few hours
b. Lesser dose - slowly developing paralysis - beginning with difficulty in masticating feed - drooping eyelids and ears - stiff clumsy gait

C. Post Mortem Lesions - None
D. Diagnosis - Difficult - Signs - Lab confirmation - 66-100 Mortality in cattle
E. Prevention and Treatment:
1. No treatment
2. Remove source of contaminated feed
3. Formalized Bacterin-Toxoid

IX. Enterotoxemia (Clostridium perfringens - Welchii)

A. Criteria for Development:
1. Toxogenic strain
2. Abundance of growth promoting nutrients
3. Partial or complete stasis of the gastro-intestinal tract

B. Major Toxogenic Strains:
Type and Toxin
A - alpha
B - beta and epsilon
C - beta
D - epsilon

C. Diagnosis:
1. History:
a. Sudden death - steers on full feed or best doing calves
2. Lesions:
a. Hemorrhagic enteritis with Type C
b. Mucoid and catarrhal enteritis w/Type D
c. Hemorrhages in the thymus
d. Hemorrhages on intestinal serosa and diaphragm
e. Increased straw-colored pericardial fluid
3. Intestinal Smears:
a. Preponderance of large gram positive rods and absence of normally found gram negative flora
4. Mouse neutralization
5. Urine glucose (Type D)

X. Immunizing Products

A. Bacterins:
1. *C. Chauvoei* Bacterin
2. *C. Chauvoei - C. Septicum* Bacterin
3. *C. Chauvoei - Pasteurella* Bacterin
4. *C. Chauvoei - C. Septicum - Pasteurella* Bacterin
5. *C. Novyi-Sordellii* Bacterin
6. *C. Chauvoei -Septicum-Novyi* Bacterin
7. *C. Chauvoei-Septicum-Novyi-Sordellii* Bacterin
8. *C. Hemolyticum* Bacterin
9. *C. Chauvoei-Septicum-Novyi-Sordellii-Perfringens* - Type C & D Bacterin

B. Bacterin-Toxoids:
1. *C. Botulinum* Type *C Bacterin*-Toxoid
2. *C. Novyi* Bacterin-Toxoid
3. *C. Novyi Sordellii, Perfringens* Type C & D Bacterin-Toxoid
4. *C. Perfringens* Type *C Bacterin*-Toxoid
5. *C. Perfringens* Type *D Bacterin*-Toxoid
6. *C. Perfringens* Type *C & D Bacterin*-Toxoid

C. Toxoids:
1. *C. Perfringens* Type C Toxoid
2. *C. Perfringens* Type D Toxoid
3. *C. Perfringens* Type C & D Toxoid
4. *C. Perfringens* Type D - Tetanus Toxoid
5. Tetanus Toxoid

D. Antitoxins:
1. *C. Perfringens* Type C Antitoxin
2. *C. Perfringens* Type D Antitoxin
3. *C. Perfringens* Type C & D Antitoxin
4. Tetanus Antitoxin

Salmonellosis in the Bovine Animal

Vaughn A. Seaton, *D.V.M.*
Professor and Head
Veterinary Diagnostic Laboratory
Iowa State University
Ames, Iowa 50010

Salmonella organisms are gram negative rods that are parasitic in man and animals and usually are viewed as enteric organisms.

The first "salmonella" organisms were isolated in 1885 by Smith and Salmon from swine. In 1888, the second isolote of the "salmonella" group was from a man who had eaten raw meat from a diseased cow (1). Salmonella was the name proposed for the group of organisms in honor of D.E. Salmon, the first chief of the United States Bureau of Animal Industry.

It is interesting to note that in 1934, 44 serotypes were recognized. This number gradually increased to 962 in 1966 and has continued to some 1,300 now. However, most salmonella isolated now belong to a small number of serotypes (3). The disease is believed to be worldwide and in cattle it appears to have dramatically increased in the last few years. In fact bovine salmonellosis has been recognized as an important cattle disease only since the late 1950's (4). Part of this increase may be more apparent than real because of better isolation techniques, and especially a greater awareness of the disease in cattle. It has, of course, been recognized for a long time as a major dis-

ease in swine.

Salmonellosis is a disease of all animal species caused by a variety of different species of salmonella and is manifested usually by an acute septicemia, acute enteritis, or chronic enteritis (2).

The morbidity rate is generally high and in certain cases, such as young calves, malnourished animals or stressed animals due to shipping, calving, weaning too early, or the presence of concurrent infections, it can result in very high mortality rates reaching 30 to 70%. Even those animals that survive the acute form of the disease may suffer long-standing chronic problems.

In order, the most common serotypes in cattle, according to Doctor E.M. Ellis, Veterinary Services Laboratory, U.S.D.A., A.P.H.I.S., Ames, Iowa, as the result of having serotyped thousands of bovine cultures, are as follows: 1. *S. typhimurium;* 2. *S. typhimurium* var. copenhagen; 3. *S. dublin;* 4. *S. newport;* 4. *S. st. paul;* 6. *S. anatum.*

Salmonellosis has been a problem in human health from infected milk, meat, and poultry products. Often this is occasioned by careless handling or contamination after the milk leaves the cow, or in the case of meat and poultry it may be the result of contamination during slaughter or processing. Most all salmonella species in animals should be considered as a potential source of salmonella infection in man.

Transmission

Although most salmonellae are capable of causing disease in animals if the conditions are conducive, we find that certain salmonella serotypes are more likely to cause disease in different species of animals. For instance, in cattle, *Salmonella dublin* is considered host adapted but is more important in Great Britain, for example, than the United States (10). *Salmonelta typhimurium* is the most common serotype causing bovine salmonellosis in the United States. However, *Salmonella typhimurium* seems to be a pathogen for nearly all animals including man and is not primarily an infection of any particular species.

Salmonella organisms can often be cultured from normal appearing animals; however, the mere presence of the organisms do not always indicate disease. In fact, it is believed that many animals, especially adults, are likely to be latent carriers until some debilitating disease or stress occurs and thereby increases the likelihood of the infection becoming an active disease.

Salmonella typhimurium can remain viable for up to seven months in soil, water, feces and on pasture (6). This illustrates the point that environmental conditions greatly affect viability of salmonella. Drying and sunlight are, of course, detrimental to salmonella as they are to many bacterial pathogens.

Salmonella infection in animals is generally acquired by ingestion of the organisms. Transmission is generally from adults to young either directly or by contamination of premises including pens, water, feed, troughs, or buckets. This often occurs after clinically normal but carrier animals are purchased and placed into the herd.

Fecal contamination of feed and water serve as primary sources of contamination for ingestion by other animals. In the Nebraska cases discussed by Dr. Clair Hibbs of the North Platte Diagnostic Laboratory, it appeared that a heavier incidence of their cases occurred along streams. He indicated they had confirmed more cases in 1974 than the last four years altogether (5).

Kahrs, et. al., reported an epidemiologic investigation of an outbreak of fatal enteritis and abortion associated with dietary change and *Salmonella typhimurium* infection in a dairy herd in New York State (7).

Death and abortion in the cows was caused by *Salmonella typhimurium* in a large dairy herd in which the source of the infection was never determined. They were unable to isolate salmonella from either the feed or the water, however, the infection was an explosive outbreak indicating a common source of infection. It appeared to be associated with feeding green chopped hay. Kahrs postulated three different conditions which may have contributed to the outbreak. One theory is that salmonella organisms were present, but went undetected in the green chopped hay in spite of rigorous culture attempts; another was that all the cows were carriers and that the biologic equilibrium of the intestinal flora was upset when the feeding of green chopped hay was started, thereby initiating the septicemia from the intestinal tract and, thirdly, that a massive dosage of *Salmonella typhimurium* was present in some other medium and that the feed change occurred only coincidentally.

At any rate, that case report, I believe, exemplifies many salmonella outbreaks in that the exact source is next to impossible to identify.

Pathogenesis

After ingestion the salmonella organisms colonize the digestive tract and may invade the blood stream thereby setting up an acute septicemia, especially in young or debilitated older animals.

If the infection becomes generalized, the mesenteric lymph nodes, spleen, and liver are excellent sites of infection and from a post-mortem diagnostic point of view, serve as excellent sources for the laboratory isolation of the salmonella.

In septicemic cases, salmonella organisms can, on occasion, be isolated from milk. This is believed to be especially true of *Salmonella dublin* (8). This is one common way the organisms can be transmitted to newborn calves being milk fed.

In speaking of *Salmonella dublin,* Richardson suggested that latent carriers are probably activated to fecal excretion at parturition and showed that they could be detected at calving by culture of rectal and vaginal swabs together with the rectal swab of the neonate (9). One wonders if this may be true of

Salmonella typhimurium and others thereby helping explain the very early occurrence of salmonellosis in neonatal calves.

Richardson suggests that *Salmonella dublin* is activated at parturition from latent carrier cows and therefore can be considered a genital as much as an enteric condition.

Clinical Features

In cattle, the peracute form occurs generally in newborn calves up to two weeks old and is characterized clinically by depression, high temperature of 105° to 107°F and often death within 48 hours.

If actue septicemia does not cause immediate death, acute enteritis is usually the next clinical sign as evidenced by severe watery diarrhea which may contain blood spots and fibrin within a couple of days. The nature of the diarrhea seems to vary according to severity of the infection and duration of the disease before either recovery or death. Diarrhea is initially watery and profuse. Later, passage of blood in feces is to be expected. Dehydration develops very quickly and death may follow in two to five days.

If infected calves survive two to three weeks, and few do, there are often fibrin casts either expelled or found on post-mortem. It appears from necropsy reports in our laboratory and from the veterinary diagnostic laboratory at North Platte, Nebraska, that casts are most often associated with *Salmonella enteriditis*. That is only an empiric observation and may not be confirmed by others.

If the calves live through the first few days of the infection, there is often a secondary pneumonia which follows. In a large percentage of these cases *Pasteurella sp.* organisms are isolated. This reflects a debilitated condition with severe dehydration and emaciation apparent. Electrolyte imbalance becomes clinically important. Often recovered calves never do well and may actually be an economic liability.

Arthritis is said to be prominent in young calves and abortion is common in adults infected by salmonella. However, in Iowa, we see neither involvement with any regularity and cannot say that either is typical of cases submitted to the Veterinary Diagnostic Laboratory. It appears that calves do not usually remain carriers after infected. They either recover or die. The carrier state seems to be initiated and persists in older cattle.

Another hazard is in clinics or hospitals, for instance, where surgery is performed, thus providing the stress necessary to enable a salmonella outbreak to occur. The diarrhea can be profuse, watery, and almost always becomes hemorrhagic in the adult. In fact, a hemorrhagic diarrhea in the adult should make you immediately consider salmonellosis in addition to coccidiosis and mucosal disease.

Necropsy

At necropsy with the acute form of salmonellosis, lesions may vary from none visible to those of a generalized septicemia such as congested mucous membranes and blood engorged organs, including the spleen and possibly a few ecchymotic hemorrhages on both serosal and mucosal surfaces of the intestinal tract. Intestinal lesions are primarily in the small intestine but the abomasum and large intestines are sometimes involved.

In calves the undeveloped rumen is often full of milk or milk replacer and the intestinal contents are watery to hemorrhagic and may contain bits of fibrin. In longer standing cases, fibrin casts may be present. The mesenteric lymph nodes are generally enlarged.

In adults and long standing cases in the young, necrosis of mucosal epithelium of the intestinal tract may be prominent, even to the stage of ulceration and sloughing.

Severe dehydration is apparent clinically but is dramatic at post-mortem. If the calf has lived several days after onset of diarrhea, the fetal fat reserves are depleted and the tissues are thin and dry.

Pneumonia is commonly found at post-mortem and may well be figuring prominently in the ultimate cause of death.

Differential Diagnosis

I could add nothing to the standard textbook descriptions of the diseases to be differentiated from salmonellosis.

Clinical signs of the various diseases could be discussed *ad infinitum* and due to the varieties of signs depending on ages affected, stage of infection, and presence or absence of concurrent infections a hard and fast differentiation clinically is extremely difficult.

It is my conclusion that definitive differential diagnosis is one that requires laboratory assistance.

Salmonellosis in calves must be differentiated from its most common look-alike, *E. coli* infection. High temperatures are constant findings in salmonellosis and not so in colibacillosis. Bacterial culturing is the best method of differentiation in these two diseases.

In cases where the animal is passing blood, coccidiosis is a diagnostic problem. Coccidiosis can be equally explosive and transmitted as rapidly as salmonella infection. Differentiation can usually be resolved by fecal smears or flotations in which the oocysts of coccidia should be readily apparent. Temperatures are not usually as elevated as in salmonellosis, if at all.

BVD or mucosal disease is easily confused with a salmonella infection; however, the mucosal lesions of the dental pad, esophagus, abomasum, and necrosis of the Peyers patches as well as the slower spread from animal to animal is indicative of the former condition rather than salmonellosis. BVD is generally found in older animals than most salmonella infections. Both diseases can occur, of course, in adults.

BVD infected calves generally survive longer than those with salmonellosis. Laboratory confirmation may be needed as concurrent infections may occur.

In adults, Johne's disease can be confused with salmonellosis but again the slower spread and the presence of acid-fast organisms from rectal scrapings are indicative of Johne's and not salmonella.

Reovirus and coronavirus in young calves can best be differentiated from salmonella by FA techniques, culture attempts, and the lack of response to treatment in the instances of viral infection.

Treatment

The mechanism of how a diarrhea occurs is a complex subject. Basically it is initiated by inflammation of the intestinal mucosa. This initial irritation causes a disruption in the normal patterns of both secretion and absorption by the intestinal cells and a disruption of the ability to absorb nutrients.

The inflammation causes an increased secretory action from the cells in an effort to counteract the irritation, bringing fluid containing electrolytes into the lumen. There is also a decreased ability to absorb water. Oftentimes an initial intestinal stasis occurs. This condition permits bacterial growth in the intestines to be especially active and contributes to further colonization and inflammation. The goal in therapy is to stop the organism multiplication, stop irritation and replace water and electrolytes. A regimen of therapy should be directed toward those ends.

It has been suggested that one of the problems of therapy arises even when bacteriacidal action is successful and the salmonella organisms are killed. Lysis may occur releasing endotoxin. The calves may then proceed to die of endotoxic shock.

Antibiotics, sulfas and electrolytes are used heavily in therapy. It appears that regardless of what is used, treatment is often a frustrating and sometimes futile effort. Nitrofurazone compounds, tetracyclines, gentamicin, ampicillin and sulfas are the most commonly used. Chloromycetin would be widely used if it were not illegal to do so.

Antibiotic *in vitro* sensitivity reactions indicate that bovine salmonella isolates are most often sensitive to gentamicin, nitrofurans, vetisulid, and chloromycetin. It appears that, in talking to practitioners in Iowa, triple sulfa IV, plus oral sulfa boluses are the drugs of choice. Sulfonamides in the drinking water are often suggested, if flavored to make it palatable. Electrolyte therapy and rehydration measures are to be recommended in addition to antibacterials, especially in young calves. Where possible the IV drip method, using about 4,000 cc. in the first four (4) hours, followed by 4,000 cc. per day for the next day or so, in a 100 to 125 pound calf, gives the best response.

Bacterins are of doubtful value after infection is established and appears to be little used prophylactally except in some instances as mixed bacterins.

Control

Introducing carrier animals into the herd or group is the most common cause of new infection, followed by contaminated feed and water. Obviously avoiding the above are the best control measures but may be difficult to manage because neither carriers nor contaminations are easily detected.

Conclusion

In conclusion it has been said, "Salmonella infection in man and animals is the sum of the forces that have contributed to the contamination of human foods and animal feeds at various points in the production, processing, distribution, and final preparation as well as the physical condition, age, and stress of the host at the time of exposure" (11).

References

1. Merchant and Packer, Veterinary Bacteriology and Virology, Iowa State University Press. - 2. Blood and Henderson, Veterinary Medicine, Page 464. - 3. Edwards, Ewing, Enterobacteriaceae, 2nd Edition, Burgess Publishing Company. - 4. Ellis, E.M., Editor, Cultural Methods For Detection Of Animal Salmonellosis and Arizonosis, in press. - 5. Hibbs, C., personal communication. - 6. Josland, S.W. (1951) Aust. Vet. Journal 27, 264. Watts, P.S. and Wall, M. (1952) Aust. Vet. Journal 28, 165. - 7. Kahrs, R.F., Bentinch-Smith, J., Byorh, G.R., Bruner, D.W., King, J.M., and Lewis, N.F., Cornell Veterinarian (1972) 62, No. 2, 175-191. - 8. Richardson, A. (1973) Veterinary Annual 14, 6-10. - 9. Richardson, A. (1973) Veterinary Record, 92, 112-115. - 10. Sojka, W.J. and Field, H.I. (1970) Veterinary Bulletin, Weybridge, 40 (7) 515-531. - 11. Wilson, Walker and Pfow, Proceedings, Seventy-Fourth Annual Meeting of the U.S. Animal Health Association, 1970, Page 436.

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