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H5N1 Outbreaks and Enzootic Influenza

Robert G. Webster,*† Malik Peiris,†‡ Honglin Chen,†‡ and Yi Guan†‡
*St. Jude Children's Research Hospital, Memphis, Tennessee, USA; †University of Hong Kong, Hong Kong, SAR, China; and ‡Shantou University Medical College, Shantou, Guangdong, China

ABSTRACT

Ongoing outbreaks of H5N1 avian influenza in migratory waterfowl, domestic poultry, and humans in Asia during the summer of 2005 present a continuing, protean pandemic threat. We review the zoonotic source of highly pathogenic H5N1 viruses and their genesis from their natural reservoirs. The acquisition of novel traits, including lethality to waterfowl, ferrets, felids, and humans, indicates an expanding host range. The natural selection of non-pathogenic viruses from heterogeneous subpopulations co-circulating in ducks contributes to the spread of H5N1 in Asia. Transmission of highly pathogenic H5N1 from domestic poultry back to migratory waterfowl in western China has increased the geographic spread. The spread of H5N1 and its likely reintroduction to domestic poultry increase the need for good agricultural vaccines. In fact, the root cause of the continuing H5N1 pandemic threat may be the way the pathogenicity of H5N1 viruses is masked by co-circulating influenza viruses or bad agricultural vaccines.

INTRODUCTION

Influenza is an ancient disease that has infected humans at irregular intervals throughout recorded history (1). While the 1918 "Spanish" influenza is the best recorded catastrophic influenza pandemic, similarly severe pandemics occurred earlier, when the human population of the world was much smaller, and they will occur again. Our challenge is to understand all aspects of the influenza virus, the hosts and their response, and the virus global impact so that we may be better prepared to face the inevitable next influenza pandemic.

The influenza virus that appears most threatening is the avian H5N1 strain that since 2003 has infected >130 persons in Vietnam, Thailand, and Cambodia and has killed more than half of them. Nonetheless, the H5N1 influenza threat is viewed with disturbing complacency; a frequently heard statement is "since the virus has not adapted to continuing human-to-human transmission by now, it is unlikely to do so in the future." Such complacency is akin to living on a geologic fault line and failing to take precautions against earthquakes and tsunamis.

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THE SOURCE

Influenza A viruses are perpetuated in the wild birds of the world, predominantly in waterfowl, in which the 16 subtypes (which differ by 30% in their hemagglutinin [HA] nucleotide homology) coexist in perfect harmony with their hosts (2,3) (Figure 1).

In these natural hosts, the viruses remain in evolutionary stasis, showing minimal evolution at the amino acid level over extended periods. This fact indicates that the influenza-bird association is ancient; this lack of change is surprising because influenza viruses are segmented, negative-stranded RNA viruses that have no quality-control mechanisms during replication and are highly prone to variation. After transfer to a new type of host, either avian or mammalian, influenza viruses undergo rapid evolution. However, all 16 HA subtypes, including H5 and H7, have until recently been considered to be benign in their natural hosts. This benign equilibrium between the influenza virus and its host may have changed.

GENESIS OF H5N1 VIRUS

Before 1997, no evidence had indicated that H5 influenza viruses could infect humans and cause fatal disease. The H7 influenza viruses were known to cause conjunctivitis in humans, and serologic studies provided evidence of sub-clinical human infection with the subtypes prevalent in avian live poultry markets (4). The precursor of the H5N1 influenza virus that spread to humans in 1997 was first detected in Guangdong, China, in 1996, when it caused a moderate number of deaths in geese and attracted very little attention (5). This goose virus acquired internal gene segments from influenza viruses later found in quail (A/Quail/HK/G1/97 [H9N2]) and also acquired the neuraminidase gene segment from a duck virus (A/Teal/HK/W312/97 [H6N1]) before the goose virus became widespread in live poultry markets in Hong Kong and killed 6 of 18 infected persons (6,7). This H5N1 virus was eradicated by culling all domestic poultry in Hong Kong, and the genotype has not been detected since that time. However, different re-assortants continued to emerge from goose and duck reservoirs (8) that contained the same H5 HA glycoprotein but had various internal genes. The H5N1 viruses continued to evolve, and in late 2002, a single genotype was responsible for killing most wild, domestic, and exotic waterfowl in Hong Kong nature parks (9,10). This genotype of H5N1 spread to humans in Hong Kong in February 2002, killing 1 of 2 infected persons (11), and was the precursor of the Z genotype that became dominant. The Z genotype spread in an unprecedented fashion across Southeast Asia, affecting Vietnam, Thailand, Indonesia, Cambodia, Laos, Korea, Japan, China, and later Malaysia. Further analysis showed that the H5N1 influenza viruses that caused outbreaks in poultry in Japan and Korea were genetically different from those in the other countries (the V genotype) (12,13). The phylogeny of the recent Z genotype viruses showed that viruses isolated in Vietnam and Thailand formed a cluster that remained distinct from those isolated in Indonesia. To date, >140 million domesticated birds have been killed by the virus or culled to stem its spread; as of December 2005, >130 persons have been infected in Vietnam, Thailand, Indonesia, Cambodia, and China, and 70 have died (42 in Vietnam, 14 in Thailand, 8 in Indonesia, 4 in Cambodia, and 2 in China).

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These recent H5N1 influenza viruses have acquired the unprecedented and disturbing capability to infect humans; to cause neurotropic disease and a high proportion of deaths in waterfowl in nature; to cause death in and be transmitted among felid species, including domestic cats (14); and to cause neurotropic disease and death in ferrets and mice (15). These incremental changes intensify concern about this H5N1 virus pandemic potential.

These traits are likely to have been acquired initially by re-assortment in 2001 and 2002, when a plethora of different genotypes were detected in poultry markets and later in farms in Hong Kong (13). These genes were presumably acquired from viruses found in waterfowl in Southeast Asia, but the actual gene donors have not yet been identified. Since late 2002, the Z genotype has become dominant, but phylogenetically distinguishable viruses have continued to co-circulate in Indonesia and western China. These characteristics have been acquired mainly through mutations in the RNA polymerase (PB2) gene, insertions in the HA gene, and deletions in the NA and non-structural (NS) genes. Thus, the H5N1 viruses continue to evolve, initially by re-assortment and more recently by mutation and deletion (16,17). While most H5N1 influenza viruses isolated from avian species in Asia since 1997 are highly pathogenic in gallinaceous poultry, they show heterogeneous pathogenicity in other species. In domestic ducks, the pathogenicity of the H5N1 viruses varies from high to non-pathogenic. In ferrets, most avian isolates replicate and cause respiratory tract infection, while a few strains are highly pathogenic and neurotropic (causing hind leg paralysis), and the virus has been isolated from the brain (15). In contrast, all isolates from humans are highly pathogenic to ferrets. A similar pattern is found in experimental infection of mice, in which most avian isolates cause respiratory infection.

Figure 1. Emergence of H5N1 influenza virus and control options. A nonpathogenic H5 influenza virus is believed to have spread...

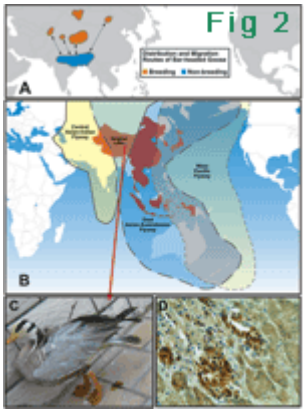

Figure 2. Migration routes of Asian birds. A) Distribution and migration routes of bar-headed geese (courtesy of P. Leader)...

MECHANISM OF SPREAD

Were the highly pathogenic H5N1 viruses transferred within and between countries by persons, poultry, or fomites? In previous outbreaks of highly pathogenic H5 and H7 infection in multiple countries, the spread was directly attributable to humans. The main way influenza virus is spread in poultry is by movement of poultry and poultry products; establishing good bio-security measures on poultry farms is therefore an important defense. The poultry industry is a huge, integrated complex in Asia, and a number of firms have branches in China, Vietnam, Thailand, and Indonesia.

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Nonetheless, the involvement of multiple lineages of H5N1 argues against human-mediated spread from a single source. Live poultry markets are an amplifier and reservoir of infection (18) and probably play a role in the maintenance and spread of the virus in the region.

However, a number of other factors unique to affected Asian countries make control difficult. Backyard flocks are common in the region, and these domesticated birds are not subject to any bio-security measures.

Fighting cocks are prized possessions and are often transported long distances. Fighting cocks may also play a role in the spread of infection and in transmission to humans.

Many of the affected countries have a weak veterinary infrastructure and are facing highly pathogenic avian influenza outbreaks for the first time. The migrant ducks that commonly wander through rice fields scavenging fallen rice seeds are another potent mechanism for the spread of infection.

Role of Domestic Ducks

After late 2002, when H5N1 viruses had killed waterfowl in Kowloon Park in Hong Kong, most avian H5N1 isolates isolated in Vietnam, Thailand, and Indonesia were highly pathogenic to chickens and domestic ducks. However, by late 2003 and early 2004, some avian isolates were non-pathogenic to ducks but retained their pathogenicity to chickens (19). Genetic analysis of these isolates showed evidence of multiple variants within single specimens (20). On Madin-Darby canine kidney (MDCK) cells, these viruses formed a mixture of small and large plaques that had different biologic properties. Viruses that formed large plaques were usually highly pathogenic to ducks and ferrets, whereas viruses that formed small plaques were usually non-pathogenic to both birds and ferrets. Some virus isolates formed small plaques that were pathogenic to ducks. Thus, plaque size was not a marker of pathogenicity. When ducks were orally infected with the original mixed population of H5N1 viruses, most birds died, but some excreted virus for an extended period (up to 17 days); during this time, viruses that were non-pathogenic to ducks were selected. Serologic testing of these ducks showed hemagglutination inhibition (HI) and neutralizing antibodies against the original dominant virus in the mixture; thus, immune clearance had caused the selection of the minor variants. The viruses shed on day 17 had become non-pathogenic to ducks, although they remained highly pathogenic to chickens. Sequence analysis of the HA showed that these viruses differed from the original dominant virus at multiple amino acids and were antigenically distinguishable in HI tests. Therefore, H5N1 viruses circulating in avian populations in Southeast Asia are clearly heterogeneous. Notably, this phenomenon has repeatedly been reported for other influenza viruses that are in the process of altering their interspecies transmission, including European avian H1N1 viruses that were transmitted to pigs (21), H9N2 viruses that were transmitted to pigs and humans, and now H5N1 viruses that are transmitted from ducks to humans. How these mixtures of co-dominant viruses are generated in a quasi-species is unresolved. Suggested mechanisms include mutator mutations or partial heterozygotes, but a satisfactory explanation is not available (22).

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A subdominant population of H5N1 viruses is presumably selected in ducks after the immune response clears the dominant virus. The subdominant population appears to be uniformly non-pathogenic to ducks, as if this is the natural situation for influenza in the duck. Whether further selection will occur against the polybasic cleavage site in the HA and the pathogenicity-determining sites in PB2 and NS remains to be seen.

These viruses loss of pathogenicity to ducks, but retention of pathogenicity to chickens and presumably to humans, has been a problem associated with their eradication.

In Vietnam, for example, disease signs were used as the criteria for identifying H5N1 infection in ducks. Thus, the duck has become the Trojan horse of highly pathogenic H5N1 influenza in Asia (20).

Role of Migratory Birds

Migratory waterfowl are generally believed to be the main reservoir of all 16 subtypes of influenza A viruses, including H5 and H7 subtypes. However, less agreement is found regarding the role of migratory waterfowl in the initial spread of highly pathogenic H5N1 viruses across eastern Asia in 2003. The isolation of highly pathogenic H5N1 from herons, egrets, and peregrine falcons in Hong Kong in 2003 and 2004 leaves no doubt that wild migratory birds can be infected and may spread disease to local poultry flocks. The outbreak in Qinghai Lake (16,17) proves that these highly pathogenic H5N1 influenza viruses are transmissible among migratory waterfowl. The migration route of shorebirds in the east Asian-Australasian flyway does overlap the areas that have had H5N1 outbreaks, although the virus has been notably absent in Taiwan, Malaysia (except for occasional outbreaks near the Thai border), and western Australia (Figure 2). The role of migratory birds in the transmission and spread of highly pathogenic H5N1 viruses is still unclear. However, the recent outbreak of H5N1 infection in bar-headed geese and other species in Qinghai Lake is a cause for concern because these birds migrate southward to the Indian subcontinent, an area that has apparently not been affected by H5N1 avian influenza. If the virus were to become entrenched in India, its geographic range would be substantially extended, and the pandemic threat would increase accordingly (17). A mutation in the PB2 gene (residue E627K) associated with pathogenicity in mammals (16,17) has been found in viruses isolated from birds in Qinghai Lake; this finding has caused concern that this mutation will be transferred to other migratory birds (e.g., wild ducks) and will be spread because not all infected birds die.

Although culling domestic poultry to contain the spread of highly pathogenic H5N1 virus is considered an acceptable agricultural practice, culling migratory birds is not acceptable to any international authority (Food and Agriculture Organization of the United Nations [FAO], the World Organization for Animal Health [OIE], the World Health Organization [WHO]). The idea of culling migratory birds must be strongly discouraged, for it could have unknown ecologic consequences. Instead, since highly pathogenic H5N1 has been demonstrated in migratory birds, the poultry industries of the world must adapt measures such as increased bio-security (Figure 1), the use of vaccines, or both.

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Early detection and aggressive control measures allowed Japan, South Korea, and Malaysia to eradicate H5N1 virus soon after its introduction into those countries poultry flocks, demonstrating that rapid and determined responses can keep the virus from gaining a foothold. In other countries in Asia, delayed detection and response caused the virus to become entrenched across a wide region, and eradication at this stage has become a formidable undertaking.

AGRICULTURAL VACCINES

The need for H5N1 vaccines for domestic poultry is increasing. Adopting a policy to use vaccines in poultry is an important decision for agricultural authorities in countries such as Thailand (a major poultry exporter) and Vietnam. Both countries are investigating their specific needs. While considerable data exist on the efficacy of influenza vaccines in domestic chickens, little comparable information is available regarding ducks. The pros and cons of the use of vaccines in poultry have been reviewed (23).

Current technologies permit discrimination between vaccinated and naturally infected birds; however, vaccines are not standardized on the basis of antigen content. "Good" and "bad" agricultural vaccines are in use.

Good Agricultural Vaccines

Good agricultural vaccines provide protection from disease despite lack of a close antigenic match between the vaccine and circulating strain and reduce the virus load below the level of transmissibility. They do not provide sterilizing immunity: vaccinated birds may excrete low levels of virus after challenge infection. Sentinel unvaccinated birds are kept in each house to monitor for virus shedding, antigenic drift, or both.

Bad Agricultural Vaccines

Bad agricultural vaccines prevent disease signs but do not prevent shedding of transmissible levels of virus. They also promote undetected spread of virus on farms and to live poultry markets and promote antigenic drift. China and Indonesia have adopted poultry vaccination to control H5N1, and Vietnam has begun vaccine trials in poultry. However, the resurgence of H5N1 in Indonesian poultry and pigs (24) and the detection of H5N1 in apparently healthy birds in live poultry markets in China (17) suggest that some vaccines are of suboptimal quality or that co-infection masks disease. The adoption of a vaccine strategy for H5N2 virus in Mexico in the 1980s reduced disease signs but has not eliminated the H5N2 virus from the region; instead, vaccination may have contributed to the virus widespread presence in Central America and to its antigenic drift (25).

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H9N2 AND CROSS-PROTECTION

The clinical signs of infection with highly pathogenic H5N1 virus may be masked by cross-protection by other influenza subtypes, but this fact is often overlooked. During the initial outbreak of highly pathogenic H5N1 in Hong Kong in 1997, chickens in the live poultry markets exhibited no disease signs, yet samples from apparently healthy chickens, ducks, and quail showed highly pathogenic H5N1 in each of the poultry markets surveyed (26). Surveillance showed that multiple influenza subtypes were co-circulating, including 2 lineages of H9N2, the first represented by the G1 lineage (A/Quail/Hong Kong/G1/97 [H9N2]) and the other by G9 (A/Chicken/Hong Kong/G9/97 [H9N2]).

The G1 lineage has the same 6 internal gene segments as the index H5N1 human isolate (A/Hong Kong/156/97 [H5N1]) and is believed to have been the donor of these genes during re-assortment that produced the original H5N1 human strain in 1997 (27). In laboratory studies, chickens previously infected with H9N2 (A/Quail/Hong Kong/G1/97 [H9N2]) were protected from disease signs and death when challenged with highly pathogenic H5N1, but the chickens shed H5N1 virus in their feces (28). Further studies in inbred chickens established that the cross-protection was due to cell-mediated immunity and that it could be transferred by CD8+ T cells but not by antibodies (29). The possible effect of co-circulating influenza viruses on the pathogenicity of highly pathogenic H5N1 in Vietnam, Thailand, and elsewhere in Asia has not been resolved.

To date, no other subtypes of influenza A viruses have been reported in poultry in Vietnam or Thailand. Surveillance of live poultry in Hong Kong and in Nanchang (30) suggests that other influenza A viruses are co-circulating in live poultry markets and on duck farms. Definitive information is required to understand the ecology of influenza and the possible masking of disease signs caused by H5N1.

CONCLUSION

Conventional wisdom about pandemic influenza holds that a pandemic is inevitable and that the only question remaining is "When?" The H5N1 virus continues to evolve and spread, with additional human infections occurring in Vietnam, Cambodia, Indonesia, China, and Thailand. If this virus acquires human-to-human transmissibility with its present fatality rate of 50%, the resulting pandemic would be akin to a global tsunami. If it killed those infected at even a fraction of this rate, the results would be catastrophic. While the high pathogenicity of the Qinghai bar-headed goose isolate is a continuing threat to poultry and humans, perhaps the most insidious threat comes from unobserved transmission through wild and domestic ducks. The isolation of H5N1 virus from bar-headed geese in Qinghai Lake in southern China in 2005 originated from unobserved infection in poultry markets and suggests that highly pathogenic H5N1 viruses continue to circulate unseen among poultry in China (17). We cannot afford simply to hope that human-to-human spread of H5N1 will not happen and that, if it does, the pathogenicity of the virus will attenuate. Notably, the precursor of the severe acute respiratory syndrome (SARS)–associated corona virus (31) repeatedly crossed species barriers,

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probably for many years, before it finally acquired the capacity for human-to-human transmission, and its pathogenicity to humans was not attenuated. We cannot wait and allow nature to take its course. SARS was interrupted by early case detection and isolation, but influenza is transmissible early in the course of the disease and cannot be controlled by similar means. Just 1 year before the catastrophic tsunami of December 2004, Asian leaders rejected a proposed tsunami warning system for the Indian Ocean because it was too expensive and the risk was too remote. This mistake must not be repeated in relation to an H5N1 avian influenza pandemic. We must use this window of opportunity to prepare and to begin pre-pandemic implementation of prevention and control measures.

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Dr Webster is a professor in the Division of Virology, Department of Infectious Diseases at St. Jude Childrens Research Hospital. The major focus of his research concerns influenza viruses in wild aquatic birds and their role in the evolution of new pandemic strains for humans and animals.

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Editors Note:
Bird flu threatens to be a pandemic of huge magnitude. The only way to face it and hopefully defeat it is preparedness and information. In this context this article from the journal Emerging Infectious Diseases is reproduced.
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Article 7

Clinical Study on Hjarre's Disease in Poultry

K. A. Shah and S. Qureshi
Institute of Animal Health and Biological Products, Zakura, Srinagar
Department of Animal Husbandry, Kashmir

INTRODUCTION

Hijarre's Disease (Coligranuloma) is a disease of adult chicken and turkey, characterized by nodular granulomas in liver, mesentery and walls of intestine. It is caused by Escherichia Coli, a gram -ve , non-acid fast non-spore forming bacillus. Besides coligranuloma, E-coli has more oftenly been incriminated in number of other clinical conditions which include coli bacillosis, coli septicemia, egg peritonitis, arthritis and air saculitis etc. causing decrease in production high mortality rate and condemnation of carcass at slaughter, thereby resulting in major economic losses to the poultry Industry. Coli granuloma is relatively uncommon coliform disease and may cause mortality as high as 75% in an individual flock (calnek et. al 1991). Perusal of literature reveals very meagre information on the occurrence of disease. This communication probably constitutes first report on Hjarre's disease in Kashmir.

MATERIALS AND METHODS

Occurrence of sudden sporadic deaths among adult stock in an organized poultry farm farmed basis for this study. The affected flock was dull, depressed and anorectic, reared on deep litter and had been already vaccinated against new castle disease, Infectious bursal disease and Marek's disease as well. In order to elucidate cause of mortality, thorough necropsy examination of fresh carcasses was conducted. Morbid material was aseptically collected and subject to culture sensitivity test. Tissue smears were prepared by crushing individual nodules between two glass slides (Randall 1991) and stained with Ziehl-Neelsen staining method (Zahoor 2003).

RESULTS AND DISCUSSIONS

Pathological lessions detected at necropsy were pathognomic for coli granuloma. These included, coagulation necrosis and enlargement of liver, hard nodular granulomas in the mesentry, walls of intestine, particularly cecum congested and swollen spleen with abnormal contours, congested viscera with atrophied bursa normal peripheral nerves and bone marrow corroborating with reports of Calneck et. al 1991, Randall 1991 and Jordan 1990.

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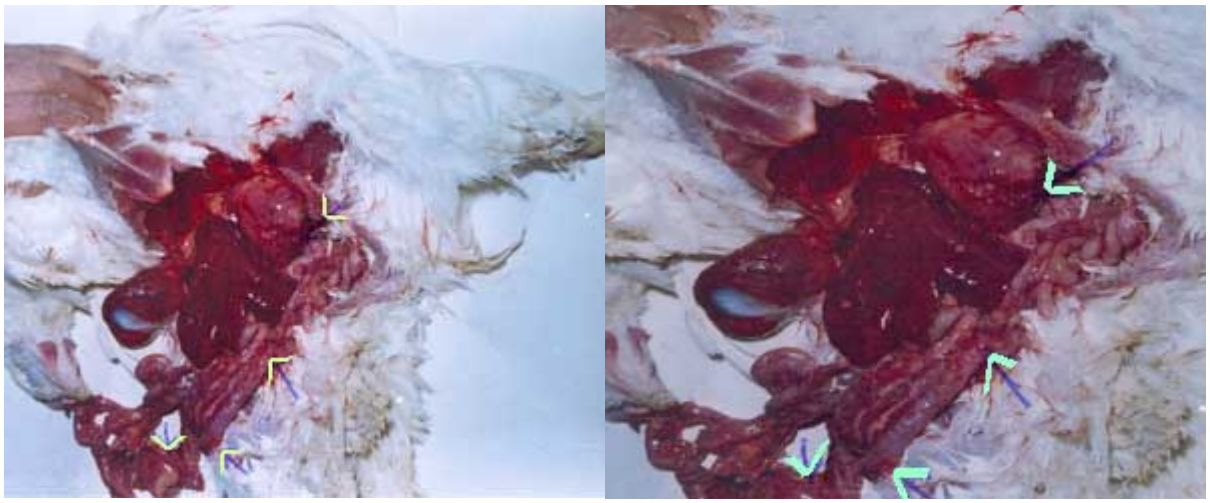

Moreover, tissue smears prepared from nodules did not reveal presence of any acid fast bacillus on Ziehl-Neelsen staining, thus ruled out the possibility of Avian tuberculosis. Bacteriological examination of morbid materials carried out in accordance to procedures of Edward and Ewing 1972 and Cruik Shank at. al. 1975 indicated infection with Escherichia coli. Isolate was found resistant to sulfha drugs, cephalaxine, ciprofloxacine, ampicillin and amoxycillin and sensitive to enrofloxacin furazolidons, sparfloxacin ofloxacin and gentamicin showing maximum zone of inhibition for enrofloxacin and minimum for gentamicin. E.coli was earlier isolated from coligranuloma and other coliform diseases in domestic birds by Gross (1961), Harry and Hemsley (1965), Bolin (1986), Jordan (1990) and Khursheed and Pampori (2003). Enrofloxacin 300 mg per litre of drinking water for 5 days supported with Homeopathic Therapy of 'Thuja' 200x four drops per litre of drinking water prepared in a quantity to be consumed within two hours in morning for twenty days controlled the disease effectively. Antibiotics were widely used to combat various types of coliform disease in birds and animals (Cloud et. al. (1985), Philphs and Case (1980), Khursheed and Pampori loc. cit.). Thuja was reported to be very effective in treatment of warts and tumorous/nodular growths in man (Shah 2003) and animals (Rai et. al. (1991) and Varshney and Paliwal (2000)).

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Article 8

Management of Recurrent Cervical Prolapse in a Pleuriparous Cow in Estrum- A Case report

Mujeeb-ur-Rehman Fazili
Assistant Professor, Division of Veterinary Surgery
Faculty of Veterinary Sciences and Animal Husbandry, Alusteng, Srinagar, Kashmir, India

INTRODUCTION

Typically, prolapse of the vagina is a condition of the ruminants in late gestation. Occasionally it is seen after parturition and rarely it occurs unconnected with pregnancy or parturition (Arthur et. al., 1993). In this paper attempt has been made to put on record a case of recurrent prolapse of cervix in a pleuriparous cow in estrum.

CASE HISTORY AND CLINICAL EXAMINATION

A crossbred Jersey cow aged thirteen years having calved tenth time eight months ago was presented with the history that some mass protruded through the vaginal orifice during recumbancy when the cow was in the heat a fortnight ago. The prolapse had been retained by vaginal sutures applied by a Para veterinarian. These sutures dislodged by the cow a week later and the prolapse recurred the next day. On clinical examination the animal had normal temperature, pulse and respiration. Its surface had only minor injuries. The animal was standing with little straining.

MANAGEMENT

Epidural anesthesia with six ml of 2% procaine hydrochloride (Novacaine) was achieved. The prolapsed mass was washed clean, dressed with povidone iodine (Wokadine) lotion and replaced. The replaced cervix was retained by the Buhnr's technique (Arthur et. al., 1993 and Roberts, 1982). Supportive therapy included administration of Thiacla, 450ml intravenously; 10ml each of pheneramine maleate (Avil), oxytetracycline hydrochloride (Wolicycline DS) and diclofenac sodium (Zobid) were given intramuscularly for three days. 10ml injectable phosphorus (Tonoricin) was given intramuscularly on third post-operative day. The owner was advised to stall the animal on a forward slope. On the next heat, two weeks later the animal showed recurrence of the prolapse. The Buhner's nylon tape was found intact yet whole of the cervix has prolapsed through it.

The replacement of the prolapse was effected with some difficulty under epidural anesthesia. Buhner's technique was supplemented by Caslick's vuvloplasty (Arthur loc. cit, Khar loc. cit). Antibiotic and anti-inflammatory therapy was continued for three days.

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RESULT AND DISCUSSION

The animal did not show recurrence of prolapse during six months of the observation period. The condition has been reported to occur in older cows and buffaloes after several calvings (Khar loc. cit.). it has been suggested that the anatomical anchorage of the genital tract is less efficient in affected animals (Arthur loc. cit). Deficiency of calcium and a disturbed calcium and phosphorus ration has been observed in both cows and buffaloes with antepartum prolapse of the vagina (Khar loc. cit). The ligamentous relaxation due to predominance of estrogens may increase the mobility of the vagina (Arthur loc. cit). The main ovarian hormones produced during proestrus and oestrus being estrogen might have lead to the prolapse. According to Arthur (loc. cit) Buhner's technique is the best means of retaining the replaced vagina. This technique along with calcium and phosphorus supplementation failed to give the desired effect in the present case where the prolapsed organ mainly involved cervix (but not vagina only) and had occurred unrelated to pregnancy or parturition. Therefore Caslick's operation had to be conducted additionally for achieving the desired effect.

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Article 9

Spirochaetosis- A Devastating Tick Borne Poultry Disease in the Trans Himalayas

Mir Afzal Haq Kanth
Livestock Development Officer
Department of Animal Husbandry, Kashmir

INTRODUCTION

Almost all the vertebrate animals, except the fishes somehow or the other are infested with ticks. The arthropods cause significant losses to the Indian GDP. These losses are attributed to the bites which cause among other things anemia, transmission of diseases such as viral, bacterial, rickettsial and protozoan. Spirochaetosis is one such disease in poultry caused by a soft tick called Argas persicus. This tick is a serious pest of the chicken and it causes a great loss to the poultry farmer. It is particularly problematic for the poor poultry farmers of the trans Himalayan belt comprising the hilly areas of Jammu & Kashmir, Himachal Pradesh, Arunachal Pradesh, Assam, parts of Uttar Pradesh, and also areas in Nepal, Bhutan and Burma where the use of timber and mud is in vogue during poultry house construction. This soft tick thrives in the cracks and crevices of these poultry houses and remains a constant irritant to the farmer.

BIONOMICS OF ARGAS PERSICUS

Its shape is oval. It is yellowish brown in color when unfed and slaty blue after engorging on blood of host. In these sexes are separate and the two sexes look almost identical. The female lays eggs in the cracks and crevices of the poultry house and under the bark of trees in batches of 100-250. Each female lays 4 to 7 batches of eggs in her lifetime. The eggs are small, spherical and brown in color. The six legged larvae hatch from the eggs in 10-21 days. These feed on the host and often remain attached for up to six days under their wings. They may also drop off and hide in the crevices. After around seven days they molt and become nymphs. There are two nymphal stages, each of which last for about two weeks. The ticks engorge once in the nymphal stage and then the 2nd nymph molts into the adult. The nymph and the adults hide in sheltered spots and attach with the hosts during the night, feeding for about two hours and causing restlessness and irritation to chickens. These ticks are very tenacious and can live for 3 to 5 years without feeding.

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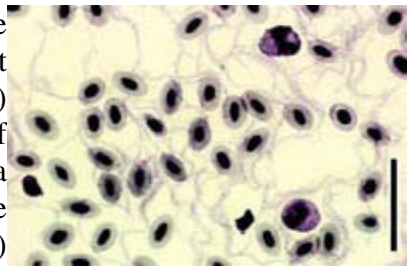
DISEASE TRANSMISSION



This Argas persicus (soft tick) is responsible for the transmission of one important and serious disease, almost ranking second to Newcastle Disease (Ranikhet Disease) only, called spirochaetosis. The causative agent of spirochaetosis is the spirochaete bacterium Borrelia anserinum (Borrelia gallinarum), an actively motile filamentous organism usually about 8 to 20 microns (µ) long.

CLINICAL SYMPTOMS

Following the bite of the soft tick there is an incubation period of 5 to 9 days. Death may take place after an acute illness lasting for 4 days or more. Chronic cases may last for a fortnight. The mortality rate is very high. Recovered birds may retain the infection Pyrexia, weakness, drowsiness, ruffling of feathers, anorexia, anaemia, diarrhea, intense thirst drop in egg production, the affected birds assume a crouching posture with the head hanging down and eyes closed, emaciation and finally paralysis spreading from the extremities. The disease has been found in chicken, turkeys, geese, ducks etc.

DIAGNOSIS

1. May be made by blood examination during the acute stage.

2. In acute cases spleen may be 4 or 5 times its normal size.

3. The liver is enlarged, fatty and sometimes shows necrotic foci.

4. The blood does not coagulate.

TREATMENT

(A)Curative

(i) Several arsenical preparations such as Soamin, Salvarsan, Atoxyl, Sulopharsenol etc. Sulpharsenol is injected by the intramuscular or intravenous route at a dose of 2 to 4 grams dissolved in 2 to 4ml of distilled water. Recovery is usually obtained after one injection.

(ii) Single dose of Penicillin usually affects a complete cure. 15000 I.U. of Penicillin subcutaneously is very effective.

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(B) Prophylactic

(i) Spirochaetosis freeze dried vaccine @ 1ml intramuscularly renders immunity for up to 18 months

(ii) Tick Control: The following measures are indicated:
(a)Practical Measures -Housing should be tick proof, without cracks and crevices. Important factor is to minimize the use of unprocessed timber. Also fumigation of the house be taken up at least twice a year.
-Quarantining of fresh arrivals till one is satisfied of their being tick free.
-Burning of weeds in farm premises.
-Rodents serve as mobile museums for the various stages of ticks. Rodent control will minimize chances of onward transmission to birds.
-For treatment of infected premises the birds must be removed from their run and houses to be placed in wooden crates in which the attached larvae will drop off within 10 days from their bodies. Meanwhile the houses will be cleaned an fumigated efficiently. The waste material is best burned, while the wooden crates could be sprayed clean. Various acaricides are available to this end.
(b) Research Trends:
Use of repellents (dimethyl phthalate, diethyl tohimide etc.)
Use of hormones.
Irradiation techniques.
Biological control.
Male sterile techniques.
Genetic control.
Host resistance.

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Article 10

Drug Resistance

Aijaz Ahmad Banday
BVSc & AH, MVSc (Veterinary Microbiology)

INTRODUCTION

The most serious current problem in relation to the use of anti microbial drugs in the control of infectious diseases is the increasing frequency with which bacterial resistance to some of these substances has developed. In practice this means that the treatment of specific infection with an antibiotic agent known to be potentially active against a particular microbial species may be ineffective when used for the treatment of certain outbreaks of that infection.

HOW DRUG RESISTANCE IS DEVELOPING

Detailed investigations have revealed that the property of multiple drug resistance can be transferred from the resistant strains of Escherechia to Shigella in the intestinal tract of the patients as well as in vitro under laboratory conditions. Further investigations have shown that the transfer of multiple drug resistance can take place among the majority of genera of family Enterobacteraciae as well as in certain other genera. These observations as well as epidemiological aspects of this problem have lead to world wide investigations in relation to the control of infectious diseases in animals and man by anti microbial drugs. It is now known that within the family Enterobacteraciae the multiple drug resistance may be transferred by conjugation from one bacterium to another by means of episomes known as R factors (resistance factors) in association with resistance transfer factor s (RTF).

The episomes that consist of DNA occur in the cytoplasm of the donor bacterium and multiply independently of the chromosomal DNA. Thus a bacterium with an R factor only is a cell that is resistant to one or more anti microbial drugs but is not able to transfer this resistance to another susceptible cell. on the other hand, a bacterium with both an R factor and RTF is both resistant and capable under suitable conditions of transferring the resistance to another susceptible host. It is now known that this transfer of antibiotic resistance from one bacterium into another can take place not only in the normal bacterial flora of the intestines of animals (e.g. calves, chickens, pigs) but also in the absence of antibiotics in the environment; that the frequency with which in vivo transfers take place will be higher when colonization of the bacteria occurs in the intestines and that transfer can occur between bacteria belonging to the same genius for example from Escherechia coli to Escherechia coli or from one related genus to another for example Escherechia coli to Salmonella typhimurium.

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WHAT TO EXPECT AND HOW TO HANDLE IT

This newly acquired knowledge means the original assumption that drug resistance developed in bacterial population by a natural process of selection over a prolonged period of time and only in the presence of the particular antibiotic, is not the sole method by which the resistance can occur. These latest investigations have shown that by means of R factors and RTF microbial resistance to drugs may also develop quickly and can be transferred from one bacterial species to another and the process can continue in the absence of the antibiotic from the microbial environment. During recent years the widespread use of antibiotic in the field of the veterinary medicine and their further use as additives in animal foodstuffs to promote growth, have resulted in the development of increasing numbers of bacterial strains possessing resistance to many of the drugs in common use and this is becoming particularly evident among the strains of Salmonella and Escherechia. Although it's possible for human intestinal bacterial pathogens to develop drug resistance from the bacterial strains derived from animals, but present evidence would indicate that this may not be the most important mode of development of drug resistance currently observed in man, but it may be more commonly the results of antimicrobial therapy in vogue through human medicine. However both the veterinary and medical aspects of drug resistance are serious problems and the use of drugs in veterinary medicine which also apply to the treatment of human diseases must be made with discretion.

Editors Note:
This brief yet visionary article had been written by the author almost two decades back. Tragically Dr. Aijaz Ahmad Banday died at a very young age. The loss of a scientist of repute, as the author was, is irreparable. Kashvet and VetScan pay tribute to this son of the soil and feel honored by including this article of his in the issue.

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For more information mail to **vetscan@kashvet.org**