AVIAN POX

D. N. Tripathy

College of Veterinary Medicine

University of Illinois, Urbana, Illinois 61801

Avian poxviruses - fowl, pigeon, turkey, canary, junco, quail, sparrow, and psittacine poxviruses are members of the genus Avipoxvirus of the Poxviridae family. Fowl poxvirus is the type species of the genus. Because of similar clinical manifestation in different avian species, only fowlpox is described here.

Fowlpox is a slow-spreading disease, and the virus can persist in a susceptible population for a long time. The disease occurs in two forms - cutaneous (slides 1, 2) and diphtheritic (slides 3, 4). Fowlpox may be suspected when skin lesions erupt on various parts of exposed skin (cutaneous form) of affected chickens. A mild form of the disease may remain unnoticed, with only small focal lesions, usually on the comb and wattles. In severe form of the disease, generalized lesions may occur on any part of the body, i.e., comb, wattle, corner of the mouth, around the eyelids, angle of the beak, ventral surface of the wings, legs, (slides 5) and vent. Skin lesions may be small and discrete or may involve large areas through the coalescence of adjoining lesions (slide 6). Coalescence of the lesions around the eyelids can cause complete closure of one or both eyes (slide 2). The small focal nodules of the skin, initially vesicular, enlarge rapidly because of proliferation of the virus in the epithelium and infiltration by the inflammatory cells. The surface of the lesions is moist for a short time but dries soon, with a rough irregular surface which becomes yellowish-brown to dark-brown. Removal of such lesions, if not completely dry, leaves a hemorrhagic moist

surface. When the scab is dry, however, it drops off, leaving a scar. The virus often also affects the mucous membrane of the mouth (slide 4) nares, pharynx, larynx, esophagus, and trachea (slide 3) causing white or opaque eruptions which coalesce and expand rapidly, later becoming ulcerated and covered with a yellowish caseous necrotic exudate. Mucous membranes of mouth, larynx, pharynx, and trachea (diphtheritic form) undergoing the extensive fibrinonecrotic process develop a diphtheritic membrane. A hemorrhagic surface is left when the diphtheritic membrane is removed. Lesions in the mouth, tongue, and esophagus interfere with the feeding, and lesions of the trachea often result in the formation of tracheal plugs (slide 3). In such cases, there is serious difficulty in respiration, with signs of gasping, and suffocation may result. This form of the disease may simulate signs of laryngotracheitis.

In layers, fowlpox causes a drop in egg production, and in young chicks, growth is reduced and feathering may be abnormal. Mortality occurs in birds with generalized lesions or with the diphtheritic form of fowlpox. Recovered birds are immune.

The lesions of fowlpox develop after an incubation period of 4 to 8 days, and in protracted severe infections may last as long as 8 to 9 weeks.

Histologically, fowlpox lesions show hyperplasia of the infected epithelium. Affected cells are enlarged and contain eosinophilic cytoplasmic inclusions (slide 7) with hematoxylin eosin stain. Smears prepared from the lesions and stained by the Gimenez stain, reveal elementary bodies when examined under oil immersion (slide 8). Viral particles with typical poxvirus morphology are observed in negatively stained preparations (slide 9) and in ultrathin sections of lesions (slides 10, 11).

Fowlpox virus can be isolated from the cutaneous or diphtheritic lesions from infected birds. Approximately 0.1 ml of the tissue suspension is inoculated on the chorioallantoic membrane (CAM) of 9-to-12-day-old developing chicken embryos. Inoculated embryos are incubated at 37C and are examined for pocks on the CAM 4 to 5 days later (slide 12). The virus multiplies in the cytoplasm of the infected cells with the formation of inclusion bodies (Bollinger bodies) or elementary bodies (Borrel bodies). The virus causes proliferation of the epithelium with ballooning of the cells. The cytoplasmic inclusions vary in size and shape. These inclusions can be stained by haematoxylin eosin (slide 13), acridine orange (slide 14), Giemsa (slide 15), and Feulgen reactions (slide 16). The specificity of the viral inclusions can be determined also by fluorescent antibody (slide 17) and immunoperoxidase methods (slide 18) and by blocking reaction (slide 19).

Susceptible chickens infected by cutaneous scarification develop lesions at the site of inoculation in 5 to 10 days. Generalized lesions may appear later during the disease. Histopathologic examination of the lesions reveals characteristic cytoplasmic inclusions.

Primary chicken embryo or chicken kidney cells support the growth of fowlpox virus. Adaption of strains to cell culture is necessary for plaque formation since not all strains form plaques.

Immunity against fowlpox develops following vaccination (fowlpox or pigeon pox vaccine) or as a result of natural infection. Both humoral and cell-mediated immunity can be detected after vaccination.

Humoral antibody response can be measured by agar gel precipitation, passive hemagglutination, virus neutralization, fluorescent antibody, immunoperoxidase, and enzyme-linked immunosorbent assay (ELISA).

DIFFERENTIAL DIAGNOSIS

Lesions due to pantothenic acid or biotin deficiency in young chicks or by T_2 toxin, may be confused with pox lesions. The diphtheritic form of fowlpox, with involvement of trachea and larynx, may simulate signs of laryngotracheitis. Histopathological examination of the trachea from birds infected with fowlpox show hyperplasia of the epithelium with eosinophilic cytoplasmic inclusions. Laryngotracheitis is caused by a herpes virus and produces intranuclear inclusions. Diphtheritic pox lesions in doves and pigeons may be differentiated from lesions caused by Trichomonas gallinae.

Acknowledgements

Slide numbers 1 and 4 were kindly provided by Drs. H. L. Shiaprasad and slide number 19 was provided by Dr. A. M. Watrach.

Slide 1 Cutaneous pox lesions in a naturally infected bird involving head area.

Slide 2 Cutaneous pox lesions (comb and eyelids) in an experimentally infected bird.

Slide 3 Diphtheritic pox lesions in a naturally infected bird showing a tracheal plug.

Slide 4 Diphtheritic pox lesions in the mouth of a naturally infected bird.

Slide 5 Cutaneous pox lesion (feet) in an experimentally infected bird.

Slide 6 Cutaneous pox lesion in an experimentally infected bird.

Slide 7 Section of cutaneous pox lesion showing eosinophilic cytoplasmic inclusion bodies.

Slide 8 Smear of a cutaneous pox lesion stained with Gimenez stain - red staining inclusions with elementary bodies. Some elementary bodies are dispersed free.

Slide 9 Fowlpox virus in a negatively stained preparation.

Slide 10 Fowlpox virus particles in ultrathin sections from a fowlpox virus infected lesion.

Slide 11 Fowlpox virus particles in ultrathin section showing core and lateral bodies.

Slide 12 Chorioallantoic membrane (CAM) of developing chicken embryo showing pocks produced by fowlpox virus.

Slide 13 Section of infected chorioallantoic membrane (CAM) showing cytoplasmic eosinophilic inclusions of fowlpox virus (H & E stain).

Slide 14 Section of infected CAM showing cytoplasmic inclusion (greenish-blue stain) of fowlpox virus (Acridine orange).

Slide 15 Section of infected CAM showing cytoplasmic blue-stained inclusions of fowlpox virus (Giemsa stain).

Slide 16 Section of infected CAM showing cytoplasmic inclusions of fowlpox virus (Fuelgen reaction indicating presence of DNA material).

Slide 17 Section of fowlpox virus infected skin stained with fluorescein isothiocyanate labeled fowlpox antibody. Only inclusions (greenish) are staining.

Slide 18 Section of infected CAM showing cytoplasmic inclusion (dark brown) of fowlpox virus - stained by horseradish peroxidase labeled antibody against fowlpox virus. ? wrong

Slide 19 Section of infected CAM as in slide (9) - Immunoperoxidase blocking reaction with unlabeled antibody against fowlpox virus. Structural evidence of inclusions is present without the dark stain, as a result of blocking.

DNT:jq:0116F
08/16/85