Review article

Zoonotic Diseases from Poultry

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Abstract

A wide variety of zoonotic disease risks exists in poultry. Popularity of backyard and smaller production flocks is increasing, as is public concern about global poultry disease events. This paper discusses several of the more common poultry zoonotic diseases, their prevalence and presentation in both poultry and humans.


Introduction

As veterinarians with multi-species training, when we hear the word “zoonosis”, we automatically think about dogs with rabies, calves with ringworm, and puppies with roundworms. But what about poultry? What are the zoonotic diseases of poultry? The recent cases of H7N9 avian influenza moving from poultry into people makes us think about chickens and public health. Also, the increasing popularity of “urban chickens”, or backyard poultry, means that many clinics and laboratories may be presented with sick birds and need to advise clients on potential human diseases that could be transmitted from poultry or their products.

This paper will serve as a review of the zoonotic disease problems that poultry present. In general, there are two ways that humans can get diseases from poultry. One is being around the live birds and the other is foodborne, through exposure to, or consuming meat or egg products from the infected birds.

What are the zoonotic threats from live chickens?

Avian influenza is at the top of the list of zoonotic threats, if only because of serious global zoonotic events over the last decade. You would have to be living in a cave not to have seen headlines about “bird flu” and the people it has killed. So what is it, how does it affect birds, and how does it get into humans and cause disease? Here are the basics...

Influenza viruses have a complicated classification system that many think of as an alphabet soup. They are first divided into A, B, or C influenza. The Type A category contains most of the human influenza viruses and is also the only one seen in most domestic animals, including birds. All influenza A viruses have two prominent proteins – hemagglutinin (H) and neuraminidase (N). There are 17 varieties of the hemagglutinin protein and 9 varieties of the neuraminidase protein. As a result, all influenza A viruses are categorized according to which type of H and which type of N, resulting in the H#N# designation, or subtype. In contrast to other classes of animals, birds can be infected with all possible combinations H and N, or a total of 153 influenza A subtypes. Most of these viruses do not cause any kind of disease in the birds and so are referred to as “low pathogenic avian influenza” (LPAI) strains. Waterfowl, especially, are known to allow replication of numerous types of avian influenza viruses in their intestinal tract and
these subsequently become disseminated as they fly and defecate in the air. Some of these “low pathogenic influenza” viruses can infect poultry below and within poultry they have been known to mutate to become virulent and so a “low pathogenic avian influenza virus” can change to a “highly pathogenic avian influenza virus” (HPAI) and cause disease. To date, all of these highly pathogenic viruses have been of the H5 or H7 subtypes. As a result, all H5 or H7 viruses detected in birds are considered “notifiable” because of the possibility that they will mutate and become virulent and kill poultry.

Outbreaks of HPAI in poultry are seen sporadically. Infected poultry will die rapidly, often within 48-72 hours of infection. Transmission is extensive and an entire flock will become diseased and die. The pathogenesis involves infection of capillaries with resulting edema and hemorrhage in multiple areas, soon followed by multi-organ failure (Figure 1).

Human influenza viruses have historically been of the H1, H2, or H3 subtypes. Consequently, when an H5N1 was isolated from a sick boy in Hong Kong in 1997, the world took note. H5 subtypes are not supposed to infect humans, they are bird viruses! This, or a related virus, subsequently spread through poultry throughout Asia, much of Africa, and eventually Europe. There were more than 600 people infected, with a 59% mortality rate (49). Most infected people had close contact with poultry or were family members with intimate contact with sick people. The disease in humans was similar to the disease in animals, with multi-organ system failure. In humans, there was often a terminal Acute Respiratory Distress Syndrome and respiratory failure. H5N1 continues to circulate, but at a very low level, as international efforts have been largely successful in containing the spread. It is worth noting that this H5N1 virus has never occurred in the Americas.

Other avian influenza virus subtypes, in addition to H5N1, have been known to infect humans (27, 37). The H7 subtype has done so on numerous occasions. In 2002, in the Netherlands, H7N7 virus caused massive poultry morbidity, mortality, and culling and resulted in 80 human infections, including one fatality (13). H7N3 subtypes have occurred in Chile, two different parts of Canada, and Mexico, with some human infections documented (4, 5, 22, 44). In all of these, with the exception of the one fatality in the Netherlands, the predominant clinical presentation in the human patients was conjunctivitis. All were classified as HPAI, meaning that it was very virulent for poultry.

Additionally, the H9 subtype, on rare occasions, has infected humans with some respiratory impairment, but no deaths (30, 38). These viruses were of the LPAI variety.

Of concern now is the recently diagnosed LPAI H7N9, which, in just the first month of its discovery in China, caused 129 human illnesses, with an 18% mortality rate (23). This particular strain is LPAI, meaning there are no apparent clinical signs in birds, so it is difficult to know which birds are infected. Cases in humans were recorded from several provinces and most cases had some contact with poultry. Closure of the live bird markets decreased the rate of development of new cases.

**Newcastle Disease**

After HPAI, the other very serious disease of poultry that strikes fear into poultry owners and regulators globally is Newcastle disease. This disease too has a zoonotic potential, but is much less severe in humans than its cousin, avian influenza.

Newcastle disease is second only to avian influenza in its tremendous impact on poultry health. The highly variable single stranded RNA virus, classified by pathotypes, presents a constant threat in endemic areas and an ongoing challenge and economic cost even in developed poultry industries. Within the one serotype, pathotypes of Newcastle disease based on clinical signs and lesions vary in virulence and severity from mild subclinical infections to velogenic strains where mortality approaches 100% (9). (Figure 2).

It is thought that nearly every species of bird is susceptible and 241 susceptible species have been documented (26). Wild birds, game birds, and the spread of the virus via movement of people and equipment represent significant exposure risks for domestic poultry.

Newcastle disease virus was first documented as a human zoonosis many decades ago. While mild, self-limiting infections in poultry workers and researchers are occasionally documented, fortunately the virus does not represent a significant or severe threat to human health and human-to-human spread has never been reported. Clinical infections in humans usually result from accidental exposure to vaccines or fluids from infected birds or carcasses. Commonly, conjunctivitis develops within 24 hours and is sometimes accompanied by lacrimation, palpebral edema, pain, and, rarely, fever, chills, depressed appetite and photophobia (45). Recently, a case of severe interstitial pneumonia was attributed to this virus in a transplant patient; severe immunosuppression may allow for human infection (18).

Newcastle disease virus may also yield a future benefit to human health as it is the subject of much current research centered on its ability to induce apoptosis in several types of human cancer cells, including prostate and breast cancer (17, 42). It is hoped that further research with Newcastle disease virus will be able to exploit its oncolytic ability for human tumor immunotherapy (51).

**Avian tuberculosis**

Birds get mycobacteria, can it move to humans? The answer is yes, but not very often.

*Mycobacterium avium* infections are found worldwide in a myriad of birds, from poultry and game birds to wild and zoological species. In poultry, avian tuberculosis, caused by *M. avium* serovars 1, 2, and 3, is
most often encountered in mature backyard birds and rarely seen today in commercial flocks due to modern management practices (11).

Figure 1. Chicken infected with HPAI H5N1. There is extensive edema of the tissues of the neck.

Figure 2. Newcastle disease. A virulent strain has caused hemorrhage and necrosis in the conjunctival lymphoid tissue of a chicken.

In birds, the bacteria first form yellow to white tubercles in the small intestine and later spread to other organs, particularly liver, spleen and bone marrow. The resulting progressive granulomatous disease leads to emaciation and eventual death. The durable acid-fast bacilli are spread via the fecal-oral route when infected birds contaminate the environment through the shedding of tubercle bacilli.

In humans, infection with *M. avium* from birds is extremely rare in immunocompetent individuals and most often occurs in immunocompromised patients, such as AIDS/HIV patients (7, 47). The majority of serovars isolated in humans (both immunocompromised and immunocompetent) do not include serovar 2, the most common isolate in poultry (15).

Figure 3. Colibacillosis in a chicken. Avian pathogenic *E. coli* can create an extraintestinal infection in multiple organs. Here, there is polyserositis.

And here are two diseases most commonly acquired from turkeys

Ornithosis

*Chlamydophila psittaci* infections occur in more than 400 species of birds, often in an unapparent manner (3). In poultry, a well-defined clinical disease syndrome is recognized only in turkeys, where it presents as a mild respiratory infection. In other poultry species, the disease may be unapparent because there are several genotypes of *C. psittaci*, which tend to infect only certain species and these species-specific genotypes vary in their virulence for birds as well as humans.

There are numerous reports of humans obtaining the infections from turkeys. Infection, of either turkeys or humans, is through inhalation of contaminated aerosols. Infected birds, whether clinically ill or asymptomatic, will shed the organism in respiratory and ocular secretions, and feces. The organism survives drying and will become aerosolized when environmental conditions allow. Once in the human, the organisms infect epithelial cells of the respiratory tract and can then enter macrophages to disseminate through the body and create extensive necrotizing and inflammatory lesions in visceral organs such as the liver. Data from slaughter plants indicate a very high rate of human infection, with up to 70% of turkey plant employees being seropositive (10). Areas of greatest infection/exposure are in the receiving rooms (live birds) and also in the evisceration stations. This is probably due
to secretions from live birds and exposure to air sacs and respiratory tree at the evisceration stations.

Studies from Belgium have indicated a high rate of positivity in chicken broilers as well; although, there seems to be less transmission to humans from broilers, probably because of a difference in genotypes present (10). In France, there have been documented cases of severe clinical disease in workers at duck farms with no evidence of disease in any of the ducks. Additionally, seropositivity of ducks was very low and yet they were intense shedders of *C. psittaci* (28).

**Erysipelas**

The word “erysipelas” comes from the Greek, meaning “red skin”. This term has a very different meaning in human and veterinary medicine and so creates considerable confusion when talking about zoonotic diseases. In animals, erysipelas means disease due to *Erysipelothrix rhusiopathiae* and is seen most commonly in pigs and poultry. But in humans, erysipelas means skin rash and systemic infection due to streptococcal bacteria. However, humans can be infected with *E. rhusiopathiae*, and this infection in people is called “erysipeloid”. So, *E. rhusiopathiae* in animals is erysipelas and *E. rhusiopathiae* in humans is erysipeloid.

Erysipelas in poultry is primarily seen in turkeys where it causes a septicemia with extensive hemorrhages in multiple tissues, especially muscle, and fibrin thrombi, which clog the liver creating hepatic necrosis. There is cyanosis of the skin; hence, the infection in turkeys is commonly known as “blue comb.” Outbreaks have been recorded rarely in chickens, ducks and quail (33, 34, 48). Pathogenesis of the organism is related to its ability to spread through tissue and evade phagocytosis (48).

In humans, erysipeloid is most frequently seen in people who handle fish as the organism tends to be abundant in fish slime and so it may often be called “fish handler’s disease.” It is also known to occur regularly in abattoir workers, and there have been many cases associated with poultry plant personnel. It presents as a sharply defined, gradually spreading, oozing, painful swelling surrounding a pre-existing skin lesion. The organism enters most commonly through skin wounds and creates a local infection that may spread to the draining lymph nodes and create a lymphadenopathy (48). Infection is usually self-limiting, although penicillin is curative. Occasionally, the infection in humans may become septicemic and, if it infects the heart valves, the mortality rate is 38% even with antibiotic therapy (20).

**What are the zoonotic threats from poultry products?**

**Salmonella**

To many, *Salmonella* is synonymous with “chicken”. Chickens can be infected with numerous different serovars of *Salmonella*, most of which will cause disease in humans. Some serovars, such as *S. pullorum* and *S. gallinarum* are specific for chickens and will cause disease in chickens but are not infectious for humans. There are many others which will effectively colonize the intestinal tract or reproductive tract of chickens and do not cause any untoward effects in the bird, yet be shed into feces or eggs and cause serious human disease.

For instance, *Salmonella typhimurium* and *Salmonella Heidelberg* can infect chickens without causing any apparent disease and spread to other hosts, including humans, to cause disease. Problems occur at slaughter when materials from the digestive tract end up contaminating carcasses. Then, inadequate hygiene measures in the home during food preparation will result in transmission to humans and serious disease. In a national study examining chicken carcasses in 15 different Brazilian cities, 2.7% of carcasses harbored *Salmonella* spp. Eighteen different zoonotic serotypes were identified. Of particular concern was that 53% of the strains cultured were resistant to one or more antibiotics (31). Turkeys can also harbor these human-disease-producing salmonellas, but the prevalence of infection is much lower (12).

And what about eggs? The chicken female reproductive tract can be infected with *Salmonella enteritidis* and the hen will not show any clinical signs, but the organism will be shed into the eggs and will cause disease in humans. Thorough cooking of eggs kills the organism. Human disease problems arise with uncooked egg products in food, for example Caesar salad dressing, or egg nog.

Once in the human gastrointestinal tract, *Salmonella* bacteria have a survival strategy that allows them to persist, enter epithelial cells, and evade host destruction. The organism stimulates a secretory diarrhea and also destroys epithelium, creating ulcers and an effusive diarrhea. The endotoxin on *Salmonella* results in cytokine activation and can lead to septic shock (39).

All of these are food-borne problems. But *Salmonella* infections, and even extensive outbreaks, have been recorded due to contact with live chickens, especially from mail-order hatcheries (16).

**Campylobacter**

The winner in this category of foodborne illness, in terms of overall number of cases, is definitely *Campylobacter*. Campylobacteriosis in humans, most often caused by *Campylobacter jejuni*, is the leading cause of bacterial diarrhea in developed countries and a leading cause of diarrhea in children under the age of 5 years in developing countries (50). Estimated annual cases in the US alone range from 850,000 (40) to more than 2 million (14) and worldwide over 70% of cases are linked to consumption of chicken meat (2). Cooking will kill the organism but the problem arises with poor food
preparation practices and the co-mingling of uncooked poultry meat with food that will be consumed fresh. People contract the usually self-limiting, sporadic gastroenteritis within 2 to 5 days of ingesting the organism and the abdominal cramping, watery to bloody diarrhea, and occasional fever may last anywhere from a day to a week. Rarely, death (usually in immunocompromised individuals) or grave sequelae such as Guillain-Barré syndrome, reactive arthritis, nephritis, myocarditis, pancreatitis or septic abortion may occur following infection. Guillain-Barré, in which an auto-immune response results in paralysis, is thought to occur in roughly 1 out of 1,000 reported Campylobacter infections (1). Disturbingly, increasing antibiotic resistance has been documented in Campylobacter; demonstrated resistance to fluoroquinolones led to the recent prohibition of their use in the US poultry industry (43).

Once associated with vibrionic hepatitis in laying hens (36), today campylobacteriosis is a clinically unapparent condition in poultry. The organisms are common inhabitants of avian digestive tracts and poultry may support extensive colonization without any adverse effects (6). Once present in a house, the bacteria spread quickly, infecting more than 90% of birds within 2 weeks (8). The ubiquity of the bacteria in the environment makes eradication and prevention of infection at the farm level nearly impossible. During processing, one infected carcass can easily contaminate the entire production line and thus, much of the control measures in place focus on this stage of production.

**E.coli**

*Escherichia coli* is a commensal of birds and mammals, including humans, worldwide and the beneficial nonpathogenic strains far outnumber pathogenic ones. Avian colibacillosis, occurring most commonly in chickens, turkeys, and ducks, may be localized or systemic and is caused by opportunistic virulent strains, most of which are extraintestinal *E.coli* (25). Disease often occurs due to poor management, poor sanitation, or as a secondary infection following respiratory or immunosuppressive disease. Avian colibacillosis is the most common bacterial disease of poultry and one of considerable economic concern worldwide. In addition to outright morbidity and mortality, colibacillosis and colisepticemia are responsible for substantial condemnation losses at the processing plant.

Avian pathogenic *E.coli* are most commonly serotypes O1, O2, and O178, as classified by the O antigen on the antigenic portion of the LPS molecule (19). Fecal contamination, especially from rodents or wild birds, may introduce pathogenic strains into a flock. Contaminated water or feed and insect vectors are other sources of exposure. Following infection via transmucosal or transdermal routes, the bacteria can cause a wide range of disease processes, usually named for the affected tissue (e.g., coliform omphalitis, cellulitis, salpingoperitonitis, airsaculitis, etc.), depending on condition of the bird and/or other concurrent infections. (Figure 3).

Although most strains affecting poultry are species-specific, poultry are experimentally susceptible to the human enterohemorrhagic *E.coli* O157:H7 and may shed it for months (41). More commonly found in ruminants, this strain associated with diarrheal disease outbreaks in humans has also been isolated in turkeys (21). Research has also shown that avian pathogenic *E.coli* in poultry, especially in pigeons but in chickens as well, may carry some genes shared by Shiga toxin *E. coli* responsible for disease in humans (35). Perhaps most concerning to human health is not the threat of direct infection from poultry sources but the fact that strains of avian pathogenic *E.coli* share similar serotypes, virulence factors, and genes and plasmids for antibiotic resistance with human strains (29, 32). Strains carrying virulent and antibiotic resistance factors similar to those that cause cystitis and meningitis in people have been isolated in retail poultry products (24). Avian pathogenic *E.coli* strains have shown the ability to rapidly acquire antibiotic resistance (when exposed to antibiotics) and, alarmingly, are capable of transmitting these resistant clones and resistance plasmids directly to poultry workers (46).

**Conclusion**

Although infections in humans are relatively rare; there are substantial zoonotic risks from poultry and poultry products that veterinarians and physicians alike should be aware of and keep in mind in their daily practice. Global outbreaks of poultry disease will likely continue to be headline news and raising small flocks of backyard birds continues to grow in popularity alongside the locavore and slow food movements. Whether presented with a beloved pet chicken or simply questioned about a current event, veterinarians need to be prepared to counsel and educate the public on the myths and realities regarding the risks of poultry zoonoses.

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