

Humpsore: Stephanofilariasis With Reference to Andaman and Nicobar Islands

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Abstract

Humpsore or stephanofilariasis is caused by *Stephanofilaria assamensis*, transmitted by fly *Musca conducens* in different animal species particularly in bovine and bubaline species in eastern region of India including Andaman and Nicobar Islands. It causes pruritus, damage in the hair follicles, skin glands leads to alopecia and severe ulcerative nodular dermatitis, exudation, granulation, ulceration and incrustation. It causes delayed puberty in heifers, reduced milk production, increased inter-calving interval and reduced fertility rate in milch cows. Various therapeutic measures include levamisole hydrochloride, organophosphorous compounds, lvermectin, petroleum jelly, tobacco ointment, etc. are used in alone or combination of two or more for effective control of stephanofilariasis. Control and eradication of stephanofilariasis may be successfully achieved by targeting life cycle of *M. conducens*. Success of the treatment is depending upon the control fly infestation and microfilaria in the blood of the affected animals. Simultaneously, other managemental procedure such as blanket treatment of the affected animals, improvement of the general cleanliness and hygiene has reduced the fly population and prompt treatment of fresh injuries or cuts and intermediate host free environment are to reduce the prevalence of humpsore.

Key words: Stephanofilariasis, prevalence, clinical symptoms, pathophysiology, treatment, control measures

Introduction

Livestock enterprise constitutes an important part in wealth of a country as it provides draft power, leather, manure, milk and meat to the vast majority of the people throughout the world. Humpsore is caused by S. assamensis, which widely affects the bovine, bubaline and caprine species, elephant, black rhinoceros and Nilgi in India, common in eastern region of India particularly in Assam, Tripura, West Bengal, Odisha and Andaman and Nicobar Islands and also in other states such as Andhra Pradesh, Telangana, Bihar and Gujarat, besides Southeast Asian countries (Rai et al., 2010). Stephanofilariasis causes pruritus, damage to the hair follicles and skin glands and alopecia. It also causes ulcerative nodular dermatitis, exudation, granulation, ulceration and incrustation, depending on the stage. It causes unclean or unhygienic milk production. Stephanofilariasis is not only affecting the hump but also affects other parts of the body, inner canthus of the eye, scrotum, udder and sternal area (Watrelot-Virieux and Pin, 2006). Size of the humpsore varies from a few cm to more than 30 cm and this is endemic to Andaman and Nicobar Islands (Rai *et al.*, 1990; Rai *et al.*, 1992) as these islands have typically hot and humid climate throughout the year and rainfall is extending more than 8 months with higher relative humidity (average 80%), which favour the un-interrupted growth of parasites, flies, insets, lice, etc.

Stephanofilaria can be characterized as the agent of a disease commonly observed in exuberant high occupancy pastures with large quantities of wet faeces, principally in hot and rainy seasons (Sutherst *et al.*, 2006). Five species of the genus Stephanofilaria have been described as bovine parasites in a number of regions of the world; their intermediate hosts are *Haematobia irritans*, *Musca conducens*, *Musca planiceps* and *Musca autumnalis* (Riviera and Aycardi, 1985). Stephanofilariasis is transmitted biologically by *M. conducens* in Andaman and Nicobar Islands (Rai *et al.*, 1995). This disease affects both male and female animals. The affected male is unsuitable for draught and plough purposes and affected females lose their productivity, growth rate and fertility rate. Stephanofilariasis causes delayed

puberty in heifers and diminished milk yield, increased inter-calving interval in milch cows. Thus, the farmers suffer severe economic losses. It is also considered as a zoonotic disease; however, its occurrence in humans is rare (Novaes et al., 2006). It was also reported that stephanofilariasis has showing higher prevalence rate in exotic and its crossbreds (20.17%) than in zebu (16.14%) cattle (Singh et al., 2002). Further, Johnson and Toleman (1988) reported that the prevalence rate was lower in light coloured than in dark coloured cattle in zebu type. Various workers attempted to eradicate the disease and variation in success rate both in India and in other parts of the world is due to indirect life cycle of the parasite and non-availability of effective and economical treatment protocol for this disease. This review explains about the incidence, pathophysiology, treatment and control of humpsore in cattle and buffaloes with special reference to Andaman and Nicobar Islands.

Etiological factor

A breach in the continuity of epidermis of the skin is the pre-requisite condition for the subsequent development of humpsore, continuous ocular discharge from the inner canthus of the eye and feeding of the discharge by flies, injuries in the base of the horn and base of the ear, abdominal wall or other regions favour for development of the humpsore. Breach is the origin of the lesion in the skin which becomes enlarged by secondary bacterial infections (Staphylococcus aureous, Staphylococcus albus, etc.). Severity of the lesions varies with different predilection sites. It is observed that the humpsore lesions mostly on or around the hump and also observed on the back, ventral surface of the body, anterior and posterior to the navel and on the abdomen. Topographical studies suggest that S. zaheeri is a well adapted and widespread parasite whereas S. assamensis is confined mainly to the eastern parts of India. A humid tropical climate with thick vegetation appears to be ideal environment for Stephanofilaria species in general and for S. assamensis in particular. Therefore the stephanofilariasis is more prevalent in cattle and buffaloes of Andaman and Nicobar group of Islands.

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Economic importance of stephanofilariasis

The disease is located mainly on or near the regions of the hump and neck and it causes extensive skin damage. The value of the animals is reduced due to the ugly looking lesion leads to huge economic loss to the farmers (Ibrahim et al., 2013). Severe infections can cause considerable stress to the affected animals which inturn affects the health and wellbeing. Humpsore can be particularly annoying for dairy cattle in endemic regions; it considerably hampers the manual or mechanical milking and makes it impossible to comply with hygienic measures for milking. This disease causes loss of milk production, reduced working capacity and damage in the hide. Damaged hides can be downgraded and even rejected at slaughter. Affected females, particularly milch cows/buffaloes exhibit poor growth rate, reduced milk production and fertility failure. Thus, farmers suffer heavy economic losses due to humpsore.

Clinical examination

The disease can be diagnosed clinically by direct visual method and report from the disease register of the farm. The sore size is varied from a few cm to more than 30 cm. Most important clinical signs is intense pruritus, which is characterised by rubbing of the sore with wall, pillar, trees or fencing which causes a central ulcer or excoriation of the parts, which is complicated by screw worm fly and secondary bacterial infections. In initial stages, the skin is covered by gravish white crust which favours for stephanofilarial infection. The non-ulcerated lesions frequently become excoriated favouring further deposition of stephanofilarial larvae and gradually obtain the characters of ulcerated lesions resulting into partial or completely devoid of hairs. The ulcerated lesions have central ulcers of various sizes and shapes surrounded by larger peripheral crusty areas in most of the lesions with irregular boundary. The discharge of the ulcers is pureblood, serum or blood mixed serum. The surface of the ulcers is frequently hemorrhagic and moist but dry surface covered by thin blackish or brownish crusts and a number of flies are seen feeding on the discharges. The colour of the crusty/scabby part is found to vary from gravish white to blackish and this part is raised from the

ulcerated part or normal skin surface. The thickness of the crusty part of the lesions is up to 2.3 cm according to character and degree of crust formation, compared to the normal skin thickness of about 2.5 mm. The shape of the lesions is characteristically circular although roughly rectangular, triangular or irregular shaped lesions are common (Rahman and Khaleque, 1974). Close contact of the animals and negligent management of cutaneous abrasions could be the cause of higher incidence of humpsore under farm conditions. It is also observed that the humpsore lesions mostly on or around the hump and also on the back, ventral surface of the body, anterior and posterior to the navel and on the abdomen (Dewan, 1971).



Fig 1: Cattle with Humpsore

Clinically two types of lesions are considered for descriptive and diagnostic purposes. Early lesions are smaller in size (about 3 to 4 cm in diameter) characterized by formation of thin, mildly granular crusts in most lesions and rarely laminated. The old lesions, on the other hand, are larger in size and had coarsely granular crusts with many cracks and crevices with or without centrally located ulcer. Both types are partially or completely hairless. These lesions are again classified according to severity depending on amount of crusts, exudation, size of lesion and ulceration into 3 types such as mild, moderate and severe.

Pathological Examination

Microfilaria of *S. assamensis* is observed in peripheral blood smears of cattle (Das, 1955) and Sen *et al.* (1956) recovered microfilariae from humpsore lesions. The

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disease is transmitted by M. conducens, the biological vector and distributed throughout the world (Rai et al., 2010). These flies become infected with microfilariae when they bite or feed on the wounds that cause the worms in the skin of cattle and other final hosts. Microfilariae develop into infective larvae (L3) inside the flies in about 3 weeks. Such flies re-infect their hosts while feeding. The fly also carries the microfilariae from infected animals in proboscis and inoculates the microfilariae to healthy animals by biting. Patnaik (1970) found that the larva of S. assamensis develops in M. conducens and become infectivity after 23-25 days at 25°C. Poor condition and high rainfall are the predisposing factors of humpsore especially in north-eastern part of India including Andaman and Nicobar islands. Gross morbid lesions of the humpsore are to be examined systematically. Clinical signs coupled with skin biopsies provide the best means of establishing a definitive diagnosis.

Histopathological study revealed that there is marked hyperkeratosis of stratum conium of epidermis and discontinuity or loss of integrity of the superficial layer of the epidermis. There is severe dermatitis and also the proliferation of fibrous connective tissue and a diffuse infiltration of the dermis by mononuclear inflammatory cells and eosinophils (Johnson et al., 1986) indicate that there is hyperkeratosis and parakeratosis in the epidermis. Cross and longitudinal section of stephanofilarial parasites are seen in tissue sections which are encapsulated with fibrous connective tissue in hair follicle. Microfilariae and eosinophils are found in the dermis. Microfilariae are found enclosed within limiting membranes in the dermis immediately adjacent to the stratum germinativum. Adults are found in cysts at the base of hair follicles or in the base of the rete pegs and are surrounded by zones of inflammatory cells, predominantly mononuclear cells. The deeper layers of the dermis are contained perivascular aggregations of lymphocytes, histiocytes and eosinophils. Probably, these findings are related to the death of the parasite and the consequent sensitization of the host. There is an extensive proliferation of fibrous connective tissue (non-neoplastic) at the reticular area of the dermis.



The concentration of the worms or microfilaria in the lesions revealed in ear-sore that one worm per 1.58 sq cm, 3.89 sq cm and 12.3 sq cm, skin areas of chronic, moderate and typical sore lesions, respectively (Agrawal, 1977) and sex ratio of 1:3 with predominance of female worms in humpsore. It appears that the two sexes may differ in their antigenicity or adaptability, one sex being more vulnerable to host reactions. Sharma Deorani (1967) revealed that the histopathology of humpsore by recognising lesions in four stages, *i.e.* sub-acute, acute, desquamative and reparative. The worms were found in both the dermis and the epidermis. Microfilariae were observed only in the desquamative and subacute stages. The reparative or healing stage was characterised by the presence of thick contracted scar tissue but the absence of worms.

Stephanofilariasis with reference to Andaman and Nicobar Islands

Andaman and Nicobar Islands has typically tropical humid climate throughout the year and annual rainfall extending more than 8 months with higher relative humidity (average 80%) favours the un-interrupted growth of the parasites, flies, insets, lice, etc. Disease pattern in all the major islands is more or less similar to that of the adjacent regions of Southeast Asian Island countries. The disease treatment protocols, control models, developed in Andaman and Nicobar Islands will largely be applicable to such regions also. However, occurrence of certain diseases may vary, particularly those which spread due to group grazing or prevalent population of intermediate hosts. Prevalence and factors responsible for increasing the incidence of humpsore and its effect on health and wellbeing, production and reproduction of the dairy herds in Andaman and Nicobar islands are depicted in Figure 2.

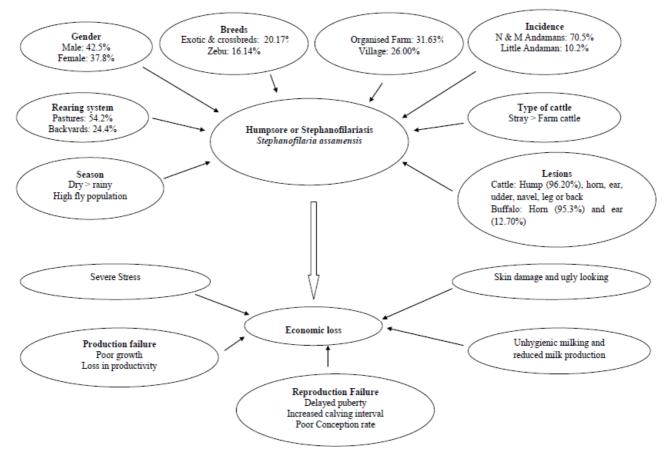


Fig 2: Incidence of Humpsore and its effect on production and reproduction in Andaman and Nicobar Islands (Rai *et al.*, 1994; Rai *et al.*, 1995)

The humpsore parasite becomes adult and starts laying eggs at around 4-6 months in sore (Srivastava and Dutta, 1963). The sore either undergoes reparative stage or become crusty during the month of July (Rai et al., 1995). These eggs ingested by fly (M. conducens) and develop the larval stage I, II and III in inside of the body of the fly within 25-35 days (Srivastava and Dutta, 1963). The infective larva (stage III) reaches in the saliva of the fly and may get deposited in the skin injuries during the feeding of the fly on the wounds. Thus, the infective larva deposited during September may become mature by January and this vicious life cycle continues. The infective larva inside the injury on the host feeds on live cells resulting in gradual increase in the size of the wound and excretion of serum, dead cells etc. in form of oozing from the wound, which attracts the flies further. Repeated deposition of larvae is required to develop a full-fledged sore and if the wound is made fly-proof, the sore may heal/



undergo reparative stage and further spread is prevented (Rai *et al.*, 1995). However, the filariids are known to be long-lived and it is difficult to accept that animals can get rid of the infection in as short a span as 6-8 months. It appears more likely that the dry season causes the latent lesions to flare up into the clinical form due to reinfection and increased numbers of vector bites, thus, apparently raising the proportion of infected animals. Therefore, the treatment should be conducted once in six months or twice in a year in Andaman and Nicobar Islands.

Therapeutic approaches

Completely approved treatments for stephanofilariasis are not available for cattle and buffaloes and different treatment protocols have varied success rate (Table 1). The therapeutic approaches were assessed on the basis of skin healing and normal posture of the animal.

SI. No.	Treatment Protocol	References
1	Surgical removal or cauterizing agents	Mishra (1969)
2	Ointment made from 64 preparations (conch shell ash, lead monoxide, sulphur, tobacco, etc.)	Hassan (1969)
3	Application of Petroleum jelly	Patnaik (1970)
4	Neguvon 6% liniment + Asuntol 6% dusting powder	Patnaik (1970)
5	Antimosan (s/c) + 1% gentian violet	Ahmed and Ali (1973)
6	Trichloropon applied with 6-10% petroleum jelly or castor oil	Rahman and Khaleque (1974)
7	8% Trichlorophon + 4% sulphonamide ointment (Healed within 20-26 days)	Baki and Dewan (1975)
8	Parentral administration of 8% Trichlorphon (very effective)	Baki and Dewan (1975)
9	Antimony potassium tartrate + Phenothiazine (4-8% ointment)	Dutta and Hazarika (1976)
10	Tobacco ointment (80% of cases cured)	Dutta and Hazarika (1976)
11	External application of Antimony potassium tartrate	Dutta and Hazarika (1976)
12	Trichlorophon (6-10%) in petroleum jelly or castor oil (Cured within 7 days)	Rahman and Khaleque (1974)
13	Supona 20 and Sumithion @ 4% concentration (Toxic signs @ 6%)	Das et al. (1977)
15	Levamisole hydrochloride + blanket treatment	Rai et al. (1994)

Table 1: Available therapeutic measures for Humpsore

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SI. No.	Treatment Protocol	References
16	Levamisole HCl + Zinc oxide ointment (mild and moderate size healed within 5-13 days)	Rai <i>et al.</i> (1994) Rai and Ahlawat (1995)
17	Less than 2 cm diameter healed within 5-6 days	
18	2-5 cm diameter healed with 7-8 days	
19	5-8 cm diameter healed within 10-14 days	
20	8-12 cm diameter healed within 15-18 days	
21	more than 12 cm diameter healed within 17-25 days	
22	Ivermectin + Levamisole + Mastilep ointment	Choudhury and Das (2012)
23	Levamisole (3ml s/c) at lesion+ Dermocept ointment (healed in 11 days)	Phukan <i>et al.</i> (2005)
24	Ivermectin + Topicure spray (healed within 21 days)	Puttalakshmamma et al. (2012)
25	15% Tobacco ointment	Al Masud et al. (2017)
26	Ivermectin + Zinc oxide ointment twice daily	Islam et al. (2018)

Prevention and control

The control and eradication of stephanofilariasis in bovines can be successfully achieved especially by targeting the life cycle of the intermediate host, *Musca conducens*. Simultaneously, other measures such as blanket treatment of affected animals during active phase of the parasite after cordoning the area, improvement in general cleanliness and hygiene to reduce the fly population, promptly attending to fresh injuries/cuts and concerted efforts to make the intermediate host sterile for parasite may be undertaken (Rai *et al.*, 2010).

Acknowledgement

The work was supported by NABARD for project "Humpsore treatment by Trimodel therapy to improve the Production and Reproduction Performances of milch cows in South Andaman".

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Received : 21st June 2019

Accepted : 14th October 2019