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Original Article

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Abstract

Testicular atrophy is a major cause of culling in boars. Pathogenesis of this change is poorly understood. Therefore, we aimed to investigate pathological changes of the genital tracts of culled boars with testicular atrophy. Twenty-eight genital organs of culled boars collected from slaughter houses in Thailand were pathologically examined. According to testicular weight, the organs were allocated into 4 groups: severe atrophy (n = 12), moderate atrophy (n = 4), mild atrophy (n = 4), and normal control (n = 8). In the severe atrophic group, the mean weight of atrophic testes (175.1 ± 51 g) was lower than those of the normal control boars (376.1 ± 72 g) ($p < 0.001$). By gross examination, 5 boars (42%) in the severe atrophic group had bilateral atrophic testes, but the remaining animals (58%) were unilaterally affected. Examination of cut surface revealed extensive fibrosis in those severe atrophic testes with less degree in the other groups. Microscopically, severe testicular fibrosis, severe degeneration and collapsed seminiferous tubules were frequently observed in those severe atrophic testes with frequently lymphocytic infiltration. Fibrosis and degeneration were minimal in the remaining groups, and were absent in the control group. Examination of epididymides revealed sperm granulomas in the epididymal heads of severe atrophic testes (n = 2) and in mild atrophic testis (n = 1). No significant relationship between age of the boars and the occurrence of testicular atrophy was found. In conclusion, severe fibrosis and degeneration were the most striking lesions in testicular atrophy. Allthough the causes of these changes remain unclear, our findings revealed useful information for clinical examinations of genital organs in boars.

Keywords: boars, testicular atrophy, testicular degeneration, testicular fibrosis

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บทคัดย่อ

การศึกษาทางพยาธิวิทยาของสภาวะอัณฑะฝ่อลีบในพ่อสุกรที่ถูกคัดทิ้ง

คมกฤช เทียนคำ [1] เผด็จ ธรรมรักษ์ [2] สว่าง เกษแดงสกลวุฒิ [1] ธิติ อันตรเสน [1]*
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การฝ่อลีบของอัณฑะในพ่อสุกรเป็นสาเหตุที่สำคัญของการคัดทิ้งพ่อสุกร พยาธิกำเนิดของความผิดปกติดังกล่าวยังไม่เป็นที่เข้าใจอย่างถ่องแท้ การศึกษาในครั้งนี้จึงมีวัตถุประสงค์เพื่อศึกษาการเปลี่ยนแปลงทางพยาธิวิทยาของระบบสืบพันธุ์พ่อสุกรที่ถูกคัดทิ้งจากฟาร์ม โดยทำการเก็บตัวอย่างอวัยวะสืบพันธุ์ของพ่อสุกรจำนวน 28 ตัว จากโรงฆ่าสัตว์ ชั่งน้ำหนักอัณฑะ และอิพิดิไดมิส จากข้อมูลของน้ำหนักอัณฑะจึงได้แบ่งพ่อสุกรออกเป็น 4 กลุ่ม ตามความรุนแรงของการฝ่อลีบของอัณฑะ ได้แก่ กลุ่มที่มีการฝ่อของอัณฑะรุนแรงมาก (n = 12) กลุ่มที่มีการฝ่อของอัณฑะปานกลาง (n = 4) กลุ่มที่มีการฝ่อของอัณฑะเล็กน้อย (n = 4) และกลุ่มควบคุมที่มีอัณฑะเป็นปกติ (n = 8) ในกลุ่มที่มีการฝ่อของอัณฑะอย่างรุนแรง อัณฑะที่ฝ่อมีน้ำหนักเฉลี่ย (175.1 ± 51 กรัม) น้อยกว่าค่าเฉลี่ยของกลุ่มควบคุม (376.1 ± 72 กรัม) อย่างมีนัยสำคัญทางสถิติ ($p < 0.001$) และมีพ่อสุกรจำนวน 5 ตัว (42%) ที่อัณฑะฝ่ออย่างรุนแรงทั้งสองข้าง ส่วนสุกรที่เหลือทั้งหมด (58%) มีอัณฑะฝ่ออย่างรุนแรงเพียงข้างเดียว การตรวจทางมหพยาธิวิทยาพบสภาวะไฟโบรสิสรุนแรงในกลุ่มสุกรที่มีการฝ่อของอัณฑะอย่างรุนแรง จุลพยาธิวิทยาของอัณฑะที่ฝ่อลีบรุนแรงพบสภาวะไฟโบรสิสรุนแรง ที่บริเวณเนื้อเยื่อระหว่างท่อผลิตอสุจิ และพบการเสื่อมและยุบตัวลงของท่อผลิตอสุจิ ร่วมกับการแทรกของเซลล์ลิมโฟไซต์ สภาวะไฟโบรสิสและการเสื่อมของท่อผลิตอสุจิมีความรุนแรงน้อยในกลุ่มอื่นๆ และไม่พบรอยโรคดังกล่าวนี้เลยในกลุ่มควบคุม นอกจากนี้ยังพบสเปิร์มแกรนูโลมาที่ส่วนต้นของอิพิดิไดมิส ในพ่อสุกรที่มีอัณฑะฝ่อรุนแรง 2 ตัว และในกลุ่มฝ่อเล็กน้อย 1 ตัว การศึกษาในครั้งนี้ไม่พบความสัมพันธ์ระหว่างอายุและการฝ่อของอัณฑะอย่างมีนัยสำคัญทางสถิติ โดยสรุปสภาวะไฟโบรซิสและการเสื่อมอย่างรุนแรงของอัณฑะเป็นรอยโรคที่พบได้มากที่สุดในอัณฑะที่ฝ่อลีบ แม้ว่าสาเหตุของรอยโรคนี้ยังไม่สามารถระบุได้ชัดเจน แต่การศึกษาในครั้งนี้เป็นประโยชน์อย่างยิ่งในการตรวจระบบสืบพันธุ์ของพ่อสุกรทางคลินิก

คำสำคัญ: พ่อสุกร การฝ่อลีบของอัณฑะ การเสื่อมของอัณฑะ การเกิดไฟโบรซิสของอัณฑะ

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Introduction

Atrophy or diminishing in size of testes is related to male infertility both in domestic (Montagna et al., 2001) and wildlife animals (Tiller et al., 1997). Testicular atrophy can also be induced by experimentally transgenic modification in mice (Antonangeli et al., 2009) as well as immune castration in boars (Hilbe et al., 2006) or administration of toxic substances (Saito et al., 2005). In human patients with acquired immunodeficiency syndrome (AIDS), testicular atrophy occurs with seminiferous tubule atrophy and interstitial fibrosis, but these changes could be the consequence of human immunodeficiency virus infection in genital tract or an impact of wasting condition (De Paepe and Waxman, 1989; Mhawech et al., 2001).

Testicular atrophy is a result of severe degeneration of germ cells epithelium which probably caused by genetic disorder or aging process. Age-related testicular degeneration and poor semen quality were evident in bulls (Kumi-Diaka et al., 1981) and stallions (Fukuda et al., 2001; Turner and Zong, 2012). In stray dog population, testicular degeneration with small size of testes is more common in old dogs (Ortega-Pacheco et al., 2006). In human patient, age-related changes of male reproductive performance are controversy; however, several studies revealed the evidence of age-related changes in semen quality (Zhu et al., 2011; Stone et al., 2013).

Regardless of testicular degeneration, small testicular size may be due to hypoplastic condition associated with chromosomal defect known as Klinefelter syndrome XXY as described in many species including boars (Kopp et al., 2008), bulls (Molteni et al., 1999) and human (Lanfranco et al., 2004). This syndrome consists of hormonal deficiency, testicular hypoplasia and completely loss of spermatogenesis.

In boars, pathological changes of testis result in poor semen quality and infertility leading to economic loss due to early culling. Klinefelter syndrome and segmental aplasia of Wolffian ducts were shown to be the causes of azoospermia in boars with decreased testicular weight (Kopp et al., 2008). In contrast to these rare abnormalities, testicular degeneration was more commonly seen in slaughtered boars (Teankum et al., 2008). Recently, an investigation on inbred Duroc boars with asymmetric size of testes showed that chronic orchitis, severe degeneration and fibrosis were the causes of atrophic changes of testes (Noguchi et al., 2013). This study also suggested that noninfectious orchitis might be involved in atrophic processes. A direct effect of viral infection can induce severe inflammation and atrophic changes of testicles as shown in boars inoculated with rubulavirus (Ramirez-Mendoza et al., 1997). Other viruses such as pseudorabies virus can cause periorchitis with increased scrotal fluid, but not much alteration was observed in testes (Miry and Pensaert, 1989)

It seems that testicular atrophy could be induced by various causes, and its pathogenesis is poorly understood. The primary causes are difficult to definitely identify. To gain a better understanding of this atrophic change, we aimed to investigate pathological features of atrophic testes of culled boars collected from Thai slaughter houses in western region.

Materials and Methods

Boars and sample collection: Twenty-eight male genital organs of boars were collected from slaughter houses in the western region of Thailand. The organs were submitted to the Department of Veterinary Pathology, Chulalongkorn University, Nakornpathom province and Bangkok, Thailand. The reproductive organs were macroscopically examined. The testes and epididymides were separated and weighed. Testicular diameter, length and circumferences were measured using a metric-scale ribbon tape. The testes were sliced sagittally, and cut surfaces were examined for pathological changes.

Three slices of specimen were taken from proximal, middle, and distal part of each testis, and fixed in 10% formalin. Then other 3 pieces of testes were collected with the same manner and fixed in Bouin's solution for 18 hours before further processing. The degrees of testicular degeneration and fibrosis were assessed by gross and histopathology, (+1 : mild, +2 : moderate, and +3 : severe). Other parts of the genital organs including three parts of epididymides (head, body and tail), prostate glands, seminal vesicles, bulbourethral glands, urethra, glans penis, and prepuce were collected and fixed in 10% formalin. All samples were routinely processed for histopathology (haematoxylin and eosin staining).

Based on gross and histopathological findings, 20 out of 28 cases were diagnosed as testicular atrophy, and were included in this study. According to the severity of testicular atrophy assessed by criterion shown in Table 1, the animals were allocated into 3 groups: severe atrophy (n = 12), moderate atrophy (n = 4), and mild atrophy (n = 4). The boars without testicular lesions were considered as normal control boars (n = 8).

Statistical analysis: Descriptive statistics and frequency analysis were conducted for all reproductive parameters. Relationship between age and testicular atrophy was analysed by Wilcoxon rank sum test. Difference of testicular weight between each was analysed by Pair *t* test. Difference of testicular weight between the control group and atrophic groups was analysed by using General Linear Model (GLM) procedure of SAS software. A probability (*p*) less than 0.05 was considered to be significantly difference. The results are expressed as means (± SEM).

Results

Clinical observations: All samples were collected from slaughter houses in Nakornpathom, a province in the western part of Thailand. The boars with testicular atrophy were of different breeds. They were culled due to various reasons, including poor semen quality, lameness, small size of testes and sickness. Average age of the boars with severe testicular atrophy (29.8±11 months) was higher than the moderate and mild atrophic groups; however, statistic correlation between age and testicular atrophy could not be established due to missing data of age.

In boars with severe testicular atrophy (n = 12), five boars (42%) were bilaterally affected (Fig 1C, 1D); the remaining cases (58%) were unilaterally affected (Fig 1A, 1B). The boars with severe testicular atrophy belonged to Duroc (n = 5), Landrace (n = 3), cross-bred Duroc-Pietrain (n = 2), and Large White breed (n = 1). In the moderate atrophic group, the

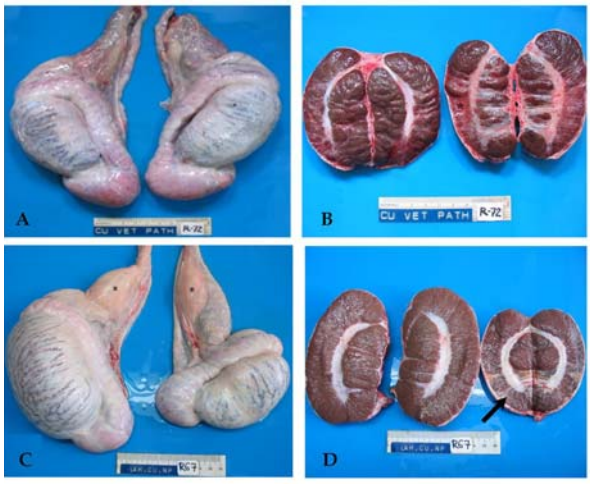

Figure 1 Severe testicular atrophy: boar 9 with severe bilateral atrophy (A); White strips on the cut surface resembling to fibrosis, and the compressed grayish white areas (*) indicating atrophy of seminiferous tubules (B); boar 7 with unilateral atrophy of the right testis with marked enlargement of spermatic cord lymph nodes (*) (C); white streaks on cut surface of testes resembling fibrosis (arrow)

boars belonged to Duroc (n = 3) and Duroc-Pietrain breed (n = 1), and were unilaterally affected. The boars in mild atrophic group belonged to Duroc breed (n = 3) and crossbred Duroc-Pietrain (n = 1), and they were also unilaterally affected. The control boars consisted of Duroc (n = 4), Landrace (n = 2) and Large White (n = 2) breeds, the animals were culled due to leg problem (n = 4) and poor semen quality (n = 4).

Size and weight of testicles and epididymides: Mean weight and size of testes and epididymides of boars are shown in Table 2. There was no significant difference in testicular weight between the left side and the right side in the control group. In boars with severe testicular atrophy, mean weight of atrophic testes was significantly lower than those of the control group ($p < 0.001$), but there was no significant difference between the mild atrophic group and the control group. Testicular size including length, diameter and circumference of severe atrophic testes were lower than those of the control group. In all atrophic groups, mean weights of epididymides of the atrophic testes were lower than those of the contralateral sides, but not statistically significant.

Macroscopic findings: In normal control boars, cut surface of the testes was bulgy, greyish and rather firm in consistency without evidence of pathological changes. The genital lesions of boars in the severe atrophic group are summarized in Table 3. The important findings of those testes were severe fibrosis and degeneration with flabby and soft consistency in some cases. Testicular fibrosis was severe in this group appearing as multifocal compressed white areas or diffused white streak on cut surface (Fig 1B, 1D). The fibrotic lesion was less in the other groups. Severe fibrosis was usually accompanied with testicular degeneration, and the lesion was present in both atrophic testes and the contralateral side with normal weight.

Testicular degeneration appeared as compressed areas of tan colour usually corresponding to severe degeneration of seminiferous tubules confirmed by histology. In severe atrophic group, the testicles were small and soft in consistency, and it was difficult to distinguish between normal and degenerated areas.

A single solitary nodule of Sertoli cell tumour was observed in one boar with severe bilateral atrophic testis (boar 1). Intratesticular haemangiomas were detected in a boar with moderate atrophic testis. Pathological detail of these tumours will be further investigated.

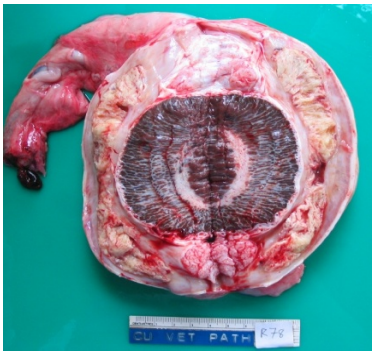

Figure 2 Cut surface of severe proliferative periorchitis, tunica vaginalis was covered by thick fibrous tissue and fibrino-necrotic mass. The testis was severe atrophy with severe fibrosis, left testis of boar 10.

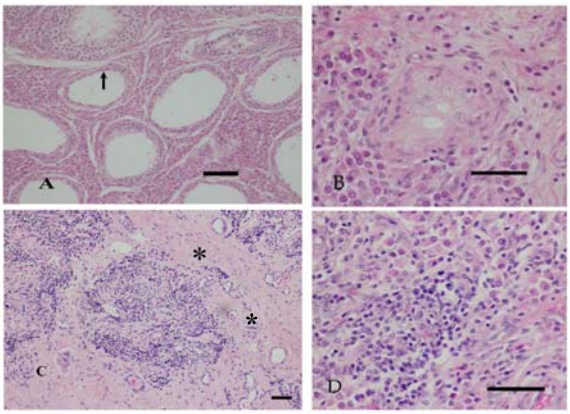

Figure 3 Histopathology of severe atrophic testes, boar 3: Degenerated seminiferous tubules with complete loss of tubular epithelium or lined by one layer of Sertoli cells (arrow), boar 3, bar = 100 μm (A); collapsed seminiferous tubules with lymphocytic infiltration, boar 3, bar = 50 μm (B); severe fibrosis of interstitial area (*) with lymphocytic infiltration, right testis of boar 10, bar = 50 μm (C); lymphocytic infiltration in interstitium, boar 10, bar = 50 μm (D), H&E stain.

Table 1 Average age of boars with diagnostic criterion of testicular atrophy

Groups	Severe atrophy (n = 12)	Moderate atrophy (n = 4)	Mild atrophy (n = 4)	Control boars (n = 8)
Average age (months)	29.8 ± 11*	19 ± 7	24 ± 10	34.6 ± 7
Gross				
Testicular size	marked small	small	slightly smaller than contralateral sides	normal size / no marked differences in weight of both testes
White areas on cut surface	prominent multifocal	a few areas	a small white streak	undetectable
Histopathology	multifocal extensive areas of seminiferous tubule atrophy	multifocal areas of seminiferous tubule atrophy	focal area of seminiferous tubule atrophy	no remarkable lesion or focal mild degeneration

*SD

Table 2 Summary of size and weight of testes and epididymides (n = 28)

Groups	Severe atrophy (n = 12)		Moderate atrophy (n = 4)		Mild atrophy (n = 4)		Control boar (n = 8)	
	Atrophic testes (n = 16)	Contralateral side (n = 8)	Atrophic testes (n = 4)	Contralateral side (n = 4)	Atrophic testes (n = 4)	Contralateral side (n = 4)	Left side (n = 8)	Right side (n = 8)
Testes								
Weight (gr)	175.1 ± 51[a]	391 ± 80[b]	216 ± 37	421 ± 124	311 ± 90	363 ± 98	382 ± 78[b]	370 ± 69.4[b]
Diameter (cm)	6.3 ± 0.9	8.5 ± 0.7	6.9 ± 0.5	8 ± 0.8	7.6 ± 1	8.1 ± 0.6	8.8 ± 1.3	8.9 ± 1.6
Length (cm)	9.7 ± 1.3	13.9 ± 1.7	10.3 ± 2.4	13.3 ± 1.3	13 ± 0.8	14.3 ± 0.5	13.8 ± 1.9	13.5 ± 2.1
Circumference (cm)	16.1 ± 2.3	22.9 ± 1.9	18.3 ± 2.3	22 ± 2.7	19.5 ± 0.6	20.8 ± 1	22.2 ± 1.3	22.3 ± 1.2
Epididymides								
Weight (gr)	151.3 ± 26.8	226.7 ± 72.8	174.5 ± 80.6	251 ± 144.7	190.3 ± 31.2	226.3 ± 57.9	164.4 ± 27.2	161.5 ± 39.1

[a, b] Different superscripts indicate a significant difference ($p < 0.001$), values are expressed as means ± SEM

Table 3 Summary of pathological findings of boars with severe atrophic testes (n = 12)

No.	Age (m)	Br	Body weight (kg)	Testicular weight (gr)		Testicular lesions					
						Atrophic testis			Contralateral side		
				Left	Right	Fibrosis	Degen	Non-sup orchitis	Fibrosis	Degen	Non-sup orchitis
1	na	na	na	167*	198*	+3	+3	+2	+3	+3	+2
2	30	D	270	176*	263	+1	+3	+2	+1	+3	+2/calcified
3	36	D	200	170*	155*	+3	+3	+1	+3	+3	+1
4	na	D	320	130*	185*	+3	+2	+1	+3	+2	+1
5	42	LR	340	510	235*	+3	+2	-	+3	+1	-
6	na	LR	370	390	65*	+3	+3	+3	+3	+3	+3
7	24	D	340	395	150*	+2	+2	-	+1	+1	-
8	30	LR	290	245*	420	+1	+3	-	+1	+3	-
9	36	D	294	120*	135*	+1	+2	+1	+1	+2	+1
10	36	LW	275	670*†	245*	+3	+3	+2	+3	+2	+2
11	17	Pt/D	180	665	250*	+3	+2	+2	+2	+1	-
12	8	Pt/D	290	175*	370	+3	+3	+3	+1	-	+2

* : atrophic testes, † : weight include periorchitis mass, Br : breed, D : Duroc, LR : Landrace, LW : Large white, Pt/D : Pietran/Duroc 50%, (-) : no lesion, (+1) : mild, (+2) : moderate, (+3) : severe

Histopathology: In the severe atrophic testes, seminiferous tubule degeneration was multifocal, characterised by loss of germinal epithelium and the tubules were lined by only one cell layer resembling Sertoli cells (Fig 3A) with occasional, cytoplasmic vacuoles. Spermatic giant cells were also observed in some tubules. In the compressed areas with greyish-white colour, seminiferous tubules collapsed (Fig 3B) with increased fibrous tissue infiltration in inter-tubular areas (Fig 3C). Various degrees of nonsuppurative orchitis characterised by lymphocytic infiltration in interstitial areas as well as in peritubular areas were observed mostly in atrophic areas of the severe atrophic testicles (Fig 3D). This lesion was mild in the moderate and mild atrophic groups, and it was not seen in the control group. However, focal mild degeneration was detected in one control boar.

In boar No. 10 of severe atrophic group, severe proliferative fibrinous periorchitis was seen in the left testis with thickening tunica by fibrous tissue (Fig 2) and covered by fibrino-haemorrhagic mass and neutrophilic debris. *Staphylococcus* spp. was cultured from this lesion. Severe atrophic change appeared also in the contralateral side. Marked infiltration of lymphocytes, tubular atrophy and fibrosis was observed in both testes of this case. In addition, moderate periorchitis was seen in another boar with moderate atrophic testis.

Pathological changes of epididymis and other accessory organs: In all groups of testicular atrophy, epididermides showed various lesions including epididymal oedema mainly at the head of epididymis. The lesion was seen in the severe atrophic (n = 5), moderate atrophic (n = 2) and mild atrophic (n = 2) groups. Epididymal cyst containing clear fluid with about 1 cm in diameter of ovoid shape located between the head of the epididymis and testis was seen in 1 boar.

Sperm granulomas in epididymal heads were observed in 2 cases of the severe atrophic group and in 1 case of the mild atrophic group. Histopathology revealed granulomatous inflammation

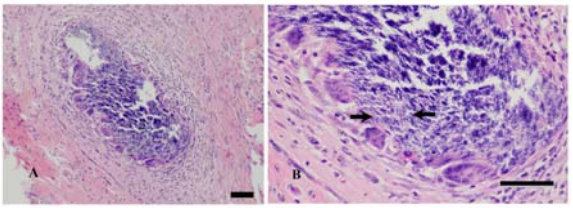

Figure 4 Spermatic granulomatous inflammation in the epididymal head of boar 2; accumulation of macrophages, multinucleated giant cells and lymphocytes surrounding necrotic mass with remnant of spermatozoa (A) (bar = 50 μm); higher

magnification of Fig 4A with multinucleated giant cells and the remnant of spermatozoa (arrows) (B) (bar = 50 μm), H&E stain.

characterised by an accumulation of macrophages, epithelioid cells, multinucleated giant cells and lymphocytes at the edge of the necrotic mass with remnant of spermatozoa (Fig 4). There were no lesions in the accessory sex organs of all boars.

Discussion

Testicular atrophy results in severe alteration of reproductive performance in boars leading to economic loss due to early culling of young boars. The current study revealed that the atrophic change of testes was attributed to extensive testicular fibrosis and degeneration concurrently with frequent chronic inflammatory reactions.

In this study, collapse of degenerated seminiferous tubules with interstitial fibrosis was prominent in the atrophic testes. Fibrosis was extensive in the severe atrophic testes, and it was also prominent in the contralateral testicles with normal weight, but the lesion was not detected in the control group. This lesion of interstitial fibrosis was in agreement with another study on inbred Duroc boars with small size of testicles (Noguchi et al., 2013). These changes in human testes were considered to be the sequence of peritubular cells transformation into myofibroblast producing extracellular matrix in interstitial areas (Apa et al., 2002).

Similar to the previous report on fibrotic testes in boars (Noguchi et al., 2013), various degrees of lymphocytic infiltration was frequently observed in interstitial tissue indicating chronic inflammatory responses. As in some cases of severe unilateral testicular atrophy, chronic interstitial orchitis appeared in both testes. In addition, severe proliferative periorchitis was observed in one boar with severe bilateral atrophic testes. These findings was similar to the case report of periorchitis in a 25-year-old man (White et al., 2006), suggesting that inflammatory processes could be the causes of atrophic testes. It is essential to further investigate whether this lesion involves infectious agents or not.

In bulls with intensive rearing conditions, testicular fibrosis was frequently observed in all ages, and association with bovine respiratory syncytial virus infection in some herd was mentioned (Barth et al., 2008). Atrophy of testes was observed in a mule deer population with velvet-covered antler; however, an effect of age groups could not be concluded (Tiller et al., 1997). Seminiferous tubules atrophy with fibrosis is common in old horses (Fukuda et al., 2001), but age-related changes are not confirmed in human or in boars. In this study, most of the boars with severe testicular atrophy were older than the moderate or mild atrophic groups. However, statistic correlation between age and testicular atrophy could not be established.

In the severe atrophic testes, degeneration of seminiferous tubules was commonly seen, and most tubules collapsed, concomitantly with interstitial fibrosis. These findings are similar to previous study on azoospermic boars with arrested spermatogenesis inducing testicular atrophy (Kopp et al., 2008). The etiology of these degenerative changes may include aging, high environmental temperature (Kanter et al., 2013), genetic or chromosome abnormality (Bolor et al., 2006). In general, mechanisms of germ-cell degeneration depend on apoptotic process (Tao et al., 2006). Disruption of spermatogenesis is also caused by hormonal disturbances, e.g. gonadotrophins, estrogens, androgens. Decrease in testosterone activity (Sofikitis et al., 2008) or over exposure to estrogen (Pinto et al., 2008) result in reduction in sperm production and subsequent degeneration of seminiferous tubules. Moreover, degeneration process can be induced by various toxic substances such as cancer therapeutic drugs and zearalenone (Kim et al., 2003 and Boekelheide, 2005). In addition, some viral infection can result in apoptosis of germ cell epithelium and multinucleated cell formation in seminiferous tubules leading to degeneration of testis as shown in experimental PRRSV-infected boars (Sur et al., 1997). Although PRRSV infection was not investigated in this present study, but the occurrence of PRRS was intensive in pig population where samples were taken (Thanawongnuwech et al., 2004).

This study showed that bilateral atrophy of testicles was seen in the severe atrophic group, the remaining animals were unilaterally affected. These data indicated that minimal or moderate changes in testicular weight could imply minimal degree of testicular atrophy. Thus the prevalence of testicular atrophy may be underestimated, as difference in testicular size between the left side and the right side in boars is often seen in the field. Interestingly, the Sertoli cell tumour and testicular haemangioma were found in the atrophic testes. It is not known whether these tumours were related with atrophic changes or just coincidental findings.

Pathological changes of epididymides were not obvious among the atrophic groups. The mean weight of epididymides of the atrophic testes was lower than the contralateral sides, probably because of lower sperm production in those atrophic testicles. Microscopically, mild perivascular lymphocytic infiltration often seen in the interstitium of epididymis may imply local immune responses to some antigen. Oedema of epididymal head was an interesting lesion detected in some boars. Although the cause of this particular change is still obscured, association with porcine circovirus infection was documented (Opriessnig et al., 2006). In addition, the epididymal cyst and sperm granulomas observed both in the severe and mild atrophic groups may be only a coincidence, because these lesions were common in slaughtered boars (Teankum et al., 2008).

In conclusion, various degrees of degeneration and fibrosis were prominently observed in the atrophic testes of boars concurrently with other minor changes. Although the primary causes of such lesions need to be elucidated, the pathological changes found in this study provided a better understanding in the detail of testicular atrophy in boars. These findings are also useful for swine

practitioners in clinical examination of reproductive organs of boars.

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