Review on Avian Salmnellosis and its Impact

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Abstract: Avian Salmonellosis is an infectious bacterial disease caused by different species of *Salmonella*. The common causative agent in poultry is *Salmonella Pullorum, galinarum, Arizona, Typhimurium* and *enteridtis* which cause Pullorum, fowl typhoid, paraclonal infection and paratyphoid disease respectively. Pullorum disease can cause severe clinical illness in young chicken. *Salmonella Arizona* can affect turkeys. *Salmonella Typhimurium* can affect wide range of hosts including humans. Avian salmonellosis can be transmitted with the mode of transmission; horizontally and vertically. It can also be transmitted to humans by feeding contaminated poultry products like meat and egg. The pathogenesis is due to the bacteria invades the gastro intestinal tract of birds and cause inflammation of the intestine. The bacteria are phagocytized by macrophages and through the blood stream and the lymphatic system it can spread to the reticuloendothelial cells like liver and spleen where multiplication can takes place. Salmonellosis can be diagnosed by culturing and identification of the bacteria by using serological test like Enzyme-linked immunosorbent assays. Nicropcy indicates Pathological lesion is found on the tissue of liver, kidney, spleen, intestine and ovary. Treatment of avian salmonellosis is not satisfactory because the drug cannot eliminate the agent from the body. Once birds are infected they remain carrier and shed the bacteria throughout their life. Prevention of salmonellosis can be done by controlling movement of rodents, birds and peoples to the farm, strict sanitation and vaccination of birds. Avian salmonellosis has zoonotic importance. Its zoonotic impact can be reduced by applying Hazard Analysis Critical control point system and hygienic feeding and drinking methods with effective public education to reduce the risk. Salmonelosis in poultry causes heavy economic loss through mortality and reduced production. This review paper gives an overview on avian salmonellosis.
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1. Introduction

Salmonellas are the common facultative intracellular organisms that survive in the phagolysosome of macrophages and can therefore evade the bactericidal effect of antibody and cause salmonellosis. Compared to other organisms of the same family, *Salmonellas* are relatively resistant to various environmental risk factors. *Salmonellas* have been shown to be resistant to drying even four years, especially in dried feces, dust and other dry materials such as feeds and certain foods. Prolonged survival in water and soil has also been described. Because of these characteristics it is challenging to different sectors (Myint *et al.,* 2004). Poultry sector is one of the sectors being challenged by Salmonellosis. It is even under a gradual decline in developing countries and is highly constrained by diseases among other factors (FAO, 2007). The current rates of mortality due to diseases from day old to adult chicken are estimated to be 20 to 50% (Tadele and Yilma, 2004). Among diseases, fowl typhoid and Pullorum disease are expected to incur heavy losses to the poultry industry as it is the case (Bell et al., 1990; Bouzoubaa et al., 1992). *Salmonella* are introduced in poultry farms by several ways, including day-old infected chicks, domestic animals, human, equipment, water and feed. Once the farm is contaminated, it is very difficult to eliminate *Salmonella* from the environment. Factors such as the presence of wild birds, rodents, domestic animals and insects, as well as intensive production systems and multiple age flocks keep *Salmonella* on farms, which compromise eradication methods. Infected breeder flocks are responsible for vertical transmission. Although *Salmonella* is typically not a pathogen in the gut of the chicken, systemic infection can cause serious disease (fowl paratyphoid) in the bird. This usually results from contamination of egg shells from infected breeder hens and spread of the organism into the respiratory tract during hatching which may result in

potentially high morbidity and mortality in young chicks (Sanchez *et al.,* 2002). In laying hens, *Salmonella* can be highly invasive leading to systemic infections that can potentially be deposited in the internal contents of eggs by trans-ovarian transmission following colonization of the intestinal tract (Ibrahim *et al.,* 2013). The eggs are contaminated either from the ovary tissue or on their passage through the cloaca. Fowl are the specific host of *Salmonella enterica serovars Pullorum* and *Gallinarum*, which cause Pullorum disease and fowl typhoid, respectively. *Salmonella Arizona* can affect turkeys and cause paraclonal infection. Other serotypes with no specific host, such as Enteritidis and Typhimurium cause paratyphoid infection and may infect chickens and persist in the final poultry product, inducing or not clinical disease during rearing (keery, 2010). Non thyphoidal Salmonellosis is one of the most common and widely distributed foods borne intestinal disorder of humans associated with contaminated meat and poultry is caused by non-host specific *Salmonella* species (Enteritidis and *Typhimurium)*. It is estimated to cost nations billions of dollars annually there by draining funds that could have been used for development (Radostits *et al.,* 1994; WHO, 1988). *Salmonella* remains the main food borne bacterial diseases in human and many of the world outbreaks are related to food containing poultry products. Thus, the control of *Salmonella* in poultry flocks is crucial for poultry industry success. The increasing costs or impracticality of improvements in biosecurity, hygiene and management, coupled with the increasing problems associated with antibiotic resistance, suggests that vaccination in poultry will become more attractive as an adjunct to existing control measures (Pavic *et al.,* 2008).

Therefore, the objectives of this seminar paper are; to briefly describe the etiology, epidemiology, pathogenesis and clinical and pathological findings of avian salmonellosis; to review the treatment, zoonotic impact, economic significance, control and prevention of avian salmonellosis.

2. Salmonellosis

2.1. Historical perspective

The genus *Salmonella* was named after Daniel Elmer Salmon, an American veterinary pathologist. While Theobald Smith was the actual discoverer of the type bacterium (*"Salmonella enterica"* var. Choleraesuis) in 1885 from porcine intestine. Dr. Salmon was the administrator of the United States Department of Agriculture (USDA) research program, and thus the organism was named after him. Smith and Salmon had been searching for the cause of common hog cholera and proposed this organism as the causal agent. Later research, however, would show that this organism (now known as *Salmonella enterica*) rarely causes enteric symptoms in pigs, and was thus not the agent they were seeking (which was eventually shown to be a virus). However, related bacteria in the genus *Salmonella* were eventually shown to cause other important infectious diseases. The genus *Salmonella* was finally formally adopted in 1900 by J. Lignieres for many species of *Salmonella*, after Smith's first type species Salmonella Choleraesuis (Girmay, 2013). The first report of laboratory confirmed outbreak of food borne salmonellosis described an episode in which 58 persons in 25 different families who had eaten beef developed acute gastroenteritis; one died. Gartner isolated the 'Gartner-bacillus' from infected cow from which the meat came, and from organ of fatal case. Kauffman determined that the 'Gartner-bacillus' from this outbreak was serotype Enteritidis, but outbreak of 'Gartner-bacillus' was of serotype Dublin and possibly other serotypes. Mice, rabbit, goat, and guinea pig were affected when inoculated with the bacillus. In the following years, several out breaks of salmonellosis affecting man and animals were reported and old concept of 'meat poisoning' was linked with the etiologic agent *Salmonella*. Subsequently, human salmonellosis occurred primarily among individuals who ate meat from infected animals, mainly cattle, but also pigs or goats (Teklu, 2008 and Chebo, 2014).

2.1.1. Characteristics of Salmonella

The family *Enterobacteriaceae* consists of gram negative, facultatively anaerobic, non-spore forming and rods. *Salmonella* conforms to the general definition of the family. Members of the genus are motile by peritrichous flagellation except *S. enterica ser. Pollorum* and S. *enterica ser. Gallinarum,* which lack flagella. Non motile variants can also arise as result of a faulty assembly of flagellar subunits or deficiencies in the motor functions in these appendages. Salmonellae are chemoorganptrophic, with an ability to metabolize nutrients by the respiratory and fermentative pathway. An important characteristic of the Salmonella bacteria is that they are able to grow and multiply outside living host organisms, thus having greater survival chances than otherwise (Gray and Fedorka, 2002).

2.1.2. Biochemical characteristics

Salmonellae are generally unable to ferment lactose, sucrose or salicin, although glucose and certain other monosaccharides are fermented with production of gas. They are usually catalase positive, oxidase negative, and reduce nitrates to nitrites. The organisms use citrate as the sole carbon source, decarboxylate lysine, arginine and ornithine and produce hydrogen sulphide. The methle red reaction is positive, the Vogus–Proskauer test is negative and indole is negative. Phenylalanine is not delaminated, urea is not hydrolysed, gelatin is not liquefied rapidly

in nutrient media and neither DNAase nor lipases are produced. *Salmonellae* may harbor temperatures phages or plasmids that code for metabolic characters used in identification (e.g. H2S, lactose or sucrose fermentation) (Teklu, 2008).

2.1.3. Growth and physical characteristics

Salmonellae are able to grow on a large number of culture media and produce visible colonies well within 24 hours at about 37^{o}C. The parameters of pH, water activity (a_w), nutrient content and temperature are all interrelated for *Salmonellae*, as they are for most other bacteria. The pH for optimum growth is around neutrality (between 6.6 and 8.2), with value above 9.0 and below 4.0 being bactericidal. A minimum growth pH of 4.05 has been recorded for some (with hydrochloric and citric acids), but depending on the acid used to lower the pH, the minimum may be as high as 5.5. Aeration was found to favor growth at lower pH values (Teklu, 2008 and Chebo, 2014). *Salmonellae* grow in the temperature range of 2-47 ^{o}C with rapid growth between 25 and 43^{o}C (D'Aoust, 2000; 2001). The lowest temperature at growth has been reported are 5.3^{o}C for S. Heidelberg and 6.2 ^{o}C for S. *Typhimurium.* Temperature around 45^{o}C has been reported by several investigators to be upper limit for growth. Regarding available moisture, the growth inhibition has been reported for a_w value below 0.94 in media with neutral pH, with a_w value being required as the pH is decreased toward growth minima (Coetzer *et al.,* 1994). Compared to other gram negative, *Salmonellae* are relatively resistant to various environmental factors. The survival of *Salmonellae* for prolonged period of time in foods stored at freezer and ambient temperature is documented. The viability of *Salmonellae* in dry foods stored at ≥25^{o}C decreases with increased storage temperature and with increased moisture content (WHO, 1988). Although the low aw in dry foods is not conducive to bacterial growth, the condition does promote survival of *Salmonellae* for prolonged period of storage at ambient and elevated temperature. The resistance of *salmonellae* to heating depends on medium in which they are kept; greater heat resistance observed in *Salmonellae* grown in nutritionally rich media than of *Salmonellae* grown in minimal media (Seifert, 1996; D'Aoust, 1997). *Salmonella* is sensitive to heat and will not survive temperature above 70^{o}C. They are killed rapidly by autoclaving at 120^{o}C. *Salmonella* have been shown to be resistant to drying events for years, especially in dried feces, dusts, and other dry materials such as feeds and foods. Prolonged survival in water and soil has been described. The usual disinfectants (creolin 3%, chalk milk 5%, caustic soda 2%) inactivate the pathogen in a few minutes. *Salmonella* are quit sensitive to gamma and beta radiation (Molla, 2004).

2.1. Etiology

Scientifically, *Salmonella* classification is described under: Domain: Bacteria; Phylum: Protobacteria; Class: Gamma Protobacteria; Order: Enterobacteriale; Family: Enterobacteriaceae; Genus: *Salmonella*(kemal, 2014). *Salmonella* species are Gram negative, facultative anaerobic rods. *Salmonella* species are classified into serovars (serotypes) based on the lipopolysaccharide (O), flagella protein (H), and sometimes the capsular (VI) antigens. There are more than 2500 known serovars. Within a serovars, there may be strains that differ in virulence. *Salmonella* nomenclature is not completely standardized. According to the latest nomenclature, which reflects recent advancement in taxonomy, the genus *Salmonella* consists of three species (spp): *S. enterica*, the type species, *S. bongori*, former subspecies V and S. subterranean (Chebo, 2015). *S. enterica* consists of six subspecies: *S. enteric subsp. enterica*, *S. enteric subsp. salamae, S. enteric subsp. Arizonae, S. enteric subsp. diArizonae, S. enteric subsp. houtenae and S. enteric subsp. indica* (Beshatu, 2014). The etiological agent of fowl typhoid is *Salmonella* Gallinarum, Pullorum by *Salmonella* Pullorum, paracolon infection by *Salmonella* Arizona and paratyphoid infection caused by *Salmonella Typhimurium, Salmonella Enteritidis* and *Salmonella* Heidelberg (Dunkley *et al.,* 2008).

2.2. Epidemiology

2.2.1. Transmission

Salmonella are introduced in poultry farms by several ways, including day-old infected chicks, domestic animals, human, equipment, water and feed. Once the farm is contaminated, it is very difficult to eliminate *Salmonella* from the environment. Factors such as the presence of wild birds, rodents, domestic animals and insects, as well as intensive production systems and multiple age flocks keep *Salmonella* on farms, which compromise eradication methods (Bercheir *et al.,* 2003). Transmission of *Salmonella* Pullorum is primarily through the egg but also occur via direct or indirect contact with infected birds. Rarely there is also occurrence of wound infection. *Salmonella* Gallinarum has much higher tendency to spread among growing or mature flocks. But it is also transmitted via egg and produce lesion in chicks and poults similar with *Salmonella* Pullorum. *Salmonella* Arizona is transmitted by infected egg from the hen. The three potential means of spreading of *Salmonella* Enteritidis and *Salmonella* Typhimurium are fecal-oral, contaminated feed and sexual contact in breeder birds. Spreading of the infection between birds is a large concern as a single infected hen can theoretically spread the disease throughout the rest of the flock (Kahin, 2005). *Salmonella* Enteritidis colonizes the gastrointestinal tract in several areas, mainly the crop

and caeca. The presence of *Salmonella* Enteritidis in the caeca enables shedding to occur when feces are passed. Feed can become contaminated either from the raw material themselves or during processing and is therefore also another potential source of infection for hens and chicks (Boever, 2005). There is also the potential for *Salmonella* Enteritidis to spread via sexual contact. Previously unmated hens would become infected with *Salmonella* Enteritidis if inseminated with*Salmonella* Enteritidis contaminated semen. Vertical transmission occurs in birds, with contamination of the vitelline membrane, albumen and possibly the yolk of eggs (keery, 2010).

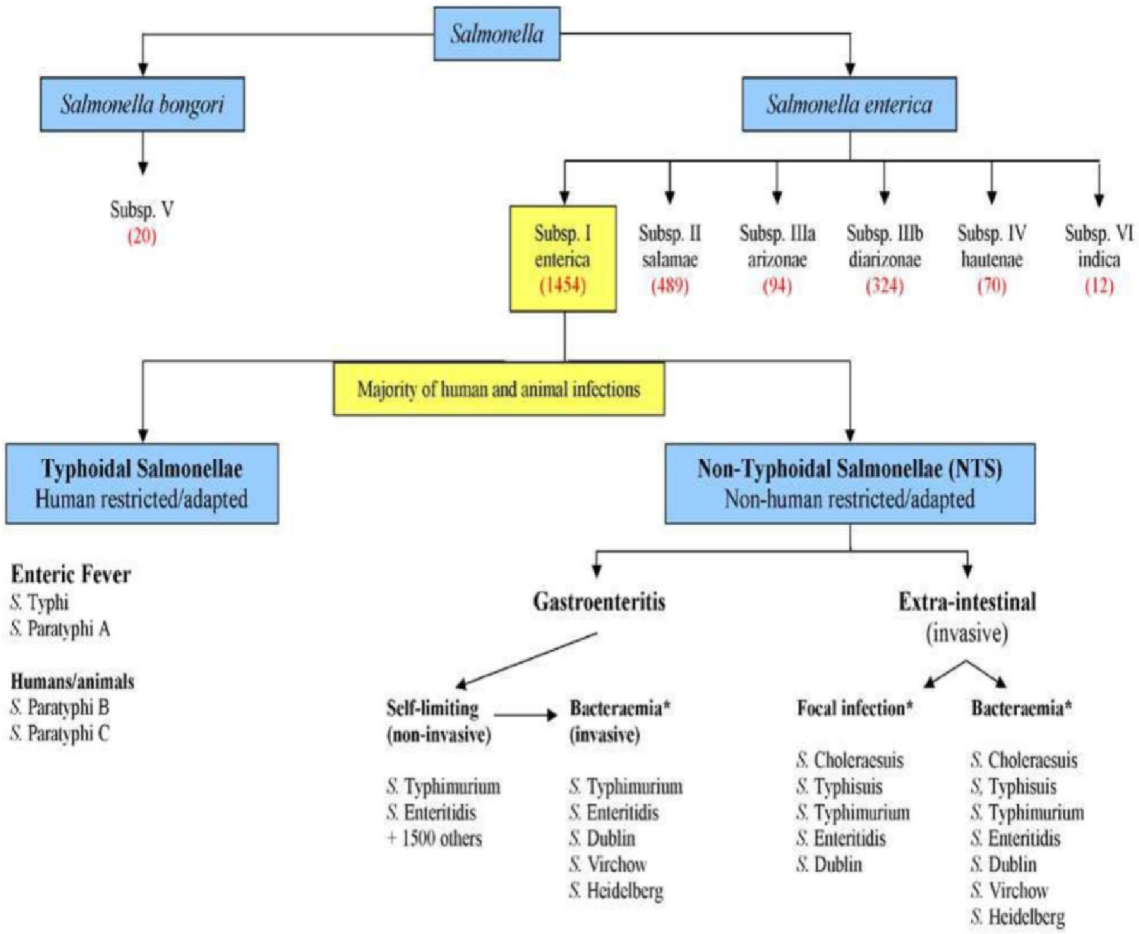

Figure 1: Classification of the genus *Salmonella* (Langridge *et al*., 2008).

2.2.2. Prevalence

Although *Salmonellae* have been isolated in poultry flocks of various species, including both broiler and layer breeds, estimates of the prevalence of *Salmonellae* in commercial poultry and their environments have varied considerably. While they may reveal true differences in the distribution of *Salmonella* across geographic regions and management systems, they may also be simply due to differences in the techniques used to determine the *Salmonella* prevalence. The apparent prevalence of *Salmonella* differs depending upon sample types, collection and handling methods and detection technique (Kabir, 2010). Chickens are the natural hosts for the highly host adapted biovars *Salmonella* Gallinarum and *Salmonella* Pullorum, but natural outbreaks have also been reported in turkeys, guinea fowl, quail and pheasants. Fowl typhoid is per acute, acute or chronic form of disease affecting mostly adult chickens, whereas Pullorum disease affects the very young chickens, mostly 2−3 weeks of age. In the adult it tends to be chronic (Medina *et al.,* 2013). Fowl typhoid is frequently referred to as a disease of adult birds; still, there are also reports of high morbidity and mortality in young chickens. *Salmonella* Gallinarum can produce lesions in chicks, which are indistinguishable from those associated with Pullorum disease. Paratyphoid infection the prevalence of the

disease varies widely by geographic location and season. Arizona infection primarily affects turkeys, with chickens affected occasionally (Kahin, 2005). A certain percentage of chickens that survive from the initial infection become carriers with or without presence of clinical signs and pathological lesions. Crowding, malnutrition, and other stressful conditions as well as unsanitary surroundings can exacerbate mortality and performance losses due to salmonellosis, especially in young birds (Kabir, 2010).

2.3. Pathogenesis

The pathogenicity of *Salmonella* depends on the invasive properties and the ability of the bacteria to survive and multiply within the cells, particularly macrophage. The main site of multiplication of these bacteria is the digestive tract. Following invasion through the intestinal mucosa, caeca tonsils and Payer's patches, the organisms are engulfed by macrophages, and through the blood stream and/or lymphatic systems, they spread to organs rich in reticuloendothelial tissues, such as liver and spleen, which are the main sites of multiplication. In case of inadequate body defense mechanism, they may lead to second invasion and be localized in other organs, particularly ovary, oviduct, myocardium, pericardium, gizzard, yolk sac and/or lungs (Kumara *et al.,* 2013). The outcome of infection with *Salmonella* depends essentially on three factors: the infective dose, predisposing factor influencing the host and the level of immunity (kemal, 2014). In the bird challenge, *Salmonella* Typhimurium rapidly causes inflammation of the intestinal mucosa, but *Salmonella* Pullorum preferentially targeted the bursa of fabricius prior to eliciting intestinal inflammation. Pullorum disease manifests itself predominantly as an enteric disease of chickens, while fowl typhoid shows signs of septicemic disease. Both biovars can cause septicemic infections, which may be acute or chronic, but unlike *Salmonella* Pullorum, *Salmonella* Gallinarum is capable of producing per acute infection and hemolytic anemia in both young and adults. *Salmonella* Gallinarum is extremely pathogenic to young broiler chicks. Fowl typhoid is indistinguishable from Pullorum disease unless the etiological agent is isolated and identified (Kabir, 2010). Poultry species can be infected by host-specific and non-host specific *Salmonella* serotypes. *Salmonella* Gallinarum and *Salmonella* Pullorum cause severe disease and death of birds compared with other known Salmonella serotypes. Avian systemic salmonellosis has three phases: invasion, systemic infection and the resolution of the infection. The third phase can have three results: the clearance of the bacteria, death of the birds due to infection and, partial clearance of the bacteria, which leads to a subclinical carrier state (Ibrahim *et al.,* 2013). The biology of pullorosis is markedly different when compared to fowl typhoid, which causes high mortality. Pullorosis induces an increase of *Salmonella* in the spleen resulting in an infection of the reproductive tract. In chickens, it produces systemic disease in some special cases, such as the laying period, in chicks in the first two weeks of life or after viral diseases. *Salmonellas* are not native members of the gut micro biota, but young chicks are readily colonized. They become localized in the caeca tonsils and can occur in the upper part of the small intestine and in the gizzard and proventriculus. Because most birds infected with *Salmonellas* become symptomless carriers, they constitute a reservoir of the organisms, which is source for infection (Revolledo, 2012).

2.4. Clinical sign

Clinical signs observed in broilers in the spontaneous outbreaks of salmonellosis were of variable nature and as such could not be considered of major diagnostic significance. In general the diseased birds showed dullness, severe depression, anorexia, appeared listless, stood motionless about with head sunk onto the chest and with both eyes closed. Most of the infected chicks developed progressive weakness, complete inappetance, increased thirst and droopy wings with ruffled feathers. Watery to mucoid greenish yellow diarrhea was the most characteristic clinical sign in acute cases. Lameness was also recorded in birds in a few outbreaks. In few cases mild respiratory distress was also observed (Nazir *et al.,* 2012). In per acute cases of paratyphoid, birds mostly died without showing any premonitory signs. However in most of the other outbreaks of paratyphoid, birds showed depression with closed eyes, ruffled feathers, fecal soaked vent feathers, unabsorbed yolk and profuse diarrhea. In acute cases of Pullorum disease in birds having history of diarrhea, pasting of vent with loose whitish faecal material. Clinical signs in chicks and poults include anorexia, diarrhea, dehydration, weakness and high mortality. In mature fowls, fowl typhoid and Pullorum disease are manifested by anorexia; drop in egg production, increased mortality, reduced fertility and hatchability. *Salmonella* Pullorum infected adult birds may or may not exhibit any clinical signs, or they cannot be detected by their physical appearance (Ashwani *et al.,* 2014). Infection transmitted via egg or hatchery usually results in mortality during the first few days of life and up to 2-3 weeks of age (Kahn, 2005). In a setting of fertile egg with a few, infected embryos there may be reduced hatchability, the newly hatched birds appears weak or soon die and others that develop bacteremia, morbidity and mortality began to increase around the 4th and 5th days (Charlton, 2000). Chicks dying after hatching often exhibit on signs of ill health before death but older chicks appear sleepy, huddle together with eye

closed, a "pot-bellied" appearance and sometimes urates stains in vent region (Gordon and Jordan, 1982). Death of birds takes place from second day and decline by about 5th day and usually stops by about 8th month (Chauhan and Roy, 2007).

2.5. Post mortem findings

The occurrence and types of gross lesions are highly variable depending on the course of the infection, the virulence of the organism, and the resistance of the host. The lesions in chicks affected with fowl typhoid were indistinguishable from those associated with Pullorum disease. In typical cases of fowl typhoid, appearance of bronze colored liver was characteristic and prominent lesion. The bronze discoloration of the liver was observed more frequently at 7 to 15 days of age. Livers of affected chicks of this age group also revealed numerous grayish necrotic foci or necrotic patches, reddish hemorrhagic foci which were distributed uniformly on their surfaces. The chicks which died in early age group also showed pale discoloration, enlargement and congestion of liver along with mild distension of gall bladder. In few of these cases diffused areas of necrosis were also observed (Kumara *et al.,* 2013). In case of Pullorum disease the newly hatched chickens, which die from the disease, may not show any gross lesions. Some of them may show hemorrhagic streaks on normally yellowish liver of newly hatched chicks. The chicks, which die later, may show grayish necrotic spots of 1 to 2 mm size in the liver, raised, white spots on heart and spleen (Chauhan and Roy, 2007). In acute cases of Pullorum disease in birds having history of diarrhea, pasting of vent with loose whitish faecal material, impaction of cloaca was observed in affected birds. In addition to above changes in Pullorum disease, unabsorbed yolk and yolk sac infection were observed during first week of early age. The grossly observed lesions in paratyphoid infection included necrotic foci on liver, pericarditis with the presence of fibrinous exudates in pericardial sac, perihepatitis, presence of grayish white nodules on ventricular region of heart (very rare), congestion of intestines and kidneys, yolk sac infection. The lesions in caeca were more prominent and severe in cases of paratyphoid (Nazir *et al.,* 2012).

2.6. Diagnosis

Bacterial culture and detection is the only way of making a definitive diagnosis of salmonellosis and of exactly determining the serotype. However, culturing the organism is unreliable for various factors like, the method used to collect samples, the amount of sample submitted variation in sheidling of the organism, and the bacteriological method used (Ashwani *et al.,* 2014). The whole blood test is used for rapid diagnosis of *Salmonella* Pullorum/Gallinarum on the farm, being a relatively reliable diagnostic test under certain circumstances. In the laboratory, the tube agglutination test is the method of choice for export and diagnostic purposes for samples from all species of farm animals. Enzyme-linked immunosorbent assays are available for some serovars and may be used for serological diagnosis and surveillance. PCR is a highly specific and sensitive for detection of *Salmonella*. DNA probe can also be used for detection of *Salmonella* in the poultry (Hossain *et al.,* 2006).

2.7. Differential Diagnosis

Avian salmonellosis must be differentiated from aspergillosis and collibacilosis. In chicks, the white nodules in internal organs can be confused with Marek's disease. In adult carriers, infections with coliform bacteria must also be considered (Boever, 2005).

2.8. Treatment

A number of anti-bacterial agents will reduce the morbidity and mortality, however, no treatment is likely to effect the complete elimination of the carrier from uninfected flock (Jordan, 1994). The addition of furazolidone to the mass at a level of 0.04% for 10 days is generally effective in reducing mortality and if instituted early in the outbreak the carrier rate is low (Gordon and Jordan, 1982). In Pullorum and fowl typhoid disease treatment of infected flocks to alleviate the perpetuation of the carrier state is not recommended. In case of paracolon infection antibiotics are given by day old poults to minimize mortality. But birds may still carry and shed the organism even after treatment. In paratyphoid infection several antibacterial drugs help to minimize mortality but do not eliminate the disease from the flock. Polymixin B was the most sensitive drug for treating Salmonellosis infection. Also Salmonella infection is highly sensitive to chloramphenicol (Kumara *et al.,* 2013).

2.9. Prevention and control

Prevention and control programs for infections aim at protecting the health of the birds, ensure the safety of the consumers, and strengthen the reliability of the poultry production chain. In Pullorum the risk of vertical transmission may be minimized by bacteriological and serological monitoring of breeding chicken lots, resulting in *Salmonella*free birds; by purchasing birds more resistant to Salmonella infection by culling birds that are carriers of the microorganism; by treatment of eggs that are still in the sheds, and careful incubation of dirty and cracked eggs. In fowl typhoid the prevention and control methods are the same with that of Pullorum disease (Elina *et al.,* 2012). The best method of control is by preventing the introduction of *salmonellae* with infected birds by high standard of management and flock security. However, once a primarily breeding flock is infected with *Salmonella* which have an

affinity for poultry, it is very difficult to eliminate. Administration of intestinal flora from adult birds has been shown under some circumstances to protect chickens against challenge with *salmonellae* (Jordan, 1994). Good bio-security is essential including all-in-all-out policy forms, where all birds are of the same and all brought in at the same time and disposed to allow for proper cleaning and disinfection between crops (Gracey, 1999). Good hatchery sanitation and a vigorous program of detecting and eliminating reactors from breeding stock (Nesheim *et al.,* 1979). Live and attenuated vaccines have been used worldwide, and their efficacy has been demonstrated in challenge trials. The aim of a live attenuated vaccine should be to reduce the bacterial virulence while maintaining its immunogenicity. Other attenuated vaccines include autotrophic and metabolic drift mutants. Further research is needed to evaluate immunological interactions among the host and *Salmonella*, avoiding empirical methods in developing new vaccines and investigating ways to prevent the infection. Vaccines against *Salmonella* infections in chickens and other food-producing animals require protection at both mucosal (gut) and systemic levels (Revolledo, 2012).

3. Zoonotic Importance

Salmonellosis is a common human intestinal disorder primarily associated with *Salmonella* contaminated meats and poultry. Contaminated poultry is a common source of human infection. The disease has assuming increasing importance in recent years, because of the much more frequent occurrence of human salmonellosis, with animal salmonellosis as the principal reservoir. Food prepared with contaminated raw eggs, egg products or insufficiently heated poultry meat or is the major source of the human *Salmonella* infections (Molla *et al.,* 2002). In addition, good hygiene practices during food preparation in the kitchen, adequate refrigeration and adequate heating also help to prevent *Salmonella* infections. The increasing costs or impracticality of improvements in biosecurity, hygiene and management, coupled with the increasing problems associated with antibiotic resistance, suggests that vaccination in poultry will become more attractive as an adjunct to existing control measures (Pavic *et al.,* 2008). Hazard Analysis Critical control point system has been successful and is being followed by measures to reduce the number of *Salmonella* entering processing plants through live animals. In developed countries most of the large scale operations are run by private sectors and it is compulsory to register annually before the importation of a new stock. The objective of this regulation is to encourage poultry breeder farmers to control or eradicate salmonellosis in their farms in order to produce *Salmonella* free commercial chicks and safe guard the industry and consumer from zoonotic salmonellosis (Priyantha *et al.,* 2011).

4. **Economic Importance**

Salmonelosis in poultry causes heavy economic loss through mortality and reduced production (Khan*et al.,* 1998). The consequences of Salmonella in domesticated birds can be complex and generally go well beyond the immediate effects on affected producers. These diseases have numerous impacts, including: productivity losses for the poultry farms (e.g. production losses, cost of treatment, market disturbances), loss of income from activities using poultry resources, infection of human (morbidity, food safety and quality), prevention or control costs (public expenditure) and suboptimal use of production potential (Sanchez *et al.,* 2002). The most direct economic impact of these diseases is loss of production and/or productivity, and ensuing income losses for farmers. If the farm economy is diversified or if there are other opportunities to generate income, the impacts can be mitigated. However, if the economy depends on one or some of the vulnerable products, the impacts can be serious, and local food security can be threatened. The economic impact also depends on response strategies adopted by farmers and possible market adjustments (Elina *et al.,* 2012). Economic loss due to FT outbreak could be very high, not only from loses due to mortality but also from other costs that may be involved, such as removal of dead animals, disinfection, and preparation of hen house for the entrance of new flocks (Shivaprasad, 2000).

Conclusion and Recommendations

Avian salmonellosis is one of the most important diseases of poultry. It affects both domestic chickens and wild birds. Now a day there is an increase in the incidence of the disease as the production system is intensified. The disease can be introduced by numerous ways to the farm. It can cause higher morbidity and mortality to the poultry especially Pullorum and fowl typhoid one. It has greater economic impact by decreasing egg production, reducing growth and causing death of birds. And it can also cause reducing market of poultry products by lowering the number people consuming egg and meat of chickens. It has highly zoonotic impact. The main public health concern is it can cause drug resistance to peoples. It can be prevented by applying different methods like avoid contact of birds with humans, rodents and other birds outside the farm and public education. Based on the above conclusions the following recommendations are forwarded.

- The poultry farm should be prevented by applying strict extinction and control methods of

rodents and other animals including human being entering to the farm.

- Strict sanitization of the poultry house and equipment is important in order to prevent contamination of feed.
- Chicken should be vaccinated against salmonellosis.
- Public health hazard should be minimized by applying strict sanitation in the house hold and Hazard Analysis Critical Control Point system in processing units of poultry products.

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References

1. Allaoui, A., Rhazi, F., Bouchrif, B., Karraouan, B., Oumokhtar, B., Aboulkacem, A. and Essahale, A. (2014): Occurrence and antimicrobial- resistant Salmonella serovars isolated from turkey carcasses and giblets. *AcaJouMic Rea*. 2: 5-12.
2. Ashwani, K., Etsay, K., Yohaness, T., Tsegabirhan, K., Kassaw, A. and Tsegay, T. (2014): Seroprevalence of Salmonella Gallinarum Infection in Chicken Population of Parts of Tigray and Addis Ababa by Plate Agglutination and Microagglutination Tests. *MEJS*. 6: 35-36.
3. Bercheir, A., Fernandes, S. and Gama, Q. (2003): Occurrence of Salmonellosis in laying hens. Brazil: institute of poultry production. Pp: 56-62.
4. Boever, W. J. (2005): Salmonellosis. Lowa: lowa state University College of veterinary medicine. Pp: 5-7.
5. Bouzoubaa, K., Lemainguer, K. and Bell, J. G., 1992. Village chickens as a reservoir of Salmonella Pullorum and Salmonella Gallinarum in Morocco. *Prev. Vet. Med*., 12: 95-100.
6. Charlton, B.R. (2000): Avian Disease Manual, 5thedn, USA, International Book Distributing Co. pp. 101-119.
7. Chauhan, H.V.S. and Roy, S. (2007): Poultry Disease, Diagnosis and Treatment.3rd ed, New age International (p) Ltd, PP. 18-29.
8. chebo, B. (2015): prevalence and antimicrobial susceptiblity profile of salmonella in slaughtered cattle and lactating cows in central ethiopia. msc thesis, addis ababa university college of veterinary medicine and agriculture, bishoftu, ethiopia.
9. Dessie, T. and Jobre, Y., (2004). A review of the importance and control of Newcastle disease in Ethiopia. *Ethiop. Vet*. J., 8:71-81.
10. Dunkley, K., Callaway, T., Chalova, V., Reynolds, J., Hume, M., kubena, L., Nisbet, D. and Ricke, S. (2008): Salmonella ecology in the avian gastrointestinal tract. USA: Department of Poultry Science, Texas A&M University. Pp: 26-27.
11. Eliana, T., Ana, K., Greice, S., Renato, L., Antonio, C., and Ana, C.(2012): Important Aspects of Salmonella in the Poultry Industry and in Public Health.(ed)Biological Institute - Advanced Technological Research Center of the Poultry Agribusiness - Agriculture Secretariat of the State of Sao Paulo, Descalvado/SP Brazil. PP 195-196.
12. FAO, 2007. Poultry sector country review-Ethiopia. FAO animal production and health division, emergency centre for transboundary animal diseases, socio economics, production and biodiversity unit, p 3.
13. Girmay, G. (2013): Evaluating the relative resistance of different poultry breeds to Salmonella Typhimurium. MSc thesis, Addis Ababa University College of Veterinary Medicine and Agriculture, Bishoftu, Ethiopia.
14. Gordon, R.F. and Jordan, F.T.W. (1982): Poultry Disease, 2nd ed, Balliere Tindal, Pp. 9-75.
15. Hossain, S., Chowdhury, E., and Haider, G.(2006): Avian Salmonella infection: isolation and identification of organisms and histopathological study. *Bangl. J. Vet*. Med. 4: 07–12.
16. Ibrahim, M., Emeash1, H., Ghoneim, N., and Abdel-Halim, M.(2013): Seroepidemiological Studies on Poultry Salmonellosis and its Public Health Importance. *J. World's Poult. Res*. 3: 18-23.
17. Kabir, L. (2010): Avian Colibacillosis and Salmonellosis: A Closer Look at Epidemiology, Pathogenesis, Diagnosis, Control and Public Health Concerns. *Int. J. Environ. Res. Public Health* 7: 89-114.
18. Kahin, (2005): The Merck veterinary manual. 9th ed. U.S.A: Kahin and CO., INC, Whitehouse station, N.J., and Merial limited a Merck and Aventis company. Pp 1455-1459.
19. Kassaye, A., Lencho, T., and Mesele, A. (2010): Prevalence of Salmonella\Infection in Intensive Poultry Farms in Hawassa and Isolation of Salmonella species from sick and dead chickens. *Ethiop. Vet*. J.14: 116-119.
20. Keery, L., (2010): Salmonella Enteritidis Control Programs in the Canadian Poultry Industry.

Canada: British Columbia Ministry of Agriculture and Land. Pp 3-4.
21. Kemal J. (2014) A Review on the Public Health Importance of Bovine Salmonellosis. *J VeterinarSciTechnol* 5: 175.
22. Kumara, D., Mishra, S., and Lather, D. (2012): Pathomicrobial studies on Salmonella Gallinarum infection. India: LalaLajpatRai University of Veterinary and Animal Sciences. Pp 727-728.
23. Langridge, G., Nair, S. and Wain, J. (2008): Invasive Salmonellosis in Humans. In Böck, R. C. I. A., Kaper, J. B., Neidhardt, F. C., Nyström, T., Rudd, K. E. and C. L. Squires (eds): EcoSal-Escherichia coli and Salmonella: cellular and molecular biology, ASM Press, Washington, D.C. Available from: http://www.ecosal.org/.
24. Medina, E., Fanos, T., Mesula, G., Teferi, D., and Tariku J. (2013): Sero and media culture prevalence of Salmonellosis in local and exotic chicken. Afr. J. *Microbiol. Res*. 7: 1041-1044.
25. Molla, B., Alemayehu, D., and Salah, W. (2002). Sources and distribution of Salmonella serotypes isolated from food animals, slaughterhouse personnel and retail meat products. *Ethiop.J.Health Dev*.16: 65-68.
26. Molla, W. (2004): Cross sectional study on *salmonella*in apparently healthy slaughtered sheep and goat at Addis Abeba and Modjo abattoir. Msc thesis, Addis Abeba University Faculty of Veterinary Medicine, Debrezeit, Ethiopia.
27. Myint, M. (2004): Epidemiology of *Salmonella* contamination of poultry meat products. U.S.A: Department of Veterinary Medical Sciences. Pp 2-3.
28. Nazir, S., Kamil, A., Darzi, M., Mir, M., and Nazir, K (2012): Pathology of Spontaneously Occurring Salmonellosis in Commercial Broiler Chickens. *J. World's Poult. Res*. 2: 63-69.
29. Nesheim, M.C., Austic, R.E. and Card, L.E. (1979): Poultry Production. 12thed, In Great Britain by Bailliere Tindal, London. Pp. 270-271.
30. Netsanet, B., Berihun, A., Nigus, A., Abreha, T., and kalayou, S.(2012): Seroprevalence of SalmonellaPullorum infection in local and exotic commercial chicken from mekelle area. *REDVET*.13: 2-15.
31. OIÉ (2000): Salmonellosis. In: Manual standards for diagnostic test and vaccines, 4th ed.
32. Pavic, A., Groves, P., and Coex, J. (2008): Control of SalmonellainPoultrythrough Vaccination and Prophylactic Antibody Treatment. Australia: Faculty of Science, University of New South Wales. Pp 116-117.
33. Priyantha, M., Vipulasiri, A., and Gunawardana, G. (2011): Salmonella control in poultry breeder farms in Sri Lanka: Effects of oral antibiotic treatment on whole blood agglutination test with SalmonellaPullorum antigen. *Int. J. Livest. Prod*. 3: 21-24.
34. Radostits, O.M., Blood, D.C., and Gay, C.C. (1994): Diseases caused by Salmonella species. In: Veterinary Medicine, A Text Book of the Diseases of Cattle, Sheep, Pigs, Goats, Horses. 8 th ed., Baillie Tindall, London. Pp 730-745.
35. Rovelledo, L.T. (2012): Avian Salmonellosis, vaccines and immune mechanisms of protection. Bahia, Brazil: Salvador, Pp: 4-7.
36. Sanchez, S., Charles, L., Lee, M., Maurer, J., and Doyle, M. (2002): Animal sources of salmonellosis in humans. *JAVMA*.221: 490-495.
37. Seifer, H.S.H.(1996):Tropical animal health. Kluwer Acadamic Publisher, Dorotrecht. Pp 368-371.
38. Shivaprasad, H.L. (2000): Fowl Typhoid and Pullorum disease. *Rev. Sci.Tech*. 19: 405-424.
39. Sockett, P.N. (1991): The economic implications of human Salmonella infection, a review. *J. Appl. Bacteriol*. 71: 289 – 295.
40. Tadele, G., Asrade, B., Bayleyegn, G., and Ali, M. (2014): Sero-prevalence of Fowl Typhoid and Pullorum Disease from Apparently Healthy Chickens in Eastern Ethiopia. *J VeterinarSciTechnol*. 5: 1-3.
41. Teklu, A. (2008): Prevalence and serotype distribution of Salmonella in slaughtered sheep and goat and abattoir environment in an export abattoir, Modjo, Ethiopia. MSc thesis, Addis Ababa University Faculty of Veterinary Medicine, Debrezeit, Ethiopia.
42. World Health Organization (1988): Salmonellosis Control: The Role of Animal and Product Hygiene, Technical Report Series 774, World Health Organization, Geneva.

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