Fowl pox is a slow-spreading viral disease of chickens, turkeys, and many other birds characterized by cutaneous lesions on unfeathered skin of the head, neck, legs, and feet and/or by diphtheritic lesions in the upper digestive and respiratory tracts.

Among poultry, pox occurs frequently in chickens and turkeys. Among other birds, pigeons, canaries, and psittacines are frequently infected and the disease is seen occasionally in many wild birds. Perhaps all birds are susceptible. The disease occurs in all age groups except the recently hatched and is worldwide in distribution.

Fowl pox is an ancient disease and in the distant past was mistakenly thought to be related to small pox and chicken pox of man. The characteristic pox inclusion bodies (Bollinger bodies) and the smaller elementary bodies within them (Borrel bodies) were studied by Drs. Bollinger and Borrel, respectively, in 1873 and 1904. In the United States, pox has been a common and frequently reported disease of poultry. In recent years there has been increased interest in pox in wildlife and caged birds, which are being submitted to diagnostic laboratories in increasing frequencies.

Fowl pox is caused by a large DNA Avipoxvirus of the family Poxviridae. Many strains of virus are recognized and naturally infect the species given in their name. Some common examples are:

- fowl poxvirus (type species)
- quail poxvirus
- turkey poxvirus
- mynah poxvirus
- pigeon poxvirus
- psittacine poxvirus
- canary poxvirus

Poxviruses appear to be closely related, however, a strong host specificity is found with most poxvirus strains. In some instances, exposure to one of the viruses in the group engenders immunity to that virus and one or more of the other viruses in the group. Poxvirus isolates from Hawaiian forest birds (alala and apapane poxvirus strains) are more related to each other than to fowl poxvirus indicating genetically distinct poxviruses in this region. Perhaps all strains are host-modified variants of what was once a single virus.

The various strains of avian poxvirus are morphologically identical. Strain classification has traditionally depended upon the cross-protection test in birds but these are not practical for routine diagnosis. Restriction endonuclease analysis of DNA has been successful in differentiating strains.

Recovery from poxvirus infection usually results in a strong, enduring immunity to later exposure to the same virus. Also, in turkeys and chickens vaccination is usually quite effective in preventing pox. Recently, however, several outbreaks of fowl pox have occurred in vaccinated chickens.

Virus is present in lesions and in desquamated scabs. Poxvirus is quite resistant to environmental factors and persists in the environment for many months.

Most poxviruses stimulate the formation of inclusion bodies in infected epithelium. Intracytoplasmic inclusions (Bollinger bodies) contain elementary bodies (Borrel bodies). Bollinger bodies are quite large and readily identified microscopically.
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EPIDEMIOLOGY

1. The virus-containing crusts (scabs) formed on the skin are desquamated into the litter. Virus persists in the environment and may later infect susceptible birds by entering the skin through minor abrasions. Mechanical transmission via cannibalism is thought to play a significant role in some outbreaks. Respiratory tract infection can result from inhalation of aerosolized virus-containing feathers and scabs.

2. Certain mosquitoes, and possibly other blood-sucking arthropods, can transmit virus from infected to susceptible birds. Mosquitoes remain infective for several weeks. Mosquito-transmitted outbreaks may result in rapid spread.

3. Poxvirus infection can result from mechanical transmission from toms to turkey hens via artificial insemination.

CLINICAL SIGNS

1. In poultry, onset often is gradual and the disease may go undetected until cutaneous lesions are numerous and obvious in the flock. The disease spreads slowly and severe outbreaks may last many weeks. Turkey pox infection is generally more chronic than fowl pox infection. Canaries can have systemic infection with high mortality. Signs vary somewhat with the two overlapping forms of pox:

   A. Cutaneous form

   This form predominates in most outbreaks. Birds often show few signs other than a mild to moderate reduction in rate of gain, a temporary loss in egg production, or a lack of flock vigor. Mortality is low if the disease is uncomplicated.

   B. Diphtheritic form

   Lesions in the upper respiratory or digestive tract may result in dyspnea or inappetence, respectively. Lesions in the nasal cavity or conjunctiva lead to nasal or ocular discharge. Mortality is low to moderate and is often due to suffocation or starvation and dehydration.

LESIONS

1. Cutaneous lesions vary in appearance according to whether the papule, vesicle, pustule, or crust (scab) stage is observed. In most outbreaks the terminal reddish brown to black scab stage [Fig. 1; Fowl Pox; UC Davis] is present on at least some of the birds presented for diagnosis. Papules, the initial lesions, are light-colored nodules in the skin. Vesicles and pustules are raised, usually yellow. Occasionally, small papilloma-like lesions occur. Lesions usually occur on the unfeathered skin of the head [Fig. 2; Fowl Pox; UC Davis] and neck but may occur around the vent or on the feet or legs. Cage birds and wild birds often have lesions on the feet or legs and these may appear as horny growths.

2. Diphtheritic lesions are raised, buff to yellow plaques on mucous membranes. They usually predominate in the mouth [Fig. 3; Fowl Pox; NCSU] but may be present in the sinuses, nasal cavity, conjunctiva, pharynx, larynx, trachea, or esophagus. Diphtheritic lesions often accompany cutaneous lesions but may occur alone in some birds.

3. Turkey pox [Fig. 4; Fowl Pox; UC Davis] has been observed in turkeys previously vaccinated with fowl pox vaccine. Occasional birds develop lesions on the conjunctiva, mouth, and upper digestive tract. Economic loss is often due to poor feed conversion.

4. Microscopically, epithelial hyperplasia with eosinophilic cytoplasmic inclusion bodies [Fig. 5; Fowl Pox; UC Davis] and surrounding inflammation are observed.
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DIAGNOSIS

1. Typical skin lesions are very suggestive of the disease. The diagnosis can be confirmed by demonstrating intracytoplasmic inclusion bodies in stained sections or in scrapings of the lesions.

2. Typical skin lesions can be reproduced in a susceptible bird of the same species. Ground lesion material should be inoculated into scarified skin or empty feather follicles and should produce a typical pox "take" at the application site in about 5-7 days.

3. Virus-containing lesion material will produce pocks on the dropped chorioallantoic membrane of embryonated chicken eggs. The lesions contain typical intracytoplasmic inclusion bodies.

4. Some poxvirus strains, particularly turkey pox, may not have demonstrable inclusion bodies in tissue sections. Electron microscopy may be helpful in these cases.

CONTROL

1. Pox can be prevented in chickens, turkeys, pigeons, canaries, and quail by vaccination. Vaccination is usually done when the birds are 4 weeks of age but can be done at any age if necessary. Pullets should be vaccinated 1-2 months before production begins.

2. Chickens and pigeons usually are vaccinated by the wing web-stick method. An applicator with two slotted needles is dipped in vaccine and thrust through the wing web. Turkeys may be vaccinated by the wing web route but lesions may be transferred to the head from the vaccination site. Vaccination by a drumstick-stab method when 2-3 months old is the recommended route. Turkeys retained as breeders should be revaccinated.

3. Pigeon pox vaccine is now widely used in chickens either alone or in combination with fowl pox vaccine. Chickens purchased as replacements for layers should be revaccinated if the initial vaccination occurs prior to 10 weeks of age. Pigeon pox vaccine can cause severe reactions in pigeons if not applied properly.

4. Turkeys are usually vaccinated with fowl pox vaccine. Turkey pox, quail pox, and canary pox vaccines are commercially available when circumstances indicate that these strains are the causative agents. Fowl and pigeon pox vaccines are not cross-protective with these strains. Fowl pox vaccine should not be used to vaccinate pigeons.

5. Vaccination produces a small lesion ("take") at the site of vaccination. A generous sample of the birds should be examined for vaccination lesions about 5-7 days after vaccination. Takes caused by turkey pox vaccine generally appear later (8-10 days after vaccination) than those caused by fowl pox. A large percent of those birds should have takes or revaccination is necessary.

6. Broilers are not vaccinated unless there is pox in the area. Broilers may be vaccinated with a mild tissue culture fowl pox vaccine administered subcutaneously at 1 day of age. This vaccine does not produce a visible take, but may result in a small number of birds that exhibit central nervous system (CNS) signs at 4-12 days postvaccination.  *In-ovo* injection of this vaccine may magnify the number of chicks exhibiting CNS reactions.

7. Control cannibalism with proper beak trimming and reduced environmental light intensity.

8. Fowl pox is currently being employed as a vector for recombinant vaccines.

TREATMENT

There is no satisfactory treatment for pox.