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Keith Prasse

Iowa State University

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Incidence and Etiology of Gastric Ulcers in Swine

Keith Prasse*

INCIDENCE OF THE DISEASE

There seems to be either the acute, sudden-death syndrome or the subclinical, asymptomatic condition seen only at the time of slaughter. Generally only one pig in a herd dies, but losses do vary from a single pig to approximately 50% death loss do occur with stress from management or environmental changes, other changes, other disease, or immunization.

One slaughter house observation (representing the subclinical syndrome) reported a 53% incidence. Another similar study of 443 slaughtered hogs showed 86% abnormal cornification of stomach linings, which is an early stage of any beginning ulcer.

The age of the swine affected clinically varies from baby pigs to adult hogs, but the disease is most prevalent in 80 to 120 pound hogs. No significant differences in incidence have been noted between sexes or breeds.

The disease is found under varied forms of management. At first confinement was felt to increase the incidence. However, the disease is more frequently recognized in well-managed and well-cared-for herds. Conditons demanding or resulting in rapid growth seem to be a factor.

A 1963 report on the disease stated that variation in temperature is a factor in precipitating the clinical case. Most deaths occurred in late spring and early winter. Extreme cold or extreme warmth didn’t seem to be important, but rapid change in temperature did. It is interesting to note a statement made in the 1939 article: “There is a striking seasonal increase in incidence of swine stomach ulcers observed in late summer and early fall.”

ETIOLOGY OF THE DISEASE

The information available on the etiology of this condition is limited to suppositions by various authors, and to several experiments involving nutritional studies, feed processing, and feed additives. The most definite statements made regarding etiology concern infectious agents.

During the eight year slaughter house study ending in 1939 Streptococcus were repeatedly isolated from every active lesion. The authors stated that this organism was either the primary cause or a secondary invader of the ulcer. They chose to believe it was the primary cause. The question remains; was this ulcerative condition the same as is now being diagnosed? In recent reports infectious organisms are seemingly discarded as etiologic possibilities. Bacteriological examinations show no consistant pattern and no ulcers have been reproduced from isolated organ-

* Keith Prasse is a junior in the College of Veterinary Medicine.

170 Iowa State University Veterinarian
isms. If an infectious organism is involved, it is of low infectivity.\textsuperscript{9, 11, 1} A research report from Kansas indicated a fungi-ulcer correlation.\textsuperscript{5} Candida\textit{ albicans} was isolated from 47\% of all ulcerated stomachs and only 13\% of all the stomachs were with no ulcers. This study included 600 porcine stomachs. Another author reported \textit{C. albicans} as being consistently isolated from stomach scrapings, but it was not regarded as a primary etiologic factor of this disease.\textsuperscript{3}

Ulceration of the gastric mucosa is frequently encountered in chronic infections of other diseases such as hog cholera, swine erysipelas, necrotic enteritis and infectious gastroenteritis.\textsuperscript{8} Hyostrongylosis also results in ulcerative gastritis.\textsuperscript{4}

Regarding nutritional causes, deficiency of vitamin E was suggested by Kowalsczgr in 1960.\textsuperscript{9} Abnormal cornification found in slaughter animals is an early stage in ulceration. This suggests subclinical vitamin A deficiency.\textsuperscript{3} However, excess amounts of vitamins A, E, and K have no effect in lowering the incidence of swine gastric ulcers."

Deaths in swine due to gastric hemorrhage occurred during nutritional experiments on parakeratosis.\textsuperscript{9} One ration being used was high in calcium (1.3\% of the ration). Twenty-three of 35 pigs on this ration had ulcers. The author did not attempt to relate the calcium to the ulcers.

Another recent nutritional experiment was designed to determine the effects of heat-treated corn and gelatinized corn on daily gains and feed utilization.\textsuperscript{10} Death due to esophagogastric ulcers defeated the original purpose of the experiment. Three different groups of hogs were fed heat-treated corn. There was a resultant 39\% mortality. Necropsy of the remaining hogs at slaughter showed animals with less acute ulcers. The total ulcer incidence in the three lots was 50\%, 66\% and 50\% respectively. The control lots for the above received normal raw corn. No ulcers were found.

Another group of hogs was divided. Half were fed gelatinized corn. Half were fed gelatinized corn plus 1.5 mg thiamin, 50 micrograms of vitamin \textit{B}_{12} (both per pound of ration), and 5–10\% fish meal. There was no difference in incidence of ulcers between the two groups, but a total of 53\% of the hogs had ulcers. The machine used to heat treat and gelatinize the corn for these experiments was similar to that used in pelleting processes.

Shortly after this research was published an article appeared in a trade journal, \textit{Feedstuffs}, regarding the pellet machine mentioned above.\textsuperscript{5} It stated that the machine was not that of a standard feed company and that the corn had been ground to a meal before treating as well. At the same time other research from Kansas indicated pelleted feed did increase ulcer incidence.\textsuperscript{5} New research was done;\textsuperscript{5} Wisconsin workers studied over 4,000 swine stomachs at a slaughter house and found a 20\% incidence of ulcers. These hogs came from diversified conditions, but most of the farms did not feed a complete pelleted ration. Also, a feed company studied two groups of pigs with the only variable being pelleted feed and meal feed. They found no difference in ulcer incidence between the groups.

Corn and soybeans grown in dry or otherwise unusual seasons accumulate dangerously high levels of nitrates. Interference with vitamin A synthesis and metabolism caused from metabolism of these nitrates may result in epithelial damage and weakness at points of stress such as the esophageal portion of the stomach.\textsuperscript{11} Another causative theory suggested is as follows: Perhaps an inherited or genetic weakness of the esophageal area of the stomach in pigs unaccustomed to the severe stress of modern feedlot housing and concentrated rations is present.\textsuperscript{11}

The same authors who suggested the last two theories summarized their opinion about the etiology of this condition as follows. Most findings appear to support a toxic theory; the fact that the condition went virtually undiagnosed until the last ten years supports the toxic theory involving feed additives or feed components. Widespread use of commercially prepared feed and a recent change by the feed industry from animal protein to all plant protein may also point toward a toxic principle.

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Anaphylactoid Reaction To Strain 19 Brucellosis Vaccine in Calves

by KENNETH W. SCHUMANN, B.S.*

INTRODUCTION

Brucellosis is recognized as a disease affecting mainly domestic animals and as a mild to severe disease in humans. Transmission to humans is usually from the animal population; therefore control of the human form demands control in the domestic animals. In addition, the economic loss to the livestock industry due to Brucellosis is great, estimated at $25,000,000 annually.5

Since chemotherapy of the disease is relatively ineffective, prevention and eradication are the important means of control. With the discovery of the attenuated strain of Brucella abortus known as strain 19 by Buck,7 vaccination with this organism has become the standard prevention program. Calves are usually vaccinated between the ages of four and eight months.

Strain 19 is an attenuated rather than avirulent strain and has been known to cause the disease in cattle18 and humans.21,22 Vaccination with Strain 19 has been known to cause anaphylactic shock or an anaphylactoid reaction in calves.1,5,11,18

INCIDENCE AND OCCURRENCE

Anaphylactoid reactions to Strain 19 appear to be infrequent. I could locate no reports of this reaction in adult cattle. The reaction is peculiar to certain individuals but a higher incidence has been reported in certain herds or certain localities.11 It is not confined to the United States.

CLINICAL SIGNS

The anaphylactoid reaction usually comes on fairly suddenly, within a few hours after vaccination. A sudden and severe dyspnea develops accompanied by muscle shivering, weakness, and uneasiness. Edematous swellings are seen around the eye, udder, anus and vulva. Increased salivation, bloat and diarrhea may or may not occur. A drop in blood pressure and a decreased body temperature seem to be characteristic. Fluid sounds may be heard on auscultation of the thorax. The animals often die in a matter of hours but occasionally recover. A case of al-

* Ken Schumann is a junior in Veterinary Medicine at Iowa State.