Clinical and Retrospective Studies of Obstetric Problems of the Domestic Fowl in Zaria, Nigeria

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Abstract: Most female birds produce at least an egg at some point in their life time with or without a mate. A fertile egg is however produced when a bird is mated. The process of egg formation is the result of anatomical, physiological and nutritional factors influenced largely by genetic, disease and extrinsic environmental stimulus. Oviposition involving repeated or larger clutch sizes may functionally exhaust the reproductive tract thereby posing risks of metabolic and physiological drain on the bird. The birds’ reproductive anatomy is such that it does not prevent eggs from undergoing reverse peristalsis. A ten-year study (2001-2010) of obstetric problems of the chicken revealed prevalence of (9.8\%) as reproductive abnormalities, and incidences of oophoritis (39\%), egg-yolk peritonitis (22\%), salpingitis (19\%), egg bound/binding (3\%), atrophied (7\%), and ruptured (2.0\%) oviduct, cystic ovary and oviduct (1.7\%), neoplasms (3\%), various forms of prolapses (2\%), ectopic egg (1\%) and oochitis (0.3\%). Postmortem clinic records showed that most of the obstetrical cases involved ovary and oviduct and were seen in exotic breeds of chickens. This study highlights prevalence, possible causes and remedies to some obstetric problems of the domestic fowl. Required attention must be given to these problems for optimum egg productivity.

Introduction

Most female birds will produce at least an egg with or without a mate during their life cycles. Domestic poultry are primarily maintained to ensure reasonable meat and egg production. The cost of producing an egg in commercial laying birds correlates well with the intensity of egg production per laying bird which must be maintained at optimum to ensure profitability and subsequent sustainability of poultry production as an enterprise. In most developing nations, and especially in Nigeria, one of the major problems affecting commercial layer birds is drop in egg production (Abdu et al., 2002). This has led to the either suspension or total closure of commercial poultry ventures as revenue generation would not maintain their viability. Staff retrenchment, negative impacts on poultry owners’ livelihoods and regional decline in poultry products are some of the immediate and future consequences of commercial poultry farm closure.

Many reproductive abnormalities have earlier been reported to adversely affect economic gain of poultry production due to losses in total egg production and mortality in birds (Peckhan, 1972; Kaikabo, 2007). Abdu et al., 2002 and Kaikabo et al., 2007, have evaluated common reproductive abnormalities of the domestic fowl in Nigeria. They incriminated diseases and husbandry practices as key factors. Recently, Rosen, 2012, showed there were underlying husbandry and/or management issues associated with the reproductive diseases and conditions of laying birds to which clinical signs were almost always vague. Though obstetric problems of birds were believed to be due to multiple causes, emphasis has always been on diseases that cause death of laying birds (Hurgeford, 1969; Peckan, 1972; Abdu et al., 2002; Kaikabo et al., 2007).

It is obvious that genetic, pharmacological, nutritional and environmental influences play major roles in reproductive systems of birds and subsequently on egg production (Roskopt and Woerpel, 2000; Esmail, 2011; Musa et al., 2012; Esmail, 2013). This study evaluated and critically looked at possible remedies to major reproductive abnormalities of the domestic fowl in Zaria, Nigeria.

Materials and Methods

Clinic records of the avian and aquatic clinic of the Veterinary Teaching Hospital, Ahmadu Bello University, Zaria were studied for 10 years (2001-
2010). Gross abnormalities observed at postmortem affecting any part of the reproductive tract or complications resulting from it at different parts of the abdominal cavity were considered for this study. Clinical cases managed were also highlighted. The retrospective obstetric abnormalities the chicken studied were age, type and flock size specific. A case was considered to be a farm that reported clinical case and diagnosed based on history, clinical and laboratory findings. Relevant literatures related to causes and possible remedies of these reproductive conditions were referred to.

Results
About 3, 280 non obstetric avian cases were recorded during the period under study. A total of 320 reproductive abnormalities were recorded in this period out of which 6 cases from different flocks were clinically diagnosed and managed successfully. The obstetric problems recorded represent 9.8% of all avian cases presented to the clinic and 1.9% clinically managed. The major reproductive abnormalities were mainly those involving the ovary and oviduct. Few other problems involving the peritoneal cavity were mainly due to complications from ovary and oviduct and a few others were congenital.

Table 1 is a summary of the major reproductive abnormalities of chicken in Zaria. All abnormalities recorded were in exotic breeds and birds aged 21-30 had the highest (62%) incidence. Table 2 shows relative percentages of all chicken reproductive abnormalities referenced by age, breed, type and flock size.

CLINICAL, POSTMORTEM FINDINGS AND MANAGEMENT OF CHICKEN REPRODUCTIVE DISORDERS.

Oophoritis
Oophoritis is the inflammation of the ovary resulting from neoplastic, mechanical, or infectious causes. The normally turgid yellow ovules become wrinkled, haemorrhagic or discolored accompanied usually by premature rupture and spillage of yolk materials into the abdominal cavity. In this study, oophoritis or follicular regression was seen to be the most commonly observed reproductive disorder characterised by mild to severe congestion and haemorrhages of the ovarian follicles (Fig.1). Signs of oophoritis are non specific and therefore not diagnostic and may include anorexia, weight loss, depression, cessation of egg production, and sudden death. A diagnosis of oophoritis is made through a patient’s history and physical examination. Radiographic imaging reveals an enlarged soft tissue density in the region of the ovary, and there would be coelomic fluid present if concurrent coelomitis existed. Diseases like virulent Newcastle disease (vND), fowl typhoid, fowl cholera, avian influenza and colibacillosis were diagnosed to be responsible for this condition.

Treatment of infectious oophoritis includes broad-spectrum antibiotic therapy based on bacterial isolation and antimicrobial sensitivity results.

Salpingitis
This is an inflammatory condition of the entire oviduct or it may be restricted to only the shell gland (uterus) manifested by haemorrhages and congestion. It is usually a sporadic individual bird problem, though flocks affection in cases of Mycoplasmosis, Salmonellosis and Colibacillosis has been observed. Lesions seen may affect the abdominal air sacs, peritoneal cavity or as descending inflammation into tubular organs or infection may originate from the cloaca orifice and ascend (Rosen, 2012). Early lesions may be presented as irregular reproductive mucosal surface, erosions or small ulcerations and edema that may progress to adherent fibrinopurulent exudates or to yellow cheesy exudates. The oviduct becomes nonfunctional and ovaries may atrophy (Doneley, 2010; Rosen, 2012). In the cases we have seen at postmortem so far, some portions of the uterus or the entire oviduct were inflamed.

Egg yolk peritonitis (coelomitis)
Egg yolk is caustic capable of eliciting mild histolytic to severe inflammatory responses, it also serves as a good medium for bacterial growth (Roskopt and Woerpel, 2000). Furthermore, the condition mainly results from inability of released ova to enter the infundibulum, it may also result from obstruction or rupture of oviduct or reverse peristalsis (Doneley, 2010, Rosen, 2012). Occasionally, such ectopic ova are resorbed without incidence of peritonitis, but many times, it is characterised by abdominal distention, depression, anorexia and lethargy (Rosen, 2012). The diagnosis of this condition is based on history of egg laying and presenting clinical signs. In the laboratory, complete blood count revealing leukocytosis with left shift neutrophilia which is indicative of inflammatory responses is very helpful (Roskopt and Woerpel, 2000, Doneley, 2010). Abdominocentesi or laparatomy may confirm this condition. At postmortem, egg yolk is observed in the abdominal cavity. In especially chronic cases, the yolk formed clumps in the abdominal cavity with adherence of almost all visceral organs, air sacculitis and fibrinous deposits (Fig. 2). Clinical cases are usually managed by corticosteroids and broad spectrum antibiotics. Aspirin in water or juice given 8 hourly was found to be helpful. Surgical intervention with supportive care of vitamins, fluids and warmth has been successful (Roskopt and Woerpel, 2000; Rosen, 2012).

Egg bound/dystocia
It is a condition in which there is difficulty in laying or an egg cannot be laid and is lodged in the cloaca (Riddell, 1997; Foster and Smith, 2009). Egg bound occurs when an egg does not pass at a normal rate through the oviduct and often leads to dystocia, caused mainly by malnutrition, stress, obesity,
myopathies, lack of exercise, systemic disease, injury from a previous dystocia, or malformed eggs (Bowles, 2002; Doneley, 2010; Rosen, 2012). Clinical experience has revealed excessively large normally or abnormally positioned egg to be responsible for this condition in recent times (Fig. 3). Clinical signs associated with such cases were intermittent straining, pigeon-like posture and ruffled feathers. Though signs of egg-binding and dystocia were reported to be vague, they included severe depression, straining, persistent tail wagging, wide stance, failure to perch, coelomic distension, dyspnea, and sudden death in other bird species (Walter, 2000; Bowles, 2002). The bound egg may compress the local blood vessels and surrounding nerves, causing lameness, paresis/paralysis, and decrease organ perfusion as a result pressure necrosis may occur and metabolic disturbances of abnormal defecation and micturition may also occur (Bennett, 1997; Bowles, 2002, Rosen, 2012). Radiography and ultrasonography are required to localize and characterise the egg (Rosen, 2012). Clinical cases presented to us were managed at first by manipulation through intrauterine egg lubrication and manual removal, and secondly by ovarioectomy where bound eggs were carefully crushed within the uterus with sterile thumb forceps when manual traction failed. After crushing, eggs contents were “milked out”, egg shells were carefully removed using sterile thumb forceps and oviducts were lavaged liberally with warm physiological saline. Medically, cases were managed post operatively using oxytocin, antibiotics, corticosteroids and fluids; this treatment schedule has been advocated by Foster and Smith, 2009. Accordingly, some patients may require Stabilization and supportive care of oxygen, fluid therapy, nutritional support, pain management, and, when indicated, parenteral calcium (Bennett, 1997; Roskopt and Woerpel, 2000; Bowles, 2002; DeMatos and Morrissey, 2005). Prostaglandining-2-alpha (PGF2α) can be applied locally to relax the uterovaginal sphincter for ease of egg passage, because administration of PGF2α when the uterovaginal sphincter is closed causes the shell gland rupture if the sphincter fails to dilate (Bowles, 2002; Rosen, 2012).

**Egg binding**

This condition can occur in the cloaca, oviduct or uterus and a times leading to uterine prolapse (Fig. 4). It is a common but emergency condition in pet birds like budgerigars, canaries, cockerels and love birds. Obstruction by fully developed eggs, calculi, low calcium level, infection and uterine damage are some of the major causes of this condition. The obstruction often occurs in the distal part of the oviduct whereby eggs become continuously trapped and such trapped eggs at pelvis may crush the kidneys (Roskopt and Woerpel, 2000; Anne and Girl, 2006). The condition is characterized by sudden death in small species, somnolence, tail wagging, excessive abdominal straining, pain, restlessness and ruffled feathers are some of the accompanying clinical signs. Depending on the cause, treatment is by gentle manipulation and gentle steam heating. Calcium injection at 0.15-0.25mg/kg b.wt, oxytocin at 0.2 I.U per bird, prophylactic antibiotics and steroids has been advocated depending on the cause (Roskopt and Woerpel, 2000). Figures 4 & 5 show cases of egg binding that due to fully developed and shell less eggs detected at postmortem in our clinic.

**Ectopic egg/Internal layer**

This is a condition in which soft shelled or fully formed egg are found in the peritoneal cavity (Riddell, 1997), occasionally seen in small birds like budgerigars and cockatiels (Roskopt and Woerpel, 2000). This condition is also seen in commercial chicken (Abdu, 2002), it is believed to be caused by reverse peristalsis of the oviduct, thereby discharging soft-shelled to fully developed egg within it into the abdominal cavity (Riddell, 1997), or eggs which have developed outside the reproductive tract (Foster and Smith, 2009). All the cases seen so far were at postmortem involving either fully formed or soft-shelled eggs in the abdominal cavity (Fig. 6). From literatures, the condition is often manifested clinically by distended abdomen and a penguin-like posture (Riddell, 1997). The management of this condition is entirely surgical.

**Atrophy of the oviduct**

Atrophy, hypoplasia and atresia of the uterus of laying birds have been documented (Bennett, 1997; Walter, 2000; Abdu, 2002). Atrophy may be associated with severe stress, chronic infections, certain intoxications etc while hypoplasia is a common sequel of early infectious bronchitis infection in especially immature pullets. Affected birds may have developed ovaries but partially developed oviducts given it the name “false layer”. In such birds yolk materials are deposited in the abdominal cavity leading to egg yolk peritonitis. In hereditary atresia of the oviduct, yolks and albumin deposited cannot pass through the oviduct and are refluxed back into the abdominal cavity (Bowles, 2002; DeMatos and Morrissey, 2005, Rosen, 2012). This condition was observed at postmortem in already laying flocks that had frequent history of drop in production. At the time of examination the ovaries were regressed and inactive while the oviducts ranged from gross atrophy to almost complete absence (Fig. 7).

**Oviduct impaction**

This condition sporadically occurs in especially cull hens when the oviduct is occluded by masses of yolk, coagulated albumen, shell membrane or even fully developed eggs (Riddell, 1997). Usually, yolk-like materials in the oviduct appear as concentric rings with the odour of a cooked egg (Abdul et al., 2002), such eggs may be enclosed by shell membranes and may be found in the abdominal cavity. Pullets that were induced to lay early appeared more susceptible. Clinically, birds presented with this condition may be
seen in a penguin-like posture, they lack balance with distended firm abdomen and uterus which may impinge on abdominal organs. Fig. 8 is a postmortem finding that showed oviduct impaction in 1-year bird from a flock of actively laying birds that had stopped laying months ago, presented dead with enlarged abdomen.

**Prolapse**

It is a condition in which part of the reproductive tract is pushed inside out and protrudes from the vent. This can be caused by straining when excessively large sized eggs are laid especially by poorly developed young or obsessed hens (Dunedin, 2000; Rosen, 2012). Other circumstances in which this condition occurs include higher stocking density leading to cannibalism, flocks with poor beak trimming and increased light intensity. When oviposition occurs, there is normally eversion of the vaginal or uterine mucosae which may be slow to retract afterwards; this can lead to pecking by other pen mates causing trauma and edema that may prevent retraction. Straining continues due to irritation of the mucosa resulting into oviduct prolapsed (Klapheke, 2007; Rosen, 2012).

Many forms of prolapses have been identified which include: vent, cloacal, oviduct, oviduct and cloaca (Roskop and Woerpel, 2000). In this report cloacal prolapsed is evident (Fig. 9). The condition can be controlled by avoiding the predisposing factors mentioned above and can be managed surgically using purse-string, cloacopexy, partial hysterectomy or by gentle manipulation. Oxytocin is administered to hasten oviduct shrinkage. Recurrence of this condition often occurs post-management, and therefore, salvage is the best option (Roskop and Woerpel, 2000). Our study revealed indiscriminate use of poultry egg boosters to be responsible for various prolapse conditions in chicken in the study area which in some cases had resulted into severe flock cannibalism. Chickens coming into lay early and obsessed chickens with a lot of fat deposits in their abdominal cavities had commonly been affected by prolapsed conditions. The condition was often managed by isolation of affected chickens, manual return of prolapsed organ and liberal application of gentian violet spray around the vent to prevent further cannibalism. In severe cases surgery was the only option.

**Neoplasm of the ovary and oviduct**

Ovarian neoplasm is frequently seen in avian leucosis especially Marek’s disease and lymphoid leucosis. Adenocarcinomas have also been reported in chickens. Neoplasm of the oviduct occurs rarely. Adenocarcinoma of the oviducts mainly affect the magnum and may be highly invasive and widespread in the peritoneum. This neoplasm may interfere with oviduct function and thus affecting egg production. The incidence of avian leucosis especially Marek’s disease has been reported to be high and on the increase in the study area (Musa et al., 2013), and this correlates well with frequency of ovarian and oviduct neoplasm (Fig. 10 a & b).

**Cystic oviducts and ovary**

Cystic right oviducts are naturally prevalent in the chicken because only the left components of a paired embryonal reproductive system develop post hatching. If the right oviduct wall contains significant glandular tissues, fluid secretions will accumulate resulting into cysts (Fig. 11) with affected hens appearing to have abdominal ascites on palpation.

Ovarian cysts (Fig. 12) have been diagnosed in many avian species, including cockatiels, canaries, budgerigars, pheasants, and domestic waterfowl. Endocrine disorders, ovarian anatomic abnormalities, and neoplastic conditions have been associated with the development of ovarian cysts in birds (Bowles, 2002, Rosen, 2012). The patient’s history may reveal cessation of current or previous egg production. Owners may report chronic reproductive behavior that does not result in an egg, or decreased performance in breeding hens. Advanced cystic ovarian disease may cause depression, hyporexia, and weight loss. Abdominal distension, often the result of a secondary coelomitis, and related clinical signs such as ascites and dyspnea may be observed in the affected bird (Bowles, 2002; Rosen, 2012).

Radiographic and ultrasonography will often reveal the fluid-filled cyst, or multiple cysts, in the area of the ovary. Ovarian cysts can be large and may overlap onto themselves as they grow. Fluid aspirated from ovarian cysts is usually clear to straw-colored and often of low cellular content (Bennett, 1997; Bowles, 2002; Rosen, 2012). Ovarian cysts can be secondary to neoplasia or oophoritis, therefore exploratory laparoscopy or surgery may be indicated to determine if there is underlying disease (Rosen, 2012). At postmortem we observed ovarian cysts to be of different sizes, shapes and contained clear fluids (Fig. 12).

Treatment goals for the avian patient diagnosed with ovarian cysts include deflation of the cyst and treatment of associated diseases, such as coelomitis, ovarian granuloma, and/or neoplasia (Rosen, 2012). Drug usage, behavioral changes, environmental, and dietary intervention to decrease stimulation of ovarian activity and production of hormones that may perpetuate ovarian cysts can be reliable remedies. Aspiration of cysts and salpingohysterectomy with partial ovarioectomy may be beneficial to completely resolve ovarian cysts (Rosen, 2012).

**Orchitis**

Ochitis could be infectious or noninfectious in nature. Infectious orchitis can occur due to ascending infections, hematogenous spread of pathogens, or bacterial dissemination from nearby organs. Noninfectious causes of orchitis are rare in
occurrence. The major cause of ochitis in this report was *Salmonella gallinarum*. Grossly, the testicles were misshapen and haemorrhagic (Fig. 13). Diagnostic test results may reveal a leukocytosis with a relative heterophilia. The tests may be visually enlarged with diagnostic imaging modalities such as radiographs, ultrasound, and endoscopy. A definitive diagnosis of orchitis is made through cytology, bacterial culture and antimicrobial sensitivity testing, and histopathologic examination of samples from affected testes. The recommended treatment protocol for orchitis includes broad-spectrum antibiotics, pending sensitivity results, and pain management (Bennett, 1997; Doneley, 2010; Rosen, 2012). This case from a breeder flock was successfully managed using ciprofloxacin in drinking water.

**Vent pecking/cannibalism**

Cannibalism occurs when birds peck at the feathers, toes, heads and vents of other birds. This is usually associated with bleeding which induces further pecking. We have observed the condition to often occur in laying birds and could be so serious resulting into evisceration and eating of the intestine resulting into death of affected chicken. The predisposing factors were frequent prolapse in especially young or over weight hens. Stressful factors like overcrowding, overheating and bright light or could be dietary in nature (salt, vitamins, protein or insufficient feed) have been reported to cause the condition. This condition is not common in well managed free-ranged flock (Duredin, 2006; Doneley, 2010). Management is by isolation and wound treatment. Prevention is better achieved by adequate spacing and ventilation, not too bright light, offering several feeds and grains during the day to keep birds busy, introduce rooster, use of red light may help in some cases (Duredin, 2006).

**Pharmacological influence on egg production**

Several different hormones and prostaglandins are believed to play significant roles in the reproductive activity of birds. PGF2 as an example causes shell gland contractions and prostaglandin E2 relaxes the uterovaginal sphincter and vagina and also cause shell gland contractions. There are no anatomical structures to prevent the egg from undergoing reverse peristalsis, physical disturbances of laying hens and certain drugs can cause egg peritonitis, soft- shelled egg, shelled or mishapened eggs, adrenergic drugs cause relaxation of the oviduct while cholinergic drugs cause oviduct constriction (Roskopt and Woerpel, 2000).

Acetylcholine, oxytocin and vasotocin produce expulsion of uterine egg. For a shell to be fully developed, metabolic carbon dioxide must be converted to hydrogen carbonate within the shell gland catalysed by carbonic anhydrase. Drugs such as sulphonamides and acetazolamide inhibit carbonic anhydrase hence leading to thinning of egg shell (Roskopt and Woerpel, 2000).

**Nutritional influences on egg production**

Protein deficiency leads to decreased number of eggs and high chick mortality, oestogens and androgens in females stimulate bones to make calcium available for shell formation. Therefore, deficiency of this will lead to increase in thin shelled eggs or egg bound incidence. Calcium also affects uterine contraction, deficiency will lead to decrease in uterine contraction, vitamin D₃ is associated with poor bone development (Roskopt and Woerpel, 2000).

**Discussion**

There are few reports of reproductive abnormalities of birds in Nigeria, and the incidences of such problems in chickens have earlier been reported to be low (Peckham, 1972, Abdu et al., 2002). Hutt et al., 1956 observed low incidence of oviduct dysfunction, ovarian dysfunction and presence of soft-shelled egg in the peritoneal cavity. Accordingly, Abdu et al, 2002 observed most obstetric problems to have occurred in improved commercial laying birds where owners of such birds had put in huge financial investment thus made such owners promptly submitted dead and diseased birds to veterinary clinics for diagnosis and treatment. It is also the views of Abdu et al, 2002 that local chickens in comparism are economically viable, had less active reprodutive organs producing fewer eggs per year, not stressed under the intensive management system, hence were less prone to reproductive disorders and had less chances of presentation for clinical attention.

The most common reproductive abnormality observed in this study was oopheritis which was similarly documented by Abdu et al, 2002. Egg yolk was reported to be a very good medium for bacterial growth and other microorganisms, there is therefore a high possibility of bacterial infection associated with egg yolk found in the peritoneal cavity, also bacterial and viral diseases that have affinity for the reproductive tract of birds could lead to frequent oopheritis and egg yolk peritonitis. The close proximity of ovary to many organs like the lungs, liver, kidney, spleen, intestine and air sacs could make it highly likely to ascending infections from these organs.

Indiscriminate use of poultry drugs like egg formulas and sulphur containing drugs are common in the study area. These drugs are of frequent use to boost egg production and treat certain poultry diseases in commercial farms which have lead to oversized eggs and consequent higher incidences of egg bound and egg binding conditions. Crisis of decline in egg production have been reported by farmers following use of sulphur or sulphur containing drugs.

Ochitis due to infectious agents like bacteria especially salmonella in cocks is of serious significance not only because of infertility that leads to low number of hatchable eggs, affected birds are...
also at high risk of vertical transmission of the organism to their upspring.

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Table 1. Yearly distribution of major reproductive disorders of chicken diagnosed at ABUVTH, Zaria, Nigeria (2001-2010).

| Year | Non reproductive disorders | Reproductive disorders | Year specific rate [%] |
|------|-----------------------------|------------------------|-----------------------|
| 2001 | 390                         | 33                     | 8.5                   |
| 2002 | 348                         | 30                     | 8.6                   |
| 2003 | 298                         | 29                     | 9.7                   |
| 2004 | 300                         | 36                     | 12.0                  |
| 2005 | 349                         | 37                     | 10.6                  |
| 2006 | 293                         | 46                     | 15.7                  |
| 2007 | 353                         | 48                     | 13.6                  |
| 2008 | 328                         | 28                     | 8.5                   |
| 2009 | 295                         | 20                     | 6.8                   |
| 2010 | 326                         | 13                     | 6.0                   |
| Total| 3,280                       | 320                    | 100                   |

Table 2. Incidence of reproductive disorders of chicken diagnosed at ABUVTH, Zaria, Nigeria (2001-2010)

| Reproductive disorder | Number of cases | Incidence [%] |
|-----------------------|-----------------|---------------|
| Oophoritis            | 125             | 39.0          |
| Egg yolk peritonitis  | 70              | 22.0          |
| Salpingitis           | 61              | 19.0          |
| Egg bound/binding     | 10              | 3.0           |
| Atrophic oviduct/ovary| 22              | 7.0           |
| Ruptured oviduct      | 6               | 2.0           |
| Cystic oviduct/ovary  | 5               | 1.7           |
| Neoplasms             | 10              | 3.0           |
| Prolapse              | 7               | 2.0           |
| Ectopic egg           | 3               | 1.0           |
| Ochitis               | 1               | 0.3           |
| Total                 |                 | 100           |
Table 3. Age, breed and flock size distribution of reproductive disorders of chickens diagnosed at ABUVTH, Zaria, Nigeria (2001-2010).

| Item       | Number affected | Incidence [%] |
|------------|-----------------|---------------|
| Age        |                 |               |
| 16-20 weeks| 25              | 7.8           |
| 21-25 weeks| 10              | 3.1           |
| 26-30 weeks| 15              | 4.7           |
| 31-35 weeks| 10              | 3.1           |
| >35 weeks  | 260             | 81.3          |
| Breed      |                 |               |
| Local      | 3               | 0.9           |
| Exotic     | 317             | 99.1          |
| Flock size |                 |               |
| <100       | 10              | 3.1           |
| 100-500    | 70              | 21.9          |
| 500-1000   | 210             | 65.6          |
| >1000      | 30              | 9.4           |

Fig. 1: Oophoritis showing severe congestion and haemorrhages of the ovarian follicles

Fig. 2: Egg yolk peritonitis with adhesions of and fibrin deposits on visceral organs of a layer

Fig. 3: Egg bound (large egg just proximal to cloaca) caused by non patent uterus in a chicken

Fig. 4: Egg binding that resulted into ectopic egg in a commercial chicken

Fig. 5: Egg binding (developed egg in the uterus) and shell-less egg in the magnum
Fig. 6: Ectopic egg (soft-shelled) in the abdominal cavity of a chicken

Fig. 7: Atrophy of oviduct with regressed ovary from actively laying flock of commercial chicken with history of decrease egg production.

Fig. 8: Oviduct impaction in a commercial chicken

Fig. 9: Cloacal and vent prolapse in a laying commercial bird

Fig. 10 (a): Ovarian and oviduct neoplasms in Marek’s diseased chickens.

Fig. 10 (b): Ovarian and oviduct neoplasms in Marek’s diseased chickens.
Fig. 11: Cystic right oviduct (arrow), and developed egg in left oviduct of a commercial chicken

Fig. 12: Cystic ovaries and atrophied oviduct in a commercial chicken

Fig. 13(a): Ochitis in layer breeder (misshapened and haemorrhagic testicles)

Fig. 13(b): Ochitis in layer breeder (left and right testicles)