

THERAPEUTIC TRIALS IN AN OUTBREAK OF HAEMORRHAGIC SEPTICAEMIA IN BUFFALOES

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Haemorrhagic Septicaemia (HS) is an infectious disease of cattle and buffaloes. It is an endemic problem in some of the Asian countries. In Pakistan, it is cause of gigantic economic losses to livestock industry and enormous consternation to the dairy farmers. The disease is caused by the specific serotype of *Pasteurella multocida* Robert type I (Aslam, 1986). The causative organism is the normal inhabitant of nasopharynx and tonsils of healthy ruminants (Buxton and Fraser, 1977) and is responsible for clinical manifestations in immunocompromised animals. The disease is not uncommon in rainy season, but outbreaks of the disease have been recorded through out the year (DeAlwis, 1992). The disease incidence is potentiated by variety of factors such as age, burden of infection, host defence mechanism and immune status of animals. Recently, an outbreak of the disease in buffalo calves was recorded and controlled successfully by therapeutic agents.

HISTORY

Livestock Production and Extension (LPE) Department purchased eighty calves (2-3 years old) from small dairy holders over a period of 3 months for breed improvement programme. These calves were kept at Livestock Production Research Institute (LPRI) Bahadar Nagar (Okara) and were not vaccinated.

RESULTS AND DISCUSSION

An outbreak of HS was recorded in these calves during February, 1997. Seventy seven calves were the victim of the menace. Seventy four calves manifested rise in body temperature (106°F), respiratory distress, nasal discharge, drooping of saliva, cyanotic conjunctiva and hot painful swelling of throat, while three showed oedema of the brisket and protrusion of tongue. One of the calves of the latter group was slaughtered. The long bones of the slaughtered calf were processed in laboratory for isolation and identification of the pathogen. The isolated bacterium was capsulated, non-motile, non-sporulated, bipolar and gram negative cocco-bacillus. The isolated bacteria induced 100% death in experimentally inoculated mice

within 24 hours (Shigidi and Mustafa, 1979; Das, 1996). The bacterium unable to grow on MacConkey's agar and capable of fermenting glucose, fructose, mannitol and sucrose without production of gas. The bacterium failed to ferment L-arabinose, lactose, maltose and dulcitol. The bacterium was positive for catalase, oxidase, indole and H₂S production and was negative for voges Prauskaur's test and urease activity (Chandrasekaran *et al.*, 1981). Moreover, fresh colonies of the bacterium on blood agar tested through plate agglutination test with hyperimmune serum against *P. multocida* Roberts type I (procured from Veterinary Research Institute, Lahore) showed a positive reaction (Anonymous, 1992).

The remaining 76 animals were treated with different combination of antibiotics along with the use of antipyretics. The causative bacterium of the menace was capsulated that is mainly composed of lipopolysaccharide. The toxemia induced by the lipopolysaccharide (endotoxin) triggers arachidonic acid pathway in residential macrophages, resulting in production of host derived inflammatory mediators such as prostaglandins, leucotriene, interleukin-I etc. These when present in sufficient quantity might result in vasodilation, hypotension, pyrexia and other circulatory perturbations (Levin, 1990; Slauson and Cooper, 1990). Selective inhibitors of arachidonic acid metabolism (steroid and non-steroid anti-inflammatory drugs such as Delofenic sodium) along with broadspectrum antibiotics such as tetracycline, amoxycolin, floxatril etc, showed beneficial effects (Table 1). Cold water application before medication also mitigated the body temperature and consequently improves the recovery rate (Raza, 1996).

The treated animals incepted rumination within 24 hours post medication, while body temperature reduced to normal after 60 hours (Table 2). All the infected animals were cured with any of the four combinations of drugs except two animal: one receiving Floxtril plus Skimadin, was slaughtered on account of its recumbency stage and one calf died before receiving any treatment. The postmortem examination of dead and slaughtered calves revealed serogelatinous fluid particularly in the submandibular, throat and brisket

Table 1: Response of the infected animals to therapy

Group of Calves	Therapy	Calves treated	Recovery (%)
I	Amoxycolin (Vet Med) 1 ml/10 Kg B.Wt., IM Tribrissen 48% (Welcome) 1.5 ml/30 Kg B.Wt. IM Novasul (Waseem Impex) 1 ml/20 Kg B.Wt. IM	14	100
II	Amoxycolin (Vet Med) 1 ml/10 KG B.Wt., IM Demicon 33.33% (Vetcon) 30 ml/ 50 Kg B.Wt. IV Slone. M (Selmores) 1 ml/120 kg.	20	100
III	Tetroxy LA (CJM) 1 ml/ 10 Kg B.Wt. IM Demicon 33.33% (Vetcon) 30 ml/ 50 Kg B.Wt. IV Dexafar (Prix) 1 ml/20Kg	22	100
IV	Floxatril (Prix) 1 ml/20 Kg B.Wt. IM Skimadin (Thenis) 30 ml/50 Kg B.Wt. IV	20	95

Table 2: Body temperature of animals after therapy

Time (hours) post-treatment	Temperature of treated calves (°F) Group of calves			
	I	II	III	IV
00	104.0	104.2	105.8	104.9
12	103.8	103.8	104.6	103.8
24	103.2	103.0	104.5	103.5
36	102.9	102.0	104.1	103.7
48	103.0	102.5	103.0	102.8
60	102.4	102.1	102.5	102.0
72	101.8	101.8	102.2	101.9
84	101.6	101.2	101.6	101.4

Attaining of normal body temperature and respiration with medication was basic criteria for declaring effective treatment. These group of calves were medicated as shown in Table 1.

region, sever enteritis, haemorrhages on muscles, petechial and ecchymotic haemorrhages and pericarditis were invariably seen on the heart (DeAlwis, 1992).

In summary, *Pasteurella multocida* was isolated from long bones of the dead animals suffering from haemorrhagic septicaemia. The steroid/non-steroid preparations and broad spectrum antibiotics induced 100% recovery of the infected animals.

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