

Review Article

LEG WEAKNESS IN COMMERCIAL BROILER CHICKEN- AN OVERVIEW

G. Raj Manohar*, A.V. Omprakash and P. Kanagaraju**

*Assistant Professor, Department of Poultry Science, Madras Veterinary College,
Chennai-600 007

**Professor and Head, Poultry Research Station, Chennai-600 051.
Tamil Nadu Veterinary and Animal Sciences University
E-mail: rajmanovet@gmail.com (**Corresponding Author*)

Introduction

Success in commercial broiler chicken enterprise depends to a greater extent on minimizing the incidence of disease occurrence and elimination of conditions producing stress to the birds which may result in economic losses. Diseases of the locomotor system are gaining economic significance in poultry industry.

Broiler Chicken has a capacity to grow very fast and produce tender meat. They are being developed for high growth rate through genetic selection. This produces early muscle growth without a simultaneous increase in the skeletal development resulting in leg weakness and disorders.

A normal gait is an integrated function of the nervous, muscular and skeletal systems. A failure in any of the above component will clinically result in leg weakness or lameness. This will cause increased culling rate or mortality in broilers.

According to Butterworth (1999),

Weakness-deficient in strength or power, easily broken, bent, weak point, defect.

Lameness-disabled by injury or defect in a limb especially foot or leg, limping or unable to walk normally.

Leg weakness due to Infectious conditions include

- (a) Bacterial chondronecrosis with osteomyelitis (BCO or femoral head necrosis, FHN)
- (b) Tenosynovitis and arthritis
- (c) Infectious Stunting Syndrome (ISS)

(d) Viral induced neoplasia.

Bacterial chondronecrosis with osteomyelitis or femoral head necrosis

According to McNamee and Smyth (2000), bacterial infection of growing bone are the commonest cause of lameness in growing fowl.

The term bacterial chondronecrosis with osteomyelitis (BCO) have been described under a variety of names including osteomyelitis (a bacterial infection of the metaphysis), femoral head necrosis, long bone necrosis, proximal femoral degeneration, bacterial chondritis with osteomyelitis and bacterial chondronecrosis.

Aetiology

In bacterial chondronecrosis with osteomyelitis, Staphylococcal infection (*S. aureus*) is the most common bacterium recovered from egg and joint infections of poultry (Skeeles, 1997). It has been frequently recovered from bone infections of commercial broilers.

Other bacteria recovered from infected bone include coagulase positive *Staphylococci* eg. *S. xylosus* and *S. simulans* and other agents such as *E.coli*, *Mycobacterium avium*, *Salmonella spp.* and *Enterococcus* (McNamee and Smyth, 2000).

Occurrence

Bacterial chondronecrosis appears to occur when Staphylococci (usually *S. aureus*), overcome the bird's local immune response, circulate in the blood stream, escape local destruction by circulating leucocytes and then establish themselves, forming micro-abscesses. These small abscesses may cause infarction and local metaphyseal bone necrosis. Staphylococcal osteomyelitis/chondronecrosis may affect the vertebral skeleton resulting in lameness and disability due to spinal cord damage. In broilers, the 'right condition' for colonization appear to be present between 25 and 45 days of age but a small number of cases may be seen as early as 14 days.

Clinical signs and pathology

Birds affected with bacterial chondronecrosis usually progress fairly rapidly from mild to severe lameness. They will have corresponding changes in gait (Kestin *et al.*, 1992), frequently use a wingtip for support when walking if severely affected and may demonstrate signs of discomfort (vocalization) when the proximal femur or tibia is manipulated (Thorp, 1996).

Macroscopically, bacterial chondronecrosis with osteomyelitis may appear as focal areas of yellow caseous exudate or lytic areas which cause affected bones to be fragile

(Skeeles, 1997). Lesions vary from small pale areas adjacent to the growth plate to the medullary cavity.

Prevention

The use of antibiotics is unlikely to provide long term solutions due to the inherent resistance of the organisms and the concerns over increasing the development of antibiotic resistance (McNamee and Smyth, 2000).

McNamee *et al.* (1999) showed that younger the birds were exposed to *S. aureus*, higher the incidence of bacterial chondronecrosis with osteomyelitis.

Bacterial interference has been used as a successful strategy to prevent Staphylococcal infection.

Arthritis and tenosynovitis

Bacterial and viral infection

These conditions result from the colonization of synovia (sheaths) which results in inflammation of the hock (arthritis of the tibiotarsus–tarso-metatarsus) and associated ligaments and tendons (inflammation of the gastrocnemius and digital flexor tendons – tenosynovitis). Affected birds are usually depressed and unable to walk due to pain (Jordon, 1996).

The incidence of bacterial (Staphylococcal) tenosynovitis is lower in broilers than in broiler breeders and accounts for 3-4 % of cases of lameness post-mortem. In the commercial broilers harvested at 6 or 7 weeks of age, Staphylococcal tenosynovitis is usually seen only in the acute phase, but in broiler breeders at 7-12 weeks of age, subsequent chronic fibrosis may be seen.

Mycoplasma infection

M.gallisepticum, *M. synoviae* and *M. iowae* have been implicated in acute chronic lameness caused by arthritis, synovitis and tenosynovitis.

Clinical signs

Clinical signs and pathology include lameness with swollen tendons and joints due to oedema and thickening of periarticular tissue, particularly the synovial membranes and the production of a viscous yellow to clear exudate in the tendon sheaths and joint cavities (Butterworth, 1999). Morbidity of affected flocks was usually 5-15 % but varied from 1-75 %.

Prevention

Several chemotherapeutic agents have been successful in preventing the spread in infected flock particularly tiamulin, lincomycin-spectinomycin, tylosin and tetracyclines. In

the long run, effective control may be only successful by elimination of the disease by culling. Treatment of breeders with chemotherapeutic agents does not prevent vertical transmission but dipping eggs in antibiotics or incubation, heat treatment is effective in limiting vertical transmission.

Viral infection

The viral condition has been reported as a significant problem in broiler chickens. Reovirus and Adenoviridae have been most commonly linked to leg disorders in chickens. Affected chickens at 4-6 weeks of age showed low mortality (<5 %) but high morbidity (Rosenberger and Olson, 1991). Incidence in an affected flock has been found to vary from 1-10 %. The affected chickens are usually 3-8 weeks of age with a number of poorly grown chickens (Heide, 1977). In the case of viral infection, there is no specific treatment and severely affected chickens have to be culled.

Infectious Stunting Syndrome (ISS)

Infectious stunting syndrome (ISS) has also been referred to as Helicopter disease, brittle bone disease, pale bird syndrome, malabsorption syndrome, runting and stunting syndrome.

ISS has been reported in young chicks from most countries with intensive broiler production. Prevalence of severely stunted but active chickens is low, usually 1-5 %. Incidence varies for flocks infected from 5-20 %.

Aetiological factors include Picorna virus like particles, characterised by implication as a cause (Reece and Frazier, 1990). Impaired digestion resulting in poor absorption of nutrients has also been suggested as a cause because evidence of enteritis has been found (Bracewell and Randall, 1984).

Clinical signs include stunted growth and swollen joints. One of the feature is the presence of severely stunted chickens which remain small despite voracious appetites. The stunted nature of the chickens is noticeable as early as 4-8 days of age. The problem is more noticeable after two weeks when the unaffected chickens grow rapidly. Histological examinations reveal ricket-like changes in the tibiotarsi of 2-4 week old broiler chickens.

Cold stress appears to increase the severity of stunting and adequate temperature should therefore be maintained (Reece, 1996). Economic losses can be reduced by culling (up to 10 % of the flock). Treatment with massive doses of vitamins (A, D and E) has been known to ameliorate some effects.

Viral induced neoplasia

Viral induced neoplasia includes osteopetrosis, myelocytomatosis and Marek's disease which are not very prevalent. Osteopetrosis is any condition characterized by abnormal growth and modelling of bone. It is rare in chickens.

Myelocytomas involves overgrowth of the bone marrow and abnormal protuberances on the surface of various bones including the tibiotarsi have been reported in broilers.

Marek's disease is caused by infection of young susceptible chickens with Herpes virus. It usually affects chickens from 6 weeks of age, but more usually after 7 weeks. For this reason, the incidence of Marek's disease as a cause of neurological lameness in broilers is very low. Vaccines are available which give effective protection under normal commercial conditions.

REFERENCES

- [1] Bracewell, C.D. and C.J. Randall, 1984. The infectious stunting syndrome. *World Poult. Sci.*, **40**, 31-37.
- [2] Butterworth, A., 1999. Infectious components of broiler lameness – a review. *World. Poult. Sci.*, **55**, 327-350.
- [3] Heide, L. Vander, 1977. Viral arthritis / tenosynovitis -a review. *Avian Pathol.*, **6**, 271-284.
- [4] Jordon, F.T.W., 1996. Staphylococci. In : *Poultry Diseases*. London, pp. 66-69.
- [5] Kestin, S.C., T.G. Knowles, A.E. Tinch and N.G. Gregory, 1992. Prevalence of leg weakness in broiler chickens and its relationship with genotype. *Vet. Rec.*, **131**, 190-194.
- [6] Kestin, S.C., S. Gordon, G. Su and P. Sorensen, 2001. Relationship in broiler chickens between lameness, live weight, growth rate and age. *Vet. Rec.*, **148**, 195-197.
- [7] Mcnamee, P.T. and J.A. Smyth, 2000. Bacterial chondronecrosis with osteomyelitis of broiler chickens : a review. *Avian Pathol.*, **29**, 253-270.
- [8] Mcnamee, P.T., J.J. McCullagh, J.D. Rodgers, B. Thorp, H.J. Ball, T.J. Connor, D. McConaghy and J.A. Smyth, 1999. Development of an experimental model of bacterial chondronecrosis with osteomyelitis in broilers, following exposure to *Staphylococcus aureus* by aerosol and inoculation, chicken anaemia and infectious bursal disease viruses. *Avian. Pathol.*, **28**, 26-35.
- [9] Reece, R.L., 1996. Infectious stunting syndrome. In : *Poultry Diseases* edited by F.T.W. Jordon and M. Pattison. Saunders Company, London.

- [10] Reece, F.N. and J.A. Frazier, 1990. Infectious stunting syndrome of chickens in Great Britain : field and experimental studies. *Avian Pathol.*, **19**, 723-758.
- [11] Rosenberger, J.K. and N.O. Olson, 1991. Reovirus infection. In : *Diseases of poultry 9th edition* (Eds. B.W. Calnek, H.J. Barnes, C.W. Beard, W.M. Read, H.W. Yoder Jr.) pp. 639-674. London, Wolfe Publishing Ltd.
- [12] Skeeles, K.J., 1997. Staphylococcosis. In : B.W. Calnek, Barnes, H.J., Beard, C.W., McDougald, L.R. and Y.M. Saif (Eds). *Diseases of poultry 10th edition*, Chapter II (pp. 247-253). Ames, IA : Iowa State University Press, U.S.A.
- [13] Thorp, B.H., B. Ducro, C.C. Whitehead, C. Farquharson and P. Sorensen, 1993. Avian tibial dyschondroplasia: the interaction of genetic selection and dietary 1, 25 dihydroxy cholecalciferol. *Avian Pathol.*, **22**, 311-324.