

**PATHOLOGICAL CHANGES IN SKIN LESIONS OF CAMEL  
(*Camelus dromedarius*) IN RELATION TO *Staphylococcus  
aureus* INFECTION**

ऊँटों (केमिलस ड्रोमेडिरिस) में त्वचीय विकृतियों का  
स्टेफाइलोकोकस आरियस संक्रमण से संबंधित व्याधिकीय परिवर्तन

**CHANDRA PRATAP SINGH**

B.V.Sc. & A.H.

**THESIS**

**MASTER OF VETERINARY SCIENCE**

**(Veterinary Pathology)**



। पशुधनं नित्यं सर्वलोकोपकारकम् ।

**2017**

**Department of Veterinary Pathology  
College of Veterinary and Animal Science**

**Rajasthan University of Veterinary and Animal Sciences,  
Bikaner – 334001**

**Pathological changes in skin lesions of camel  
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परिवर्तन

# **THESIS**

Submitted to the  
Rajasthan University of Veterinary and Animal Sciences,  
Bikaner

In partial fulfillment of the requirements for  
the degree of

**MASTER OF VETERINARY SCIENCE**

**IN**

**Veterinary Pathology**

**FACULTY OF VETERINARY & ANIMAL SCIENCE**

**By**

**CHANDRA PRATAP SINGH**

**2017**

**Rajasthan University of Veterinary and Animal Sciences,  
Bikaner-334001**

**College of Veterinary and Animal Science, Bikaner**

**CERTIFICATE – I**

Date.....

This is to certify that **Mr. Chandra Pratap Singh** had successfully completed the comprehensive examination held on .....as required under the regulation for the degree of **Master of Veterinary Science**.

**(Dr. Hemant Dadhich)**  
**Head**

Department of Veterinary Pathology  
College of Veterinary and Animal Science  
Bikaner

**Rajasthan University of Veterinary and Animal Sciences,  
Bikaner-334001  
College of Veterinary and Animal Science, Bikaner**

**CERTIFICATE – II**

Date.....

This is to certify that this thesis entitled “**Pathological changes in skin lesions of camel (*Camelus dromedarius*) in relation to *Staphylococcus aureus* infection**” submitted for the degree of **Master of Veterinary Science** in the subject of **Veterinary Pathology** embodies bonafide research work carried out by **Mr. Chandra Pratap Singh** under my guidance and supervision and that no part of this thesis has been submitted for any other degree. The assistance and help received during the course of investigation has been fully acknowledged. The draft of the thesis was also approved by the Advisory Committee on .....

**(Dr. Hemant Dadhich)**  
Head  
Department of Veterinary Pathology  
College of Veterinary and Animal Science  
Bikaner

**(Dr. Manisha Mathur)**  
(Major Advisor)

**(Dr. G.S. Manohar)**  
Dean  
College of Veterinary and Animal Science  
Bikaner

**Rajasthan University of Veterinary and Animal Sciences,  
Bikaner-334001**

**College of Veterinary and Animal Science, Bikaner**

**CERTIFICATE – III**

Date.....

This is to certify that the thesis entitled “**Pathological changes in skin lesions of camel (*Camelus dromedarius*) in relation to *Staphylococcus aureus* infection**” submitted by **Mr. Chandra Pratap Singh** to Rajasthan University of Veterinary and Animal Sciences, Bikaner, in partial fulfillment of the requirement for the degree of **Master of Veterinary Science** in the subject of **Veterinary Pathology** after recommendation by the external examiner was defended by the candidate before the following members of the examination committee. The performance of the candidate in the oral examination on his thesis has been found satisfactory. We therefore recommend that the thesis be approved.

**(Dr. Manisha Mathur)**  
Major advisor

**(Dr. Hemant Dadhich)**  
Advisor

**(Dr. Tribhuwan Sharma)**  
Advisor

**(Dr. S.C.Goswami)**  
Dean P.G.S Nominee

**(Dr. Hemant Dadhich)**

Head

Department of Veterinary Pathology  
College of Veterinary and Animal Science,  
Bikaner.

**Approved**

**Dean**

Post Graduate Studies  
Rajasthan University of Veterinary and Animal science, Bikaner

**Rajasthan University of Veterinary and Animal Sciences,  
Bikaner-334001**

**College of Veterinary and Animal Science, Bikaner**

**CERTIFICATE – IV**

Date.....

This is to certify that **Mr. Chandra Pratap Singh**, of the Department of **Veterinary Pathology**, College of Veterinary and Animal Science, Bikaner has made all corrections/modifications in the thesis entitled "**Pathological changes in skin lesions of camel (*Camelus dromedarius*) in relation to *Staphylococcus aureus* infection**" which were suggested by the external examiner and the advisory committee in the oral examination held on..... . The final copies of the thesis duly bound and corrected were submitted on..... , are enclosed herewith for approval.

**(Dr. Hemant Dadhich)**

Head

Department of Veterinary Pathology  
College of Veterinary and Animal Science,  
Bikaner

**(Dr. Manisha Mathur)**

Major Advisor

**(Dr. G.S. Manohar)**

Dean

College of Veterinary and Animal Science,  
Bikaner

**Approved**

**Dean**

Post Graduate Studies

**RAJUVAS**, Bikaner Enclosed: One original and two hard bound copies of the thesis, forwarded to the Dean, Post Graduate Studies, **RAJUVAS**, Bikaner through the Dean, College of Veterinary and Animal Science, Bikaner

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## 1. INTRODUCTION

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The camel (*Camelus dromedarius*) is an important animal component of the fragile desert eco-system. Even in the modern era of machines, the efficiency of camel in desert cannot be replaced. The proverb “Ship of the desert” named its epithet on account of its indispensability as a mode of transportation and draught power in desert but the utilities are many and are subject to continuous social and economic changes.

Scientific classification of the dromedary (single humped) camel is as follows: order: *Artiodactyla* (even-toed ungulates); suborder: *Tylopoda*; family: *Camelidae*; sub family: *Camelinae*; genus: *Camelus*; with two species named Bactrianus (two-humped) and Dromedarius (one-humped).

Camel is used in agriculture and many other allied works (Knoess, 1977). In earlier days, camel was used as baggage animals by pilgrims, and also in ancillary military purposes (HMSO, 1956). Water carrying is another important function carried out by the camel both for nomads and static settlements from distant water sources (Hopkirk, 1980). Besides providing meat, milk, hair, hide and organic manure, camel also serves for riding purposes (Higgins, 1986). Within the arid regions, the camel-breeding tribes have maintained a dominant position over other societies by virtue of the ability of the camel to exploit even poor grazing ranges (Sweet, 1965).

The camel tolerates high temperature, solar radiation and water deprivation. it has ability to support a very high degree of water loss amounting to as much as 30 per cent of its initial body weight. Due to the extremely concentrated urine and insignificant sweat, the camel does not loose water. The temperature of skin remain cool due to coarse and well ventilated hairs on its back which allow evaporation to

take place on the surface of the skin. Thriving on a diet of thorny, fibrous and often very salty plants of poor quality, camel converts the scattered vegetation of desert into highly nutritious food and other form of energy. The huge plate like feet themselves are less damaging to soil structure than the smaller cloven hooves of the other common domesticated animal species. This ability of camel also makes the animal most suited for the desert and semi-desert areas.

A recent FAO/CIRAD/KARKARA workshop estimated global camel milk output as about 5.3 million tonnes. Camel milk is slightly saltier than cow's milk, three times as rich in vitamin C and known to be rich in iron, unsaturated fatty acids and vitamin B (Gahlot, 2007).

Camel milk is reported to have bactericidal and viricidal properties (Yagil, 2000) and inhibits various pathogenic bacteria (Barbour *et al.*, 1984). Biala and Gnan (1998) expressed their views that camel played an important role in the diet of people in the Arab world by providing meat and milk during 21<sup>st</sup> century.

World camel population is estimated to be around 25.89 million spread across 47 countries. About 85 per cent of the camel population inhabits mainly Eastern and Northern Africa and rest in Indian subcontinent and Middle East countries. Somalia has the highest camel population (7.0 million) (NRCC, VISION 2030). India stands tenth in the world ranking with 0.51 million camels (FAOSTAT, 2011). The majority of world's camel population is of dromedary type except small population of Bactrian camels in central Asia. Around 80 per cent is possessed by Rajasthan (0.325 million) and rest camel population is found in Gujrat, Haryana and U.P. (Indian livestock census, 2012). The Rajasthan government on June 30, 2014 declared the camel as the state animal and also in the process of drafting a new law to prevent slaughter, illegal trade and transportation of camel and announced to include camel milk in its Food Security Programme (THE HINDU, 2014).

According to Faulkner (1978), camel hide is especially valued for its large size and the quality of the leather obtained. Wool and hair are also used for manufacturing of tent cloths, blankets etc. Usta *et al.* (1997) have described preparation of decorative items like lamps, shades, decanter or goblets, jewellery, pen stand, table ware etc. by processing camel hide and are decorated with gold painting after giving it a shape known as “Ustakala”.

The skin is the largest organ of the body and the anatomical and physiological barrier between the animal and environment. It provides protection from physical, chemical and microbiological injury. Its sensory components perceive heat, cold, touch etc, skin functions in a limited way as secretory and excretory organ besides immunoregulation and thermoregulation. The skin surface has antibacterial and antifungal properties. In camels, the skin is attached rather tightly to the underlying tissue and is relatively immobile. This is a disadvantage when the animal is attacked by biting and flying insects, particularly in view of its short and ineffective tail.

The camel has a low susceptibility to diseases but skin involvements like contagious skin necrosis, dermatitis, wounds, abscesses or similar problems were commonly observed in camels (Rutter and Mack, 1963; Semushkin, 1968; Edlesten and Pegram, 1974; Domenech *et al.*, 1977).

The primary habitat of *S.aureus* is in the nasal passage, on the skin and hair of human and warm-blooded animals. The transmission of the organisms may occur through skin lesions, contaminated food, including milk and other animal products (Le Loir *et al.*, 2003; Boerema, *et al.*, 2006).

*Staphylococcus spp.* especially *S.aureus* is one of numerous infections worldwide, with clinical manifestations including skin and soft-tissue infection, sepsis and pneumonia (Monecke *et al.*, 2014; Steinig *et al.*, 2015).

The skin infections due to *Staphylococci* are contagious skin necrosis, dermatitis, wounds, abscesses or similar lesions are a problem in camel. The infection is chronic and difficult to treat medically depending on among other factors, the pathogenicity of the *Staphylococcal* strain present (Wernery, 2000). The disease is not fatal but due to reduced working efficiency it causes great economic losses. Sometimes the animal becomes of no use because of widespread abscesses or wounds over the whole body which are difficult to manage and even the antibiotic therapy does not work satisfactorily

The literature regarding microbiology of the skin wounds and abscesses in camel is scarce. Different workers (Kataria, 1999; Qureshi *et al.*, 2002; Rathore, 2012) have isolated *Staphylococcus aureus* from skin lesions but information remains incomplete as far as strain identification and strain variation is concerned since this organism shows variations in phenotypic expressions (Boerlin *et al.*, 2003) and acquires antibiotic resistance with remarkable proficiency (Booth *et al.*, 2001).

*Staphylococcus aureus* is gram positive, ubiquitous, pathogenic bacteria found mainly in nose and skin of animals. It is known to cause variety of suppurative infections and has always been a major cause of wounds and abscesses and mastitis in animals. Over the past few decades the organism has come up as a leading cause of hospital and community acquired infection in human subjects causing endocarditis, deep-seated abscesses and bacteremia leading to toxic shock syndrome. The disease caused by *S. aureus* is not always fatal but an indirect great economical loss is incurred due to reduced working efficiency. The abscesses and wounds caused by *S. aureus* spread rapidly over the body surfaces and become very difficult to manage making animal useless for any economical purpose.

The *staphylococcal* alpha-toxin (*hla*) is a major virulence factor contributing to *Staphylococcus aureus* pathogenesis. *hla* genes codes

for *Staphylococcal* toxins. It is a pore-forming toxin and has cytolytic, haemolytic, dermonecrotic, and lethal activities. A wide range of cell types, including erythrocytes, monocytes, lymphocytes, macrophages, epithelial cells, fibroblasts, and keratinocytes, are affected by alpha-toxin (Bhakdi *et al.*, 1988; Bhakdi and Jensen., 1991; Song *et al.*, 1996).

The pathogenicity of *S.aureus* is related to the production of a wide variety of exoproteins including alpha and beta haemolysins which contribute to its ability to cause diseases in many mammalian species (da Silva *et al.*, 2005). Alpha–haemolysin or alpha toxin is considered a main pathogenicity factor because of its haemolytic, dermonecrotic and neurotoxin effect and it is governed by *hla* gene.

In Rajasthan, so far very little efforts have been made to study the aetiology, prevalence and pathology of various dermatological disorders in camel. Keeping in view of these facts the present investigation has been undertaken with following objectives:

1. To find out the occurrence of various pathological conditions of skin lesions in camel.
2. To study the gross and histopathology of various pathological conditions of skin lesions in camel.
3. Genotypic characterization of *Staphylococcus aureus* and its correlation with histopathological changes of skin lesions in camel (*Camelus dromedarius*).

## 2. REVIEW OF LITERATURE

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### 2.1 DERMATITIS

Leese (1927) described in detail the contagious skin necrosis in camels. The condition begins as painful swelling and then ulcer is formed which sometimes heal or may remain unhealed. Pus may be formed in the lesion and the lymphatic gland draining the affected part of the skin becomes swollen, hot and painful.

Peck (1939) believed contagious skin necrosis to be a symptom of salt deficiency. Hot, hard, painful swelling appeared on the body especially on the neck and hind quarter which became irritant within few days. The camel bites and scratched the lesions to the abscesses, or sinuses developed and necrotic skin was sloughed.

Pullen (1950) described cutaneous impetigo contagious as *staphylococcal* or *streptococcal* infection being evident as discrete, thin walled vesicles and bullae. These ruptured forming erosions covered with purulent crust.

Ogilvie (1957) described cellulites in man as inflammatory affection of ordinary fibrous and fatty tissue found in subcutis. Grossly the affected tissue was swollen. On section there was variable amount of yellowish or yellowish red liquid containing pus. Microscopically the capillaries and venules were greatly dilated lined by swollen endothelium and filled with polymorphs which were often accompanied by infiltration of lymphocytes, plasma cells and eosinophilic leucocytes and a few fibroblasts in dermis.

Rutter and Mack (1963) reviewed the diseases of camel into two parts. They described that contagious skin necrosis was characterized by necrotic lesions of the skin and abscesses in various part of the body and was widespread in Egypt, Sudan and Somalia. They further

pointed out that it was common in India where the camels seemed more susceptible than the camel in Africa.

Kouba (1964) reported various animal disease problems in Mongolian People's Republic. He found that skin necrosis was common skin disease in camels.

Cohrs (1967) described furuncle in all animals as abscess forming inflammation of skin involving hair follicle or sebaceous gland showing projecting nodule exuding pus. *Staphylococcus aureus* organism was isolated from it. Further, formation of necrotic plug and its sloughing was followed by filling of cavity with granulation tissue.

Semushkin (1968) reported that *staphylococcal* disease (contagious skin abscesses) could affect 5 to 20 per cent of Bactrian camel population.

Eldelsten and Pegram (1974) carried out a survey during 1970-72 and studied mild form of contagious skin necrosis. Lesions consisting of a firm swelling which burst after 5-10 days leaving a circular discharging sinus in the centre of the gluteal region but also occurred in the inguinal, perineal and lower cervical region.

Domenech *et al.* (1977) in Ethiopia reported a chronic suppurative disease known as Mala leading to the formation of internal as well as external abscesses is apparently more important than contagious skin necrosis and usually found on the lower neck or on the hump

Buchnev *et al.* (1978) considered that *staphylococcal* infections were one of the important diseases of camels in the USSR and *Staphylococcal* disease, also called contagious skin abscesses was found widespread among Bactrian camels in central Asia.

Chaudhary (1978) reported that abscess was a localized collection of pus in any part of the body, produced by a pyogenic irritant, the inflammation being limited to one area producing a pus

filled cavity with damaged but still living wall. In the process of abscess formation two chief changes occur side by side in the tissues, a progressive emigration of polymorphonuclears leucocytes, which come to the tissue or gradual distinction and disappearance of the tissue elements. The special cells of the part become necrosed, break down into granular material and melt away, while the supporting connective tissue fibrils, capillaries etc digested and disappear. The accumulation of leucocytes still goes on and the tissue gradually becomes replaced by pus, in which remains of the dead tissue are present.

Veen and Barnes (1978) studied cutaneous ulcers of camels from various parts of Northern Nigeria. The ulcers which sometimes reached a diameter of 20 cm were pathologically characterized by extremely dense collagenous tissue, extensive infiltration by eosinophils.

Nicholls and Rubria (1981) studied *staphylococcal* dermatitis in cows showing pustules, sores or generalized exudative dermatitis in a herd. Here mastitis was associated with *Staphylococcus aureus*. These organisms were also recovered from the skin lesions.

Nesbitt (1983) classified pyoderma in canines into surface, superficial and deep forms. Surface pyoderma included pyotraumatic dermatitis and skin fold pyodermas. It showed focal or multifocal erythematous erosions in the former and excoriation, hyperkeratosis and hyperpigmentation in fold of skin in the later. Superficial pyoderma mainly involved hair follicles forming erythematous papule, pustule and crust, leading of the hyper pigmented maculae. Deep pyoderma include furunculosis, callous and interdigital pyoderma.

Ismali *et al.* (1985) observed oedematous skin disease in camels. *Corynebacterium ovis* was isolated in pure culture from closed lesions. *C. ovis* was also found associated with *Staphylococcus aureus*.

Carter (1986) stated that various infections of the skin of many animals, subcutaneous abscesses caused by *Staphylococcus species*.

Rook *et al.* (1987) described cellulites in man as an acute, sub acute or chronic inflammation of loose connective tissue. It occurred as a complication of wound, ulcer or other skin lesions, grossly characterized by redness and swelling.

Krick and scott (1989) described cellulites in dogs, unrelated to age or sex showing follicular inflammation, skin necrosis and sloughing of affected part. *Staphylococci* were isolated from these lesions.

Mohmed *et al.* (1990) during a study of camel diseases in the Sudan reported that abscesses were sporadic incidence that causes loss of weight. The causative various bacteria include *Staphylococcus species*.

Hazarika *et al.* (1991) studied experimental *staphylococcal* dermatitis in rabbits. Gross lesions produced were erythematic and necrosis. Biopsy revealed marked haemorrhage, oedema, necrosis and infiltration of mononuclear and polymorphonuclear cells both in epidermis and dermis. Increased fibroblastic activity was noted in and around infiltration.

Nguhiu-Mwangi *et al.* (1991) reported non suppurative limb cellulites with skin necrosis and sloughing in the cattle. It seemed to be encouraged by the paucity of tissue between the skin and the bone by the poor vascularity of the area and by the causative bacteria.

Dioli and Stimmelmayer (1992) reported that singular or multiple external and internal abscesses are a very common health problem in camels. Several organisms have been isolated from abscesses such as *Corynebacterium spp.*, *Staphylococcus spp.*, *Pseudomonas spp.*, and *Actinomyces spp.*

Yager and scott (1993) described pyoderma as primary or secondary and superficial or deep. All bacterial pyoderma were

secondary to some exogenous and endogenous triggering factor. *Staphylococcus aureus* were often isolated from bacterial pyoderma in ruminants. Superficial pyoderma involved the epidermis and portion of the hair follicle forming papules, transient pustules and annular areas of crusting, scaling and alopecia. Deep pyoderma involved dermis and sub cutis including cellulites, furunculosis and abscess. Further cellulitis was also described in domestic animals as a severe, deep, diffuse suppurative to necrotic dermatitis in which area of infection was poorly confined showing extensive oedema with friable, dark discoloured, devitalized skin which sloughed easily.

Kaaden and Wernery (1995) described *Staphylococcal* dermatitis in camels and was assigned to be one of the important and common bacterial disease inflicting camels.

Scott *et al.* (1995) described pyoderma in canine as surface, superficial and deep. The surface bacterial infection included pyotraumatic dermatitis and pyotraumatic folliculitis. Superficial pyoderma included impetigo mucocutaneous pyoderma, superficial bacterial folliculitis, furunculosis, cellulites, subcutaneous abscess, bacterial pseudomycetomas and mycotic granuloma.

Jones *et al.* (1996) described folliculitis in animals showing thickened, indurated and hairless skin. Histologically, the lesions were inflammatory showing necrotizing changes in hair follicle. Dermis near these lesions showed micro abscesses, aggregate of epithelioid cells and foreign body giant cells. Further, furunculosis was also described showing abscesses in dermis, hair follicles or sebaceous glands. Furthermore cellulites were also described in domestic animals as skin necrosis and sloughing of the affected part showing diffuse suppurative to necrotic dermatitis with follicular inflammation.

Yagoub and Mohamad (1996) investigated incidence and reported clinical observation and etiology of contagious skin necrosis in

Sudan. Out of 2284 camels, 131 (5.73 per cent) were affected with contagious skin necrosis.

Yeruham *et al.* (1996) noticed that impetigo appeared in a dairy cattle herd in association with intensive showering of the lactating cows during the summer months involving the perineum, the udder and the ventral surface of the tail. *Staphylococcus* was isolated from 7 of 8 samples from these cows.

Abu Elzcin *et al.* (1998) observed a highly contagious skin disease in a herd of dromedary camel consisting of 700 animals of different age groups in Saudi Arabia. The disease primarily struck young stock and then spread to the adults. The morbidity rate was 24 per cent while mortality zero per cent.

Agab (1998) reported common disease in Sudan during one year (March 1991 to Feb. 1992). Out of total 3932 diseased camels, wound and abscesses were found in 315 camels, contagious skin necrosis was found in 96, contagious ecthyma in 65, pox in two and papilloma virus infection in one case. He found that contagious skin necrosis, contagious ecthyma and camel pox affected mainly young calves. The death rate is higher in calves under 6 months of age.

Bihani *et al.* (1998) observed 6 cases of necrotic dermatitis during the year 1996-97 having lesion on shoulder, neck and ventral aspect of tail. The lesion started as painful pustular swelling of 4-8 cm diameter. The scab formation took place within 2-3 days which could be peeled off easily leaving a raw red granulomatous surface. Purulent discharge was also observed from the site the lesion.

Sarmah *et al.* (1998) studied experimental *staphylococcal* dermatitis in goats. Gross lesions included erythema, necrosis and abscess formation. The microscopic changes included edema, hemorrhage and necrotic changes. Blood vessels surrounding the necrotic areas were congested showing increased fibroblastic activity.

Pasricha and Gupta (1999) described cellulitis in man as an infection of the deep dermis and subcutaneous tissue producing a large painful area of redness and swelling. This was usually located on the leg, forearm or hand associated with fever.

Wernery (1999) has described various infectious diseases of camelids. Bacterial skin diseases are mainly of two types *Staphylococcus aureus* dermatitis and Pseudotuberculosis.

Monle *et al.* (2000) noted clear histopathological changes of inflammatory response in skin locations inoculated with  $2 \times 10^7$  cfu *Staphylococcus aureus*. Microscopic evaluation revealed a dense infiltrate of neutrophilic granulocytes with visible bacteria in deep dermis and subcutaneous fat after 6hr. In addition, abscess formation in the superficial musculature was noted. Surrounding these abscesses, a diffuse infiltrate of neutrophilic granulocytes and mononuclear cells in dermis dominated, but sparse Perivascular infiltrates were also seen.

Rollefson *et al.* (2001) reported that contagious skin necrosis in camels caused by a number of bacteria including *S. aureus*.

Khanna (2002) described various histopathological conditions in dogs and reported that in intraepidermal vesicular and pustular dermatitis, hydropic degeneration of basal cells and intercellular oedema were found microscopically. In Panniculitis, there was a circumscribed area of Panniculitis and the cellular infiltrate comprised mainly of macrophages and neutrophils whereas in Seborrheic dermatitis epidermal hyperplasia and spongiosis revealed an enlargement of sebaceous gland.

Mehta *et al.* (2002) reported the disease profile of Jaisalmeri camel and found that among conditions, skin wound and saddle gall occurred in 7.67 per cent camel.

Nagase *et al.* (2002) reported that in dairy animal *S.aureus* causes pustular inflammation of skin and other organs of serious nature.

Quinn *et al.* (2002) reported that the *Staphylococcus* are pyogenic, they were associated with abscesses formation and suppuration. Pus is composed of the debris of dead leukocyte and living and dead bacteria can be surrounded by intact phagocytic cells and fibrin strands. A fibrous capsule was eventually formed around an abscess.

Schwan *et al.* (2003) reported skin infections, wound infections, and abscesses were predominant types of infections caused by *S. aureus*. These infections affect the epidermis, dermis, and subcutaneous layers of the skin. In a wound model, there is a lot of tissue destruction as the result of the burn, so tissue damage by the  $\alpha$ -hemolysin or even  $\delta$ -hemolysin may not be necessary to allow for bacterial dissemination.

Terada *et al.* (2006) analysed epidermal hyperplasia and inflammatory changes in the dermis of the skin sites of SDS\_SpA treated mice. The dermis was densely infiltrated with various subtypes of leukocytes, including eosinophils and mast cells. Focal accumulations of eosinophils were observed in the sub cut layer. In contrast, mice treated with SDS alone showed only minor skin alteration.

Bengoumi *et al.* (2007) reported two kinds of cutaneous abscesses in camels, i.e. lymphadenitis and skin abscesses. *Staphylococcus aureus* was isolated in 91% of lymphadenitis cases and 88% of skin necrosis cases.

Koutinas *et al.* (2007) investigated the flock of 290 sheep, total 20 ewes (6.9%) with an age range of 1.5–4 years, found to have developed a nonpruritic dermatitis characterized by alopecia,

hypotrichosis, erythema, hyperpigmentation, crusting, superficial ulcers, exudation and thickening of the skin that was localized mainly to the peri-ocular area. The main histopathological feature of this skin disease was a superficial and deep, perivascular to interstitial eosinophilic and mononuclear dermatitis.

Viana *et al.* (2008) reported that *Staphylococcal* mastitis was the main cause for culling adult does from rabbitries. However, very few scientists reported this condition on rabbits. The objective of this work was to improve the mammary pathological knowledge in natural cases of chronic mastitis through a histomorphological classification and to compare the differences between the lesions caused by several *S. aureus* genotypes. In order to define the spectrum of gross pathological changes, the macroscopically characteristics of the mammary glands of 130 does with chronic mastitis were recorded. The number of glands was registered, which varied between 8 and 10 mammary glands per animal. The incidence of affection of the different glands were also studied. Mastitis cases were classified according to defined histopathological criteria (abscess-type, rosette-type, sandwich-type and mixed-type). The abscess-type was the most frequently diagnosed (64.4% of the animals). This type was characterized by the presence of one (unifocal) or several (multifocal) well differentiated abscesses of variable size. The purulent material was mainly present heterophils of different grades of degeneration and of debris, together with large clumps of Gram-positive bacteria.

Dalis *et al.* (2010) carried out a study to determine bacteria associated with pathology of bovine dermatophilosis in north central Nigeria. Skin samples obtained from 211 cattle with skin lesions suspected to be dermatophilosis were processed for bacteriology and histopathology. Histopathology revealed hyperplasia of the epidermis, parakeratosis, necrosis, cellular infiltration of the hair follicles and papillary dermis, diffuse cellular infiltration of the reticular dermis and

folliculitis were also observed in some sections. It was concluded that the histopathological lesions observed could be due to *D. congolensis* complicated by secondary bacterial infection. The need to investigate the role of bacteria particularly that of *S. aureus* in the development of bovine dermatophilosis was emphasized.

Oryan *et al.* (2010) observed case of erythema multiforme in an 11 years old mare which had received no medication was presented with a history of foaling. This horse displayed a gradual outbreak of erythematous maculae's, raised papules, non alopecic and non pruritic plaques over the lateral aspects of the neck and shoulders to rump without involvement of the dorsum and ventral abdomen. The distribution of these skin lesions was bilateral but asymmetric. Histopathological findings included mild hydropic degeneration and scattered apoptotic keratinocytes especially at different layers of the epidermis. The dermis showed a marked full-thickness oedema particularly at epidermodermal junction with moderate diffused infiltration of mixed eosinophils, lymphocytes, plasma cells, and macrophages at different portions of the dermis.

Bath *et al.* (2011) reported full-depth necrosis of the epithelium with severe ulceration and acute tissue necrosis in skin biopsies. There was prominent oedema, rhabdomyolysis and acute neutrophilic infiltration in some parts of the subcutis. A pathological anatomical diagnosis of severe multifocal necrotic dermatitis accompanied by exudation and ulceration was made. The lesions were found to be consistent with a dermatotoxic insult.

Hanaa *et al.* (2011) examined fifty adult she camels for teat abnormalities at kerdasa slaughter house at Egypt. Teats were examined pathologically and microbiologically. Gross examination of teats revealed 76.5% showed teat lesions. The total bacterial recovery rate was 36% and the isolated bacteria were *S. aureus* 14.5%, *E. coli* 14%, Coagulase-negative staphylococci, 4%, *Bacillus species* 1.5%, *P.*

*hemolytica*, 1.5% and *P. aeruginosa* 1.5 %. While total mycotic recovery rate was 26.5% with isolation of *C. Albicans* 9.5%, *A. fumigatus* 9%, *A. niger* 2%, *mucor* 2.5% and *cladosporium* species 8.5%. Histopathological examination of teats revealed two types of thelitis, Necrotic suppurative thelitis (25.5%) and necrotic non suppurative thelitis with diffuse fibrosis (38%). They concluded that *S. aureus* and *C.albicans* were the most prevalent isolates from she camel thelitis.

Foster (2012) reported cutaneous manifestations of *staphylococcal* infection in livestock species. In cattle, *staphylococcal* infections may present as folliculitis or as impetigo. Both may present as mild forms of a group of conditions loosely termed udder dermatitis, which has various clinical presentations and does not always involve *staphylococci*.

Hamed *et al.* (2012) reported characteristic dermal lesions of contagious skin necrosis in ten camels, these lesions were demonstrated on different parts of the animal body as an area of skin necrosis in which the skin looked black in colour and not covered with hair. These sharply separated from the surrounding healthy skin and were cold and very hard in consistency. When an area of necrosed skin was detached, circular ulcer of varying diameter, usually 2-10 cm remained and clearly demarcated from surrounding healthy tissue.

Mathur *et al.* (2012) collected one hundred eighty seven skin biopsies showing frank macroscopic lesions were from camels for histopathological study of various types of dermatitis along with haemato-biochemical parameters and isolation of organisms. Perifolliculitis, folliculitis and frunculosis conditions were recorded in 11.68 per cent cases of dermatitis in camel.

Soong *et al.* (2012) reported *Staphylococcus aureus* as the major cause of skin and soft tissue infection in the United States. Invasive USA300 infection has been attributed to several virulence factors, including protein A and the  $\alpha$ -hemolysin (*hla*), which causes

pathology by activating host signaling cascades. They showed that *S. aureus* exploits the proinflammatory bias of human keratinocytes to activate pyroptosis, a caspase 1-dependent form of inflammatory cell death, which was required for staphylococci to penetrate across a keratinocyte barrier. Keratinocyte necrosis was mediated by calpains, Ca (2+)-dependent intracellular proteases whose endogenous inhibitor, calpastatin, is targeted by Hla-induced caspase 1. Neither Panton-Valentine leukocidin nor protein A expression was essential, but inhibition of either calpain or caspase 1 activity was sufficient to prevent *staphylococcal* invasion across the keratinocytes. These studies suggest that pharmacological interruption of specific keratinocyte signaling cascades as well as targeting the hla might prevent invasive skin infection by *staphylococci*.

Anthony (2013) reported that biopsy should be considered if immune-mediated disorders or neoplastic conditions are suspected. Consideration of these differentials is also recommended when treatment has been pursued, and failures or relapses have occurred. In most cases, especially suspected, skin biopsies should be read out by a dermato-histopathologist. Acute changes including leukocytoclastic vasculitis, thrombosis and vessel wall necrosis are often scarce and can easily be overlooked; when present, they may provide a diagnosis. Vessel wall thickening and hyalinization, along with epidermal hyperplasia or papillomatous, may be detected in chronic lesions. If secondary bacterial infection is severe, it is recommended to clear this before taking the biopsy. For hyperplastic and nodular lesions, such as skin of severe CPL patients, a double punch biopsy is recommended, whereby a smaller 6 mm punch biopsy is introduced into the opening previously created by the skin sampled using an 8 mm biopsy. This technique will allow the practitioner to reach the deep dermis and subcutis for conditions such as CPL and panniculitis, respectively.

Psalla *et al.* (2013) reported that equine pastern vasculitis was clinically challenging and the underlying etiopathogenesis was unclear. The aims of this retrospective study were to establish histopathological criteria for pastern vasculitis, to look for an underlying cause, to investigate whether the histopathological lesions are associated with a distinct clinical picture, to assess if and how the clinical picture varies, and to determine the treatment response. Skin biopsies and clinical data from 20 horses with a diagnosis of vasculitis of the distal extremities were investigated and histology was compared to biopsies from healthy horses. It was concluded that intramural inflammatory cells, leukocytoclasia with nuclear dust, thickening and oedema of the vessel walls, and microhaemorrhages are highly specific histological findings in equine pastern vasculitis.

Tkaczyk *et al.* (2013) reported that alpha toxin promotes severe skin lesions and a defect in bacterial clearance and histological examination of the skin lesions found that the mice infected with wild type exhibited extensive necrosis of the skin and subcutaneous fatty tissue, severe muscle necrosis and inflammatory infiltrate, necrotic lesions became large ulcer associated with extensive tissue necrosis and minimal submuscularis inflammatory infiltrate, granulation tissue formation and visible bacteria.

Hassaneen *et al.* (2015) reported that pathologically the udder of she camel with *staphylococci* showed oedema with abscess grossly. Histopathological revealed pus, necrotic debris and inflammatory cells in the dermis.

Jubb *et al.* (2015) classified various forms of dermatitis on histological basis and reported as Perivascular, interface, Vasculitis, nodular and diffuse, Intraepidermal, Subepidermal vesicular and pustular, Perifolliculitis, folliculitis and furunculosis, Panniculitis, fibrosing, ulcerative and Seborrheic dermatitis.

Kobayashi *et al.* (2015) reported that *Staphylococcus aureus* causes many types of human infections and syndromes most notably skin and soft tissue infections. Abscesses are a frequent manifestation of *S. aureus* skin and soft tissue infections and are formed, in part, to contain the nidus of infection. Polymorphonuclear leukocytes (neutrophils) are the primary cellular host defence against *S. aureus* infections and a major component of *S. aureus* abscesses. These host cells contain and produce many antimicrobial agents that are effective at killing bacteria, but can also cause non-specific damage to host tissues and contribute to the formation of abscesses. By comparison, *S. aureus* produces several molecules that also contribute to the formation of abscesses. Such molecules include those that recruit neutrophils; causes host cell lysis, and are involved in the formation of the fibrin capsule surrounding the abscess. They review the current knowledge of the mechanisms and processes underlying the formation of *S. aureus* abscesses, including the involvement of polymorphonuclear leukocytes, and provide a brief overview of therapeutic approaches.

## **2.2 BACTERIOLOGICAL STUDIES**

Yadav *et al.* (1971) isolated staphylococci from bovine udder and they found all the strains to produce both alpha and beta haemolysin, the former being in lesser amounts.

Arzo (1973) isolated *S. aureus* from skin lesion of camels produced golden yellow and white colonies on milk salt agar and nutrient agar.

El-Seedy *et al.* (1990) described fistulus wither in 93 working camel. Eighty seven samples were bacteriologically positive for *staphylococcus aureus*, *Corynebacterium pyogenes*, *C. ovis* and *Escherichia sp.*, predominantly. *Proteus vulgaris* and *Klebsiella sp.* were also isolated sporadically. Among the strict anaerobic organism

recovered from cases of fistulus withers were *Clostridium perfringens* and *Fusobacterium necrophorum*.

Ismail *et al.* (1990) isolated a variety of bacteria from closed abscesses on the thoracic regions, shoulder, abdomen, head and limbs of one humped camels. *Staphylococcus aureus*, *Corynebacterium pyogenes*, *C. pseudotuberculosis*, *Streptococcus pyogenes*, *E. coli*, *Klebsiella sp.*, *Proteus vulgaris*, *Proteus mirabilis*, *Pseudomonas aeruginosa*, *Clostridium perfringens* and *Fusobacterium necrophorum*.

Quinn *et al.* (1994) recorded difference in colony pigmentation of *S. aureus* from cattle, human and other domestic animals. The coagulase positive *S. aureus* has been recorded to be the most common cause of human and animal's *staphylococcal* infection viz. abscess formation, suppuration, botryomycosis, folliculitis, furuncles, cellulitis scaled skin syndrome and postoperative wound infections of various sites.

Yagoub and Mohamed (1996) investigated incidence, clinical observations and aetiology of contagious skin necrosis in Sudan. The size of lesion varies from 5 to 7 cm in diameter and was distributed to neck, flank, head, tail, abdomen and hind limb. Bacteria isolated include *Staphylococcus sp.* (30 per cent), *Corynebacterium pyogenes* (11.8 per cent), *Lactobacillus* (11.0 per cent), *Escherichia coli* (5.1 per cent), *Bacillus sp.* (10 per cent), *Micrococcus sp.* (9.3 per cent), *Proteus sp.* (4.1 per cent), *Streptococcus sp.* (3.61 per cent), *Nocardia sp.* (1.8 per cent), *Erysipelothrix sp.* (1.2 per cent), *Actinomyces sp.* (1.2 per cent), *Aerococcus sp.* (1.0 per cent), *Pasturella sp.* (1.0 per cent), *Actinobacter sp.* (0.7 per cent) and *Aeromonas sp.* (0.7 per cent). The predominant bacterial species was *Staphylococcus sp.* either as pure isolate or in mixed culture.

Qureshi (2002) reported that all of the isolates of *S. aureus* strains obtained from camel skin abscesses and wounds produced complete, partial or both types of haemolysis on sheep blood agar.

Tadesse and Molla (2002) studied bacterial pathogens associated with abscesses and cutaneous wounds of camels. Out of 105 swab sample they found *Corynebacterium pseudotuberculosis*, *Streptococcus agalactiae*, *Staphylococcus aureus* and *Corynebacterium pyogenes* were the major isolates.

Solanki (2004) investigated that twenty one *S. aureus* isolates from camel skin wounds and abscesses showed complete haemolysis, 13 isolates exhibited incomplete haemolysis and 12 isolates did not show any haemolysis. None of the isolates showed both types of haemolysis.

Abdalla and Salim (2008) isolated bacteria from 40 samples of contagious skin necrosis in camels from Alshowak and El Gadaref areas, El Gadaref State, Sudan. The percentage and frequency of isolation of various bacterial genera and species was recorded. Thirteen species were identified that belonged to eight different genera. The dominant bacterial species encountered in contagious skin necrosis was *Staphylococcus aureus* (32.36 per cent) present either as a single isolate or mixed, in variable frequencies, with other bacterial species that belonged to the genera, *Actinomyces*, *Bacillus*, *Corynebacterium*, *Enterobacter*, *Escherichia*, *Pseudomonas*, *Salmonella* and *Staphylococcus*.

Kalka-Moll *et al* (2008) isolated *S. aureus* from subcutaneous abscesses in human.

Qureshi and Kataria (2012) characterized 40 *S. aureus* from camel wound samples and recorded 15 isolates to produce golden yellow, 24 to produce yellow and one to produce white colony

Sun *et al.* (2012) reported that yellow to orange colony colour of *S. aureus* was one of the classical criteria for identification of the species. However, the pigmentation of *S. aureus* was not a very stable character. In this study, *S. aureus* grown in biofilm pattern was nearly

colorless. Meanwhile, *S. aureus* grown in anaerobic and aerobic patterns was colorless and yellow, respectively.

Ajayi *et al.* (2013) reported that samples were inoculated on Mannitol Salt agar (MSA) and incubated at 37°C for 24h and 48h. Colonies which appeared yellow on MSA were subjected for further tests. Examination indicated that the organism was Gram positive after the Grams staining technique and that they appeared in clusters and spherical (cocci) in shape under the microscope.

Valarmath *et al.* (2013) collected different types of wounds samples from 19 patients during March-April 2011 in Namakkal, Tamil Nadu, India. Four types of bacterial species were isolated and identified by selective culture medium and standard biochemical tests from the collected wound samples. Each wound samples showed one or more bacterial isolate, totally 78.9% of samples exhibited 24 isolates. Among them, *Staphylococcus aureus* (54.1%) was the predominant isolate, second most was *Klebsiella pneumoniae* (20.8%) followed by *Pseudomonas aeruginosa* (16.6%) and the lowest percentage was recorded by *Escherichia coli* (8.3%). Highest occurrence was observed in burn wound samples (44%) and second most is the accident and post-operative wound samples (25%) followed by skin wound samples (24%).

Habib *et al.* (2015) characterized *S. aureus* by cultural and biochemical properties isolated from various sources such as blood, pus, milk, secretions, discharge, injuries, cuts, surgical and nonsurgical wound of sheep, goat, dogs, buffaloes, camels, horses, cattle, poultry birds and humans. The species did not vary in their morphological, staining and cultural characteristics and showed similar characteristics on different culture media through it was isolated from different species.

Tiwari *et al.* (2015) isolated microflora associated with wound and skin affections on cattle, buffalo, dogs, goats, sheep, camel and

horses skin. The most abundant bacterial species encountered in wound was *Staphylococcus aureus* (36.22 per cent) present either as pure isolate or mixed with other bacteria.

### 2.3 Genotypic identification

Aarestrup *et al.* (1999) studied that genotypically, the gene encoding  $\alpha$ -haemolysin was detected in all isolates from human and bovine sources, and found a significant difference in the prevalence of the gene encoding  $\beta$ -haemolysin between the bovine (96%), human carrier (56%) and isolates from septicaemia (57%).

Booth *et al.* (2001) suggested that all the 128 *S. aureus* isolates from various sites of infections showed presence of *hla* gene giving 534 bp product size amplicon.

Salasia *et al.* (2011) reported that most of the *S. aureus* isolated from bovines (84%) and humans (73%) harboured the *hla* gene.

Singh *et al.* (2011) identified 107 *S. aureus* isolates from sahiwal, 128 isolates from Karan fries and 111 isolates from Murrah buffalo and further checked the prevalence of toxic genes. The *hla* gene responsible for alpha haemolysis was found in all the isolates with 550 bp amplicon size.

Rathore (2012) isolated 15 *S. aureus* from camel wound samples and subjected them to PCR amplification targeting 23S rRNA gene using species specific primers for genotypic confirmation. With the PCR, a species specific amplicon of 1250 bp was produced by all the isolates.

Hassuny (2014) isolated 18 *S. aureus* from human throat swabs of which only four isolates had *hla* gene. Yadav *et al.* (2015) reported that the prevalence of *hla* gene was found in 96.8% out of 32 isolates in both cattle and buffalo isolates with a single amplicon of 534 bp. Yu *et al.* (2015) detected *hla* gene in 95.3% isolates of 128 *S. aureus* from human patients with skin and soft tissue infections. Dhirendra (2016)

reported the isolation of *S. aureus* from camel skin wound sample and subjected them to PCR amplification targeting 23SrRNA gene using species specific primer for genotypic confirmation.

Sharma *et al.* (2016) described that a total 102 *S. aureus* isolates were obtained from different sources and identified with conventional methods and later confirmed genotypically using species specific primer for ribotyping based on 23SrRNA gene.

### 3. MATERIALS AND METHODS

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For the present study, skin portions showing frank lesions were examined to identify the various skin problems commonly prevailing in camels. The samples were examined for histopathological and bacteriological aspects. Details of cases taken for the investigation, processing of tissue showing frank lesions and bacteriological studies have been presented.

#### **3.1 Cases taken for investigation**

Camel of either sex, different age and breeds reported to various clinics at Bikaner, Jodhpur, Jaisalmer district and adjacent areas were taken for the present study.

#### **3.2 Bacteriological sampling and isolation**

##### **A. Bacteriological sampling in cases of superficial lesions**

Top of pustule/papule or crust was lifted with a sterile 25 gauge needle. A swab was gently pressed on the exposed surface and allowed time for the contents to be absorbed. Then, swab was placed in sterilized nutrient broth medium for further processing.

##### **B. Bacteriological sampling in cases of deep lesions**

The surface of the affected area was clipped and disinfected with 70 percent ethanol and allowed to dry. The area was then gently squeezed to express exudates and then sampled with swab.

##### **C. Bacterial sampling in under scribed lesions**

These were obtained by biopsy and transferred into sterile transport medium rather in 10 per cent formalin.

### **3.3 Bacteriological Media**

#### **Mannitol salt agar medium**

To prepare the medium 1g of beef extract, 10 g of peptone, 75 g of sodium chloride and 0.025 g of phenol red were dissolved in 700 ml of double glass distilled water and pH of the medium was adjusted to 7.4. Agar-agar type 1 was added at the rate of 2% for final volume and volume of the medium was made to 800 ml, and autoclaved at 121°C for 15 min. 200 ml of 5% D-mannitol solution prepared in glass distilled water and sterilized separately at 10 lbs pressure for 15 min, was added to the basal medium before pouring in sterilized petri dishes.

### **3.4 Sample Collection**

In the present investigation, total 95 skin samples of camel were collected for histopathological in 10% formal saline and for bacteriological investigation with sterile absorbent swabs soaked in nutrient broth.

### **3.5 Bacterial isolation**

#### **Genotyping**

##### **(A) 23SrRNA gene based identification (Ribotyping)**

The ribotyping for 23SrRNA was carried out as per the method described by Straub *et al.* (1999). The forward and reverse primer used are given:

Primer 1 – 5' ACGGAGTTACAAAGGACGAC 3'

Primer 2 – 5' AGCTCAGCCTTAACGAGTAC 3'

#### **Preparation of bacterial cell culture**

a. Samples collected for bacterial isolation were grown overnight in 25 ml nutrient broth in shaker incubator at 37°C.

b. The bacterial culture was then centrifuged at 15000 rpm for 15min to obtain pellet and supernatant was discarded.

## Isolation of DNA

DNA isolation was carried out as per the method of Nachimuthu *et al.* (2001) with some modifications.

- a. Bacterial cultures were pelleted and washed two times with 500 $\mu$ L PBS and centrifuged 5000 rpm for 5 min.
- b. Final cell pellet was resuspended in 1 ml of Tris-EDTA (TE) solution.
- c. 100 $\mu$ l of lysozyme solution (conc. 3 mg/ml) was added and mixture was then incubated at 37°C in water bath for 15 min.
- d. 100 $\mu$ l of 10% SDS solution and 2  $\mu$ l of Proteinase K (10 mg/ml) solution was added and incubated at 60°C in water bath for 1 h with gentle mixing of the contents at every 10 min interval.
- e. After 1 h of incubation 0.75 ml of DNA extraction buffer was added and further incubated for 30 min at 60°C in water bath.
- f. Phenol: Chloroform: Isoamyl alcohol mixture (25:24:1) was prepared and 0.5 ml of it was added to the DNA preparation and mixed gently for about 10-15 min.
- g. Above mixture was centrifuged at 15000 rpm for 15 min at 20°C and upper aqueous phase containing DNA was transferred to another tube.
- h. To this mixture, 0.5 ml of cold isopropanol was added and the tube was replaced in ice for 15 min and then centrifuged at 15000 rpm for 10 min at 20°C.
- i. The supernatant was discarded and the pellet was dissolved in 0.5 ml of cold 70% ethanol and centrifuged at 10000 rpm for 10 min at 20°C.
- j. Supernatant was discarded and the tubes were inverted on a filter paper for 5 min. The tubes were then covered with

parafilm with a small hole in it and left at room temperature for overnight to allow evaporation of alcohol

- k. Next morning the pellet was redissolved in 50µl of TE buffer and left for 24 h for dissolution of the pellet.

#### **Agarose gel electrophoresis:**

Agarose gel electrophoresis was carried out in a horizontal, submerged electrophoresis unit analysis to check the integrity of DNA. Each DNA sample was mixed with one-fifth volume of the gel loading buffer. For electrophoresis, 0.8 per cent agarose gel in TBE buffer containing ethidium bromide (0.5-1 µg) was used.

After the addition of tracking dye (bromophenol blue), each sample was loaded in the well of gel and electrophoresis was carried out at 100 V at room temperature for about 1-2 h depending upon the length of the gel or till the dye migrated more than half of the length of the gel. At the end of the electrophoresis, the gel was visualized under UV transilluminator and photographs were obtained.

#### **Amplification of DNA:**

**Table-1:** The reaction mixture (total volume 25 µl)

De-ionised water	11.3 µl
10x Taq Buffer A containing	5 µl
MgCl <sub>2</sub>	3.0 µl
dNTP-mix(10mM)	1.0 µl
Primer-1(10 pM/µl)	1 µl
Primer-2(10 pM/µl)	1 µl
TaqDNA polymerase(5U/µl)	0.2 µl
DNA(25ng/µl)	2.5 µl

**Table-2:** The PCR cycling conditions for 23SrRNA gene.

Cycles	Step	Temperature (°C)	Time
Cycle 1	Denaturation	94	5 min
	Primer annealing	55	30 s
	Primer extension	72	75s
Cycle 2-37	Denaturation	94	40 s
	Primer annealing	55	60 s
	Primer extension	72	75 s
Cycle 38	Denaturation	94	1 min
	Primer annealing	55	1 min
	Final extension	72	3 min

The PCR products, after addition of 2 µl of trekking dye were resolved on 1% agarose gels prepared in 1.0 x TBE buffer containing 0.5 µg/ml of ethidium bromide and 250 bp marker was used as molecular marker. The amplification products were electrophoresed for 1 h 30 min at 100 V. The gel was then visualized under U.V. transilluminator.

### **(B) *hla* gene amplification**

The method of Booth *et al.* (2001) was used for the amplification of *hla* gene following the protocol as mentioned below.

The following sequences for two primers were used:

Primer 1- 5' GGT TTA GCC TGG CCT TC 3'

Primer 2- 5' CAT CAC GAA CTC GTT CG 3'

**Table-3:** The PCR cycling conditions for *hla* gene

Cycles	Step	Temperature (°C)	Time
Cycle1- 30	Denaturation	94	30s
	Primer annealing	53	60 s
	Primer extension	72	30s
Cycle 31	Final extension	72	3min

The PCR products, after addition of 2 µl of trekking dye were resolved in 1% agarose gels prepared in 1.0 x TBE buffer containing 0.5µg/ml of ethidium bromide and 250 bp DNA ladder was used as molecular marker. The amplification products were electrophoresed for 50-60 min at 100 V. The gel was then visualized under U.V. transilluminator.

### **3.6 Histopathological processing of tissue**

The skin portion showing lesions were collected and preserved in 10 per cent formal saline after cutting the affected parts. The parts of affected tissue measured 2-5 mm thickness and presenting the lesions with normal tissue, were used for fixation and further histopathological examination.

For histopathological examination, processing of tissues was done by paraffin embedding using acetone and benzene technique (Lillie, 1965). The tissue sections of 4-6 micron thickness were cut and stained with hematoxylin and eosin staining method as a routine.

## 4. RESULTS

In the present investigation, a total of 95 skin samples of camels of different age groups, sex and breeds were examined for histopathological observations of various skin affections. The various types of dermatitis identified in 67 cases on the basis of their histopathological characteristics. These conditions were as under-

**Table-4, Fig-1: Various Type of Dermatitis in camels.**

S.N.	Conditions	No. of cases	Percentage
<b>1.</b>	<b>Dermatitis</b>	<b>67</b>	<b>70.52</b>
(i)	Perivascular dermatitis	8	11.94
(ii)	Interface dermatitis	5	7.46
(iii)	Vasculitis	03	4.47
(iv)	Nodular dermatitis	03	4.47
(v)	Diffuse dermatitis	6	8.95
(vi)	Intraepidermal vesicular and pustular dermatitis	8	11.94
(vii)	Subepidermal vesicular and pustular dermatitis	7	10.44
(viii)	Panniculitis	04	5.97
(ix)	Perifolliculitis, folliculitis and furunculosis	10	14.92
(x)	Ulcerative dermatitis	04	5.97
(xi)	Interstitial dermatitis	03	4.47
(xii)	Seborrheic	02	2.98
(xiii)	Fibrosing dermatitis	4	5.97
	<b>Total</b>	<b>67</b>	

## **4.1 Pathological studies**

### **(A) Dermatitis**

Dermatitis was recorded to be positive in 67 (70.52 per cent) of the total cases. These were classified on the basis of histopathological characteristic in the following types:

#### **Perivascular Dermatitis**

This condition was recorded in 8 (11.94 per cent) cases.

Grossly, the skin appeared as erythematous, itchy patches and plaques. Sometimes there were presence of vesicles on the legs and trunk region (Fig.2).

Microscopically, the predominant inflammatory reaction is centered around the dermal blood vessels. There is presence of mild to moderate number of polymorphonuclears and mononuclear infiltration around the superficial dermal blood vessels (Figs.3, 4 and 5). There were presence of epidermal hyperplasia and hyperkeratosis in some cases (Fig.6). Some cases revealed varying degrees of angiogenesis and granulation tissue formation along with mononuclear infiltration around the dermal blood vessels (Fig.7). Some cases revealed epidermal hyperplasia typified by marked rete ridge formation (Fig.8).

#### **Interface dermatitis**

This condition was recorded in 5 (7.46 per cent) cases.

Microscopically, in this type of dermatitis, the dermo-epithelial junction showed hydropic degeneration of epidermal basal cells alongwith cellular infiltration predominantly of lymphocytes and plasma cells (Fig.9). There were presence of parakeratosis and perivascular infiltration of lymphocytes (Fig.10).

### **Vasculitis**

This condition was recorded in 3 (4.47 per cent) cases.

Microscopically, there was heavy angiocentric infiltration of neutrophils in and around the blood vessels. The major vascular changes were endothelial swelling (Fig.11). Some cases showed focal necrosis, fibrinoid changes and occlusive thrombi composed of platelets, fibrin, red blood cells and neutrophils. There was also presence of haemorrhages in some cases (Fig.12).

### **Nodular Dermatitis**

This condition was recorded in 3 (4.47per cent) cases.

Grossly, there was presence of single or multiple firm brown nodules with ill defined borders, seen most usually on the extremities and neck region (Fig.13).

Microscopically, there was presence of discrete clusters of inflammatory cells present mainly in the dermis (Fig.14). The cellular infiltration characterized by predominantly neutrophilic, histocytic, eosinophilic or mixed cellular infiltrate. These nodules were usually multiple, large and solitary masses (Figs.15 and 16).

### **Diffuse Dermatitis**

This condition was recorded in 6 (8.95 per cent).

Grossly, there were presence of annular erythrodermic, hypo pigmented scarring alopecia. In some cases, firm papules and nodules were found on the extremities.

Microscopically, diffuse dermatitis denotes cellular infiltration which was densely spread and discrete cellular aggregates were not easily recognized (Fig.17). There were mixed cellular infiltration in most of the cases but in some cases, the predominant infiltrating cells were neutrophils, histocytes and eosinophils (Fig.18). Sometimes there was collagen degeneration in the dermal tissue.

### **Intraepidermal vesicular and pustular Dermatitis**

This condition was recorded in 8 (11.94 per cent) cases.

Microscopically, there were presence of intracellular oedema in the epidermis alongwith ballooning degeneration and acanthosis. The area showed inflammatory infiltration mainly of neutrophils and few eosinophils, the inflammatory cells accumulate in the vesicles (Figs.19 and 20).

### **Subepidermal vesicular and Pustular Dermatitis**

This condition was recorded in 7(10.44 per cent) cases.

Microscopically, this kind of dermatitis revealed dermo-epidermal separation, severe subepidermal oedema and cellular infiltration. There were formation of subepidermal vesicle and pustules due to hydropic degeneration of basal cells. There was also severe intercellular oedema with blowout of basement membrane zone (Fig. 21).

### **Panniculitis**

This condition was recorded in 4(5.97 per cent) cases.

Grossly, there was presence of subcutaneous nodules located on the neck and trunk region has fluctuating swelling in some cases. Some nodules ruptured, from which purulent material was discharged.

Microscopically, there was a circumscribed area of panniculitis in which the cellular infiltrate mainly consists of neutrophils and macrophages (Figs.22 and 23). In some cases, the red blood cells were found in the center of lesions surrounding by inflammatory infiltration and necrotic debris (Fig.24).

### **Perifolliculitis, Folliculitis and Furunculosis**

This condition was recorded in 10 (14.92 per cent)

Grossly, in perifolliculitis a wide spread alopecia on the various parts of the body was found (Fig.25) whereas in folliculitis and furunculosis, erythematous follicular papules were usually seen on abdominal area.

Microscopically, perifolliculitis and luminal folliculitis showing inflammatory infiltration of neutrophils, lymphocytes around the wall and lumen of follicle (Fig.26).

In folliculitis, some cases showed hyperplasia of epidermis and accumulation of inflammatory cells mainly of neutrophils within follicular wall with presence of melanin in hair shaft (Fig.27). Some cases showed luminal folliculitis in which inflammatory cells present in lumen of follicle (Figs.28, 29 and 30). Sometimes there was granulomatous inflammation with suppuration and heavy neutrophilic infiltration (Fig.31).

In furunculosis, there were destruction of majority of follicular epithelium which was destroyed by the inflammatory reaction with release of hair shafts and keratin debris in the dermis. Pyrogranulomatous inflammatory infiltration was also observed in few cases of furunculosis (Figs. 32 and 33).

### **Ulcerative Dermatitis**

This condition was recorded in 4 (5.97 per cent) cases.

Grossly, the lesion represents a typical, solitary and situated at the neck and shoulder area. The area contain brown crusts (Fig.34) which was firmly thickened part of the skin surrounds the ulcer.

Microscopically, there was focal dermoepidermal separation and inflammatory exudate, haemorrhages and lymphocytic infiltration in dermis (Fig. 35). Some cases revealed epidermal hyperplasia and

parakeratosis was common feature of the ulcer edge (Fig. 36). In some cases, layers of inflammatory exudate between collagen bundles and subjacent superficial perivascular lymphocytic infiltration in the dermis were frequently noticed. Both dermis and epidermis were occupied by neutrophils, presence of immature capillaries and some cases showed haemorrhages in the dermis (Fig.37).

### **Interstitial Dermatitis**

This condition was recorded in 3 (4.47 per cent) cases.

Microscopically, interstitial dermatitis was characterized by the infiltration of cells between interstitial spaces of collagen bundles of the dermis (Fig. 38). The infiltrate was poorly circumscribed, mild to moderate in intensity and haemorrhages seen in interstitial space of dermis (Fig.39).

### **Seborrheic Dermatitis**

This condition was recorded in 2 (2.98 per cent) cases.

Grossly, the lesions were reddish brown, scaly plaques having alopecia and follicular cast present on the surface on different parts of the body.

Microscopically, there were hyperplastic superficial dermatitis having usually a marked keratinisation defect characterized by orthokeratotic or parakeratotic hyperkeratosis, follicular keratosis and dyskeratosis (Fig. 40). In most of the cases, the epidermis was normal but in few cases slight increases in thickness of epidermis. Hyperplasia and spongiosis alongwith enlargement of sebaceous glands was observed. The mild perivascular cellular infiltration mainly consists of lymphocytes and plasma cells (Figs. 41 and 42).

## **Fibrosing Dermatitis**

This condition was recorded in 4 (5.97 per cent).

Microscopically, there was extensive fibrosis occurred due to destruction of collagen fibres in the epidermal and dermal tissue (Fig. 43). Some cases revealed extensive fibrosis in dermis along with oedema and infiltration of lymphocytes (Fig. 44).

## **4.2 Bacteriological Studies**

### **Genotypic characterization of *S.aureus*:**

#### **(A) 23SrRNA gene based identification (Ribotypic)**

In the present investigation, out of 95 skin samples of camel, 49 samples showed presence of different Gram's positive bacteria after growth on Mannitol salt agar medium (Figs. 45 and 46). Out of 49 Gram's positive bacteria, only 30 isolates could be confirmed as *S. aureus* through 23SrRNA based ribotyping with an amplicon of 1250 bp. (Table: 5 and 6, Figs. 47 and 48).

**Table-5 Site wise number of isolates used for 23SrRNA based ribotyping**

S.No.	Site	No. of sample	No. of isolates obtained (code numbers)
1.	Neck	3	2(1,3)
2	Chest pad	4	3(7,15,24)
3.	Tail	10	6(2,9,23,30,39,43)
4.	Abdomen	3	1 (35)
5.	Nostril	4	2 (27,41)
6.	Hump	10	6 (5,19,25,37,40,45)
7.	Fore Legs	7	4(4,11,33,43)
8	Hind Legs	8	6(6,8,13,17,22,31)

**Table-6: Isolates confined to be *S.aureus* based on 23SrRNA based ribotyping**

S.No	Gene type	Isolate numbers	Total isolates	Amplicon Size(bp)
1	23SrRNA	1,2,3,4,5,6,7,8,9,11,13,15,17,19,22,23,24,25,27,30,31,33,35,37,39,40,41,43,45,48	30	1250

**(B) *hla* gene :**

Production of Dermonecrotic, haemolysin  $\alpha$  toxin is governed by *hla* gene, and is important virulence factor in *S.aureus*. It is a cytolytic pore forming toxin. Hla toxin not only lyses cells but suppresses host immune response also. In the present investigation, all the confirmed 30 *S. aureus* strains isolated from camel were subjected to PCR amplification of these *hla* haemolytic and dermonecrotic genes. In our finding of the 30 isolates, 28 (93.33%) produced amplicons of 534 bp and had *hla* gene, two isolates were found negative for *hla* gene. (Table-7 and Figs.49 and 50).

**Table-7: Isolates having Alpha toxin gene (*hla*) in *S.aureus* isolated from camel with skin lesions**

<b>S.No</b>	<b>Gene type</b>	<b>Isolate numbers</b>	<b>Total isolates</b>	<b>Amplicon Size(bp)</b>
1	<i>hla</i>	1,3,4,5,6,7,8,9,11,13,15,17,19,22,23,24,27,30,31,33,35,37,39,40,41,43,45,48	28	534

**Table-8 Pathological changes of skin lesions of camel (*Camelus dromedarius*) in relation to *hla* gene**

<b>30 <i>S.auerus</i> isolate</b>	<b><i>hla</i> gene</b>	<b>Extent of lesion</b>	<b>Necrosis</b>	<b>Granulation Tissue</b>	<b>Inflammatory infiltrate</b>	<b>Epidermal hyperplasia</b>
Sample No1	+	++	++	-	+	-
Sample No2	-	+	-	+++	+++	++
Sample No 3	+	+	+	-	+	-
Sample No 4	+	++	++	-	+	-
Sample No 5	+	+++	+++	-	++	-
Sample No 6	+	++	++	-	+	-
Sample No 7	+	++	++	-	+	-
Sample No 8	+	+	++	-	+	-
Sample No 9	+	+++	+++	-	++	-
Sample No 11	+	++	++	-	+	-

Sample No 13	+	+	+	+	++	-
Sample No 15	+	+	+	-	+	-
Sample No 17	+	+++	+++	-	+	-
Sample No 19	+	+++	+++	-	++	-
Sample No22	+	+	+	+	+	-
Sample No 23	+	++	++	-	++	-
Sample No 24	+	++	++	-	+	-
Sample No 25	-	++	-	+	++	+
Sample No 27	+	++	++	-	+	-
Sample No 30	+	+	+	-	+	-
Sample No 31	+	+++	+++	-	++	-
Sample No 33	+	++	++	-	+	-
Sample No 35	+	++	++	-	+	-

Sample No 37	+	+	+	-	++	-
Sample No 39	+	+	+	-	+	-
Sample No 40	+	+++	+++	-	++	-
Sample No 41	+	++	++	-	+	-
Sample No 43	+	+	+	+	+	-
Sample No 45	+	++	++	-	+	-
Sample No 48	+	+++	+++	-	+	-

(- ) no necrosis, no granulation tissue

(+ ) Mild extent of lesion, Mild necrosis, Mild granulation tissue, Mild inflammatory infiltration

(++) Moderate extent of lesion, Moderate necrosis, Moderate granulation tissue, Moderate inflammatory infiltration

(+++ ) Severe extent of lesion, Severe necrosis, More granulation tissue, Severe inflammatory infiltration

Microscopically, *hla* gene positive *S. aureus* revealed severe necrosis in skin and muscle, minimal inflammatory infiltrate, absence or mild formation of granulation tissue and severe extent of lesions where as mild and moderate extent of lesions, no tissue damage, more infiltration of inflammatory cells and more granulation tissue was observed in tissues infected with *hla* negative *S. aureus* (Figs. 52 and 53) (Figs. 55 and 56) (Table- 8)

## 5. DISCUSSION

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The attempt had been made to identify and characterize the bacteriological and pathological status of various skin conditions in camel. The present investigation was carried out at Bikaner, Jodhpur, and Jaisalmer districts situated in the arid and semiarid region of Rajasthan. In this study, 95 cases of camel suffering from various skin affections were studied in which the maximum incidence of dermatitis was recorded.

The results obtained in this study for various parameters have been discussed in detail as per the classification followed for various skin disorders in the text.

### 5.1 DERMATITIS

The present investigation revealed 70.52 per cent incidence of dermatitis which is very higher as compared to the incidence recorded by various authors *viz.* Semushkin (1968) as 5 to 20 per cent, Kriz (1982) as 28 per cent and Yagoub and Mohammad (1996) as 5.73 per cent whereas, Dashtseren *et al.* (1984) reported upto 80 per cent incidence of dermatitis in camel. The incidences recorded in present investigation appear to be higher which might be due to extreme variation in the environment temperature and climatic conditions responsible for growth of variety of causal agents.

#### (A) Pathological studies

##### Perivascular Dermatitis

The gross lesions in perivascular dermatitis were observed as erythematous itchy patches and plaques in this investigation were in agreement with Zaitoun *et al.* (2000), who noticed wide spread skin eruptions and plaques on the skin of camel. Microscopic findings such as polymorhonuclears and mononuclear leucocytic infiltration around

the blood vessels alongwith epidermal hyperkeratosis and hyperplasia observed, have also been described by Jubb *et al.* (2015) in domestic animals whereas Scott *et al.* (1995) while describing the perivascular dermatitis in dogs, reported the predominant inflammatory reaction centered on superficial and deep dermal blood vessels similar to the described in the present text. The similarity in the findings observed in camel and dogs might be due to presence of thick hair coat on the body which is suitable medium for growth of bacteria. Nagai *et al.* (2000) reported perivascular infiltration of mononuclear cells in the dermis in the cases of dermatitis in dairy cattle and supports well the finding of present investigation.

### **Interface Dermatitis**

The microscopic features such as hydropic degeneration of epidermal basal cells, inflammatory infiltration predominantly of lymphocytes and plasma cells and parakeratosis were observed. The similar lesions were also described by Munz (1992) in a study on infectious skin diseases of camels, Scott *et al.*(1995) also reported the hydropic degeneration of epidermal cells in cases of interface dermatitis in dogs. Joshi (2013) reported vacuolar changes of basal cells with sparse perivascular lymphocytic infiltrate.

### **Vasculitis**

Notherlfer *et al.* (1994) described the vascular changes in the case of dermatitis. The findings observed by the authors such as endothelial swelling, focal necrosis, presence of RBC's and neutrophils are in agreement with results of present study. The similar finding was also described by Jubb *et al.* (2015).

### **Nodular Dermatitis**

The gross changes like nodules on the extremities and neck region observed in positive cases of nodular dermatitis were also reported by Edelsten and Pegram (1974) in camel and Scott *et al.* (1995) in dogs. The histological characteristics like presence of clusters of inflammatory cells mainly in the dermis have also been described by Jubb *et al.* (2015) for nodular dermatitis in domestic animals whereas Scott *et al.* (1995) observed similar finding in dogs for this condition.

### **Diffuse dermatitis**

Scott *et al.* (1995) described the gross and microscopic changes of this type of dermatitis in small animals. Grossly, there was presence of alopecia, papules and nodules on extremities. Microscopically, widely spread cellular infiltration of neutrophils, histocytes, eosinophils and sometimes collagen degeneration in the dermis were in corroboration with the findings recorded in this study. Whereas, extensive infiltration of eosinophils in the cases of dermatitis recorded in present study were akin to the previous report of Veen and Barnes (1978) who also found extensive infiltration of eosinophils in the cases of dermatitis in camel in Nigeria. The climatic condition of the area under study in Nigeria was nearly similar and they confirm the finding of this study.

### **Intraepidermal Vesicular and Pustular Dermatitis**

The histopathological characteristic of this type of dermatitis were identified in the present study, revealed intercellular and intracellular oedema in the epidermis alongwith inflammatory cells predominantly of neutrophils and eosinophils. This has also been explained earlier by Veen and Barnes (1978) in camels. Khanna (2002) also reported hydropic degeneration of basal cells and intercellular oedema in a study of dermatitis in dogs in the same study area. Narnaware *et al.* (2013) revealed hydropic degeneration of

keratinocytes, hyperplasia of epidermis, hyperkeratosis and intraepidermal pustule with mononuclear cellular infiltration.

### **Subepidermal Vesicular and Pustular Dermatitis**

This type of dermatitis revealed dermo-epidermal separation, subepidermal oedema and cellular Infiltration. Ismail *et al.* (1985) observed subepidermal oedema in a study on skin diseases in camels which attest the findings recorded in the present study. Similar observations were also described by Jubb *et al.* (2015) in domestic animals mainly in cattle which also contain thick skin as in case of camel.

### **Panniculitis**

The gross and microscopic alterations observed in this study for panniculitis were in agreement with the description of Jubb *et al.* (2015). Authors reported gross characteristics lesions such as subcutaneous nodules having fluctuating swelling which were observed in this study at neck and trunk region of affected camels. The microscopic features in panniculitis were infiltration of neutrophils and macrophages. Khanna (2002) also observed the areas of panniculitis alongwith infiltration of polymorphonuclears and mononuclear leucocytes in dogs and supports the observation reported in camels.

### **Perifolliculitis, Folliculitis and Furunculosis**

The present study revealed gross changes in the condition as alopecia on the various parts of body alongwith formation of papules. Microscopic alteration observed in perifolliculitis, folliculitis and furunculosis were infiltration around the follicle, in the wall as well as lumen of follicle and rupture of wall with inflammatory infiltration respectively. These gross and microscopic findings have also been explained well by Jubb *et al.* (2015) in large domestic animals and Scott *et al.* (1995) in the small animal dermatological disorders.

### **Ulcerative Dermatitis**

Leese (1927) described ulcer formation in the cases of contagious skin necrosis in camels. The similar dermatological changes were also observed by Veen and Barnes (1978) while studying cutaneous ulcer of camels in Nigeria. Authors further studied the lesions histopathologically and characterized them with collagenous tissue alongwith inflammatory infiltration. In the present study, microscopically, layer of inflammatory exudates between the collagen bundles, neutrophilic and lymphocytic were observed which was in conformity with those described by Veen and Branes (1978). Coigno *et al.* (1985) characterized the lesions as chronic ulcers with dermo-epidermal separation beneath the basal lamina and by hyperkeratosis adjacent to ulcers.

### **Interstitial Dermatitis**

In the present text this condition has been identified on the basis of histological feature characterized by infiltration of cells between interstitial spaces of collagen bundles of the dermis. The similar kind of dermatitis was explained by Scott *et al.* (1995) in the dermatological disorders in small animals and Jubb *et al.* (2015) described interstitial dermatitis in large domestic animals with similar microscopic features as described in the present text.

### **Seborrheic Dermatitis**

The gross characteristics of seborrheic dermatitis as described by Jubb *et al.* (2015) in large domestic animals such as presence of scaly plaques and alopecia, presence of follicular cast on the surface of various parts of body of animals have also been observed in the affected camels included in present investigation. Similarly microscopic features *viz.* keratinization defect, hyperplasia and spongiosis, enlargement of sebaceous glands were observed. These finding are also in line with the finding suggested by Jubb *et al.* (2015). Khanna

(2002) reported that in seborrheic dermatitis, epidermal hyperplasia and spongiosis led to an enlargement of sebaceous glands which confirms the findings of present investigation. Borda *et al.* (2015) described that superficial perivascular and perifollicular inflammatory infiltrates, composed mainly of lymphocytes and histiocytes in association with spongiosis and hyperplasia, and parakeratosis around follicular opening.

### **Fibrosing Dermatitis**

The microscopic features in this condition were fibrosis in the epidermal and dermal tissue along with inflammatory infiltration. These features in fibrosing dermatitis have also been described by Khalafalla (1998) in the dermatitis of camels in Sudan. The proliferation of fibroblasts occurred due to destruction of collagen fibres of dermis by the action of bacterial toxins.

### **(B) Bacteriological Studies**

#### **23SrRNA gene based identification (ribotyping)**

In our study, the overall *S. aureus* prevalence was recorded as 61.22%. Similar genotypic method of *S. aureus* identification have been used by Rathore and Kataria (2012), Ariyanti *et al.* (2011), Dhirenda (2016) for *S. aureus* isolates from elsewhere.

#### ***hla* gene**

In our study, the overall *hla* gene prevalence was recorded as 93.33%. The prevalence of *hla* gene recorded in the present study was similar to observation of Haveri *et al.* (2007) who recorded the prevalence of 97.4% and Yu *et al.* (2015) recorded the prevalence of *hla* gene as 95.3% in *S.aureus*. Likewise, Dhirendra (2016) recorded the prevalence of *hla* gene as 96.15%.

### **Histopathological changes of skin lesions of camel in relation to *hla* gene**

Microscopically, *hla* gene positive *S. aureus* revealed severe necrosis in skin and muscle, minimal inflammatory infiltrate, absence or mild formation of granulation tissue and severe extent of lesions where as mild to moderate extent of lesions, no tissue damage, more infiltration of inflammatory cells and more granulation tissue was observed in tissues infected with *hla* negative *S. aureus*. Similar histopathological finding also reported by Kennedy *et al.* (2010). Tkaczyk *et al.* (2013).

*S. aureus*  $\alpha$ -hemolysin, coded by *hla* gene is a pore-forming cytotoxin and is required for full virulence of a septic wound infection. It has got broad range of cellular specificity and has been recognized as an important cause of injury in the context of both skin necrosis and lethal infection (Powers *et al* 2012 and Berube *et al.* 2013). It is secreted as a water soluble monomer, capable of binding and oligomerization into a heptameric structure on the host cell membrane where A-disintegrin and metalloprotease 10 (ADAM10) act as the cellular receptors for this toxin. The molecular transformation on susceptible host cells culminates by formation of a membrane-perforation 1–3 nm  $\beta$ -hairpin lined amphipathic pore through the eukaryotic lipid bilayer, allowing for the flow of  $\text{Ca}^{2+}$  and  $\text{K}^+$ , ATP through barrel of the pore formation and cellular lysis and tissue barrier disruption at host interfaces lined by epithelial or endothelial cells (Wilki *et al.* 2010, Inoshima *et al.* 2011).

## 6. SUMMARY

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The camel has been a necessity of man-kind in the desert and semi desert areas since its domestication, about 2500 to 3000 years ago. With the rapid advancement of science and technology more mechanization has taken place and the traditional means of transport are being ignored, but the camel is still an indispensable mode of transport in the desert zone.

The camel has a low susceptibility to diseases but skin involvements like contagious skin necrosis, dermatitis, wounds, abscesses or similar problems were commonly observed in camels. The disease caused by *S. aureus* is not always fatal but an indirect great economical loss is incurred due to reduced working efficiency.

The present investigation was carried out from January 2016 to December 2016. During this period a total number of 95 samples of skin lesions in Camel of either sex, different age and breeds were reported for various skin affections at different clinics at Bikaner, Jodhpur, Jaisalmer district and adjacent areas and skin lesions samples were taken for the present study.

Out of 95 samples, 67 (70.52 per cent) cases showed dermatitis. The cases were classified according to gross and histopathological observations for different skin conditions which were also subjected to microbial isolation studies for the purpose of identification of etiological agents as well as to characterize the health status of animals affected of various skin affections.

Dermatitis was further sub-classified on the basis of the histopathological characteristics as perifolliculitis, folliculitis and furunculosis (14.92 per cent), intraepidermal vesicular and pustular dermatitis (11.94 per cent), perivascular dermatitis (11.94 per cent), subepidermal vesicular and pustular dermatitis (10.44 per cent), diffuse

dermatitis (8.95 per cent), interface dermatitis (7.46 per cent), fibrosing dermatitis (5.97 per cent), panniculitis (5.97 per cent), ulcerative dermatitis (5.97 per cent), interstitial dermatitis (4.47 per cent), nodular dermatitis (4.47 per cent), vasculitis (4.47 per cent) and seborrheic (2.98per cent).

Out of 95 skin samples, 49 samples showed presence of different Gram's positive bacteria after growth on Mannitol salt agar medium. Out of 49 Gram's positive bacteria, only 30 (61.22 per cent) isolates could be confirmed as *S. aureus* through 23SrRNA based ribotyping with an amplicon of 1250 bp. All the confirmed 30 *S. aureus* isolates were subjected to PCR amplification of *hla* haemolytic and dermonecrotic gene. In our finding of the 30 isolates, 28 (93.33 per cent) produced amplicons of 534 bp and had *hla* gene, 02 isolates were negative for *hla*.

Histopathologically, *hla* gene positive *S. aureus* found to produced severe necrosis in skin and muscles, minimal inflammatory infiltrate, absence or mild formation of granulation tissue and severe extent of lesions where as mild and moderate extent of lesions, no tissue damage, more infiltration of inflammatory cells and more granulation tissue was observed in tissues infected with *hla* negative *S. aureus*.

In this study, camels were found more prone to perifolliculitis, folliculitis and furunculosis which were recorded in 10 (14.92 per cent) cases followed by intraepidermal vesicular and pustular dermatitis as well as perivascular dermatitis in 8 (11.94 per cent) cases. These conditions were commonly found in camel and the bacteria isolated were *S. aureus* which is a saprophytic bacteria cause's purulent lesion in skin.

Of the total 49 bacteria infected lesions, 30 were infected with *S. aureus*. Amongst the total confirmed *S. aureus* cases most of the

wounds were infected with *hla* (+ve) *S. aureus*. There was difference in the severity of wounds infected with *hla* (+ve) isolates viz a viz *hla* (-ve) isolates and *hla* (+ve) isolates lead to more tissues necrosis as compared to *hla* (-ve) isolates indicating its role in severity of wounds.

## 7. LITERATURE CITED

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# **PATHOLOGICAL CHANGES IN SKIN LESIONS OF CAMEL (*Camelus dromedarius*) IN RELATION TO *Staphylococcus aureus* INFECTION**

**DEPARTMENT OF VETERINARY PATHOLOGY  
COLLEGE OF VETERINARY AND ANIMAL SCIENCE, BIKANER  
RAJASTHAN UNIVERSITY OF VETERINARY AND ANIMAL SCIENCES,  
BIKANER**

**Submitted by:  
Advisor:**

**Chandra Pratap Singh  
Dr. Manisha Mathur**

## **ABSTRACT**

In the present study, a total of 95 skin samples were collected from various district of Rajasthan. Out of 95 skin samples, 67 samples were positive for dermatitis and 49 samples showed presence of different Gram's positive bacteria after growth on Mannitol salt agar medium. Out of 49 Gram's positive bacteria, only 30 (61.22 per cent) isolates could be confirmed as *S. aureus* through 23rRNA based ribotyping with an amplicon of 1250 bp. All the confirmed 30 *S. aureus* isolates from camel were subjected to PCR amplification of *hla* haemolytic and dermonecrotic gene. In our finding of the 30 isolates, 28(93.33 per cent) produced amplicons of 534 bp and had *hla* gene, 02 isolates were negative for *hla* gene.

Pathological studies revealed dermatitis in 67 (70.52 per cent) cases. Various form of dermatitis were observed *viz.* Perivascular dermatitis (11.94 per cent), Interface dermatitis (7.46 per cent), Vasculitis (4.47 per cent), Nodular dermatitis (4.47 per cent), Diffuse dermatitis (8.95 per cent), Intraepidermal vesicular and pustular dermatitis (11.94 per cent), Subepidermal vesicular and pustular dermatitis (10.44 per cent), Panniculitis (5.9 per cent), Perifolliculitis, folliculitis and furunculosis (14.92 per cent), Ulcerative dermatitis (5.97 per cent), Interstitial dermatitis (4.47 per cent), Seborrhic (2.98 per cent), Fibrosing dermatitis (5.97 per cent).

In present study, camels were found more prone to perifolliculitis, folliculitis and furunculosis followed by intraepidermal vesicular and pustular dermatitis, perivascular dermatitis.

Histopathologically, *hla* gene positive *S. aureus* revealed severe necrosis in skin and muscles, minimal inflammatory infiltrate, absence or mild formation of granulation tissue and severe extent of lesions where as mild and moderate extent of lesions, no tissue damage, more infiltration of inflammatory cells and more granulation tissue was observed in tissues infected with *hla* negative *S. aureus* .

# ऊँटों (केमिलस ड्रोमेडिरिस) में त्वचीय विकृतियों का स्टेफाइलोकोकस आरियस संक्रमण से संबंधित व्याधिकीय परिवर्तन

पशु व्याधिकी विभाग  
पशु चिकित्सा एवं पशुविज्ञान महाविद्यालय, बीकानेर  
राजस्थान पशु चिकित्सा एवं पशुविज्ञान विश्वविद्यालय, बीकानेर।

उपस्थापन :  
उपादेष्टा :

चन्द्र प्रताप सिंह  
डॉ. मनीषा माथुर

## अनुक्षेपण

वर्तमान अध्ययन में राजस्थान के विभिन्न जिलों से 95 त्वचीय नमूने एकत्रित कर उनका अध्ययन किया गया। 95 त्वचीय नमूनों में से 67 नमूने त्वक्शोध के लिए सकारात्मक थे तथा 49 नमूनों में मेनीटाल सॉल्ट अगार पर संवर्धन के पश्चात् विभिन्न ग्राम पॉजिटिव बैक्टीरिया की उपस्थिति देखी गई। इन 49 ग्राम पॉजिटिव बैक्टीरिया में से 23 एस.आर.आर. एन.एन. के माध्यम से मात्र 30 (61.22%) एस. ऑरियस आइसोलेट्स की पुष्टि की गई जो 1250 बी.पी. के एक एम्पलीकॉन राइबोटाइपिंग पर आधारित है। ऊँटों से अलग किये गए सभी 30 एस. ऑरियस की पुष्टि पीसीआर प्रवर्धन जो कि एच.एल.ए. रक्त संलायी व त्वक्गलक जीन पर आधारित था, से की गई। प्रस्तुत अन्वेषण में 30 आइसोलेट्स में से 28 (93.33%) से 534 बी.पी. के एम्पीकॉन प्राप्त हुए तथा 2 आइसोलेट्स एच.एल.ए. जीन के लिए नकारात्मक पाए गए।

व्याधिकीय अध्ययन से त्वक्शोध (70.52%) पाया गया। त्वक्शोध के विभिन्न रूप देखे गये जैसे कि बाह्य वाहिन्य शोध (11.94%), इंटसकेस त्वक्शोध (7.46%), वाहिन्य शोध (4.47%), ग्रन्थामय त्वक्शोध (4.47%), प्रस्तुत त्वक्शोध (8.95%), चर्माग्यन्तर पुटिकापन एवं पिड़की (11.94%), अधः त्वचीय पुटिकापन एवं पिड़की (10.44%), पेनीकुलाइटिस (5.97%), बाह्य स्यानिका दोष, स्यूनिका दोष एवं फुरन कुलोसिस (14.92%), व्रणीय त्वक्शोध (5.97%), अन्तशालीय त्वक्शोध (4.47%), त्वक्स्नेही-स्त्राव त्वक्शोध (2.98%), तत्पुरुजा त्वक्शोध (5.97%) पाये गये।

प्रस्तुत अध्ययन में ऊँटों में प्रमुख रूप से बाह्य स्यूनिका दोष, स्यूनिका दोष एवं फुरन कुलोसिस इसके पश्चात् चर्माग्यन्तर पुटिकापन एवं पिड़की, बाह्य वाहिन्य त्वक्शोध पाए गए।

ऊतक व्याधिकीय एच.एल.ए. जीन सकारात्मक एस.आरियस में त्वचा व मॉसपेशिया का गंभीर परिगलन, कम से कम सूजन घुसपैठ, अभाव या दानेदार ऊतक के हल्के गठन, घावों का गंभीर विस्तार जबकि एच.एल.ए. जीन नकारात्मक एस. आरियस में घावों का हल्का व मध्यम विस्तार, ऊतकों में कोई नुकसान नहीं, शोध कोशिकाओं और अधिक दानेदार ऊतक की अधिक घुसपैठ देखा गया।

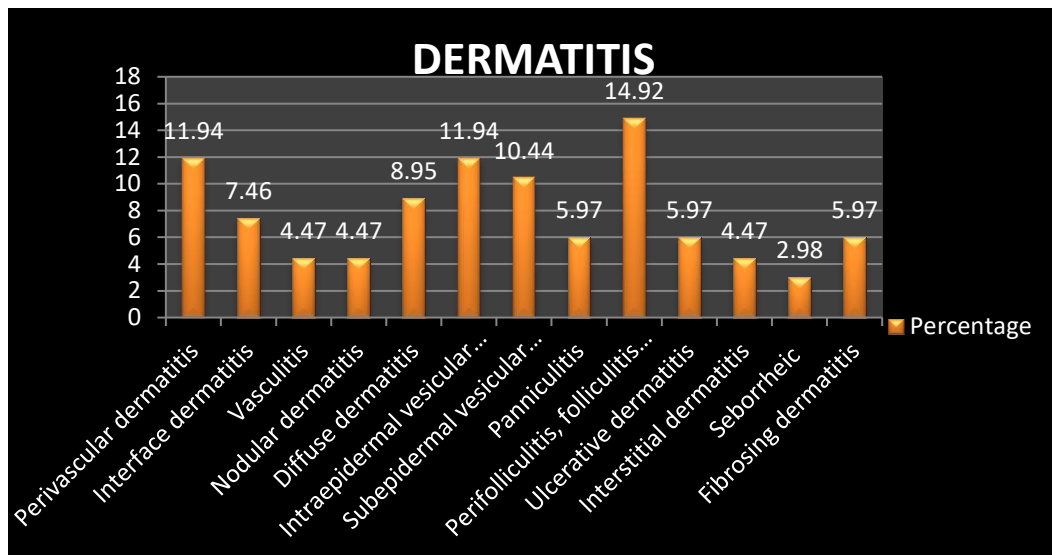


Fig. 1 Photograph showing various type of dermatitis in camels.



Fig. 2 Photograph of Camel having dermatitis showing erythematous patches and plaques on the legs

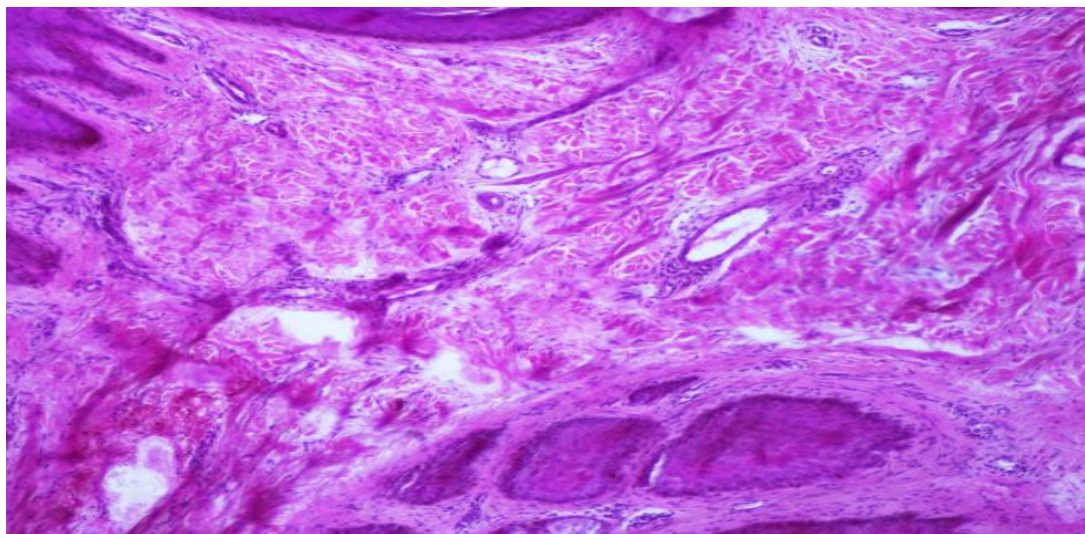


Fig. 3 Microphotograph of skin having perivascular dermatitis showing polymorphonuclear and mononuclear infiltration around the blood vessel. H&E 100X.

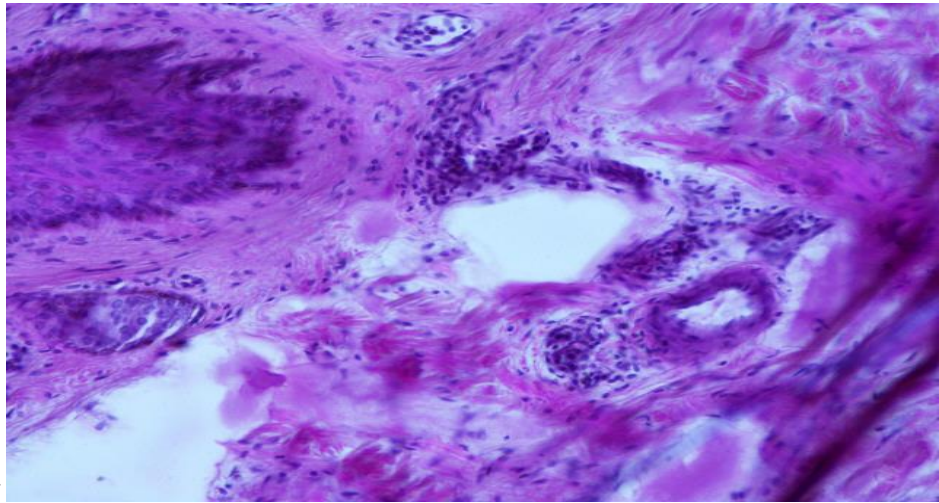


Fig.4 Microphotograph of skin having perivascular dermatitis showing polymorphonuclear and mononuclear infiltration around the blood vessel. H&E 200X.

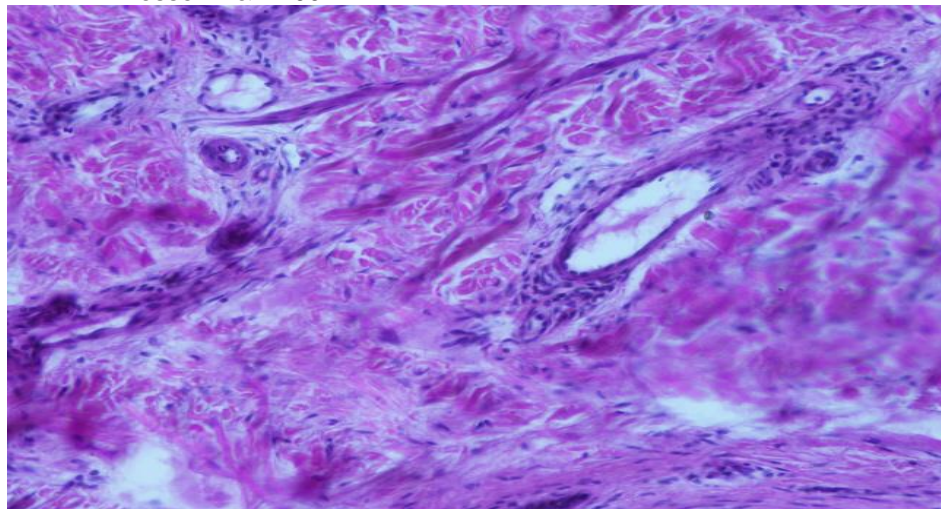


Fig.5 Microphotograph of skin having perivascular dermatitis showing polymorphonuclear and mononuclear infiltration around the blood vessel. H&E 200X.

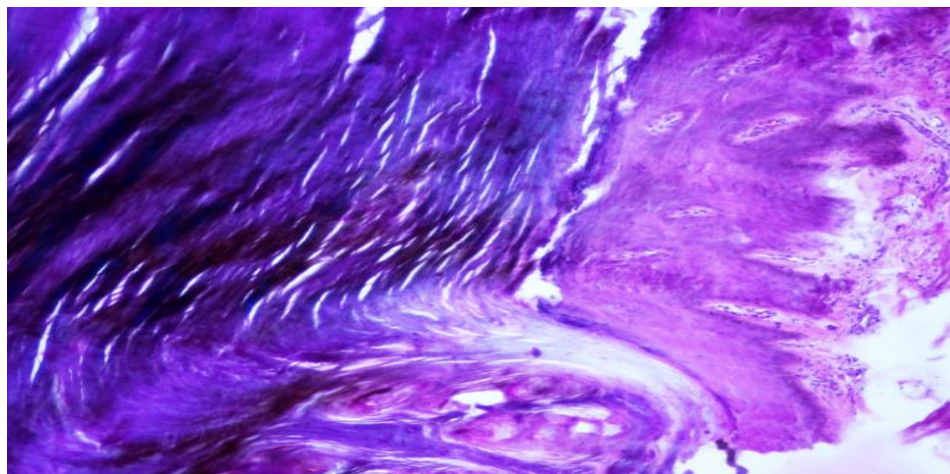


Fig.6 Microphotograph of skin having perivascular dermatitis showing epidermal hyperplasia and hyperkeratosis. H&E 100X.

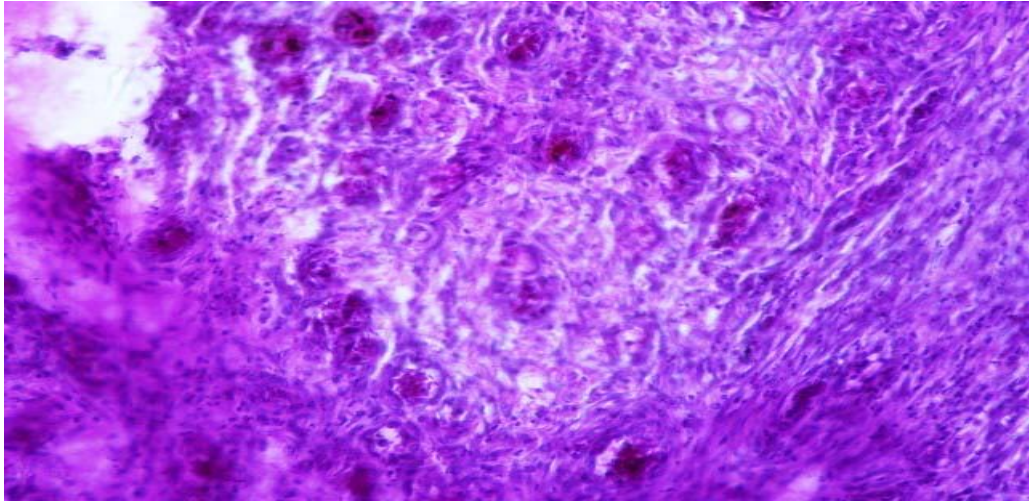


Fig.7 Microphotograph of skin having perivascular dermatitis showing newly formed blood vessels surrounded by inflammatory infiltration. H&E 100X.

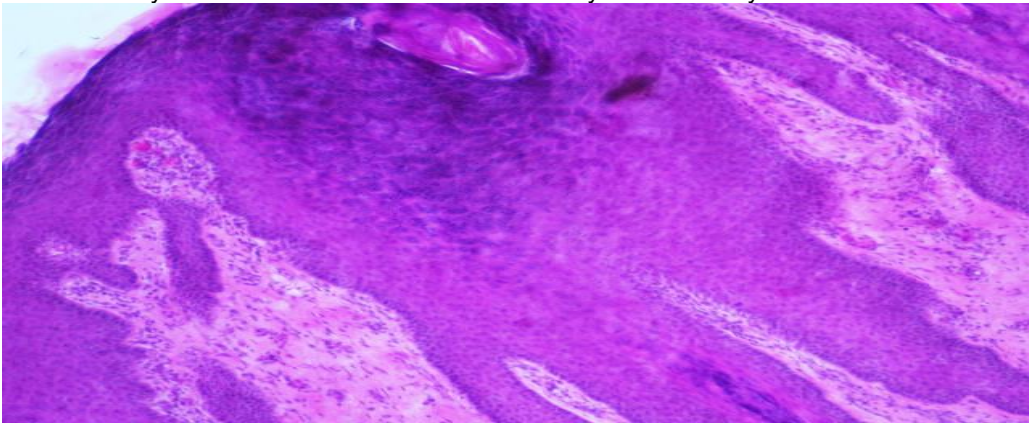


Fig.8 Microphotograph of skin having perivascular dermatitis showing epidermal hyperplasia typified by marked rete ridge formation. H&E 100X.

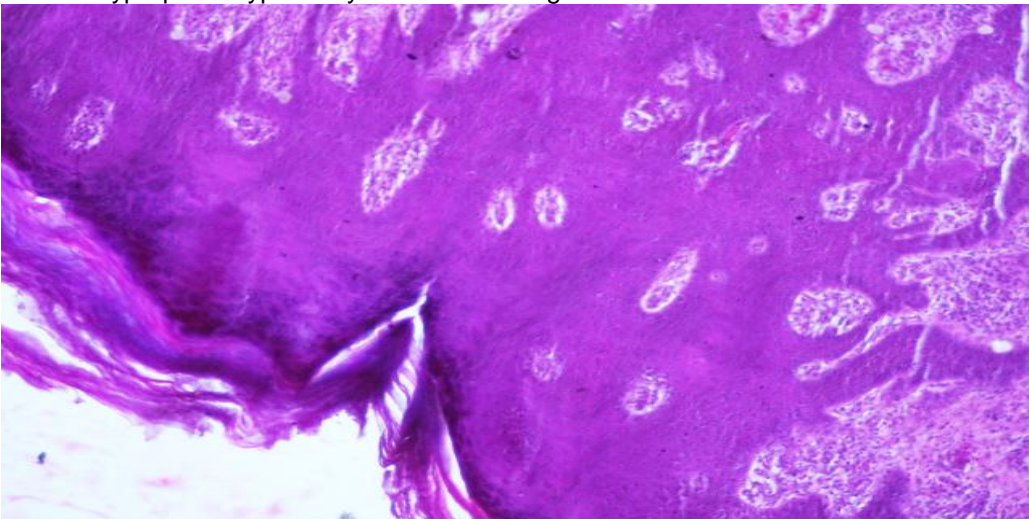


Fig.9 Microphotograph of skin having interface dermatitis showing hydropic degeneration in basal epidermal cell with cellular infiltration. H&E 100X.

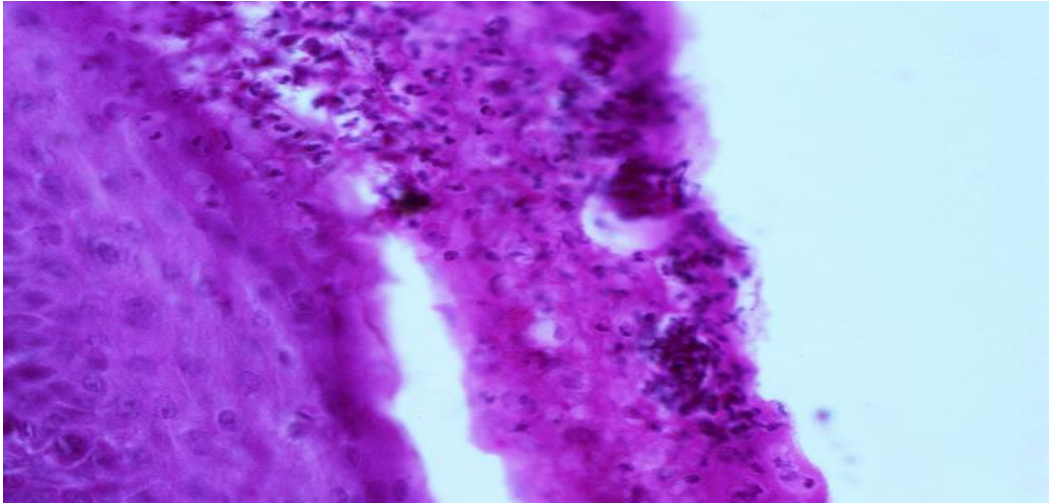


Fig.10 Microphotograph of skin having interface dermatitis showing parakeratosis and infiltration of lymphocytes. H&E 400X.

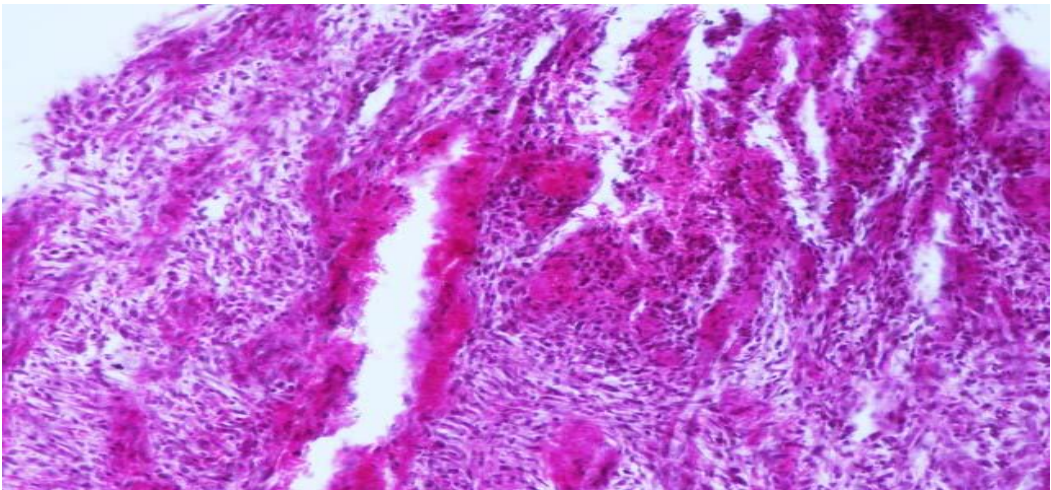


Fig.11 Microphotograph of skin having vasculitis showing inflammatory infiltration around blood vessels along with RBC's and haemorrhages at places. H&E 100X.

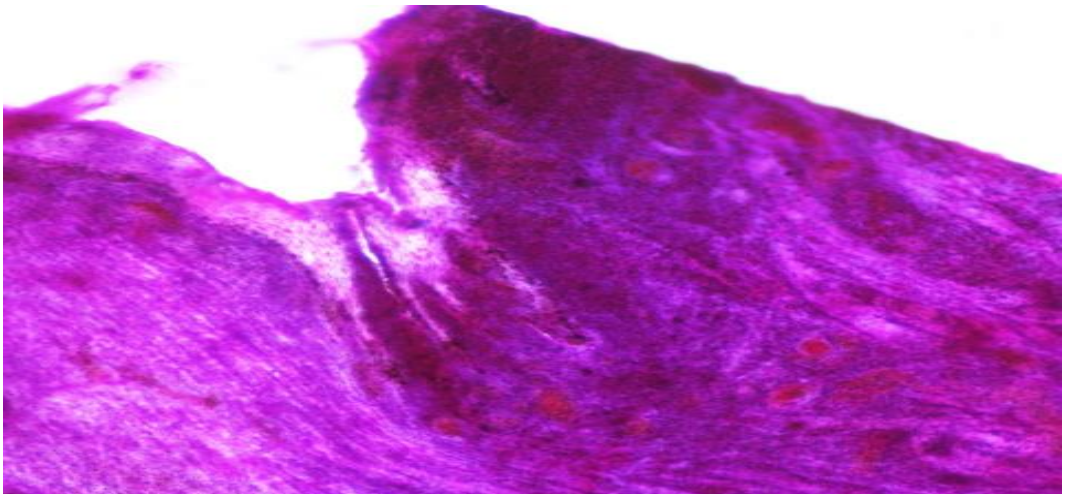


Fig.12 Microphotograph of skin having vasculitis showing focal necrosis and inflammatory infiltration around blood vessels along with RBC's and haemorrhages at places. H&E 100X.



Fig. 13 Photograph of a camel having dermatitis showing firm of nodule of skin.

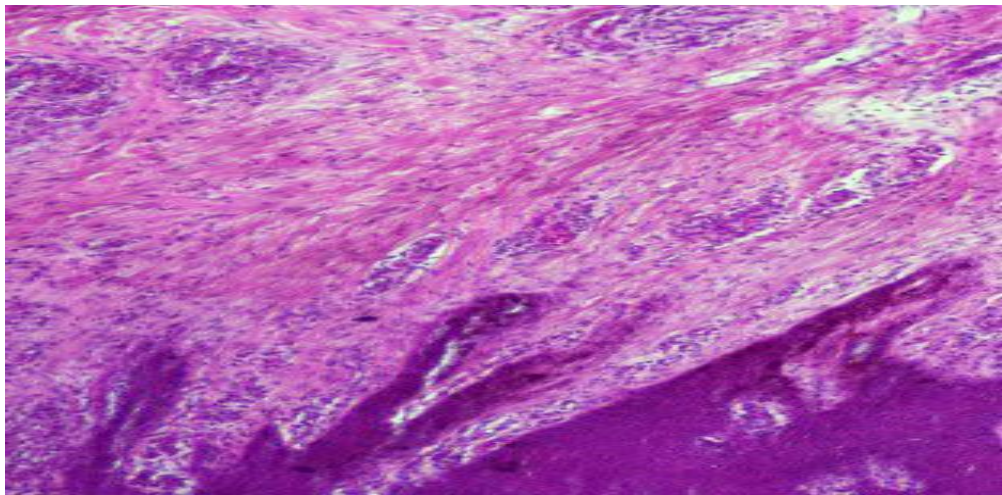


Fig.14 Microphotograph of skin having nodular dermatitis showing nodules of inflammatory cells predominantly of mononuclear in dermis. H&E 100X.

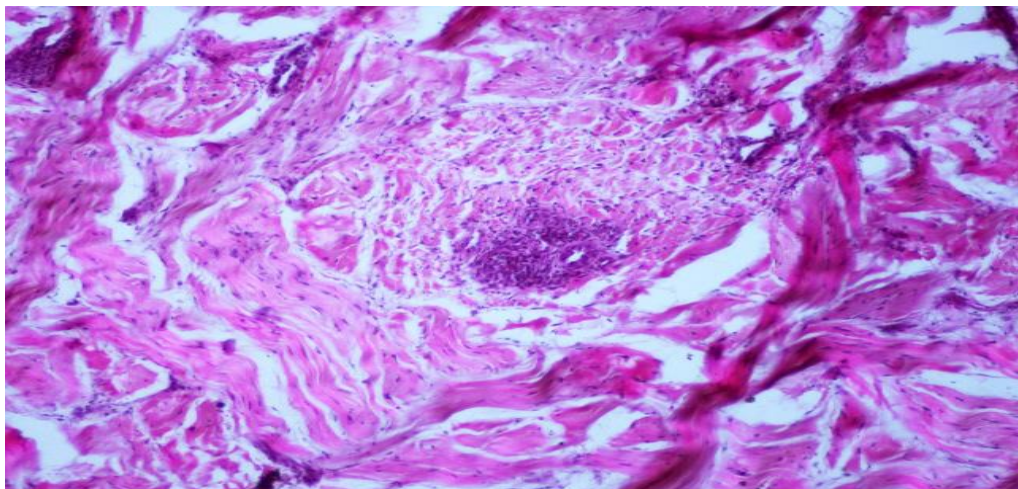


Fig. 15 Microphotograph of skin having nodular dermatitis showing discrete cluster having neutrophils, histocytes and eosinophils in the dermis. H&E 100X.

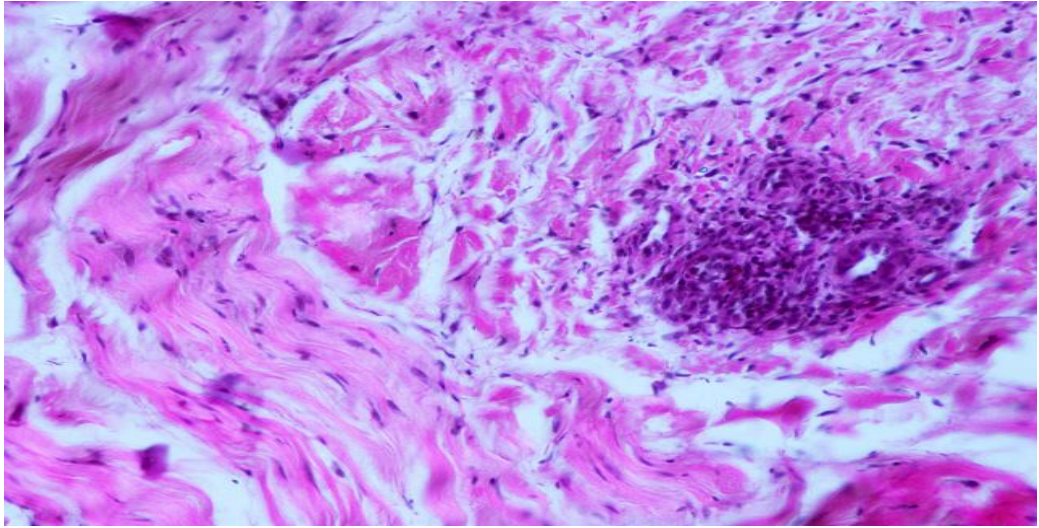


Fig.16 Microphotograph of skin having nodular dermatitis showing discrete cluster having neutrophils, histocytes and eosinophils in the dermis. H&E 200X.

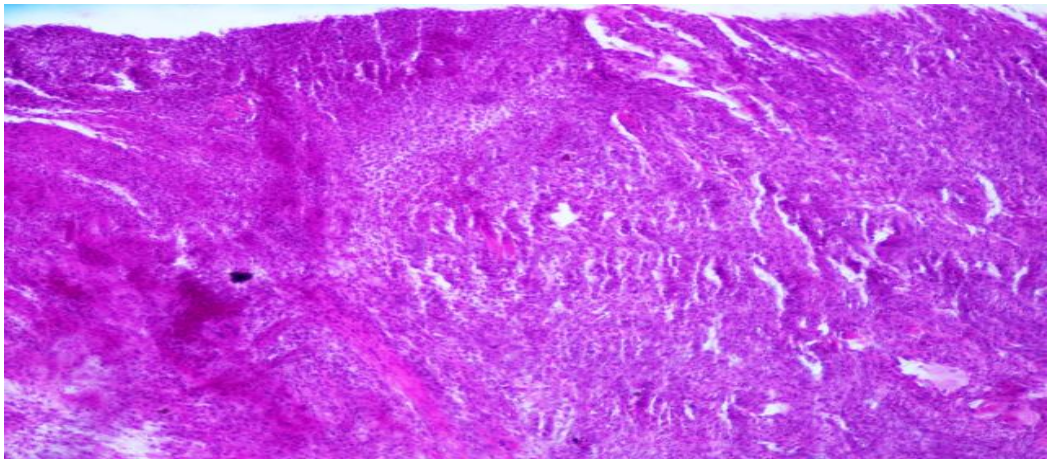


Fig. 17 Microphotograph of skin having diffuse dermatitis showing diffusely spread inflammatory infiltration in the dermis. H& E 100X.

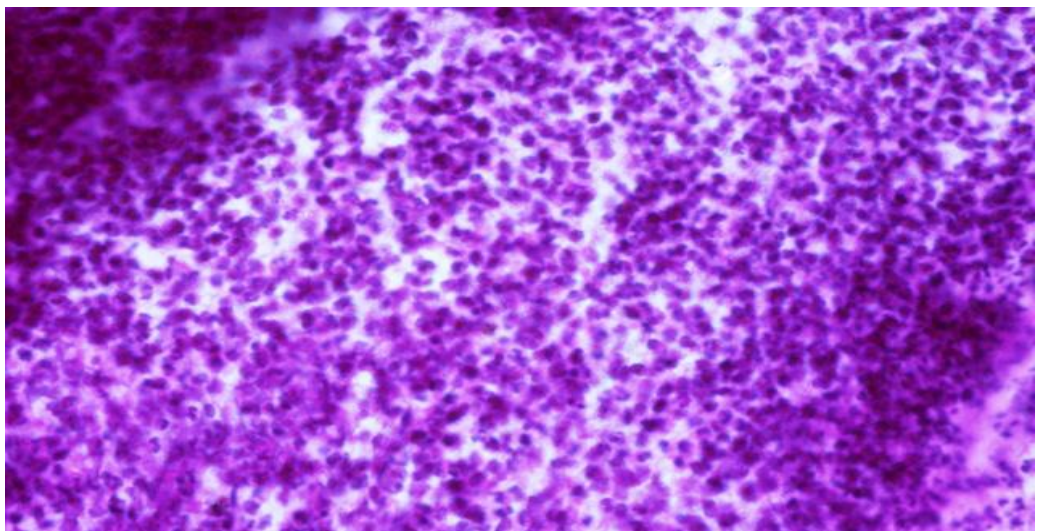


Fig.18 Microphotograph of skin having diffuse dermatitis showing neutrophils and eosinophils in the dermis. H&E 400X.

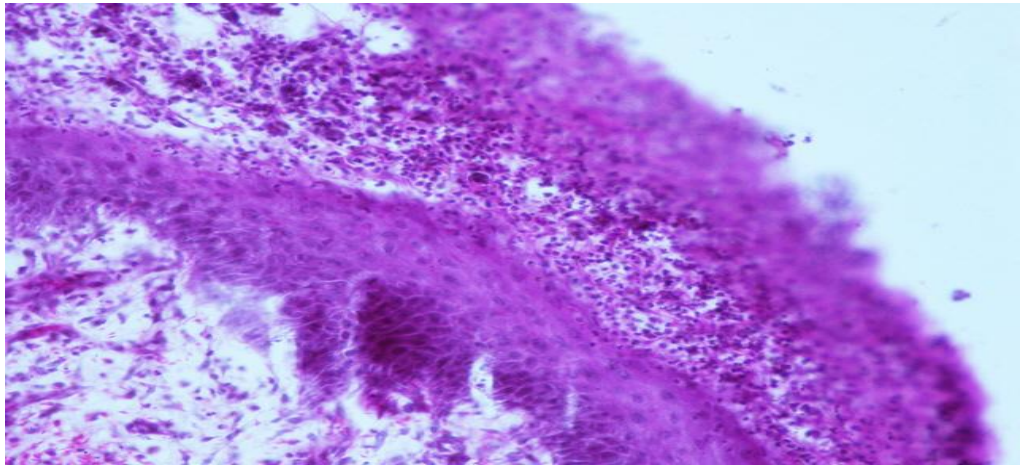


Fig.19 Microphotograph of skin having intraepidermal vesicular and pustular dermatitis showing degenerated inflammatory cells in the vesicles. H&E 200X.

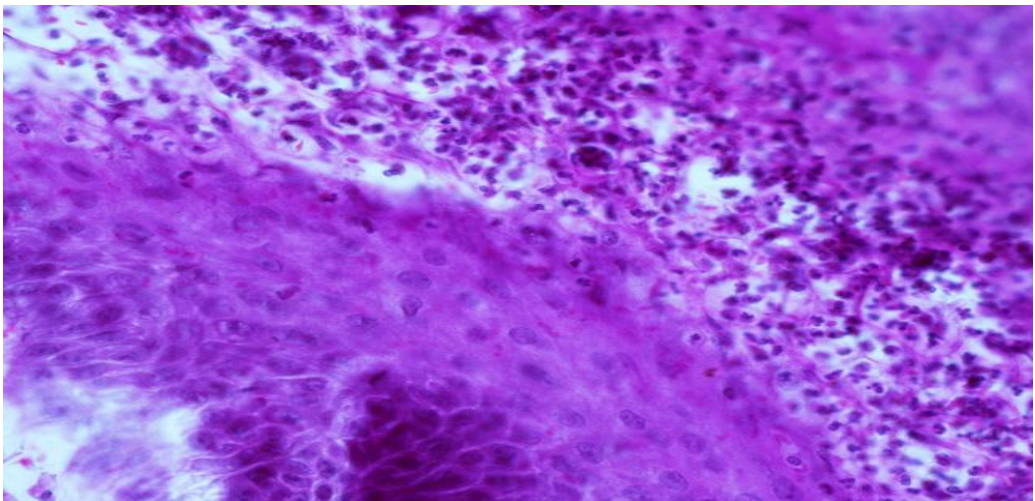


Fig.20 Microphotograph of skin having intraepidermal vesicular and pustular dermatitis showing degenerated inflammatory cells in the vesicles.H&E 400X.

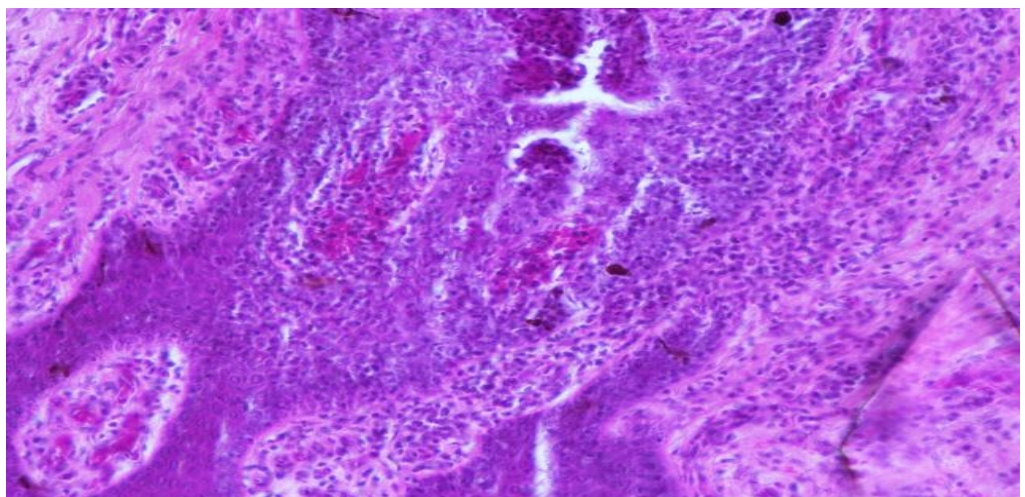


Fig. 21 Microphotograph of skin having subepidermal pustular dermatitis showing severe cellular infiltration. H&E 100X.

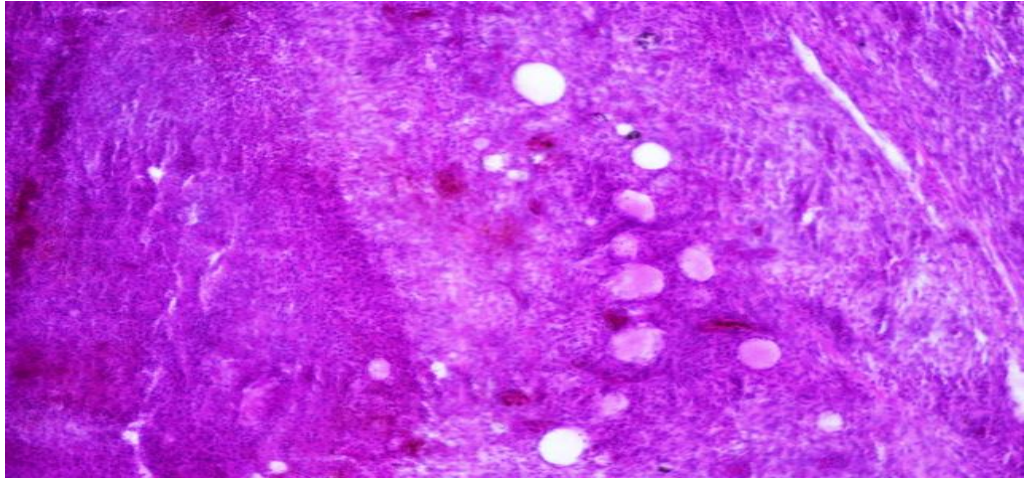


Fig.22 Microphotograph showing areas of panniculitis along with cellular infiltration predominantly of neutrophils and macrophages. H&E 100X.

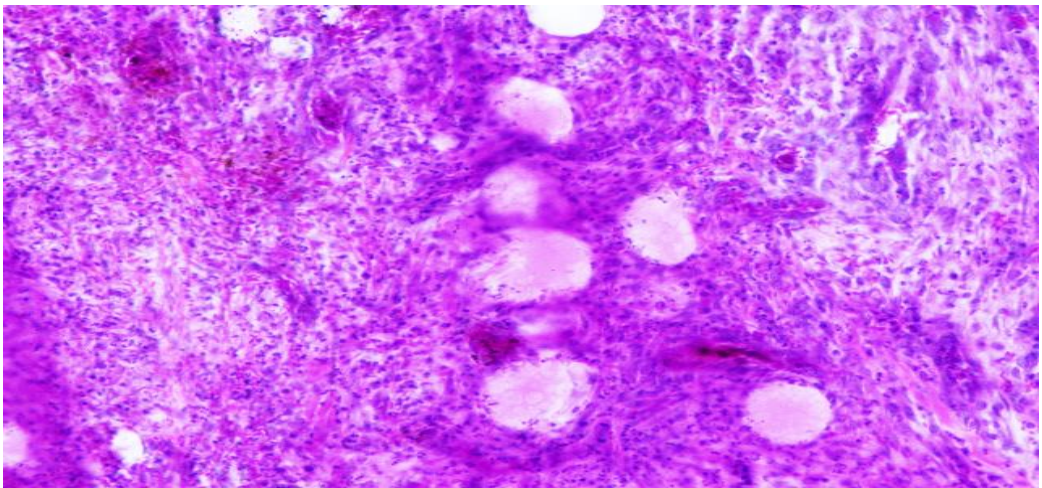


Fig.23 Microphotograph showing areas of panniculitis along with cellular infiltration predominantly of neutrophils and macrophages. H&E 200X.

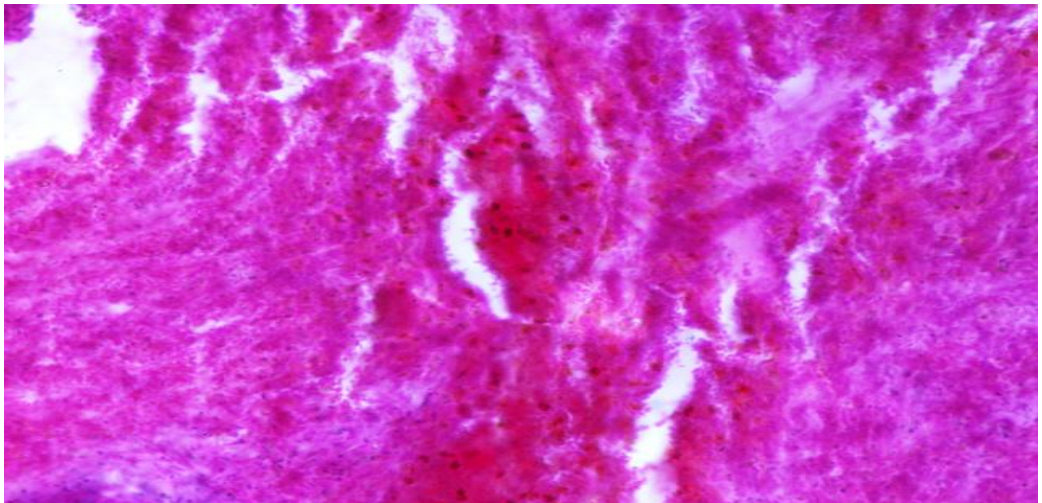


Fig.24 Microphotograph of panniculitis showing RBC's along with inflammatory infiltration and necrotic debris. H&E 200 X.



Fig.25 Photograph of a camel showing wide spread alopecia.

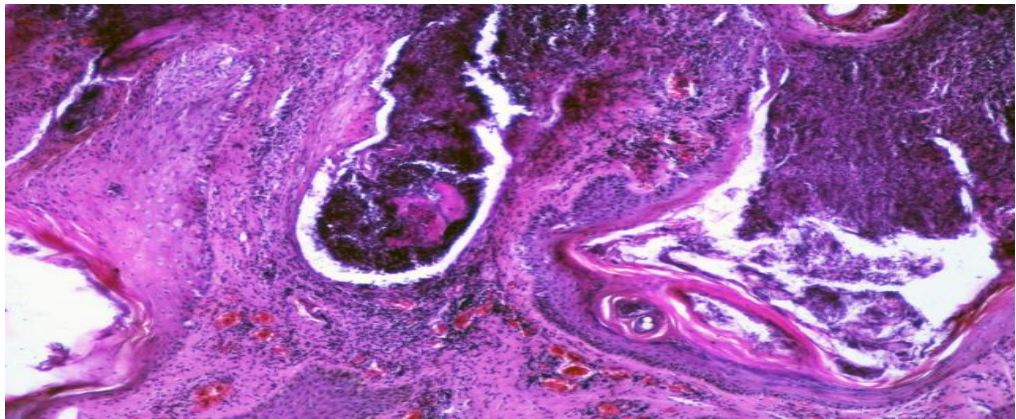


Fig.26 Microphotograph of skin having perfolliculitis and luminal folliculitis showing inflammatory infiltration of neutrophils, lymphocytes around the wall and lumen of follicle. H&E 100X.

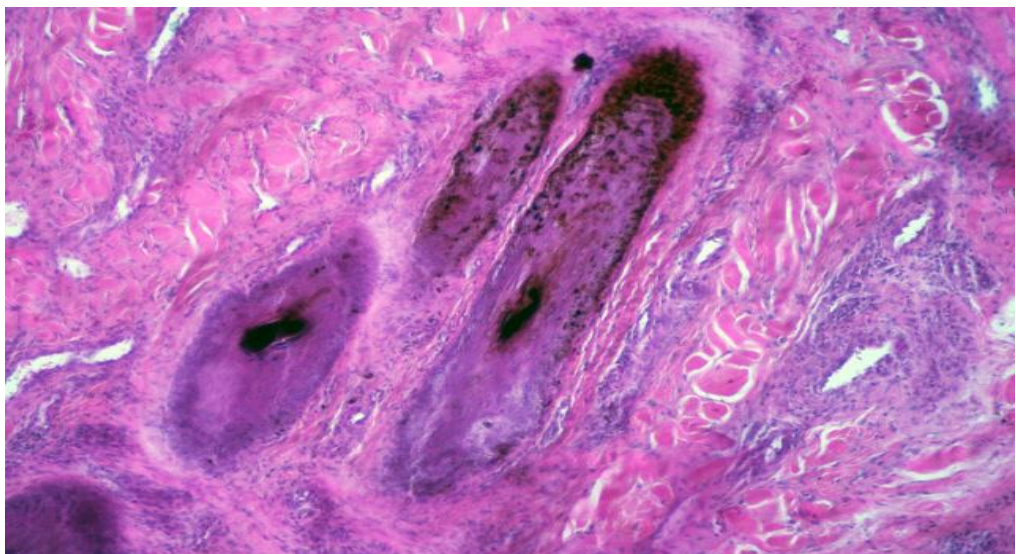


Fig.27 Microphotograph of skin having folliculitis showing inflammatory infiltration of neutrophils and melanin pigment in the wall of follicle. H&E 100X.

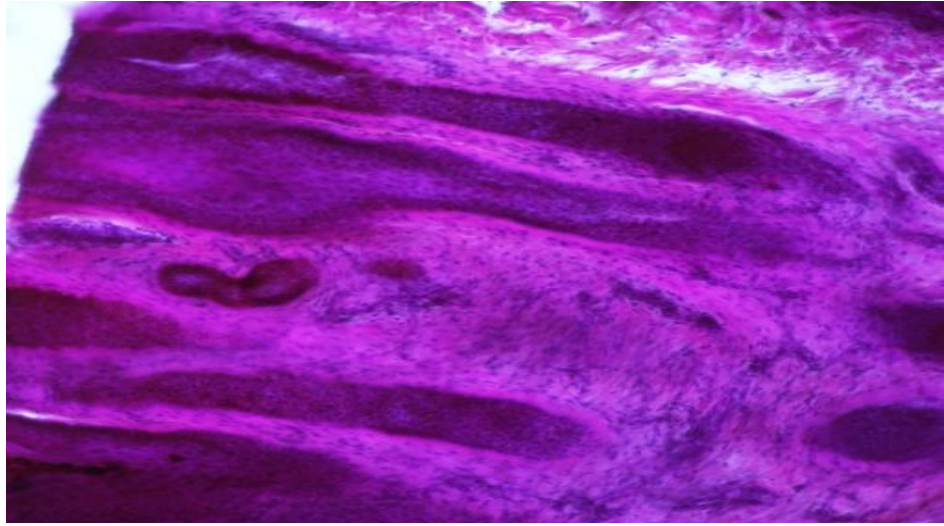


Fig.28 Microphotograph of skin having luminal folliculitis showing inflammatory cells in lumen of follicle. H&E 100X.

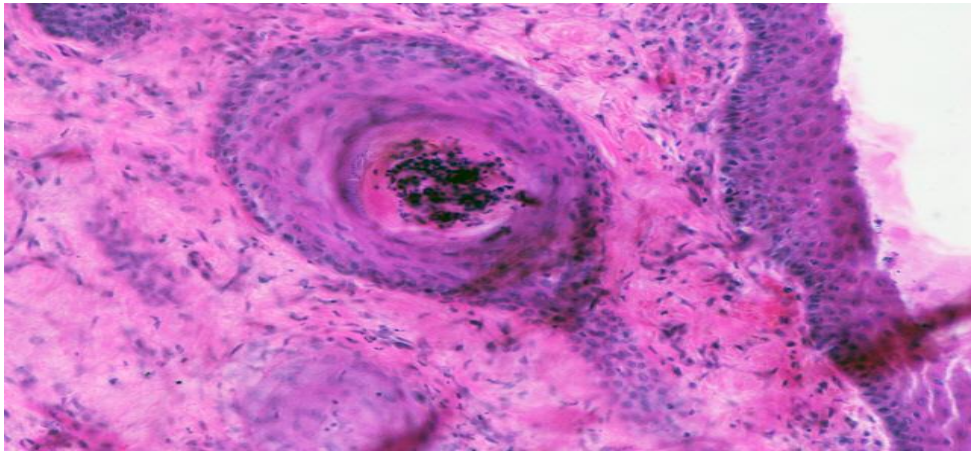


Fig.29 Microphotograph of skin having folliculitis showing inflammatory cells in lumen of follicle. H&E 200X.

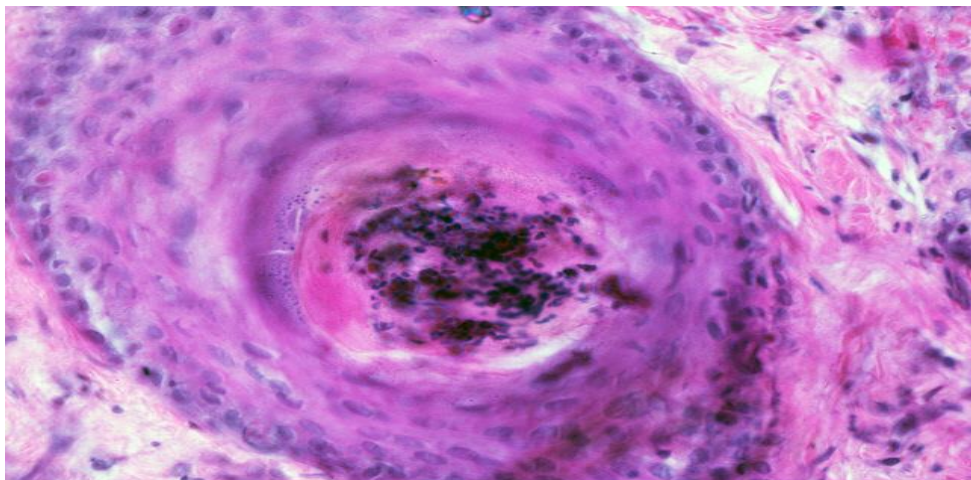


Fig.30 Microphotograph of skin having folliculitis showing inflammatory cells in lumen of follicle. H&E 400X.

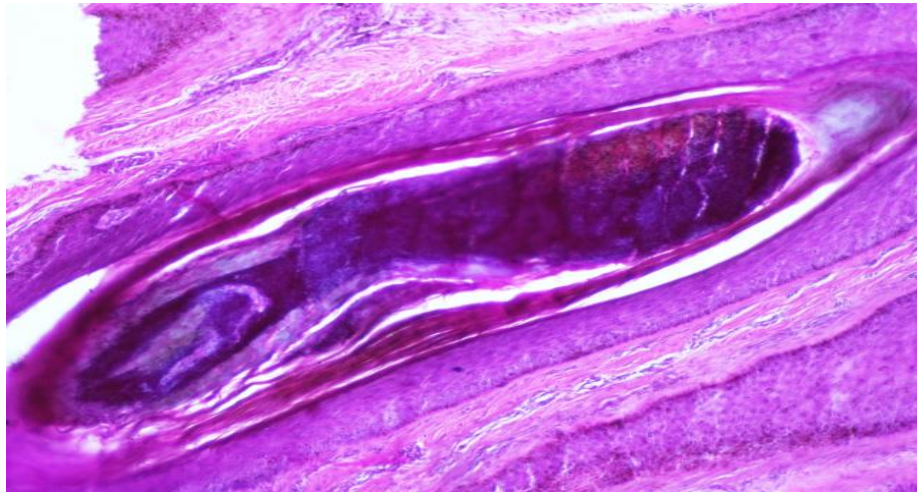


Fig.31 Microphotograph of skin having folliculitis showing granulomatous inflammation with marked neutrophilic infiltration in the follicle. H&E 100X.

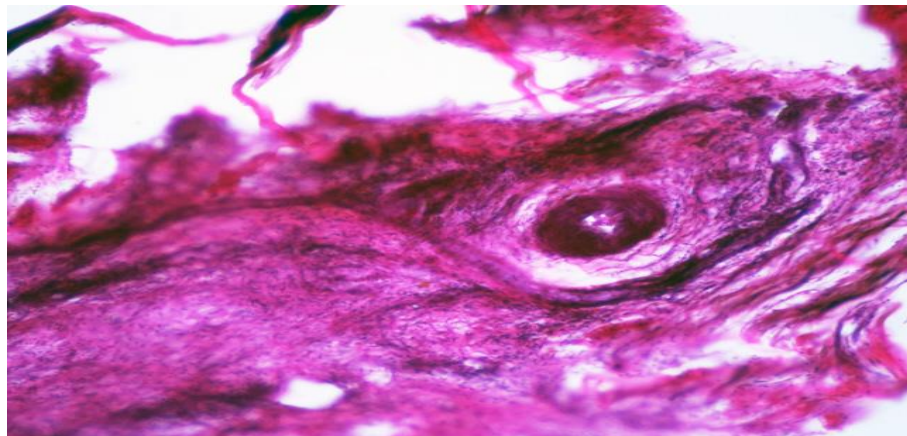


Fig.32 Microphotograph of skin having furunculosis showing destruction of follicular epithelium, inflammatory infiltration and predominantly of mononuclears. H&E 100X.

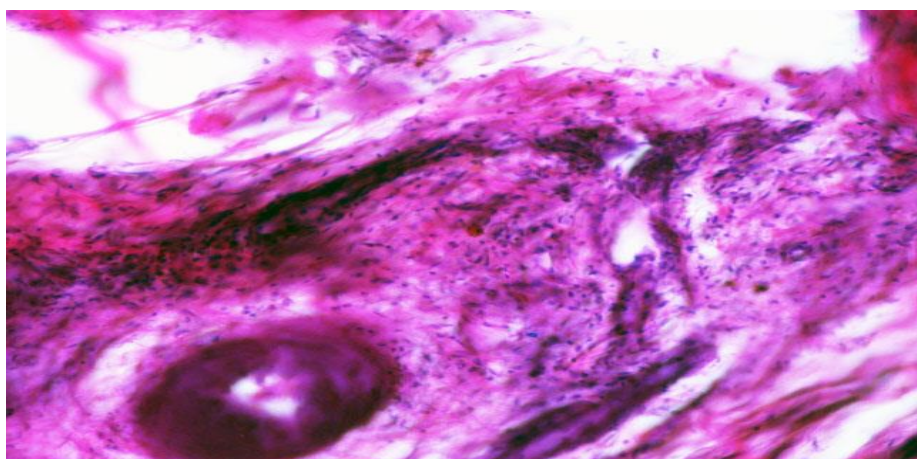


Fig.33 Microphotograph of skin having furunculosis showing destruction of follicular epithelium, inflammatory infiltration, predominantly of mononuclears. H& E 200X.



Fig.34 Photograph of a camel having ulcerative dermatitis showing brown crusts at the neck region.

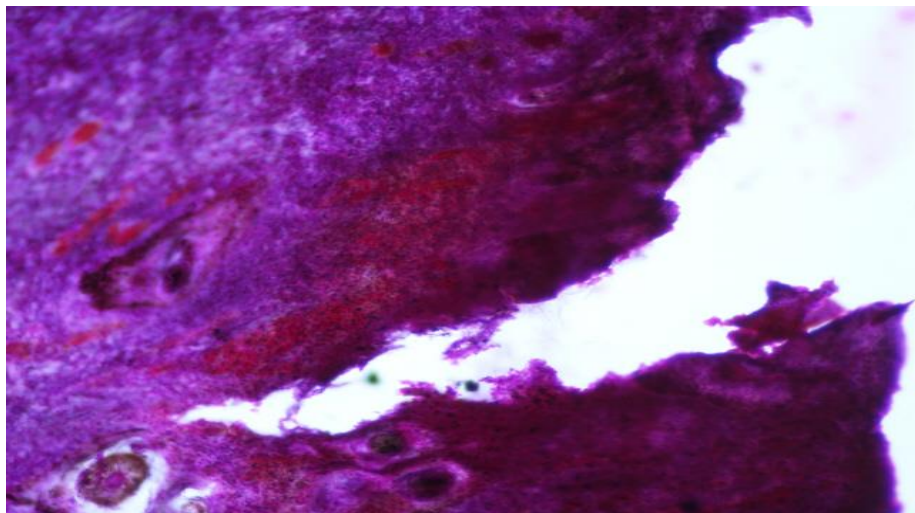


Fig.35 Microphotograph of skin having ulcerative dermatitis showing inflammatory exudate, haemorrhages and lymphocytic infiltration in the dermis. H&E 100X.

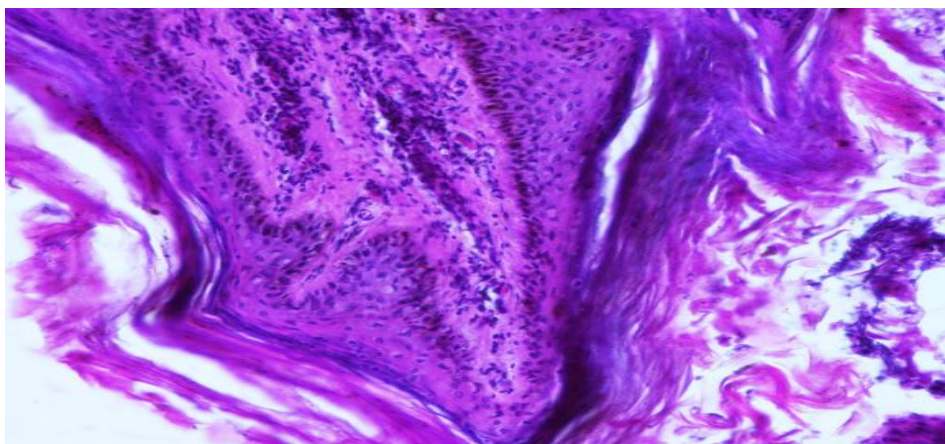


Fig.36 Microphotograph of skin having ulcerative dermatitis showing epidermal hyperplasia and parakeratosis. H&E 200X.

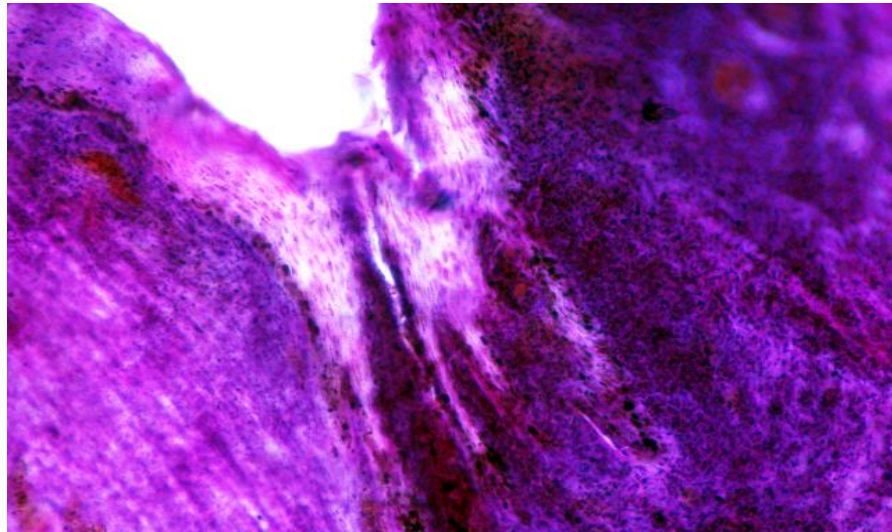


Fig.37 Microphotograph of skin having ulcerative dermatitis showing inflammatory exudates, haemorrhages and lymphocytic infiltration in the dermis. H&E 200X.

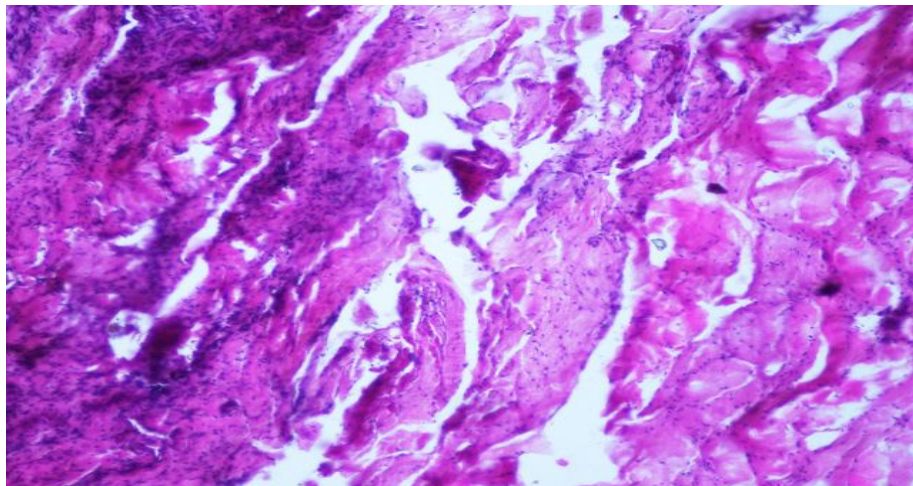


Fig.38 Microphotograph of skin having interstitial dermatitis showing inflammatory infiltration between collagen bundles of dermis. H&E 100X.

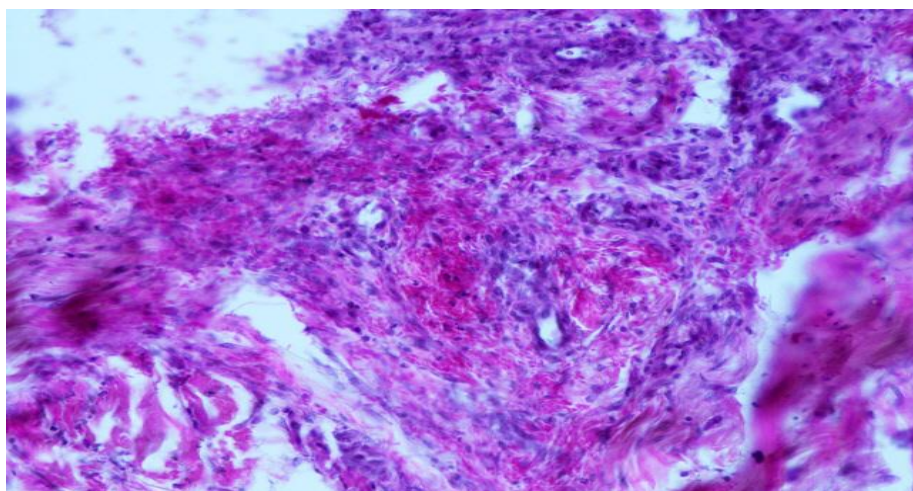


Fig.39 Microphotograph of skin having interstitial dermatitis showing haemorrhages and infiltration of inflammatory cells in the interstitial spaces of dermis. H&E 200X.

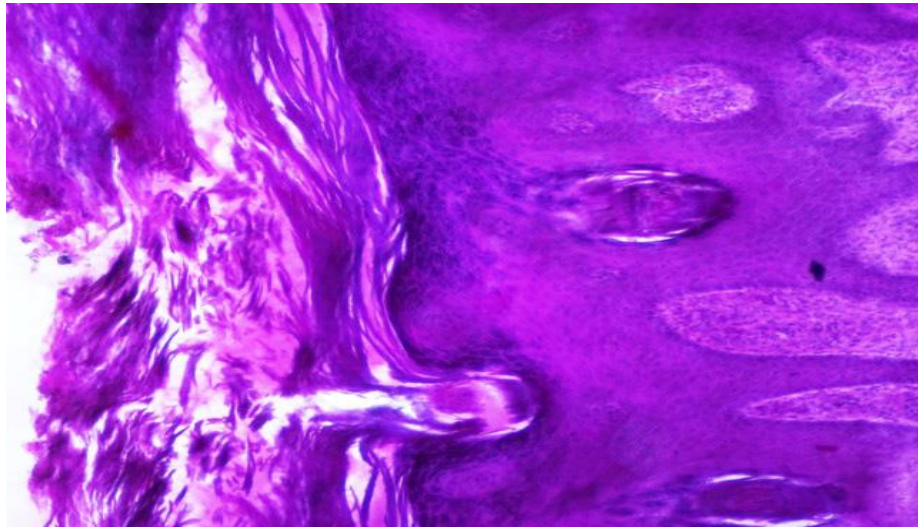


Fig.40 Microphotograph of skin having seborrheic dermatitis showing hyperkeratosis, thickening of epidermis with mononuclear infiltration in underlying dermis. H&E 100X.

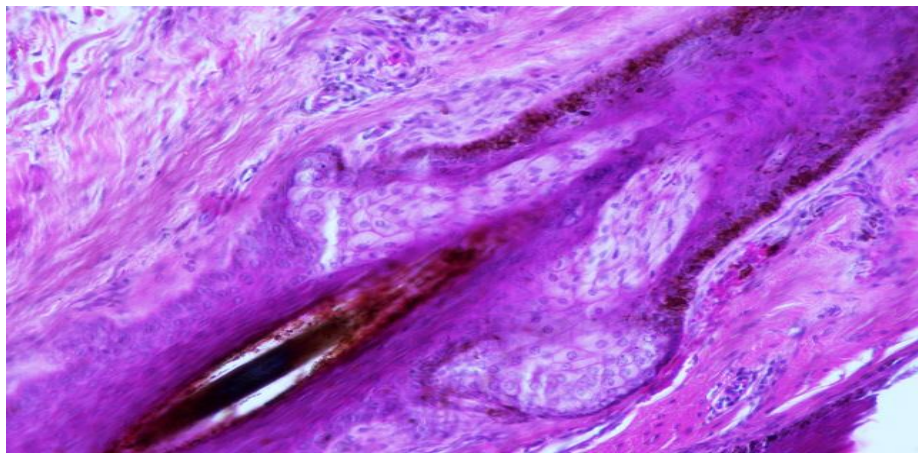


Fig.41 Microphotograph of skin having seborrheic dermatitis showing Hyperplasia of sebaceous glands. H&E 200X.

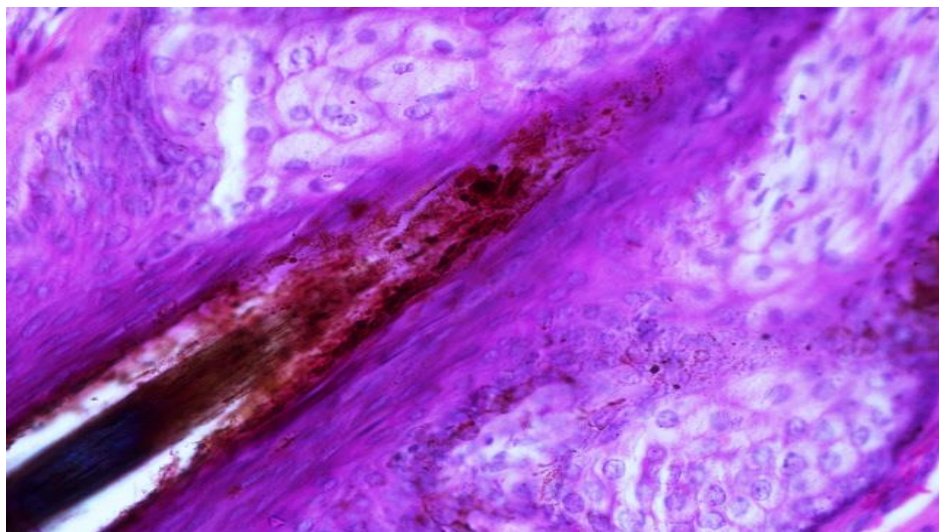


Fig.42 Microphotograph of skin having seborrheic dermatitis showing hyperplasia of sebaceous glands. H&E 400X.

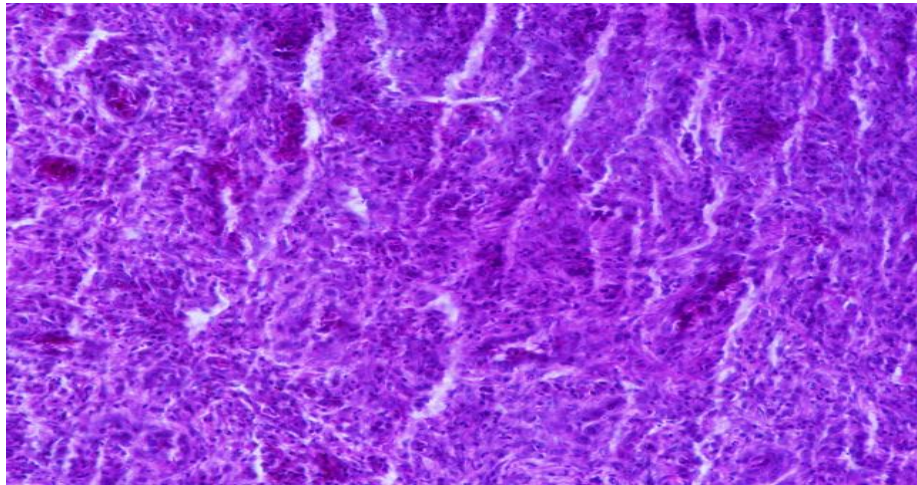


Fig.43 Microphotograph of fibrosing dermatitis showing extensive fibrosis in dermis along with infiltration of lymphocytes. H&E 100X.

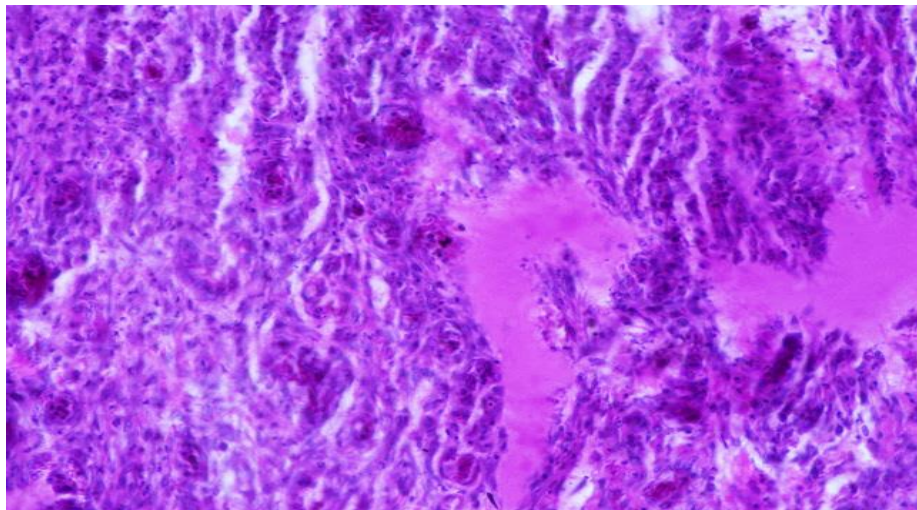


Fig.44 Microphotograph of fibrosing dermatitis showing extensive fibrosis in dermis along with oedema and infiltration of lymphocytes. H&E 200X.

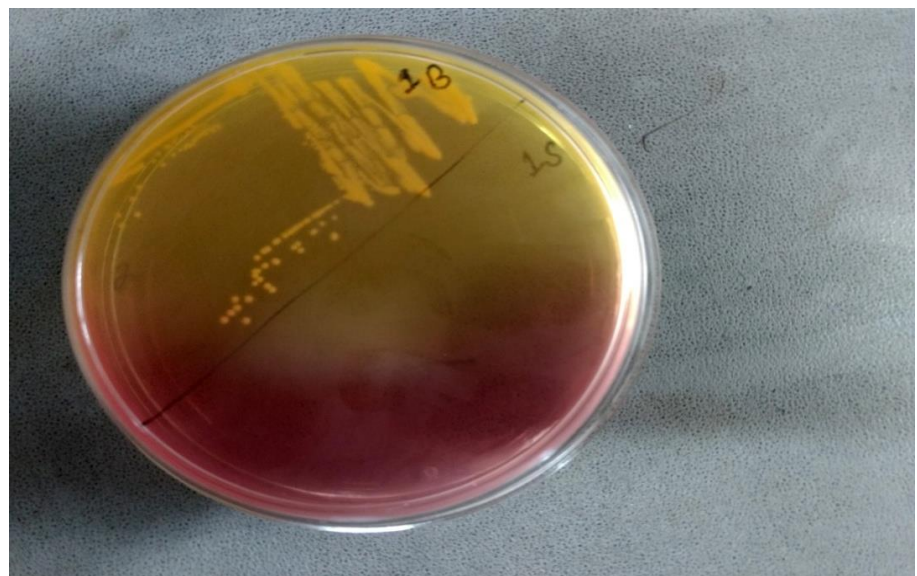


Fig.45 Photograph showing yellow colonies of *S. aureus* in Mannitol Salt Agar

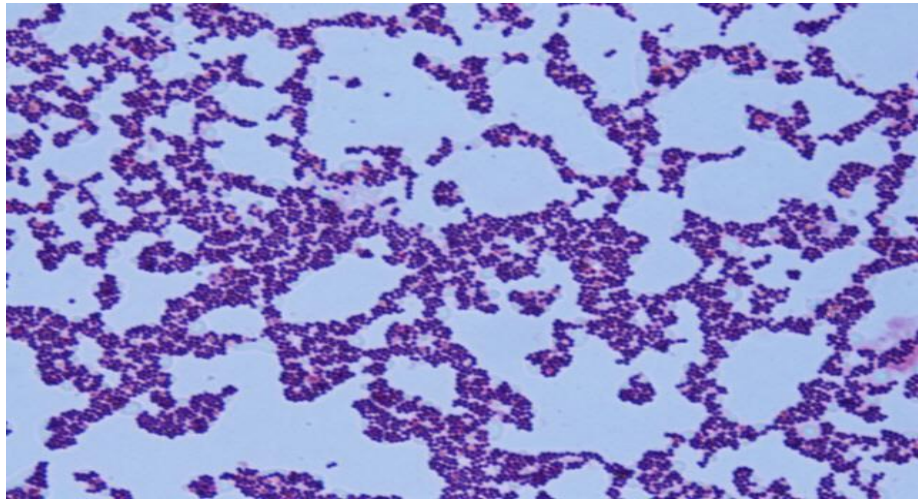


Fig.46 Photograph showing *S. aureus* grape cluster like morphology on Gram stain. 1000X.

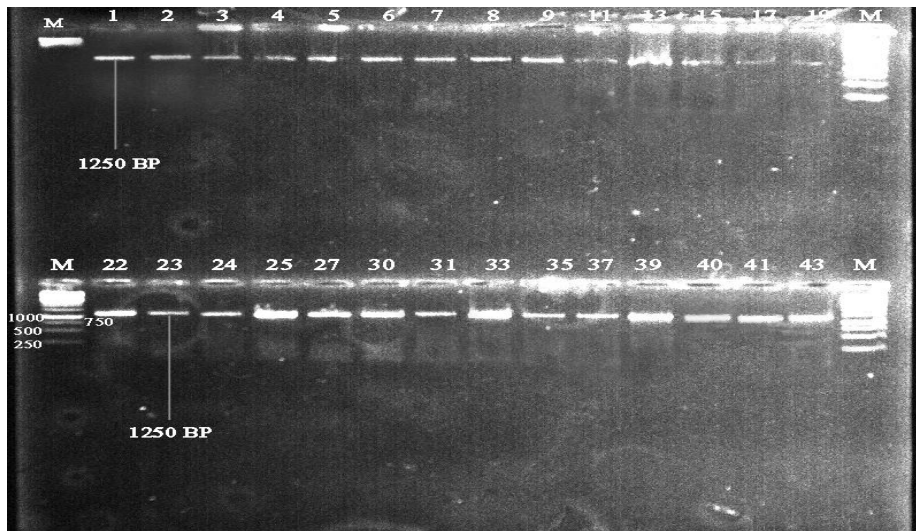


Fig.47 23SrRNA ribotypic of *S.aureus* obtained from camel skin lesions M-Molecular marker (250bp).

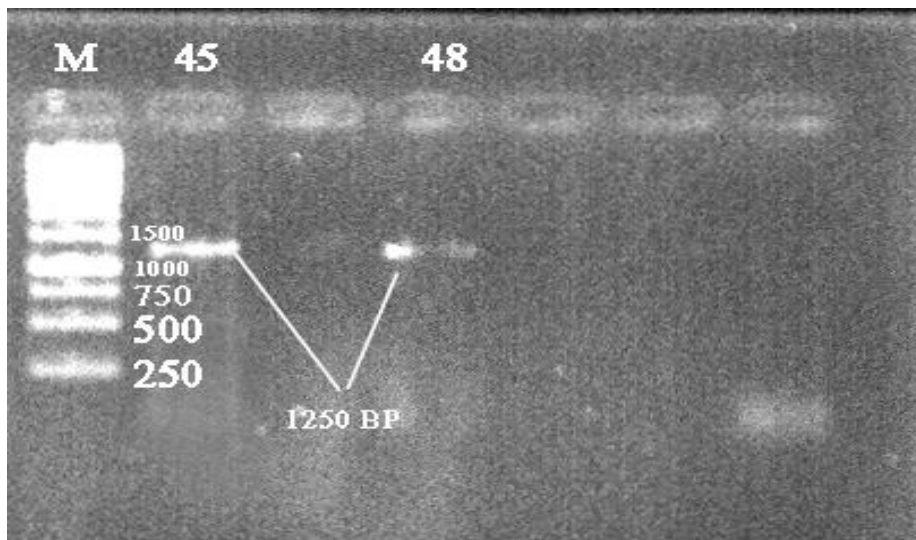


Fig.48 23SrRNA ribotypic of *S.aureus* obtained from camel skin lesions M-Molecular marker (250bp).

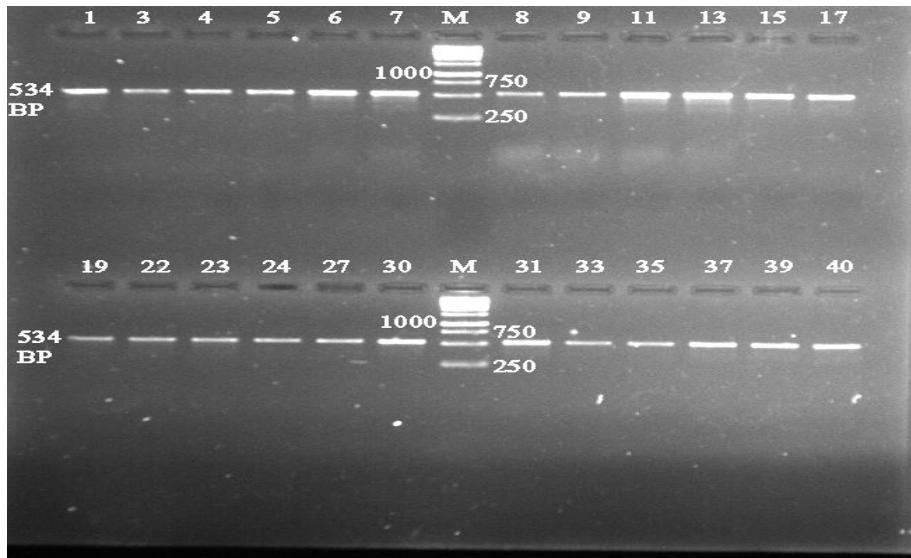


Fig.49 Agrose gel electrophoresis of amplicons of *hla* gene *S.aureus* isolates obtained from camel skin lesions M-Moleculer Marker (250bp).

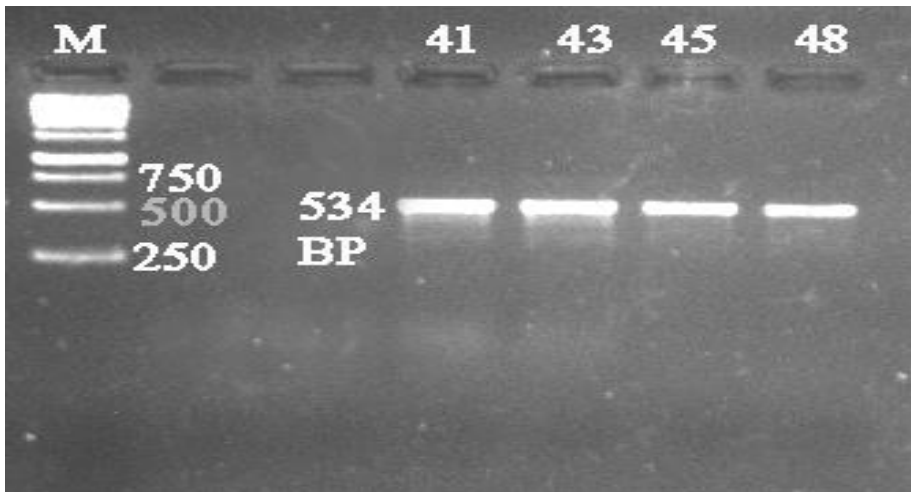


Fig.50 Agrose gel electrophoresis of amplicons of *hla* gene *S. aureus* isolates obtained from camel skin lesions M-Moleculer Marker (250bp).



Fig.51 Photograph of a camel showing extent of lesion from *hla* positive *S. aureus* sample.

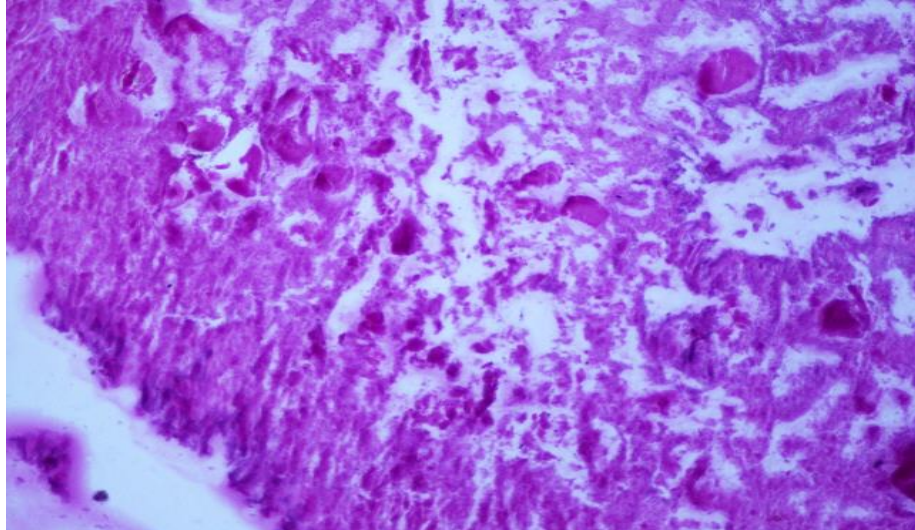


Fig.52 Microphotograph of skin showing severe muscle necrosis.(*hla* positive) H&E100X.

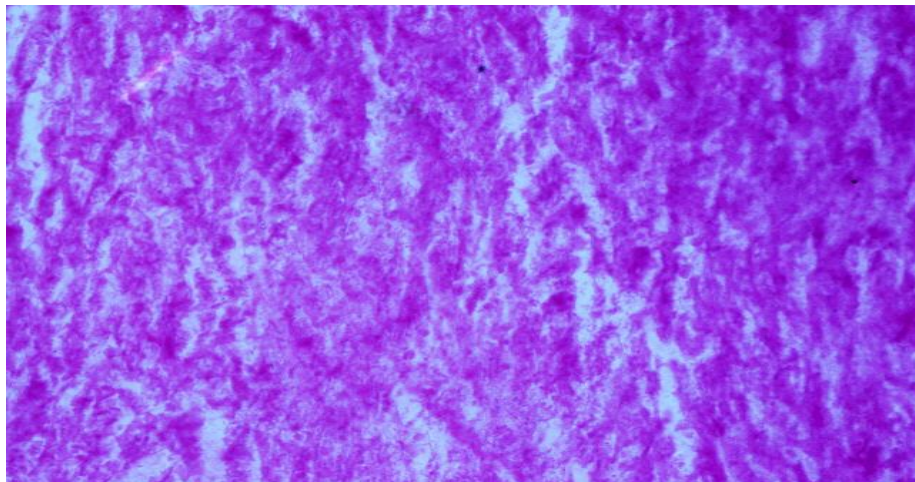


Fig.53 Microphotograph of skin showing severe muscle necrosis.(*hla* positive) H&E 100X.



Fig.54 Photograph of a camel showing extent of lesion from *hla* negative *S. aureus* sample.

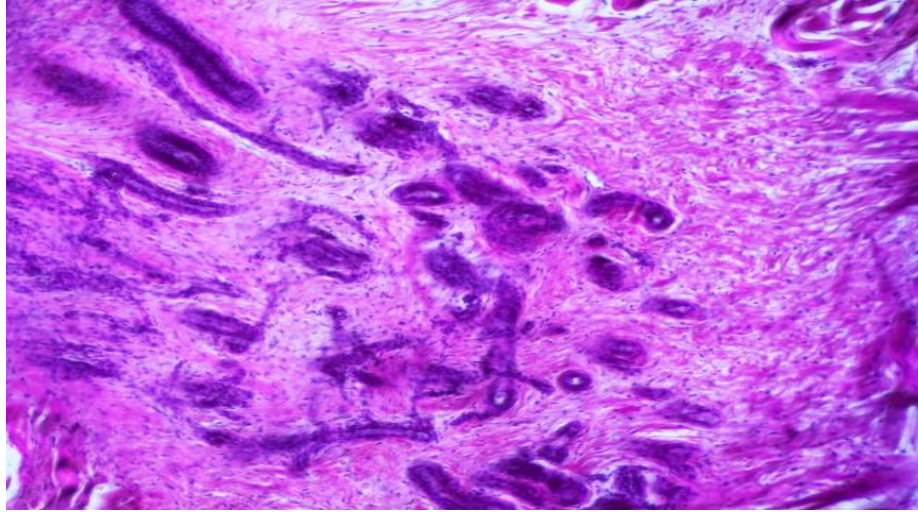


Fig.55 Microphotograph of skin showing angiogenesis. (*hla* negative) H&E 100X.

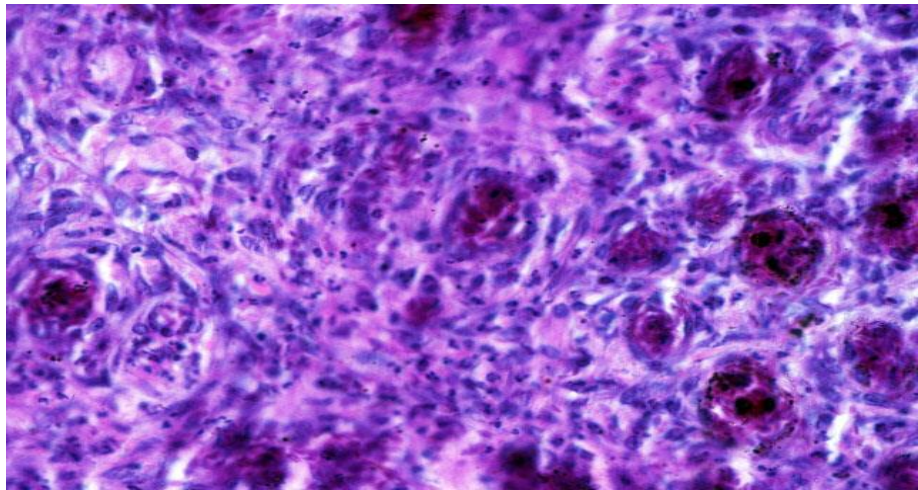


Fig.56 Microphotograph of skin showing extensive granulation tissue. (*hla* negative) H&E 100X.