous glands of the skin, with abundant secretion of exudate and excoriation of the epidermis without itching, leading to dehydration, increasing depression and death caused by *Staphylococcus hyicus* (L'Ecuyer, 1967; Wegener and Skov-Jensen, 1992; 2006).

History and distribution

Jones, (1956) first described the disease as an independent nosological unit.

In the second half of the 20th century in America, Europe and Australia, there were reports of a new skin disease in newborn pigs with high mortality, called "exudative epidermitis". Until then, diseases similar to exudative epidermis have been referred to as exudative dermatitis, pustular dermatitis, exfoliative dermatitis, necrotic dermatitis, infectious dermatitis, seborrhea oleosis and eczema, but their etiology has not been determined with certainty (Genev et al., 1960; Vachev, 1977). The causative agent of the disease was first described by Sompolynsky (1953) as *Micrococcus epidermidis* and only in the late 1970s was it determined as a representative of the genus *Staphylococcus*, group *S. hyicus* (L'Ecuyer, 1967; Wegener, et al., 1993; Taylor, 1995).

In our country, Genev et al. (1960) described a mass case of staphylococcal infection in suckling pigs, growing pigs and adult pigs. For the first time, Stoyanov et al. (1973) diagnosed exudative epidermitis in pigs by isolating 31 strains of staphylococci. Later, Vachev (1977) reported on dermonecrosis in pigs caused by staphylococci. In practice, exudative epidermitis occurs frequently, but is not well known and is confused with other skin diseases (Stoyanov, 1977; Ivanov, 1997; Djurov and Pankin, 1993; Dimitrova, 2012).

Etiology

The main cause of EE is deemed to be pathogenic strains of *Staphylococcus hyicus,* (*S. hyicus)* harbouring toxins. A key feature of the pathogenesis of this skin disease is the presence of bacterial strains that express exfoliative toxins known as ExhA, ExhB, ExhC, ExhD, SHETA and SHETB. The causative agent of exudative epidermis is a gram positive cocci in single cells, in pairs and short chains and clusters of aerobic and facultative anaerobic (Victor et al., 2013). The causative agent *Staphylococcus hyicus, subsp. hyicus*, can be isolated from the typical skin lesions (16%), but also from the mucous membranes of the head (7%), conjunctivae (12%), genitalia of sows (17%), from the foreskin of boars, as well as from organs of sick and dead pigs. Placing a pure culture of *S. hyicus* on the skin of a non-immune pig is sufficient to reproduce the disease. There are reports of many predisposing factors for the manifestation of exudative epidermitis and can be of economic significance as a cause of mortand and a cause of poor growth rate. (Wegener and Skov-Jensen, 1992).

S. hyicus is a Gram-positive, coagulase-negative cocci that forms 2 mm white, nonhemolytic colonies after 24 h of culture on blood agar and pink colonies on McConkey agar. Produces capsules, protein A and exfoliative toxin 29–30 kDa. A four antigenically different exfoliative toxins have recently been identified, and are throught to correlate with clinical disease, and its proposed designation is SHETB. It differs from other porcine staphylococci in its biochemical relationships. 6 serological types have been identified and at least 5 of them can be isolated in cases with clinical manifestation of EE. It differs antigenically from staphylococci in other animals. (Sato et al., 1991; Wegener and Schwarz, 1993; Andresen, 1998; Andresen, 2005).

S. hyicus is resistant to desiccation but does not form spores and is easily inactivated by heat. At a temperature of 70 °C, it lasts up to 60 min. For disinfection, iodine preparations, 2% chlorine lime, 1–2% formalin, 2% sodium hydroxide, etc. are used. It is sensitive *in vivo* to many antibiotics, including penicillins, cephalosporins, tetracyclines, lincomycin, tylosin, fluorinated quinolones, tiamutin and some aminoglycosides (Stoyanov et al., 1973; Stoyanov, 1977; Taylor, 1995; Whiteman, 2004; Popova, 2009; Dimitrova, 2012). Based on antibiotic disc sensitivity test, the organism was sensitive to rifampin, levafloxacin, ciprofloxacin and norfloxacin and was resistant to amoxicillin, erythromycin, chloramphenicol and ampicillin. (Victor et al., 2013).

There may be other staphycoccal species involved, and an exfoliative toxin similar to ExhB has been isolated from *S. chromogenes* isolated from healthy pigs. Experimental infection of pigs with this isolate led to clinical signs of EE (Andresen, 2005). An exfoliative toxin gene similar to ExhC, was isolated from a field isolate of *Staphylococcus sciuri,* that was associated with field infection and EE in piglets from a breeding herd (Chen et al., 2007).

Epizootological features

Pigs from 1 to 10 weeks of age, regardless of sex and breed, are most susceptible to epidermal exudative disease, with peak incidence in the first week. It has been established that, in addition to suckling pigs, weaned pigs under 3 months of age can also get sick, but less often, as well as single adult pigs. The resistance of pigs to infection increases with increasing age (Taylor, 1995; Ivanov, 1997). Pigs descended from infected growers or animals in which *S. hyicus* is endemic are generally nonsusceptible. This suggests that immunity plays an important role in disease prevention. *S. hyicus* causes no disease in humans. (Timothy, 2012).

The source of infection are sick pigs and adult infected pigs. *S. hyicus* in carriers pigs is harbored in the nasal cavity and conjunctiva, and vagina of sows and prepuce of boars. The spread of the infection over long distances occurs with the movement of healthy pigs-carriers. Outbreaks are seen following the introduction of a carrier animal and gilts to a non-immune herd. The newborn piglets are probably infected during parturition, and cross-contamination can occur when weanlings from different litters are group-housed. The causative agent is emitted with exudate and desquamated epithelium of the skin. This bacterium is very persistent in the en-

vironment, and aerosol transmission is possible. Spread by other species is of little concern. (Wegener and Skov-Jensen, 1999; Timothy, 2012). An important role in the occurrence and worsening of the disease is played by certain predisposing factors, such as the presence of other bacterial and vesicular viral diseases, insufficient nutrition in terms of zinc and vitamins, parasitosis, poor breeding conditions, poor hygiene and high humidity, immunological inadequacy of the young pigs, disturbed microflora and skin integrity, MMA syndrome in sows and genetic susceptibility. With the simultaneous impact of etiological and predisposing factors, sporadic cases of affected pigs increase, as a result, the disease develops into an enzootic. The manifestation is year-round, with a tendency for stationarity, but it can also be self-limiting within 3–4 months, if the importation of infected pigs is stopped. Morbidity varies significantly and can cover entire pigs, and can reach 20%, and mortality depends on the age of the pigs and can be up to 10% (Stoyanov, 1977; Ivanov, 1997; Dimitrova, 2012; Schwarz et al., 2021).

Pathogenesis

S. hyicus may directly penetrate the skin, a traumatic breach of the epidermis is the most common inciting factor for EE. The first changes are noticed as early as 12 hours after infection as reddening of the skin as a result of the multiplication of the causative agent between the cells of the epidermis, where microcolonies are formed. Inflammation and hyperplasia of the corneal layer occurs, followed by erosion. The damage deepens and reaches the germinal layer of the epidermis and the papillary layer of the dermis, which is accompanied by abundant fat secretion and serous exudate. The serous exudate passes into crusted deposits, which necrotize, and erosions of different sizes are formed under them. Pigs rapidly emaciate, become comatose and die from dehydration, loss of serum proteins and electrolytes. Are Several virulence factors described for *S. hyicus.* Exfoliative toxins are considered the most important virulence factor in the development of EE in pigs. The culture filtrate of *S. hyicus* could cause exfoliation in the skin of piglets and suggested this was due to exotoxin production. Six exfoliative toxins have been identified in *S. hyicus.* Four of these were identified in Denmark (Andresen, 1998) and two in Japan (Sato et al., 1999). Sick pigs acquire active immunity, which protects them from reinfection and relapses. Piglets from immune mothers receive passive colostral immunity, which protects them from disease for 6–8 weeks. The death is related to the resulting dehydration, loss of serum protein and electrolytes. (Taylor, 1995; Ivanov, 1997; Whiteman, 2004; Timothy, 2012).

Clinical signs

Stoyanov et al. (1973) distinguish three forms of the disease – superacute, acute and chronic, and other authors four – superacute, acute, subacute and chronic. The early clinical signs of exudative epidermitis (EE) are lethargy and erythematous skin. Pigs aged 5 days to 2 months are susceptible, and older pigs are more resistant. The signs progress to an exudative dermatitis and crusting. Severely affected piglets of the litter may die in 24 hours to 10 days. Adult animals may be mildly affected with small areas of EE on their backs and sides (Wegener and Skov-Jensen, 1999).

The peracute form is usually observed in pigs during the first week after birth. They are affected some or all of the pigs in the farrowing. It takes 24 to 48 h, with mortality reaching 100% in affected piglets. It occurs suddenly, with the appearance of red painful spots and blisters on the skin, depression, anorexia, exicosis and general weakness. The entire surface of the skin then becomes reddened, wrinkled, and covered with an oily, gray-brown exudate, passing into dirty brown crusts, chiefly around the eyes, mouth, ears, and on the abdominal wall. The temperature rarely rises. (Djurov and Pankin, 1993; Taylor, 1995; Wegener and Skov-Jensen, 1999; Whiteman, 2004).

The acute form of EE is more common in 3-10-week-old pigs and develops within 3–5 days, but with a lower mortality rate. The skin of infected pigs is covered with an oily exudate or dirty brown plaques to blackish brown scales, most commonly in the folds around the eyes, ears and neck. At first, the skin lesions are 1–2 cm in diam-

eter, but after 3–4 days they expand to include the cheeks, carpal joints, abdominal wall and rump. The skin acquires an oily appearance, which is why the disease is also called "oily disease" of pigs. Blisters and ulcers appear on the snout and tongue of sick pigs. Loss of appetite and dehydration are constant signs. Pruritus (itching) and fever are not observed. Diarrhea, hypodynamia, hypothermia, coma and death (up to 90%) were observed in one part of the pigs, and dehydration, exhaustion and death occurred in the remaining pigs, but to a lesser extent. In young pigs, death occurs within 3–5 days. Higher morbidity and mortality is found in small pigs. Usually, in adult animals, the disease is benign. Skin lesions are limited. They develop slowly and do not merge with each other. Lethality is low, but recovery is slow and pig growth is delayed (Taylor, 1995; Ivanov, 1997; Wegener and Skov-Jensen, 1999; Whiteman, 2004; Victor et al., 2013).

The *subacute form* of the disease proceeds protractedly, the exudate forms brown scabs on the skin, protruding above the surface, especially around the eyes, ears, abdomen, carpal joints and less often on the whole body. In this form, the mortality rate is even lower.

A *chronic course* of the disease is observed in a small percentage of affected pigs, where only limited areas of the body are affected. Thickening and wrinkling of the skin, thick crusts that crack deeply are found. Adult animals may be mildly affected with small areas of exudative epidermitis on their backs and sides. In older pigs, skin lesions are limited, do not confluent, development is protracted, development is suppressed, and the outcome of the disease is favorable. Slowed growth and reduced herd productivity by 35% are characteristic of sick pigs. In adult pigs, the disease varies in severity, but mainly manifests as localized lesions on the back and flanks. In addition to skin lesions, *S. hyicus* infection in adult pigs can also cause arthritis, abortion, metritis and vaginitis. In the chronic course of the disease, the growth of the affected pigs slows down and in general the productivity of the herd decreases (Stoyanov et al., 1973; Wegener and Skov-Jensen, 1992; Djurov and Pankin, 1993; Taylor, 1995; Dimitrova, 2012).

Pathological changes

In the autopsy of suckling pigs that died from the peracute and acute form of exudative epidermitis, Djurov and Pankin (1993) found carcasses in poor or medium preservation, with signs of dehydration and staining of the anal area with diarrheal feces. Macroscopic changes in exudative epidermitis are characterized by red spots, bubbles, sticky exudate, crusts, thickening and cracking of the skin, and in some cases erosive stomatitis. These lesions are most often found on the ears, around the eyes, on the ventral thorax, and on the abdomen. After removing the crusts, red spots are found. The subcutaneous connective tissue in the affected areas is impregnated with a fibrous transudate. The liver is dystrophic and the kidneys are pale and edematous. The ureters and renal pelvis are dilated, pale, filled with a serous infiltrate and yellowish crystals of urate and sodium chloride. Superficial lymph nodes are enlarged, with a juicy cut surface. The brain is also edematous. In the subacute and chronic form of EE, the affected pigs are severely dehydrated and have a bad smell. The skin lesions found were extensive and varied in number and severity among pigs from the same litter. Inflammation of the external ear and of the head in general, incl. of the mouth and tongue. Subcutaneous lymph nodes are swollen. (Djurov and Pankin, 1993; Victor et al., 2013).

Histopathological changes

Microscopic findings are the presence of both a superficial and deep pyoderma that may extend to involve the subcutis, and the presence of a brownish exudate. (Thompson, 1988; Taylor, 1992). Hypertrophy of the sebaceous glands, inflammatory changes and many microabscesses with staphylococci are found in the skin. A precipitate of dead cells is found in the renal pelvis and ureters, and sometimes pyelonephritis is also found. (Djurov and Pankin, 1993; Taylor, 1995; Whiteman, 2004).

Diagnosis

Sings and lesions are usually adequate for diagnosis of EE in young piglets. The confirmation can be made by isolation of *S. hyicus* or by histo-

pathology (Stoyanov, 1977; Ivanov, 1997; Taylor, 1995). Material from scraped scabs and crusts, contents of blisters on the nose and tongue, skin and pieces of internal organs (spleen, liver, kidneys and lymph nodes) are sent for bacteriological examination. Bacteriological examination is carried out to isolate and identify the causative agent. The growth is reported in meat peptone broth and blood agar with the addition of 7.5% sodium chloride. Selective media containing potassium thiocyanate or less than 10% NaCl may aid isolation. The biochemical and coagulase activity is investigated. Coagulase-positive are from 24% to 56% of the isolates. An indirect enzyme-linked immunosorbent assay (ELISA) test for the detection of toxins ExhA, ExhB, and ExhC, was developed as an alternative to phage typing (Andresen, 1998). The susceptibility of *S. hyicus* to antimicrobial agents is tested. Pieces of the damaged skin areas and pieces of liver and kidneys are sent for pathomorphological examination.

In a differential diagnostic aspect, it is necessary to exclude necrotic dermatitis, parakeratosis, trichophytosis, scabies, swine pox, swine circovirus disease, pellagra and avitaminosis. Scabies and ringworm are excluded by microscopic examination (Stoyanov, 1977; Ivanov, 1997; Taylor, 1995; Whiteman, 2004; Dimitrova, 2012).

Prevention and treatment of diseases

Autogenous bacterins made from strains cultured from a particular herd and given to non-immune sows are useful to protect the litters of newly introduced sows. The exfoliative toxin and the bacterial cells should be included as antigens when the vaccine is made. An indirect ELISA or phage typing can be used to select a toxigenic strain for vaccine production (Andresen, 1998).

General preventive measures include repair of the production premixes and regular high-quality disinfection of the pens for pregnant sows and farrowing pens. Before giving birth, sows are bathed with disinfectant solutions (Feziasept, Aseptin, Disinfect-B, C-4, chlorhexidine or providone-iodine shampoos) and placed in dry, clean and disinfected farrowing boxes. Nutrition is improved and a lack of vitamins, micro and macro elements in the feed is not allowed. Sows and pigs are regularly treated against scabies. Isolation of affected animals and keeping them in separate groups will help to contain and eradicate the disease. (Ivanov, 1997; Dimitrova, 2012; Timothy, 2012).

Treatment

Treatment with antibiotics will reduce the severity of the dermatitis and aid recovery. This bacteria is potentially susceptible to several antibiotics: however, plasmid-mediated resistance is common. The choice should be based on sensitivity testing whenever possible (Laber et al., 2002; Timothy, 2012). According to Popova (2009), these are some penicillins and cephalosporins, macrolides, amphenicols and tetracyclines, depending on the results of the antibioticograms and the severity of the infection. Futagawa-Saito et al. (2008) tested 207 strains of *S. hyicus*, of which 150 exfoliative toxin-producing and 57 non-toxinogenic strains, for susceptibility to 13 antimicrobial agents. The established frequency of resistance to penicillin and ampicillin was 76.8%; to erythromycin – 56%; trimethoprim-sulfamethoxazole – 28.5%; chloramphenicol – 24.2%; kanamycin – 19.8%; and doxycycline – 1.4%. Resistance to chloramphenicol and trimethoprim-sulfamethoxazole was markedly higher in the toxigenic strains, while resistance to kanamycin and erythromycin was higher in the non-toxigenic strains. The established resistance to 2 or more antimicrobials is 85.5% in general, but noticeably higher in toxigenic strains – 89.3%, against 75.4% in non-toxigenic ones.

Sick pigs are isolated in dry rooms and treated parenterally and topically. The reports indicate that *S. hyicus* is resistant to many antimicrobial agents. (Wegener and Skov-Jensen, 1992; Timothy, 2012). Penicillins (amoxacillin 7 mg/kg for 3 days), macrolides (tylosin, tilmicosin) and lincomycin (10 mg/kg for 3–5 days), tetracyclines (oxytetracycline 10 mg/kg for 3 days) are prescribed, tiamulin (tiamutin at 10 mg/kg, for 3 days), aminoglycosides (gentamicin, kanamycin and amikacin at 10 mg/kg, for 3–5 days), fluoroquinolones (enrofloxacin, pefloxacin, marbofloxacin and ciprofloxacin at 3–4 mg/kg, for 3 days),

potentiated sulfonamides (sulfamethoxazole in combination with trimethoprim) (Dimitrova, 2012). Injection of the antimicrobial is recommended, but it may be given orally in less severe cases. A high proportion of staphylococci isolated from pigs affected with exudative epidermitis were found to be resistant to β-lactam antimicrobials. Park et al. (2013) investigated β-lactam resistance in *S. hyicus* and *S. aureus* isolated from pigs by disc diffusion method, where they found 73.3% resistance. Methicillin resistance can be identified in a variety of staphylococcal species isolated from pigs. The resistance may be passed from one species to another species of staphylococci. These data suggest that transmission to or from the porcine pathogen *S. hyicus* may also occur.

Disinfecting baths with iodophors and douching skin lesions with rivanol, sulfathiazole, tetracycline, gentamicin, etc. ointments are effective. Good results are obtained with topical cloxacillin at 10,000 UE/g lanolin, with 1% hydrocortisone in combination with parenteral cloxacillin. Disinfection baths of sick pigs with iodophors in a concentration of 0.5–1% (Jozan, Disinfect B) have proven to be very effective. The recommendations have included spraying the pigs several times with solutions such as Nolvasan or Virkon. Sick pigs are provided with water ad libitum. (Djurov and Pankin, 1993; Taylor, 1995; Ivanov, 1997; Laber et al., 2002; Whiteman, 2004).

Metaphylaxis

Sows from the affected herds are treated metaphylactically for 7 days before farrowing with appropriate medicinal premixes (tylosin-prem., tilmicosin-prem., lincomycin-prem., oxytetracycline-prem., doxycycline-prem., Tiamutin-prem. in the doses indicated by the manufacturer) (Whiteman, 2004; Dimitrova, 2012).

Immunoprophylaxis

In stationary farms, sows are vaccinated in the third month of pregnancy with inactivated vaccines, and pigs – at weaning. Prevention is obviously better than treatment because piglet mortality can be high. (Andresen, 1998; Dimitrova, 2012).

Measures in case of proven exudative epidermitis: – isolation and therapy of sick pigs; - metaphylactic treatment of healthy animals through the feed or drinking water with any of the above-mentioned antimicrobial agents; – the feed provides an optimal amount of zinc, selenium, vitamin E and biotin; – the feed of sows can be medicated with appropriate antibiotic premixes; – non-technological regrouping of pigs is avoided; – good hygiene is maintained and regular and high-quality disinfection is carried out (Djurov and Pankin, 1993; Taylor, 1995; Ivanov, 1997; Dimitrova, 2012; Victor et al., 2013; Fraile, 2013).

From the overview of exudative epidermitis in pigs, it is clear that during the last 20 years in different countries of the world, significant studies have been carried out on the etiology, pathogenesis, diagnosis, prevention and control of the disease. At the same time, in our country, due to the change in the ownership of pig farms, from state to private, and some structural and functional changes in the activity of the veterinary service, systematic studies on the distribution, diagnosis and control of exudative epidermitis in pigs have not been conducted, which is why there is no current data on the disease. Taking into account the changed conditions under which pig farming is carried out in the country, as well as a number of new, primary diseases in pigs, often occurring in association with secondary bacterial infections, it becomes clear the need for new studies on exudative epidermis, which will benefit both for practicing veterinarians as well as for owners of pig farms of different sizes, technology and health status.

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