INFECTIOUS STUNTING SYNDROME IN CHICKENS: A REVIEW

Tahseen A. Abdul-Aziz, Department of Poultry and Fish Diseases, College of Veterinary Medicine, AL-Ameria, Baghdad, Iraq.

Synonyms

Brolier runting syndrome, runting and stunting syndrome, femoral head necrosis, brittle bone disease, maldigestion syndrome, malabsorption syndrome, pale bird syndrome, helicopter chick, infectious proventriculitis.

Definition

An infectious syndrome of broiler chickens characterized by stunted growth, poor feathering, leg weakness, poor feed conversion, and loss of pigmentation of the skin. The syndrome was initially reported in Netherland (Kouwenhoven et al., 1978a), and have since been described in England (Bracewell and Wyeth, 1981), U.S.A. (page et al., 1982), Australia (pass et al.,1982) and Canada (Riddell and Derow, 1982). The various names of the syndrome reflects the variety of clinical signs and gross lesions observed, and although reported conditions share many clinical signs and pathological changes, it is not certain that all reports describe the same disease entity.

Aetiology

The infectious nature of the syndrome was first demonstrated by its contact transmission (Vertommen et

al., 1980a); also the syndrome could be reproduced in one-day-old and one week-old chickens by oral inoculation of intestinal filtrate or crude intestinal homogenate prepared from affected chickens (Kouwenhoven et al., 1978a, b; Bracewell and wyeth, 1980; vertommen et al., 1980a). There are evidence of vertical transmission of the syndrome (Wyeth and chettle, 1985). The syndromes seems to be caused by a virus. Several viruses were either isolated or visualized by electron microscope in the tissues of affected birds. Candidate viruses include reovirus (Van der Heide et al., 1981; Baxendale, 1982; page et al., 1982; pass et al., 1982; Heironymus et al., 1983; Robertson et al., 1984), enteorvirus (McNulty et al., 1982; Decaesstecker et al., 1986), parvovirus (Kisary et al., 1984; Kisary, 1985), calicivirus (Wyeth et al., 1981), togavirus (Frazier et al., 1986), and a novel unclassified virus (Farmer and Taylor, 1985). However, the exact role of each of these viruses is still questionable.

Avian reoviruses have been isolated from the intestine and other organs of chicks affected with stunting syndrome (Vertommen et al., 1980a; Baxendale, 1982; Page et al., 1982; pass et al., 1982; Robertson et al., 1984). Inoculation of isolated reoviruses in on - day-old chicks induced growth retardation and diarrhea (Vertommen et al., 1980a; Baxendale, 1982; page et al., 1982). Enterovirus-like particles were identified during the first week of life in the gut contents of broiler chickens which later developed runting (McNulty et al., 1984); inoculation of one-day-old chicks with gut contents containing the enterovirus-like particles resulted in abnormal faces, poor feathering, and poor growth. Viral particles, suggested to be belong to parvoviridae, were detected in the intestinal homogenate

of 10-day-old chickens affected with stunting syndrome (Kisary *et al.*, 1984). Calicivirus-like particles were detected in the intestinal contents of chickens suffering from the stunting syndrome (Wyeth *et al.*, 1981). Togavirus-like particles have been observed in the epithelial cells lining the pancreatic ducts of chickens from a flock with stunting syndrome (Frazier *et al.*, 1986)

A striking feature of the syndrome is the development of very early age resistance (vertommen, 1982). It is difficult to reproduce stunting by one week of age, and almost impossible by two weeks of age (Bracewell and Randall, 1984).

Clinical Signs

Clinical signs of the syndrome have been reported by Koumenhoven *et al.* (1978b), Bracewell and Wyeth (1981), Bracewell and Randall (1984), Reece *et al.* (1984), and Farmer (1986). The most characteristic clinical feature is the uneven growth of chickens; the runting in growth usually becomes obvious between the first and third week of life. Clinical signs that may be observed during the first week of life, in up to 50% of chickens in the flock, include listlessness, diarrhea, dropping eating, and spherical profile of the body due to buffing out of the feathers. By one to two weeks of age, 0.1-20% of chickens in the flock are stunted but active with varacious appetite. Stunted chickens pass off droppings which contain large amounts of poorly digested food. Some stunted birds start to grow after the second week, whereas others begin to grow after four weeks of age. However at the time of slaughter (7 to 8 weeks of age) affected chickens are very underweight. Feathering abnormalities have been observed at three to four week of

age; the body feathers are rough and buffed, and the development of adult plumage is delayed, so that the juvenile feathers are usually present on the head and neck. The primary wing feathers are poorly and irregularly grown resulting in "helicopter chick" appearance. Loss of pigmentation from the pigmented areas of the skin has been observed (vertommen *et al.*, 1980; page *et al.*, 1982) and was attributed to failure in the absorption of yellow pigment of corn in the ration.

Gross lesions

The abdomen of affected bird is often distended; the distension is more obvious when the skin over the abdomen is stripped (Bracewell and Wyeth, 1981; Riddell and Derow, 1985). Abdominal distension is due to the distension of intestine, and in some cases, the intestinal loops are visible through abdominal wall. The intestine is variably pale and contains undigested food with liquid and, or, orange-tinged mucoid material. The caeci may be distended with gas and frothy fluid, or contain yellowish, thin, unpleasantly odor material (Kouwenhoven *et al.*, 1978a page *et al.*, 1982; Bracewell and Randall, 1984; Riddell and Derow, 1985). Proventricular lesions (proventriculitis/ proventricular hyperplasia) have been reported in naturally and experimentally infected birds (Kouwenhoven *et al.*, 1978a; page *et al.*, 1982). The proventriculus is enlarged and its wall is thickened and turgid. The mucosal glands are engorged and the mucosal surface may be coated with fibrinous necrotic material or contains hemorrhages. The gizzard has been reported to be small in size and flaccid (Kouwenhoven *et al.*, 1978a; Good, 1982; page *et al.*,1982). Pancreatic lesion is characterized by atrophy of one or more lobes to almost complete replacement of

the pancreas with a thin band of fibrous tissue; affected part of the pancreas may be white and firm (Randall et al., 1981; Reece et al., 1984; Riddell and Derow 1985). Skeletal abnormalities have been observed (Page et al., 1982; Riddell and Derow, 1985); the bones of affected bird are brittle and the growth plates of long bones are widened. The head of the femur is easily broken at its junction with the shaft, particularly when the legs of affected chickens are spread during necropsy. Tibial dyschondroplasia has been reported on two occasions (Brien, 1980; Vertommen et al., 1980a). Others also reported hydropericardium (Good, 1980; Page et al., 1982) and atrophy of bursa of Fabricius and thymus (Good, 1982; Barr et al., 1983; Brien, 1983; Reece et al., 1984; Riddell and Derow, 1985)

Microscopic lesions

Range of histopathological changes, which vary in extent and severity, have been described in the pancreas of affected birds (Randall et al., 1981; pass et al 1982; Reece et al., 1984; Rinddell and Derow, 1985; Martland and Farmer, 1986). In some cases, there are vacuolation and partial or complete depletion of Zymogen granules of acinar cells; the vacuoles may contain eosinophilic hyaline inclusions. There may be few necrotic acinar cells. In other cases, the acinar epithelium is flattened from columnar to cuboidal or squamous with dilation of acinar lumens. In severely affected pancreas, there are atrophy of many acini with marked interstitial and capsular fibrosis. Fibrosis tends to obliterate much of the exocrine tissues, and several lobes have small distorted acinar remnants or small branching ductules embedded in a mass of fibrous tissue. Fibrosis may be accompanied by infiltration with macrophages,

lymphocytes, and heterophils. In the intralobular pancreatic ducts, lesions vary from dilation of the lumen, with flattening of the epithelium, to narrowing of the lumen, with degeneration of epithelial lining and thickening of the wall. The wall of some ducts is thick, necrotic, edematous, and infiltrated with mononuclear leukocytes; the lumen may be obstructed by inspissated mucus, bacteria, and cellular debris. The interlobular pancreatic ducts may be stenosed or occluded, with focal vacuolation and necrosis of epithelium. Some times the wall of the ducts is edematous, necrotic, and inflamed, or thickened with granulation tissue.

A proportion of affected birds have lesions in the intestine, including inflammation, necrosis, and cyst formation in the crypts of Lieberkuhn (Vertommen *et al.*, 1980b).

The proventriculitis, if present, is characterized histologically by hyperplasia of glandular epithelium, with infiltration of mononuclear leukocytes in the glandular tissue and mucosa. Occasionally, there is focal necrosis of glandular tissue, or haemorrhages in the mucosal villi. Interglandular fibrosis was seen in some cases (Kouwenhoven *et al.*, 1978; page *et al.*, 1982).

Histological changes in the bursa of Fabricius consist of atrophy of lymphoid follicles, depletion of lymphocytes in the medulla, and hyperplasia of reticuloepithelial cells, (Bracewell and Randall, 1984). In the thymus, thymic lobes are smaller than normal, and the separation between cortex and medulla is idistinct (Reece *et al.*, 1984).

Microscopic lesions in the bone were also described. Page *et al.*, (1982) found a transverse and vertical clefts extend across the growth plate, with focal necrosis of the cartilage. Riddell and Derow (1985)

reported thickening of zone of proliferation and poor vascularization of zone of hypertrophy.

Biochemical changes in blood

These include an increase in the alkaline phosphatase activity (Vertommen *et al.*, 1980a, Barr *et al.*, 1983) and a decrease in the amylase and glutathione peroxidase activity (Barr *et al.*, 1983) and caroten pigment (Vetommen *et al.*, 1980a, Ruff 1982).

Pathogenesis

The mechanism of clinical signs and lesions is not fully understood. Impaired nutrient utilization has been reported in stunted birds (Liburn *et al.*, *1982; Ruff, 1982; Barr et al.*, 1983). It is thought that infection of the digestive tract, with subsequent damage, very early in life impairs the absorption of essential nutrients. But farmer (1986) commented that blockage of pancreatic ducts and atrophy of the pancreas resulting in deficiency of digestive enzymes which leads to maldigestion and retarded growth of affected birds. The maldigestion prevent essential nutrients to be available in absorptive forms.

What is ahead

The aim of researches is to identify the definite causative agent. This step will help for better understanding of pathogenesis of the syndrome, and for finding a vaccine to control it.

REFERENCES

Barr, D.A. Sinclair, A.J.; Reece, R.L. Embury, D.H.; Gould, J.A.; Hooper, P.T. and Smart, I.J. (1983).

Stunting syndrome in broiler chickens. Vet. Rec. 113: 380

Baxendal, W. (1982). Broiler runting syndrome: report of a seminar. World's poult. Sci. J. 38: 224

Bracewell, C.D. and Randall, C.J. (1984). The infectious stunting syndrome. World's poult. Sci. J. 40: 31-37.

Bracewell, C.D. and Wyeth, P. (1981). Infectious stunting of chickens. Vet. Rec. 109: 64

Brien, J.D.P. (1983). Stunting syndrome in broiler chickens. Vet. Rec. 113: 203.

Decaesstecker, M.; Charlier, G. and Meulemans, G. (1986). Significance of parvoviruses, entero-like viruses and reoviruses in the aetiology of the chicken malabsorption syndrome. Avian pathol. 15: 769-782.

Farmer. H. (1986). Research progress on infectious stunting syndrome. Poult Int. 25: 12-13.

Frazier, J.A.; Farmer, H. and Metland, M.F. (1986). A togavirus-like agent in the pancreatic duct of chickens with infectious stunting syndrome. Vet. Rec. 119: 209-210.

Good, R.E. (1982). The pale bird syndrome. Poultry Digest. 41: 278-279.

Heironymus, D.R.K., Villegas, P. and Kelven, S.H. (1983). Indentification and serological differentiation of several reovirus strains isolated from chickens with suspected malabsorption syndrome. Avian Dis. 27: 246-254.

Kisary, J. (1985). Experimental infection of chicken embryos and day-old, chickens with parvovirus of chicken origin. Avian pathol. 14: 1-7.

Kisary, J.; Nagy, B. and Bitay, Z. (1984). Presence of reoviruses in the intensive of chickens showing stunting syndrome. Avian Pathol. 13: 339-343.

Kouwenhoven, B.; Davelaar, F.G. and Van Walsum, J. (1978a). Infectious proventriculitis causing runting in broilers. Avian Pathol. 7: 183-187.

Kouwenhoven, B.; Vertommen, M.S. and Eck, J.H.H. Van (1978b). Runting and leg weakness in broilers: involvement of infectious factors. Vet. Sci. Commune. 2: 253-259.

Liburn, M.S.; Edwards, H.M. and Jensen, L.S. (1982). Impaired nutrient utilization associated with pale bird syndrome in broiler chicks. Poult. Sci. 61: 608-609.

Martland, M.F. and Farmer H. (1986). Pancreatic duct obstruction in a sunting syndrome of broiler chickens. Vet. Rec. 118: 531-534.

McNulty, M.S.; Allan, G.M.; Connor, T.J.; McFerran, J.B. and McRacken, R.M. (1984). An Entero-like virus associated with the runting syndrome in broiler chickens. Avian Pathol. 13: 429-439.

Page, R.K.; Fletcher, O.J.; Rowland, G.N.; Gaudry, D. and Villegas, P. (1982). Malabsorption syndrome in broiler chickens. Avian Dis. 26: 618-625.

Pass, D.A.; Robertson, M.D. and Wilcox, G.E. (1982). Runting syndrome in broiler chickens in Australia. Vet. Rec. 110: 386-387.

Randall, C.J.; Wyeth, P.J. and Higgins, R.J. (1981). Pancreatic lesions in stunting broilers. VEt. Rec. 109: 125.

Reece, R.L.; Hooper, P.T.; Tate, S.H.; Beddome, V.D.; Forsyrh, W.M.; Scott, P.C. and Barr, D.A. (1984). Field, clinical and Pathological observations of a runting and stunting syndrome in broilers. Vet. Rec. 115: 483-485.

Riddell, C. and Derow, D. (1985). Infectious stunting and pancreatic fibrosis in broiler chickens in Saskatchewan. Avian Dis. 29: 107-115.

Robertson, M.D.; Wilcox, G.E. and Kibenge, F.S.B. (1984). Prevalence of reoviruses in commercial chickens. Aust. Vet. J. 61: 319-322.

Ruff, M.D. (1982). Nutrient absorption and changes in blood plasma of stunted broilers. Avian Dis. 26: 852-859.

Van der Heide, L.; Lutticken, D. and Horzinek, M. (1981). Isolation of avian reovirsus as a possible etiologic agent of osteoporosis (brittle bone disease; femoral head necrosis) in broiler chickens. Avian Dis. 25: 847-856.

Vertommen, M. (1982). Broiler runtiung syndrome: report of a seminar. World's poult. Sci. J. 38: 223.

Vertommen, M.; Eck, J.H.H. Van; Kouwenhoven, B. and Kol, N. Van. (1980a). Infectious stunting and leg weakness in broilers: I. Pathology and biochemical changes in blood plasma. Avian Pathol. 9: 133-142.

Vertommen, M.; Van der Laan, A. and Veenendaal-Hesselman, H.M. (1980b). Infectious stunting and leg weakness in broilers II. Studies on alkaline phosphatase isonzymes in blood plasma. Avian Pathol. 9: 143-152.

Wyeth, P.J. and Chettle, N.J. (1985). Infectious stunting syndrome: evidence of vertical transmission. Vet. Rec. 117: 465-467.