Sudan University of Science and Technology
College of Veterinary Medicine

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Review of Literature on Viral, Bacterial and Parasitic Diseases of the One-humped Camel (*Camelusdromedarius*)

إستعراض الأدبيات في مجال الأمراض الفيروسية، والبكتيرية، والطفيلية التي تصيب الإبل وحيدة السنام

Graduation research project B.V.M. (V) – 2017/2018

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بسم الله الرحمن الرحيم

﴿أَفَلَا يَنظُرُونَ إِلَى الْإِبِلِ كَيْفَ خُلِقَتْ﴾

(صدق الله العظيم).

سورة الغاشية (17).

In The Name of Allah, the Most Gracious, the Most Merciful,

﴿Do they not look at the camels,

how they are created﴾

Al-Ghashiyah (17).

Dedication

We dedicate this work to our Mothers, Fathers, Brothers, Sisters and Friends with deep Love and Sincerity.

Acknowledgements

We thank our Allah who gave us the aptitude and patience to conduct and finish this work.

We are grateful to Professor/ Mukhtar Taha Abu-Samra for his continuous support, supervision of this work and fatherly compassion.

Our deep thanks and respect to all academic and technical staff of the College of Veterinary Medicine, Sudan University of Science and Technology.

Finally we are indebted and very grateful to our parents for their love and support over the years.

Introduction and justification of the literature search on the diseases of the one-humped camel (*Camelus dromedarius*)

Figure 1: *Camelus dromedarius* (brown Rashaidi).

The Genus *Camelus* was probably among the last of the major domestic species to be put to regular use by man. References of the one-humped camel as a domestic animal is mentioned in the Wholly Koran and the Old Testament of the Christian Bible but there is little direct evidence for an exact time of domestication.

Since its domestication 3000-4000 years ago; the one-humped camel (*Camelus dromedarius*) accompanied humans and provided many facilities in arid and semiarid areas. It is an indispensable species of domestic animal. It had been exploited by man in Asia and Africa in arid and semiarid areas- often being the only supplier of food and transport for people. It is a multipurpose animal that can be used for milk, meat, wool, hide, transport, races, tourism, agricultural work and beauty contests. No other domestic animal is able to provide as many variable services to humans.

Past and present experiences proved that the dromedary camels have very special anatomical characteristics, and many varied physiological mechanisms, which enable the animals to live, reproduce and produce milk and meat, and to work under extreme conditions of heat and aridness - even during periods of drought when cattle, sheep and goats barely survive.

The ten first countries in the world according to their camel population are Somalia, Sudan, Ethiopia, Niger, Mauritania, Chad, Kenya, Mali, Pakistan and India. According to FAOstat (2009) statistics, the Sudan with a camel population of 4.5 million heads, is the second country in the world after Somalia. The camel population in the Sudan is 18 % of the whole population

of camels in the world, with estimated camel population growth higher than the world's growth.

Abu-Samra (1970- 2018) reported that the camel potentials as a safety valve for present and future food security for pastoralists, agro-pastoralists, and even for urban populations is very promising. He recognized 13 constraints facing the development of this important species of domestic animal, among which: meager authentic reports on the various diseases especially in the newly growing intensive systems of camel husbandry, and decrease in birth and population growth rates resulting from high mortality rates among newly born camel calves at an early age from pneumonia and neonatal diarrhea. For these reasons a rigorous search of the literature was conducted on viral, bacterial and parasitic diseases that affect this important species of domestic animals with the hope that it will be of great help to veterinary practitioners, veterinary students, and researchers working on camels.

Bibliography

Abu-Samra (1970- 2018). Personal Communication.
FAOstat (2009). Food and Agricultural Organization of the United Nations, Rome, Italy.

Abstract

The present document was prepared because the authentic and refereed literature on camel diseases is meager, and many contradicting literature is encountered in different diseases of camels. For these reasons an extensive and rigorous search of the literature was conducted on the viral, bacterial and parasitic diseases of camels. In this document only correct data are included and are documented and illustrated with figures of the clinical picture of the disease and the pathological changes caused by these diseases in different organs.

We hope that the current research document will be useful to veterinarians practicing on camels, research workers interested in camel research, and veterinary students in the different universities. We also hope that this document will save them the effort of verifying correct and authentic information related to camel diseases.

ملخص البحث

لقد تم إجراء هذا البحث نسبة لشح الأدبيات الموثقة و المحكمة في مجال أمراض الإبل المختلفة، ووجود العديد من الإختلافات والتضارب في كثير من الأدبيات في مختلف أمراض الإبل، وعليه لقد تم إجراء مسح مكثف وصارم للأدبيات في الأمراض الفيروسية، والبكتيرية، والطفيلية التي تصيب الإبل وحيدة السنام مع التأكد من صحتحها وذلك بتوضيح العديد من هذه الأمراض إكلينيكياً و الآفات المرضية في الأعضاء المصابة بهذه الأمراض.

نرجو أن يكون هذا البحث مفيداً للأطباء البيطريين الممارسين للمهنة في مجال الإبل، وكذلك الباحثين الراغبين في إجراء البحوث في مجال أمراض الإبل، و الطلاب في كليات الطب البيطري المختلفه. كما نأمل أن يوفر عليهم هذا البحث مهمة التقصي عن صحة المعلومات المتعلقة بهذه الأمراض.

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Chapter 1

Viral Diseases of the One-humped camel (*Cemelus dromederius*)

Viruses Causing Clinical Disease in the One-humped Camel

- Rabies: *Lyssavirus*.
- Camel pox: *Orthopoxvirus cameli*.
- Contagious ecthyma (Orf): *Parapoxvirus camel strain*.
- Papillomatosis (Warts): *Papillomavirus*.
- Influenza: *Influenza*virus A.
- Neonatal diarrhoea: *Rotavirus* and *Coronavirus*.
- Borna disease (BDV): *Bornavirus* (progressive viral polioencephalomyelitis).
- Equine Herpesvirus (EHV-1): *Varicellovirus* (Neurological disorders, abortion).
- Rift Valley Fever (RVFV): *Phlebovirus*.
- Parainfluenza: *Parainfluenza virus* (PI-3).
- Peste Des Petits Ruminant (*Morbilivirus*).

Non-pathogenic Viruses for which the One-humped Camels were only Sero-positive

- Enzootic bovine leucosis (BVL).
- Ovine pulmonary adenomatosis (OPA).
- Equine infectious anemia (EIA).
- Caprine arthritis encephalitis (CAE).
- Foot-and-mouth disease (FMD).
- *Adenovirus.*
- *Parainfluenza virus 1, 2, 3.*
- *Bovine respiratory syncytial virus (BRS).*
- African horse sickness (AHS).
- Bluetongue (BTV).
- Mucosal disease (MDV).
- Vesicular stomatitis (VSV).
- Bovine viral diarrhea (BVDV).
- Infectious bovine rhinotracheitis (IBR).

Camel pox

Figure 2: Severe infection of a camel with camel po x showing multiple papules and nodules of pock lesi ons involving the head.

Etiology:

- Camel pox is a contagious skin disease caused by *Orthopoxvirus cameli* of the family *Poxviridae*. (Fowler , 2002).

Epidemiology:

- The virus is transmitted by direct contact with infected animals or by contaminated objects.
- Camel pox is widely distributed, and occurs wherever camel husbandry is practiced.
- The disease may be benign or malignant irrespective of sex, and usually affects 2-3 years old camels.
- The malignant form of the disease usually affects young camels that did not receive antibodies against the virus from their mothers resulting in high mortalities (15-30 %) in newborn calves which may be as young as 3-5 days of age. (Kritz . B , 1982).

Clinical signs:

- The incubation period of the disease is about 7-10 days, and the initial signs of the severe disease is marked elevation of body temperature (41.6 °C). The infected camels show anorexia, restlessness, nasal discharge, lacrimation and tendency to drool saliva.
- The lesions of pox start to appear in the form of erythematous wheals, followed by enlargement of the mandibular lymph nodes, pendulous lips, appearance of papules on the lips, eyelids and nostrils and swelling of the lips and nostrils.
- The papules develop into nodules with simultaneous appearance of thin-

walled vesicles containing serous exudate.

- The nodules and vesicles spread all over the head, neck, medial aspects of the legs, and perineum, but in many cases the lesions involve the udder, scrotum, and vagina, around the anus, buccal and nasal mucosa.
- The vesicles persist for 3-10 days and enlarge in size as the result of accumulation of the serous fluid, and change to pustules.
- The affected camels rub their lips against their body and rub other areas with their head.
- The pustules persist for a period of 7-10 days, and become dry forming a thick scab which persist for another 7-10 days after which period the scab covering the pustules is shed off leaving intact areas; or rupture as the result of rubbing and scratching and become invaded by secondary bacteria, and heal leaving scars.
- Vesicles are the early lesions seen in the oral cavity which later rupture leaving bleeding ulcers, the nasal cavity is inflamed, and in severe cases pock lesions may involve the trachea, esophagus and lungs.
- Females abort and males may develop orchitis.
- Newborn calves find great difficulty in eating or sucking their dams because of the lesions involving the buccal cavity, and while, some newborns experience difficulty in respiration others may develop pneumonia.
- Some camels develop keratitis followed by corneal opacity.(Maclachlan and Dubovi , 2011).

Diagnosis:

- The clinical picture.
- Examination of scabs from the lesions under the electron microscope.
- Isolation of the pox virus.
- Serological tests (AGPT and ELISA).
- Histopthological examination and presence of intracytoplasmic inclusion bodies in ballooned cells. (Fowler , 2002) .

Differential diagnosis:

- Camel pox should be differentiated from contagious ecthyma and papillomatosis. (Fowler , 2002).

Treatment:

There is no specific treatment for pox, but it is always preferable to give the following supportive treatment:

- Long acting oxytetracycline by the intramuscular route.
- The nostrils, eyes and muzzle should be cleaned and disinfected and

smeared with ointments containing, a larvicide, antihistamines and antibiotics.

- Animals showing anorexia are fed with 20 % glucose by the intravenous route.(Kritz . B , 1982).

Prevention and control:

1) Isolation and supportive treatment of infected animals.
2) Restriction of camel movement to and from infected areas.
3) Inactivated and live attenuated vaccine, but live vaccine is preferred as they provide long lasting immunity (Fowler , 2002).

Contagious pustular dermatitis
Contiguous ecthyma (Orf)

Figure 3: Camel infected with contagious pustular dermatitis showing lesions covered with cracked crusts involving the lips and mouth commissures.

Etiology:

- Orf is a contagious skin disease caused by *Parapoxvirus,* of the family *Poxviridae*. *(seyed et ., al , 2015)* .

Epidemiology:

- The virus is transmitted by direct contact with infected animals or by contaminated objects. Small injuries on the lips from browsing on thorny trees and bushes facilitate infection.
- Camel orf is widely distributed, and occurs wherever camel husbandry is practiced.
- The disease mostly infects calves although adult animals can be affected.
- The morbidity rate is high 50-80 % but mortality is low 10-15 %.
- Animals that recover become immune. (Fowler , 2002).

Clinical signs:

- The initial sign of contagious ecthyma is elevation of body temperature, lacrimation, depression and anorexia.
- The animal develop papules around the mouth and swelling of the face and neck.
- Papules develop into nodules involving the lips. These change into vesicles and then pustules which persist for about ten days after which period they become covered with multi-fissured scab. The scab becomes dry, hemorrhagic and ulcerated due to rubbing and scratching of the affected areas against hard objects and other parts of the body.
- The lesions in most of the cases is localized at the mouth commissures and nostrils. However, in newborn calves and young camels; the disease is acute and the lesions extend involving the mucosa of the mouth (buccal cavity and gums below the incisor teeth), and nostrils.
- Camel calves become weak and emaciated, and die due to their inability to graze or suckle their dams.
- There is edema of the eyelids, lips, muzzle and nostrils, and the parotid, submaxillary and cervical lymph nodes become enlarged.
- In case lesions involve all parts of the body; they are prevalent on the distal part of the legs, inner thighs and vaginal area.
- In adult camels the disease observed was of moderate severity.(Seyed et ., al , 2015)**.**

Diagnosis:

- The clinical picture, development and distribution of the lesions in the different parts of the body.
- Examination of scabs under the electron microscope.
- Agar gel precipitation test (AGPT) and (ELISA) are suitable serological tests for diagnosis using samples from the lesions.
- The virus can be identified using the polymerase chain reaction (PCR). (Seyed et., al ,2015).

Differential diagnosis:

- Contagious pustular dermatitis should be differentiated from pox and papillomatosis. (Seyed et .,al , 2015)**.**

Treatment:

There is no specific treatment for Orf, but it is always preferable to give the f ollowing supportive treatment:

- Long acting oxytetracycline by the intramuscular route.
- The nostrils, eyes and muzzle should be cleaned and disinfected and smeared with ointments containing, a larvicide, antihistamines and antibiotics.

- Animals showing anorexia are fed with 20 % glucose by the intravenous route. (Fowler , 2002)**.**

Prevention and control:

1) Isolation and supportive treatment of infected animals.
2) Restriction of camel movement to and from infected areas.
3) There is no available vaccine to protect camels from Orf. (Fowler , 2002) .

Camel papillomatosis

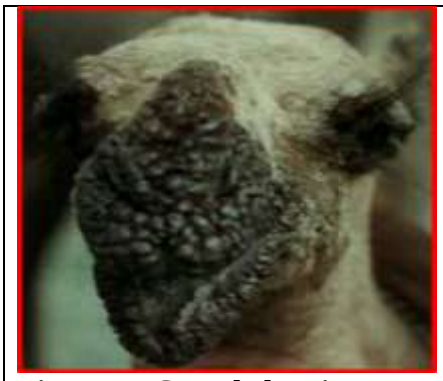

Figure 4: Camel showing sever e papillomatosis involving the l ips, nostrils and submandibula r area.

Etiology:

- Camel papillomatosis is caused by species specific *Papillomavirus* of the family *Papovaviridae*. (Fowler , 2002).

Epidemiology:

- Papilloma virus infections occur worldwide in animals as well as man.
- The disease is rare in camels affecting camels of 6 months to two years of age.
- The virus is transmitted by direct contact.
- The *Papillomavirus* affects the basal cells of the epithelium causing excessive cell growth and development of typical wart lesions. (Khalafalla , 1998).

Clinical signs:

- Lesions develop on the lips, nostrils and submandibular area as multiple sessile warts, which appear as round cauliflower-like horny masses.(Khalafalla , 1998) .

Diagnosis:,

- Electron microscopy of infected tissues reveals the *Papillomavirus*.

(Fowler , 2002).

Differential diagnosis:

- Papillomatosis should be differentiated from pox and contagious pustular dermatitis. (Khalafalla et., al ,1998).

Treatment:

- Camels are treated with auto-vaccine from surgically removed warts by injecting 3-7 ml of the vaccine subcutaneously. The warts recede within 8-10 days.
- Due to the antigenic variants of the papilloma virus a specific vaccine should be developed for each individual herd. (Fowler , 2002).

Rabies

Figure 5: A rabid camel biting its fore limb to the extent of bleeding.

- Rabies is an infectious viral disease of all warm-blooded animals including man, caused by *Lyssavirus* transmitted through the bite of a dog or a wild animal, and characterized by nervous signs, paralysis, and death.

Etiology:

- Rabies is caused by *Lyssavirus* of the *Rhabdoviridae* family. (Khan , 2015).

Epidemiology:

- Rabies is widely prevalent in Africa and Asia.
- The most frequent animals for the transmission of rabies to camels are stray dogs and wild carnivores including foxes, hyenas and wolves.
- The mode of transmission is usually through a bite.
- The incubation period ranges from one to several months.
- Rabies is a fatal disease with 100 % mortality. (Fowler , 2002).

Clinical signs:

- The furious form is most common in camels, the affected animal shows behavioral changes including restlessness, aggression and viciousness, hyperexcitabilty, and irritation signs of agitation to include attacking of inanimate objects, pica, itching and scratching with self-inflicted bites to the forelimbs, hyper-salivation and muscle tremor. This excitative stage lasts for one to three days, and is followed by the paralytic stage during which period there is paralysis of throat, difficulty of swallowing, signs of colic, sternal recumbence, and paralysis of the hind legs.
- During the paralytic phase which can last one or two days before death, the camel attempts to yawn continuously, which is a typical clinical sign of rabies in the dromedary. (Fowler , 2002) .

Diagnosis:

- History of bite.
- Clinical signs.
- Presence of eosinophilic intracytoplasmic inclusion bodies (**Negri bodies)** in brain cells is considered diagnostic.
- Laboratory confirmation by fluorescent antibody in brain smears and enzyme linked immunosorbent assay. (Khan , 2015) .

Treatment and control

- There is no treatment for rabid animals, and vaccination against rabies is the safest and surest protection against the disease.
- A dose of 1 ml of inactivated rabies vaccine induces good but short term protection of camels, and a booster dose of vaccine every 6-8 months after primary vaccination guarantees sufficient protection against rabies.
- To control rabies, mass vaccination of all stray dogs and immediate elimination of any camel suspected of being rabid is necessary. (Fowler , 2002) .

Rift Valley Fever (RVF)

Etiology:

- Rift valley fever virus is a member of the genus *Phlebovirus*.
- It is a peracute or acute zoonotic disease of domestic livestock and wild ruminants characterized by abortion and death among young animals . (Fowler , 2002).

Epidemiology and geographic distribution:

- Rift valley fever virus infection had been reported in Kenya, Maurtania, Senegal, Sudan, Egypt, Congo, Zambia, Mozambique, Madagascar, Saudi Arabia and Yemen.
- The virus is transmitted by infected mosquitoes of the genera and species:

Culex zombaensis, Mansonia Africana, Aedes quasitunivittatus and *Aedes albopctus.(Farendeze and White , 2011).*

Clinical signs and diagnosis:

- In adult camels the disease is usually subclinical, but there is significant increase in abortion rates and mortality, associated with epizootics of Rift Valley Fever.
- A seroepidemiological survey was conducted in the Sudan to determine the prevalence of RVFV antibodies and to identify the potential risk factors associated with RVFV.
- Seropositivity among the Sudanese One-humped camel was conducted in Khartoum State. A cross sectional study was conducted in 240 randomly selected camels from four localities. Sera sampled were tested for the presence of RVFV-specific immunoglobulin G (IgG) antibodies using a competitive enzyme-linked immunosorbent assay (c-ELISA). Rift valley fever virus seropositivity was recorded in 23 out of 240 camels, prevalence rate of 9.6 %.
- Age and heavy rainfall were recorded as potential risk factors for contracting RVF. (Fowler , 2002) .

Prevention and control:

- Infected animals must be isolated.
- Camels in endemic areas should be separated from sheep and cattle.
- Since mosquitoes play a significant role in the spread of infection, application of residual insecticides to animals and their pens and barns will decrease the number of new cases.
- All susceptible animals in epidemic areas must be vaccinated using killed or attenuated virus vaccines. However, pregnant animals may abort following vaccination with the attenuated live virus vaccine.(Farendeze and White , 2011)**.**

Parainfluenza -3 (PI-3)

Etiology:

- Parainfluenza is caused by *parainfluenza* virus 3. (Fowler , 2004).

Transmission:

- Transmission of *Parainfluenza* virus 3 is by contact with infected camels (Fowler , 2002).

Clinical Signs:

- The disease was reported in two herds of racing camels. In the first herd there was rhinitis with clear nasal discharge becoming mucopurulent within two to three days, while in the second herd, there were signs of

laryngitis and tracheitis, which is more debilitating and highly contagious. (Al.Ani , 2004).

Diagnosis:

- Elisa, direct immunofluorescent test, and PCR. (Fowler , 2002) .

Treatment:

- Antibiotics and rest for infected race camels. (Al.AnI , 2004).

Neonatal calf diarrhea

Figure 6: Camel calf with neonatal diarrhea showing hind quarters and tail soiled with dry feces.

- Diarrhea is considered as the most serious problem affecting camel neonates because of the serious economic losses to camel owners and animal resources sector.
- The serious complications that may occur resulting from diarrhea comprise; toxemia, dehydration, academia, loss of essential electrolytes, and ultimate death of a large percentage (20-40 %) of camel neonates.

Etiology:

- Neonatal camel diarrhea is usually caused by mixed infection and in most of the cases more than one infectious agent is recognized and are as follows:

1) *Rotavirus Group A* belonging to the family *Reoviridae* and *Torovirus* belonging to the family *Coronoviridae*.

2) Infection by one or both viruses is usually accompanied by bacterial infection namely *Escherichia coli*- enterotoxigenic K 99, *Salmonella spp.*, *Shigella spp.*, and *Clostridium perfringens*. (Fowler , 2002) **.**

Occurrence and transmission:

- The morbidity rate of neonatal calf diarrhea is (83 %) and the mortality rate is (20-40 %) in many countries where camel husbandry is practiced.
- The disease affects calves soon after birth to the age of three months.
- Young camels less than one year of age may become infected with *Coccidia* of the Genus *Eimeria*.
- Transmission is by ingestion from contaminated udder of the dam soiled with mud and dirt, and the stress of cold weather exacerbate the disease. (Celement et .,al , 1995).

Clinical signs:

- Profuse watery diarrhea when the causative agent is viral, and has an offensive odor when the infectious agent is bacterial.
- The diarrhea ranges from white to dark in color soiling the hindquarters and tail of the neonate.
- The appetite of the neonate calf for suckling its dam is decreased or is completely absent when the condition becomes complicated.
- Initially there is slight elevation of body temperature.
- The continuous profuse diarrhea and anorexia, result in dehydration lethargy and weakness, sunken eyes, loss of skin elasticity, emaciation, occasional blindness, profuse lacrimation, interrupted moaning, arching of the back, quietness, inclination for isolation and recumbence with normal or subnormal body temperature.
- The disease is very severe and in most of the cases the diarrhea is bloody resulting in pallor of the mucous membranes.
- If the diarrhea, dehydration and academia are not promptly corrected, the neonate will suffer from haemoconcentration, peripheral circulatory failure and shock, and the neonate become comatose, recumbent, with cold extremities, weak heart sounds with increased rate, slow but deep shallow respiration indicating ischemia of the brain and elevation of blood urea level as the result of decreased perfusion of the kidneys. (Mylrea , 1966).

Diagnosis:

1) Examination of feces under the ordinary and electron microscope.
2) Complete CBC and serum profile.
3) The virus is differentiated by its characteristic shape resembling wheels.
4) Isolation of the virus in tissue culture.
5) Isolation of the bacteria in suitable culture media.
6) ELISA, Latex agglutination and polyacrylamide gel electrophoresis, Toxin neutralization test, are all suitable serological tests for diagnosis of the disease. (paton and paton , 1998)**.**

Treatment:

- The hair coat of neonates is dried off using a towel to reduce heat loss.
- The neonates are treated with antibiotics and the diarrhea is controlled by the oral administration of suitable astringents.
- In case of dehydration the neonate is treated orally using feeding bottles fitted with large teats containing 2-5 % solution of sodium chloride, electrolytes, dextrose, glycine, sodium bicarbonate dissolved in water. The neonate is also given milk either by suckling its dam or using feeding bottles. (Fowler , 2002).

Prevention and control:

1) One week before term; pregnant she camels should be transferred to a warm, capacious well ventilated and disinfected parturition enclosure with floor covered with a thick layer of fresh and clean sand.

2) Immediately after delivery the udder of the she camel should be washed and disinfected, and this process is repeated every time before the newborn calf suckles its dam until making sure it received enough colostrum.

3) All feeding utensils used for the neonates should washed and disinfected.

4) Pregnant she camels should be vaccinated 1-3 months before term and another dose immediately after parturition using Coroniffa RC vaccine.

5) Sick newborn calves should be treated as described above. (Fowler , 2002) **.**

Peste des Petits ruminant

Etiology:

- *Morbilivirus* is the causative agent of Peste Des Petits Ruminant in the dromedary. (Fowler , 2002).

Transmission:

- The disease is transmitted by direct contact.(Domench et,al...2016)

Clinical signs:

- Colic and difficult respiration.
- Yellow diarrhea which becomes bloody and pregnant animals abort.
- Sudden death in 80 % of pregnant or recently delivered she camels. (Khalafalla et,al , 2010) .

Diagnosis:

- Elisa and PCR. (Domench et.,al ,2016).

Treatment:

- No specific treatment.
- Astringents to stop diarrhea, fluid replacement therapy to prevent

dehydration, and antibiotics to combat secondary bacterial infection. (Fowler , 2002).

Rinderpest (cattle plaque) – Reported in llamas but not confirmed in dr omedaries which are seropositive

Etiology:

- Rinderpest in llamas is caused by *Morbilivirus*. *(Constable et .,al , 2017)* .

Transmission:

- Transmission is by direct contact via aerosol droplets, shed in urine, feces and milk. (Murcia and Palmaini , 1976) .

Clinical signs:

- The incubation period of the disease 3-5 days.
- Elevation of body temperature, anorexia, labored respiration, and decreased milk yield.
- Congestion of the mucous membrane of the conjunctivae, oral, and nasal cavities.
- In severe infections there is erosion and ulceration of the epithelial lining of the entire oral cavity, drooling of saliva, severe bloody diarrhea, prostration caused by involvement of gastrointestinal tract, final drop in body temperature, and finally the animal dies from dehydration and shock. (Fowler , 2002).

Diagnosis:

- Clinical signs, gross and histopathological changes. (Murcia and Palmaini , 1976).

Treatment:

- No specific treatment.
- Astringents to stop diarrhea, fluid replacement therapy to prevent dehydration, and antibiotics to combat secondary bacterial infection. (Fowler , 2002) .

Infectious bovine rhinotrichitis- Reported in llamas but not confirmed i n dromedaries which are seropositive

Etiology:

- *Bovine herpesvirus* -1 is the causative agent of infectious bovine rhinotrichitis in llamas. (Fowler , 2002).

Transmission:

- Cattle may be an important source of infection to llamas when they graze together.
- Transmission is by direct contact through respiratory or reproductive routes. (Dermott et .,al ,1997).

Clinical signs:

- Four clinical forms of the disease have been recognized: The *respiratory* and *the genital* forms in adult animals, and the *enteric* and *encephalitic* forms in calves.

Respiratory form

- After an incubation period of 5-14 days, there is fever, nasal and ocular discharge and red swollen conjunctivae, drop in milk yield, mouth breathing and salvation.
- Hyperemia of the nasal mucosa, necrotic areas on nasal septum, secondary bronchopneumonia, and abortion.

Genital form

- Frequent urination, tail elevation, edema of the vulva and puerile or reddened vaginal mucosa, and mucoid or purulent exudate in the vagina.

Enteric form

- Severe oral and stomach areas of necrosis in new born animals with high mortality rates.

Encephalitic form

- Depression, excitement, and high mortality rates. (Fowler , 2002).

Diagnosis:

- ELISA and PCR. (Intisar , 2009).

Treatment:

- Antiviral therapeutic agents have been used in human *herpesvirus* infection, but these agents have not been administered to llamas. (Fowler , 2002).

Akabane disease

Etiology:

- Akabane disease is caused by *Bunyavirus.* (Khan , 2015).

Transmission:

- A mosquito and culicoides-borne virus. (Al.Ani , 2004).

Epidemiology:

- The disease is endemic in animals in the Middle East countries, Turkey, Australia, Japan, and Kenya. (Khan , 2015).

Clinical signs:

- Abortion of pregnant animals and/ or congenital anomalies such as arthrogrypsis, hydroencephaly and microencephaly of the newborn animal. (Al.Ani , 2004).

Diagnosis:

- Clinical signs.
- Neutralizing antibodies were detected in a high percentage of camels in many countries. (Al.Ani , 2004).

Treatment:

- No specific treatment. (Khan , 2015).

Bibliography

Al-Ani, F.K. (2004). Camel Mangement and Diseases. 1st Edition, Dar Am mar Book Publisher, Amman, Jordan.

Barani.S.M, Mohebbi.M.R, Varshovi.H.R, Naslaji.A.N. (2015). Journal of a griculture sscience and technology. PP 345-351.

Khan ,C.M. (2015) . Merck veterinary manual 9th edition , India .

Edward,S .R .Newman ,H. and White, H. (1996). The virulence of British is olated BHV ,1 in relationship to viral genotype British Journal .,147 ,

Khalafalla, A.L. Ibrahim, M.E., Sabagh, L., Al-busada, K.A. and AL-Mubar ak, A.I. (2015). Virology Journal, 12: 124.

Kritz B .(1982) A study of camel pox in somilia Jouranl of Comparative Pat hology 92 ,1-8.

Mombeni, E.H, Mombeni, M.G., Varshovi, H.R., Khalaj, M., Keuarkohi, M . and Gondarzi, M. (2013). Revue de Elevage Et Medicine Veterinaire des p ays tropicux. 66: 113- 115.

Fowler, M. E. (2010). *Medicine and Surgery of Camelids*. 3rd Edition, Wiley Blackwell publication, Ames, Iowa, USA.

Mylrea, P. (1966) . Digestion in young calves fed whole milk ad libitium an d it's relationship to calf scour. Res vet act 7:407-416.

Maclachlan, N. and Dubovi E.J. (2011).Fenner's Veterinary Virology ,5TH e dition , PP 330-340 .

Chapter 2

Bacterial Diseases of the One-humped camel (*Cemelus dromederius*)

Botulism

- *Clostridium botulinum* is cosmopolitan in distribution.
- One or more of the types (A to G) is probably toxic to all vertebrates.
- No cases have been reported in the new world camels (NWCs), but the disease has been reported in a group of dromedaries in the old world camels (OWCs) in Chad (Fowler, 1998).

Etiology:

- *Clostridium botulinum* is a straight Gram-positive rod with subterminal spores at a pH near or above neutrality (Wernery and Kaaden, 2002).
- The spores are resistant to heat and are only killed at 121°C for 15 minutes, while the toxins of *C. botulinum* are destroyed at 100 °C for 15 minutes.
- *Clostridium botulinum* is a strict anaerobe and even small traces of oxygen inhibits its growth.
- Eight different neurotoxins are produced by this organism.

Clinical signs:

- Similar clinical signs have been observed in camels and in most other animals and are as follows (Fowler, 1998):

1. Initially, there is incoordination, muscle weakness, and recumbence.
2. Progressive flaccid paralysis of all skeletal muscles including the respiratory muscles.
3. Body temperature is not elevated.
4. Dilatation of the pupils.
5. Cyanotic mucous membranes.
6. Decreased salivation.

Diagnosis:

- *Clostridium botulinum* may be cultured from the normal digestive tract making its isolation from affected animals of no diagnostic significance.
- A definitive diagnosis can only be made by the injection of filtrates of suspected feed materials or gut contents into mice or guinea pigs. Control animals are given simultaneous injections of protective doses of specific antitoxin (Fowler, 1998).

Treatment:

- There is no specific treatment for diseased animals suffering from botulism, apart from the intravenous administration of hyperimmune

serum specific to the toxin type involved (Wernery and Kaaden, 2002).

- As the type of *C. botulinum* responsible for the disease in animals is generally not known until sometime has relapsed, it is possible to mix antisera before administration.
- Old world camels (*Camelus dromedarius*) should receive 5 ml of each type of antiserum and repeated within 24 hours (Wernery and Kaaden, 2002).
- Good nursing is essential when treating camelids suffering from botulism.

Anthrax

Figure 7 a: A camel infected with anthrax showing blood oozing from the nostrils.

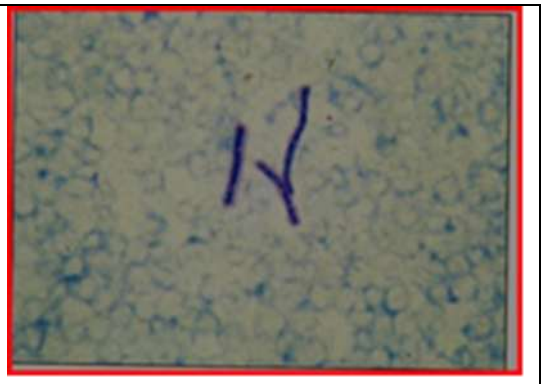

Figure 7 b: Rectangular rods of *Bacillus anthracis* in a blood smear from the ear vein of a dead camel infected with anthrax – McFadyean's stain.

- Anthrax is an acute, septicemic disease, which can affect camelids. (Wernery and Kaaden, 1995; Fowler, 1998).

Etiology:

- *Bacillus anthraces* is an aerobic sporulating bacterium, which is a Gram-positive, non-motile, cylindrical rod.
- *Bacillus anthraces* spores develop only in the presence of oxygen at temperatures above 12°C. It grows on ordinary solid media and no haemolysis is produced on blood agar (Wernery and Kaaden, 1995; Fowler, 1998).
- Under low magnification the colonies give the appearance of a Medusa-like head or a woman's curly hair.
- In the infected animal it forms a capsule, which can be demonstrated by special stains. In organ smears the bacilli lie either singly or in short chains forming the so-called bamboo- stick form.

Clinical signs:

- The first clinical sign of anthrax is fever (42 ° C, 108 ° F), but this may be missed (Fowler, 1998).
- The general signs of total anorexia, stomach stasis, colic, haematuria, and haemorrhagic diarrhoea may be more evident.
- Sudden death may occur without premonitory signs being observed.
- If the animal survives for twenty-four hours, subcutaneous swellings may be seen on various parts of the body.
- Haemorrhagic discharges may exude from all body openings.
- Dyspnoea indicates pulmonary involvement.
- The animal becomes severely depressed, convulsive, or comatose and may die in one to three days (Fowler, 1998).

Diagnosis:

- Anthrax may be confused with other diseases that cause sudden death or produce septicaemia (Fowler, 1998).
- *Bacillus anthraces* is easily cultured from the tissues of the carcass.
- In case anthrax is suspected, complete necropsy should be avoided to prevent further contamination of the soil with the organism.
- A small quantity of blood is sufficient for the laboratory to make a direct smear or culture.
- A fluorescent antibody test is also available (Fowler, 1998).

Treatment:

- If a rapid diagnosis is made, *B. anthraces* is susceptible to many antibacterial agents, including penicillin and tetracyclines.
- Therapy should be continued for five or more days, depending on the response (Fowler, 1998).

Tuberculosis

Figure 8: Extensive areas of casea ted pus caused by tuberculosis in volving the udder of a she camel.

- Tuberculosis (Tb) is a chronic, contagious, granulomatous disease (Wernery and Kinne, 2012).

Etiology:

- *Mycobacterium tuberculosis* is the causative agent of tuberculosis. It is non-motile and non-sporing acid-fast rods of different lengths (Wernery and Kinne, 2012).

Clinical signs:

- Tuberculosis is a debilitating disease, so weight loss and emaciation are typical.
- Diarrhea and dyspnoea may accompany lesions in the respective organ systems. (Fowler, 1998).
- Enlargement of superficial lymph nodes, recumbence and death.
- Sometimes the disease is not observed in animals with severe lung lesions, and animals are occasionally found dead without showing clinical signs (Wernery and Kinne, 2012).

Diagnosis:

- A definitive diagnosis can be made only at post-mortem examination by demonstration of typical gross lesions, followed by histopathology and confirmatory bacterial culture. More rapid diagnosis can be made using polymerase chain reaction (PCR) assays (Wernery and Kinne, 2012).
- Intradermal tuberculin testing, which is the classical diagnostic test, often gives non-specific reactions in camelids (Wernery and Kaaden, 2002).

Treatment:

- Valuable zoo artiodactylous if treated with isoniazid (5 to 10 mg/kg/day) controls infection and supresses tuberculin reaction, but if treatment is discontinued, the tuberculin response may reappear because the infection is still present (Fowler, 1998).

Paratuberculosis (Johne's disease)

Figure 9: Severe inflammation o f the intestines in a camel infecte d with paratuberculosis-(Johne' s disease).

Etiology:

- Johne's disease (paratuberculosis) is a chronic, granulomatous disease

caused by an acid-fast bacterium (*Mycobacterium avium paratuberculosis*), which is a ubiquitous organism throughout the world in temperate, subtropical, and tropical countries (Fowler, 1998).

Clinical signs:

- Paratuberculosis is a disease characterized by a delayed onset, and variable expression of signs.
- In adult cattle the disease is characterized by chronic diarrhea and weight loss.
- In camelids, sheep and deer, chronic weight loss is the primary sign, with diarrhea appearing in the terminal stage of the disease.
- The major clinical signs of the camel paratuberculosis are greatly similar to that of the paratuberculosis in cattle.
- The main manifestations of the disease in camels at the clinical stage are intermittent diarrhea, reduced milk yield, dehydration, emaciation and intermandibular edema (Ahmed Al-luwaimi, 2015).
- Clinically affected animals are usually afebrile and have a good appetite until the terminal stages of the disease.
- The diarrhea is devoid of blood and fibrin, and usually has no special odour (Fowler, 1998).

Diagnosis:

- Paratuberculosis can be diagnosed by culture, allergic and serological tests.
- Bacteriological culturing of the feces is the most sensitive and specific test for *M. avium* spp. *Paratuberculosis*, but may require up to 16 weeks to obtain the results.
- Biopsy specimens of intestinal mucosa and faecal smears stained by the Zeal Nielsen stain yields characteristic clumps of *M. avium* spp. *paratuberculosis* organisms. However, examination of feces will detect only about 25 % of subclinical execrators.
- Intradermal testing with avian tuberculin or "Johnin" produced from M. *avium* spp. *paratuberculosis* gives unsatisfactory results. (Wernery and Kaaden, 2002).

Treatment:

- No treatment is effective in reversing a clinical case or in clearing a carrier animal.
- Once a diagnosis of Johne’s disease in a camel is confirmed, the animal should be euthanized to avoid continued contamination of the environment (Fowler, 1998).

Pseudotuberculosis (Caseous lymphadenitis)

Figure 10 a: Pseudotuberculosis abscess at the base of the neck and anterior to the left shoulder of a camel.

Figure 10 b: Pulmonary cavern caused by pseudotuberculosis in a camel.

- Caseous lymphadenitis is a chronic and subclinical disease (Wernery and Kaaden, 2002).

Etiology:

- Cor*ynebacterium pseudotuberculosis* is the causative agent of pseudotuberculosis. It is a short, irregular ovoid, Gram-positive rod almost resembling a coccus.
- In smears made from abscesses, the bacterium shows a marked pleomorphism (Wernery and Kaaden, 2002).

Clinical signs:

- The superficial form of caseous lymphadenitis is characterized by infection and abscess formation of external lymph nodes of the submandibular, parotid, pre-scapular, subiliac, popliteal and supramammary lymph nodes, while the visceral form is characterized by abscessation of the lungs, liver, kidneys, uterus, spleen and the mediastinal and bronchial lymph nodes (Saeed and Alharbi, 2014).

Diagnosis:

- Histological examination reveals caseous necrosis of the lymph nodes with a lymphoid and epithelioid reaction, but giant cells were not observed (Nashed and Mahmoud, 1987; Wernery and Kaaden, 2002).

Treatment:

- A combination of penicillin and erythromycin are used for the treatment of pseudotuberculosis in camels.
- The intravenous injection of dimethyl sulfoxide (DMSO) and 20 mL Baytril for 12 days are also used for the treatment of pseudotuberculosis in camels (Wernery and Kaaden, 2002).

Tetanus

- Tetanus in camelids is rare and the degree of susceptibility of the OWC and NWC camelids is unknown.
- Since external wounds are very common and antibodies to tetanus have been detected in dromedaries with no disease, it may be concluded that camelids are resistant to tetanus (Wernery and Kaaden, 2002).

Etiology:

- *Clostridium tetani* is the causative agent of *tetanus*.
- The disease only develops when deep wounds with devitalized tissue are contaminated with soil or feces containing *C. tetani* spores (Fowler, 2002).

Clinical signs:

- The disease in camels is manifested by typical jaw spasms, stiff neck, rigid tail and stiff gait developing within 14 days after exposure.
- Other signs include dyspnoea, erected ears and fixed staring (Wernery and Kaaden, 2002).

Treatment:

- The affected camel should be kept in a quiet, dark place.
- Tetanus antitoxin is injected at the dose rate of 3000 IU units under the skin.
- Procaine penicillin is injected intramuscularly for at least 7 days.
- Propionylpromazine (Combelen) is injected to sedate the animal.
- Methocarbamol (Robaxin) is injected subcutaneously to relax the muscles and to calm the animal.
- The infected wound is opened to expose it to the air (*Clostridium tetani* is anaerobic), the necrotic tissue is curetted, the fluid is drained, and the wound is dressed with an antiseptic such as potassium permanganate.
- If the animal cannot eat, feed with milk, oatmeal gruel and linseed gruel by working it into its mouth from the side (Köhler-Rollefson, Mundy and Mathias, 2001).

Pasteurellosis

- *Pasteurella multocida* is a normal respiratory flora of camels and other animals, but it becomes pathogenic and causes the disease when the resistance of the camel is lowered by harmful environmental influences such as sudden changes in weather, transportation for long distances, deficiencies of dietary nutrition and heavy parasitic infestation as trypanosomiasis (El-Hofy, 2017).

Etiology:

- The species of concern in camelids are *Pasteurella multicida* in OWCs and *Mannheimia haemolytica* in NWCs.
- These organisms are small Gram negative rods or coccobacilli, non-spore forming and facultative anaerobes. They produce several proteinaceous exotoxins that are important in the pathogenesis of the disease (Fowler, 2002).

Clinical signs:

- The first sign of the disease is fever over 40 °C.
- Swollen, painful lymph nodes, especially in the angle of the jaw and at the bottom of the neck.
- The camel does not chew or feed and may grind its teeth.
- The mucous membranes of the eyes and mouth are dark red.
- Frequently the affected camel shows signs of colic, weak cough, rapid shallow breathing, salivation and rapid pulse.
- The feces is blood-stained or tarry in color, and the urine is coffee or chocolate-like in color.
- Pregnant females abort.
- The disease terminates in death of the affected camel within 2–8 days after the first clinical signs appear (Köhler-Rollefson, Mundy and Evelyn Mathias, 2001).

Diagnosis:

- Proper diagnosis can only be made on the epidemiology, clinical signs, pathology and isolation of *Pusteurella* organisms from blood, liver, spleen, kidney and lymph nodes.
- Specimens of the bone marrow are useful diagnostic aid in cases that have been dead for some time (Wernery and Kaaden, 2002).

Treatment:

- In the early stages of the disease the affected camel can successfully be treated with amoxycillin, tetracyclines or sulphonamides.
- Sulphadimidine given orally at the dose rate of 110 mg/kg body weight for 4 successive days produces good recovery (Köhler-Rollefson, Mundy and Mathias, 2001).

Brucellosis

Figure 11: A camel infected with *Brucellosis* causing severe swelling and enlargement of the right testis.

- Brucellosis is a contagious disease caused by the bacteria of the genus *Brucella*. *Brucella* bacteria are Gram-negative coccobacilli, which are non-motile and non-spore-forming (Wernery and Kaaden, 2002).
- Brucellosis is a highly infectious zoonotic disease.
- Brucellosis is the most important worldwide distributed zoonotic bacterial disease.
- The disease causes serious economic losses due to marked decrease in milk production, abortions, retention of the placenta, and public health hazard on being zoonotic.

Etiology:

- The disease in dromedaries is mainly caused by *Brucella melitensis*, but they are susceptible to *B. abortus*.

Epidemiology and transmission:

- Brucellosis occur by direct and indirect transmission.
- The factors that predispose to infection are:

1) Animals are more prone to infection during the breeding season.
2) Adult animals are more prone to infection than young ones.
3) Susceptibility to infection increases during pregnancy and lactation.
4) The percentage of infection in camels reared in farms and enclosures is higher than in camels raised under entire grazing systems.

Clinical signs:

- Non pregnant dromedaries experimentally infected with a field strain of *B. abortus* developed only mild, transient clinical signs including reduced appetite, slight lameness and bilateral lacrimation (Abu Damir et al., 1989).

- Orchitis and epididymitis have also been associated with brucellosis caused by *B. abortus* and *B. meletensis* (Tibary et al., 2006).
- Other conditions caused by the disease were retention of placenta, placentitis, uterine infections, fetal death and mummification, delayed maturity and infertility, and arthritis and hygroma in male animals (Ramadan et al., 1998; Tibary et al., 2006; Ahmad and Nemat, 2007; Musa et al., 2008).

Diagnosis:

- History, epidemiological information, clinical signs especially of abortions, and post-mortem picture of the fetus.
- Culture of the organism from milk, semen, vaginal exudates, uterine exudates covering epidermal membrane of the aborted fetus, aborted fetuses and fetal exudates. *Brucella* organisms can be recovered from the placenta, but more conveniently in pure culture from the stomach and lungs of aborted foetuses (Wernery and Kaaden, 2002).
- Rose Bengal test (RBT), and milk ring test (MRT) are useful for rapid screening but are not specific, and need to be confirmed by other serological tests such as:
- Standard tube agglutination test (SAT).
- Complement fixation test (CFT).
- Enzyme linked immunosorbent assay (ELISA).
- Polymerase chain reaction (PCR) is very specific and enables the identification the species of *Brucella*.

Treatment:

- *Brucella melitensis* is sensitive to many broad - spectrum antibiotics.
- Camelids with positively diagnosed brucellosis should be treated for two to three weeks to eliminate development of the carrier state in the female.
- Regulations in some countries that are certified brucellosis free may require euthanasia of positive reactors (Fowler, 2002).
- Treatment of brucellosis in dromedaries is prohibited because of latency, and thus each case serves as a potential source of infection to other camels and humans.

Blackleg (black quarter)

- The OWC and NWC are more resistant to blackleg infections than bovines (Wernery and Kaaden, 2002).

Etiology:

- *Clostridium chauvoei* is the causative agent of black leg (Al- Ani, 2004).

Occurrence:

- Blackleg occurs in young camels on pasture in Africa and India.

Clinical signs:

- The disease is characterized by high fever, lack of appetite, depression, lameness, and swellings that appear on the shoulders and hindquarters.
- Affected muscles produce a characteristic emphysematous crackling sound on palpation due to the presence of gas under the skin, which is produced following muscle necrosis by the growing bacteria.
- The skin over the swelling is usually normal, but in the centre of the muscles, it may undergo dry gangrene, and when the muscle is incised, it is usually swollen, contain gas and black in color.
- The animal also shows dyspnoea and grunting (Al-Ani, 2004).
- Death occurs within a few days (Köhler-Rollefson, Mundy and Mathias **(**2001; Al-Ani, 2004).

Diagnosis:

- Gram stained impression smears of affected tissue reveals Gram positive, rod bacilli (Fowler, 2002; Al-Ani, 2004).
- Positive fluorescent antibody test (Fowler, 2002). Commercial fluorescein-labelled antisera are available for *Clostridium chauvoei*, *C. septicum*, *C. novyi*, and *C. sordellii* (Al-Ani, 2004).

Treatment:

- A high dose of procaine penicillin is recommended by intramuscular route for 5-7 days (Al-Ani, 2004).
- A commercial toxoid vaccine containing *Clostridium chauvoei* alone or in combination with other clostridia species is available; it induces effective protection (Al-Ani, 2004).

Mastitis

- Mastitis is not common in camels, but when it does occur, prompt attention is necessary to avoid loss of function in one or more quarters or even death.
- Camelid mastitis occurs in the same forms as seen in dairy cattle, namely, subclinical, peracute, acute, and chronic (Fowler, 2002).

Etiology:

- Mastitis is caused by a wide variety of bacteria namely: *Staphylococcus aureus*, *Bacillus*, *Corynebacterium bovis*, *Streptococcus agalactiae*, *Escherichia coliuberis* and *Pasteurella haemolytica*.

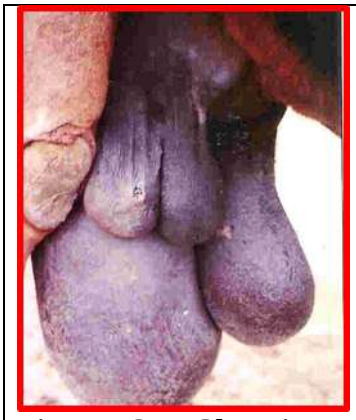

Figure 12 a: Chronic mastitis causing severe enlargement and deformity of the left fore and hind

quarters of the udder of a she camel.

Figure 12 b: Gangrenous mastitis causing severe enlargement, deformity and ulceration of the left fore and hind quarters of the udder of a she camel.

Epidemiology:

- The above mentioned bacteria enter the udder through the teat.
- Animals may become infected if the animal enclosure is dirty.
- Inexperienced persons milking camels using wrong milking techniques.
- People milk camels with dirty hands.
- The udder or teats when injured cause obstruction of the teat canal making milking impossible.
- Tick wounds on the udder (*Amblyomma* ticks) predispose to mastitis.
- Wrong practice of tying the teats with string or with dirt to prevent the calf from suckling.
- Mastitis cannot be transmitted directly from one animal to another, but it can be spread when healthy animals are milked after milking a mastitic camel without washing the hands in between (Köhler-Rollefson, Mundy and Mathias, 2001).

Clinical signs:

- Subclinical mastitis is only detected by culturing a milk sample or by testing the milk with one of the mastitis tests such as the California Mastitis Test (CMT). Indurated areas may be palpable within a gland, but there are no systemic signs, and the gland is not swollen or hot.
- Chronic mastitis results in periodic changes in the quality of milk.
- Acute mastitis is usually seen just before or within a few days after parturition and is characterized by heat, swelling, hardness of the affected gland, and evident pain on palpation. The secretion may be watery, haemorrhagic, thickened, stringy, or odorous.
- The female may refuse to allow the calves to suckle because of the pain, and new-borns may refuse to nurse because of the unpalatable secretion.
- The calves may develop gastroenteritis or septicaemia from ingestion of the pathogen.
- The she camel may show anorexia and have a low-grade fever.
- Peracute mastitis is usually seen within a few days of parturition, and may have all of the signs of acute mastitis in addition to severe depression and toxaemia, and may even develop gangrene, septicaemia, and death (Fowler, 2002).

Diagnosis:

- Diagnosis of mastitis is based on a thorough physical examination; evaluation of the secretion for consistency, color, viscosity, presence of debris, and sediment.
- Evaluation with the CMT.
- Culture and sensitivity.
- Ultrasonography may be used to locate walled-off abscesses.

Differential diagnosis:

- Preparturient and postparturient edema occurs in camelids and must be differentiated from mastitis (Fowler, 2002).

Treatment:

- Mastitis treatment should be based on culture and sensitivity.
- The streak canals can be easily traumatized when using bovine antibiotic mastitis ointments, and the treating person must be fully aware of the anatomical particularities of the of the camelid's mammary gland.
- In severe cases, the exudate should be removed from the gland three to five times daily by gently massaging the udder.
- Peracute and sometimes acute mastitis require parenteral treatment with antibiotics along with local infusion (Wernery and Kaaden, 2002).
-

Pneumonia

- There are many different types of pneumonia in camelids, depending on the cause of the disease and the parts of the lungs infected (Köhler-Rollefson, Mundy and Mathias, 2001).

Etiology:

- The causal bacterial agents of camelid pneumonia are similar to those causing pneumonia in livestock and horses.
- Most infectious cases result from opportunistic bacteria.
- Septicaemic animals usually develop pneumonia, and the most common agent isolated is *Escherichia coli*.
- Other causes of pneumonia include inhalation of toxic vapours.
- *Actinomyces lamae* may produce abscessation of the lung.
- Aspiration of stomach contents occurs in the orphaned neonate being fed from a bottle or while being stomach tubed. Passive regurgitation during anaesthesia is a significant risk. If surgery entails prolonged left lateral or dorsal recumbence, it is advisable to intubate the trachea with a cuffed tube and to position the head and upper neck so that stomach contents can flow freely from the mouth (Fowler, 2002).

Clinical signs:

- The clinical signs are exaggerated in the neonate and include dyspnoea, coughing, elevated body temperature, variable nasal exudation, depression, and anorexia.
- Sounds heard at auscultation vary with the degree of exudation and consolidation (Fowler, 2002).

Diagnosis:

- A haemogram should be conducted. Bacterial or fungal infections, show an elevation of leukocyte count and left shift.
- Radiographic evaluation is useful.
- A trans-tracheal wash can be used to collect material for culture and sensitivity. This is performed by aseptically preparing an area over the trachea in the mid-cervical region and a 15-gauge needle is inserted between the tracheal rings. A sterile catheter is threaded through the needle, and while the neck is on a horizontal position 5 to10 ml of a non-bacteriostatic water or saline is pumped into the trachea with a pumping action of the syringe to aspirate exudate. Collected material should be examined for cytology and cultured (Fowler, 2002).

Treatment:

- Broad-spectrum antibiotic therapy in association with anti-inflammatory drugs is recommended as well as proper general nursing and supportive treatment.
- The antibiotics of choice are: Trimethoprim/ sulfadiazine, procaine penicillin G, gentamycin and oxytetracycline.
- The anti-inflammatory drugs include: Flunixin meglumine and daexamethasone (Wernery and Kaaden, 2002).

Salmonellosis

- Salmonellosis is a serious bacterial infection that affects calves over 2 weeks of age. It is the most important disease of suckling calves, causing up to 20 % mortalities among them (Köhler-Rollefson, Mundy and Mathias, 2001).

Etiology:

- Salmonellosis is caused by members of the genus *Salmonella* belonging to the family *Enterobacteriaceae*, whose members are Gram-negative coccobacilli; with the exception of *S. gallinarum pullorum*, all *Salmonellae* are motile with peritrichous flagella (Wernery and Kaaden, 2002).

Clinical signs:

- Salmonellosis causes yellowish or greenish-grey, foul-smelling diarrhoea, which often contain blood (Köhler-Rollefson, Mundy and Mathias, 2001).
- The disease often causes fever, but dehydration with sunken eyes, and dry mucous membranes are prominent clinical signs.
- In very acute cases, animals die within 24–48 hours after the clinical signs appear, however, mortalities usually occur, after 1–2 weeks.
- If the disease progresses into the septicaemia form; the body temperature rises rapidly, and the animal becomes dull, does not react to external stimuli, shows inco-ordinated movements, then becomes unable to stand, and has difficulty in breathing.
- The lungs and joints may become inflamed (Köhler-Rollefson, Mundy and Mathias, 2001).

Diagnosis:

- *Salmonellae* have simple nutrient requirements and growth *in vitro* is therefore possible on many different media. However, selective procedures are used for the isolation of *Salmonella* from specimens that contain a mixed flora.
- Colonies characteristic for *Salmonellae* can be easily serotyped.

Serotyping is based on the 0 (somatic) and H (flagellar) antigens (Wernery and Kaaden, 2002).

Treatment:

- In case salmonellosis is suspected in an animal, treatment should be started immediately. If the animal did not receive treatment within 24–48 hours, the animal is likely to die.
- Once the septicaemia form has developed, treatment is usually not successful – especially if the infection has moved into the lungs.
- The animal is treated with oxytetracycline given orally as well as through injection (Köhler-Rollefson, Mundy and Mathias, 2001).

Colibacillosis

- Colibacillosis is a diarrhoea caused by infection with *Escherichia coli* bacteria (Köhler-Rollefson, Mundy and Mathias, 2001). It usually occurs as a secondary infection following milk scour, rotavirus or coronavirus infections (*Neonatal Calf diarrhea*).

Etiology:

- *Escherichia coli*, the causative agent of colibacillosis is a ubiquitous, Gram-negative, enteric bacterium.
- The organism may be a constituent of the normal flora of the intestinal tract, but under favourable conditions, it may become pathogenic (Fowler, 2002).

Clinical signs:

- Camel calves suffer from dysentery abdominal pain, anorexia and dehydration, and death occurs within a few days (Wernery and Kaaden, 2002).
- The calf becomes weak, frequently lying, resting or sleeping.
- The affected calf shows high body temperature (above 40 °C), anorexia, and watery, yellowish diarrhea and dehydration (Köhler-Rollefson, Paul Mundy and Evelyn Mathias, 2001).

Diagnosis:

- Clinical signs and lesions are not diagnostic.
- Isolation and identification of the organism is necessary for confirmation of diagnosis.
- Colibacillosis may also be seen as a secondary infection accompanying such diseases as enterotoxaemia.
- The lesions are consistent with the organ systems involved in the infection (Fowler, 2002).

Treatment:

- Gentamycin at the dose rate of 5 mg/kg by the intramuscular route for 5 consecutive days.
- Trimethoprim at the dose rate of 12 mg/kg by the intramuscular route for 5 consecutive days.
- Chloramphenicol at the dose rate of 10 mg/kg by the intramuscular route for 7 consecutive days.
- Scourban or Diaclean or kaolin and charcoal or bismuth subnitrate and charcoal as anti-diarrheic and multi-vitamins orally.
- Glucose saline by the intravenous and subcutaneous routes.

Dermatophilosis

Figure 13 a: A young camel showing scab covered generalized lesions of dermatophilosis.

Figure 13 b: Typical "railroad tracks" of *Dermatophilus congolensis* organism in a smear from infected material. Giemsa stain.

- Dermatophilosis is a skin disease mainly of young camels, but adults are also infected. The disease is zoonotic and people can get infected if they come into contact with infected animals (Köhler-Rollefson, Mundy and Mathias, 2001).

Etiology:

- *Dermatophilus congolensis* belonging to the order *Actinomycetales* is the causative agent of dermatophilosis.
- *Dermatophilus congolensis* is distinguished by branching hyphae subdivided by transverse and longitudinal septae forming parallel rows of cocci (Wernery and Kaaden, 2002).

Clinical signs:

- Calves have high skin involvement than adult camels, while adults had a higher incidence of hair matting.
- The disease in adult camels is characterized by serous exudation and hair matting involving the hind limbs and abdomen and less frequently other

parts of the body.

- Removal of the hair mats with dried exudates forming scab, leave raw hyperemic skin covered with pus underneath.
- The lesions heal leaving areas of 1-3 cm in diameter covered with white crusts.
- The lesions of dermatophilosis in adult camels persist for ≥ six months.
- In camel calves less than one year of age the disease is more severe affecting most of the skin especially the head, neck, chest, flank, and upper fore and hind limbs.
- Infected camel calves developed alopecia, and yellowish brown crusts on the affected areas.
- The infected calves show enlarged regional lymph nodes, anorexia and their general health condition deteriorates resulting in emaciation terminating in death in advanced cases.

Diagnosis:

- *Dermatophilus congolensis* is cultured and grows well on sheep and ox blood agar and in brain-heart infusion agar. The plates should be incubated at 37 °C under aerobic and increased CO_2 tension.
- Gram-stained smears of scab material show Gram-positive hyphae divided transversely and longitudinally forming parallel chains of cocci typical of "railroad tracks" (Abu-Samra, 1978).
- ELISA is used for the detection of antibodies against dermatophilosis in camels (Wernery and Kaaden, 2002).

Treatment:

- Successful treatment of dermatophilosis with terramycin or procaine penicillin and streptomycin has been reported.
- Infected dromedaries are treated twice with LA Terramycin intravenously (Wernery and Kaaden, 2002).
- The scabs are removed and the areas cleansed daily with an iodine solution for 7 days.
- The lesions heal within 4 weeks.
- Shearing of badly affected areas with long hair is often an important additional method of further reducing the development of lesions (Wernery and Kaaden, 2002).

Bibliography

Abu-Samra, M.T. (1978).*Dermatophilus* infection: the clinical disease and diagonsis. *Zentralblatt fur Veterinarmedizin*. **B 25:** 641- 651.

Ahmed M. Alluwaimi, A. M. (2015). Paratuberculosis in camel (Camelus dromidarius), 21; 83-88.

El-Hofy, F. I. (2017). *Pasteurella multocida* in camels. pp171.

Fowler, M. E. (2010). *Medicine and Surgery of Camelids*. 3rd Edition, Wiley Blackwell publication, Ames, Iowa, USA.

Gwida, M. ELGohary A,Melzer F, Khan I, Gosler U, Neubauer H (2012). Brucellosis in camel. pp 353-354.

Kohler-Rollefson, I; Mundy, P. and Mathias, E. (2001). *A Field Manual of Camel Diseases* (*Traditional and modern health care for the dromedary*).CTA, Technical Centre for Agricultural and Rural Cooperation (ACP-EU), Postbus, Wageningen, The Netherland.

Saeed, E. M. A. and Alharbi, K. B. Alharbi (2014). Morel's Disease and Caseous Lymphadenitis. pp79-80.

Wernery, U. and Kaaden, O. (2002). *Infectious Diseases of Camelids*. 2nd Revised and Enlarged Edition, Blackwell Science publication, Berlin, Germany.

Chapter 3

Parasitic Diseases of the One-humped camel (*Cemelus dromederius*)

Trypanosomiasis

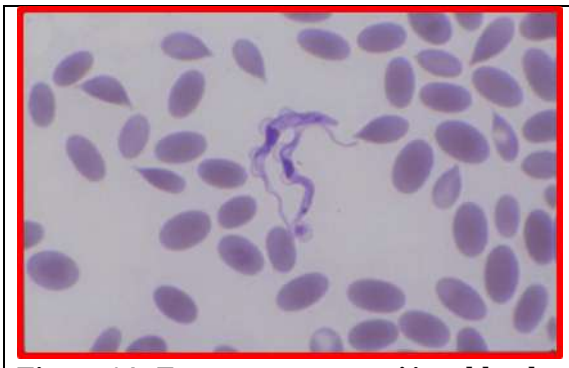

Figure 14: *Trypanosoma evansi* in a blood smear from a camel infected with trypanoso miasis.

- The trypanosomes of mammals are subdivided into: stercoraria and salivaria based on the mode of development in their insect vectors and vertebrae host (Wernery and Kaaden, 2002).

Etiology:

- *Trypanosoma evansi* is the main causative agent of trypanosomiasis in camels. It is one of the salivarian trypanosomes. It is long slender, having prominent undulating membrane, long free flagellum and small subterminal kinetoplast (Wernery and Kaaden, 2002).

Clinical signs:

- Camels infected with trypanosomiasis show fever, shivering, lacrimation, reduced appetite, lose weight, develop drooping hump, unable to walk long distance, shows edema in the feet, brisket, and eyelids.
- The coat becomes rough, and the animal develop diarrhea, the mucus membrane become pale, and the odor of urine changes due to the presence of ketone bodies.
- Abortion occur in all stages of pregnancy, milk production is reduced and in some cases the animal may become blind and show nervous signs.
- Infected newborn calves die (Rottcher, Schillinger and Zweygarth, 1987).

Diagnosis:

- Clinical sings.
- Microscopic examination of wet, thin or thick blood films.
- Elisa, IFAT, capillary agglutination test, and passive haemgglutiation test (Elazhari Mohamed, 1992).

Treatment:

- Suramin and quianapyramine are recommended for the treatment of *T. evansi*, most of the drugs used for cattle trypansomasis are either not curative or are too toxic for camel (Rottcher, Schillinger and Zweygarth, 1987).

Tritrichomoniasis

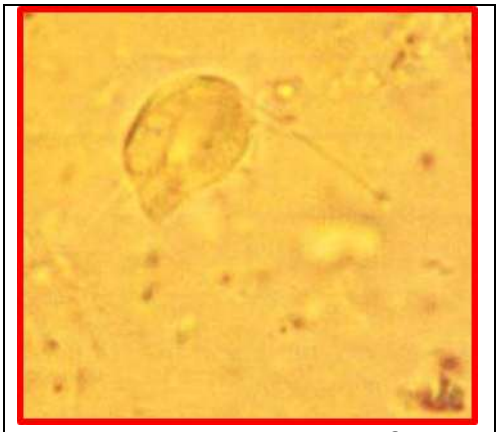

Figure 15: *Tritrichomonus foetus* i n vaginal washings of a she camel .

Etiology:

- *Tritrichomonus foetus* is the causative agent of trichomoniasis in camels. It is pear shaped, and possesses three free flagellae arising from a basal body at the anterior end, and a fourth flagella extends backwards to form undulating membrane along the side of the organism continuing as free flagellum (Wernery and Kaaden, 2002).

Clinical signs:

- *Tritrichomonus foetus* is commonly found in the digestive and reproductive systems and also found in other organs. The organism causes venereal disease characterized by early fetal death and infertility (Wernery and Kaaden, 2002).

Diagnosis:

- Presence of the parasite in fresh smears from the cervical and vaginal mucus or preputial washing, and discharge from the uterus.
- Clean samples may be cultured in special media and PCR (Wernery and Kaaden, 2002).

Treatment:

- Dimetridazole, diminazene aceturates, ipronidazole, metronidazole.
- Affected organs are rinsed with acridin and iodine preparations give positive effect (Wernery and Kaaden, 2002).

Sarcocystiosis

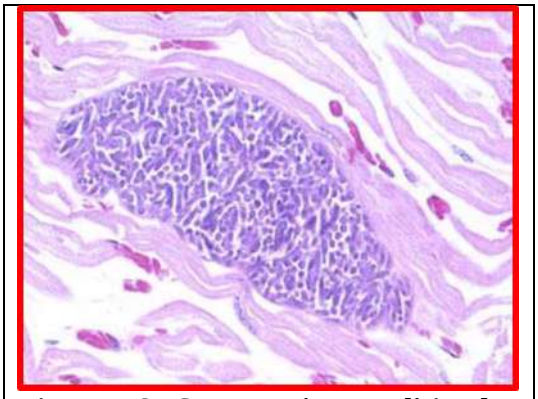

Figure 16: *Sarcocystis cameli* in the heart muscle of camel.

- Sarcocystis are parasites using two hosts to complete their life cycle. Carnivores are the final host and herbivores are intermediate hosts (Wernery and Kaaden, 2002).

Etiology:

- *Sarcocystis cameli* is the causative agent of sarcocystosis in camels. Two different cysts were recognized: One is thin-walled found in the diaphragm, heart, and esophagus, and the other is thick-walled found only in the esophagus (Wernery and Kaaden, 2002).

Clinical signs:

- Camels affected with sarcocystosis shows anorexia, lethargy, develop pyrexia, become restless and anemic.
- Post-mortem examination reveals hemorrhages in several organs and muscles, and immature cysts containing metrocytes in the brain (Dubey et al., 2015).

Diagnosis:

- Identification of sarcocysts is by the typical ultrastructure of their wall. Macroscopic cysts are seen during meat inspection or at necropsy, and microscopic cysts are often found accidentally in histological sections of muscles.
- Serological tests, fecal examination of the final host, and PCR (Wernery and Kaaden, 2002).

Treatment:

- Treatment and control are very seldom applied.
- The only way to control of sarcoystis infection is by breaking the life cycle of the parasite (Wernery and Kaaden, 2002).

Coccidiosis

- Coccidiosis of camels is an intestinal protozoan infection caused by apicomplexan parasites of genus *Eimeria* (Sazmand and Joachim, 2017).

Etiology:

- *Eimeria cameli* and *E. dromedari* are the most prevalent coccidia infecting both bactrian and dromedary camels. There are five types of *Eimeria* species found in camels and two *Isopora* species (Wernery and Kaaden, 2002).

Clinical signs:

- Camels infected with coccidiosis show loss of appetite, enteritis, diarrhea and poor weight gain (Sazmand and Joachim, 2017).

Diagnosis:

- Clinical sings, demonstration of oocysts in smears of freshly collected feces or by using the concentration technique.

Treatment:

- Sulphonamides, oxytetarcycline give good results (Elazhari Mohamed, 1992).
- Anticoccidial drugs are used to control outbreaks in livestock (Wernery and Kaaden, 2002).

Toxoplasmosis

- Toxoplasma is an intracellular protozoan parasite of man, animal and birds. It is worldwide in distribution (Elazhari Mohamed, 1992).

Etiology:

- Toxoplasma in camels is caused by *Toxoplasma gondii*, and its prevalence is much higher in females than males and in adults than young camels (Elazhari Mohamed, 1992).

Clinical signs:

- Toxoplasmosis in camels causes mild dyspnea, anorexia, abortion, and accumulation of fluids in the ventral half of pleural cavity (Elazhari Mohamed, 1992).

Diagnosis:

- Dried smears fixed in methanol and stained with Wright-Giemsa stain, ELISA, and IFAT (Elazhari Mohamed, 1992).

Treatment:

- Pyrimethamine in combination with sulphadiazine, toltrazuril (Wernery and Kaaden, 2002).

Balantidiasis

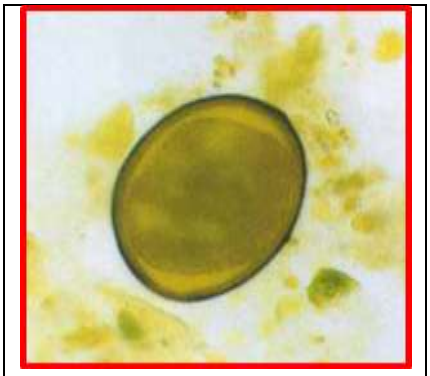

Figure 17: *Balantidium coli* t rophozoite from camel intest ine.

-
- *Balantidium* species are often seen in the lumen of the caecum and large intestine of mammals such as swine, human, and non-human primates (Sazmand and Joachim, 2017).

Etiology:

- *Balantidium coli* is the only species associated with disease in camels (Wernery and Kaaden, 2002).

Clinical sings:

- *Balantidium coli* in camels causes ulceration of the mucosa of the large intestine, severe enteritis, diarrhea, and the cysts are viable in moist feces (Wernery and Kaaden, 2002).

Diagnosis:

- Fecal examination where trophozoites are seen moving around (Al-Ani, 2004; Sazmand and Joachim, 2017).

Treatment:

- Ampicillin, trimethoprim and anti-inflammatory drugs such as flunixin meglumine (Al-Ani, 2004; Sazmand and Joachim, 2017).

Tick Borne Diseases (theileriosis and babesiosis)

- *Theileria* and *Babesia* are protozoan Genera belonging to the order *Piroplasmida* commonly transmitted by ticks (Wernery and Kaaden, 2002).

Theileriosis

Etiology:

- *Theileria* parasite as tick borne pathogen for camel is still not confirmed, but DNA of *Theileria equi*, *Theileria mutans*, *Theileria annulata*, and *Theileria ovis* has been detected in peripheral blood of dromedaries worldwide (Sazmand and Joachim, 2017). However, *Theileria camelensis* and *T. dromedarii* were diagnosed in camels, and are transmitted by the tick *Hyalomma dromedarii* (Al-Ani, 2004).

Clinical signs:

- Camels show fever, swelling of superficial lymph nodes, loss of appetite, and loss of weight, lacrimation, high morbidity, but no mortalities (Sazmand and Joachim, 2017).

Diagnosis:

- Blood smears, and PCR (Sazmand and Joachim, 2017).

Treatment:

- Buparvaquone (Sazmand and Joachim, 2017).

Babesiasis

Etiology:

- *Babesia cameli* is the only species diagnosed in camels; *Hyalomma dromedarii*, *H. marginatum* and *H. impressum* ticks are thought to be the vectors (Al-Ani, 2004).

Clinical signs:

- Camels show haemolytic anemia, hemoglobinurea, hemoglobinemia, anisocytosis, and polychromasia (Wernery and Kaaden, 2002; Al-Ani, 2004).

Diagnosis:

- Blood smears and PCR (Sazmand and Joachim, 2017).

Treatment:

- Buparvaquome (Sazmand and Joachim, 2017).

Fascioliasis

- Fascioliasis is a parasitic infection with wide distribution.
- It is an important foodborne trematode.
- The parasite requires snails as intermediate hosts, and both *Fasciola hepatica* and *F. gigantica* have snails of different genera (Sazmand and Joachim, 2017).

Etiology:

- *Fasciola hepatica* and *F. gigantica* cause fascioliasis in camels.
- The flukes live in the bile duct and the eggs are discharged into the bile duct and then to the intestine and excreted through the feces (Al-Ani, 2004, Fowler, 2010).

Clinical signs:

- Light infection is usually asymptomatic, but heavy infestation causes diarrhea, loss of weight and even death. Young worms cause massive destruction of the liver parenchyma (Al-Ani, 2004).

Diagnosis:

- History, clinical signs, seasonal occurrence, identification of snails, examination of the feces for fluke eggs, and ELISA (Wernery and Kaaden, 2002).

Treatment:

- Rafoxanide administered orally at a dose 7.5 mg/kg every two weeks 2-3 times is recommended.
- The snails are destroyed using molluscicidal drugs such as copper sulphate and copper pentachlorphenate (Al-Ani, 2004).

Haemonchosis

- *Haemonchus* is an important round worm parasite infesting the abomasum of various ruminants (Al-Ani, 2004).

Etiology:

- *Haemonchus longistipes* is the common abomasal nematode of camels. The male worm is 19- 26 mm in length and the female is 27-42 mm in length (Wernery and Kaaden, 2002). *Haemonchus placei* was also encountered in the abomasum of camels (Al-Ani, 2004).

Clinical signs:

- The disease in camels causes pica, diarrhea, loss of weight, anemia, edema, hypoproteinaemia, hypoalbuminaemia, hyperglobulinaemia, increase in the blood urea nitrogen and eosinophilla (Elazhari Mohamed, 1992).

Diagnosis:

- Clinical signs, fecal examination, larval culture, worm eggs (Elazhari Mohamed, 1992).

Treatment:

- Thiabendazole, phenothiazine (Elazhari Mohamed, 1992).

Hydatidosis

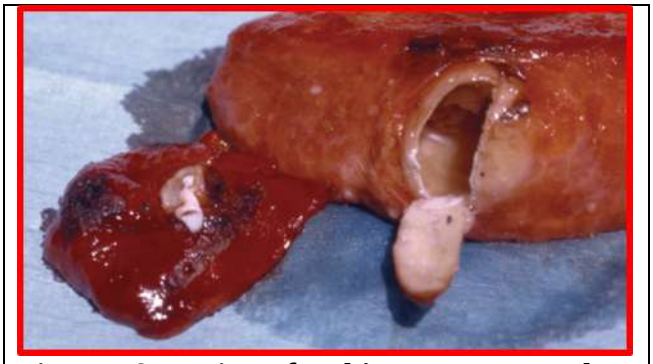

Figure 18: Lesion of *Echinococcus granulosus* in the lung of a camel.

- *Echinococcus granulosus* worm is a cestode parasite that lives in the intestine of dogs and other carnivores as final hosts.
- A number of mammals act as intermediate hosts in the developmental cycle of the parasite. Camels as well as other animals, become infected by ingesting the parasite eggs containing the embryo called onchospheres or hexacanths.
- Upon ingestion by the intermediate host, the embryos are released from their surrounding membranes by the action of the digestive juices, bore actively into the intestinal wall and enter the intestinal blood vessel.
- Cysts of various sizes develop in various camel organs especially the lungs and liver. These cysts are referred to as hydatid cysts and represent the larval stage of the parasite. *Echinococcus granulosus* worm and its cysts in the different intermediate hosts are worldwide in distribution (Al-Ani, 2004).

Etiology:

- Hydatidosis in camels is caused by *Echinococcus granulosus*, a small tape worm of 2-7 mm long found in the intestine of dogs and other carnivores. The worms have a scolex, neck, and consists of three or four

proglottids. They passes their eggs through the feces of the final host (Fowler, 2010).

Clinical signs:

- No clinical signs appear, and the cyst/cysts are seen only after the animal is slaughtered or after postmortem. Many large cysts may be found in the lung and cause respiratory distress especially in racing camels, or in the liver and interfere with liver function, or in many other organs (Al-Ani, 2004).

Diagnosis:

- Presence of hydatid cyst in slaughtered animals, fecal examination of the definitive host for eggs of *Echinococcus granulosus*, microscopic examination of the tapeworm, radiographic examination, ELISA, and PCR (Wernery and Kaaden, 2002).

Treatment:

- No treatment can destroy the hydatid cyst, and the cysts can only be removed surgically after being injected with formalin to avoid rupture and contamination of the area it occupied (Fowler, 2010).
- Prevention of the disease is by, elimination of stray dogs, safe disposal of infected slaughtered offals, and treatment of pet dogs by manosonil every 3-6 months (Al-Ani, 2004).

Coenurosis

- *Coenurus cerebralis* is found in the brain and spinal cord of herbivorous mammals including camels.
- *Coenurus cerebralis* is the intermediate stage in the life cycle of the cestode worm *Taenia multiceps* which lives in the small intestines of dogs, foxes, coyotes and jackals, and *Coenurus cerebralis* is considered as the larval stage of *Taenia multiceps*, which measures 40-100 cm in length, while *Coenurus cerebralis* cysts are 5-6 cm in diameter.
- The development of *Coenurus cerebralis* is more or less similar to hydatid cysts, but most frequently develops in the central nervous system of herbivores including camels (Wernery and Kaaden, 2002; Al-Ani, 2004).

Clinical signs:

- The clinical signs vary with the location of *Coenurus cerebralis* cyst in the brain and spinal cord.
- The signs may be behavioral abnormalities, circling, ataxia, hypermetria, blindness, deviation of the head, paralysis, convulsions, hyperexcitability,

prostration and emaciation (The Center for Food Security and Public Health, 2005).

Diagnosis:

- Only detected and confirmed at post-mortem, and at meat inspection (Wernery and Kaaden, 2002).

Treatment:

- Praziquantel, epsiprantel, mebendazole, fendezole to prevent infection of the definitive host against infection with the tape worm.

Cysticercoses

Etiology:

- *Cysticercus tenuicollus* the intermediate stage (larval stage) of the mature tape worm *Taenia hydatigena* which lives in the small intestine of the dog and fox is rarely encountered in the liver and abdominal cavity of camels.
- The mature cestode in the intestine of dogs and foxes is 70-500 cm in length.
- *Cysticercus cameli* (*dromedarii*) is another intermediate stage (larval stage) of the mature worm *Taenia hyaenae*, which infests various species of hyena is encountered in the heart, diaphragm and muscles of the camel (Al-Ani, 2004).

Clinical signs:

- The *Cysticerci* cause no harm in the intermediate host, but economic losses occur due to regulations governing meat inspection. Presence of the cysts in the liver may predispose the animal to black disease.

Diagnosis:

- Finding the proglottids or eggs in the feces of dogs, presence of the cysts at post-mortem or meat inspection of camels.

Treatment

- Prazquantel, epsiprantel, mebendazole, fendendazole to prevent infection of the definitive host against infection with the tape worm.

Monieziasis

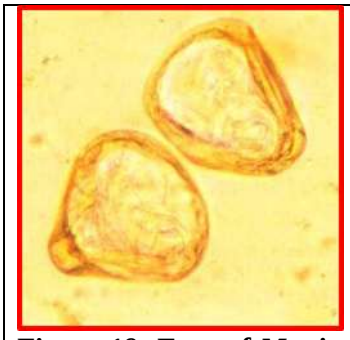

Figure 19: Eggs of *Moniez ia expansa* in camel feces.

- Monieziasis is caused by the species of the genus *Moniezia* tapeworms, which are found in ruminants worldwide (Fowler, 2010).

Etiology:

- *Moniezia expansa* and *M. benedeni* cause monieziasis in camels.
- *Moniezia* are long tapeworms reaching 6m in length.
- The tapeworm has unarmed scolex with no rostellum or hooks, but have 4 suckers, and the proglottids are broader than long, and the eggs are triangular in shape (Wernery and Kaaden, 2002).

Clinical signs:

- Heavy infection impairs nutrition and causes diarrhea, debility, and obstruction of the intestine (Wernery and Kaaden, 2002).

Diagnosis:

- Moniezia eggs found in the feces of affected camels have characteristic triangular shape (Al-Ani, 2004).

Treatment:

- Copper sulphate is the drug of choice, but various other medications have been used to treat camels and include niclosamide, dichlorophen, albendazole and cambendazole (Al-Ani, 2004).

Lungworms

- *Dictyocaulus viviparous* occur in the bronchi of camels and other ruminants (Al-Ani, 2004).

Etiology:

- The male *Dictyocaulus viviparus* is 4-5.5 cm and the female is 6-8 cm long, the life cycle is distinct (Al-Ani, 2004), and can be summarized as follows:

- The females lay larvated eggs, which are coughed up and swallowed.
- The first- stage larvae which are passed in the feces are 0.3-0.36 mm long.
- The third larval stage is the infective stage and infection occur by ingestion.
- Larvae penetrate into the intestinal wall and pass via the lymph vessels to the mesenteric lymph nodes where they develop into the fourth-stage larvae.
- The larvae continue their migration to the lungs via the thoracic duct and right heart.
- The fifth-stage larvae are produced in the lungs by the fifteenth day and sexual maturity is reached on the twenty-second day post-infection (Al-Ani, 2004).
- Other lungworms identified to infest camels are *Dictyocaulus filaria* occur in the bronchi of other ruminants but rarely in camels. The male is 3-8 cm and the female is 5-10 cm long. The worms have a milk white color and the intestine shows a dark line (Al-Ani, 2004).

Clinical signs:

- The worms live in small bronchi resulting in catarrhal parasitic bronchitis, coughing, increased respiratory rate, pulmonary edema and emphysema (Al-Ani, 2004).
- Secondary bacterial pulmonary infections may occur.
- Young animals tend to be more severely infected than older animals (Al-Ani, 2004).

Diagnosis:

- Diagnosis is based upon finding the first stage larvae in fecal samples using the Biermann apparatus, and finding a massive number of larvae in the major bronchi at necropsy (Al-Ani, 2004).

Treatment:

- Antihelmintic drugs, and artificial vaccination by using irradiated larvae has been developed for cattle and can be used to protect camels in endemic areas (Al-Ani, 2004).

Meningeal-worm

Etiology

- *Parelaphostrongylus tenuis* which occurs in the cranial venous sinuses and subdural space of the white tailed deer (Wernery and Kaaden, 2002), may infest camels (Al-Ani, 2004).
- The adult is about 39-91 mm in length. Migration of the larvae in the spinal cord cause neurological deficits (Al-Ani, 2004).

Clinical signs

- There is no clinical sings but fetal neurological disease occur in the aberrant host, migration of the larvae in the spinal cord cause neurological sings such as cephalomalacia, lameness, ataxia, stiffness, circling, blindness, hypermetria, abnormal position of the head, local hemorrhage from the spinal cord leads to death (Wernery and Kaaden, 2002; Al-Ani, 2004).

Diagnosis

- No ante-mortem diagnosis is possible, and there may or may not be increase in eosinophils in the cerebrospinal fluid (Fowler, 2010).

Treatment

- No definitive treatment or preventive program has been used.
- Ivermectin is effective against the stage prior to entering the spinal cord.
- A molluscicidal drug could be used to destroy the snails and slugs that serve as intermediate hosts (Fowler, 2010).

Nodular worm

- The nodular worm is a strongyloid nematode found in the small and large intestine of camels and other ruminant (Al-Ani, 2004; Fowler, 2010).

Etiology:

- *Oesphagostomum venulosum* is found in small and large intestine of camels and other ruminants. The male is 11-16 mm and the female is about 13-24 mm long.
- *Oesphagostomum columbianum* occurs in the colon of camels and other ruminants. The male worm is 12-16.5 mm and the female is 15-21.5mm long, and have a direct life cycle (Al-Ani, 2004).
- After ingestion of the infective larvae escheatment occurs in the small intestine, and larvae penetrate into the wall of the intestine where they coil up against the muscularis mucosa and cause cyst formation.
- A marked reaction against each larva takes place, leukocytes especially eosinophils and foreign body giant cells, collect around the parasite and the focus becomes encapsulated by fibroblasts (Al-Ani, 2004).

Clinical signs:

- Severe enteritis, nodule formation, and inflammation in the wall of the intestine (Wernery and Kaaden, 2002).

Diagnosis:

- Examination of the feces by Biermann's technique, and ELISA (Wernery and Kaaden, 2002).

Treatment:

- Ivermectin, Thiabendazole, tetramisol, oxfendazole, morantel give good results (Al-Ani, 2004).

Eye worm

Figure 20: *Thelazia* worms in the conjunctival sac of a camel.

- *Thelazia rhodesii* and *Thelazia leesei* occur in the conjunctival sac or lachrymal ducts of camels (Al-Ani, 2004).

Etiology:

- *Thelazia rhodesii* is milky white worm.
- Males are 8-12 mm and females are 12-18 mm long (Al-Ani, 2004).
- The life cycle is indirect and the intermediate host is *Musca* species.
- The first larvae of *Thelazia rhodesii* enter the gut of the fly from the eye secretions of the definitive host and penetrate the ovarian follicles of the fly. They then develop and become second stage larvae, which grows and molt to become the third infective stage larvae. The third stage larvae leave the ovarian follicles of the fly and migrate to the mouth parts of the fly, where they are transferred to camels.

Clinical sings:

- The eye worm of camels is of little pathogenic significance. However, sometimes it may cause irritation, cloudy cornea, marked lacrimation, conjunctivitis, and keratitis (Al-Ani, 2004).
- One or both eyes may be infected without clinical sings. However, irritation result in lacrimation, conjunctivitis, keratitis, and in severe infection the cornea may become opaque (Wernery and Kaaden, 2002; Al-Ani, 2004).

Diagnosis:

- Finding the parasite in the conjunctival sac or eggs in lacrimal secretion (Wernery and Kaaden, 2002; Al-Ani, 2004).

Treatment:

- The parasite can be mechanically removed, or under sedation with local anesthetic diethylcarbamazine in a concentration of 2 mg/L injected in the conjunctival sac, or by injecting 2 ml of levamisole in the subconjunctival sac, or subcutaneous injection of ivermectin (Al-Ani, 2004; Fowler 2010).

Myiasis (fly strike)

- Different types of flies have been reported to cause fly strike in camels.

Blow fly

- The most important blowfly that attacks camels is *Lucilia cuprina*.
- *Lucilia cuprina* is greenish to bronze in color, strong and their olfactory sense is highly developed.
- The female fly lays clusters of light yellow eggs in carcasses, infected wounds, soiled areas, and matted fur around infected sores and discharges.

Clinical signs:

- The maggots attack the skin and soon begin feeding on the skin and other tissues.
- This causes considerable stress to the infested camel, which may be seen rubbing and biting the infested parts of the body (Wernery and Kaaden, 2002, Al-Ani, 2004).

Treatment and control:

- Treatment is by using suitable insecticides and larvicides, and control is by shearing long fur.

Screw worm

- Screw worm is given to the larvae of *Collitroga*.
- The larvae of the fly *Chrysomnyia bezziana* is the cause of the Old World Screw worm myiasis (OWS).
- It occurs in Africa from the south of the Sahara to northern South Africa, Arabia including South Arabia, Yemen, Oman, United Arab Emirates, Iraq, Jordon and Syria, the Indian subcontinent and South Asia from Southern China through the Malay Peninsula and the Indonesian and Philippine islands to New Guinea.
- The larvae of the fly *Chrysomnyia Hominivorax* is the cause of the New World Screw Screwworm myiasis (NWS).

- It occurs in North Central and South America – from the Southern United States through Mexico, Central America, the Caribbean islands, and the northern countries of South America to Uruguay, Chile and Argentina.
- Unlike other species of blowfly, female screwworms are obligatory parasites and do not lay their eggs on carrion.
- The female fly deposit clusters of 50-150 eggs at the edge of the wound on a host.
- Virtually any wound is attractive, whether natural (from fighting, thorns, disease and tick) or manmade (from branding, castration and treatment).
- The larvae hatch in 10-12 hours and grow to maturity in about 3-6 days, after which they leave to pupate on the ground. The mature larvae are about 15 mm long and are well armed with spiny bands around the body segments. The pupal period lasts for three days or one to several weeks for *Chrysomnyia bezziana* and for three days for *Chrysomnyia Hominivorax*.
- Cattle, sheep, goats, and horses suffer frequently, but other animals including camel may also be affected.

Clinical signs:

- The maggots penetrate into the tissues, where they liquefy the tissue, and extend the lesion considerably.
- The lesion, develops a foul smelling odor.
- Larvae feeding on the skin and underlying tissues of the host cause myiasis, which can be fatal.
- Mature larvae are pink in color and 1-2 cm long. They are pointed anteriorly and blunt posteriorly. Two dark lines are visible reaching from the blunt posterior to the middle of the body and have rows of dark fine spines on the anterior part of each segment.

Flesh fly

- The larvae of the flesh fly *Sarcophaga dux* may be found in the skin of camels.
- The larvae of the old world flesh fly, *Wohlfarthia magnifica*, occur in the Mediterranean, Arabia, Turkey and Russia affecting the inner margin of the vulva mucosa of Bactrian camels.
- *Wohlfarthia nuba* occurs in man and animals especially camels in the Sudan, Ethiopia and eastwards to Karachi.
- The fly may deposit its larvae in lesions around the eyes or elsewhere on the body. They penetrate the skin and develop in the subcutaneous tissues, and secondary bacterial infection develops, and produces intense irritation and inflammation of tissues.

Treatment and control:

- The wound should be washed thoroughly and the maggots should be destroyed to prevent them from pupating.
- The wound can be packed with gauze soaked in a mixture of oil and chloroform (1:2) for a few minutes. The gauze is removed from the wound and the maggots are removed with a forceps. This is repeated for 24 hours.
- Dichlofenthion as 1 % aerosol is used to treat screw worm infection (Al-Ani, 2004).
- Direct prevention from infection is achieved by dipping or spraying with insecticides such as organophosphorus compounds (Wernery and Kaaden, 2002; Al-Ani, 2004).

Camel Nasal Bot Fly

- The larval stage of *Cepalopina titillator* occurs in the nasal cavity, frontal sinus and pharynx of Arabian and Bactrian camels.
- They cause nasopharyngeal miyasis in Iraq, Egypt, Saudi Arabia, North Africa and other areas where camel rearing is practiced.
- The adult fly is about 8-11 mm in length, with the upper part of the head orange and lower part pale yellow with dark frontal stripe. The head is partially covered by silvery pruinosity. Antennas are separated by heart shape convex area. The eyes are separated from each other, especially in the female (Al-Ani, 2004).

Figure 21 a: Sagittal view of the head of a camel showing severe hyperemia, congestion and hemorrhagic tracts in the nasal septum and pharynx of a camel caused by the nasopharyngeal larvae of the fly *Cephalopina titillator*.

Figure 21b: *Cephalopina titillator* larvae in the nasopharynx of a camel.

Clinical signs:

- The larvae of this fly are deposited in the nostrils of camels and molt twice while attached to the nasal passages and pharynx, and two cycles may occur each year.
- Most cases develop no obvious signs, while others develop nasal discharge, restlessness, head shaking, frequent sneezing and snoring during respiration.
- In rare cases when the damage caused by the larvae is severe; neurological signs appear as the result of secondary bacterial infection due to larval migration to the brain (Al-Ani, 2004).

Diagnosis:

- The larvae may be seen in the nasal cavity by the laryngoscope, and at post-mortem larvae of different stages can be found in the nasal cavity and pharyngeal area (Al-Ani, 2004).

Treatment:

- In endemic areas subcutaneous injection of ivermectin at the dose rate of 200 µg/kg body weight is recommended.
- Other medications such as rafoxinide at the dose rate of 7.5 mg/kg orally or ruelene at the dose rate of 110 mg/kg orally were found effective (Al-Ani, 2004).

Sarcoptic mange

Figure 22 a: A camel with advanced lesions of sarcoptic mange, showing complete alopecia, thick crust formation, wrinkling, folding, cracking and fissuring of the skin of the hind quarter and tail

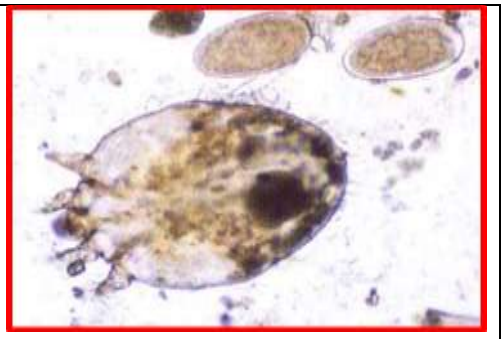

Figure 22 b: *Sarcoptes scabiei* with eggs in skin scrapings from an infected camel.

- Sarcoptic mange is probably the most important ectoparasite in camels.
- The mite is a burrowing mite causing a severe disease in all domestic animals especially camels, sheep and goats (Abu-Samra, Hago, Aziz and Awad1981; Abu-Samra, Imbabi, and Mahgoub, 1981; Ibrahim and Abu-Samra, 1985; Nayel and Abu-Samra, 1986).
- The disease causes serious economic losses, may result in death in case the infection is severe, and is dreaded by camel owners (Abu-Samra, Hago, Aziz and Awad1981; Abu-Samra, Imbabi, and Mahgoub, 1981; Ibrahim and Abu-Samra, 1985; Nayel and Abu-Samra, 1986).
- The parasite complete its life cycle in the tunnels they burrow into the stratum corneum and deep in the skin (Nayel and Abu-Samra, 1986; Al-Ani, 2004).

Etiology:

- *Sarcoptes scabiei* causes sarcoptic mange in camels. The mites have round body with short legs, the caudal tow pair of legs do not extend beyond the margins of the body, with bell-shaped caruncles on the long non-segmented pedicels in the leg of the male and only the anterior two pair of females. The male has no copulatory disc and the head is broad. The female mites measure about 0.5 mm, and the male about 0.3 mm (Fowler, 2010).

Clinical signs:

- Initially, the camels become restless showing intense pruritus manifested by rubbing or biting and gnawing the affected areas on the skin. Severe erythema with many papules and nodules (0.5 – 1cm) are seen on the head, neck, flanks and thighs.
- The papules and nodules oozed serous exudate, and the scratching and rubbing causes alopecia (1 -1.5 cm). The hair is initially moist, and then become dry 7-10 days later. New papules and nodules appear in many areas, and the coat becomes rough, naked and had moth eaten appearance.
- There is crust formation, secondary excoriation, but the camels show no change in all sings of health.
- In the late stage the skin become bald, and the animal become weak and loses condition. The skin had an offensive odor and become thickened, wrinkled into folds, and showed fissuring and cracking oozing blood.
- In the terminal stage of the disease the camel decreases its food and water intake and become emaciated. It developed edema on the legs and foot pads, become unable to work or walk for long distances, and eventually die (Nayel and Abu-Samra, 1986).

Diagnosis:

- Deep skin scrapings from the lesions examined in 20 % KOH for the mite and its different developmental stages (Nayel and Abu-Samra, 1986).

Treatment:

- The camel is washed with soap and water followed by scrubbing the animal with 15 % aqueous solution of salicylic acid, and finally washed with Gammatox or Sebacil EC 50 or injected subcutaneously with ivermectin (Nayel and Abu-Samra, 1986).

Ticks

Etiology:

- Many types of ticks have been reported to infest camels in different parts of the world, such as *Ornithodoros savingyi*, *Amblyomma lepidum*, *Rhipicephalus spp.*, *Dermacentor spp.*, *Boophilus microplus*, *Ixodes holocyclus* and *Hyalomma spp.* (Al-Ani, 2004).

Clinical signs:

Ticks produce many deleterious effects on their camel host (Al-Ani, 2004).
These deleterious effects can be summarized in the following:

1) Skin damage by the attachment of the tick resulting in secondary bacterial abscessation and myiasis.
2) Heavy infestation of camel prepuce lead to phimosis and coital problems.
3) Heavy infestation may cause production losses due to anemia, irritation, allergic responses, and emaciation.
4) Ticks have the ability to act as mechanical or biological vectors for viruses, rickettsia, bacteria, spirochetes, protozoa and helminths.
5) Ticks secrete in their saliva a neurotoxin which can cause paralysis in camel calves.

Diagnosis:

- Finding of the ticks attached to the skin of the affected camels (Wernery and Kaaden, 2002; Al-Ani, 2004).

Treatment and control:

- Acaricides, alteration of the environment, clearance and burning of vegetation, the use of tick repellents (organophosphorus acaricides, diamidine acaricide, and amitraz) have repellent action when applied by spraying (Al-Ani, 2004).
-

Pediculosis

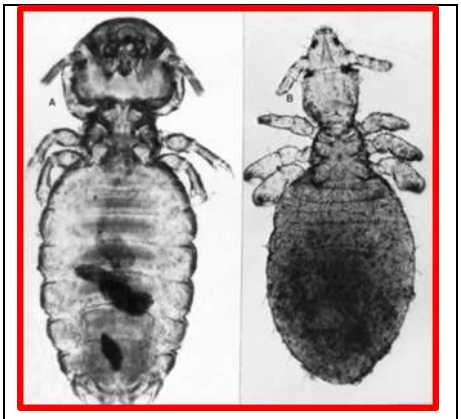

Figure 23: Camel biting louse (*Damalinia breviceps*) - left, and camel sucking louse (*Microthoracius cameli*) - right.

- There are two orders of lice *Anoplurida* which is sucking lice and *Mallophagida* which is biting lice (Wernery and Kaaden, 2002).

Etiology:

- The eggs of lice are usually cemented to the hair of the host. They hatch in one to three weeks (mean 12 days), and three nymphal stages take 12 days to produce adults, which reach sexual maturity in one to three days.

Clinical signs:

- Lice cause irritation to the host resulting in restlessness, inability to eat or rest well, and may injure themselves by scratching their body due the irritation caused by the lice, milk and meat production are decreased and fur quality is diminished, with the coat becoming rough and shaggy (Al-Ani, 2004).
- Camels infested with sucking lice show pruritus licking, scratching, and rubbing their body, the skin becomes rough with secondary bacterial infection resulting in damage of hides.
- In heavy louse infestation the camel becomes anemic (Wernery and Kaaden, 2002).
- Camels infested with biting lice show pruritus, lusterless ragged coats and in heavy infestations they show matted wool, alopecia and self-trauma (Wernery and Kaaden, 2002).

Diagnosis:

- It is easy to make a diagnosis by finding the lice or by detecting the eggs "nits" on the fur (Al-Ani, 2004).

Treatment:

- Pyrethrins, chlorinated and organic phosphates, hydrocarbons, and carbamates proved to be effective.
- Ivermectin is not affective in biting lice but affective against sucking lice (Fowler, 2010).

Flea Infestation

- Fleas are wingless insects with laterally compressed bodies, and about 1.5-4 mm in length.

Etiology:

- *Vermipsylla dorcadia* infest camels.
- The female flea lays up to 20 eggs at a time. The oval glistening eggs are deposited in the dust or dirt or on the host.
- The eggs are about 0.5 mm long, rounded at the poles and pearly white in color.
- The larvae may hatch in 2-16 days after the eggs have been laid, and the larvae are creamy yellow in color. They are very active and hide themselves from light. They are found in crevices in the floor and resting places of the host animal. A moderate temperature and a high degree of humidity are favorable for their development which takes 7-10 days or longer.
- The mature maggot is about 6 mm long.

Clinical signs:

- A large number of fleas cause irritation, debilitation, decreased milk and meat production and cause loss of fur.
- Blood sucking by adult fleas cause anemia and may transmit camel plague caused by *Pasteurella pestis.*

Treatment and control:

- Treatment is by reducing the breeding habitat and the use of insecticides (Fowler, 2010).

Horse Fly

Figure 24: *Tabanus* fly.

- *Tabanidae* insects, are large robust flies with powerful wings and large eyes.
- *Tabanus taeniola* and *Tabanus philoliche spp.* play as mechanical vectors for anthrax, *Trypanosoma evansi* and many other diseases.
- The bites of *Tabanidae* are painful and irritating, and camels are restless when troubled by these flies.
- The flies lay about 400-600 egg in the vicinity of water, usually on the leaves of plants. The eggs are about 2 mm long and light in color when laid, but turn dark after a while.
- The larvae hatch after 4-7 days and drop into the water or mud into which they disappear.
- The larvae feed on small crustacea and grow for 2-3 months, and perform several ecdyces and then pupate.
- The whole life cycle takes about 4-5 months under favorable conditions.
- The adult flies are seen in summer and are very fond of sunlight.
- The females are well known blood suckers and feed every three days chiefly on large animals including camels.

Treatment and control:

- Control of tabanids is very difficult.
- Spraying of animals and animals' houses with insecticides, keeping the animals away from the place where flies abound during the hot part of the day, and pouring kerosene on stagnant water (Al-Ani 2004).

Stable Fly

- *Stomoxys calcitrans* known as the stable fly have prominent forward directed pointed proboscis.
- It lives all over the world.
- Both males and females are blood suckers attacking camels and other animals.
- The fly requires about 3-4 minutes to take its meal and changes its position or fly to another animal to continue its feed.
- The fly mechanically transmits a number of diseases such as surra (Al-Ani, 2004).

House Fly

- *Musca domestica* is the common house fly.
- The fly has a cosmopolitan distribution and is important as a mechanical carrier of various infectious agents including viruses, bacteria, protozoa, and also acts as intermediate host for many helminthes (Al-Ani, 2004).

Bibliography

Abu-Samra, M.T., Hago, B.E.D., Aziz, M.A. and Awad, F.M. (1981): Sarco ptic mange in sheep in the Sudan. *Annals of Tropical Medicine and Parasito logy*. **75:** 639-645.

Abu-Samra, M.T., Imbabi, S.E. and Mahgoub, E.S. (1981): Mange in domes tic animals in the Sudan. *Annals of Tropical Medicine and Parasitology*. **75:** 627-637.

Al-Ani, F.K. (2004). *Camel Mangement and Diseases*. 1st Edition, Dar Am mar Book Publisher, Amman, Jordan.

Dubey, J. P.; Hilali, M.; Van Wilpe, E.; Calero-Bernal, R.; Verma, S. K.; and Abbas I. E. (2015). A review of sarcocystosis in camels and redescripti on of *Sarcocystis cameli* and *Sarcocystis ippeni* sarcocysts from the one-hu mped camel.

Elazhari Mohamed G. El. (1992). Infectious Diseases in Camels, faculty of veterinary science university of Khartoum Sudan.

Fowler, M. E. (2010). *Medicine and Surgery of Camelids*. 3rd Edition, Wiley Blackwell publication, Ames, Iowa, USA.

Ibrahim, K.E.E. and Abu-Samra, M.T. (1985): A severe outbreak of sarcopti c mange among goats naturally infected with a sheep strain of *Sarcoptes sca biei*. *Revue d' Elevage ET de Medecine Veterinaire des pays Tropicaux*. **38:** 258-265.

Nayel, M.N. and Abu-Samra, M.T. (1986): Sarcoptic mange in the one-hum ped camel (*Camelus dromedaries*). A clinico-pathological and epizootiologi cal study on the disease and its treatment. *Journal of Arid Environments*. **10 :** 199-211.

Schillinger, R. and Zweygarth, E. (1987-2017). Parasitic Disease of Camel i n Iran - A literature review. PP 2-8 .

The center for food security and public health 2005, Taenia infection page 1-8 .

Wernery, U. and Kaaden, O. (2002). *Infectious Diseases of Camelids*. 2nd Re vised and Enlarged Edition, Blackwell Science publication, Berlin, Germany .