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# Distribution and aetiology of valvular endocarditis in suckling and weaned piglets in Serbia

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**Abstract:** There are limited data available in the literature about the frequency and distribution of endocarditis in suckling and weaned piglets. The goal of this study was to investigate the frequency and distribution of lesions from cases of spontaneous valvular endocarditis in young piglets and to determine the bacterial agents involved in those lesions. Valvular endocarditis lesions in suckling piglets were detected in 21/454 cases (4.62%) and in 20/306 cases (6.53%) in weaned piglets. Streptococci were the dominant bacterial species isolated in 68% of the cases; *S. suis* was identified in 48.7% of the cases, *S. pyogenes* in 7.3% and *S. spp.*, which could not be further identified, were cultured in 12.1% of the cases. *E. rhusiopathiae* was identified as the causal agent of endocarditis in four cases in the weaned piglets. In three cases, *A. suis* and *S. aureus* were isolated, and, in two cases, *A. pleuropneumoniae* was identified. *Y. enterocolitica* was isolated form one case of valvular endocarditis in a weaned pig. The finding of valvular endocarditis caused by *Y. enterocolitica* is an uncommon finding in pigs, and to the authors' knowledge, there are no other reports of this agent being the cause of endocarditis in pigs. The involvement of *Y. enterocolitica* in endocarditis might have been assisted by the predisposing role of immunosuppressive viruses, such as porcine reproductive and respiratory syndrome.

**Keywords:** swine; piglets; endocarditis; bacteria

Valvular endocarditis is the most common acquired lesion of the porcine endocardium (Loynachan 2012). Such lesions are noted in clinically normal, slaughtered pigs at a level of 2% to 10% (Jones 1980). In many studies, it is established that *Streptococcus suis* and *Erysipelothrix rhusiopathiae* are the dominant agents of valvular endocarditis in pigs (Esgleas et al. 2005; Jensen et al. 2010; Loynachan 2012). However, from cases of spontaneous porcine endocarditis, other bacterial species have also been isolated, such as *Streptococcus porcinus, Staphylococcus aureus, Staphylococcus hyicus, Trueperella pyogenes, Actinobacillus pleuropneumoniae, Actinobacillus suis, Escherichia coli, Pasteurella multocida, Listeria monocytogenes, Fusobacterium necrophorum* and *Enterococcus faecalis* (Pedersen et al. 1984; Katsumi et al. 1997). Regardless of the aetiology, the mitral valve is most commonly affected and then, by the order of the frequency, the semilunar aortae, tricuspid and semilunar a. pulmonalis valves. Although it has been described that valvular endocarditis can occur in all age groups of pigs, ex-

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cept in cases of experimental infections (Jones 1982; Johnson et al. 1986), there is very little information available about the naturally occurring distribution of lesions and causes of valvular endocarditis in young animals, particularly in suckling and weaned pigs. Therefore, in this retrospective study, we report the frequency and patterns of the distribution of the lesions from cases of spontaneous valvular endocarditis in suckling and weaned piglets and the results of the bacteriological examination of the corresponding lesions.

## MATERIAL AND METHODS

In total, 454 suckling (from 2 kg to 10 kg) and 306 weaned (from 10 kg to 25 kg) dead piglets from conventional farrow-to-finish pig farms were necropsied at the Veterinary Institute Belgrade from January 2018 to March 2019. The animals were from seven farms originating amongst different regions in Serbia and were 1 to 12 weeks of age. All the farms were continuous flow operations, and had a poor management system of health issues, including biosecurity, inadequate sanitation and housing-environmental management shortcomings. All the farms included in the study vaccinated against erysipelas (sows in the first third of pregnancy; piglets at 85 days) and six farms vaccinated against porcine circovirus 2 (PCV2). The post-mortem findings for these submissions were varied and included systemic (porcine reproductive and respiratory syndrome – PRRS (84 cases), PCV2 – systemic disease (14 cases), Glässer's disease (78 cases), respiratory [porcine respiratory disease complex – PRDC (125 cases)], swine pleuropneumonia (87 cases), enteric [colibacillosis (102 cases)], swine dysentery (33 cases) and skin [exudative epidermitis (67 cases)] diseases as well as other conditions caused by bacteria such as streptococci (134 cases), *A. suis* (19 cases) and *S. aureus* (17 cases). At necropsy, particular attention was paid to the inflammatory changes on the endocardium. The hearts were opened in the direction of the blood flow with an incision through the atrium and right ventricle to the right ventricular apex and then along the right ventricular wall adjacent to the interventricular septum into the pulmonary outflow tract. The left side was incised from the atrium to the left ventricular apex to expose the endocardial surface and mitral valve; subsequently, the mitral valve cusp was incised to expose the outflow tract into the aorta. The lesions were characterised by gross inspections.

From the forty-one hearts with macroscopically visible valvular endocarditis lesions, avoiding contamination, the material was sampled for a histological and bacteriological examination.

Table 1. Primers used for the detection of the pathogens studied in this work

| Pathogens | Oligonucleotides | Reference |
|---|---|---|
| *E. rhusiopathiae* | F: AGATGCCATAGAAACTGGTA<br>R: CTGTATCCGCCATAACTA | Makino et al. (1994) |
| *S. aureus* | F: ATAGAGATGCTGGTACAGG<br>R: GCTTCCGATTGTTCGATGC | Hookey et al. (1998) |
| *S. pyogenes* | F: AAAGACCGCCTTAACCACCT<br>R: TGGCAAGGTAAACTTCTAAAGCA | Liu et al. (2005) |
| *A. suis* | F: GAGCTGGGAAGCTCGACTAT<br>R: CCCCCATCTTCAAACAGGAT | Kariyawasam et al. (2011) |
| *A. pleuropneumoniae* | F: TGGCACTGACGGTGATGA<br>R: GGCCATCGACTCAACCAT<br>nested F: GGGGACGTAACTCGGTGATT<br>nested R: GCTCACCAACGTTTGCTCAT | Schaller et al. (2001) |
| *Y. enterocolitica* | F: AATACCGCATAACGTCTTCG<br>R: CTTCTTCTGCGAGTA ACGTC | Wannet et al. (2001) |
| Multiplex PCR<br>*S. suis*<br>serotype 2 (plus 1/2) | *S. suis* F: CAGTATTTACCGCATGGTAGATAT<br>*S. suis* R: GTAAGATACCGTCAAGTGAGAA<br>serotype 2 (plus 1/2) F: GTTGAGTCCTTATACACCTGTT<br>serotype 2 (plus 1/2) R: CAGAAAATTCATATTGTCCACC | Marois et al. (2004) |

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## Histopathology

Samples of the valves' tissue were fixed by immersion in 10% buffered formalin. The fixed tissue was dehydrated, embedded in paraffin wax, sectioned at 5 µm, and stained with haematoxylin and eosin (H&E). The slides were examined under a light microscope.

## Bacteriology

For the bacterial isolation, Columbia agar with 5% defibrinated ovine blood and MacConkey agar were used (Hi Media, Mumbai, India). For the *A. pleuropneumoniae* isolation, an *S. aureus* culture as the source of the V factor and a chocolate agar with a Poly Vitex supplement (BioMerieux, Marcy l'Etoile, France) were used.

The plates were incubated in an aerobic condition on 37 °C for 24 to 48 hours. The bacteria were identified using the colony morphology, haemolytic pattern on the blood agar and a further microscopic examination (Gram staining), standard biochemical methods, a serogrouping agglutination test of streptococci of Lancefield Groups A, B, C, D, F and G (Microgen Bioproduct Ltd., Camberley, U.K.) and a commercial test (BBL – Crystal, Becton Dickinson, Cockeysville, USA). The bacterial isolates were further tested by PCR (polymerase chain reaction) using a DreamTaq PCR Master Mix kit (Thermo Scientific, Vilnius, Lithuania) and the primers described for the DNA detection of *E. rhusiopathiae*, *S. aureus*, *S. pyogenes*, *A. suis*, *A. pleuropneumoniae* and *Y. enterocolitica* (Makino et al. 1994; Hookey et al. 1998; Schaller et al. 2001; Wannet et al. 2001; Liu et al. 2005; Kariyawasam et al. 2011) (Table 1). For the detection of *S. suis* serotype 2 (plus 1/2), a multiplex PCR (Multiplex PCR kit; Qiagen GmbH, Hilden, Germany), as described previously, was applied (Marois et al. 2004).

## RESULTS

The incidence and distribution of the lesions from the cases of naturally-occurring valvular endocarditis in the piglets studied is presented in Table 2. Of the 760 (454 suckling and 306 weaned) piglets, examined macroscopically, visible valvular endocarditis was diagnosed in 5.39% (41/760) of the

Table 2. Incidence and distribution of the valvular endocarditis in the suckling and weaned piglets

| Production stage | Positive | Mitral valve | Mitral and semilunar aortic valves | Mitral and tricuspid valves | Tricuspid and semilunar a. pulmonalis valves | Mitral semilunar aortic tricuspid semilunar a. pulmonalis valves |
|---|---|---|---|---|---|---|
| Suckling piglets (2–10 kg) | 21/454 (4.62%) | 17/454 (3.74%)<br>*S. suis* (8)<br>*Streptococcus spp.* (3)<br>*S. pyogenes* (2)<br>*A. suis* (2)<br>*S. aureus* (1)<br>*A. pleuropneumoniae* (1) | 2/454 (0.44%)<br>*S. suis* (2) | 1/454 (0.22%)<br>*S. suis* (1) | 1/454 (0.22%)<br>*S. suis* (1) | – |
| Weaned piglets (10–25 kg) | 20/306 (6.53%) | 13/306 (4.24%)<br>*S. suis* (4)<br>*Streptococcus spp.* (2)<br>*S, aureus* (2)<br>*S. pyogenes* (1)<br>*E. rhusiopathiae* (1)<br>*A. suis* (1)<br>*A. pleuropneumoniae* (1)<br>*Y. enterocolitica* (1) | 3/306 (0.98%)<br>*E. rhusiopathiae* (2)<br>*S. suis* (1) | 2/306 (0.65%)<br>*S. suis* (1)<br>*E. rhusiopathiae* (1) | 1/306 (0.32%)<br>*S. suis* (1) | 1/306 (0.32%)<br>*S. suis* (1) |
| Total | 41/760 (5.39%) | 31/760 (4.07%) | 5/760 (0.65%) | 3/760 (0.39%) | 2/760 (0.26%) | – |
| Positive | 41/41 (100%) | 31/41 (75.6%) | 5/41 (12.1%) | 3/41 (7.31%) | 2/41 (4.87%) | – |

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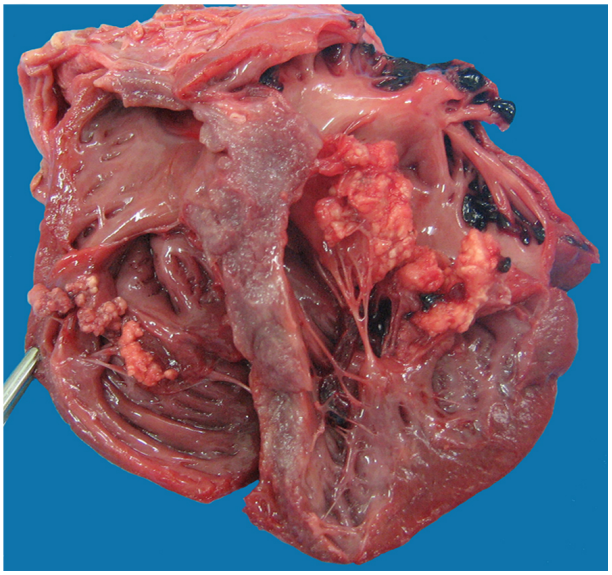

Figure 1. Valvular endocarditis of a weaned piglet caused by *Streptococcus suis.* Multiple, yellow-red thrombotic masses, (vegetations) of different shapes, on the mitral and tricuspid valves can be seen

cases. Valvular endocarditis lesions in the suckling piglets were detected in 21 cases (21/454, 4.62%). The mitral valve was involved in 20 of the cases and was the only valve affected in 17 of the cases, the mitral valve and the semilunar aortae valve were affected in two of the cases, the mitral valve and tricuspid valve were affected in one of the cases, and the tricuspid valve and semilunar aortae valve were affected in one of the cases. In the weaned piglets, valvular endocarditis was diagnosed in 20 cases (20/306, 6.53%) with only the mitral valve being affected in 13 of the cases, the mitral valve and the semilunar aortae valve were affected in three of the cases, the mitral valve and tricuspid valve were affected in two of the cases and the tricuspid valve and semilunar aortae valve were affected in one of the cases.

In the mild cases, the endocardial lesions were characterised by the presence of solitary yellow-red nodules on the leaflets' ending. Severe lesions were characterised by deep valve tissue damage with the formation of nodous, mushroom or cauliflower thrombotic vegetations (vegetative endocarditis) (Figure 1).

Thrombotic masses were found on the atrioventriculares valves on the atrial surface of the valves and on the semilunar valves on the ventricular surface, near to the cusps' endings. In the cases in which thrombotic deposits were situated on one valve, they had a verrucous shape only (verrucous endocarditis), whereas, in the cases of the extended and intensively inflammatory processes, mushroom and cauliflower shapes were dominant. In one case (a weaned pig), all the valves were affected by inflammatory processes.

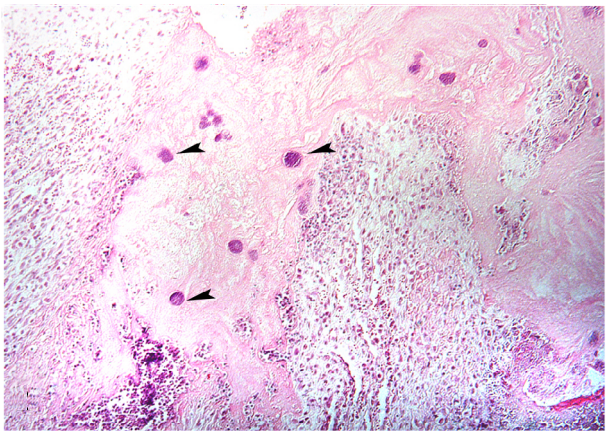

Figure 2. Cusp of the mitral valve of a suckling piglet with *Streptococcus suis* endocarditis. Abundant masses of fibrin with embedded bacterial colonies and infiltration with neutrophils are present (H&E)

The main findings in the histopathological examination of the affected valves were the partial replacement of valve tissue by a mixture of fibrin, neutrophils and mononuclear inflammatory cells, necrotic debris, mineralised foci and plaques. The thickened valves had a notably increased fibroblastic proliferation.

In the lesions, accumulated layers of fibrin with numerous embedded bacterial colonies underlaid by a zone of infiltrated leucocytes and granulation tissue were noted (Figure 2).

The bacterial agents isolated and information about the co-morbidities in the 41 cases of naturally-occurring valvular endocarditis in suckling and weaned piglets are presented in Table 3.

Streptococci were the dominant bacterial species isolated in 68% (28/41) of the cases; *S. suis* was identified in 48.7% of the cases (20/28), *S. pyogenes* in 7.3% (3/28) and *S. spp.*, which could not be further identified, were cultured in 12.1% (5/28) of the cases. *E. rhusiopathiae* was identified as the causal agent of endocarditis in four of the cases in the weaned piglets.

Also, in three cases, *A. suis* and *S. aureus* were isolated, and in two cases *A. pleuropneumoniae* was identified. *Y. enterocolitica* was isolated from the one case of valvular endocarditis in a weaned pig.

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Table 3. Bacterial agents isolated from the valvular endocarditis lesions and information about the comorbidities in 41 suckling and weaned piglets

| Bacterial species | Suckling piglets (2–10 kg) | Comorbidities in suckling piglets | Weaned piglets (10–25 kg) | Comorbidities in weaned piglets | Total (%) |
|---|---|---|---|---|---|
| *S. suis* serotype 2 (plus 1/2) | 12/454 (2.64%) | traumatic stomatitis (5)<br>mono/polyarthritis (3/1)<br>PRRS viremia (2)<br>skin abscess (1) | 8/306 (2.61%) | skin abrasions (4)<br>PRRS viremia (2)<br>fibrinous *polyserositis (1)*<br>PRDC (1) | 20/41 (48.7%) |
| *S. pyogenes* | 2/454 (0.44%) | skin abscesses (2) | 1/306 (0.32%) | skin abrasions (1) | 3/41 (7.3%) |
| *Streptococcus spp.* | 3/454 (0.66%) | fibrinous *polyserositis (2)*<br>dermatitis/epidermitis (1) | 2/306 (0.65%) | skin abscess (1)<br>fibrinous *polyserositis* (1) | 5/41 (12.1%) |
| *E. rhusiopathiae* | 0/454 (–) | – | 4/306 (1.30%) | PRDC (1)<br>PRRS viremia (2)<br>fibrinous *polyserositis (1)* | 4/41 (9.7%) |
| *A. suis* | 2/454 (0.44%) | *A. suis* – septicaemia (2) | 1/306 (0.32%) | *A. suis* – septicaemia (1) | 3/41 (7.3%) |
| *S. aureus* | 1/454 (0.22%) | monoarthritis (1) | 2/306 (0.65%) | exudative epidermitis (2) | 3/41 (7.3%) |
| *A. pleuropneumoniae* | 1/454 (0.22%) | App – pleuropneumonia (1) | 1/306 (0.32%) | App – pleuropneumonia (1) | 2/41 (4.8%) |
| *Y. enterocolitica* | 0/454 (–) | – | 1/306 (0.32%) | PRRS viremia (1) | 1/41 (2.4%) |
| Total | 21/454 (4.62%) | – | 20/306 (6.53%) | – | 41/760 (5.39%) |

PRDC = porcine respiratory disease complex; PRRS = porcine reproductive and respiratory syndrome

## DISCUSSION

The results of the frequency and distribution of the lesions in spontaneous valvular endocarditis in the suckling and weaned piglets support the general pattern of valvular endocarditis reported in the literature for older pigs (Guarda and Negro 1989; Bauman and Bilkei 2002; Loynachan 2012). However, the predilection of the mitral and semilunar aortae valves for valvular endocarditis, regardless of the aetiology in these age groups of pigs are not described as strongly as in this study.

In addition, the analysis of the distribution of the lesions showed that tricuspid and semilunar a. pulmonalis valves were affected in two cases (one case in a suckling pig and one case in a weaned pig), and from the author's knowledge, this is the first description of such patterns of the distribution of the lesions of valvular endocarditis in suckling and weaned piglets. For the localisation of the observed thrombotic masses (on the atrioventricular and semilunar valves on the atrium and ventricular surface), we have no further explanation than that what was proposed earlier for slaughtered pigs, that the solid and less flexible dense connective tissue (through its content of collagen fibres) which supports the endocardium on the ventricular side, forms a barrier that forces the inflammation within a valve toward the atrial chamber (Jarplid et al. 1997).

The multiple valvular endocarditis lesions observed in both age groups of pigs were a part of the primary diseases in which these lesions represent an accompanying finding. In all the related cases, according to the clinical background, post-mortem findings and bacteriological results, a streptococcal disease/infection was most frequently diagnosed. Thus, the damaged gingival epithelium (associated with a poor teeth clipping technique) with the subsequent gingivitis and periodontal inflammation (traumatic stomatitis), and the inflammatory lesions of the joints (arthritis) in suckling piglets, and skin wounds (lesions and abrasive skin damage) arising from injuries from poor pen structure and/or fighting, combined with the probable persistent streptococcal colonisation of the tonsils in the weaned piglets, were the most important entry sites for the streptococci. The subsequent valvular endocarditis is a sequel to an extracardiac streptococcal infection associated with one or more episodes of bacteraemia (Kumar et al. 1992; VanVleet and Ferrans 2007). Consequently, streptococci were most frequently isolated (21/48, 68%) from the valvular endocarditis lesions with *S. suis*

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being the main infectious agent of valvular endocarditis in both age groups of the piglets.

Although naturally occurring bacterial valvular endocarditis in pigs is generally known to be caused by *E. rhusiopathiae* (Bauman and Bilkei 2002; Jensen et al. 2010; Loynachan 2012), the bacteriological results from our study showed a lower incidence than expected in the aethiopathogenesis of the valvular endocarditis in the suckling piglets (valvular endocarditis caused by *E. rhusiopathiae* was not detected in suckling piglets), in contrast to its detection rate in the weaned piglets (9.7%). The reasons that contribute to the considerable incidence of *E. rhusiopathiae* valvular endocarditis in weaned piglets, despite the vaccination programme against erysipelas, may be: affecting the shortening duration of the maternal immunity, a greater impact on the increasing susceptibility of the piglets to bacteria, and facilitating the easier transmission of pathogens including *E. rhusiopathiae* from older to younger pigs in the conventional farrow-to-finish system.

The prevalence of spontaneous valvular endocarditis in both age groups of pigs caused by other identified bacteria than the streptococci, *E. rhusiopathiae*, is correlated with the incidence of the primary diseases they cause, whereby more diverse bacteria were detected from the valvular endocarditis lesions among the weaned piglets. This is somewhat expected, due to the high infection pressure (in the continuous flow system) with both bacteria and viruses, especially those originating from older animals and, therefore, the occurrence of various underlying diseases in the weaned piglets. The pathogenesis of valvular endocarditis caused by other bacteria depends on the virulence factors of the pathogens and their specific mechanisms of activity, which, from primary infection sites, facilitate bacterial colonisation of the endocardium (Kumar et al. 1992; VanVleet and Ferrans 2007). In most cases, the pathogens spread and adhere to the walls of the blood vessels, with microcolonies being established by septic emboli (VanVleet and Ferrans 2007).

One interesting finding was the detection of the *Y. enterocolitica* isolates in the valvular endocarditis lesions in a weaned pig. *Y. enterocolitica* in pigs normally causes little (diarrhoea in weaned pigs) or no disease (Thomson et al. 2001) and although this bacterium has been isolated from 15 cases of severe acute endocarditis in humans (Krajinovic et al. 2007) there is no reported case of valvular endocarditis in swine (nor in any other farm animals) caused by this agent. However, the bacterial cultures yielded a pure growth of *Y. enterocolitica* in this case, suggesting that this organism may be implicated in the development of the valvular endocarditis lesion. Furthermore, evidence shows that *Y. enterocolitica* binds to the cellular fibronectin and several collagen types, through the membrane, protein Yad A (Schultze-Koops et al. 1993) strengthens this possibility, although the presence of this protein in this isolate was not assessed.

In this case, it could be speculated that numerous underlying conditions can facilitate a *Y. enterocolitica* infection followed by colonisation of the tonsils and/or intestines. The endocardium may have been infected during a subsequent episode of the bacteraemia. Among many underlying diseases and conditions that can lead to *Y. enterocolitica* bacteraemia, several may have particular significance, such as a PRRS active infection, PCV2 – a systemic or enteric disease and stress-induced immunosuppression (Butts and Sternberg 2008).

In conclusion, the spontaneous valvular endocarditis in the suckling and weaned piglets, reported here, represents the accompanying lesions associated with the underlying diseases.

Thus, in most cases (68%), a streptococcal disease/infection has been diagnosed, and streptococci, particularly *S. suis* (48.7%), were the dominant bacterial species isolated in both age groups of the piglets. *E. rhusiopathiae* was detected as a causative agent of valvular endocarditis in the weaned piglets only (4). The other cases were associated with *A. suis* and *S. aureus* (3) or *A. pleuropneumoniae* (2). In general, the frequency and patterns of the distribution of the lesions on the heart valves are very similar to those reported in older pigs, however, lesions on the tricuspid and semilunar aortae pulmonalis valves only, in cases of naturally occurring valvular endocarditis, have not been reported on earlier in either suckling or weaned piglets.

Although most of the infections by *Y. enterocolitis* in pigs have been reported as being asymptomatic, to our knowledge, this is the first reported case of valvular endocarditis caused by this bacterium in pigs.

## Conflict of interest

The authors declare no conflict of interest.

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