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# THE PREVALENCE OF NEW BORN CALF-CAMEL SCOURS WITH SPECIAL REFLECTION TO EPIDEMIOLOGY, BACTERIAL ETIOLOGY AND PHYSIOLOGY PROCESSING AT TAIF, KSA

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**ABSTRACT**

Camels are the top herds wealth in KSA, since is considered as heavy animal population, breeding resist more dangerous diseases. Calf-camels are precious nuclei for veterinary farms. It's catch easily infection and ended by death. Present study based on the more infectious diseases at nursery period for neonatal calf-camels. The study was conducted at Taif, during 2012. Taif is a rich area of camels population and breeding. Data were collected included numbers of total calf-camels aged 0-14 days, scoured calf-camels, morbidity and mortality rates according the clinical signs and specimens collected for examination. Total calf-camels under study were 1200, morbidity rates were 240(20%) from all examined calf-camels, born at winter 130(54.2%) and at summer 110(45.8%). Mortality rates were 109(54.4%) from the scoured calf-camels, at winter 57(52.3%) and at summer 52(47.7%). The predominant isolates were *E. coli*(56.3%), *Clost. p.*(53.3%), *Cam. j.* (29.6%), *Sal. t.*(18.8%), *Prot. v. (*15.4%), *Ent. f.*(15%), *Br. a.*(14.2%), ETEC (9.6%) and *Yer. e.*(9.2%). The haematology parameters were at winter and summer as follows Blood pH 7.13 and 7.12, Hb 11.1 and 11.0 gm/dl, total RBCs 10.3 and $10.2X10^6$ /Cumm, PCV 52.1 and 52.3%, and total WBCs 11.2 and $11.7X10^3$ /Cumm respectively. The biochemical parameters were at winter and summer as follows total protein was 8.14 and 8.15 g/dl, sodium 117.1 and 117.2 m.Eq/L, potassium 7.3 and 7.4 m.Eq/L, chloride 69.5 and 69.8 m.Eq/L, urea 38.3 and 38.6 mg/dl, finally createnine 3.1 and 3.2 mg/dl respectively.

***Key words:*** *Hb, RBCs, PCV, Packed Cell Volume, WBCs.*

## INTRODUCTION

Calf scours is not a single disease entity. It is a clinical syndrome associated with several diseases characterized by diarrhoea. Absorption of fluids from the intestine is altered, with life threatening electrolyte imbalances occur; that is, the scouring calf losses fluids, rapidly, dehydrates, and suffers from electrolyte loss and acidosis. Infectious agents cause initial damage to the intestine, but actual deathfrom scours usually results from dehydration, blood acidosis, and loss of electrolytes (Mylrea, 1966). Neonatal calf diarrhoea (NCD), known as calf scour, is a common disease affecting the newborn calf. The most critical period is in the first few days following birth of the calf. Greatest losses occur when calves are kept in close confinement, where the transmission of the causative agents of NCD is enhanced by their buildup in the environment. The diarrhoea and other clinical signs seen with the disease are caused by the interaction of any of several possible infectious causes and predisposing factors such as lack of colostrum, failure to absorb colostrum antibody, poor nutrition and environmental effects. NCD is a costly disease, with losses estimated to be over 250million $annually and death loss of up to25% of the U.S. calf crop (Mylrea, 1966). The calves submitted for necropsy in their first two weeks of life had a watery and yellowish diarrhoea which was sometimes tinged with blood. These calves had diarrhoea for not more than 3days when they were received and most of them had signs of dehydration of variable severity. Most of the calves submitted alive had not been treated. At necropsy, the following gross lesions were generally observed, dehydration and the whole intestinal tract distended by a yellow watery content which often contained gas and poorly digested milk or only the large intestine distended by fluid. The intestinal mucosa was usually normal on gross examination. Whatever the etiology was, the clinical signs and gross lesions were almost the same (Morin *et al*., 1976). An outbreak of calf scours occurred among the offspring of a herd, calves born to heifers showed severe diarrhoea starting in 5-7days after

birth. Dehydration, inappetence and dejection developed within 24-48hrs. (Snodgrass *et al*., 1980). Diarrhoea occurs during the first few days of life. *E. coli* that causes the disease which possesses special attributes of virulence that allow them to colonize the small intestine and produce entero-toxins causes hypersecretion of fluid into the intestinal lumen. ETEC are shed in environment by infected animals in the herd and are ingested by newborn calves soon after birth. There is some natural immunity to ETEC, it often fails to protect calves born and raised under modern husbandry conditions. Hence, methods have been developed to stimulate protective immunity by vaccination of the dam, so protective antibodies are transferred passively to calves through the colostrum (Stephen, 1985).

Scours occurs when normal movement of water and out of the digestive tract is disrupted, resulting in water loss and dehydration. Loss of body fluids through diarrhoea is accompanied by loss of body salts, this fluid and electrolyte loss produces a change in body chemistry lead to severe depression in the calf and eventual death. Diarrhoea is common in newborn calves. Acute disease is characterized by progressive dehydration and death, sometimes in 12hr. In sub-acute form, diarrhoea may persist for several days and results in mal-nutrition and emaciation. The primary harm from scours is losses of water, electrolytes and salts in diarrhoea creates dehydration and alteration of the acid-base balance of the bodily fluids. Inflammation of intestinal lining impairs the calf's ability to digest nutrients, creating weight losses and the potential for hypo-glycemia.

These changes can be severe enough to result in death. Certain bacteria as *Sal.spp.* and *Clost. p*., release toxins cause harm to multiple vital organs in calves (King *et al*., 1993). Diarrhoea and deaths in newborn calf-camels were very high in KSA. Micro-organisms increasing the rates of morbidity and mortality is well estimated. Survey was conducted in diarrheic calf-camels aged 0-12months. Camel was resistant to most of the diseases. Camel was found to be susceptible to a large number of pathogenic agents.

Many factors contribute to calf mortality, among which is calf diarrhoea (Agab, 1993). Scours is often caused by pathogenic *E. coli.*, exposed to a cold, wet environment, lack of adequate colostrum antibodies, and exposed to soil bacteria. Calf scours is a broad, descriptive term referring to diarrhoea in calves. Calf scours is not a specific disease with a specific cause, actually a clinical sign of a disease complex with many possible causes (Clement *et al*., 1995). Calf-camel scours was an economically important disease which causing great losses in calf-camels all over the world (Mohammed *et al*., 2003). The major bacterial infectious agents that have been implicated in calf scours are *Sal. spp*. *E. coli*, K99, and *Clost. spp*. the most common pathogens in scouring calves less than 2months of age. (Acha *et al*., 2004). Calf scours causes major economic losses because of high mortality and morbidity, more than 50% of deaths of unweaned calves were due to diarrhoea (Smith, 2009). The only seasonal effect on incidence was for calf scours within 0-14 days of birth which was higher at winter compared to summer. The medium ages at occurrence of calf scours 0-14 days of birth (Charles *et al*., 1988). Calf-camel scours was 8% aged 0-12 months. (Mashref *et al*., 2012).

High calf mortality was considered one of the major constraints to higher productivity within calf-camels regarded the major causes of death (Salih *et al*., 1998). Mortality in calf-camel populations was 39.9% found to be higher aged less than 6 months in Sudan due to calf-camel scours (Ali *et al*., 2005). Bacteria was the most of fatal scours caused among newly born calf-camel, which produce toxins that degrade the intestinal lining. The calf responds to these toxins by pumping large amounts of water into the intestinal tract to flush out the toxin, bacteria were *E. coli*, *Sal. spp*., *Clost. p*., and other bacteria. *E. coli*, reported the greater incidence of isolated with greater frequency. Single infections are common, but mixed infections e.g., *E. coli*, *Sal.*,*E. coli*is the most common bacteria associated with calf scours. It causes calfscours by secreting a toxin that damages the cells lining the gut. *E. coli*, K99 causes enough damage to lining cells that large volumes of electrolytes and fluids are lost and death may occur (Abubaker *et al*., 2006). *E. coli*single most important cause of bacterial scours in calves, cause diarrhoea must first colonize or adhere to the calf's gut. It do so by means of very fine, fuzz-like protrusions known as pili or fimbriae. These pili may possess K99 antigen. *E. coli* strains possess K99 antigen are called entero-toxigenic *E. coli* (ETEC), whichhave the ability to produce toxins in the intestines. Most newborn calves are exposed to *E. coli*from the environment, when sanitation is marginal. Manure from healthy cows and faeces from scouring calves provide a source of *E. coli* for calves aged16-24hrs. The younger calves are greater the chance for death from progressive and severe dehydration.

The most common micro-organisms causing diarrhoea *E. coli* was 66% (Salih *et al*., 1998). ETEC, K99 strain runs a rapid, fatal course, toxins cause so much fluid to be pumped into the intestine, calf usually dies before external signs of diarrhoea. This type of scours is one of the few that occur within the first 3 days of life. Rapid form of *E. coli* often with no evidence of diarrhoea. Colostrum deprived calves usually die of this form of *E. coli*that affects calves within 10-14 days of age, usually within the first week, it was isolated 81% from diarrheic calves, while *E. coli* isolated 27.3% from young camels in KSA (Abubaker *et al*., 2006). *E. coli*and *Prot. spp*.

were the incriminated for calf-camel scours (Fouda and Al Mujialii, 2007). Prevalence of calf-camel scours was 8.0% aged 0-12 months, the main causes were *Br. a*. 8.98%, *E. coli*58.2%, ETEC 7% and *Ent. spp*. 8.8% respectively. (Mashref *et al*., 2012). *Sal. spp.* invades the mucosa of the small intestine causing inflammation and erosion of the intestinal lining. Infected calves can shed the organism in faeces, urine, saliva, and nasal secretions, which can survive in the environment for months.

The most common *Sal.* serotype is group B, typically *Sal.t.* Calves are usually severely affected, do not drink milk or milkreplacer. There is a high mortality rate among infected calves, with death occurring within 12-48 hrs. after the first signs appear, usually affect calves that are over 10 days old, especially *Sal. t.* and *Sal.d*.cause diarrhoea in calves aged 2-12 wks. old. *Sal. spp.* produce entero-toxins but are also invasive and produce inflammatory change within the intestine. The main causes group *Sal. spp*. were 12% (Mashref *et al*., 2012). *Clost. p.* infections were commonly known as entero-toxemia which is fatal and is caused by toxins released by various types of *Clost. p*.,it has a sudden onset and affected calves become listless, and strain or kick at their abdomen. *Clost. p.* is normally found in the intestine of cattle and can survive for months in the soil. There are many more cases of *Clost. spp.* infection that involves the abomasum, usually in calves aged 2-5 wks. Acute death with moderate bloating is often found. *Clost. p.* type A may be found in association with bloat. *Clost. p*. types A, B, C, and E produce a variety of necrotizing toxins and cause a rapidly fatal haemorrhagic enteritis in calves. Infection with type B or C is a common cause of enteritis and dysentery, that usually affects calves less than 10-14 days of age, revealed *Clost.p.* in 25.9%. (Zakia, 2004). *Camp. J.* and *Yer. e.* may be present in the faeces of calves with diarrhoea, prevalence of calf-camel diarrhoea was 8.0% in ages 0-12 months. The main causes were *Br. a*. that was 8.98%. (Mashref *et al*., 2012).

Calf scours cause dehydration, inappetence and dejection developed within 24-48 hrs., *E. coli*, K99 antigen was resulted (Snodgrass *et al*., 1980). The signs of diarrhoea attributed to dietetic errors, suckling managements, fermentation of milk and formation of lactic acidosis causing irritation of the mucosa of the gastro-intestinaltract leading to mal-digestion, mal-absorption, hyper-peristaltic and rapid passage of gastro-intestinalcontents resulting in significant increases of moisture of the faeces, increased respiratory rates with labored respiration may be trend to the decrease of blood pH which eliminate the excess of carbondioxide (El-Sheikh, 1987; Radostitis *et al*., 2000; El sheikh *et al*., 2004 and Radostits *et al*., 2007).

Scours syndromes represent the most serious digestive problems among newborncalves causing economic losses to producers due to high morbidity and mortality rates, the causesof diarrhoea are multifactorial included interactionbetween; calf, environment, nutrition and infectiousagents. It causesvarying degree of dehydration, gastroenteritis, bodyfluid loss and various body fluid changes (Smith, 2009). Dehydration andacidosis due to loss of body fluids and electrolytes includedbicarbonate, sodium, potassium and chlorides infaeces. The acidosis was manifested clinically byhyper-ventilation and increase the respiratory rate.Higher concentration of potassium inblood resulted in bradycardia and cardiac arrhythmia oreven death. The increases of Hb, PCV may be attributed to dehydration and thereduction of water content in the vascular space (Coles, 1986; Radostitis *et al*., 2000). Metabolic acidosis lead to excessive loss ofbicarbonate in faeces,excessive production of lacticacid in body tissue by anaerobic glycolysis, organicacid production by abnormal gut flora and limitation ofrenal excretion of hydrogen ion (El-Sheikh, 1987). The slight increase in serum totalprotein screened the excessive loss of body fluids andconcentration of some blood component. However, protein loss by catabolism or by leakage into intestinallumen was lowered in diseased calf (Scott *et al*., 2004).

The elevation of serum urea andcreatenine attributed to hypo-volemic, reduced renalperfusion rate and function as well as increasing thecatabolism of protein by increasing the degree ofdehydration (Deshpande *et al*., 1993; Schlerka and Baumgartner, 1995). The gradual decreases of serum sodium andchloride with gradual increases of serum potassiumlevels in diarrheic calves which loss of sodium and chloride with intestinal secretion which associated with diarrhoea (Kaneko *et al*., 1997; Radostitis *et al*., 2000). Serum chlorides follows sodium level because chloride wasusually found in the form of sodium chloride, excessive excretion of potassium in scouring faeces 17.5 times than normal. Decreaserenal tubular excretion of potassium, body madecompensatory mechanism by moving hydrogen ions incases of metabolic acidosis and during catabolism into the intracellular fluids, this movement of hydrogen ionsinto cells would force the potassium ions toextra-cellular fluids resulted in hyper-kalemia (Scott *et al*., 2004). Academia a significant increase in RBCs count, haemoglobincontent and PCV with a non-significant variation in WBCs count. Totalprotein, significant increase of serum urea and createnine as well as hypo-natremia, hypo-chloremia and hyper-kalemia (Abdel Khalek *et al*., 2012).

The aim of the present study was designed to evaluate the performance of the breeding camel herds through morbidity and mortality rates of calf-camel scoursaged 0-14 days.

## MATERIALS and METHODS

**Study area:** The study was conducted at Taif, KSA during 2012 year, Taif is a rich area for camel herds population and breeding, though selected camel breeding farms. The main targets of study were discussed with farm owners then got their approves for research work. Each farm was weekly visited to square the infected scoured calf-camels at age 0-14 days by team work, that with endorsement from farm owners for any new calf-camel scours infections for follow up. Data collected included numbers of total calf-camels at age 0-14 days in each farm, infected calf-camel by scours, morbidity and mortality rates of calf-camel scours according the clinical signs and specimens collected for examinations.

**Sample collection and preparation:** The total calf-camels was in age 0-14 days under study during 2012 year were 1200 ones. The scoured calf-camels were240, distrusted as130 at winter other and 110 at summer. A total of 240 faecal specimens from scoured calf-camels were collected in sterile plastic bags and kept in ice box for bacteriological examinations. Blood specimens were with drawn of 240 scoured calf-camels from jugular vein. The blood specimens were divided in two parts. The 1$^{st}$part was collected in sterile screwcap heparinized tube for haematology examinations, the 2$^{nd}$part was collected in sterile screwcap non-heparinized tube for serological and biochemical examinations. A total of 15 healthy calf-camels clinically normal were taken as control for the same study examinations. All specimens were tested at Al-Janadriya Veterinary Center, Taif City, KSA.

**Laboratory patterns:**

**Bacteriological examination tests:**

**Faecal samples:**Specimens were inoculated on blood agar, discrete colonies were subcultured on MacConkey and XLD. Pure cultures were preserved on brain heart infusion broth under −20 °C. Identification of *Ent. spp.*were done by using standard conventional and commercial tests was then performed, used API 20E Strep system (Bio Merieux, Cedex, France) and Micro Scan (Smith *et al*., 2008).

**Detection of ETEC antibodies:** It was detected by ELISA. (Paton and Paton, 1998).

**Detection of *Sal.*antibodies:** Strains presenting a biochemical profile suggestive of *Sal.* were submitted to additional biochemical tests, were differentiated serologically (Ewing, 1986; Popoff, 2001; Rayan and Ray, 2004).

**Detection of *Br.*antibodies:** Sera were screened using ELISA and RBPT (Alton *et al*., 1988).

**Detection of *Clost. p.* antibodies:** Specimens were isolated and identified biochemically, biologically for toxin identification by neutralization test using polyvalentand monovalent antitoxin, ELISA and PCR (Quinn *et al*., 1994; Meer and Songer 1997; Uzal and Songer, 2008).

**Haematologicaland Biochemical examination tests:** Haematology were for blood pH, haemoglobincontent (Hb), Total erythrocytes counts (TRBCs), packed cell volume (PCV) and leucocytes counts (TWBCs), and biochemical were total proteins, sodium, potassium, chloride, urea and createnine levels (Coles, 1986; Kaplan and Pesce, 1996).

**Data Analysis:** Data were summarized and analyzed using SPSS version 16 computer program, which using Epi Info version 6 statistical software, and Chi-square test at critical probability of $p<0.05$ (Coulombier *et al*., 2001).

## RESULTS

**Table 1:** The Prevalence of Morbidity and Mortality Rates of calf-camel scours According to seasons

| *No. 1200 | Morbidity *% | | Mortality *% | |
|---|---|---|---|---|
| **Seasons** | **Winter** | **Summer** | **Winter** | **Summer** |
| **No. (%)** | 130 (54.2%) | 110 (45.8%) | 57 (52.3%) | 52 (47.7%) |
| **Total** | **240/1200 = 20%** | | **109/240 = 54.4%** | |

*No: Number, *%: Percentage.

Table 1 shows the prevalence of morbidity and mortality rates of calf-camel scours according to seasons. Total calf-camels under study were 1200. Morbidity percentage were 240(20%) from all examined calf-camels, which divided at winter 130(54.2%) and at summer 110(45.8%). Mortality percentage was 109(54.4%) from the scoured calf-camels divided at winter 57(52.3%) and at summer 52 (47.7%).

**Table 2:** The percentage of bacteria isolated from scoured calf-camels according to seasons

| Seasons *No. | *Ent. f. *% | *E. coli % | *ETEC % | *Sal. T. % | *Prot. v. % | *Cam. j. % | *Yer. e. % | *Br. a. % | *Clost. p. % |
|---|---|---|---|---|---|---|---|---|---|
| **Winter No. 130** | 16.2% | 59.2% | 10.8% | 20% | 16.9% | 31.5% | 10.8% | 15.4% | 55.4% |
| **Summer No. 110** | 13.6% | 52.7% | 8.2% | 17.3% | 13.6% | 27.3% | 7.3% | 12.7% | 50.9% |
| **Total No. 240** | **15%** | **56.3%** | **9.6%** | **18.8%** | **15.4%** | **29.6%** | **9.2%** | **14.2%** | **53.3%** |

*No.: Number, *%: Percentage, *Ent. f.: Enterococcus faecalis, *ETEC: Enterotoxigenic Escherichia coli, *E. coli: Escherichia coli, *Sal. t.: Salmonella typhimurium, *Prot. v.: Proteus vulgaris, *Camp. j.: Campylobacter jejuni, *Yer. e.: Yersinia entercolitica, *Br. a.: Brucella abortus, *Clost. p.: Clostridium perfringens.

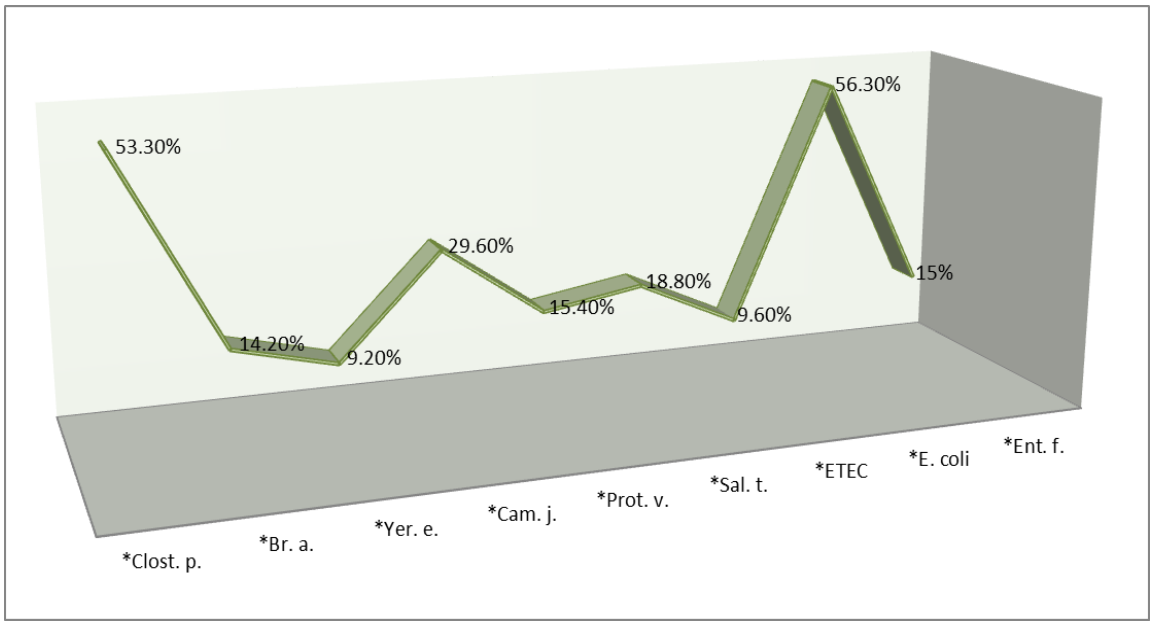


**Diagram 1:** The Percentage of Bacteria Isolated From Scoured Calf-Camels According to Seasons

Table 2 and Diagram 1 show the percentage of bacteria isolated from scoured calf-camels according to seasons. The predominant isolates were *E. coli*(56.3%), *Clost. p.*(53.3%), *Cam. j.*(29.6%), *Sal. t.*(18.8%), *Prot. v.*(15.4%), *Ent. f.*(15%), *Br. a.*(14.2%), ETEC (9.6%) and finally *Yer. e.*(9.2%). These isolates were at winter and summer 59.2 and 52.7%, 55.4 and 50.9%, 31.5 and 27.3%, 20 and 17.3%, 16.9 and 13.6%, 16.2 and 13.6%, 14.5 and 12.7%, 10.8 and 8.2%, 10.8 and 7.3% respectively.

**Table 3:** The Percentage Differences of Infections Bacteria Isolates According to Seasons

| Isolates | *Ent. f. *% | *E. coli % | *ETEC % | *Sal. t. % | *Prot. v. % | *Cam. j. % | *Yer. e. % | *Br. a. % | *Clost. p. % |
|---|---|---|---|---|---|---|---|---|---|
| **Differences (%)** | **2.6%** | **6.5%** | **2.6%** | **2.7%** | **3.3%** | **4.2%** | **3.5%** | **2.7%** | **4.5%** |

*% = Percentage, *Ent. f.: Enterococcus faecalis, *ETEC: Enterotoxigenic Escherichia coli, *E. coli: Escherichia coli, *Sal. t.: Salmonella typhimurium, *Prot. v.: Proteus vulgaris, *Camp. j.: Campylobacter jejuni, *Yer. e.: Yersinia entercolitica, *Br. a.: Brucella abortus, *Clost. p.: Clostridium perfringens.

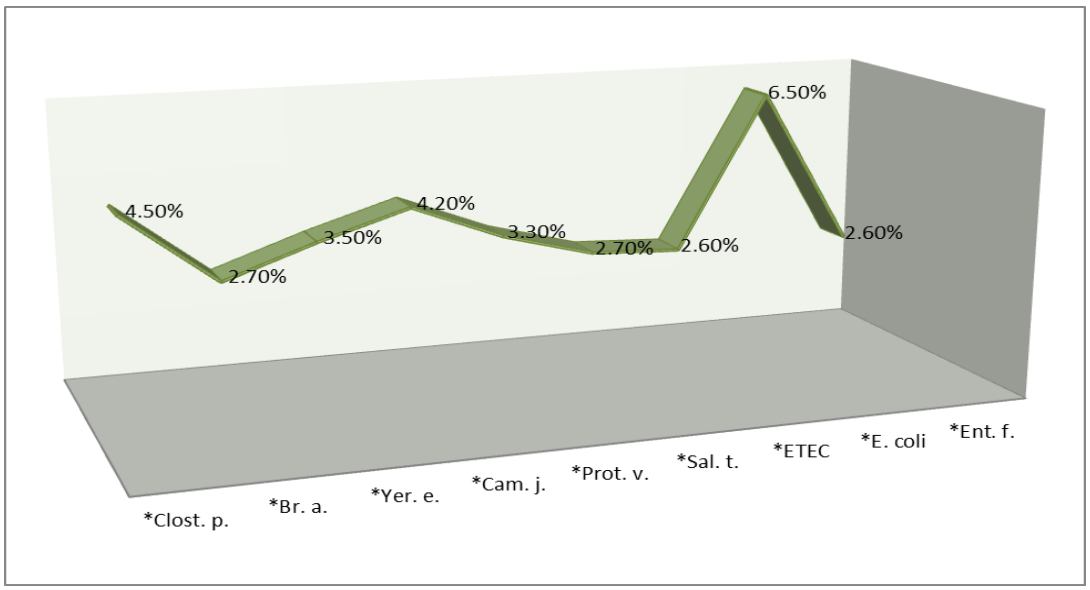


**Diagram 2:** The percentage differences of infections bacteria isolates according to seasons

Table 3 and Diagram 2 show the percentage differences of infections bacteria isolates according to seasons. The most difference in isolation *E. coli*, then followed by *Clost. p*., *Cam.j*., *Yer. e*., *Prot. v*., (*Sal. t*.;*Br. a*.) and (*Ent. f*.; ETEC) were 6.5, 4.5, 4.2, 3.5, 3.3, 2.7 and 2.6% respectively.

**Table 4:** The Mean Haematological Parameters of Infected Scoured Calf-Camels According to Seasons

| Haematology parameters | Control calf-camels | Scoured calf-camels | |
|---|---|---|---|
| | | Winter | Summer |
| **Blood PH** | **7.4** | 7.13 | 7.12 |
| ***Hb** | **9.8 gm/dl** | 11.1 gm/dl | 11.0 gm/dl |
| ***TRBCs** | **7.4 X $10^6$ /Cumm** | 10.3 X $10^6$ /Cumm | 10.2 X $10^6$ /Cumm |
| ***PCV** | **33.5 %** | 52.1 % | 52.3 % |
| ***TWBCs** | **7.6 X $10^3$ /Cumm** | 11.2 X $10^3$ /Cumm | 11.7 X $10^3$ /Cumm |

*Hb= Haemoglobin, *TRBCs = Total Red Blood Cells, *PCV = Packed Cell Volume, *TWBCs = Total White Blood Cells

Table 4 showsthe parameters were higher than normal values due to dehydration and had a very little differences between winter and summer as follows, Blood pH was 7.13 and 7.12, Hb 11.1 and 11.0gm/dl, TRBCs 10.3 and 10.2X$10^6$/Cumm, PCV 52.1 and 52.3%, finally TWBCs 11.2 and 11.7X$10^3$/Cumm.

**Table 5:** The Meanbiochemical Parameters of Infected Scoured Calf-Camels According to Seasons

| Biochemical parameters | Control calf-camels | Scoured calf-camels | |
|---|---|---|---|
| | | Winter | Summer |
| **Total Proteins** | **6.3 g/dl** | 8.14 g/dl | 8.15 g/dl |
| **Sodium** | **134.1 m.Eq/L** | 117.1 m.Eq/L | 117.2 m.Eq/L |
| **Potassium** | **5.4 m.Eq/L** | 7.3 m.Eq/L | 7.5 m.Eq/L |
| **Chloride** | **90.6 m.Eq/L** | 69.5 m.Eq/L | 69.8 m.Eq/L |
| **Urea** | **30.3 mg/dl** | 38.3 mg/dl | 38.6 mg/dl |
| **Createnine** | **1.6 mg/dl** | 3.1 mg/dl | 3.2 mg/dl |

Table 5 shows the mean biochemical values of infected camel-calves by scours according to seasons. The parameters were higher than normal values due to dehydration and had a very little differences between winter and summer as follows, total proteins was 8.14 and 8.15g/dl, sodium 117.1 and 117.2m.Eq/L, potassium 7.3 and 7.4m.Eq/L,chloride 69.5 and 69.8m.Eq/L, urea 38.3 and 38.6mg/dl, finally createnine 3.1 and 3.2mg/dl..

## DISCUSSION

Table 1 shows the prevalence of morbidity and mortality rates of calf-camel scours according to seasons. Total calf-camels under study were 1200. Morbidity percentage were 240(20%) from all examined calf-camels, which divided at winter 130(54.2%) and at summer 110(45.8%). Mortality percentage was 109(54.4%) from the scoured calf-camels divided at winter 57(52.3%) and at summer 52(47.7%). The only seasonal effect on incidence was for calf scours is within 0-14 days of birth which was higher at winter compared to summer. The median ages at occurrence of calf scours within 14 days of birth (Charles *et al*., 1988). Calf-camel scour was 8% in ages 0-12 months. (Mashref *et al*., 2012). High calf mortality was considered one major causes of death (Salih *et al*., 1998). Mortality was 39.9% in Sudan due to calf-camel scours (Ali *et al*., 2005).

Table 2 and Diagram 1 show the percentage of bacteria isolated from scoured calf-camels according to seasons. The predominant isolates were *E. coli* (56.3%), *Clost. p.* (53.3%), *Cam. j.* (29.6%), *Sal. t.* (18.8%), *Prot. v.* (15.4%), *Ent. f.* (15%), *Br. a.* (14.2%), ETEC (9.6%) and finally *Yer. e.* (9.2%). Infectious bacterial causes were *E. coli*, *Sal. spp*., *Clost. p*.,*E. coli* cause calf scours by secreting a toxin that damages the cells lining the gut. This type of *E. coli*, K-99, does not invade the gut cells nor kill calves *E. coli*as a single most important cause of bacterial scours in calves was previously stated by Abubaker *et al*., 2006.*E. coli* strains which possess K99 antigen are called ETEC, have the ability to produce toxins in the intestines. Some ETEC produce other types of pili antigens. The most common micro-organisms causing diarrhoea *E. coli* was 66% (Salih *et al*., 1998). *E. coli* was isolated (27.3%) from young camels in KSA (Abubaker *et al*., 2006). Bacteriological examination revealed *E. coli* and *Prot. spp*. were the incriminated (Fouda and Al Mujialii, 2007). Prevalence of calf-camel scours was 8.0% in ages 0-12 months. The main causes were group *Br. a*. detected in 8.98%. *E. coli* was isolated in 58.2%. *Sal. spp*. and *Ent. spp*. were detected as 12% and 8.8% respectively. ETEC was 7% (Mashref *et al*., 2012). The most common *Sal.* serotype is group B, typically *Sal. t.* There is a high mortality rate among infected calves, with death occurring within 12-48 hrs. after the first signs appear. The case usually affect calves that are over 10 days old, especially *Sal. t.* and *Sal. d.*, but occasionally other serovars, cause diarrhoea in calves 2-12 wks. old. The main causes were group *Sal. spp*. which were 12% (Mashref *et al*., 2012). *Clost. p.* type A may be found in association with bloat. *Clost. p.* types A, B, C, and E produce a variety of necrotizing toxins and cause a rapidly fatal haemorrhagic enteritis in calves. The disease in calves is rare and usually sporadic. Infection with type B or C is a common cause of enteritis and dysentery. Usually affects calves less than 10-14 days of age. The results revealed *Clost. p.* in 25.9%. (Zakia, 2004). *Camp.J.* and *Ye.e.* cause diarrhoea in neonatal calves. Prevalence of calf-camel diarrhoea was 8.0% in ages 0-12 months, the main causes were *Br. a.* detected in 8.98%. (Mashref *et al*., 2012).

Table 3 and Diagram 2 show the percentage differences of infections bacteria isolates according to seasons. The most difference in isolation *E. coli*, then followed by *Clost. p*., *Cam. j*., *Yer. e*., *Prot. v.*, (*Sal. t.; Br. a*.) and (*Ent. f.;* ETEC) were 6.5, 4.5, 4.2, 3.5, 3.3, 2.7 and 2.6% respectively. The only seasonal effect on incidence was for calf scours within 0-14 days of birth which was higher at winter compared to summer. The medium ages at occurrence of calf scours within 14 days of birth (Charles *et al*., 1988).

Table 4 Haematology parameters showed higher than normal haematological values due to dehydration. Very little differences were reduced in values between winter and summer as follows, Blood pH was 7.13 and 7.12, Hb 11.1 and 11.0gm/dl, TRBCs 10.3 and $10.2X10^6$/Cumm, PCV 52.1 and 52.3%, finally TWBCs 11.2 and $11.7X10^3$/Cumm.Most serious complication of diarrhoea are dehydration and clinical metabolic acidosis. Haematoloical estimations revealed a significant CPL increases of TRBCs. count, PCV%, Hb content with increases of TWBCs (Radostitis *et al*., 2000). The increases of Hb, PCV may be attributed to dehydration and there duction of water content in the vascular space (Coles ، 1986 and Radostitis *et al*., 2000). Metabolic acidosis attributed to excessive loss of bicarbonate in faeces, excessive production of lacticacid in body tissue by anaerobic glycolysis, organic acid production by abnormal gut flora and limitation of renal excretion of hydrogen ion (El-Sheikh, 1987). The slight increase in serum total proteins level may be attributed to the excessive loss of body fluids and concentration of some blood component. However, the protein loss by catabolism or by leakage into intestinallumen was lowered in diseased calf (Scott *et al*., 2004). The serum urea and createnine levels were increased perhaps due to hypo-volemia, reduced renal perfusion rate and function as well as increasing the catabolism of protein by increasing the degree of dehydration (Deshpande *et al*., 1993; Schlerka and Baumgartner, 1995). Mean biochemical parameters of infected calf-camels by scours according to seasons are presented in table 5. The parameters were higher than normal values due to dehydration and had a very little differences between winter and summer as follows; total proteins level was 8.14 and 8.15g/dl, sodium 117.1 and 117.2m.Eq/L, potassium 7.3 and 7.4m.Eq/L, chlorides 69.5 and 69.8m.Eq/L, urea 38.3 and 38.6mg/dl, finally createnine 3.1 and 3.2mg/dl. Most serious complication of diarrhoea are dehydration and clinical metabolic acidosis. Serum showed a significant increase in the level of protein

and createnine. Serum electrolytes levels revealed a highly significant decrease of serum sodium, a significant decrease of chloride, meanwhile the serum potassium level was significantly increased (Radostitis *et al*., 2000). The higher concentration of potassium inblood resulted in bradycardia and cardiac arrhythmia or even death (Coles ،1986 and Radostitis *et al*., 2000). Metabolic acidosis is attributed by excessive loss of bicarbonate in faeces, excessive production of lacticacid in body tissue by anaerobic glycolysis, organicacid production by abnormal gut flora and limitation of renal excretion of hydrogen ion (El-Sheikh, 1987). The slight increase in serum total protein may be attributed to the excessive loss of body fluids and concentration of some blood component. However, the protein loss by catabolism or by leakage into intestinallumen was lowered in diseased calf (Scott *et al*., 2004). The serum urea and createnine levels were increased, that could behypo-volemia, reduced renal perfusion rate and function as well as increasing the catabolism of protein by increasing the degree of dehydration (Deshpande *et al*., 1993; Schlerka and Baumgartner, 1995). There is gradual decreases of serum sodium and chlorides with gradual increases of serum potassium levels. The changes to the loss of sodium and chloride with intestinal secretion which associated with diarrhoea. Serum chlorides level usually follows sodium level because chloride is usually found in the form of sodium chloride. The increase of serum potassium level was previously stated by (Kaneko *et al*., 1997; Coles, 1986 and Radostitis *et al*., 2000). Excessive excretion of Potassium, also increases serum potassium level in scouring faeces as 17.5 times than normal. Decreasedrenal tubular excretion of potassium. The body was compensate mechanism by moving hydrogen ions in cases of metabolic acidosis and during catabolism into the intra-cellular fluids, this movement of hydrogen ions into cells would force the potassium ions to extra-cellular fluids resulted in hyper-kalemia (Scott *et al*., 2004).

**Conclusion:** The economic diseases losses of the camel herds should be controlled by regular legal vaccination of camels. It can also recommended to vaccinate the animals in KSA at regular intervals to reduce in the prevalence and losses of this disease in numbers of calf-camel. Pregnant she-camel should not be moved within the last two weeks before calving for reducing the transmission of microbial agents. Feeding colostrumto calf-camels during 0-14 daysage is strongly advised. Also maintaining the field sanitation during nursery calf-camels period is necessary. Treatments must be restricted for scoured calf-camels for cure and lowering the sources of infection.

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## مدي الاصابة بالإسهال لعجول الابل حديثي الولادة بالإشارة الخاصة الي الحالة الوبائية، المسبب البكتيري والهيئة الفسيولوجية بالطائف، المملكة العربية السعودية

***محمد سالم الحربي***

الإبل هي الثروة الأعلى في المملكة العربية السعودية، تعتبر من القطعان الجيدة في التربية والتكاثر حيث تقاوم الأمراض الأكثر خطورة. عجول الابل نواه ثمينة للمزارع البيطرية، لكنها ضعيفة جدا للعدوى حيث تنتهي بالنفوق. استنادا إلى دراسة الأمراض المعدية في فترة الحضانة لعجول الابل حديثي الولادة، أجريت هذه الدراسة بالطائف، المملكة العربية السعودية خلال عام 2012، الطائف هي منطقة غنية بالإبل للتربية والتكاثر. وتم جمع البيانات حيث شملت أعداد عجول الإبل حديثي الولادة في عمر ( 0-14يوم). تم تسجيل الاعراض الاكلينيكية لتحديد العجول المصابة بالإسهال والنافقة، تم جمع العينات للتحليل. حيث كان إجمالي عجول الابل قيد الدراسة 1200، العجول المصابة (240) 20٪ من جميع العجول قيد الفحص، وعددها في الشتاء 130 ( 54.2٪) في الصيف 110(45.8٪). وكان معدل وفيات العجول 109 ( 54.4٪) من العجول المصابة، في الشتاء 57 ( 52.3٪) في الصيف 52 (47.7%). العزلات البكتيرية كانت كالتالي :*E. coli*56.3%، *Clost. p.* 53.3%، *Cam. j.* 29.6%، *Sal. t.* 18.8%، *Prot. v.* 15.4%، *Ent. f.* 15%، *Br. a.* 14.2%، ETEC 9.6% و *Yer. e.* 9.2%. القياسات الدموية في الشتاء والصيف كانت علي النحو التالي درجة الحموضة للدم 7.13 و pH7.12، الهيموجلوبين 11.1 و 11 gm/dl، العدد الكلي لكرات الدم الحمراء 10.3 و 10.2 Cumm / $X10^6$ ، حجم الخلايا المترسبة 52.1 و 52.3 % والعدد الكلي لكرات الدم البيضاء 11.2 و 11.7 Cumm/ $X10^3$ . القياسات البيوكيميائية بينت ايضا في الشتاء والصيف كالتالي البروتين 8.14 و 8.14 g/dl، الصوديوم 117.1 و 117.2 m.Eq/L ، البوتاسيوم 7.3 و 7.4 m.Eq/L ، الكلوريد 69.5 و 69.8 m.Eq/L ، اليوريا 38.3 و 38.6 mg/dl والكريتينين 3.1 و 3.2 mg/dl .