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Degnala disease in buffaloes and cattle: A clinical review

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Abstract
Degnala disease is also called as mycotoxicosis, and having a clinical syndrome similar to chronic ergotism and is distinguished by appearance of edematous swelling, necrosis and gangrene of the extremities like on legs, tail, ears, etc. Buffaloes and cattle are more susceptible for Dagnala disease to due to feeding of paddy straw infected with fungus, Fusarium sp. Present report and history also confirmed the feeding of fungus-infected paddy straw continuously to these animals. Disease is caused by toxins secreated by mould (fungus) that grows on feed (eg. ground nut cake, maize etc.) and stored fodder that is damp. A female buffalo calf have specific signs of Degnala Disease was successfully treated with Cobalt, Manganese, penta sulphate of Copper, Zinc and Iron along with other symptomatic treatments. Animal was quick respond to the treatment and completely recovered after 15th day of treatment. Antiseptic washing of wounds with 5% copper sulphate till the culture result. After confirming the disease as degnala, the animal was treated with antidegnala liquor (*Zinc sulphate*) @ 7 ml OD for 7 days; Long-acting Oxytetracycline @ 1ml/kg b.wt IM which was repeated after 72 hours and Brotone liquid @ 40 ml OD orally for 5 days. From the first day, the owner was advised to stop feeding mould-smelling straw to the sick animal and others and to prevent contamination of lesion of animal to dust and dirt.

Keywords: Degnala Disease, Buffaloes, Cattle, Mycotoxicosis and Gangrene

Introduction
The scientific name of this disease is "Clinical gangrenous Syndrome". It is also known as "Mycotoxicosis in buffaloes and cattle". But it is popularly known as "Degnala disease" because of the fact that it was first recorded in the villages situated on the both sides of Deg rivulet it (Nala) in Pakistan by Shirlaw in 1939 [14]. The disease affects mainly buffaloes, however, cattle have also been reported to suffer. This disease is prevalent in Pakistan, India and Nepal. In these countries it has become the disease of economic importance due to high morbidity and mortality rates in the affected animals.

Aetiology
The causal agent of this disease is a fungus *viz*. *fusarium equiseti* which is found in mouldy paddy straw. This fungus produces a mycotoxin which is responsible for the development of disease. When the animals are fed mouldy and moist paddy straw, the symptoms of *fusarium* toxicity develop. Kalra, *et al.* (1972) [6] reported *Fusarium equiseti-associated* mycotoxin as a possible cause of Degnala as possible cause of Degnala disease. Irtan and Maoboll (1986) [4] reported that the fungus isolated from the paddy straw of Degnala-affected areas in Pakistan by the Common Wealth Mycological Laboratory was identified as *fusarium.* The paddy straw also contains high levels of oxalic acid, which is believed to be a contributory factor in the production of this disease (Vegad, 2002) [15].

Clinico-Epidemiological Reports
Shirlaw (1939) [14] reported the occurrence of this disease for the first time which affected a large number of buffaloes in Degnala area during the years 1939-30. He described the disease in buffaloes as associated with fever, pain in the abdomen, painful gate and anorexia.

Irfan (1971) [3] investigated Degnala disease in West Pakistan where 295 buffaloes and 10 cattle were affected. He mentioned that the disease had a seasonal incidence and the sporadic clinical cases were seen in winter months when rice straw was fed as fodder.

Kwatra and Singh (1971) [9] reported Degnala disease in buffaloes from Punjab, characterized by necrosis to tips of ears, tail and tongue and swelling of the extremities with subsequent peeling of the skin leaving open wounds. He produced the disease experimentally in buffalo calves by feeding the paddy straw collected from places, where the disease had occurred. Dhillon (1973) [2] also reported Degnala disease in buffaloes which occurred in some parts of Haryana and Panjab during the years from 1969 to 1971. He observed the similar symptoms as described earlier by Kwarta and Singh (1971) [10] in buffaloes from Punjab. Rajan *et al*. (1977) [13] reported the pathological features of necrosis of extremities in 50 buffaloes and 2 cattle affected with Degnala disease in Kerala.

Karki (1999) [8] reported Degnala disease in Banke district of Nepal where buffaloes were mostly infected. Mallick *et al*. (1990) [11] reported 6 clinical cases of gangrenous syndrome in buffaloes simulating Degnala disease from U.P. *Fusarium* sp. was isolated from the paddy straw fed to affected animals; this was the first report from U.P. Kalra and Bhatia (1990) [7] conducted epizootiological studies on the outbreaks of Degnala disease involving 370 heard from 136 villages of Haryana during the years 1968-1978. They revealed that the disease had seasonal occurrences during the winter, associated with the feeding of paddy straw. The morbidity and mortality rates were 61.60% and 13.93% respectively in buffaloes and 13.49% and 2.41% in cattle. Jadhav *et al*. (2003) [5] reported Degnala disease-like syndrome in 67 Kankrej milking cows which occurred during the three yrs period from 1994 to 1996. The disease occurred during the winter season and the incidence ranged from 15 to 50%.

Mallick (2008) [11] reviewed the clinic-epidemiological aspect of Degnala disease in Bihar state. The outbreaks of this disease were recorded first during the year 1997-98 in this state in which a large number of animals (mainly buffaloes) were affected. Since then, the disease has been occurring in this state either in the form of mild outbreaks or sporadically. But during the year 2007-08 almost after a decade, the disease broke out severely afflicting a large number of animals in some parts of this state. The reasons for the occurrence of recent outbreaks may be attributed to heavy rainfall in the year 2007 which would have favoured the excessive growth of *fusarium* in the paddy straw. The morbidity and mortality rates during these out breaks were higher in buffaloes than in cattle.

Clinical Signs

The clinical symptoms observed by the above workers comprised of lameness, disinclination to move and swelling at the lower parts of the leg at the initial state of the disease. The hind legs were involved more often than the forelegs. These symptoms were followed by the involvement of the tail and ears. The affected parts (Ieg, ear and tail) appeared tender and exhibited pain on touching. In advanced cases, the disease was generally manifested by the development of oedema, necrosis and gangrene of the extremities i.e. lower parts of legs, tail and ear lobes. The necrotic and gangrenous lesions were also observed on muzzle, nose, under and teats. The injury in the tongue was also observed in some cases which caused pain in taking food.

The other symptoms consisted of alopecia, roughness of hair cost and appearance of necrotic patches on the neck. Chest and back regions. The animals developed anorexia and milk yield decreased markedly in lactating animals. After invasion of secondary bacterial infections, the cases became complicated. Febrile reactions and diarrhoea were observed in some cases. Sloughing of skin occurred resulting in the ulceration and wounds of varying degrees. In animals have severe involvement of feet, complete separation of hooves was observed. Such animals were unable to stand and became anorectic, weak and emaciated. The animals became recumbent causing the development of bed sores. This stage was followed mostly by death.

Diagnosis

The disease was diagnosed on the basis of clinical symptoms as well as from the isolation and identification of *fusarium* fungus from the sample of paddy straw fed to animals. The disease was also confirmed by producing the disease in experimental buffalo calves on feeding the cultures of *fusarium equiseti* isolated from stray (Kwatra, 1980) [9]. Bhatia and Kalra (1981) [1] produced the disease in experimental buffalo calves by feeding contaminated paddy straw. The animals manifested the classical symptoms of the disease.

Treatment

The antidote against *fusarium* toxicity has not been developed so far Hence, only symptomatic treatment is provided to the ailing animals using a combination of drugs. The following treatment has been observed to be useful in the recovery of clinical if initiated at the early stage of the disease:

1. Meloxicam by i/m injection for 4-5 days daily for the relief of pain and inflammatory conditions of the body.
2. The injection of Oxytetracycline preferably along acting to check secondary bacterial infections.
3. Injection of anti-histaminic drug i/m for 4-5 days to prevent allergic reactions.
4. Vit-A by i/m injection for 4-5 days to maintain the integrity of cells of the body. Injection of liver extract with Vit-B complex i/m on alternate days for 4-5 days to improve the appetite.
5. Acetylarsan 23.6% soln. 10ml s/c on alternate day for 5 days. This acts as a skin tonic and restorative.
6. Regular dressing of wounds with antiseptic cream.
7. Discontinuation of feeding mouldy paddy straw. Instead green fodder and wheat bhusa should be fed. Mineral mixture of 30-40 g should be fed daily.

Among the above line of treatment, the injection of Acetylarsn appears to be a rational one probably due to antifungal activity. Karki (1999) [8] described that the oral and potential use of Arsenic sulfate 2 to 5% (also known as Degnala liquor) was effective against Degnala disease.

Prevention

1. The paddy straw should be stacked at high ground, where there is little possibility of stagnation of water.
2. It should be sufficiently dried before stacking.
3. The dirty and moist paddy straw should not be fed to animals.
4. Mineral mixture should be regularly fed to animals as a feed supplement, particularly to buffaloes stock.
5. The feeding of paddy straw should be restricted during winter if possible. If all, it has to be fed to animals. Then

it should be used after thoroughly washing with water and mixed with adequate green fodder.

Further suggestions

1. The mycotoxin (T_2 toxin) produced by the fungus *fusarium* should be identified and studied.
2. The antidote/ant mycotoxin against the *fusarium* mycotoxin needs to be developed to contain the Degnala disease.

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