Clostridium Perfringens as a Pathogen of Cattle: A Literature Review

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SUMMARY

Reports of Cl. perfringens as a pathogen for cattle are reviewed and discussed.

Introduction

Clostridium perfringens is a ubiquitous, spore-forming, soil-borne bacterium. Six toxigenic types of the organism are recognized. The major toxins produced by each type are shown in Table I. All types except F have been implicated in diseases of cattle. The object of this paper is to review the reports concerning Cl. perfringens as a pathogen of cattle.

Type A. In 1936, Rose and Edgar reported a toxemic jaundice of sheep and cattle in Australia (24). Cattle affected were mainly in the age range of two weeks to four months, but yearlings and adults were occasionally affected. Marked icterus, hemoglobinuria and nervous symptoms were features. Toxin recovered from intestinal content was neutralized by types A and D anti-sera. In 1943, Macrae, Murray, and Grant described an enterotoxemia in calves which were usually affected in the first week of life but occasionally up to two months of age (17). Diarrhea and rapidly-developing depression were the main symptoms, and no characteristic pathology was presented. Diagnosis was based on the presence of many Gram positive Cl. perfringens-like bacteria in the intestine. Only two isolates were typed; both were Cl. perfringens, type A. Schofield's description of "sudden death" of calves in 1955 was based on similar laboratory findings (26). His clinical findings were similar to those of Macrae et al.

Zaharia and Zelenka isolated Cl. perfringens, type A, from the spleens of two cows which had died from "enterotoxemia" in 1957 in Yugoslavia (28). The following year Shirley reported an enteritis in cattle which he believed to be due to Cl. perfringens, type A (27).

In 1961, Prevot, Jacotot, and Valee tabulated information on 51 cases of infection due to Cl. perfringens, type A (21). They had been diagnosed over a 12-year period. The authors reported that type A was the principal type occurring in France.

Niilo et al. (20) reported the results of experimental exposure of cattle to type A toxin. Animals given type A toxin intravenously either died within five minutes with lesions characterized by extensive hemorrhages, or survived. Intraduodenal administration of whole cultures produced no visible affect.

Type B. In 1952, Hepple reported losses

TABLE I. Major Toxins of Clostridium Perfringens

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<tr>
<th>Type</th>
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<td>F</td>
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*Differentiated by heat resistance of spores.

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in suckling calves up to ten days of age in England (15). Severe diarrhea, sometimes with blood, characterized the cases. The type B organism and its toxins were recovered from the intestine. In Italy, Quesada described a case in 1954 in which four cattle aged eight to twelve months died (22). Frank reported an enterotoxemia due to \textit{Cl. perfringens}, type B, in the United States in 1956 (8).

\textbf{Type C.} Losses in calves up to ten days of age were reported by Griner and Bracken in 1953 (13), and Griner and Baldwin in 1954 (12). These investigations brought forward convincing evidence that the toxin of \textit{Cl. perfringens}, type C, was the cause of the enterotoxemia observed. Nillo et al (20) showed that intravenous injection of type C filtrate produced progressive depression and cyanosis in calves, while intraduodenal inoculation of cultures produced no effect. Congestion of the alimentary tract was the prominent feature at necropsy.

\textbf{Type D.} The first report of bovine enterotoxemia due to this type was by Keast and McBarron of Australia in 1954 (16). The disease reported affected four mature cows in one herd, and was characterized by sudden death. Filtered intestinal contents injected intravenously into mice caused death. Type D antitoxin protected other mice against this filtrate.

In 1957 Blood and Helwig reported the loss of two calves, three months of age, from which epsilon toxin of the type D organism was recovered (3). While they reported seeing many similar cases, toxin was demonstrated in the intestine in only one instance. Helwig gave a general description of three clinical types of bovine enterotoxemia of calves in Australia due to \textit{Cl. perfringens}, but presented no real evidence to identify toxins of this organism as the cause (14). In the same year, the loss of three dairy calves from seven to ten weeks of age was described by Griner, Aichelman, and Brown in the United States (11). Type D toxin was recovered from intestinal contents.

In 1958 Griner reviewed the types of autointoxication in domestic animals due to \textit{Cl. perfringens}, and opined that only two of the reported instances of losses due to type D appeared to be sufficiently well-documented (10). The two he referred to were (16) and (11) above. In 1961 Mumford reviewed the disease as it occurred in Australia, and detailed the laboratory tests and some of the difficulties encountered in confirming diagnosis (19).

More recently enterotoxemia of cattle has been reported from Michigan (6), Mississippi (5), Ohio (9), Kansas (7), and Montana (Sharman, A., personal communication). These reports concerned cattle six months of age or older which were being fattened on heavy grain rations. Of these reports, only that of Sharman appears to be supported with sufficient evidence to definitely incriminate \textit{Cl. perfringens} type D.

Beginning in 1953, from one to sixty diagnosis of enterotoxemia of cattle have been reported annually in Alberta (1). None of these diagnoses were supported by identification of the epsilon toxin in the intestines of affected animals. Diagnosis was based on a preponderance of organisms morphologically indistinguishable from \textit{Cl. perfringens} in intestinal smears, along with clinical and pathological changes then considered indicative of the disease. In Alberta, enterotoxemia has been reported chiefly in feedlot cattle at any stage of the feeding period.

Similar losses have been reported in Saskatchewan (2). Losses considered to be due to \textit{Cl. perfringens} type D have also been reported from Ontario (Julian R., personal communication). The opinion is widely held by clinicians in these provinces that such losses can be prevented by immunization of cattle with toxoids of \textit{Cl. perfringens} type D.

In 1955, Quesada and Altiere reported the loss of eight cattle which showed lesions resembling blackleg (23). \textit{Cl. perfringens}, type D, was isolated from the three animals on which a necropsy was performed.

Maki and Tucker reported that multivalent (types B, C, D) \textit{Cl. perfringens} antitoxin may give some protection against the syndrome of acute pulmonary emphysema (18). This opinion was based on the results of prophylactic use of antitoxin in cattle on a pasture which appeared to consistently induce emphysema. Experimental reproduction of type D intoxication has been reported by Nillo et al (20). Intravenous injections produced nervous symptoms and death, and two of eight calves given whole cultures by the intraduodenal route died. Most of the calves which died displayed severe pulmonary
edema. Schofield suggested that Cl. perfringens, along with other bacteria, could produce an enterotoxemia that would result in acute pulmonary emphysema (25). He gave an intraperitoneal drip of Cl. perfringens culture filtrate to one cow. She showed dyspnea following this treatment, and had pulmonary emphysema at necropsy.

*Type E.* The only published report of disease in cattle caused by this type is that of Bosworth in 1943 (4).

**Discussion**

It is evident from this review that Cl. perfringens or its toxins have been found in a wide range of diseases of cattle. In most reports, however, a causal relationship between the organism and the host’s disease has not been clearly shown. Work on pathogenesis and experimental reproduction of disease with Cl. perfringens in cattle has been very limited. Griner and Bracken produced a hemorrhagic enterotoxemia in one calf fed a whole culture of Cl. perfringens type C, cornmeal and milk (13). Niilo, Moffat and Aver, showed that symptoms and deaths could be produced by injection of toxin or whole cultures under artificial conditions, but not by feeding. The immune status of the animals was not taken into account in any of this work. Further work on experimental reproduction and pathogenesis of disease due to Cl. perfringens is needed to clarify the role of this organism in bovine disease.

**References**