Pathology of a Broiler Disease Characterized by the Swollen Head

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(Received 26 June 1990/Accepted 30 November 1990)

KEY WORDS: broiler, swollen head syndrome.

A new disease of young broiler chickens characterized by swollen head and depression has recently been described. The disease was first reported in South Africa [4] and called “swollen head syndrome (SHS”) since then, the syndrome has been recognized in England [5, 9] and sporadic areas of other countries [7, 8]. The etiology remains uncertain but consistent findings were the recovery of pure growth of Escherichia coli from swollen head lesions [4–7] and antibodies to turkey rhinotracheitis (TRT) virus were often identified in birds from some affected flocks [6, 8, 9].

On a farm raising about 10,000 broiler chickens in Hyogo Pref., Japan, birds showing clinical signs similar to those of SHS appeared at around 25 days of age. The disease gradually spread on the farm and the signs became most apparent during the following 4 to 5 days; some of severely affected birds closed their eyes with the swollen eyelids and facial skin (Fig. 1). Mortality ranged from 3 to 7% among flocks. Five birds showing marked signs were killed and subjected to pathological and microbiological examinations. Grossly, the swollen heads were due to extensive gelatinous edema of the facial skin and peri-orbital tissues, and various amounts of caseous exudates in the lacrimal gland, conjunctival sac and facial subcutis.

Microscopically, the skin covering the swollen head was greatly thickened due to edema and inflammatory granulation tissue that developed around the caseous, coagulated exudates (Fig. 2). The exudates accompanied reactions of many macrophages and giant cells of foreign body type at the periphery, and contained numerous colonies of Gram-negative bacilli. These organisms stained intensely with carbol thionin and measured approximately 0.6 μm × 1.5−2.0 μm by electron microscopy (Fig. 3). There were often acute dermatitis with scabs or intraepidermal pustules (Fig. 4) and occasionally subchronic changes such as lymphocytic infiltration with lymph follicle formation in the dermis and subcutaneous tissue. The eyelids were markedly thickened due to hyperemia, edema and giant cell granuloma in the connective tissue plate, and acute conjunctivitis was commonly present (Fig. 5). The conjunctival epithelial...

Fig. 1. Head of a chicken showing marked swelling of the peri-orbital and infraorbital skin (arrow-heads).

Fig. 2. Periocular skin of an affected bird. The skin is greatly thickened due to edema and granulation tissues developed around subcutaneous caseous exudate. Hematoxylin and eosin stain (HE). ×29.

Fig. 3. Subcutaneous coliform granuloma of the facial skin showing caseous, coagulated exudate surrounded by foreign body giant cells. HE. ×72. Inset shows many degenerated bacilli in the exudate by electron microscopy. ×5,333.
lesions. The isolate was examined for three major capsular antigens, 01, 02, and 078, by the agglutination test using specific rabbit antisera but did not belong to any of the serotypes tested. No antibodies were detected against *Mycoplasma gallisepticum* or *Mycoplasma synoviae* by the slide agglutination test, *Haemophilus gallinarum* by the hemagglutination inhibition test and chicken anemia agent by the virus neutralization test and enzyme-linked immunosorbent assay in any of the chickens examined.

The chicken disease presented here seems to be the same condition as SHS. The characteristic signs of swollen head and the recovery of *E. coli* in pure culture from lesions were identical with those of SHS [4, 5]. The swollen head may have resulted from widespread subchronic inflammation occurring in the lacrimal ducts or glands, periorcular subcutaneous tissues and eyelids. Variable-sized, caseously coagulated necrotic exudates with multinucleated giant cell reaction were morphologically typical of coliform granuloma [1, 3]. The culture of *E. coli* yielded from these lesions and *in situ* detection of similar bacilli in stained preparations implied that the granulomatous lesions were induced by the organism. Therefore, the condition described here could pathologically be regarded as coliform granuloma developed in the facial skin.

Little is known about the pathogenesis of SHS. Avian coronavirus and paramyxovirus were concomitantly isolated from affected chickens [4] and the disease often appeared when TRT was prevalent among flocks [6, 8, 9]. Such circumstantial evidence and failure in reproducing typical SHS by inoculating chickens with TRT virus alone [4, 8] suggested involvement of TRT virus as a predisposing factor for SHS. This was fortified by detection of antibodies against TRT virus in birds of the farm from which the present SHS cases originated (Uramoto, K., et al., personal communication). *E. coli* often infects the respiratory tract of birds in combination with infectious bronchitis and Newcastle disease viruses and mycoplasma, and causes varying conditions such as airsacculitis, pericarditis, panophthalmitis and salpingitis [3]. The present SHS cases, which are seemingly another form of collibacillosis, appeared to have developed in a similar manner by a trigger action of TRT virus infection. Whether specific serotype(s) of *E. coli* is responsible for the development of SHS remains to be solved because we could not determine serotype of the isolate.

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