Pathological and Bacteriological Studies on Chicken Lamenetic Leg in El- Sharkia Province

Rehab E. Mowafy*, Naglaa M. Ali and Taha M. F.*****
Dept. of Pathology* and bacteriology***, Animals Health Research Institute (Zagazig Provincial lab.) & Dept. Of Poultry Diseases** (Assuit Provincial lab.)

Abstract
Fifty live and freshly dead chickens 2 weeks–10 months age with lamenetic legs, were collected randomly from different broiler chickens farms of El-Sharkia Province since June, 2013 for a year. Full historical data were accompanied with each case, specimens from affected foot, hock and stifle joints parts, livers, kidneys, hearts, intestine were collected for pathological and bacteriological examination, blood samples also collected for estimation of calcium (Ca) magnesium (Mg), and inorganic phosphate (P) level in serum. Examination of the ration for detection of mycotoxins was also done. Bacteriological examination revealed 37(74%) positive case for Staph aureus which high sensitive to Augmentin, gentamycin and ciprofloxacillin and all coagulase positive Staph. aureus recovered isolates were resistant to clindamycin, while other cases free from pathogen revealed 2(4%) positive cases for nutritional deficiency of previously mentioned minerals, 5(10%) cases were positive as postvaccinal reaction, while 4(8%) cases subjected to trauma due to overcrowding and finally 2 (4%) positive case for ochratoxin affections were recorded .pathological findings of Staph aureus cases revealed swollen of joints, congestion and leucocytic infiltration of some internal organs especially livers and hearts while positive cases for nutritional deficiency of previously mentioned minerals demonstrating lesions in bone and intestine, edema with stiffness of joints were recorded in postvaccinal positive cases, while ochratoxin affection positive cases revealed congested livers and kidneys with mononuclear cell infiltration. Brocken legs and hock joint dislocation were the common character of traumatic cases.

Key words:
Lamenetic Leg, Pathological, Bacteriological Studies, staphyloccuc aureus.

Introduction

The bacterium is considered to be a normal flora of the chicken isolated from the skin and feathers as well as in the respiratory and intestinal tracts **(Skeeles., 1997 and Casey *et al.*, 2007).** However, some of the common forms of S. aureus associated poultry infections include tenosynovitis **(Butterworth., 1999),** omphalitis **(Hill *et al.*, 1989),** femoral head necrosis, infected hock and stifle joints secondary to coccidiosis or vaccine reactions **(McNamee & Smyth 2000)** and "pumble foot" **(Skeeles, 1997).**

The disease has been seen in birds ranging from 14 to 70 days of age, but most cases occurred around 35 days old **(McNamee and Smyth, 2000)**. Disease is most commonly caused by Staphylococcus aureus, which is of veterinary importance in broiler and breeders. In such birds, the most common form of infec-

tion involves tenosynovitis (inflammation of tendon sheaths) and stress caused by uneven feed distribution or predispose to Staphylococcal infection. The affected joints, usually the hocks, are hot, swollen and painful ,the affected birds are usually depressed, lameness and reluctant to walk-in tenosynovitis, the synovial membranes of tendon sheaths become thickened and edematous, with fibrinous exudate within and around the tendon sheaths, **(Jordan *et al.,* 2002).** Staph aureus produce high morbidity and low or no mortality, the organism has tropism for growing bone and liver that revealed focal hepatic parenchyma and around the blood vessels while joints revealed destruction of articular cartilage with areas of necrosis and necrosis of chondrocyte **(Hebat-Allah *et al.,* 2oo6).** Lameness occurs when the ends of the long bones become enlarged and exceedingly flexible due to improper calcification. This often facilitates the development of a bowed appearance, most noticeable at the proximal end of tibia **(Wise**, **1975**). **Julian, (1998)** suggested that a low Ca/ P ratio due to a low Ca level would increase leg deformity, that nutritional deficiencies can result in skeletal disease in all birds. Also **(Claudia**, **2007)** reported Nutritional imbalances, mechanical trauma and environmental factors can also play a role in leg problems. Inoculation of 28 days old SPF (specific pathogen free) chicken with MS WVU1853 strain by IN route ,and at 14 day post exposure leads to swollen of tibio-tarsial joint that come normal few days later **(Kawakubo *et al .,* 1980)**, while inoculation of SPF one day old chicks via foot pad with the same strain, leads to gross arthritic lesions **(Lockaby *et al.,* 1998).** An Arthurs reactionis large localized reaction characterized by pain, swelling, induration and edema, it usually begins within 48 hours following immunization **(Brunswick., 2011).** The effect of ochratoxin A (OTA) revealed marked degenerative changes in the kidney and bursa and mononuclear cell infiltration was recorded in the liver. High dose (400-800ppb) affecting broiler performance and movement ,while higher doses leads to death. **(Elaroussi *et al.,* 2008).**

This study aimed to identify the most different causative agents for lameness in El-Sharkia province as possible as available ,and the associated pathological changes.

Material and Methods

Isolation and identification of Staph. aureus

A total of fifty (42 live and 8 freshly dead), (Cobb, Hubbard, Isa brown) chickens 2 weeks-10 months age collected from different flocks in El-sharkia province suffering from lameness were tested for the presence of S. aureus. Samples of affected joints were cultured on 10% NaCL broth at 37 for 24 h and subsequently inoculated onto Mannitol salt agar at 37 for 24h. S. aureus was bacterial isolates were identified by using conventional methods which included Gram staining and microscopy, catalase, oxidase, coagulase tests as described as **(Quiun *et al.,* 2002).**

Antimicrobial susceptibility test

Antimicrobial susceptibility Test **(AST)** was carried out for the coagulase positive S. aureus isolates using disc diffusion method according to **(Quiun *et al.,* 2002).** Briefly, each of the S. aureus isolates was inoculated into nutrient broth (Oxoid, UK) and incubated at 37°C for 24 hours before they were tested. About 0.1 ml of the nutrient broth culture was subsequently inoculated onto Nutrient agar plates and spread over the surface with sterile cotton swabs. Antimicrobial discs were then placed on the surface of each plate by means of antibiotic disc dispenser and incubated at 37°C for 24 hours. Diameters of inhibition zone were measured using a transparent ruler and the interpretative breakpoints for resistance were determined by comparing zone diameters as recommended by **(Quiun *et al.,* 2002).** The coagulase positive S. aureus isolates were tested against a panel of 11 antimicrobials.

Blood serum concentrations of magnesium (Mg), calcium (Ca) and inorganic phosphate (P) were assayed on an automated analyzer in 6 samples obtained from each flock by the time of observing clinical signs and gross lesions

typical for rickets. serum calcium and magnesium were determind according to **(Tietz, 1976).** Serum inorganic phosphorus was determined according to **(Young *et al.*, 1975).**

Ration analysis for fungal toxin

Samples from rations were analyzed by **Flurometer** in Animals Health Research Institute (Zagazig Provincial lab.) for detection of fungal toxin presence **(Scott & Hand, 1967).**

Pathological studies

Diseased birds were necropsied, specimens from Joints affected parts, foot, footpad, hearts, livers, intestine and kidneys were collected and fixed in 10% neutral buffered formalin solution. Paraffin sections of 5 µ thickness were prepared and stained with haematoxylin and eosin for histopathological examination **(Bancroft and Stevens, 1996)**.

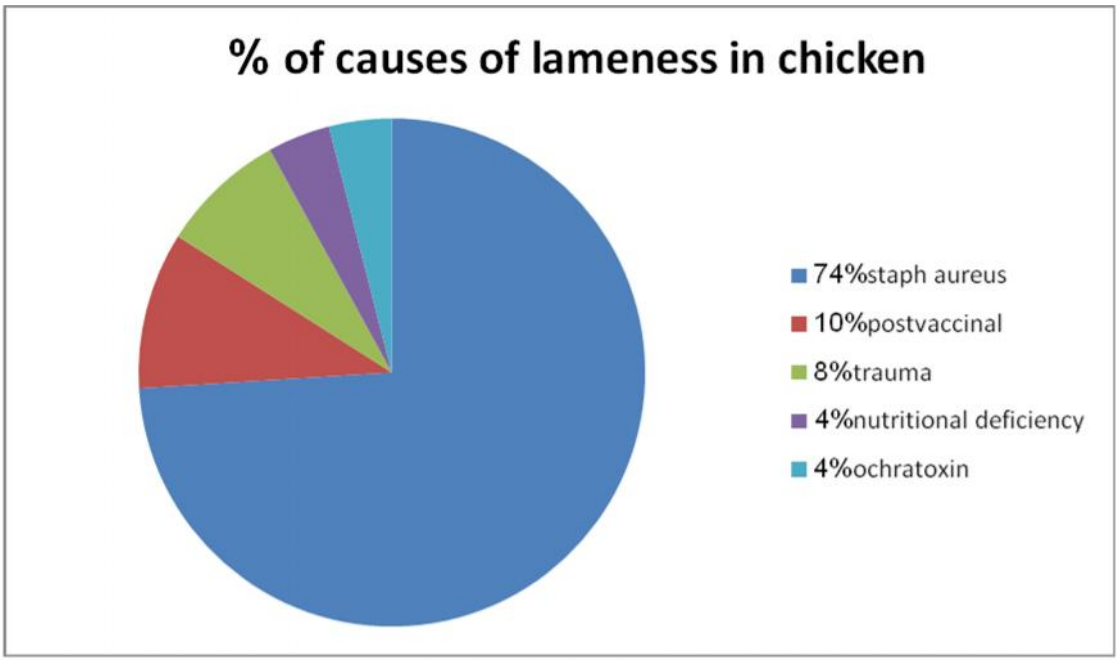
Results

Bacteriological results. A total of 37 (74%) Staph. aureus coagulase positive isolates were obtained from fifty articular tissue (foot, hock and stifyle joints) samples of chicken at 4-23 week old of age, suffering from lameness while the other 12 cases were free from pathogen.

Antimicrobial resistance of coagulase positive ,all the coagulase positive S. aureus isolated in this study were resistant to clindamycin but all of recovered isolates were susceptible to Augmentin, gentamycin and ciprofloxacillin(100%) and (91.6%) of the coagulase positive S. aureus isolates were susceptible to both vancomycin and sulphamethoxazole. This was followed by enrofloxacin (83.3%), cefriazone (75%) and tetracycline, chloramphenicol and erythromycin (50%).

Table 1. Staphylococcus aureus isolates from lamenitic chickens.

Number resistant%	Antimicrobiles
0\12(100%)	Augmentin (AMC)
0\12(100%)	Gentamycin (CN)
0\12(100%)	Ciprofloxacin (CIP)
1\12(91.6%)	Vancomycin (VA)
1\12(91.6%)	Sulphamethoxazole(SXT)
2\12(83.3%)	Enrefloxacin (ENR)
3\12(75%)	Cefriazone (CRO)
6\12(50%)	Tetracycline (TE)
6\12(50%)	Chloramphenicol (C)
6\12(50%)	Erythromycin (E)
12\12 (0%)	Clindamycin (DA)



other 13 cases free from pathogen revealed 5 (10%) cases were lamenitic as results of postvaccinal reaction, 4(8%) cases subjected to trauma, due to overcrowding and 2 (4%) cases suffering from lameness as a result of minerals deficiency cases, while2 (4%) were toxic with ochratoxin.

Blood serum concentrations results.

Six blood samples of diseased, suspected for nutritional deficiencies and normal chickens were analyzed, two cases showed hypocalcaemia (7.56& 6.93mg/dl) with 19%-26% reduction percent when compared with the normal values which were (9.43 & 9.24 mg/dl) and hypomagnesaemia (1.o2&1.08 m mol/L) with 9 %-14 % reduction percent when compared with the normal values which were (1.20 &1.18 m mol/L), while inorganic phosphorus showed decrease in values which were (4.33-4.40m mol/L) with reduction percent 2.7-8.1% when compared with the normal values which were (4.68-4.52m mol/L). Ca /P ratio was reduced and became unbalanced in comparison with normal values 2:1.

Ration analysis for mycotoxin detection.

Two broiler farms 3 &4 weeks age suspected for mycotoxicosis**,** ration analysis were done and ochratoxin was detected by high doses 760 & 800ppb. one case of each farm showed lamenitic leg with bad movement.

Pathological findings.

Clinical signs

Confined to depression, lameness, unable to stand, affected foot and hock joints are hot, swollen, painful and reluctant to walk-in tenosynovitis specially in bumble foot +ve that affecting the movement and activities of birds according to the degree of swelling.

Macroscopical findings.

Most of bumble foot +ve chickens for staph aureus demonstrated swollen foot and toe pads **(Fig.1)** synovial membranes of tendon sheaths become thickened and edematous, with fibrinous exudate within and around tendon sheath, greenish pasty material oozed from opened swelling area some were tinged with blood. congestion of liver and heart were detectable in some cases. Nutritional deficiency +ve chickens for some minerals as magnesium (Mg), calcium (Ca) and inorganic phosphate (P) showing weak, friable legs. Some sever cases showed curvature of keel bone **(Fig.12)** most cases showed enteric lesions. Postvaccinal reaction positive cases showed edema and stiffness of leg **(Fig.16),** the edematous fluid was serous in nature, internal organs appeared normal. Ochratoxin +ve severe cases showed fragil legs, Internal organs showed sever congestion specially liver **(Fig.17)** and kidney.

Microscopical findings.

Pumble foot +ve chickens for staph aureus demonstrated edema and hyalinization of the connective tissue of the skin **(Fig.2).** Interstitial aggregation of mononuclear cells in addition to hemorrhage were demonstrated in adipose tissue of footpad **(Fig. 3&4).** Necrosis of chondrocyte was also detected in cartilage **(Fig. 5)**, while bone suffering from hyperplasia of extra haversian osteoblasts represented by numerous dark basophilic lacunae containing osteocytes **(Fig.6),** while heart demonstrating congestion of cardiac blood vessel with perivascular edema **(Fig.7)**, focal aggregation of mononuclear cells with few heterophils within hepatic parenchyma was noticed **(Fig. 8&9)** thrombi with perivascular fibrosis in the hepatic tissue were seen **(Fig.10)**, while epidermis of the affected foot demonstrated massive aggregation of mononuclear cells with few heterophils **(Fig.11).** Positive cases for nutritional deficiency demonstrating osteomalicia in bone **(Fig.13),** while cartilage showed chondromalacia **(Fig.14),** intestine suffering from sloughing of intestinal villi **(Fig.15)** and parts of intestinal mucosa. Ochratoxin positive cases showed severe congestion of liver and kidney which demonstrating mononuclear cells infiltrating the renal medulla, while skin of hock joint showed hyperkeratosis of dermis and vacuolation of stratum spinosum **(Fig.18).** Postvaccinal reaction +veand traumatic cases showed no microscopical alteration neither in affected joints nor other internal organs.

Discussion

In the present study Staphylococcus aureus was isolated at the percentage of 74%,tested cases suffering from arthritis symptoms. **Selim *et al.*,(2009)** revealed isolation of (74.5%) of Staph aureus from 3weeks-12monthes old. The bacterium is considered to be normal resident of the chicken, located on the skin, feathers and in the respiratory tracts. This organism must enter the circulatory system to cause disease, thus probability of infection is increased by any injury that provides the bacteria with rout of entry, **(Butterworth**,**1999)**. Arthritis is most commonly caused by Staphaureus, Clinical signs confined to depression, lameness, swollen joint, and unable to stand, **(Hofstad *et al.*, 1984 & McNamee and Smyth, 2000).**

The most frequent sites of Staph. aureus infections in poultry were bones, tendons, sheaths and joints, especially tibio-tarsal and stifle joints **(Adayel**., **2005**).

Staph aureus is the most common bacterium recovered from leg and joint infections of poultry **(Skeeles, 1997)**, and it has been frequently recovered from bone infections of commercial broilers; **(McNamee, 1998).** In this study Staph. aureus isolated from 74% lamenitic cases suffering from pumble foot, Staphylococcus aureus has been cultured from 90% of the spontaneously occurring cases of pumble foot, depending upon avian species and environment **(Satterfield and O'Rourke, 1981)**.

Pododermatitis or foot dermatitis; is characterized by massive swelling and ulceration of the plantar and/or digital pads; causes lameness, impedes walking; and may limit access to food and water. If left untreated, pumble foot will compromise the internal tissues of the foot, such as the tendons and bones, causing osteomyelitis, synovitis, laminitis, and eventually death **(McNamee and Smyth**, **2000).**

Wei *et al.*, (1995) have suggested that skin injury around the joint may play a very important role in the development of bacterial chondronecrosis or arthritis. vaccination or minor surgical procedures are all possible routes of S. aureus entry into the bloodstream. S. aureus is considered to be normal resident of the chicken, located on the skin, feathers and in the respiratory tracts, **(Mutalib *et al.*, 1993).** This organism must enter the circulatory system to cause disease, thus probability of infection is increased by any injury that provides the bacteria with rout of entry**. McNamee & Smyth (2000) and Reasen**, **(2003),** reported that in 6 to 12-wks. old chickens, Staphylococcus arthritis caused by trauma is a chronic or secondary disease. When the mucosal or skin barriers have been compromised due to trauma or stress, S. aureus can invade the mesoderm, proliferate and cause inflammation and arthritis, **(Wilcox *et al.*, 2009).**

Joint affection occurred within 48-120 hours post infection. The affected joints showed increase of synovial fluid which was turbid and yellowish, similar findings by **(Jensen, 1990).** Staphylococci have a high affinity for collagen -rich surfaces such as the articular surface of joints, and synovial sheaths located around joints and tendons. This organism also tends to localize in the growth plate of actively growing bones, **(Jordan and Pattisson, 1996).**

In the present study, 4 cases (8%) suffering from lameness as a results of trauma, this result is agree with **(Claudia, 2007)** Mechanical trauma associated leg problems is more prevalent in fast growing chickens. Specifically handling the birds during the catching can result in femoral head necrosis whereby the head of the femur ruptures causing hemorrhage. Mechanically induced or trauma associated problems are much more frequent in fast-growing broilers, **(Julian**, **1998),** although, about 5 cases (10%) of tested and free from pathogens, suffering from lameness by the cause of postvaccination reaction. **Cargill., (2006)** accuracy of placement of the needle is critical as incorrect placement can lead to granuloma formation, lameness, the method and care of handling birds during this procedure is also important as this can contribute to lameness **(Mutalib and Boyle, 1994).** Inflammatory reaction have been observed in chicken injected intramuscular with killed vaccines such as depression, loss of appetite and present various degree of lameness. The reaction is due to circulating antigen antibodies complex prior to injection of the antigen, this result in massive swelling at the injection site that may involve the entire leg or limb **(Brunswick, 2011).**

Clinical signs and postmortem findings confined to depression, lameness, swollen joint, and unable to stand **(Hofstad *et al.,* 1984 and Jordan *et al.,* 2002).**

About 2 (4%) of tested cases exhibit lameness as a result of minerals deficiency, **Mg**, **Ca** and **P**, Lameness occurs when the ends of the long bones become enlarged and exceedingly flexible due to improper calcification. This often facilitates the development of a bowed appearance, most noticeable at the proximal end of tibia **(Wise, 1975)**. Avoiding a low Ca:P ratio in the diet have shown to reduce the incidence of leg problems in flocks **(Claudia, 2007)**. Deficiency of Ca and P or improper ratio balance lead to soft poorly mineralized bones causing flexibility in long bone causing lameness. **(Wise, 1975)** who suggested that a low Ca/P ratio due to a low Ca level would increase leg deformity. In support of this finding, **(Julian, 1998)** reported that nutritional deficiencies can result in skeletal disease in all birds. also **(Claudia, 2007)** reported Nutritional imbalances, mechanical trauma and environmental factors can also play a role in leg problems.

AYO and Minka, (2004) reported that the cause of leg lameness may be multifactor in nature, including nutrition, genetics, over feeding, lack of exercise and trauma to the lower limbs.

positive cases of ochratoxins were explained by **(Verma, 2006)** who mentioned that mycotoxin such as aflatoxins and ochratoxin lead to bone fragility and leg weakness due to their toxic effects on liver and kidney which consequently prevents the conversion of active form of vitamin D3 and its absorption. Toxins in feed and water can cause skeletal deformities and lameness in broilers chicken, **(Julian, 1998).**

All of 12 coagulase positive Staph .aureus isolates recovered were resistant to clindamycin, and all of recovered isolates were susceptible to gentamycin, augmentinand spirofoxacin, this result agree with **(Suleiman *et al.,* 2013 and Waters *et al.,* 2011),** who reported that all recovered S. aureus isolates were susceptible to gentamycin and spirofloxacillin. While **(Adyel, 2005 and Rasheed, 2011)** disagree with these results, reported that antibiotics susceptibility of Staph. aureus was found to be resistant to gentamycin and (33.3%) of isolates were resistant to Augmentin. **Otalu *et al.,* (2011)** reported that susceptibility of S. aureus isolates were (61.5%), (77%) and (84.6%) to gentamycin, augmentin and ciprofloxacin, respectively. sensitivity of the isolates to ciprofloxacin, gentamycin, and cephalexin may be a

pointer that these antimicrobials are less abused in the studied area and may possibly be recommended for treatment of Staphylococcal infections in poultry.

50% of Staph aureus isolates were resistant to erythromycin, tetracycline and chloramphenicol but **(Waters *et al.,* 2011 and Suleiman *et al.,* 2013**) recorded all Staph. aureus isolates were resistant to erythromycin. **Otalu *et al.,* (2011)** reported that all S. aureus isolates were resistant to erythromycin and tetracycline. Susceptibility of chloramphenicol (50%) in this study and these findings agree with **(Otalu *et al.,* 2011)** reported that sensitivity of S. aureus isolates to chloramphenicol were (46.9 %).

In this study the Susceptibility of Staph aureus recovered isolates to vancomycin and Sulphamethoxazole were 91.6%, but **(Otalu *et al.,* 2011)** reported that susceptibility of S. aureus isolates to vancomycin and Sulphamethoxazole were (46.2%) and (38.5%) respectively. All Staph aureus isolates were resistant to clindamycin, while **(Suleiman *et al.,* 2013)** recorded (53%) of S. aureus isolates were resistant to clindamycin, and (51.9%)of isolates were resistant to ceftriazone. Thus, sub -therapeutic use of such drugs as prophylaxes, growth promoters or inaccurate dosages given to sick flocks by unqualified personnel may likely result in plasma concentrations that are inconsistent with the desired objectives which might possibly have led to the level of resistance observed in the present study **(Suleiman *et al.,* 2013).**

Pathological findings. Pumble foot +ve cases demonstrated that the hepatic lesions in all-Staph aureus isolates were in partial aggrement with **(Hebat-Allah *et al.,* 2006)** who found that the hepatic lesions were necrosis and congestion and full aggrement with **(Selim *et al.,* 2009). Kirk skeels, (1997)** explained the formation of thrombi as sequel to the enterotoxin of staph which has adverse effect on blood vessel and blood composition. The heart lesions were in partial aggrement with **(Gehan., 1999 and Selim *et al.,* 2009)**, while the joint lesions were partially similar to that obtained by **(Hebat-Allah *et al.,* 2006 and Selim *et al.,* 2009)**. The results of skin lesions were supported by **(Gehan., 1999).**

Positive cases of nutritional deficiencies and it's association with enteric lesions were explained by **(Coles., 1986)** who mentioned that hypocalcaemia occurred due to inability of intestine to absorb principle substances in the food due to enteritis also he mentioned that serum calcium concentration decrease with hypoproteinaemia because of the reduced amount of protein bound calcium. Our results showed partial disagreement with **(Atia., 2001)** who showed hyperplasia in the articular surface and increase width of proliferative zone in the diseased duck bone in addition to edematous fibrous tissue and necrosis of chondrocyte. this may contributed to severity of cases and sex variation. while our macroscopically results were agreement with him as bone softening, easily Brocken leg.

Positive cases of ochratoxins were explained by **(Verma, 2006)** who mentioned that mycotoxin such as aflatoxins and ochratoxin lead to bone fragility and leg weakness due to their toxic effects on liver and kidney which consequently prevents the conversion of active form of vitamin D3 and its absorption, beside nutrition and management skeletal disorder found in poultry can be caused by mycotoxins in the feed

Concolusion

Staphylococcal infection is the most real important causes of arthritis leading to economic losses in the broiler chickens as high morbidity swollen joint thus we must take care to control the Staphylococcal infection in poultry

Recommendation. only 50 cases from total 63chicken with lameness were etiologically identified and used so, further studies should be applied on lameness in chicken to identify other causative agents not included in our research.

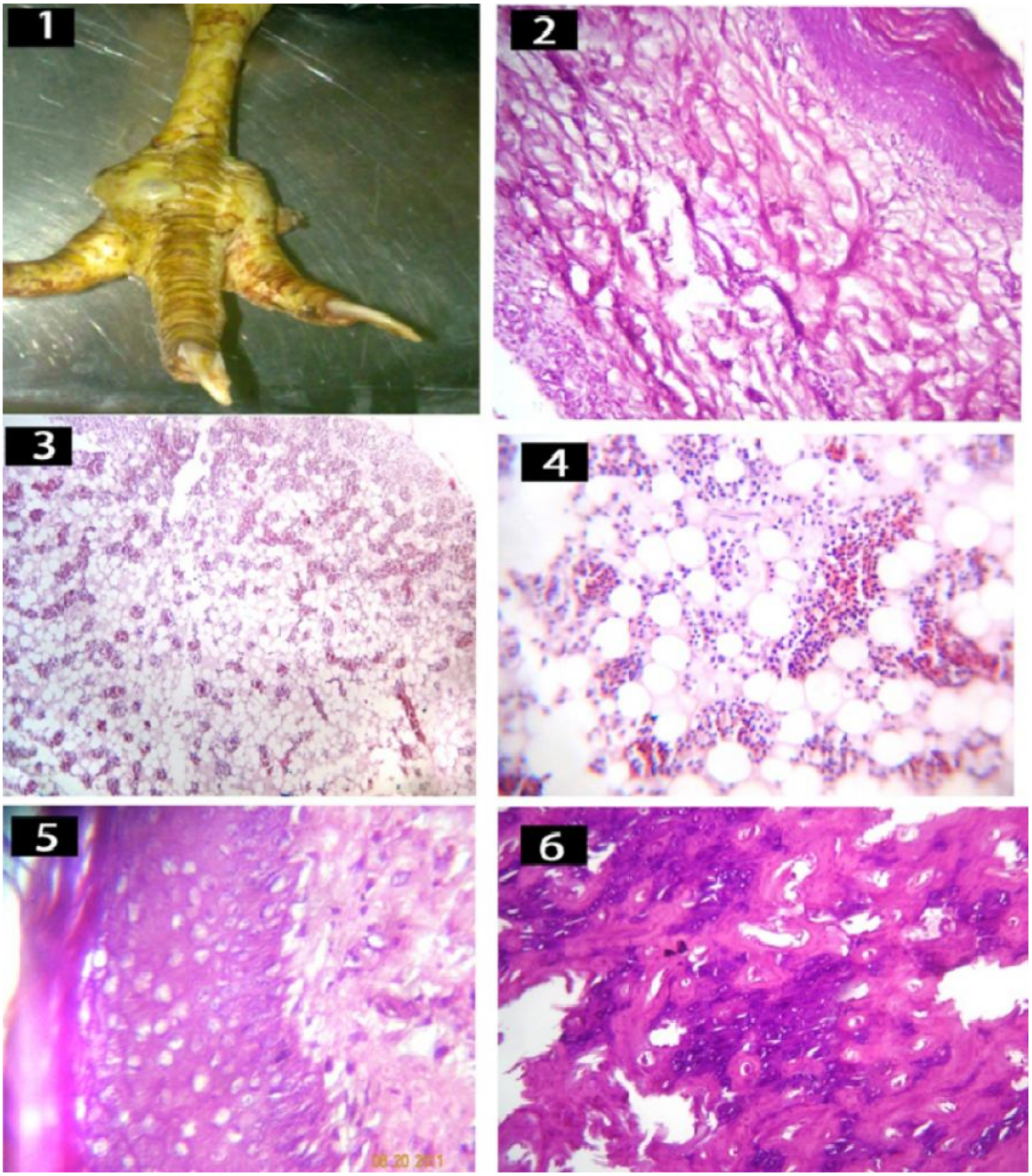


Fig (1). Foot of pumble foot positive chicken showing swollen foot and toepads.
Fig (2). Photomicrograph of skin of bumble foot +ve bird demonstrating interstitial edema and hyalinization of the connective tissue (H&EX200).
Fig (3). Photomicrograph of foot pad adipose tissue of bumble foot +ve chicken demonstrating interstitial aggregation of mononuclear cells with haemorrhage (H&Ex100).
Fig (4). High power of the previous figure to show the mononuclear cells and BCS (H&EX200).
Fig (5). Photomicrograph of cartilage of bumble foot +ve chicken demonstrating necrosis of chondrocytes(H&EX200)
Fig (6). Photomicrograph of bone of pumble foot +ve chicken demonstrating hyperplasia of extra haversian osteoblasts represented by numerous dark basophilic lacunae containing osteocytes (H&EX300).

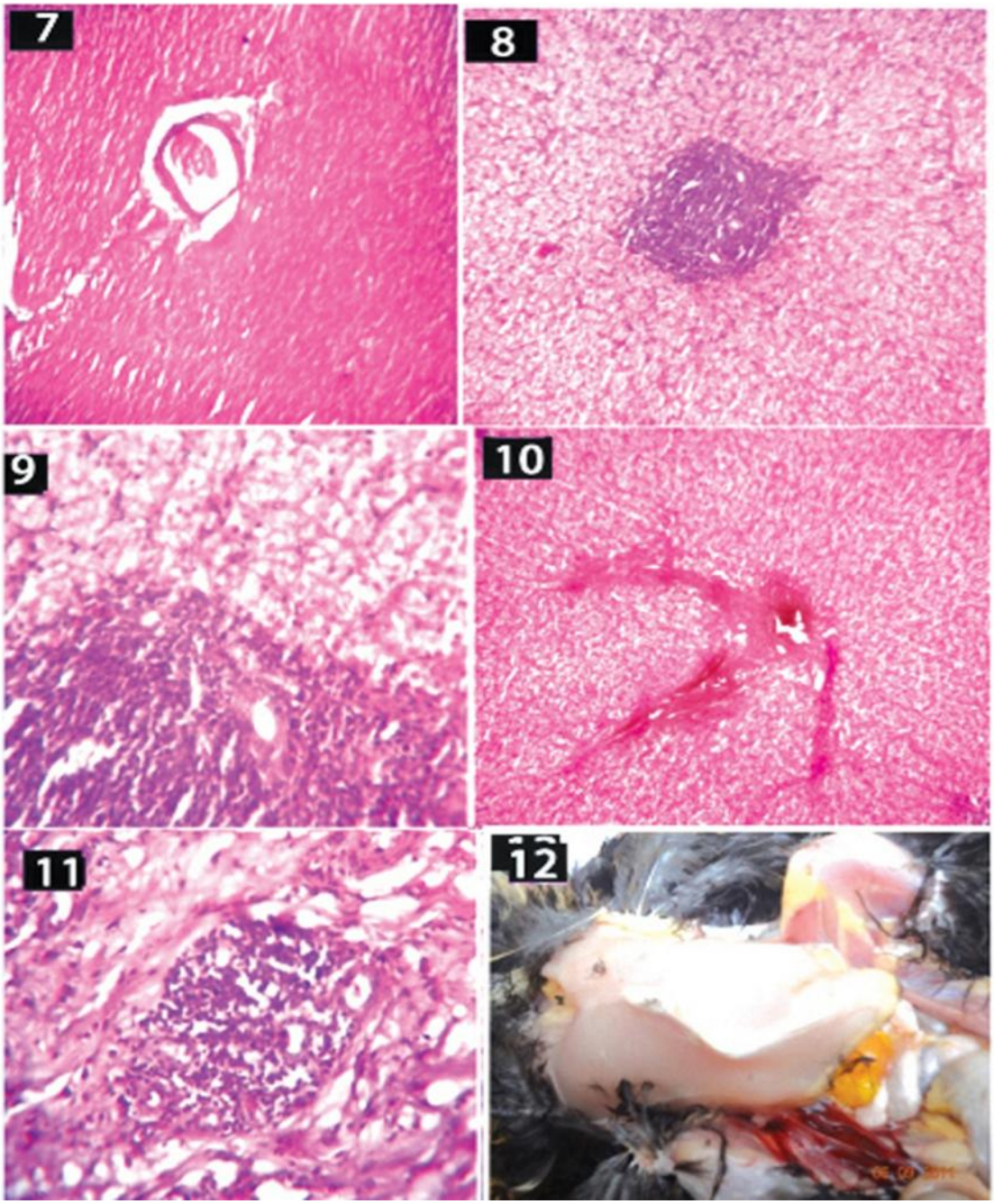


Fig (7). Photomicrograph of heart of pumble foot +ve chicken demonstrating congestion of cardiac blood vessels with perivascular edema (H&EX200).

Fig (8). Photomicrograph of liver of pumble foot +ve chicken demonstrating focal aggregation of mononuclear cells with few heterophiles within hepatic tissue (H&EX100).

Fig (9). High power of the previous figure to show mononuclear cells with few heterophile (H&EX300).

Fig(10). Photomicrograph of liver of pumble foot +ve chicken demonstrating thrombi with perivascular fibrosis (H&EX100).

Fig(11). Photomicrograph of epidermis of pumble foot +ve chicken demonstrating massive aggregation of heterophiles (H&EX300).

Fig(12). Keel bone of ca defeciency positive chicken showing irregularity and curvature.

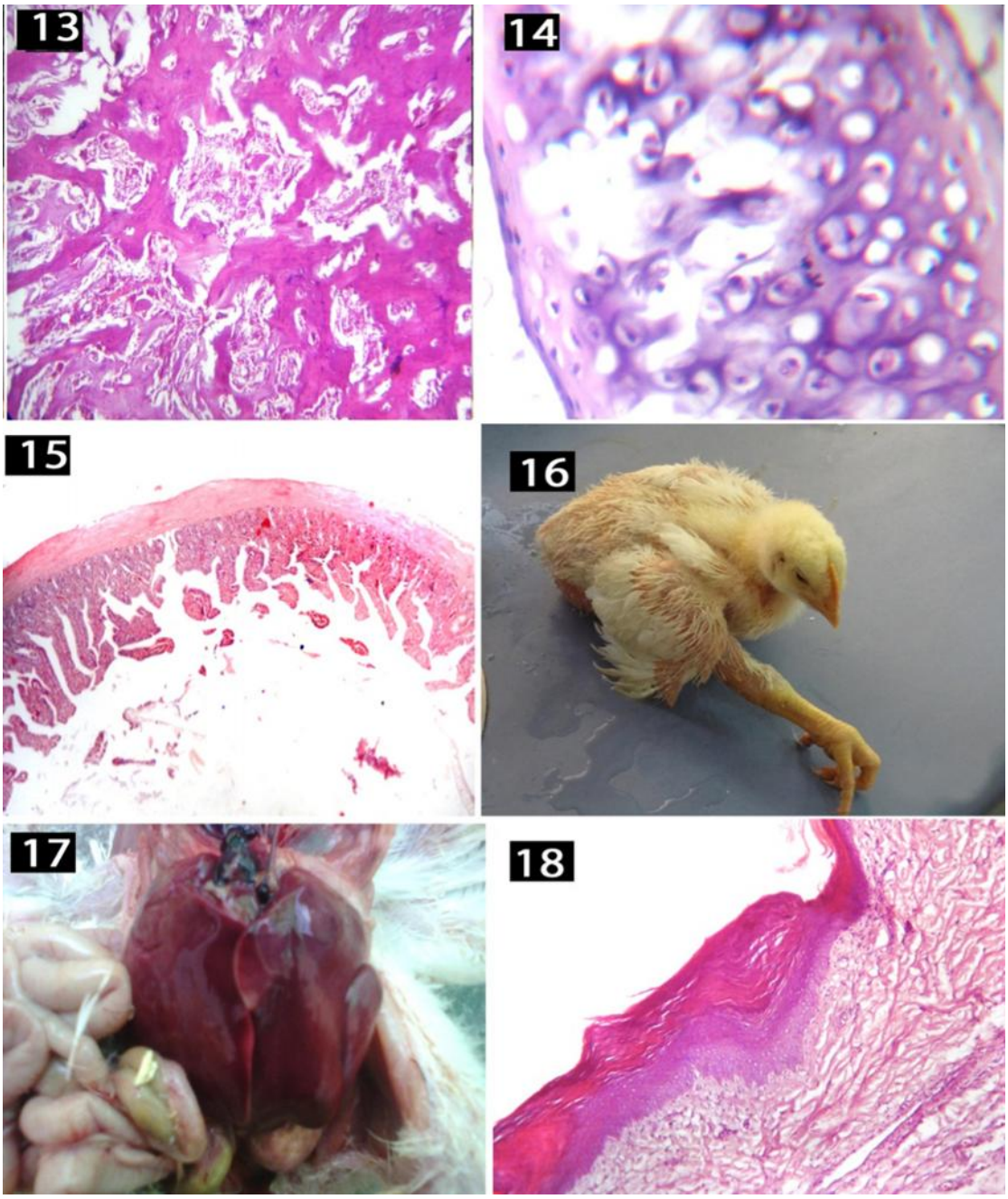


Fig (13). Photomicrograph of bone of ca defeciency +ve chicken demonstrating focal osteomalicia (H&EX300).
Fig (14). Photomicrograph of cartillage of ca defeciency +ve chicken demonstrating necrosis of chondrocyte with focal chondromalicia (H&E x300).
Fig (15). Photomicrograph of intestine of ca defeciency +ve chicken demonstrating sloughing of some intestinal villi (H&EX300).
Fig (16). Postvaccinal reaction positive chicken demonstrating edema and stiffness of leg.
Fig (17). Ochratoxin positive chicken demonstrating severely congested liver.
Fig (18). Photomicrograph of skin of Ochratoxin positive chicken demonstrating hyperkeratosis of dermis and vacuolation of stratum spinosum (H&EX200).

References

Adayel S.A. (**2005).** Incidence of Staphylococcus aureus causing Arthritis of broiler breeders. 4th Int. Sci. Conf. Mansoura.; 65-70.

Andreasen C.B. (2003). Staphylococcosis. In: Diseases of Poultry; 11th Ed, (Saif YM, ed), Iowa State Press, Ames, IA, 798-804.

Atia A.S.H. (2001). Some pathologic studies on growing ducklings suffering from locomotor disturbances ph. D thesis Fac. of vet. medicine zagazig university Pathology dep**.**

AYO, J.O. and MINKAN. S. (2004). Some major constraints on ostrich productivity in Northern Nigeria. In: Proceedings of the Annual Conference of Animal Science Association of Nigeria. 13 - 16, 2004. Ebonyi State University, 155 – 157.

Bancroft J.D. and Stevens A. (1996). Theory and Practice of Histological Technique. 4th Ed., Churchill, Livingston, New York, London, San Francisco, Tokyo.

Butterworth A. (1999). Infectious components of broiler lameness: a review. poultry science Journal, 56 (4): 327-352.

Brunswick textbook (2011). Adverse events following immunization: interpretation and clinical definitions guide version 1- the office of the chief medical office of health communicable disease control unit, pp. 1-30

Cargill P.W. (2006). Vaccine administration to poultry flocks. Merial avian business unit, UK. December.

Casey A.L, Lambert P.A. & Elliot T.S.J. (2007). Staphylococci. International journal of Antimicrobial Agents, 29, 23–32.

Claudia S. Dunkley.(2007*)* The Cooperative Extension service officers. College of agricultural and Environmental Sciences / Athens, Georgia 30602-4356

Coles, E.H. (1986). Veterinary clinical pathology 4thed. WB. Saunders company, Philadelphia, London and Toronto.

Elaroussi M.A., Mohamed F.R., Elgendy M.S., Elbarkouky E.M., Abdou A.M. and Hatab M.H. (2008). Ochratoxicosis in broiler chickens :functional and histological changes in target organs international journal of poultry science 7 (5):414-422 ,ISSN 1682-8356.

Gehan N. Abdel-Wahab (1999). Histopathologic studies of staphylococcus aureus and its toxin on chickens. M. V. Sc. thesis, fac. Vet. Med., Zagazig Univ.

Hebat-Allah, A. Mohamed; Manal H. Thabit and Neveen A. El-Nisr (2006). some studies on staphylococcus infection in chicken in Assuit Governorate. Assuit. Vet. Med. J., 5 (111):163-178.

Hofstad M.S., Barnes H.J., Clanek B.W., Reid W.M., Yoder H.W. (1984). Diseases of poultry. 8th ed. USA: Iowa state university press, Ames, Iowa; 264-265.

Hill J.E., Rowland G.N., Glisson J.R. & Villegas P. (1989). Comparative microscopic lesions in Reoviral and *Staphylococcal tenosynovitis.* Avian Diseases, 33: 401-410.

Jordan F., Pattison M., Alexander D., Faragher T. (2002). Poultry diseases. 5th ed., p. 163, W.B. Saunders;. London.

Jordan F.T., **Pattisson M. (1996).** Poultry diseases. 4th ed. London: W.B. Saunder-Company ltd: 66-67.

Jensen M.M. (1990). An overview on the pathogenesis of Staphylococcosis and an update on Staphylococcal interference. In proceedings of the Avian skeletal disease symposium. AAAP/AVMA, San Antonio,79-82.

Jensen, M.M., Downs, W.C., Morrey, J.D., Nicoll, T.R., LeFevre, S.D. and Meyers, C.M. (1987). Staphylococcosis of turkeys. Portal of entry and tissue colonisation. Avian Diseases, 31, 64-69.

Julian R.J. (1998). Rapid Growth Problems**:** Ascites and Skeletal Deformities in Broilers*,* University of Guelph**,**, Poultry Science, 1773 -1780.

Kawakubo Y., KumeK., Yoshioka M., Nishiyama Y. (1980). Histo-and immunological studies on experimental mycoplasma synoviae infection of the chicken. J. comp. pathol. 90: 457-467.

Kirk Skeeles J. (1997). Staphylococcosis. Diseases of poultry 10thEd. B. W. Calnek. Ed. Iowa state univ. Press, Ames,. 247-253.

Lockaby S.B., Hoerr F.J., Lauerman L.H., Kleven S.H. (1998). Pathogenicity of mycoplasma synoviae in broiler chicken .vet. path. 35:178-190.

McNamee, P.T. (1998). Investigation Of Bacterial Chondronecrosis With Osteomyelitis In Broiler Chickens Doctor Of Philosophy, The Queen University Of Belfast.

McNamee P.T., Smyth J.A. (2000). Bacterial chondronecrosis with osteomylitis (femoral head necrosis) of broiler chickens: Avian Pathology.; 29 (5): 477-495.

Mohan K., Shroeder-Tucker L.C., Karenga D., Dziva F., Harrison A. and Muvavariwa P. (2002). Unidentified Coryneform Bacterial strain from cases of polyarthritis in Chickens :phenotype and fatty acid profile. Avian Diseases.; 46: 1051-1054.

Mutalib A.A., **Boyle C.R. (1994).** Influence of site of inoculation of activated vaccines on the immune response in chickens. Avian Dis. 38: 857-860.

Mutalib A.A., **King J.M.**, **and McDonough P.L. (1993).** Erysiplelas in caged laying-chickens and suspected erysipeloid in animal caretakers. J. Vet. Diagn Invest.; 5: 198-201.

Otalu O.J., **Kabir J.**, **Okolocha E. C. and Umoh V.J. (2011).** Multi-drug Resistant Coagulase Positive Staphylococcus aureus from Live and Slaughtered Chickens in Zaria, Nigeria. International Journal of Poultry Science 10 (11): 871-875.

Rasheed B.Y. (2011). Isolation And Identification Of Bacteria Causing Arthritis In Chickens . Iraqi Journal Of Veterinary Sciences, 25, (2): 93-95

Satterfield, W.C., and K.I. O'Rourke. (1981). Staphylococcal bumblefoot [claw disease of birds]: Vaccination and immunomodulation In the early treatment and management. Zoo Anim, 12: 95-98.

Scott P.M. and H. and T.B. (1967). Method for the detection and estimation of ochratoxin A in some cereal products. J. Assoc. offic. Anal. Chemists 50, 366-370.

Selim, M.A., Elwan, I.F., Hassan, M. Moussa and Magda M.M. Moursy (2009). Pathological and bacteriological studies on staphylococcus Aureus infection in chickens at Sharhia Governorate. 6th Inter. Sci. Conf., Mansoura 22-23 April 2009.

Skeeles J.K. (1997). Staphylococcosis. In*:* Diseases of Poultry. 10th edition (BWCalnek editor). Iowa State University Press, Ames. 247-253.

Suleiman A., L.T. Zaria, H.A. Grema and P. Ahmad U. (2013). Antimicrobial resistant coagulase positive *Staphylococcus* aureus-from chickens in Maiduguri, Nigeria. Sokoto Journal of Veterinary Sciences. 51-55.

Tietz, N.W. (1976). Fundamentals of clinical chemistry. W.B. Saunders Co, Philadelphia. 917-919.

Quiun P.J., Markey B.K., Carter M.E., Donnelly W.J., Leonard F.C. (2002). Veterinary microbiology and microbial diseases. USA: Blackwell Publishing Company; 23-26.

Verma S. Devendra (2006). Mycotoxins affect bone structure and leg weakness**.** World Poultry Journal, 22 (11): 35-37.

Waters, A.E., T. Contente-cuomo, J. Buchhugen, C.M., Liu, L. Watson, K. Pearce, J.T. Foster, J. Bowers, E.M. Driebe, D.M. Enge, Thaler, P.S. Keim and L.B. Price (2011). Multidrug- Resistant Staphylococcus aureus in US meat and Poultry. Clin. Infect. Dis. 52: 1-4.

Wei H.J., K.Y. Cheng, C.P. Cheng and Z.L. Nin (1995). Experiment sonportal of infection of arthritis type staphyloccosis in chicken. Journal of Hunan Agricultural College, 21: 89-92.

Wilcox, C.S.J. Patterson,J. and H.W. Cheng, H.W. (2009). Use Of Thermography t- Screen For Subclinical Bumblefoot in Poultry. Science, 88: 1176-1180.

Wise D.R. (1975). Staphylococcal osteomyelitis of the avian vertebral column. Res. Vet. Sci. 12, 169–171.

Young, D.S., Pestaner, L.C. and Gibberman, V. (1975). Colorimetric estimation of phosphorus. Clin. Chem. 21:432-436.

دراسه عن مدى إنتشار مسببات العرج بمزارع الدواجن بمحافظه الشرقيه

رحاب السيد موافى* ,نجلاء محمود على **, محمد فوزى طه ***

وحده الباثولوجيا*ووحده أمراض الدواجن** ووحده البكتيريولوجى***

معهد بحوث الصحه الحيوانيه (المعمل الفرعى بالزقازيق)* & ***

(المعمل الفرعى بأسيوط) **

الملخص العربى

أجري هذا التقصى على خمسون دجاجه تسمين عمر(2أسبوع-10أشهر) تعانى من العرج وتورمات القدم بعدة مزارع بمحافظه الشرقيه و ذلك لمده عام تبدأ من يونيو عام 2013 وقد صاحب كل دجاجه تاريخ مرضى مفصل للوقوف الاولى على أسباب المرض وتدعيمها بعد ذلك بالاختبارات اللازمه وبناء عليه قام الفريق البحثى بتجميع عينات دم لقياس نسب بعض المعادن والأنسجه لتجهيزها لاجراء الفحص المجهرى ومسحات مباشره من القدم والمفاصل لعزل الميكروب المسبب للمرض وقد خضعت بعض المزارع لتحليل العلائق المستخدمه لاكتشاف مدى إحتوائها على بعض السموم الفطريه وقد تبين وجود حالات تورم قدم بكتيرى والناتجه عن ميكروب العنقودى الذهبى بنسبه 74%من اجمالى الحالات المرضيه كما تم التعرف إلى 4% من الحالات والتى تعانى من نقص فى بعض المعادن والذى يعزى أغلبها إلى اضطرابات بالامعاء كما وجد ان هناك حوالى8%من الحالات تعانى من كدمات وكسور بالأرجل نتيجه الازدحام الشديد داخل العنابر وأخيرا لوحظ وجود بعض الحالات والتى تمثل نسبه 4% والتى تعزى إلى التسمم ببعض السموم الفطريه وبالتحديد سموم الاوكراتوكسين وقد تم حصر وتوضيح لكافه النتائج و مناقشه هذه الحالات وما صاحبه من أعراض مرضيه وصعوبه الحركه وكذلك التغيرات الباثولوجيه ونتائج كلاً من العزل البكتيريولوجى وقياسات الدم وصياغتها مع التوصيه بعمل دراسات أخرى للوقوف على أسباب أخرى لحالات العرض لم تدرج بالبحث.