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Review Article

PORCINE CONGENITAL SPLAYLEG: A REVIEW

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SUMMARY

Porcine congenital splayleg (PCS) is a clinical condition of newborn piglets, characterised by muscle weakness, resulting in the inability to properly stand and walk, with affected limbs extended sideways or forwards. It is arguably the most important congenital defect of commercial piglets and causes significant economic loss to pig farmers. The aetiology and pathogenetic mechanisms for PCS are still not well understood. Various management, nutrition and genetic factors have been found associated with PCS problems, but the actual cause remain obscure. Proper management and good farm practise are essential to control this multi-factorials PCS problem.

Keywords: congenital splayleg, porcine, piglet, skeletal muscle

Introduction

Preweaning mortality in piglets constitutes a major loss to the pig industry. Congenital abnormalities account for a small but significant proportion of preweaning losses (Partlow *et al.*, 1993). They have been reported more frequently in pigs than in any other domestic animal species (Priester *et al.*, 1970). A number of abnormalities may be seen in newborn piglets or shortly after birth. Briefly, congenital conditions can be categorised as: (1) spontaneous developmental abnormalities, such as cystic lymph nodes, (2) heritable abnormalities such as congenital meningoencephalocoele, atresia ani, arthrogryposis, porcine congenital splayleg, (3) infectious agents such as congenital tremor type AI (myocloniacongenita), (4) nutritional deficiency or poisoning such as mulberry heart disease and (5) unknown causes such as bleeding navel syndrome (Taylor, 2013).

Porcine congenital splayleg (PCS), also known as straddlers or myofibrillar hypoplasia, is a clinical disease of newborn piglets. It is arguably the most important congenital defect of newborn piglets and causes significant economic impact to the industry. PCS is the inability of newborn pigs to stand and walk properly, often with their limbs extending forwards or sideways as a result of muscular weakness (Thurley *et al.*, 1967).

In 1967, the clinical term was first reported by Thurley *et al.* (1967). Since then, reports from different countries regarding congenital splayleg were published (Dobson, 1968; Cunha, 1968; Olson and Prange, 1968; Bollwahn and Pfeiffer, 1969; Lax, 1971; Svendsen and Andereasson, 1980). Reports on the prevalence vary considerably. In the United Kingdom, Ward and Bradley (1980) estimated that about 0.4% piglets were affected by splayleg, which caused an annual loss of £300,000 to the pig industry at that time. In another two studies by Dobson (1968, 1971), the overall average prevalence rates were reported at 11% and 13% respectively. A further study in Ontario indicated that PCS was the most common congenital defect with 0.87% piglets affected (Partlow *et al.*, 1993).

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Clinical signs

PCS can be found at or a few hours after birth. At birth, 2 to 3 piglets may be affected in each litter. It invariably affects the hind limbs, but occasionally affects the forelimbs. Most of the affected piglets are unable to move around or stand, although some splayleg pigs may able to move around with difficulty. The affected limbs are abducted, splayed forward or in sideways position. Often, an affected piglet is found seated on its hindquarters (Figure 1).

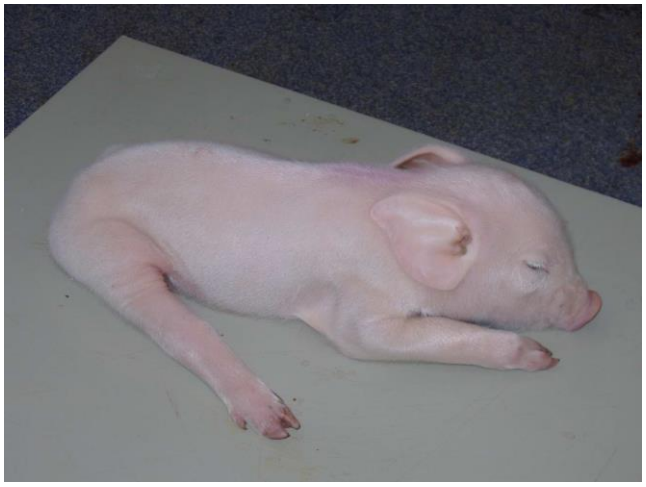

Figure 1.Porcine congenital splayleg. A 2-day-old piglet with PCS showing forward extension and abduction of hindlimbs as a consequence of muscle weakness.

An affected piglet is often separate from its healthy litter mates. Its immobility means that it cannot gain sufficient nutrients from the sow, resulting in starvation and hypothermia. Affected neonatal pigs become emaciated, weak and dirty. Abrasions and ulceration develop on the body due to long periods of lying on the floor. Splayleg pigs are more predisposed to arthritis, polyarthritis, pododermatitis and osteomyelitis of the digits due to secondary bacterial infections. The mortality rate of splayleg piglets can reach around 50%. The cause of death is either starvation or crushing by the sow. However, if supportive treatment and extra care can be provided, the affected piglet can recover after one week.

Prevalence and incidence

The condition is particularly prevalent in Landrace and Large White breeds, which are heavily muscled (Dobson, 1968; Tomko, 1993; Vogt *et al.*, 1984), although it is known to occur in virtually all commercial lines. Both male and female piglets are susceptible to PCS (Tomko, 1993; Thurley *et al.*, 1967), however, some studies indicate that male offspring are more susceptible. In one of the studies, male progeny are 1.74 times more likely than females to succumb to PCS (Vogt *et al.*, 1984). Another study showed that about twice as many males were affected as females (Van Der Heyde *et al.*, 1989). Presently, it is not clear whether PCS is related to birth weight or litter size. One study found that PCS occurs more frequently in large litters than in smaller ones (Van Der Heyde *et al.*, 1989). However, another study suggested that the occurrence of splayleg was significantly higher in small litters(Tomko, 1993).

Pathogenesis of PCS

The aetiology and pathogenesis of PCS are not known. It is considered to be a multifactorial condition. The factors that are involved are thought to include genetic and environmental factors like sow management, administration of various drugs and mycotoxins.

Treatment of pregnant sows with glucocorticoids can induce a myopathy in the newborn which mimics PCS (Jirmanova, 1983). However, there were histological differences in muscle from glucocorticoid induced and naturally occurring PCS (Ducatelle *et al.*, 1986). Dexamethasone is a synthetic glucocorticoid used primarily as an anti-inflammatory agent in various conditions, including allergic states. More recently, it was suggested that affected muscles have a reduced number of myofibrils and an increased accumulation of glycogen when compared with the muscles of normal piglets. The investigation had concentrated on the activity of glucose-6-phosphatase (G-6-Pase), a liver enzyme that breaks down glycogen reserves (Antalikova *et al.*, 1996). Another study suggested that the slippery floor in farrowing crates could predispose newborns to PCS problems (Dobson, 1971). However, this environmental factor is not likely to be a causal factor of splayleg (Van Der Heyde *et al.*, 1989). Choline is a vitamin like compound which is essential for acetylcholine synthesis. Supplementation with 2.2 to 3.0g of choline in the diet of pregnant sow until parturition was reported to reduce the incidence of PCS (Cunha, 1968). However, a subsequent study could not reproduce this finding (Dobson, 1971).

Consumption of *fusarium* F-2 toxin (zearalenone) contaminated grain by sows in late pregnancy, could lead to a higher incidence of PCS, a condition that can be experimentally reproduced by the administration of purified F-2 toxin to pregnant sows (Miller *et al.*, 1973). Increased stillbirths and neonatal mortality were also recorded. However, F-2 induced PCS piglets did not show the typical histopathological lesions described by Thurley *et al.* (1967).

Misuse of certain drugs in pregnant sows may lead to signs of PCS in newborn piglets. Administration of 3.6mg/kg/day of pyrimethamine, an anthelmintic, to pregnant Goettingen minipigs raised the incidence of PCS to 74% of the newborn, while the control group had only an incidence of 5.6% (Ohnishi *et al.*, 1989). Induction by prostaglandin before the 113th day of pregnancy could also lead to higher incidence of congenital myofibrillar hypoplasia (Bolcskei *et al.*, 1996).

Genetic factors play an important role in PCS as well. Porcine *CDKN3* gene (cycline-dependent kinase inhibitor3) which involved in cell cycling was strongly displayed in splayleg muscle (Maak *et al.*, 2003). Atrophy marker gene FBXO32 (atrogin, MAFbx) was found to be highly expressed in PCS samples (Ooi *et al.*, 2006). Recently, another four genes which are sequesosome1 (SQSTM1), structure specific recognition protein 1 (SSRP1), v-maf musculoapneurotic fibrosarcome oncogene homolog (MAF) and DNA-damage-inducible transcript 1 (DDIT4) are indicated might involve in PCS pathway as different expression levels were detected comparing genome wide gene expression of three hind leg muscles (*muscle adductores*, *musclegracilis* and *musclesartorius*) between splayleg piglets and their healthy liter mates (Maak *et al.*, 2009).

Histopathology of PCS

A variety of lesions have been described as the underlying pathological changes in congenital splayleg. The most consistent change is the presence of so-called myofibrillar hypoplasia (MFH), interpreted as an immaturity of the muscle (Ducatelle *et al.*, 1986; Thurley *et al.*, 1967). Myofibrillar hypoplasia ranges from a slight reduction of myofibrillar content to severe myofibrillar deficiency, vacuolisation, focal degeneration and necrosis. However, myofibrillar hypoplasia can also be found in clinically normal piglets. The term congenital myofibrillar hypoplasia may not therefore be the diagnostic description of PCS (Ducatelle *et al.*, 1986).

As pointed out earlier, the morphological findings of dexamethasone treatment suggest that PCS might represent a congenital form of glucocorticoid myopathy (Ducatelle *et al.*, 1986). Another study on muscle ultrastructure of PCS piglet, also showed reduced numbers of myofibrils and an increase in glycogen accumulation in comparison with muscle of normal piglets (Antalikova *et al.*, 1996). Other studies found differences between PCS piglets and piglets with experimentally-induced glucocorticoid myopathy. Naturally occurring PCS had glycogen-filled extramyofibrillar space (EMS), whereas dexamethasone splayleg had only limited glycogen in the EMS (Ducatelle *et al.*, 1986).

There were no significant qualitative differences between normal pigs and splayleg pigs aged from birth to 1 week, based on light microscopic and ultrastructural examinations (Bradley *et al.*, 1980; Ward and Bradley, 1980). The progressive clinical improvement in splayleg during the first week of life was found to be accompanied by an increase in muscle cell size, a reduction in the number of nuclei, a reduction in the severity of MFH, a reduction in the size of the extra-myofibrillar space and an increase in intracellular lipid. However similar changes were also found in normal pigs. The results indicated that light microscopy or ultrastructural morphology were not useful to diagnose splayleg due to the failure to detect any significant differences (Ward and Bradley, 1980). However, both studies took the form of visual subjective assessments. Quantitative measurements were not performed. A congenital, impaired functionality of skeletal hind limb muscles due to immaturity and/ or atrophic properties is likely to be the major patho-morphological features in PCS (Maak *et al.*, 2009).

Control and treatment of PCS

Given the uncertain aetiology and pathogenesis of PCS, it is difficult to reduce its incidence. From the husbandry viewpoint, a dry and non-slippery floor should be provided for farrowing. In addition, neonatal piglets should be protected from injury by the sow, and provided with adequate opportunities to suckle. These could reduce the incidence or the severity of PCS. Selection of breeding stock may help to control the disease. Good farm breeding records can help to identify individuals that are predisposed to the production of affected offspring. In addition, affected piglets that recover should not be used for breeding. With supportive care, adequate warmth and nutrition, affected piglets can recover from the condition. Some farmers suggested by providing sufficient of Vitamin C, the problem will subside. However, nursing is labour consuming and may not be economical as a routine in herd health management. The common treatment method used is tying together the affected limbs, with a loose "figure of 8" just above the hock joint. It appears to help the piglet to recover and gain the ability to move around. Adhesive tape can be used to tie affected legs but care must be exercised to avoid occlusion of blood flow.

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