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Ulcerative enteritis In quail, pheasant, grouse, pigeon & partridge an histophysiological - pathological study at the Kurdish triangle section

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Abstract:

Our study shows that ulcerative enteritis in some birds are an lesions in the intestinal tract, liver, and spleen. It was occurred mostly at <3 months birds with some exceptions. *Clostridium colinum* investigated by some microbiological test and by using new electrophoretic study. Histopathological observations showing ulcers, peritonitis and foci of necrosis along of the injured organ. Necrosis extends deep into tunica muscularis or serosa. Colonies of large gram positive bacterial rods are normal throughout the lesion. Here we confirm the most important changes occurred at the intestinal layers due to invasion by *C. colinum*.

Entérite ulcerative en cailles, faisan, grouse, pigeon&partridge an histophysiological - étude pathologique à l'abrégé sur Kurde section de triangle

Résumé:

notre étude prouve que l'entérite ulcerative les oiseaux insome sont des lésions dans la région intestinale, foie et spleen.It a été produit la plupart du temps à < 3 mois d'oiseaux avec le colinum d'un certain exceptions. *Clostridium colinum* étudié par un certain essai microbien et en employant de nouvelles observations électrophorétiques de study. Histopathological montrant des ulcères, la péritonite, centres de la nécrose le long de l'organ. Necrosis blessé se prolonge profondément dans des muscularis de tunica ou serosa.Colonies de grandes tiges bactériennes grampositives sont normaux dans tout le lesion.Here que nous confirmons les changements les plus importants nous sommes produits aux couches intestinales dues à l'invasion par *C.colinum*.

Introduction:

Ulcerative enteritis caused by *Clostridium colinum* which is a highly fatal enteric disease, has also been reported in other birds incl-uding chickens, turkeys, grouse, and partridge (3,27). The condition is characterized by sudden onset and rapidly - increasing mortality in a flock. It was originally known as quail disease, due to its frequent enzootic occurrence in bobwhite quail (*Colinus virginianus*), from which the causative organism derives its name (5). The mol % G + C of the DNA of *C.colinum* (43 %) is significantly higher than that of many Pathogenic clostridia (31).

Certain aspects of the natural history of *C.colinum* and ulcerative enteritis are relatively clear. Natural infection probably occurs orally and the annual recurrence of infections on some premises suggests maint-enance of *C.colinum* in soil. Quail with chronic disease can be carriers (4).

Birds less than three months of age are most commonly infected with *C. colinum*, and the disease follows a rapid and fatal clinical course. The average mortality in chickens is 10–30%, but may reach 100% in young quail. The course of the disease in a flock is usually 3 weeks with peak mortality during the second week (36).

Clinical pathology used a differrent method for inspection about the causative agents of many lesions. One of this techniques was electrophoresis that is explain the minute differ between adjucent species, which analyses of soluble cellulose proteins.

Histotechniques will be another manner for the evaluation range of causative agents at their host body (38).

The main aim of this study is to follow the major changes at the histophysiological texture of the injured sites of the quail, pheasant, grouse, pigeons, and partridge fowls bodies to determine how to treat like cases.

MATERIALS AND METHODS:

Blood sample :- fresh blood put in EDTA tubes;1gm/ml; which are sent to the lab. were analyzed dir- ectly by using autoanalyser (Ms-9).

Bacteriological test:- electrophor-esis method was used to identify of clostridial *sp*. that responsible for this case.

We are applied method of Cato, etal. (8). Blood agar and sugar fermentation was applied.

Tissue sample:- piece of intestine taken up from injured region, then the routine lab. practions which include fixation, dehydration, clea-ring, infiltration, embedding and cutting had been applied before staining the section with hemat-oxylin – eosin stain.

About 121 samples gained have a relation with this case at Kurdish triangle of Iraq, Iran, and Turkey. Work at this project continue from September,2004 and complete at March , 2005.

RESULTS:

Microscopic examinations shows that the main causative agent for the UE were *Clostridium colinum*. Other types of organisms will be a side infection to this injury. The main characters of this bacteria are include; bacilli with subterminal spores may be seen in tissue. Colonies are white, circular, convex, semitr-anslucent non–hemolytic and cell are individual. Gram – positive rods that are straight or slightly curved. Biochemically *C.colinum* ferments raffinose and does not hydrolyze gelatin.

Electrophoresis method showed that *C.colinum* had the char-acteristics originally described and produced a distinctive protein pattern (Fig.1, Lane 12).

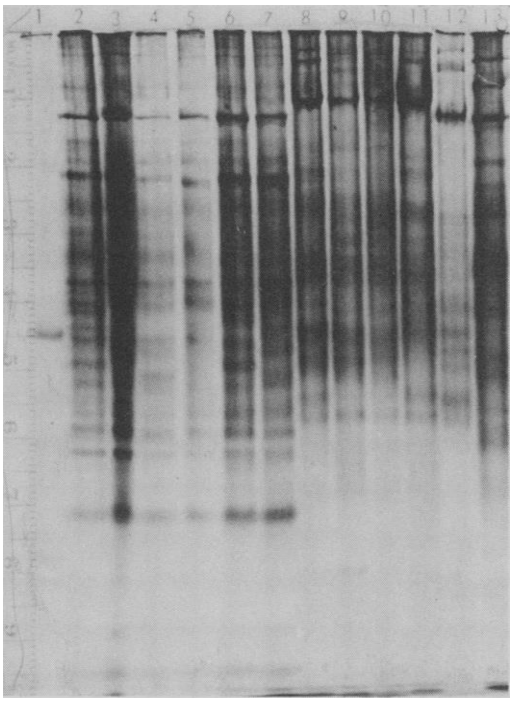

Fig.1.C.colinum by electrophoretic method line 12.

Clinical finding of the lesion explain discharging characteristic dropping streaked with urates and with a watery ring. Chronic affected birds are listless, anorectic and humped up with the neck retracted and eyes partially closed. The primary lesion are in the ceca and intestine. Hemorrhages in the wall of the acute cases. Chronic cases appear as necrotic ulcers surro-

unded by zones with yellow diph-theritic membranes having depr-essed centers. Yellow liver and hemorrhaged spleen will be seen (Fig. 2).

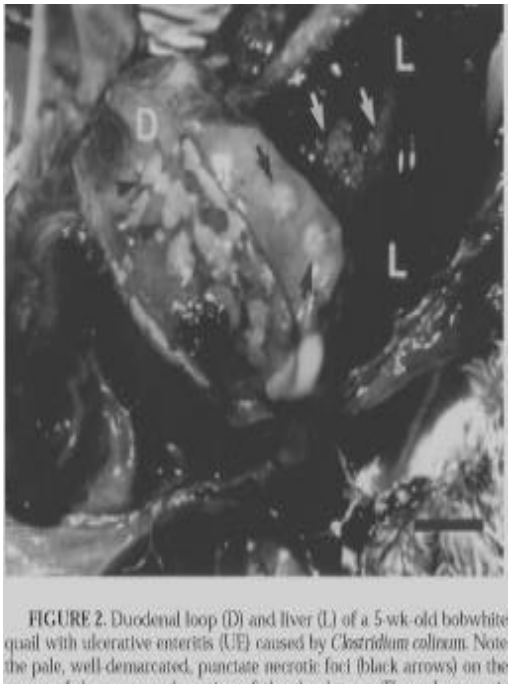

FIGURE 2. Duodenal loop (D) and liver (L) of a 5-wk-old bobwhite quail with ulcerative enteritis (UE) caused by *Clostridium colinum*. Note the pale, well-demarcated, punctate necrotic foci (black arrows) on the

Fig.2. Duodenal loop (D) and liver (L) of a 5 – wk – old bobwhite quail with ulcerative enteritis caused by C. colinum. From Porter (1998).

Blood examination tests showed an elevation of white blood cell counts to being more than 50×10^3 /mm^3, while the PCV counts declines to exceeding 8 – 10% at all the birds collected for study as showes at table – 1.

Table-1: explain the values of WBCs & PCV at each of the birds injured with EU.

Bird	WBCs $\times10^3$ /mm^3	PCV %
quail	52.5	8.5
pheasant	60	8
grouse	60.02	8.11
pigeon	51.7	10
partridge	53	9.15

The histopathologic exam revealed characteristic ulcerative enteritis involving serosa. The ulcers perforate the intestinal wall to produce peritonitis and intestinal adhesion. Well -demarcated areas of coagulative necrosis of villi with infiltrates of heterophils and mon-onuclear cells. Colonies of large Gram–positive bacterial rods are numerous throughout the lesion (Fig. 3).

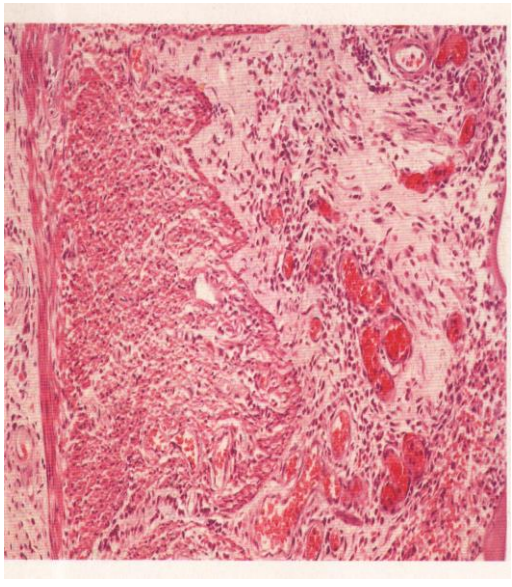

Fig. 3. Ulcerative enteritis. The mucosa and submucosa have been replaced by vascular granulation tissue. Some Gram–positive bacilli centred at the ulcers crypts. HEX 60.

DISCUSSION :

Adverse circumstances allow the bacteria to colonize the intestine, producing exotoxines that to clinical signs, leads to lesions or death (11,15). Gupte, S. (17) remember that this products may be enzymes or low effective toxins like fibr-inolysin which promotes the spread of infection by breaking down the fibrin barrier in tissues, also hyal-uronidase which split hyaluronic acid (component of connective tissue) thus facilitating spread of infection along tissue spaces. The hyperplasia of digestive mucosa in vivo have been induced by butyrate which is considerd a cytotoxic agent in several cell lines (6,9,12). This is also observed in ulcerative colitis (1,14). It is most likely that the increased cecal wall thickening observed in quails with lesions is due to the accumulation of butyrate produced from undigested lactose. The absence of any cecal thickening in quails associated with the lactose–negative strain reinforced this hypothesis (25). Underwood, J. C. E. (39), Riddell, C. (33), Jordan, F. T. W. (21), Kolmstettar, etal (23) observed that ulceration is loss of full thickness of the mucosa, and defect goes much deeper to penetrate the muscularis propria. Damaged blood vessels will have bled and the surface covered by a layer of fibrin. If the cause persists, the ulcer becomes chronic and enteric wall may result in considerable destruct-tion. The ulcers often perforates the intestinal wall to produce peritonitis and intestinal adhesion (24). Inflammation occurs by acute and chronic states will be produce systemic effects like splenomegaly which is lead to a type of fibrosis (10,18,30). Necrosis at UE in all five birds studied here will be arised due to the death of tissues from lack of oxygen or nutrients resulting from inadequate blood flow (16,34).

Avariety of enteric bacterial diseases are recognized in poultry, but the diagnosis of bacterial ent-eritis requires monitoring of clinical signs in the flock and proper use of diagnostic method such as necropsy, histopathology, bacterio-logy, and serology (37). So, clostridial species are the most obvious to injury of the poultry intestine especially *C.perfringens,C.butyricum, C.tertium, C.colinum,…* etc anaerobe clostridial types (26,29). Electrophoresis was the most procedure used lastly to differentiate the main causative

agent for the ulcerative colitis according to the species (8,13).

The animal model, quails – phea-sants – grouse – pigeons – partr-idges, presents obvious experimental advantages over the chicken model. This may be related to the fact that those birds reach maturity more rapidly. Those phenomenas has been noticed in cancer research (32) and severe lesions (2,22). Other types of birds used in this side was including lorikeets, red lories, Moluccan Cockatoo and others (19,28).

Physiology side showing an elevation at WBC_5 counts especially at severe cases, and this is correlated with immunological status of the body that will exampilified with a mechanism to countering the *C. colinum* (20). PCV parameter dec-lines may be an obvious indicator to the state of anemia that was coming from ulceration and hemorrhage state surrounding the most body organs (7,35).

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الالتهاب المعوي التقرحي في السمان والتدرج والطيهوج والحمام والحجل
دراسة فسيولوجية نسيجية – إمراضية في المثلث الكردي

الخلاصة:
نصت دراستنا على أن مرض إلتهاب الأمعاء التقرحي في بعض الطيور هو عبارة عن إلتهاب يحصل في القناة المعوية والكبد والطحال. وهو يقع غالباً في الطيور الأصغر من ثلاثة أشهر مع بعض الاستثناءات. تم الكشف عن البكتريا المسببة لهذا المرض بعدة الطرق بكتريولوجية مع استخدام طريقة جديدة للتأكيد باستخدام جهاز الترحيل الكهربائي. كما أظهرت التشخيصات المرضية النسيجية وجود تقرح مع التهاب في الغشاء البريتوني وبقع من التنخرات على طول العضو المصاب. كان وجود مستعمرات قضبانية من الجراثيم الموجبة لصبغة جرام أمراً واضحاً كذلك. لقد تم التأكيد في هذه الدراسة على أهم التغيرات الحاصلة في طبقات الأمعاء نتيجة لإصابتها ببكتريا الإلتهاب المعوي المتقرح (كلوستريديا – قولينوم).