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Review

Invited Review: Pathology and Epidemiology of FP Virus

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Abstract: Fowl Pox (FP) is a contagious slow-spreading viral disease caused by the FP virus, a DNA virus belonging to the genus avipoxvirus, subfamily chordopoxvirinae of the poxviridae family. It affects all ages, sexes, and breeds of chickens and is distributed worldwide with high prevalence in tropical and subtropical countries. FP is transmitted by direct contact with an infected chicken or indirectly by biting arthropods, (mostly mosquitoes) and through infective fomites. In chickens it is characterized by two forms: (1) The cutaneous or dry form that is characterized by wart-like proliferative lesions and scabs on the featherless areas of skin and (2) the diphtheritic or wet form that is characterized by canker lesions found in the upper digestive and respiratory tracts. In the case of the cutaneous form, the mortality rate is usually low and affected chickens are more likely to recover than those with the diphtheritic form. In the diphtheritic form, proliferative lesions involving the nasal passages, larynx, or trachea cause respiratory distress and death from suffocation. The disease can be diagnosed based on history, presence of typical lesions, and by microscopic examination of affected tissues and virus isolation. Like many other viral diseases, there is no specific treatment for FP but due to its slow-spreading characteristic, the disease can be controlled by vaccination. Therefore, vaccination, arthropod control, and good management programs are important prevention and control methods of chicken FP to be undertaken both at the field and farm level.

Keywords: FP, Chicken, Lesion

Introduction

Chicken is important to the subsistence, economic, and social livelihoods of a large human population both in urban and rural settings of developing countries. Ethiopia has a huge poultry population that is predominantly reared under an extensive traditional type of management system. Over 96% of the country's chickens are local breeds and kept mainly in a free-range system. This indigenous poultry production in Ethiopia contributed to 98.5 and 99.2% of the national egg and poultry meat production, respectively (Natnael, 2015). In recent decades, mainly to satisfy the protein demand of the growing human population, the number of exotic chickens and intensive farms are relatively increasing (Mekibib *et al*., 2018).

Poultry and eggs continue to be a hugely important source of animal protein, with poultry meat production, consumption, and trade all increased since the late 1990s. The huge poultry population has faced several challenges by several constraints including diseases. Infectious diseases are recognized as one of the major constraints because they pose an enormous economic and societal impact on farmers (Pattison *et al*., 2008).

FP is a viral disease caused by avipoxvirus belonging to the chordopoxvirinae subfamily of the poxviridae family which induces pustular, benign, and proliferative lesions of the skin and diphtheritic lesions on the mucous membrane of the digestive and respiratory passages. The disease affects both domestic and free-living birds in nature, resulting in varying morbidity and mortality. The diphtheritic form is usually more severe as it causes significant mortality and economic losses in affected flocks (Adebajo *et al*., 2012). FP is an infectious and contagious disease that occurs particularly in tropical and sub-tropical countries. Outbreaks of the disease still affect

the poultry industry in many countries. Infection occurs through the mechanical transmission of the virus to injured skin or through transmission by mosquitoes and fomites (Beytut and Haligur, 2007).

FP is a relatively slow-spreading viral infection of chickens characterized by scab-like lesions on the skin of the unfathered body parts like the combs, wattles, eyelids, legs, and mucous membranes of the oral cavity, upper respiratory and digestive systems, and/or diphtheritic (wet) membranes lining the mouth or air passages. It has been present in chickens since the earliest times and is found throughout the world. Mortality rarely occurs if the lesions are limited to the skin. However, death occurs if the oral cavity or air passages become involved. There is no treatment for FP. Control and prevention in chickens are accomplished by vaccination by the wing web method with a commercially available FP or pigeon pox vaccine (Butcher and Rossi, 1996).

Diagnosis of FP is straightforward and made based on clinical signs and lesions, while confirmation is by histopathology or immunology, or virus isolation. Infection with the FP virus causes the chicken's poor growth, poor feed conversion, and a precipitous fall in egg production (Emmanuel *et al*., 2014). Therefore, the objective of this study is to review the epidemiology, pathological lesions, and diagnostic techniques of the FP virus in poultry.

Etiology and Morphological Characterizations of FP Virus

Within the subfamily Chordopoxvirinae of the family Poxviridae, eight genera are recognized based primarily on morphological and biological characteristics. Viruses from seven genera infect mammalian species and only members of the Avipoxvirus genus infect non-mammalian hosts or birds (Afonso *et al*., 2005). Within the Avipoxvirus genus there are 10 recognized species which are FP virus, Canary pox virus, Junco pox virus, Mynah pox virus, Psittacine pox virus, Sparrow pox virus, Starling pox virus, Pigeon pox virus, Turkey pox virus, and Quail pox virus, according to the International Committee on Taxonomy of Viruses (Gyuranecz *et al*., 2013). FP virus infecting fowls, turkey pox virus infecting turkeys, and pigeon pox virus in pigeons are closely related and are not strictly host-specific (Akanbi *et al*., 2016).

FP virus is classified in the genus Avipoxvirus, subfamily Chordopoxvirinae of the family Poxviridae. Poxviruses are large, double-stranded DNA viruses that multiply in the cytoplasm of the host cell. They are the largest animal virus, with a brick shape, ranging in size from 200 to 400nm long and 170 to 200 nm wide (Emmanuel *et al*., 2014). The outer coat is composed of random arrangements of surface tubules. The virion consists of an electron-dense centrally located biconcave core or nucleoid with two lateral bodies in each concavity and surrounded by an envelope. The 288 kbp FP virus genome encodes for over 250 genes (Wulandari, 2013).

FP virus has a genome consisting of 260 genes. Of the 260 genes, 65 are homologs with chord pox genes. The FP genome is approximately 70-100 kbp larger than chord pox genomes (Chambers *et al*., 2009) which is indicating that the avipox viruses have the potential to code for more proteins than other groups of poxviruses (Siddique *et al*., 2011). Due to their large size, poxviruses can be visualized with light microscopy and fine details of the virus remain blurred. Poxvirus genomes encode the genes necessary for replication and immune modulation (Chambers *et al*., 2009).

Poxviruses are different from other DNA viruses in that the viruses replicate and mature in the cytoplasm of the infected cells. The DNA of the FP virus contains approximately 288 to 300 Kilobase pairs (Kbp) (El-mahdy *et al*., 2014).

Epidemiology of FP Virus

The geographic distribution of avian pox is worldwide. The disease was reported in birds as early as the 17th century. This widespread avian disease infects all bird families, with some seeming more susceptible (van Riper *et al*., 2002). Avipox is a highly contagious viral disease that has been documented in more than 230 species of birds worldwide (Yeo *et al*., 2019) and affects domesticated and free-ranging birds around the world (El-mahdy *et al*., 2014) and resulting in varying morbidity and mortality. Although FP is believed to be widespread in the backyard and to some extent intensively reared poultry flocks, the epidemiologic details of the disease are not quite clear in free-range indigenous chickens (Adebajo *et al*., 2012).

In chickens, FP affects all ages, sexes, and breeds. Its infection is a slowly spreading disease and an economically important disease for chickens and turkeys as it causes egg production losses and mortality, especially in commercial poultry. FP has been reported worldwide as a mild to severe poultry disease. In recent years, FP outbreaks in poultry flocks have been gradually increasing due to the emergence of variant strains of FP virus (FPV or FWPV) and the fact that the novel FPV exhibited enhanced virulence after integration of avian Reticuloendotheliosis Virus (REV) into their genomes (Siddique *et al*., 2011). Although this variant of FWPV has been found widely, the reported illness and death rates from the cutaneous form of FP in chickens have not reached 100%. The study investigated a severe outbreak of cutaneous FP in a poultry flock in northeastern China in which all infected chickens died was reported that the flock had not been vaccinated with an FWPV vaccine (Zhao *et al*., 2014).

Although it is not a highly lethal disease and shows an atypical occurrence, avian pox disease is frequently reported in avian wildlife in Brazil. Avian pox reports are common in many countries and phylogenetic studies were

described by Lüschow *et al*. (2004), Jarmin *et al*. (2006), Manarolla *et al*. (2010), and Gyuranecz *et al*. (2013) with samples from the United States, Italy, Germany, and other countries (Ferreira *et al*., 2018).

Outbreaks of FP are rare and limited in temperate climates so vaccination is less common but they are more prevalent in tropical and sub-tropical climates where control of biting insects becomes more problematic and where FP remains a significant problem for small-scale and backyard flocks, as well as for intensive, commercial farming so that vaccination becomes a pre-requisite. Although there is no evidence that more than a single serotype exists (Giotis and Skinner, 2019). The disease occurs primarily during the warm months of the year. The virus is stable and can be transmitted by direct contact with an infected chicken or by mosquitoes (Butcher and Rossi, 1996). Its incidence is variable in different areas because of differences in climate, management, and hygiene or the practice of regular vaccination (Wulandari, 2013)

Transmission of FP Virus

Wild birds and insects play an important role in the spread of pox infection (El-mahdy *et al*., 2014). The disease is spread by biting arthropods, which included mosquitoes and mites and through infective aerosols, contaminated feed or water, and skin trauma resulting from pecking by other birds (Akanbi *et al*., 2016). It is transmitted by direct contact between infected and susceptible birds. Viruses containing scabs also can be sloughed from affected birds and serve as a source of infection. The virus enters the bloodstream through the eye, skin wounds, or respiratory tract. Mosquitoes become infected from feeding on birds with FP in their bloodstream. There is some evidence that the mosquito remains infective for life. Mosquitos are the primary reservoir and spreaders of FP on poultry ranges. Several species of mosquito can transmit FP. Often mosquitoes winter over in poultry houses so, outbreaks occur during winter and early spring (Shankar, 2008).

A common indirect mode of transmission is through mosquitoes, especially Culex and Aedes spp., which act as mechanical vectors of avipoxviruses. After the insects feed on the lesions of an infected bird, the virus can remain localized on the proboscis for at least 14 days, with no evidence of further replication. During this period, virions are mechanically transmitted to another susceptible bird. Besides mosquitoes, biting midges and mites have also been reported as mechanical vectors of Avipoxvirus. Indirect transmission of Avipoxviruses also occurs via the ingestion of food and water sources contaminated with virus-containing scabs shed by infected birds. Avipoxviruses are not known to complete their replication cycle in non-avian species and bird-to-human transmission has not been documented (Yeo *et al*., 2019).

Hence birds in the free-range may be clinically or subclinically infected and develop antibodies to the FP virus through many of these ubiquitous exposures (Adebajo *et al*., 2012). Introduction of infected or "carrier" birds in a susceptible flock will cause an outbreak by direct contact, water or feed transmission and mosquitoes or other flying insects can also transmit the virus from bird to bird and also transmit the disease to nearby flocks (Intervet, 2009).

Morbidity and Mortality of FP Virus

Broilers are frequently affected by the diphtheritic form of the infection. Losses are associated with depression in growth rate and downgrading due to dermatitis although avian pox does not result in primary mortality. Infection of susceptible mature commercial egg and breeder flocks results in a decline in production (Shane, 2005). In the cutaneous form, the proliferative lesions are primarily confined to featherless areas of skin, while in the more lethal diphtheritic form, the lesions are found in the upper digestive and respiratory tracts. In the mild cutaneous form of pox disease, flock mortality is usually low, but it may be high when the infection is of a generalized diphtheritic form or when the flock is affected by a secondary infection, usually in poor environmental conditions (Beytut and Haligur, 2007).

The mortality rate in the diphtheritic form is nearing 50%, particularly in young birds (Wulandari, 2013). FP (FP) is of considerable economic importance as the disease results in a drop in egg production and retarded growth in younger birds. The chances of mortality increase when the dry form occurs together with the wet form (El-mahdy *et al*., 2014). Morbidity is 10-95% and mortality is usually low to moderate, 0-50% (https://www.thepoultrysite.com/disease-guide/fowl-pox-pox-avian-pox).

The Clinical Signs of FP Virus

The incubation period varies from 4 to 20 days (Intervet, 2009). Avian poxvirus infection is said to be characterized by Cutaneous (dry pox), diphtheritic (wet pox), and systemic and oncogenic manifestation. Although only the cutaneous and diphtheritic forms have been documented in chickens to be caused by the FP virus. The cutaneous form of pox in chicken is characterized by local epithelial hyperplasia that includes the epidermis and underlying feather follicles resulting in the formation of nodules, papules, vesicles, and eventual formation of scabs (Akanbi *et al*., 2016). The dry form or cutaneous form is also characterized by wart-like nodules and tumorous lesions on unfeathered body areas, including the feet, legs, face and around the bill and eyes (Young and Vanderwerf, 2008). The lesions heal in about 2 weeks. If the scab is removed before healing is complete, the surface beneath is raw and bleeding (Butcher *et al*., 2018).

The diphtheritic form, also known as 'wet' pox, results in the formation of lesions on the mucosal membranes of the mouth and respiratory tract (throat, trachea, and lungs) of infected birds, causing impaired breathing and difficulty in feeding (Yeo *et al*., 2019), also has respiratory signs such as sneezing, gasping, head shaking and relatively high mortality (Diallo *et al*., 2010). The prognosis with this form of the disease is poor because lesions often cause death by asphyxiation (Afonso *et al*., 2000).

A mixed cutaneous and diphtheritic forms are said to be common with the development of lesions on the comb and wattles as well as diphtheritic lesions in the mouth and/or respiratory tract of the same bird (Akanbi *et al*., 2016). Pathogenicity and clinical presentations tend to vary among birds infected with even the same Avipoxvirus strain (Yeo *et al*., 2019). The general signs of the diseases include weight loss, loss of feathers, and scaly skin of the head, neck, and back. Secondary bacterial infections are common with both forms of the disease, having the potential to cause pneumonia or other bacterial infections at the site of blistering (El-mahdy *et al*., 2014).

Pathogenesis and Pathological Lesion of FP Virus

The virus is transmitted through direct contact or vectors. A break in the skin is required for the virus to enter the epithelial cells. The cells of the mucosa of the upper respiratory tract and mouth are highly susceptible to the virus. After entering epithelial cells, it spreads from cell to cell which is helped by the production of epidermal growth factors causing the proliferation of cells. Some virus enters blood circulation and causes viremia. Through circulation, it reaches certain organs like the spleen and liver. In the epithelium of the skin, it produces pock lesions (Chauhan, 2010).

Two forms of the disease are associated with different routes of infection. Cutaneous form occurs following infection by biting arthropods that serve as mechanical vectors for viral transmission. The disease is characterized by an inflammatory process with hyperplasia of the epidermis and feather follicles, scab formation, and desquamation of the degenerated epithelium and it predisposes the host to secondary bacterial infections. The second, or diphtheric, form involves droplet infection of the mucous membranes of the mouth, the pharynx, the larynx, and sometimes the trachea (Afonso *et al*., 2000). In the diphtheritic form (wet pox), slightly elevated white opaque nodules or yellowish patches develop on the mucous membranes of the oral cavity, tongue, esophagus, or upper trachea. Nodules rapidly increase in size and often coalesce to become a yellow, cheesy, necrotic, pseudo diphtheritic, or diphtheritic membrane (Wulandari, 2013).

The systemic form of avian poxvirus infection has been documented, whereby the liver had single to multiple soft whites to yellow nodules ranging in size from 0.2- 0.5cm in diameter (Akanbi *et al*., 2016). In this form, internal tissues are mostly affected (Gilhare *et al*., 2015).

Macroscopic and Microscopic Features of FP Virus

Nodules on feather-less parts of body i.e., comb, wattle, and face. Yellowish cheese-like material in the buccal cavity. Swelling of the eyelid leads to blindness. The cutaneous form is characterized by papule and scab on a comb, wattle, face, and other feather-less parts of the body. Yellowish nodules, later on, become blackish. In diphtheritic form, there is yellowish cheese-like material on the tongue, palate, and laryngeal orifice (Chauhan, 2010).

Histological examination shows characteristic intracytoplasmic inclusion bodies in infected skin and tracheal mucosa. The diphtheritic form is recognized by the presence of nodular hyperplasia of the mucosa of the pharynx and trachea. Chickens that die of diphtheritic pox show a plug of desquamated epithelium which lodges in the glottis resulting in asphyxiation (Shane, 2005). The proliferation of epithelium in *stratum spinosum* layer of the epidermis. Cells show hydropic degeneration (Chauhan, 2010). Pneumonia in canaries. Others: Spleen, bone marrow, thymus, bursa, air sacs, dermis, etc. Some avipoxviruses are oncogenic with wart-like growth (Shivaprasad, 1998) (Fig. 1, 2, 3).

Diagnosis of FP Virus

Pox is readily diagnosed based on flock history, clinical signs, lesions, and in some instances by microscopic examination of affected tissues and virus isolation studies. Cutaneous lesions are characteristic (Butcher and Rossi, 1996). Histological examination of affected tissue will confirm the presence of intracytoplasmic inclusions (Bollinger bodies) in the respiratory mucosa and skin (Shane, 2005).

Identification of the agent by smear technique, virus isolation, and molecular methods. FP virus multiplies in the cytoplasm of epithelial cells with the formation of large intracytoplasmic inclusion bodies (Bollinger bodies) that contain smaller elementary bodies (Borrel bodies). The inclusions can be demonstrated in sections of cutaneous and diphtheritic lesions by the use of Hematoxylin and Eosin (H and E), acridine orange, or Giemsa stains. The elementary bodies can be detected in smears from lesions, for example by the Gimenez method. Electron microscopy was used to demonstrate viral particles of typical poxvirus morphology by negative staining or in ultrathin sections of infected tissues.

Serological tests by Immune responses to FP virus can be demonstrated by the use of virus neutralization, agar gel immunodiffusion, immunofluorescence, passive hemagglutination tests, enzyme-linked immunosorbent assay, and by immunoblotting (Wulandari, 2013)

Definitive diagnosis is made by isolation or growth in chorioallantois membrane (CAM) with the development of pock lesion on the membrane or by agar gel precipitation tests (AGPT). It is done by inoculation onto chorioallantois membranes of 9 to 12-day old developing chicken embryos or avian cell cultures. Eggs from specific pathogen-free flocks should be used for virus isolation (Okwor *et al*., 2012). Viruses produce a protein similar to an epidermal growth factor and multiply easily in cell cultures and on the chorioallantois membrane of embryonated eggs and form type A cytoplasmic inclusions in the cells of these culture systems (Siddique *et al*., 2011).

Treatment of FP Virus

No treatment is available. However, FP is relatively slow-spreading. Thus, it is possible to vaccinate to stop an outbreak. The wing-web vaccination method is used for chickens and the thigh-stick method for turkeys older than 8 weeks (Butcher *et al*., 2018).

Field outbreaks of FP in chickens have been managed by the removal of the nodular lesion and treatment of lesions with 2% iodine or antiseptic watery solution to prevent a secondary infection, oral administration of antibiotics and vitamins, proper husbandry practices to relieve stress, and post-infection or emergency vaccination (Okwor *et al.,* 2012).

An early form of skin lesions can be burned to prevent viremia. Avoid burning subepithelial tissue. In diphtheritic form by systemic antibiotics, antiseptic fluids on the lesions to avoid secondary bacterial infections. Removal of diphtheritic membranes in case of breathing or feeding problems. In tumor forms surgical removal of tumors. In septicemia and CNS-form systemic antibiotics might help in very few cases (Lierz *et al*., 2002).

Vaccination of FP Virus

Live vaccines against the FP virus, which cause moderate pathology in poultry and of the Avipoxvirus genus, were developed in the 1920s. The development of recombinant FP virus vector vaccines began in the 1980s. Vaccination against FP was reported as early as 1928, using live FWPV or Pigeon pox virus, which are now known to be closely antigenically related to FWPV (Skinner *et al*., 2005).

Immunization is recommended in endemic areas using a mild-attenuated avipox, a chicken-strain virus vaccine administered at approximately 8 weeks of age. In areas where early exposure occurs, the age of vaccination can be advanced. In some areas, broilers are routinely vaccinated against avian pox by subcutaneous injection at one day old. The efficacy of this procedure is questionable based on demonstrated maternal antibody interference. In areas where flocks are affected by vertically transmitted mycoplasmas, adverse vaccine reactions from the avian pox vaccine can be prevented by the administration of a pigeon-pox vaccine (Shane, 2005).

Vaccinated birds should be examined for "vaccination takes" 7 to 10 days after vaccination. A "vaccination take" is an area of swelling and scab formation at the injection site. Satisfactory vaccination in a flock is indicated by a large number of birds having "vaccination takes." Vaccinated birds should be examined for seven to ten days following inoculation. That consists of swelling of the skin or scab at the site where the vaccine was applied (Butcher and Rossi, 1996).

Vaccination with live-attenuated Viruses (FPV and Canary Pox Virus (CaPV)) and non-attenuated viruses (pigeon pox virus) is used to control this disease. FP and pigeon pox vaccines are applied by comb scarification, by the wing-web stick method, or by feather follicle immunization. Vaccination confers protective immunity 10 to 14 days after infection (Afonso *et al.,* 2000).

Prevention and Control of FP Virus

FP outbreaks in poultry confined to houses can be controlled by spraying to kill mosquitos. However, if FP is endemic in the area, vaccination is recommended. Do not vaccinate unless the disease becomes a problem on a farm or in the area. For pigeons, chickens, turkeys, and canaries, a commercial vaccine is available also for psittacines. The use of these vaccines in other bird species is very questionable as other bird species do have other avipox-strains. Cross-reactions are limited. Vaccination via wing web or on freshly plucked feather follicles with a 6-12 month protection. Vaccination into a collection of affected birds or emergency vaccination is very questionable (Butcher *et al*., 2018).

For new birds, quarantine is highly recommended for 3-4 weeks. Affected birds should immediately be isolated from the rest of the group and all birds of the affected species should be kept in the house and separated from other birds. Cleaning and disinfecting of cages, control of arthropods in the environment, and proper disposal of contaminated ground is very important to control transmission. Prevention and reduction of stress, especially overcrowding, to prevent a weak immune system and traumatic injuries to the skin. Only essential staff should be in contact with

affected animals. Feeding and cleaning healthy birds of affected species first, then the affected separated birds. Changing clothes before entering and leaving the cage. In larger collections, the disposal of infected animals can be recommended to prevent latent infective virus (Lierz *et al*., 2002).

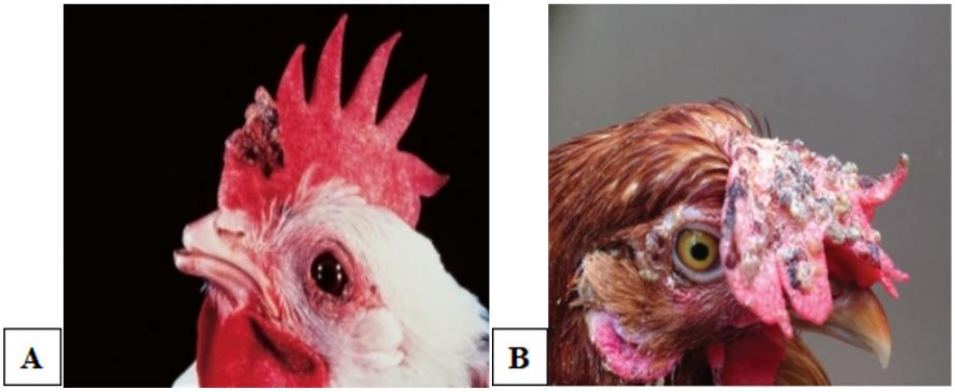

Fig. 1: (A) Focal lesion of avian pox on the comb of a hen (Shane, 2005). (B) Infected chickens manifested multifocal raised grey, crusty, verrucous, irregular nodules on the skin around the eyes and comb (Yeo *et al*., 2019)

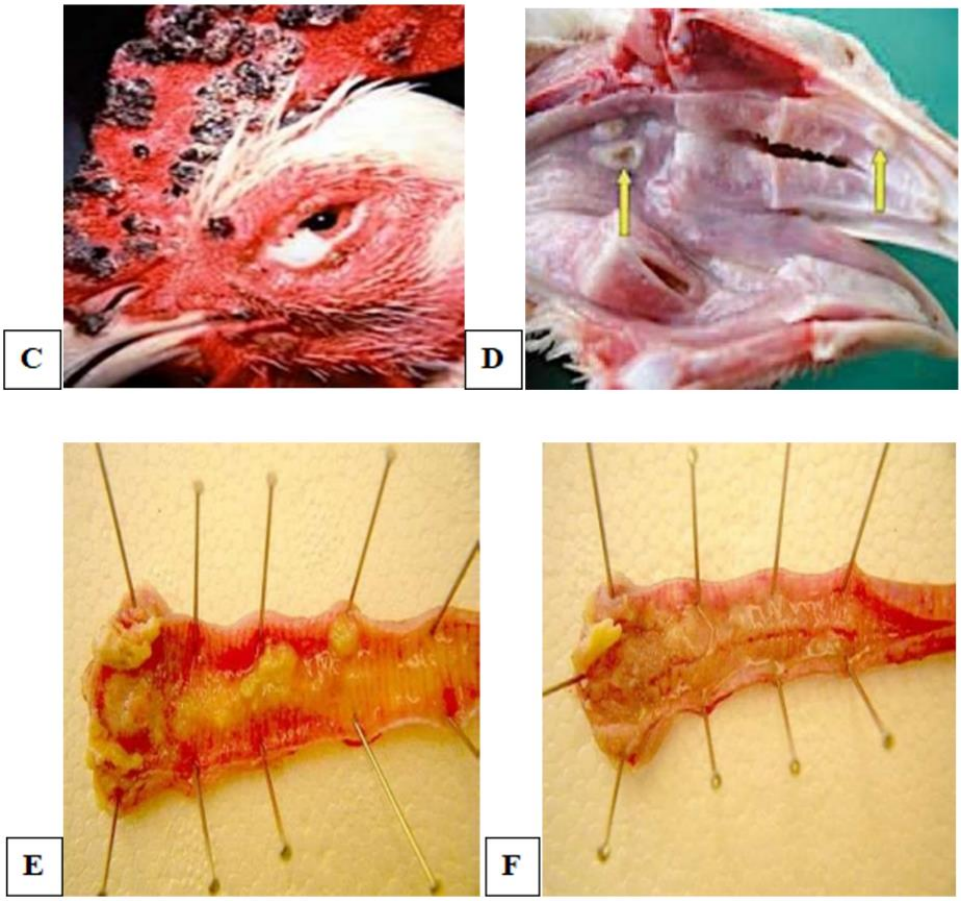

Fig. 2: (C) Dry pox results in scabs and lesions around the comb, wattle, ear lobes, and eyes**.** (D) Diphtheritic lesions look like whitish or yellowish plaques deposited and grown on the mucous coats of the buccal and nasal cavities, the sinuses, the larynx, the pharynx, the trachea, or the oesophagus (arrows). (E) Wet pox, tracheal patchy lesion. (F) Wet pox in the trachea, a thickened wall with necrotic tissue

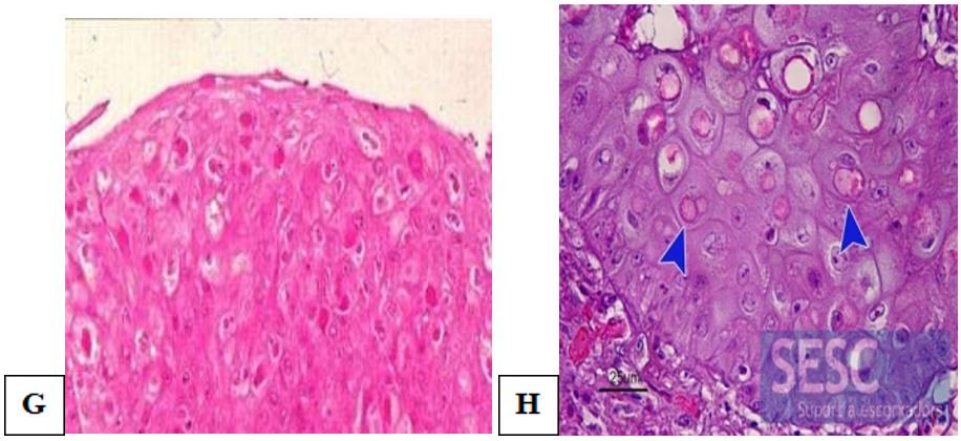

Fig. 3: Bollinger's bodies or granules: Relatively large, spheroid or ovoid (G), usually somewhat granular, acidophilic, intracytoplasmic inclusion body's (H)

Conclusion and Recommendations

FP is an infectious and contagious viral disease of poultry distributed globally. The disease has two recognized forms. The cutaneous (dry) form is characterized by nodular lesions of the skin, particularly the eyelids, comb, and thighs, a slow spread, and low mortality. The second form is the diphtheritic (wet) form, which is characterized by respiratory signs with high mortality. Both forms of the disease can be observed in a flock. Direct or indirect contact with infected birds or fomites and vectors are important means of transmission. Birds infected with the dry form usually recover in 2 to 4 weeks, but it can take several weeks to months for a flock to recover since the disease is slowly spreading. Either form of the disease will usually cause a decreased appetite, some weight loss, and a drop in egg production and young birds may have growth retardation. Although FP is an economically important disease affecting the poultry industry it is poorly studied in Ethiopia. Therefore reducing infection pressure by institutional guided control and prevention methods are among the important recommendations discussed in this review.

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Author's Contributions

Jirata Shiferaw: Preparation, development, and publication.

Rediet Wolde and Abdi Feyisa: Preparation and development

Ethics

The authors confirmed all the manuscripts written in this review articles were thoroughly cited and acknowledged. All of the authors have read and approved the final version of the manuscript.

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