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Clinical and morphological studies on spontaneous rupture of the gastrocnemius tendon in broiler breeders

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Abstract 1. Clinical and morphological studies were performed in 8 broiler breeder flocks (one hybrid) originating from two different farms in Bulgaria, with an incidence of lameness in female birds between 3 and 4% on one farm and 7 and 8% on the other. Clinical and morphological studies were performed.
2. Ruptured gastrocnemius tendons were observed in 78 broiler breeders, 24 to 31 weeks old with signs of lameness, selected for pathoanatomical study from different flocks. Unilateral ruptures predominated, over 80%.
3. A histological study of 20 samples from one farm and 8 samples from the other revealed that lesions varied between birds. In about 50% of them, haemorrhages, dystrophic and necrobiotic changes in tendons were present. In the other 50% of cases, the resolving haematoma and ruptured tendon were surrounded by fibrous tissue growth. No inflammatory changes were detected.

INTRODUCTION

Rupture of the gastrocnemius tendon (RGT) is a recognised cause of lameness in broiler chickens and broiler breeders. Initially, it was thought to be congenital rather than acquired (Riddell, 1991). Subsequently, the problem was found to be associated with viral (reoviral) tenosynovitis (Jones et al., 1981; Kibenge et al., 1982; Jones and Kibenge, 1984). It was assumed that tendon rupture occurred as a consequence of tenosynovitis and that both lesions were components of a single disease (Itacura et al., 1977). However, in some experimental studies viral isolates produced lesions in tendons other than the gastrocnemius and viruses were not always re-isolated from the lesions (Ellis et al., 1983; Duff and Randall, 1986). It was further suggested that reoviruses could not provoke ruptures by themselves, but required the involvement of other predisposing factors (Duff and Randall, 1986). Both mycoplasmas and staphylococci were also associated with RGT but this was due to secondary colonisation of the tendon subsequent to viral tenosynovitis (Kibenge et al., 1983; Dinev et al., 1995; Kleven, 2003).

In some investigations, the tendon rupture was associated with several non-infectious factors such as deficiency states, obesity, variations in tensile strength, glucosaminoglycan content, cellular structure of the tendon in the various avian species, etc. (Van Walsum et al., 1981; Cook et al., 1983a, b; Bradshaw et al., 2002). Riddell (1983) discovered that RGT was encountered only in broilers fed ad libitum but not in birds on a restricted diet. The studies of Duff and Randall (1986) showed that some spontaneous episodes of tendon rupture were present in broilers on a restricted feeding regimen. Therefore, these data could not be used for evaluation of the effect of live body weight on tendon rupture incidence. Other reports assume a traumatic aetiology, supported by histological data (Hill et al., 1988).