Deep pectoral myopathy: prevalence in 7 weeks old broiler chickens in Bulgaria

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SUMMARY

The purpose of the study was to investigate the prevalence of DPM (deep pectoral myopathy) in young broiler chickens according to their live weight during slaughtering inspection. Chickens (Ross 308 and Cobb 500) reared under intensive conditions and slaughtered on 7 weeks of age were allotted in 2 flocks of 20,000 birds each, named flock A and flock B, according to the ratio live weight / carcass weight (2.3/1.7 and 2.6/1.9, respectively). During deboning of the breast muscles, samples showing gross lesions of the m. supracoracoideus were collected and a conventional histology was performed. The overall DPM prevalence (for the 2 flocks) was 0.51% and the specific prevalence by flock was significantly higher in the flock B (P < 0.01). The frequency of early DPM lesions characterized by an acute inflammatory reaction (oedema, haemorrhages and infiltrate of macrophages and heterophils) coupled to a severe muscle discoid necrosis was significantly higher in the flock A (P < 0.05) whereas the proportion of older lesions in which the affected muscle showing a typical green colour before being replaced by fibrous or adipose tissue was dramatically increased in the heaviest chickens (flock B) (P < 0.001). These results confirm the DPM occurrence in meat-type growing birds even in young broilers (7 weeks old).

Keywords: Deep pectoral myopathy, broiler chickens, Bulgaria, histology, acute inflammatory reaction, muscle necrosis.

Introduction

Deep pectoral myopathy (DPM), also known as Oregon muscle disease or green muscle disease, was first described in 1968 as “degenerative myopathy” in turkeys [6]. Subsequently it was studied at the Oregon State University by Harper [10] and his collaborators [17]. The disease involves the wing elevating muscle known as the deep pectoral muscle or M. supracoracoideus hence; it is referred to as degenerative myopathy of the supracoracoideus (DMS).

A probable analogy can be drawn between this disease of poultry and the so-called “march gangrene” (anterior tibial syndrome) in humans [16]. The “march gangrene”, so-named because of its occurrence among the military personnel, is an ischemic necrosis of the muscle in the myofascial compartment on the anterior of human tibia following increased intra-compartmental pressure, clinically characterized by swelling, redness and pain, paralysis of the extensor digitorum brevis, and loss of sensation in the first interdigital cleft.

This disease is not preceded by vascular disease or trauma, but it is usually associated with a recent period of locomotor activity in otherwise healthy individuals [19]. The muscle is firstly swollen, pallid and oedematous, and in untreated cases this condition progresses to necrosis which has been described as appearing greyish brown and wood-hard [19]. The general reaction of the muscle is that occurring in ischemia and changes similar to those in the broiler muscle supracoracoideus may be seen [19].

Initial attempts to discover the cause of DPM were unsuccessful. Cultured tissue from affected turkeys failed to show evidence of bacterial or viral infection [9]. In addition, dietary supplementation with vitamin E, methionine and selenium also failed to reduce the incidence of DPM in turkeys [9, 19]. GRUNDER et al. [7] could not find any difference in DPM incidence between groups of turkeys fed with corn or wheat based rations. The cause of DPM appears to be not related to nutrition disequilibrium or to a pathogen.

RÉSUMÉ

Myopathie du pectoral profond : prévalence chez les poulets de chair âgés de 7 semaines en Bulgarie

Le but de cette étude a été de déterminer la prévalence de la myopathie du pectoral profond (MPP) chez de jeunes poulets de chair en fonction de leur poids vif durant l’inspection des carcasses à l’abattoir. Des poulets (Ross 308 et Cobb 500) élevés en mode intensif et abattus à 7 semaines ont été répartis en 2 groupes de 20000 oiseaux chacun, groupe A et groupe B, en fonction du rapport poids vif / poids de la carcasse (respectivement 2,3/1,7 et 2,6/1,9). Durant le désossage des filets, les échantillons présentant des lésions macroscopiques du m. supracoracoideus ont été collectés et analysés par histologie conventionnelle. La prévalence globale de la MPP (pour les 2 groupes) a été de 0,51 % et la prévalence spécifique par groupe a été significativement plus importante dans le groupe B (P < 0,01). La fréquence des lésions précoces caractérisées par une réaction inflammatoire aiguë (œdème, hémorragies, infiltrat par des macrophages et des hétérophiles) couple à une nécrose discoïde sévère du muscle a été significativement plus élevée dans le groupe A (P < 0,05) alors que les lésions plus anciennes (dans lesquelles le muscle atteint présente une coloration verte typique avant d’être remplacé par du tissu fibreux ou adipeux) se sont avérées beaucoup plus fréquentes dans le groupe B, celui des poulets les plus lourds. Ces résultats confirment l’existence de MPP chez les oiseaux sélectionnées pour la production de viande, y compris chez des poulets jeunes (de 7 semaines).

Mots clés : Myopathie du pectoral profond, poulet de chair, Bulgarie, histologie, réaction inflammatoire aiguë, nécrose musculaire.
It is generally recognized that DPM is an ischemic necrosis that develops in the deep pectoral muscle (supracoracoideus or pectoralis minor muscle) mainly because this muscle is surrounded by inelastic fascia and the sternum, which do not allow the muscle mass to shell in response to the physiological changes occurring when muscles exercised, as in wing flapping [12]. The lesion does not impair the general health in birds and it is generally found during cut-up and deboning. No public health significant problem is associated to DPM, but it is aesthetically undesirable. The fillet should be removed, whereas the rest of the carcass is still fit for human consumption [12]. However, the required trimming operations determine the downgrading of the products and produce an economic loss for the industry, especially because it affects the more valuable part of the carcass. Although this disease was first recognized in adult meat-type turkey and chicken breeders, it is becoming more and more common in meat-type growing birds [3, 8, 13]. According to SILLER [17], DPM occurs exclusively in birds that have been specially selected for breast muscle development.

The purpose of the study was to present the results obtained from slaughtering inspection and histopathology in spontaneous cases of deep pectoral myopathy (DPM) reported for the first time in broiler chickens in Bulgaria, according to the age and live weight of the birds.

Materials and Methods

ANIMALS

The investigation was carried out using the material from a large Bulgarian poultry slaughterhouse in Stara Zagora. Chickens were originated from a farm in the same province where they were reared under intensive conditions and were slaughtered on 7 weeks of age. The samples showing gross lesions were collected during the deboning of breast muscles of broiler chickens. The statistical analysis and comparison of the incidence of the lesions were made during inspection of carcasses originating from two different flocks (10 000 carcasses were inspected for each flock). The flocks consisted of 20 000 birds each, belonging to both sexes and to 2 commercial strains: Ross 308 and Cobb 500, mixed disproportionally. The mean live weight to mean carcass weight ratios were 2.3/1.7 and 2.6/1.9 in the flock A and the flock B, respectively.

HISTOLOGICAL ANALYSIS

After macroscopic examination, 10 samples with lesions from the m. supracoracoideus were taken from each flock for histological investigations. The samples were fixed in 10% neutral formalin, processed routinely and embedded in paraffin. The obtained sections approximately 5µm were stained with haematoxylin-eosin.

STATISTICAL ANALYSIS

The data were statistically processed by a chi 2 test for comparing the 2 flocks. Differences were considered as significant when $P < 0.05$.

Results

Table 1 shows the incidence and distribution of the DPM characteristic lesions for both flocks, determined on the processing line after the breast-deboning area. The overall incidence of DPM was 0.51%. The total lesions incidence was significantly higher in the flock B with the highest live/carcass weight ratio than in the flock A ($P < 0.01$). The prevalence of early lesions was twice higher in flock “A” (the birds with lower live/carcass weight ratio) ($P < 0.05$) whereas later stage lesions occurred much more frequently ($≈10$ times) in heavier birds (flock “B”) ($P < 0.001$).

Macroscopically, early lesions showed oedema, hyperaemia and haemorrhages of the supracoracoid muscle. One or both supracoracoid muscles were affected at a various extent. The changes vary by intensity from separate, focal to diffuse damage affecting the whole muscle. Commonly the lesion was sharply confined from the unaffected part of the muscle (figure 1). The fascia overlying and separating it from the superficial pectoral muscle was thickened, opaque and sometimes covered by a gelatinous matter. In older cases, the acute oedema has disappeared. The affected muscles were intensive green in contrast to the unaffected tissue and exhibited single or multiple haemorrhages (figure 2). The green areas were generally confined to the surface but gradually they penetrated more deeply into the necrotic tissue leading to the occurrence of irregular green bands of varying thickness surrounding the pale necrotic muscle in cross section. In the oldest lesions, the colour intensity of the muscle was alleviated,

<table>
<thead>
<tr>
<th>Flocks</th>
<th>Early lesions</th>
<th>Older lesions</th>
<th>Total lesions</th>
</tr>
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<tbody>
<tr>
<td>Flock A (2.3/1.7)</td>
<td>0.32%&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.05%&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.37%&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Flock B (2.6/1.9)</td>
<td>0.16%&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.48%&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.64%&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Total</td>
<td>0.24%</td>
<td>0.27%</td>
<td>0.51%</td>
</tr>
</tbody>
</table>

<sup>a</sup>Flocks were characterized by the ratio mean live weight / mean carcass weight; <br> <sup>b</sup>Different superscripts <sub>a,b</sub> in the same column indicate significant differences between flocks ($P < 0.05$) with a chi 2 test.

Table I: Incidence of DPM (Deep pectoral myopathy) in the 2 flocks of broiler chickens reared under intensive conditions in the farm in Stara Zagora province.
becoming pale yellow-green, and haemorrhages were reduced (figure 3). The necrotic muscle bundles appeared dry and brittle and usually the entire lesion was enclosed by a thick connective tissue capsule sequestrating it from the remaining muscle tissue. Sometimes the affected muscle appeared with reduced volume, fibrous and hard.

The histological investigations confirmed the acute inflammation in early DPM lesions. Degenerative necrotic changes or sometimes Zenker’s degeneration, increased acidophilia and oedema were observed. An infiltrate of inflammatory cells (mainly macrophages and some heterophil leukocytes) was found in the affected area (figure 4). The necrosis of the muscle bundle was arranged as a discoid form and the so-called discoid necrosis resulted in the separation of the individual sarcomeres (figure 5). In the more chronic lesions, the muscle fibres were almost entirely replaced by fibrous and/or adipose tissue (figure 6).

**Discussion**

In the present study, the DPM prevalence is significantly more elevated in the heavier chickens. This finding is in agreement with CRESPO and SHIVARPRASAD [5], suggesting that DPM is mostly prevalent in heavy birds. Moreover, as in the present study, the incidence of old lesions, indicating the chronicity of the muscle degeneration, was markedly higher in the biggest chickens, the DPM can...
be associated with the tendency to produce “heavy” broilers (> 3kg live weight) for breast muscle [1]. Some authors [1-4, 8] have evaluated the incidence of the muscular disease in heavy male roaster chickens reared in commercial conditions. However, it is difficult to compare these results with the current study where chickens were both males and females. Nevertheless, DPM cases in the present study were observed in young birds (7 weeks old) and this age was previously reported by RICHARDSON et al. [13] as the lowest for evidencing such muscular lesions. The mean live/carcass weight ratios of the chickens with DPM lesions are among the lowest ever reported [13]. These facts support the concept that the DPM prevalence is higher in meat-type growing birds [3, 8, 13] and according to SILLER [17], DPM occurs exclusively in birds that have been specially selected for breast muscle development, as the 2 commercial strains (Ross 308 and Cobb 500) used in this assay. Evidence that this condition only appears because of intensive selection is supported by the absence of DPM in wild turkeys, even when these birds are experimentally forced to grow [2, 4, 17].

The DPM lesions firstly exhibited an acute inflammatory aspect with oedema, hyperaemia and haemorrhages of the supracoracoid muscle. The lesions can be focal or diffuse, affecting the whole muscle and can be spread to the both supracoracoid muscles. However, the uni- to bilateral lesion ratio was not determined in the present study. The acute inflammation reaction was histologically confirmed by oedema and cell necrosis and the presence of an inflammatory infiltrate constituted mainly with macrophages and some heterophils was noted in injured muscles. Furthermore, lots of muscle fibres appeared as markedly necrotic (Zenker’s degeneration) and the discoid necrosis lead to the destruction of the muscle architecture. Older lesions were mainly characterized by a marked green colour of the affected muscles. By analogy with bile pigments it was firstly assumed that this green coloration derived from the degradation of a haemic pigment, such as haemoglobin or myoglobin, during the development of myopathy [15]. The resistance to acidic solvents shows that the pigment was covalently bound to protein [14] and in 1978, SILLER and WINGHT [15] suggested that this pigmentation may be due to the accumulation of one cytochrome, probably the cytochrome b. Thereafter, this pigmentation gradually disappeared in lesions evolving for a long time characterised by a chronic inflammation, leading to the muscle fibrosis and to the formation of a fibrous capsule in periphery [15]. Histologically, the muscle tissue gradually disappeared and was substituted with fibrous then adipose tissues, the connective tissue reaction usually beginning from the intact tendon [18].

There is a genetic predisposition of large-breasted birds to this muscular disease. Possible aetiology explanation for this disease may be related to an inadequate vasculature in meat-type birds [17]. Some evidence has been produced for a hereditary susceptibility of DPM [11]. The selection of these breeds would be associated with a gene mutation leading to apoptosis of myocytes frequently coupled to cytochrome accumulation in cytoplasm and inherent caspase activation or leading directly to the accumulation of the haemic proteins which in turn provoke the caspase cascade. Moreover, this data suggests that genetics may play an important role in the determination of DMP.

References

PREVALENCE OF DPM IN BROILER CHICKENS


