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Bacteriological and Pathological Studies on Some Aerobic and Anaerobic Bacteria Causing Diarrhoea in Camel Calves.

Hamouda, R. H., Thannaa, K. H. and Nabih, A. M.

Animal Reproduction Research Institute (ARRI) El-Haram Giza.

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SUMMARY

The present study was carried out to investigate the problem of diarrhoea among camel calves, by isolation, identification, histopathological findings and controlling of the associated infective agents (*E. coli*, *Salmonella* and *C. perfringens*). Faecal, internal organs (spleen, kidneys, liver and part of small intestine) and blood samples were obtained from 120 camel calves (aged 10 – 18 month) for microbiological examination. The isolated rate of *E. coli*, *Salmonella* and *C. perfringens* from diarrhoeic animals were (41.1%), (7.8%) and (65.6%), respectively. The isolated rate from the apparently healthy animals were (13.3 %), (0%) and (33.3%), respectively. The serological identification of the isolated *E. coli* and salmonella strains detailed that (O111/K58 and O55/K59) serovars of *E. coli* and *(S. Entertidis and S. Typhimurium)* were the most common causes of diarrhoea in camel calves. The rate of both Beta and Epsilon toxins of *C. perfringens* was (53.3%) and (23.3%), while individually was (14.4% and 3.3%) and (27.8% and 10%) in diarrhoeic and apparently healthy camel calves, respectively. The microscopical examination revealed degenerative changes with marked necrosis in the hepatic and renal tissues, spleenic depletion and desquamation of the intestinal lining epithelium indicated the toxic effect of *C. perfringens* type (C and D). In *Salmonella Typhi*miurium infection vasculitis and thrombi in blood vessels of the lamina propria and submucosa resulting in focal intestinal infarctions and ulceration, were the most important findings. Caecal glands were dilated and filled with gases and aggregation of inflammatory cells were observed in *E. coli* infection. The antibiotics used in the treatment of diarrhoea were Augmentin, Chloramphenicol, Gentamycine, Ciprofloxacin, Florofenicol, Rifampicin and Metronidazol, according to sensitivity test.

INTRODUCTION

Diarrhoea is a complex syndrome that can be caused by various infective agents proliferating in the intestinal tract. The infectious diarrhoea is an important cause of morbidity and mortality in neonatal farm animal's that results in significant economic losses especially in the beef and dairy industries (Barrington *et al,* 2002). The most commonly incriminated bacterial causes are; *E. coli* (Munos *et al.*, 1996) *Salmonella spp.* (Neser, 1994) and *C. Perfringens*. The family Enterobacteriacae that includes Gram-negative microorganisms (*E. coli* and *Salmonella spp.)* may constitute a primary cause of enteritis among newly born animals and human being (Singh *et al.*, 1979) and (Abdel Ghani *et al.*, 1987).

C. perfringens is a Gram-positive anaerobic enterotoxigenic spore-forming bacterium that causes disease in many species and harmful to neonates, also causes food poisoning to human (Miki *et al.*, 2008). Large number of toxins are produced by *C. perfringens*, some of them have a specifying virulence to the type of disease and the host range (Hatheway, 1990 and Songer, 1996). The newly born calves suffered from severe enterotoxaemia, the causative bacteria was *C. perfringens types C* and *D* which were isolated from the intestinal contents, liver, spleen and kidneys showed catarrhal enteritis and nephritis by histopathological examination (Nilo, 1980; Donald and James, 2001). *E. coli* and *Salmonella typhi*murium were the most significant bacteria responsible for heamorrhagic enteritis in one year old camel calves suffered from diarrhoea (Argenzio, 1985 and Dia *et al.,* 2000).

The present study was planned to determined the incidence, serotyping and studying the most histopathological changes in the tissues due to *E. coli*, *Salmonella Typhimurium* and *C. perfrnigens* type (C and D) which cause diarrhoea in camel calves in order to confirm the diagnosis of this problem and subsequent correct course of treatment and control.

MATERIAL AND METHODS

Samples collection:

Faecal samples, affected parts of internal organs (spleen, kidneys, liver and part of small intestine, directly after death or slaughtering) and blood samples were collected from 120 camel calves aged between 10-18 month old (30 apparently healthy and 90 diarrhoeic camel calves) from several private farms and slaughter houses with different hygienic measures. Faecal samples, affected parts of (spleen, kidneys, liver and part of small intestine) were collected in sterile plastic bags for aerobic and anaerobic examination, while the blood samples were collected in sterile vaccotainers.

The collected samples were transferred to the laboratory in ice box as soon as possible. The blood samples were centrifuged at 3000 rpm for 15m. for separation of sera and stored at -20°C in clean vials until used.

1- Bacteriological examination:-

Isolation and identification of *E. coli* and *Salmonella* according to (Konemann *et al.*, 1983 and Cruickshank et al., 1996). Morphological examination according to (Krieg and Holt, 1984). Biochemical identification: according to (Quinn et al., 2000).API 20 E (Apari Procedeir de Identificausion 20 E-BioMerieux- France): it was used as standardized identification system for Enterobacteriaceae and other non-fastidious Gram-negative rods which use 23 miniaturized biochemical tests and a data base, to confirm the identification of some isolates of *E. coli* and *Salmonella* strains.

Serotyping of *E. coli* isolates according to (Edwards and Ewing, 1972): all suspected *E. coli* isolates were subjected to serological typing by slide agglutination test using standard polyvalent and monovalent *E. coli* antisera.

Serotyping of *Salmonella* isolates according to (Edwards and Ewing, 1972): Isolates that identified by biochemical tests as *salmonella* were subjected to serological identification.

Isolation and identification of *C. Perfringens* according to (Konemann *et al.*, 1983): The growing colonies were kept in cooked meat broth tubes for identification by morphological and biochemical tests (Chai *et al.*, 2007).

Determination of the toxigenic activity and typing of *C. perfringens*: by pathogenicity in Albino Guinea pigs: weighting 350-450 g and mice neutralization test in Albino Swiss mice: weighting 20-40 g (Itodo, 1991).

Indirect solid phase Enzyme Linked Immuno-Sorbent Assay (ELISA): for determination of antitoxin titers according to (El-Idrissi and Ward, 1992b) with some laboratory modifications to facilitate the detection and estimation of the antitoxin titers against *C. perfringens* types "B" and "D" toxins in the separated sera collected from the blood samples of the newborn lambs according to (Krt, 1999) and (Uzal, *et al.*, 2003). Flat bottomed microtitre ELISA plates were coated (100 µl/well) each with washed whole cell antigen of Beta and Epsilon toxins diluted in carbonate bicarbonate buffer (PH6.9) as coating buffer 1:50. The coated plates were incubated over night at 4°C in humidity chamber, then the wells were washed 3 times with washing buffer P.B.S.T (Phosphate Buffered Saline containing 0.5% Tween 20) 200 µl/well. Blocking buffer (P.B.S. containing 2% bovine serum albumin (B.S.A.) containing 0.5% Tween 20) PH 7.4. 200µl/well was added. The plates were incubated 37°C for 24 hours in humidity chamber. After three washes with P.B.S.T., each well was inoculated with 100 µl /well of serum sample, diluted 1:100 with P.B.S, then

incubated at 37°C for 30 min. in humidity chamber. The wells were washed 3 times with washing buffer P.B.S.T, and then adding anti-ovine IgG with a horse radish peroxidase as conjugate diluted as recommended was added 100µl/well. The plates were incubated 37°C for 30 minutes in humidity chamber. The wells were washed 3 times with washing buffer P.B.S.T, and then the freshly prepared 100µl/well of substrate solution (ABTS in citric phosphate buffer with H_2O_2) was added. The plates were incubated 37°C for 10 minutes at dark place. The reaction was stopped by addition of 50µl/well of $0.2H_2SO4$. The plates were read by ELISA reader after 10 min. at 405 nm wave length according to (Snedecor, 1967). The interpretation of the results was taken in comparison with positive controls contain antitoxin only while negative controls contain diluted toxin).

Sensitivity test: was carried out using Mueller-Hinton agar (Garctikyaa-Rodriguez *et al.,* 2005). Amoxicillin, Penicillin G, Ampicilline, Chlorophenicol, Enrofloxacine, Rifampcine, Metrendazol, Erythromycin, neomycin, Doxycyclin, Florophenicol, Augmentin (Amoxicillin - clavulanate), Tetradelta, Gentamycine, Ciprofloxacin, Cephradine, rimethoprim/sulphamethexazole, Nalidxic acid, Oxytetracyclin, Norofloxacin, Streptomycin. And Tetracycline disks were used. The isolates categorized are susceptible (sensitive), intermediate or resistant according to the methods and criteria described by National Committee for Clinical Laboratory Standards.

2-Histopathological examination for the internal organs of the infected camel calves:-

Specimens from the liver, spleen, kidneys and small intestine were fixed in 10% neutral buffered formalin. The fixed samples were dehydrated in alcohol, cleared in pure xylene and embedded in paraffin blocks. Sections of 4 – 6 microns were prepared and stained with Haematoxylin and Eosin (Bancroft *et al.*, 1996).

RESULTS

The Bacteriological examination of samples revealed the recovery *E. coli*, *Salmonella* *C. perfringens*:-

As shown in **(Table 1)**, *E. coli* was isolated from diarrhoeic camel calves was (41.1%) in the higher prevalence than apparently healthy ones (13.3 %). The incidence of *Salmonella spp.* revealed that in diarrhoeic camel calves it was (7. 8%) while it was not isolated from apparently healthy one. The results of *C. perfringens* indicated that the percentage of isolation of *C. perfringens* was higher in diarrheic camel calves (65.6%) than that in apparently healthy ones (33.3%).

Serological identification of *E. coli* strains isolated from diarrhoeic camel calves:-

The results were detailed in **(Table 2)** for identification of O serogroup. O111/K58 and O55/K59 were the most common O group of *E. coli* isolated from diarrhoea in camel calves.

Serological identification of *Salmonella* strains isolated from diarrhoeic camel calves:-

The recorded results in **(Table 3)**, revealed that *S. Entertidis and S. Typhimurium* were play nearly the same role as a cause of diarrhoeia in camel calves 3/7 and 4/7 respectively.

The results of toxigenic activity and typing determination of isolated *C. perfringens*:-

as tabulated in **(Table 4)**, the higher rate of both Beta and Epsilon toxins (53.3%) was detected in diarrhoeic camel calves than that of the apparently healthy ones (23.3%).

The Beta toxin was (14.4%) and (3.3%), while the Epsilon toxin was (27.8%) and (10%) in the fecal samples of diarrhoeic and apparently healthy camel calves, respectively.

Titration of the enterotoxins of *C. perfringens* using ELISA:-

a- The titration of Beta antitoxin in the sera of camel calves: **Table5** shows that the titer of Beta antitoxin of *C. perfringens* in the apparently healthy camel calves sera varied from 40-320 which was higher than that of diarrhoeic camel calves (40-80).

b-The titration of Epsilon antitoxin in the sera of camel calves: as shown in **(Table 6)**, the range of Epsilon antitoxins titer in sera of apparently healthy camel calves was (40-640), which was higher than that of diarrhoeic camel calves (40-160).

The Drugs of choice for treatment of E. coli, Salmonella and C. perfringens Isolated from camel calves according to sensitivity test:

***E. coli*:** 1- Augmentin (Amoxycillin + Clavulanic acid) 2- Chloramphenicol 3-Cephradine 4- Neomycin 5-Gentamycine 6-Oxytetracycline

***Salmonella*:**1-Augmentin 2-Chloramphenicol 3-Gentamycine 4-Ciprofloxacin 5-Cephradine

***C. perfringens*:** The highly effective antibiotics on the isolated bacteria in the laboratory are 1-Florofenicol 2-Tetradelta 3-Gentamycin 4- Augmentin 5- Refampicin 6-Metronidazol.

A-Histopathological findings due to the infection with *E. coli*:-

1-The liver and spleen were enlarged and edematous.

Microscopically: there were degenerative changes and necrosis with heamolysis of erythrocytes in the hepatic and spleenic tissues (Fig. 1 and 2)

2- The kidneys were enlarged with some focci of cortical necrosis and heamorrhages.

Microscopically: there were necrobiotic changes in the tubular epithelium and infiltration of mononuclear cells in the interstitial tissues (Fig. 3).

3-The intestine was dilated, flaccid and filled with translucent, yellow fluid and gases. The

caecum was impacted and contained focci of hemorrhages.

Microscopically: the lesions showed heavy colonization of the mucosa by small bacilli (*E. coli*), which aggregated on the surface of the atrophic villi in the middle and lower parts of the small intestine. There was hyperplasia of the lymphoid follicles. The epithelial cells covering the tips of villi were degenerated and desquamated. Cystic dilatation of caecal glands with inflammatory cell aggregations were evident (Fig. 4).

B-Histopathological findings due to the infection with *Salmonellas*:-

1-The liver and spleen, the liver was enlarged, congested and contained multiple focci of hepatic necrosis. Spleenomagally was also seen.

Microscopically: the liver revealed moderate edema of portal area, focal area of inflammatory cell aggregations (lymphocytes and neutrophiles) and some areas of fatty changes and necrosis of most hepatocytes (Fg. 5). The spleen showed follicular spleenic depletion, hyperplasia of lymphoid follicles, marked necrosis and heamorrhages (Fg. 6).

2-The kidneys: were congested, swollen and showed necrotic focci.

Microscopically: there were degenerative changes in the renal tubules and glomeruli with marked necrosis of the renal tissues and focal areas of hemorrhages (Fig. 7).

3-The intestine: The intestinal contents were malodorous and contained mucous, fibrin and occasionally blood of aseptic tank odour. Discrete multiple focci of necrosis and ulceration were seen in the caecum and colon were also present.

Microscopically: The superficial surfaces of the intestinal villi revealed degeneration and desquamation of epithelial cells. Lamina propria was infiltrated with mononuclear inflammatory cells and moderate neutrophilic infilterations, congested blood capillaries and edema was noticed in the mucosa and submucosa. Some blood vessels were occluded by thrombi. Some of the intestinal glands appeared degenerated, necrosed and the glandular epithelium appeared hyperplastic (Fig. 8).

c- Histopathological findings due to the infection with C. *perfringens* types (C and D):-

1- The liver: Moderate congestion and the surface oozed with blood in cut section were prominent.

Microscopically: there were hyperplasia of the bile duct epithelium with fibroblastic proliferation and degenerative changes in hepatocytes, congestion of blood vessels and inflammatory cell aggregations of lymphocytes, neutrophiles and plasma cells were observed (Fig. 9).

2-The spleen: was enlarged and tinged with ecchymotic focci of haemorrhages on the surface.

Microscopically: there were lymphoid depletion with necrosis of the lymphoid

follicles, congestion, prevascular cellular infiltrations with neutrophils and giant cells. Peritrabecular vacuolar degeneration (Fig. 10).

3-The kidneys: The surface appeared pale with some necrotic focci.

Microscopically: there was marked necrosis of the tubular epithelium. The interstitial tissues were focally infiltrated with inflammatory cells consisted of neutrophiles, lymphocytes and esinophiles, cystic dilatation of the renal tubules and heamorrhages were also seen (Fig. 11).

4-The intestine: The mucosa of the small intestine revealed congestion, erosion and pin point white focci on both mucosal and serosal surfaces. The colon was distended with yellowish fluids, which appeared watery to pasty with faeces and tinged with blood .The caecum appeared dilated and flaccid.

Microscopically: The small intestine showed atrophy of the villi and necrosis of the glandular epithelium (necrotizing enteritis) associated with inflammatory cell infiltrations consisted of lymphocytes and macrophages were seen in the lamina propria with some desquamated epithelial cells. Edema and intravascular heamolysis were observed in the mucosa and submucosa, also some glandular epithelium appeared hyperplastic. The colon showed desquamation of the epithelial lining with inflammatory cell aggregations mainly lymphocytes, neutrophiles and macrophages together with some tissue debris with mucous exudate filled the lumen with congestion of blood vessels. The villus tip enterocytes was degenerated and slaughed into the intestinal lumen, leaving denuded basement membrane and the exposed membrane allow fluid leakage and attract leukocytes into the lamina propria (Fig. 12).

DISCUSSION AND CONCLUION

There are several agents that can cause diarrhoea in young animals, such as aerobic and anaerobic bacteria as well as virus, in addition to parasites and protozoa (Acosta-Marting *et al.*, 1980 and Raghib *et al.*, 2004). Gram negative bacilli were the most frequent microbial agents causing diarrhoea in camel calves that cause devastating disease conditions ranging from enteritis to death depending on the host, bacterial agents and the nature of infected host (Kusters *et al.*, 1993). *E. coli*, implicated in the diarrhoea in young animals, can produce more than 3 enterotoxin proteins and several colonization factors, which are protein surface antigens that mediate the adhesion of pathogen to small intestinal mucosa (Gaastra and Svennerholm, 1996 and Pichel *et al.*, 2000). The observation that *E. coli* was isolated from diarrhoeic camel calves (41.1%), **(table 1)** confirm the reports of Willinger (1984) and Mohamed *et al.* (1998), (40%) and Schwatz and Dioli (1992), (30%). *E. coli* was the most prevalent Gram negative bacilli recovered

from diarrhoeic neonatal camel calves (Salih *et al.*, 1997) and (Dia *et al.*, 2000). Because of *E. coli* is a normal inhabitant in small intestine of all species of animals and the soil of animal yards (Wernery and Kaaden, 2002). The serological identification of *E. coli* strains isolated from diarrhoeic camel calves revealed that O55/K59 and O111/K58, **(Table 2)** were the most common *E. coli* serovars, as investigated by Awad *et al.* (1979) and El-Morsy (1989). The incidence of isolated *salmonella* spp. from diarrheic camel calves was 7.8 %, **(Table 1)**. Similar results were recorded by Zschack *et al.* (1987), Sobhi (1997) and Salih *et al.* (1997).

The serological identification of *salmonella* strains recorded that *S. Enteritidis* and *S. Typhimurium* **(Table 3)** were the most important serotypes of *salmonella* that cause diarrhoea, as reported by (Bengoumi *et al*, 1998). The higher percentage of isolation of *C. perfringens* reported in diarrhoeic camel calves (65.6%) than in the apparently healthy lambs (33.3%) was shown in **(Table 1)**. This may be due to the implication of *C. perfringens* as a common cause of diarrhoea in camel calves especially *C. perfringens* types C and D (Timony *et al.*, 1988). The typing of isolated *C. perfringens* revealed that the rate of C. *perfringens* type C was (14.4%) in the diarrhoeic camel calves, while it was (3.3%) in apparently healthy ones. The presence of *C. perfringens* type C in the fecal samples of diarrhoeic camel calves may be due to that the microorganism is a normal flora in the intestinal tract of camel calves. On the other hand *C. perfringens* Epsilon toxin (type D) was (27.8%) in diarrheic camel calves and (10%) in apparently healthy ones. But both Beta and Epsilon toxins (type B), the cause of lamb dysentery and sever Bloody tinged diarrhoea was (53.3%) and (23.3%) in diarrhoeic and apparently healthy camel calves, respectively **(Table 4).** The high rate of *C. perfringens* (type B) in the faecal samples of diarrhoeic camel calves is of great importance as it is the main cause of lamb dysentery and death in neonatal camel calves. The titration of *C. perfringens* antitoxins in the serum samples by using ELISA (Layana *et al.*, 2006) determined that, the Beta antitoxin titre was higher in the serum samples of apparently healthy camel calves (40 to 320) than that of diarrhoeic ones (40 to 80) **(Table 5)**, Martin *et al.* (1988). The Epsilon antitoxin titre ranged from (40-640) and (40-160) in the apparently healthy and diarrheic camel calves respectively **(table 6),** Naylor *et al.* (1987). The high level of Beta and Epsilon antitoxin titres was due to the apparently healthy camel calves were received colostrum from vaccinated dam that is means passive immunity lasts 2-6 weeks (Ripley and Gush, 1983) and (Odendaal *et al.* (1988). Also may be due to the fact that the *C. perfringens* represents a part of the bacterial flora of the intestinal tract of the dams (Radostitis *et al.*, 1995). The low titres of both

Beta and Epsilon antitoxins in the sera of apparently healthy and diarrhoeic camel calves may be due to the neutralization or agglutinating and haemaglutinating circulating antitoxins and the local antitoxins present in the gut lining epithelium (Rahman *et al.*, 1998). It was concluded that *C. perfringens* toxins were the cause of severe economic losses among farm animals according to the severity of the toxin types which had been recognized, which the control measures by increasing the specific transferred antitoxins by semi-annual booster injections. The results of sensitivity test illustrated that Amoxicillin + Calvulanic acid, Chloramphenicol , Gentamycine , Ciprofloxacin , Florofenicol, Rifampicin and Metronidazol , were highly effective on isolates, which were resistant to Sulpha drugs, Streptomycin, Neomycin and Cephalexin. The miss-use of antibiotics resulted in increasing the resistance of the isolates to the currently used drugs for treatment (Abu Elamreen *et al.*, 2007).

The revealed degenerative changes were observed in all organs of infected animals with marked necrosis in the hepatic and renal tissues also spleenic depletion and desquamation of the intestinal lining epithelium, all these microscopical changes indicated the toxaemic effect of *C. perfringens* Types C and D and the role of its toxins in the disease process .The histopathological findings recorded in the present study were similar to those reported by Rawhia, (2000) and Uzal *et al.*, (2002). Upon exposure to enterotoxin, the villus tip and enterocytes degenerated and sloughed into the intestinal lumen, leaving denuded basement membranes which allow fluid leakage and attract leukocytes into the lamina propria causing necrotizing enteritis of the small intestine in the infection with *C. perfringens* type C, also there were heamorrhagic necrosis in kidneys and necro-heamorrhagic hepato-splenitis in the infection with *C. perfringens* type D, as reported by Argenzio (1985) and Donald and James (2001). The lesions in the small intestine due to the bacterial toxins induced vasculitis and thrombosis of blood vessels in the lamina propria and submucosa resulting in focal intestinal infarctions with focci of ulceration which termed (Button ulcers) due to chronic enteric Salmonellosis which reported by Shah and Hala, (1992) and Dia *et al.*, (2000). Dilatation of the caecal glands which were filled with gases and the aggregation of the inflammatory cells also the congestion of blood vessels resulted in what is called heamorrhgic colitis which due to the infection with *E, coli* as reported by (Chauhan and Singh, 1993) and Werenery and Kaaden, (2002).

It could be concluded that *C. perfringens* types C and D, *Salmonella spp.* and *E. coli* induced severe pathological alterations and were associated with high

prevalence and mortality among camel calves that reflect on the demand for regular vaccination of animals against the disease with control measures and increasing the specific transferred antitoxins by semi-annual booster injections, especially it was reported that, the disease showed no response for the therapeutic treatment.

Table 1: Occurrence of E. *coli*, *Salmonella* and *C. perfringens* isolates in diseased and apparently healthy camel calves.

State of animals	Total No. of faecal swabs	Isolated *E. coli*		Isolated *Salmonella*		No. of isolated *C. perfringens*	
		NO.	%	NO.	%	NO.	%
Apparently healthy animals	30	4	13.3%	zero	zero	10	33.3%
Diarrhoeic animals	90	37	41.1%	7	7.8%	59	65.6%

Table 2: Serological identification of *E. coli* strains isolated from diarrhoeic camel calves.

OK group of *E. coli*									
O111 K58	O55 K59	O26 K60	O119 K69	O125 K70	O128 K67	O157 K7	O20 K11	Untyped	Total
9	13	Zero	3	5	Zero	Zero	7	4	37/41

Table 3: Serological identification of *Salmonella* strains isolated from diarrheic camel calves.

Serovars of *Salmonella*			
S.Typhimurium	*S. Enteritidis*	Untyped	Total
3	4	1	7/8

Table 4: Typing of *C. perfringens* Beta and Epsilon toxins in the fecal samples of camel calves.

Animal Group	No. of examined samples	Types of *C. perfringens* toxins				Both	
		Beta		Epsilon			
		No	%	No.	%	No	%
Apparently healthy camel calves	30	1	3.3	3	10	7	23.3
Diarrhoeic camel calves	90	13	14.4	25	27.8	48	53.3

Table 5: The titration of Beta antitoxin in the sera of camel calves.

Animal Group	N0. of examined samples	Rang	Mean
Apparently healthy camel calves	10/30	40-320	95/320
Diarrhoeic camel calves	59/90	40-80	125/80

Table 6: The titration of Epsilon antitoxin in the sera of camel calves.

Animal Group	No. of examined Samples	Rang	Mean
Apparently healthy camel calves	10/30	40-640	265/640
Diarrhoeic camel calves	59/90	40-160	230/160

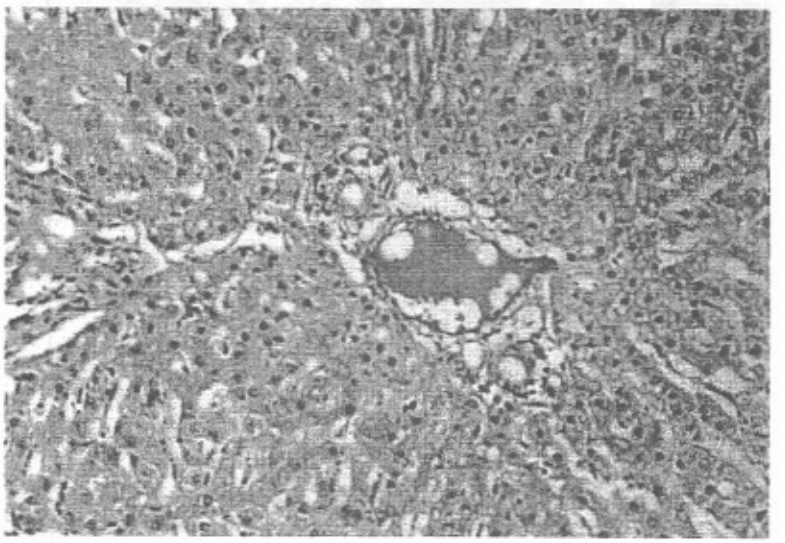

Fig 1: Liver of camel calf infected with E.coli showing marked degenerative changes and necrosis of hepatocytes. H&E(X10).

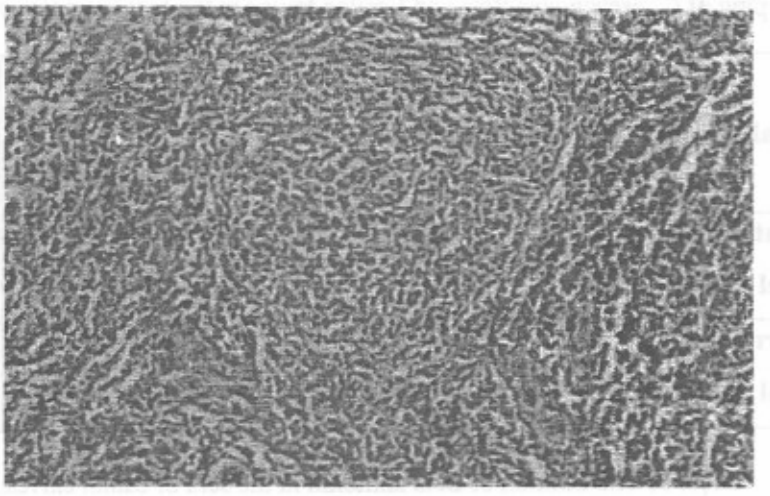

Fig 2: Spleen of camel calf infected with E.coli showing follicular deplesion and necrosis with haemolysis of erythrocytes .H&E (X10).

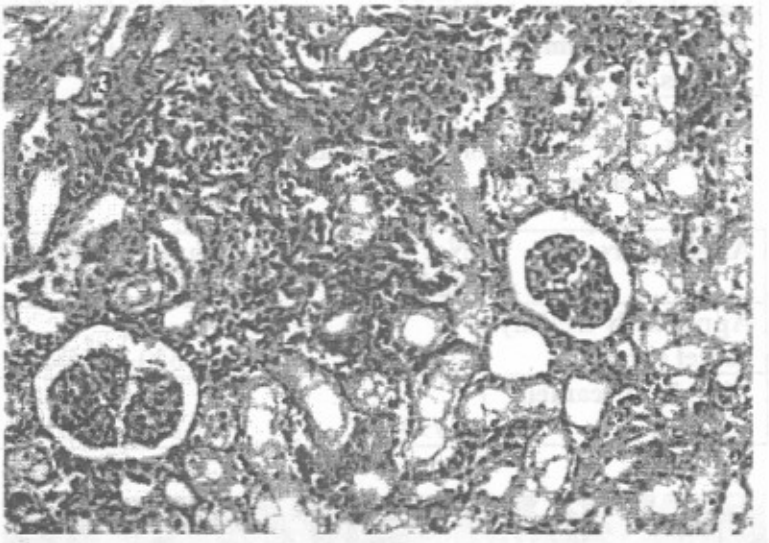

Fig 3: Kidney of camel calf infected with E.coli showing necrobiotic changes in the tubular epithelium and infilteration of mononuclear cells in the interstitial tissues.H&E(X10).

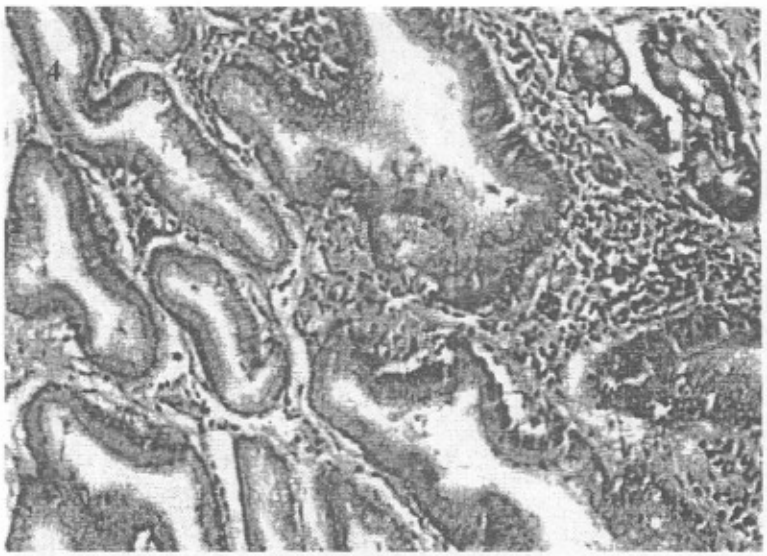

Fig 4: Intestine of camel calf infected with E.coli showing cystic dilatation of caecal glands with inflammatory cell aggregations.H&E(X10).

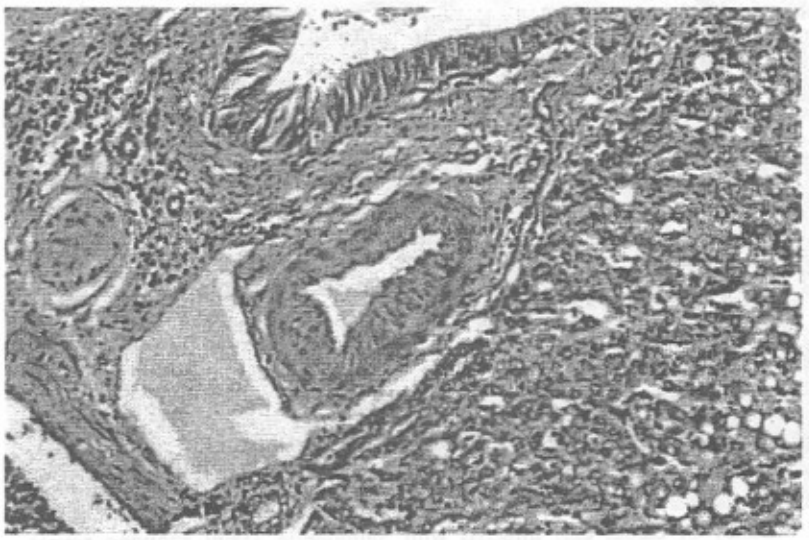

Fig 5: Liver of camel calf infected with Salmonella typhi showing edema, focal area of inflammatory cell aggregations, fatty degeneration and necrosis of hepatic cells. H&E(X10).

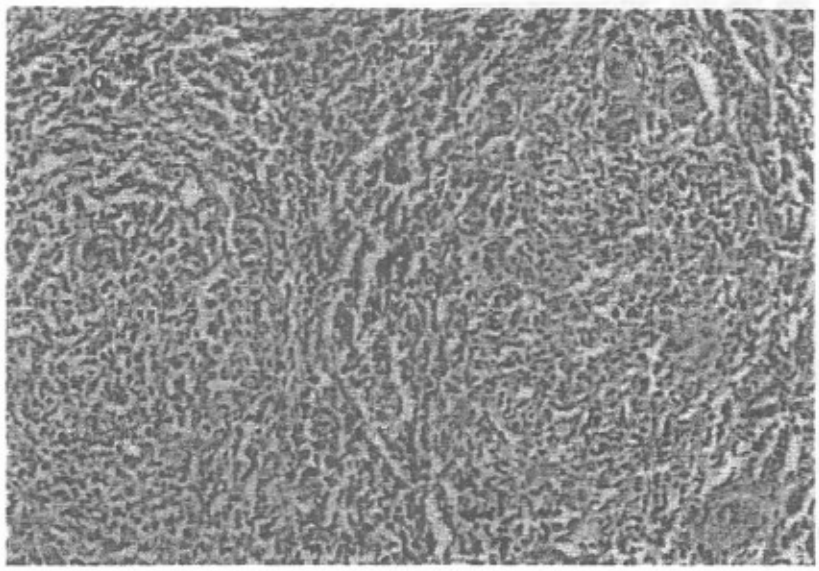

Fig 6: Spleen of camel calf infected with Salmonella typhyi showing hyperplasia with marked necrosis of lymphoid follicles and heamorrhages.H&E(X10).

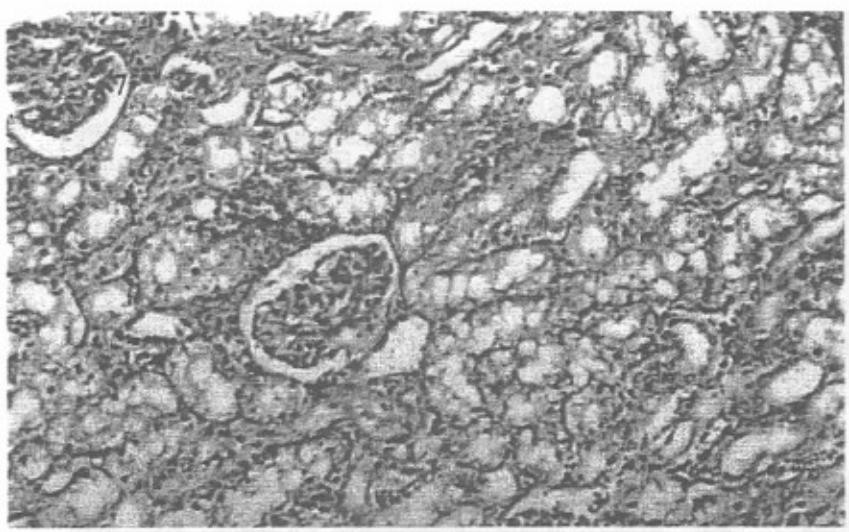

Fig7: Kidney of camel calf infected with Salmonella Typhi showing severe degenerative changes in the renal tubules and glomeruli with focal areas of hemorrhages.H&E(X10).

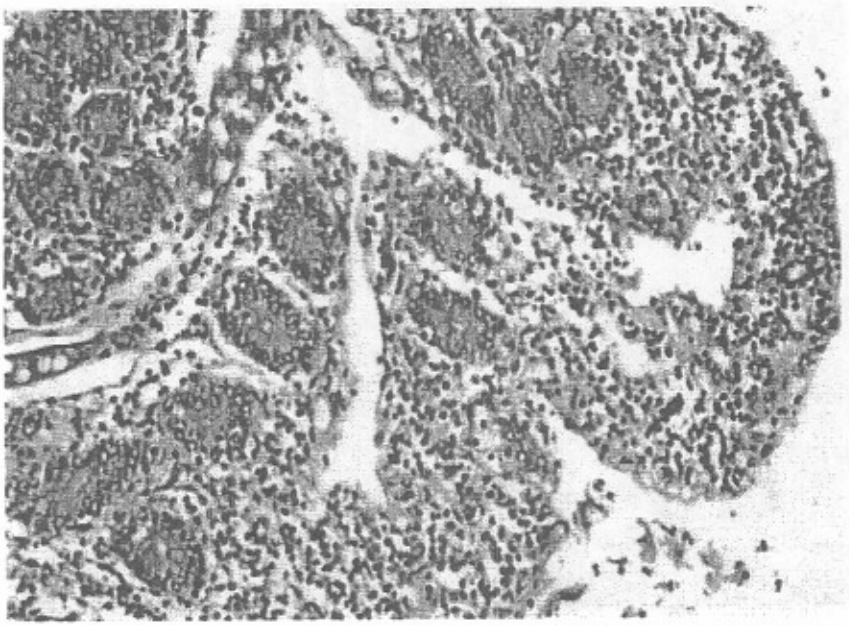

Fig 8: Intestine of camel calf infected with Salmonella Typhi showing degeneration and necrosis of the intestinal glands with desquamation of the epithelial cells of the intestinal villi which infilterated with mononuclear inflammatory cells and severe congested blood vessels .H&E(X10).

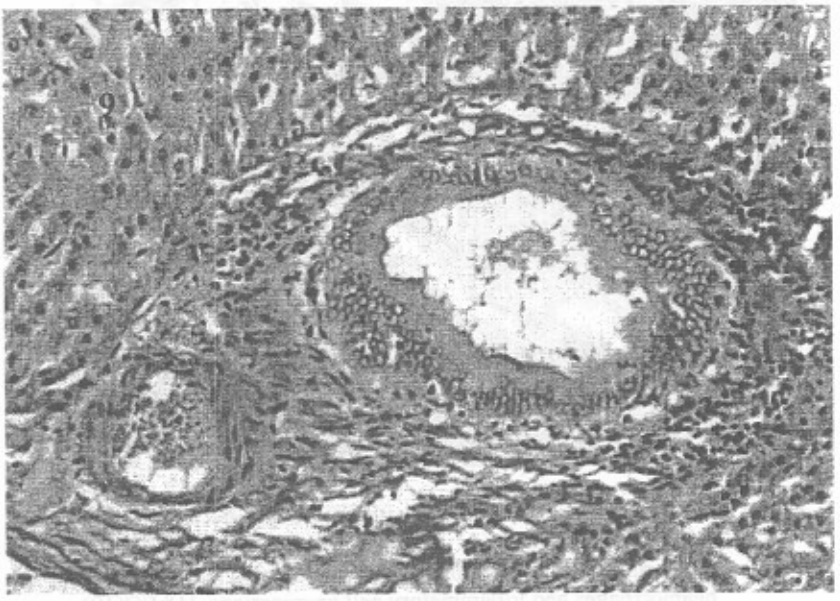

Fig 9: Liver of camel calf infected with clostridia perferinges type C&D showing hyperplasia of bile duct with fibroblastic proliferation, inflammatory cell aggregations and degenerative changes in the hepatocytes H&E(X10).

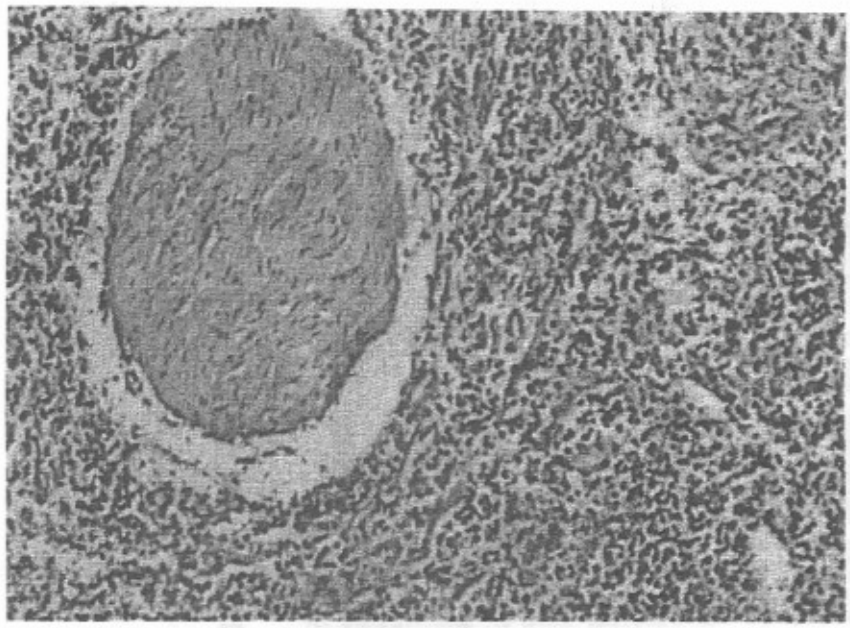

Fig 10: Spleen of camel calf infected with Closteridia perferinges type C&D showing congestion, prevascular cellular infilterations and peritrabecular vacular degeneration H&E(X10).

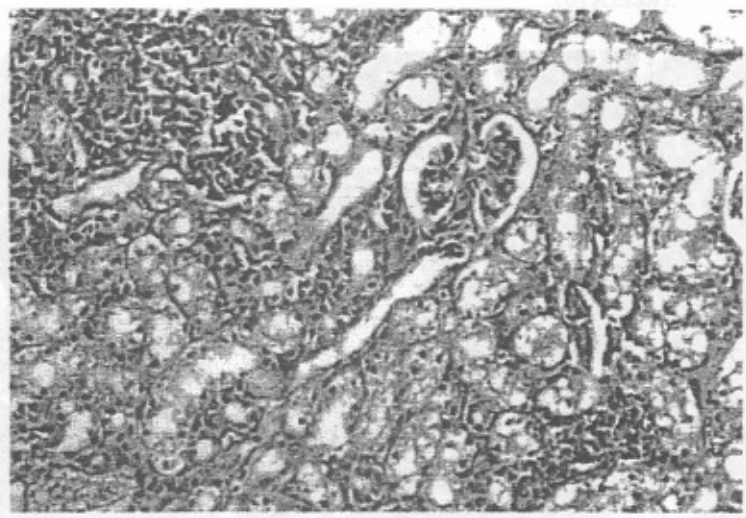

Fig 11: Kidney of camel calf infected with Closteridia perferinges type C&D showing marked necrosis of the renal tubules and glomeruli. Cystic dilatation of the renal tubules with interstitial heamorrhages and inflammatory cell aggregations. H&E(X10).

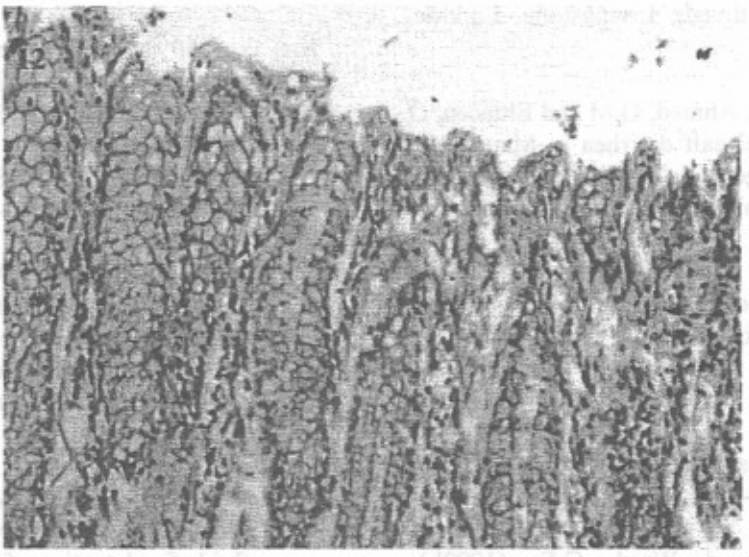

Fig. 12: Intestine of camel calf infected with Closteridia perferinges type C&D showing degeneration and desquamation of the villus tip enterocytes with inflammatory cell aggregations and the glandular epithelium appeared hyperplastic .H&E(X10).

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دراسات بكتريولوجية وباثولوجية على بعض الميكروبات الهوائية واللاهوائية المسببة للإسهال فى صغار الجمال

رمزى حمودة، ثناء كامل حسانين، أشرف نبيه

معهد بحوث التناسليات بالهرم

هذه الدراسة الحقلية صممت لايجاد التقنيات اللازمة للتغلب على مشكلة الاسهال فى الجمال الصغيرة وذلك بعزل وتصنيف ومعرفة التغيرات الهستوباثولوجية والعلاج وكذلك السيطرة على الميكروبات المسببة لهذه المشكلة. وأهم هذه الميكروبات(الميكروب القولونى، السالمونيللا والكلوستيريديم بيرفيرينجيز). لقد تم تجميع عينات البراز و الأجزاء المصابة من الأعضاء الداخلية (الكبد - الطحال- الكليتين والأمعاء) وكذلك عينات الدم من ١٢٠ بعرور (الجمل الصغير) تتراوح أعمارهم ما بين ١٠ الى ١٨ شهر وذلك للفحص البكتيريولوجى فى المعمل و الذى أسفرت نتائجه عن النسب التالية (٤١.١%)، (٧.٨%) و(٦٥.٦%) للميكروب القولونى، السالمونيللا والكلوستيريديم بيرفيرينجيز على التوالى من الجمال المصابة بالأسهال. بينما كانت النسب التالية (١٣.٣%)، (٠) و(٣٣.٣%) لنفس الميكروبات على التوالى من الجمال السليمة ظاهريا. وباجراء الفحص السيرولجى للعترات الخاصة بالميكروب القولونى والسالمونيللا التى تم عزلها، وجد أن العترات O111/K58 , (O55/K59) للميكروب القولونى والعترات (*S. Entertidis* and *S. Typhimurium*) للسالمونيللا هم أهم العترات المتسببة فى الاسهال فى الجمال الصغيرة. بينما فى الحالات المصابة بالكلوستيريديم بيرفيرينجيز وجد أن معدل السموم (البيتا والابسيلون) معا (٥٣.٣%) و (٢٣.٣%) فى الجمال المصابة بالأسهال و السليمة ظاهريا على التوالى. ولكن معدل هذه السموم كلا على حدا كان (١٤.٤%) و (٣.٣%) للبيتا و (٢٧.٨%) و (١٠%) للابسيلون فى الجمال المصابة بالأسهال و السليمة ظاهريا على التوالى. وقد أسفر الفحص الميكروسكوب عن وجود اضمحلال وتخثر لخلايا الكبد و الطحال و الكلى مع تساقط الخلايا المبطنة للأمعاء فى حالات الاصابة بميكروب الكلوستيريديم بيرفيرينجيز والذى يؤكد مدى التأثير السمى لهذا الميكروب على الخلايا. وقد شوهدت التهابات شديدة للأوعية الدموية مع بعض الجلطات والتقرحات فى طبقات الخلايا المبطنة للأمعاء فى حالات الاصابة بميكروب السالمونيللا. كما وجد تضخم فى حجم الغدد المعوية بالقولون نتيجة الالتهابات وتجمع الغازات به فى حالات الاصابة بالميكروب القولونى. وعند اجراء اختبار الحساسية للميكروبات المعزولة وجد أن الأموكسيسيللين + حامض الكلافيولينك والكلوامفينيكول والجنتاميسين والسيبروفلوكساسين والسفرادين من أقوى المضادات الحيوية تأثيرا على مكروبيى القولونى والسالمونيللا. بينما الفلوروفينيكول والتترادلتا والريفامبيسين والميترونيدازول كانت من أقوى المضادات الحيوية تأثيرا على ميكروب الكلوستيريديم بيرفيرينجيز.