

THE CLINICO-PATHOLOGY OF MALIGNANT CATARRHAL FEVER SYNDROME IN CATTLE IN IRAN

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Summary

In a 3 year project on cattle Malignant Catarrhal Fever in Iran the prevalence of the disease has been investigated and determined. The diagnosis of the disease was based on:

1. Clinical symptoms: anorexia, fever, oculo-nasal discharge, conjunctivitis, keratitis and scleral hyperaemia.
 2. Histopathological changes in the central nervous system: vasculitis
- Epidemiological studies indicate the transmission of the transmission of the disease from sheep to cattle.

Introduction

Malignant Catarrhal Fever (MCF) an acute disease of bovine occurs worldwide, (Plowright, 1968; Parihar et al, 1975; Storz et al 1976; Blood, 1983; Hoffmann et al, 1984; Buxton et al, 1985). It is a sporadic disease

but a few severe episodes are also documented (James et al, 1975; Liggitt et al 1987).

The disease is characterized by fever, profuse nasal and ocular discharge, dyspnea, diarrhea, erosions and degenerative lesions in the mucosae of the upper respiratory and throughout the alimentary tracts, kerato-conjunctivitis leading to corneal opacity and blindness, generalised lymphadenopathy, cutaneous exanthema and encephalitis.

Cattle of all ages, breeds and both sexes contract the infection. The morbidity of the disease is very low (1–5%) but its mortality is very high (90 – 100%).

In Africa a member of Herpes virus group (Alcelaphine Herpes Virus 1) has been frequently isolated from MCF cases as well as from 2 species of wildebeest (*Connochaetes gnu* and *Connochaetes taurinus*) which are inapparently infected and can be carriers. The disease that is caused by this type of Herpes Virus is called wildebeest-associated type. In other parts of the world, though the disease is believed to be caused by a viral agent and can be transmitted from cattle by transfusion for infected whole uncoagulated blood or inoculation of tissues from affected animals. The demonstration of the causative viral agent bears no concordance in tissue culture (Coulter & Storz 1979; Buxton & Reid, 1980; Buxton et al, 1985). However there is a hypothesis that sheep inapparently carry the causative agent of MCF and act as a source of infection for cattle, so in these cases it is called sheep associated MCF. MCF has been reported in a confined area of Iran, Tehran area (Etminani et al, 1972). The present paper deals with the preliminary results of a project concerned with the investigation MCF disease, and its epidemiology in Iran.

Materials and Methods

Since 1985 a total of 58 cases of Bovine Malignant Catarrhal Fever were submitted to the Dept. Of Path. at the Rezi Institute Tehran, Iran from all parts of the country, mostly from northern and western parts. The affected cattle were similar, with respect to breed, age and sex. The clinical manifestations, gross pathology and histopathology findings were studied. Attempts were made for the isolation of the causative virus, and transmission of the disease to rabbits and healthy cattle. Two hundred ml of sodium citrated blood from affected cattle were used for blood transfusion. Blood with a sevel solution as anticoagulant was used for preparing buffy coat and virus isolation in primary calf thyroid monolayer cell culture.

Necropsy samples of liver, spleen, kidneys and lymph nodes were taken aseptically, transported to the laboratory, sliced and macerated in YLE cell culture media using Tenbroock Grinder to make a 10% suspension. Rabbits were inoculated with the suspension by either 5 ml, intraperitoneally, or 0.25 ml, intracerebrally, the cattle received 200 ml of citrated blood intravenously and 20 ml of suspension intranasally and the same amount subcutaneously. Test animals were examined daily and body temperatures were also recorded, regularly.

Tissues from different organs were taken and fixed in 10% buffered formalin solution, embedded in paraffin, sectioned at 6 μ m and stained with Haematoxylin and Eosin.

Results

Virus Isolation:

Preliminary works showed that the buffy coat of affected animals induced CPE in primary thyroid cell culture but the test animals did not

show any clinical symptom of the disease. The complete results would be reported in a separate paper.

Clinical Signs:

MCF manifests different clinical signs but that of the “ Head and Eye” is the commonest form (Berkman, 1960; Ohshima, 1977). The most striking clinical symptoms in our cases were fever, profuse mucopurulent nasal discharge, ocular discharge and bilateral corneal opacity, dyspnea and snoring due to occluded nasal air passages leading to open mouth breathing. Lacrimation and thickening of the eyelids were noted in both eyes. Opacity was seen in the cornea. This-in the early stages-appeared on the corneoscleral region, gradually becoming centripetally and finally causing complete corneal opacity and blindness

Diarrhea, staggering and trembling at shoulder and nate were observed in some cases at the terminal stage of the disease. The disease followed a course of about 1–2 weeks which invariably terminated fatally due to marked weakness, prostration and suffocation.

In some cases, local areas of necrosis appeared on the hard palate, gum, gingivae and inner surface of the lips. In such cases, there were excessive salivation which was ropey and bubbly handing from the lips, In a few cases marked lesions of udder or exanthema and erythema in the skin all over the body were noted. The lesions, in the former cases caused either complete sloughing of the skin of the teats or sticky scabes. In the later cases reddened scabbed areas measuring 5–15 cm. in diameter bordered by exanthematous areas were noted.

Gross Pathology Findings

Mouth, tongue, pharynx: The mucosa was thickened and had erosions or ulcers which were covered with exudates. In some cases in the area of ulcers there were focal or diffuse necrosis of the epithelium. Also there was diphtheric membrane in the pharynx.

Eye-Bilateral corneal opacity was seen and also the eyelid were congested and thickened.

Nasal passages: There were thick, fibrinonecrotic membrane and purulent materials which covered the congested nasal mucous membranes or completely packed the nasal passages and turbinates.

Lungs: Emphysema was the prominent change noted in the lung tissue, but confined haemorrhages and areas of consolidation were seen in the lungs as well.

Abomasum: The mucosal surface showed congestion, haemorrhages and ulcers up to 3 Cm. in diameter.

Liver: The liver showed mottling in color and was slightly enlarged in consistency.

Lymph nodes and spleen: Tonsils and facial lymph nodes were enlarge up to twice of the normal size and were edematous. Other lymph nodes were slightly enlarged. In some cases splenomegaly was also noted.

Kidneys: Kidneys were nephrotic in appearance and the cortex was mottled with whitish focal areas and congestions.

Histopathology Findings

Eye: In the conjunctival epithelium ballooning degeneration and irregular hyperplastic projection into the lamina propria were usually seen. The anterior and posterior parts of the uvea were infiltrated by mononuclear cells (uveitis) and prevascular cuffing and vasculitis were observed as

well. Edema was a prominent and consistent finding noted in cornea. The corneal stroma was invaded by mononuclear cells and degenerative changes in the corneal epithelium were seen.

Brain: The lesions were composed of vasculitis, prevascular cuffing in meninges, spinal cord, cerebellum, cerebrum, thalamic area and hippocampus. The choroid plexus showed infiltration of numerous mononuclear cells. In addition, neuronal degeneration, chromatolysis and neuronophagia were noted occasionally.

Blood vessels: There were cuffing, fibrinoid degeneration or necrosis of the tunica media, adventitial histiocytes, mononuclear infiltration and hypertrophy or hyperplasia of endothelial cells.

Lung: The interstitial tissues were infiltrated with lymphoid cells. Some bronchiols showed sloughing. Lymphoid infiltration was seen around the bronchiols and arteries. Alveolar oedema was prominent and their walls showed thickening.

Abomasum: Segmental vasculitis was prominent. There was a diffuse increase of lymphoid cells in the lamina propria and epithelium.

Intestine: Vascular lesions, haemorrhages and mononuclear cells infiltrations were seen in the mucosa and submucosa layers of the terminal part of the jejunum, ileum, cecum and colon.

Lymph node: There was a hyperplasia of lymphocytes in paracortical regions. Also scattered foci of necrosis was occasionally seen.

Liver: There was infiltration of lymphoid cells in the portal triads. The endothelial cells of the capillaries in the periportal areas were congested and hyperplastic. The bile duct epithelium showed hyperplasia.

Heart: The lesions were composed of arteritis, cuffing and lymphoid infiltration.

Kidney: Focal lymphoid cell infiltration associated with cell debris were noted in cortex, medulla (focal interstitial nephritis) and around the glomerular capsules. Haemorrhages, vasculitis and prevascular cuffing were prominent throughout.

Discussion

There are but few reports (Berkmann et al, 1960; Murray & Blood, 1961; Hoffmann 1984) on sheep associated MCF to allow a thorough evaluation of the etiology and histopathology of the disease. Vasculitis is one of the specific features of MCF (Berkmann et al 1960) which confirms that it is an immunologic reaction (Whiteley et al 1985). Two major patterns of the lesions could be described:

1. Proliferation of connective tissue, endothelial and epithelial cells in blood vessels.
2. Infiltration of mononuclear cells affecting all the blood vessels. One of the signs of "Head and Eye" form is corneal opacity which is mainly due to the fibrosis with capillarization in substantia propria, (Oshima et al 1977). The ocular lesions are characterized by lymphonuclear uveitis and retinal vasculitis.

From the etiological point of view it has been recognized that cattle to cattle transmission is possible only blood transfusion and does not occur naturally (Plowright, 1964; Buxton et al 1985). It is confirmed that the close contact between sheep and cattle shows a higher incidence of the disease (Hoffmann, 1984) and sheep particularly at pre and post lambing period may be the source of infection (Harris et al 1978; Hoffmann, 1984).

Although the causative agent is not known yet (Jubb et al 1985) however it is presumed that there is similarity between sheep and wildebeest associated MCF viruses. In 3 cases, we have been able to isolate a virus in primary monolayer calf thyroid cell culture. The identification of the isolates and their pathogenicities will be reported later.

Since the authors failed to substantiate cattle to cattle transmission of the disease, they therefore support the hypothesis claiming that sheep

are the carriers of the causative agent, and hence infect the cattle one way or another. Such an idea is reflected in all our case reports, based on the fact that all the affected cattle had a history of sheep contact, either through common housing or sharing the same pasture.

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