

Sudden death associated with bleeding into digestive system of finishing pigs – a review

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Abstract

Sudden deaths of finishing pigs in modern pig herds cause economic losses and therefore draw constant attention worldwide. In the case of peracute mortality associated with gastrointestinal bleeding, pigs usually die during a short period without clinical manifestations. Necropsy can detect bleeding into various parts of the digestive system. Determining the exact aetiology of the sudden death can be difficult in many cases. Diseases and conditions such as gastric ulcers, abdominal torsion, haemorrhagic bowel syndrome and infectious diseases should be taken into account in the differential diagnosis. Because some of these diseases still have an unclear aetiology and pathogenesis, the aim of our work was to provide a summary of existing knowledge as well as to describe related pathognomonic pathological changes.

Finisher, swine, gastrointestinal bleeding, peracute mortality

The profitability and productivity of pig finishing units is affected by many factors, including nutrition, housing conditions and mortality of fattening pigs. Moreover, the mortality rate, average daily weight gain, and feed conversion ratio are important indices used to measure performance in grow-finish pigs (Brumm 1995; Whittemore 1998). It is clear that financial losses due to mortality vary greatly depending on whether pigs die at the beginning or at the end of the finishing period (Maes et al. 2001).

Finishing pigs are pigs close to the market weight and can die due to a variety of causes, including infectious diseases causing viraemia, septicaemia, and toxæmia, as well as diseases causing inflammation and changes in the cardiovascular system (endocarditis, mulberry heart disease, porcine stress syndrome), pathology in the urinary and also digestive tract (Leiting 2011). At present, a major threat to pig farming is the African swine fever which can also cause sudden death (Gogin et al. 2013; Yoon et al. 2020).

Sudden death or peracute mortality associated with haemorrhage into the digestive system of pigs is characterized by the death of a previously asymptomatic animal, mostly in a good condition, with a good appetite, and without presence of diarrhoea (Friendship 2004; Pejsak 2007; Novotný et al. 2016). It is also important to note whether the sudden death occurred with or without the presence of fever. In the case of fever, the involvement of infectious pathogens must be considered (Fedorka-Cray et al. 2000).

The objective of this paper was to review the diseases leading to sudden deaths of finishing pigs manifested by haemorrhage into the digestive system.

Gastric ulceration

Gastric ulcers are common in pigs kept under modern husbandry conditions and may cause clinical disease or death when haemorrhage or perforation occurs (Friendship 2004, 2006; Omotosho et al. 2016). Stomach erosive lesions together with pre-ulcerative parakeratosis lesions belong to the most common postmortem findings in the stomach of

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pigs at slaughter (Melnichouk 2002; Monteiro 2011). The prevalence of this disease varies a lot depending on the husbandry and feeding regimes (Berrios et al. 2019). The prevalence of lesions in the literature is reported to be about 32–65% (Christensen and Cullinane 1990; Straw et al. 1992). Some sources even report a prevalence of up to 93% (Haesebrouck et al. 2009).

The gastric mucosa of pigs is compartmentalized into four regions that differ in appearance and structure: esophageal, fundic, cardiac and pyloric. The only the esophageal region of stomach has non-glandular mucosa. It is located at the site of the esophageal entry into the stomach and is actually a continuation of the esophageal mucosa (Gandarillas and Bas 2009). Gastric ulcers are almost exclusively restricted in this non-glandular region (Kopinski and McKenzie 2007; De Witte et al. 2017). Less frequently and due to other causes such as bacterial septicaemia or an outbreak of pneumonia, ulcers can also occur in the mucous and acid-secreting regions (Monteiro 2011).

Blood loss is often so severe and acute that the pig is found dead before clinical signs have been detected (Friendship 2004). Sudden death usually involves only a small number of pigs in a grower-finisher barn, but higher numbers can occur sporadically and are usually associated with a triggering event such as feed disruption (Melnichouk 2002). Mortality due to bleeding from deep, erosive lesions may reach 1% or higher, with culling losses of 3% to 5% in such herds (Nielsen 1995). Similarly, other studies estimate approximate annual mortality attributable to gastroesophageal ulcers from 0.5% to 0.75% (Deen 1993; Melnichouk 2002).

The aetiology has not been fully understood but is likely to be multifactorial. The pig helicobacter, *Helicobacter suis*, is present in the stomach and is capable of causing chronic gastritis and ulceration (Hellemans et al. 2007; De Bruyne et al. 2012; De Witte et al. 2017). Other bacteria can ferment carbohydrate, producing organic acids and predisposing the pars oesophagea to ulceration. Pneumonia caused by infectious pathogens and infestation by the stomach worm, *Hyostromylyus rubidis*, may predispose to ulceration of the glandular portion of the stomach (Dionissopoulos et al. 2001; Berrios et al. 2019). Although in some studies with mild *Hyostromylyus* infestation, neither ulceration nor haemorrhage was recorded in the gastric mucosa of the fundic region (Sarashina and Taniyama 1986). Nutrition and management are also important in the maintenance and exacerbation of the lesions, especially finely ground or pelleted feeds may be involved (Wondra et al. 1995; Ayles et al. 1996). Stresses such as transport, starvation, interruption of feed intake, mixing and overcrowding increase the incidence of gastric ulceration in pigs (Lawrence et al. 1998). Hormonal changes have been also hypothesized to be involved (Bubenik et al. 1998).

Pathogenesis

Ulcers can develop quickly within 12 h (Davies et al. 1994). Generally, an imbalance between gastric acid secretions, changes in mucous composition and gastric mucosa protection is necessary for the development of ulcers in pigs. When the gastric mucosa is damaged, hydrochloric acid and pepsin can easily further damage the submucosa of the stomach. The particle size and physical form of the feed influences the pepsin and acid secretion in the stomach also by high fermentable carbohydrates. The ulcerative process may progress from a thickening of the mucosa at the beginning to erosions, ulceration, and haemorrhage to a perforated ulcer in the stomach (Friendship 2004; Berrios et al. 2019).

Pathological findings

Pigs that have died because of haemorrhage from ulcers appear notably pale but generally in a good body condition and without bloating. The stomach usually does not contain feed. It is often distended with clotted blood or blood mixed with ingesta and the intestines are

filled with blood or melanic content – brown digested blood (Schwartz 2002; Friendship 2004). Usually, macroscopic findings at necropsy are sufficient for diagnosis. Because a gastric ulcer death may be secondary to another clinical problem (Dionissopoulos et al. 2001), it is important to note other diseases present in the animal at the time of necropsy (Friendship 2004).

Stomachs from slaughtered pigs can be also analysed for the presence of macroscopic lesions suggesting gastric ulcer. Scores from 0 to 4 were attributed to the stomachs depending on the extent of the lesions, namely: score 0 – normal stomach; score 1 – stomach showing parakeratosis; score 2 – stomach showing active or chronic ulcer covering less than 33% of the pars oesophagea; score 3 – stomach showing active or chronic ulcer covering from 33 to 66% of the pars oesophagea; score 4 – stomach showing active or chronic ulcer covering more than 66% of the pars oesophagea (Kopinski and McKenzie 2007; Prazeres et al. 2017).

Torsion of abdominal organs

These acute abdominal accidents mainly involve torsion of the stomach and intestines. Death can be sporadic or in infrequent outbreaks (White 2002). Long-loined pigs may be more likely to develop mesenteric torsion than shorter pigs (Straw et al. 2002; Harris 2013).

Gastric torsion

Gastric torsion is a rare condition in which sudden death follows feeding. The stomach twists around the longitudinal axis of the digestive tract, also known as volvulus. This condition most often occurs following a once-daily feeding and can be prevented by feeding twice daily.

The possibility of gastric torsion is related to the capacity of the stomach and its placement. The stomach belongs to the most dilated part of the digestive tract, and its capacity ranges from approximately 3.5 l in slaughter pigs to 5 l in adults. Under pressure, the capacity increases to 8 and 12 l for slaughter and adult pigs, respectively (Lærke and Hedemann 2012). The pig stomach is not firmly fixed in its position. Its long axis is directed transversely. The empty stomach is located in the diaphragm dome, surrounded by liver lobes. However, the distended stomach may touch the right abdominal wall at the level of rib 13, just caudally from the liver. A grossly distended stomach can touch the left rib arch, it can push away the jejunal loops, coming into contact with the large intestine (Popesko et al. 1992).

Aetiology

Adult animals that are occasionally susceptible to torsion of the intestine, are more likely to suffer gastric dilation and torsion, which is equally fatal (White 2002). Deaths are usually sporadic although they can be of significance where, for example, fresh whey is being fed and bloating occurs. The predisposition is overeating with liquid feed. Prolonged over-excitement at feeding may also predispose.

Pathogenesis

It is believed that rapid ingestion of feed possibly coupled with fermentation of feed mass leads to dilatation and, in conditions of excitement, to torsion. In addition, gorging and fast intake of feed can lead to possible swallowing of large amounts of air which triggers the process of gastric dilatation (White 2002). Another explanation of the stomach dilatation and volvulus may be the dysfunction of the sphincter between the oesophagus and the stomach and the obstruction of the outflow through the pylorus caused by gastric

ulcers. This has been well documented in dogs (Parton et al. 2006). The pylorus, which is usually located ventrally and directed to the right of the body of the stomach, is twisted cranially or to the left of the midline because of gas accumulation. If the volvulus is greater than 180°, the oesophagus is closed off, thereby preventing the animal from relieving the condition by belching or vomiting. Gastric torsion and gas distension result in low blood pressure, decreased return of blood to the heart, ischaemia of the stomach, and shock. Without circulation, the stomach tissue discolours and dies. The lungs are congested as a result of the pressure on the diaphragm. Death is usually due to this pressure build-up causing either suffocation or heart failure. Mortality can also result from rupture of the spleen and loss of blood into the abdominal cavity (White 2002).

Pathological findings

It often affects the best pigs of the group that are found dead. Pigs that have died from torsion may have distended abdomens. Paleness is not a regular feature (White 2002). Torsion of the spleen and sometimes associated splenic rupture may also be present. Most pigs with gastric torsion had large amounts of gastric content composed of fluid and partly digested food (Filipov 1983; Morin et al. 1984).

Intestinal torsion

Intestinal torsion is one of the most common causes of sudden death in growing pigs between 25 and 100 kg (White 2002). Also known as intestinal volvulus or twisted gut.

The intestinal tract of the pig is suspended within the abdominal cavity below the spine. The small intestine of fully developed pigs is 16–21 m long and has a capacity of more than 20 l. The large intestine reaches a length of up to 5 m with a capacity of up to about 10 l (Lærke and Hedemann 2012). This mass of intestinal loops is attached to vertebrae by the mesentery only in a short section, which can lead to possible partial or even complete torsion of intestines.

Aetiology

It is caused by consumption of large amounts of feed in a very short time. An important provoking factor is over-feeding and abnormal fermentation of the contents of both the small and large intestine result in gas formation, increased pressure and torsion (Ruckebusch and Bueno 1976). Highly fermentable rations containing whey or other carbohydrate-rich liquid may reach the caecum, ferment, and cause torsion of the whole large intestinal mass around its root in finishers. Other factors include improper feed supply, especially when feeding is interrupted, inadequate feeder space and overstocking associated with gorging of feed, mainly in the last phase of finishing period. In addition, a very important factor is colitis, which is associated with the multiplication of bacteria in the hindgut, leading to greater feed fermentation and gas production (White 2002; Martineau et al. 2008; Thomson and Friendship 2019).

Pathogenesis

Intestinal instability is increased by excessive gas production. Sudden movements then allow the gut loops to rotate and cause a constrictive twist. Torsion of the long axis of mesentery cuts off the blood supply to the gut and causes rapid death (White 2002; Thomson and Friendship 2019).

Pathological findings

In most cases, pigs are found dead, pale, and bloated (White 2002). Intestines that have a complete obstruction of the blood flow are black and necrotic; partial volvulus is characterised by markedly red colour (Schwartz 2002). Rotation is usually counter-

clockwise when viewed from the ventrocaudal aspect of animal (Thomson and Friendship 2019). The normal position of the colon and caecum is on the left side with the apex of the cecum pointing caudally. The caecum and colon may be rotated to the right when there is partial or complete volvulus of the small and/or large intestine (Schwartz 2002). The mesenteric root should be palpated before opening the entire abdomen, with the pig in a position on the back. Torsion is palpable as a knot depending on how much of mesentery is twisted. However, only partial rotation is difficult to demonstrate at necropsy, which makes diagnosis more challenging. Mesenteric torsion obstructs the venous outflow of blood which causes blood to pool and stagnate in the intestine (Schwartz 2002; Haris 2013). Occasionally, intestinal torsion is unwinding due to transport or postmortal repositioning of the gut. However, the red discoloration of the intestine is still visible even if the volvulus has been repositioned. Similarly, rough handling of the carcass during transport can result in apparent torsions, but these can be distinguished by the absence of the intense red colour from the twisted organ (Häni et al. 1993; Straw et al. 2002; Grahofer et al. 2017).

Haemorrhagic bowel syndrome

It is also called intestinal haemorrhage syndrome, porcine intestinal distension syndrome, bloody or red gut (Martineau et al. 2008; Thomson and Friendship 2019). Haemorrhagic bowel syndrome (HBS) is a sporadically occurring disorder in fattening pigs, characterized by sudden death in combination with severe abdominal distension and intense red colouration of the intestine (Labuscagne et al. 2012; Grahofer et al. 2017). Haemorrhagic bowel syndrome primarily affects rapidly growing pigs between 4 and 6 months of age of 70 to 120 kg (Straw et al. 2002; Hollis 2006; Harris 2013). Production systems attribute mortality rates of 0.1–7% to HBS in the grow-finish phase (Schwartz 2002).

Aetiology

Exact aetiology and pathogenesis of HBS is still unclear (Hollis 2006; Martineau et al. 2008). However, several specific risk factors associated with the deaths have been defined (Schwartz 2002). Some authors allege that HBS is the result of intestinal volvulus; others declare that this condition may be associated with infectious pathogens or a combination of non-infectious and infectious causes (Häni et al. 1993; Schwartz 2002; Straw et al. 2002; Labuscagne et al. 2012; Novotný et al. 2016; Grahofer et al. 2017). Highly fermentable rations such as liquid feed or rations including whey have been most often associated with clinical cases of HBS. It is also hypothesized that deaths in the absence of intestinal torsion may be caused by excessive intra-abdominal pressure that reaches the same levels of pigs with “whey bloat” (Thomson et al. 2007). The overgrowth and (or) alteration of bacteria such as *Clostridium* or *E. coli* in the digestive tract with consequent over-production of toxic substances should be considered (Labuscagne et al. 2012; Novotný et al. 2016; Grahofer et al. 2017). This has been well documented in ruminants, where various risk factors contribute to the overgrowth of normal intestinal flora, with sequelae such as acidosis, endotoxaemia, enterotoxaemia, and death (Dennison et al. 2002). In addition, chronic dysbacteriosis of the intestinal tract due to the non-justified routine use of antimicrobials may also predispose (Grahofer et al. 2017). Of course, animal husbandry aspects are also very important, such as nutrition, management, feeding regime, nutritional hygiene, housing density, and optimal feeding space (Hollis 2006).

Pathogenesis

Excessive intra-abdominal pressure in “whey bloat” pigs cause obstruction of the mesenteric veins and obstruction of the venous return (Gudmundsson et al. 2001).

Clostridium spp., *E. coli* or other intestinal bacterial overgrowth with the production of exotoxins, enterotoxins or endotoxins may have a localized effect and can perhaps create shock-like conditions in the intestine (Schwartz 2002). Antimicrobials introduced to the feed provoke a selective pressure that may lead to long lasting changes in livestock commensal microorganisms, helping the pathogens to multiply in the gastrointestinal tract (Götz et al. 1996; Salyers and Abile-Cuevas 1997; Grahofer et al. 2017).

Pathological findings

Recently dead animals have a typical marked pallor of the skin and pronounced distension of the abdomen (Straw et al. 2002). An indication of peracute mortality is the stomach filled with fresh feed. The intestines have a similar appearance to cases of intestinal torsion, but no obvious displacement of the intestines or mesenteric torsion are detected at necropsy (Thomson and Friendship 2019). Transmural congestion affecting the serosa, intestinal wall, and mucosa is observed, but microscopic lesions are similar and not specific for either volvulus or HBS (Schwartz 2002). The small intestine is thin-walled at necropsy and filled with either clotted or unclotted blood (Hollis 2006; Harris 2013). The mesenteric vasculature is extremely engorged due to the obstruction to the venous return. The large intestine may contain tarry faecal material, but lesions of gastric and intestinal mucosa, suggestive of gastric ulceration, proliferative haemorrhagic enteropathy, swine dysentery or salmonellosis are absent (Hollis 2006). Sometimes the large intestine is without pathological findings, containing regular ingesta. A sharp demarcation is seen between the normally coloured part of the intestine and the red affected intestine (Grahofer et al. 2017).

Diseases associated with infectious causes

Not all pigs that die suddenly and are pale have stomach or intestinal accidents. Differential diagnosis includes other causes of sudden death, which, in contrast to previously mentioned diseases, are often associated with bloody diarrhoea. These include porcine proliferative enteritis, swine dysentery, and salmonellosis.

Porcine proliferative enteropathy

An acute form of porcine proliferative enteropathy known as proliferative haemorrhagic enteropathy (PHE) can in some cases cause sudden death and acute haemorrhagic diarrhoea in replacement animals and finishing pigs close to the market age (Guedes 2004).

Clinical presentation is mainly characterized by high mortality associated with anaemia and haemorrhagic diarrhoea (Pascua et al. 2015). However, some animals die without faecal abnormality and show only marked pallor (Vannucci et al. 2019). Globally, it is estimated that 96% of farm sites are infected, with approximately 30% of weaner-to-finisher pigs having lesions of variable severity and duration (Stege et al. 2000; McOrist et al. 2003).

Aetiology

The aetiological agent of PHE in swine is the obligate intracellular Gram-negative bacterium *Lawsonia intracellularis* (Lawson et al. 1993; Gebhart et al. 1993). An explosive outbreak of PHE occurs mainly in the naive pig population. Various organizational activities in farms such as transportation of pigs, changes in the feeding regimen or the diet, sudden temperature variations, overpopulation, impaired hygienic or immune status as well as a genetic predisposition could influence the appearance, the development and severity of porcine proliferative enteropathy (Bona and Bilkei 2003; Hagen and Bilkei 2003).

The pathogenesis of this more acute lesion is not well characterized. However, haemorrhage occurs concurrently with the widespread degeneration and desquamation of many epithelial cells and leakage from the capillary bed (Vannucci and Gebhart 2014; Vannucci et al. 2019).

Pathological findings

Based on morphological properties, four forms of the disease are recognised in pigs: porcine intestinal adenomatosis (PIA), necrotic enteritis (NE), regional ileitis (RI) and proliferative haemorrhagic enteropathy (PHE). In the more recent literature, the terms acute (PHE) and chronic (PIA or NE) forms are preferred (Bronsvooort et al. 2001; Jacobson et al. 2010). The intestine is dilated and the wall is thickened by serosal oedema and mucosal proliferation. Proliferative haemorrhagic enteropathy is marked by severe bleeding into the lumen of the intestine, but with underlying cell proliferation typical of proliferative enteropathy. The lumen of the ileum and proximal colon usually contain one or more formed blood clots combined with fibrinonecrotic debris. The rectum may contain black tarry faeces of mixed blood and digesta (McOrist et al. 2006; Vannucci et al. 2019).

Swine dysentery

Swine dysentery (SD) is a severe enteric disease in grower-finisher pigs, characterized by bloody to mucoid diarrhoea associated with reduced growth performance and variable mortality (Burrough 2017). Occasionally, pigs are peracutely affected and die within a few hours to days as a result of the endotoxin release (Hampson and Burrough 2019).

Aetiology

Swine dysentery is associated with strongly haemolytic, Gram-negative anaerobic *Brachyspira* species. Except *B. hyodysenteriae*, the classical agent of SD, two other strongly beta-haemolytic spp. (*B. hampsonii* and *B. suanatina*) are also agents of SD in some parts of the world (Mushtaq et al. 2015; Hampson and Burrough 2019). Outbreaks of SD are often associated with various stressors, including moving to new pens, mixing with different animals, weighing, change in feed, over-crowding, and/or extreme changes in ambient temperature. It also coincides with removal of antimicrobials used to control respiratory and enteric diseases (Hampson and Burrough 2019). Factors affecting disease transmission include environmental survival of the pathogen, animal husbandry factors (production system, production stage, farm management), role of vectors, diet influence and interaction of the microorganism with gut microbiota (Backhans et al 2010; Alvarez-Ordóñez et al. 2013).

The pathogenesis of SD is incompletely understood. Individual animal susceptibility to development of SD varies and is, in part, due to differences in the colonic microbiota (Hampson and Burrough 2019). The spirochetes must survive the acidic environment of the stomach and pass through the small intestine until ultimately reaching, colonizing, and proliferating in the caecum and colon (Burrough 2017). *Brachyspira hyodysenteriae* colonizes the mucus layer and is often found deep within crypts (Milner and Sellwood 1994; Kennedy and Yancey 1996). Colonization within the mucus layer and crypts helps further protect the spirochetes from potential oxygen toxicity at the surface, and flagellar function is important in mucus penetration (Burrough 2017). The mechanisms of tissue destruction in SD are not fully understood. However, it can be associated with disorganization of the mucin layer and its structure (Quintana-Hayashi et al. 2015), which may allow improved access of *B. hyodysenteriae* to the underlying epithelium. The haemolysin(s) may then cause local damage, resulting in epithelial sloughing. Subsequent

mucosal and submucosal invasion by secondary bacteria and the protozoan *Balantidium coli* may contribute to lesion formation (Hampson and Burroughs 2019).

Pathological findings

Lesions are characterized by haemorrhage, necrosis, and increased mucin secretions (Wilberts et al. 2014; Quintana-Hayashi et al. 2015), and are limited to all or part of the caecum through the descending colon with the spiral colon being most consistently affected in acute SD (Wilberts et al. 2014). The small intestine remains clinically unaffected (Stanton 2006).

Salmonellosis

In terms of sudden deaths, the most important is *Salmonella enterica* subsp. *enterica* serovar Choleraesuis that causes a generalized, life-threatening septicaemia with lesions in various organs (Griffith et al. 2019). Sudden death without forewarning signs occurs mainly in pigs in the acute phase of septicaemia. The first evidence of disease are dead pigs with cyanotic extremities and abdomens. In most outbreaks, mortality is high, and morbidity is variable but generally less than 10% (Wilcock and Schwartz 1992; Griffith et al. 2019; Zhitnitskiy 2020). Diarrhoea is normally not a feature of *S. Choleraesuis* infection until the third or fourth day of disease, when watery yellow faeces may be seen (Fedorka-Cray et al. 2000).

Aetiology

Host adapted *S. Choleraesuis* and non-host adapted *S. enterica* serotype Typhimurium are two of the most frequent isolated serovars recovered from clinically diseased swine (Loynachan et al. 2004). Septicaemic *S. Choleraesuis* typically affects growing pigs less than 5 months old. Intestinal salmonellosis caused by *Salmonella* Typhimurium and antigenically similar *S. enterica* serotype I 1,4,[5],12:i:- are also common in pigs (Moreno Swift et al. 2009). Unlike *S. Choleraesuis*, these salmonellas cause enterotyphlocolitis with lower mortality that occurs only after several days of diarrhoea, presumably as the result of hypokalaemia and dehydration (Griffith et al. 2019). *Salmonella* Choleraesuis outbreaks are frequently associated with “stress” factors (Wilcock 1981) that have already been mentioned in previous diseases.

Pathogenesis

Salmonella Choleraesuis is host-adapted to pigs meaning that infection is limited 99% exclusively to pigs (Uzzau et al. 2000). *Salmonella* Choleraesuis is transmitted by direct contact and by faeces or faeces-contaminated materials from clinically infected or carrier pigs. Feed and other species of animals are not a source of infection. Oral infection of pigs with *S. Choleraesuis* leads to colonization of palatine tonsil (Gray et al. 1995) and lymphoid tissues of intestine. The organism can survive within macrophages and neutrophils which serve as vehicles for *Salmonella* dissemination (Gray et al. 1996). Tissue localizations of infection then occur, the most common being liver (hepatitis), spleen (splenomegaly), lymph nodes, lung (pneumonia), brain (encephalitis) and joints (synovitis). There, the organism destroys local tissues, causes inflammation, and persists (Schwartz 1991).

Pathological findings

Cyanosis of the skin, especially on the ears, feet, tail and abdomen, is commonly found on pigs that died unexpectedly. Gross lesions include swollen mesenteric lymph nodes, splenomegaly, hepatomegaly, and lung congestion. Random white foci of necrosis are often observed on the liver (Reed et al. 1986; Wilcock and Schwartz 1992), leading

to varying degrees of icterus severity. In pigs that have displayed coughing, typical lesions associated with bacterial pneumonia such as consolidation of the cranioventral lobes can be seen (Turk et al. 1992; Zhitnitskiy 2020). Pigs dying of acute septicemia generally have no lesions in the intestine (Schwartz 1991). When pigs have been sick for several days and show signs of diarrhoea, intestinal lesions similar to the ones seen with *S. typhimurium* infections are observed: pseudomembranes on the ileum and button ulcers in the colon (Zhitnitskiy 2020).

Conclusion

Peracute mortality is associated with the death of pigs within a few hours or at night, which limits the performance of a clinical examination. Therefore, postmortem examination is the standard primary method of diagnosis. If gastrointestinal bleeding is detected, it is important to focus on the most common causes as outlined in this article. Before a detailed examination of individual parts of the digestive system, it is always necessary to evaluate the possible torsion of the abdominal organs by palpation of the mesenteric root. Final diagnosis is then made by excluding diseases that are not related to the occurrence of the identified lesions. If necessary, additional laboratory tests are used. Study of the literature has shown that the most questionable aetiology and pathogenesis is for the haemorrhagic bowel syndrome where the various causes of the onset and development of this disorder are taken into account. In an effort to minimize losses and manage outbreaks of sudden finisher mortality, it is necessary to implement measures in relation to nutrition, housing, environment and risk factors. Moreover, the improvement on-farm hygiene measures and stimulation of the immune status of animals as well as implementation of management practices that emphasize all-in/all-out production play an important role in the reduction of infectious pathogens.

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