Hemorrhagic Bowel Syndrome (HBS) in Swine

Historical reports of hemorrhagic bowel syndrome (HBS) in swine describe infrequent, explosive outbreaks of sudden deaths with intestinal hemorrhage and no apparent infectious cause. Hemorrhagic bowel syndrome does not have a single, known etiology nor are specific risk factors consistently associated with the deaths. A diagnosis of HBS is applied only after thorough efforts to rule out other causes of rapid death and intestinal hemorrhage have been completed. The most common differential diagnoses are intestinal volvulus (twisted gut), the hemorrhagic form of porcine proliferative enteritis (PPE, ileitis), gastric ulcers, bacterial toxemia (e.g. acute infections with *Salmonella* or hemolytic *E. coli*), or other causes of sudden death. Control requires that one accurately rule out other known causes of sudden death, objectively assess environment and feeding practices, and properly manage risk factors that may be present. Reports of specific, consistently successful therapeutic interventions are rare.

Objectives

- Review Hemorrhagic Bowel Syndrome.
- Outline a procedure for diagnosis of Hemorrhagic Bowel Syndrome.
- Present known risk factors and management practices for control of Hemorrhagic Bowel Syndrome.

Review and history

Sporadic cases of peracute intestinal hemorrhagic syndromes have been reported since the 1950's. Careful interpretation of historical reports is warranted since ulceration of the pars esophagea (gastric ulcers) and hemorrhagic forms of proliferative enteritis are not excluded in some discussions. In 1964, Kinnaird [1] described HBS appearance grossly and microscopically as a result of capillary “oozing” with no cause demonstrated. He suggested increased incidence with feeding of pelleted rations, with feeding liquid whey, or with high supplemental copper in diets.

In 1967, Jones [2] also described the lesions of an intestinal hemorrhagic syndrome associated with the feeding of liquid whey. Mortality was increased when feeding fresh whey compared to stored whey. Attempts to demonstrate an immune-mediated mechanism failed. Bacteriology yielded *E. coli* and *Clostridium perfringens* type A from intestine. Attempts to reproduce disease by administering filtered intestinal contents intravenously or orally failed. Others in brief reports were unable to reproduce disease by oral inoculation of *E. coli* and *Clostridium perfringens* type A isolated from affected swine. Most of the authors of these early case reports and studies did not find evidence for, nor did they suggest, infectious causation.
In 1970, O'Neill [3] proposed an allergic mechanism for an explosive outbreak in several sites in a single system but in this case not all pigs died and diarrhea was a feature. In 1972, Rountree suspected infectious causation but this outbreak, in retrospect, was probably PPE. In 1989, Ligget described an outbreak of hemorrhagic enteropathy that also had lesions typical for clotting dysfunction and was responsive to supplemental vitamin K.

A recognizable twist of the intestine (volvulus) is a variable finding [4-5] in some studies. In a Swiss report of 436 cases of HBS, intestinal volvulus was confirmed in 56% of cases examined in the first 8 years of the study; however, by more careful examination in the next 2 years it was found in 80% of cases [6]. Recent reports [7-8] implicate partial or complete volvulus as a common cause of death with pathological features typical of those described for HBS. Demonstration of a partial volvulus can be quite daunting in some cases but it is increasingly recognized in sudden deaths of individual animals.

Because known causes of similar presentation exist, some speculate that a diagnosis of HBS is actually a failure to accurately rule out other causes of sudden death with intestinal hemorrhage. Acute PPE and gastric ulcers can be overlooked. In some situations, necropsy of a dead pig is not high priority, is delegated to untrained personnel or simply may not be completely investigated. Some veterinarians suspect Clostridium sp. involvement akin to the jejunal hemorrhagic syndrome described in bovine, possibly associated with toxins elaborated by Clostridium perfringens type A. However, demonstrating the presence of bacteria that are normal flora in cases with intestinal hemorrhage does not confirm causation. Clostridia are capable of producing a variety of toxins and clostridial “blooms” probably can occur after engorgement. Antimicrobials are variably effective decreasing incidence of HBS in some herds and clostridia are not always demonstrable or isolated. Genetic tools are now in place to further characterize what role, if any, certain clostridia may have in HBS. Similarly, E. coli or other bacterial “blooms” where elaboration of exotoxins, enterotoxins or endotoxins may have localized effects can perhaps create shock-like conditions in the intestine. Others postulate direct irritation or hypersensitivity to some feed ingredient, additive, or metabolite. Immune-mediated hypersensitivity has been postulated but is very hard to prove. The infrequent cases of high mortality associated with feeding whey may be in these latter categories.

Clearly, the current perception of HBS includes several different clinical and pathological entities. Early reports described HBS as infrequent outbreaks of relatively high (up to 20%) mortality. In contrast, HBS as currently perceived is a common cause of mortality in herds in the US. Production systems attribute mortality rates of 0.1% - 7% to HBS in the grow-finish phase. Lower mortality and widespread occurrence, with lack of association with major changes in feeding practices suggest factors other than those implicated in early reports. Demonstration of a partial volvulus can be quite daunting and is likely to be inaccurately diagnosed as HBS in many cases. Hemorrhagic bowel syndrome should not be viewed as a single disease with a single cause. The algorithm provided as table 1 provides guidelines to diagnosis.

Clinical Presentation

Clinical presentation of HBS typically is finding a previously healthy pig dead between 2 and 8 months of age; most are rapidly growing pigs between 4 and 6 months of age (150 and 270 lb). The remainder of the group is asymptomatic. Perceptions and production records support a higher incidence of HBS in the summer months. Mortality rates are unpredictable with considerable variation between groups, ranging from none to 7%. On rare occasion, one may have the opportunity to observe clinical signs attributable to HBS. Affected pigs are reluctant to move, may vocalize because of abdominal pain, and stand with legs apart. Vocalization incites typical aggression by other pigs in the pen with considerable roughing of the affected pig. Open mouth breathing and generalized hyperemia is followed by recumbency, pallor, and death. Affected pigs are not observed to have diarrhea prior to death. Similar clinical signs are observed with intestinal volvulus.

Necropsy

Careful necropsy is necessary to rule out other causes of death before rendering a diagnosis of HBS. The first priority is to rule out intestinal accident such as volvulus. This is defined as a mechanical interruption of blood perfusion or drainage that may be partial or complete. Intestines that have compete obstruction of blood flow are black and necrotic (ischemic necrosis). Those with blood flow to the intestine but partial obstruction of blood drainage appear very reddened with unclotted, bloody fluid in the lumen. With intestinal accident, there may be a portion of the intestine twisted, the entire intestine may be involved, and fre-
quently may include cecum and colon. With the pig in dorsal recumbency, the abdominal wall is carefully
reflected so as not to disturb the position of intestinal tract. The normal position of the colon and cecum is
on the left side with the apex of the cecum pointing caudally. The cecum and colon may be rotated to the
right when there is partial or complete volvulus of the small and/or large intestine. Examination of the root
of the mesentery of small intestine may demonstrate a partial or complete twist or rather abrupt transition
in perfusion. The mesentery may be edematous and congested. In some cases of volvulus, there is a clear
transition of normal to affected intestine at the caudal flexure of the duodenum.

With HBS, intestinal loops are dilated with gas, flaccid and contain unclotted blood or dark bloody fluid.
The intestinal wall may have transmural congestion but not as severe as that observed with intestinal vol-
vulus. The cecum occasionally contains bloody fluid as well but normal feces are usually present in caudal
large intestine. Unfortunately these changes are not specific for HBS and can also be observed with volvu-
lus. The stomachs of pigs with volvulus have normal to large quantities of feed in the stomach. Stomachs
contain variable quantities of feed or water with HBS but can also be empty, similar to many of the other
common causes of death.

Rule out gastric ulcer, ileitis

Two other conditions most often erroneously diagnosed as HBS are gastric ulcers, and acute hemorrhagic
PPE (ileitis). One must completely examine the pars esophagea before ruling out gastric ulcer, particularly
when the intestine or colon contains dark or black blood. Acute deaths from gastric ulcer usually have car-
cass pallor without bloating. Blood and blood clots may be present in the stomach but the hallmark is ul-
ceration where esophagus enters the stomach. The stomach usually does not contain feed. Although blood
clots can be found in stomachs with acute hemorrhage from gastric ulcers, the blood that passes into the
small intestine is usually unclotted and quite dark to black by the time it reaches the large intestine. The
wall of the small intestine and colon is usually normal, although the mucosa is reddened.

The acute hemorrhagic form of PPE may have areas of terminal intestine suspiciously thickened, perhaps
with mucosal necrosis, but gross mucosal proliferation is not always discernible. In some cases the intesti-
ne is flaccid and dilated. The wall of the intestine is not as severely or diffusely congested as with HBS or
volvulus. The blood within the lumen appears fresh and often forms a rope-like clot, in contrast to unclotted,
dark bloody contents of HBS or volvulus. Diarrhea may precede death, and the stomach contains scant
ingesta with PPE. Histopathology, immunohistochemistry (IHC), or agent detection methods are required
to confirm PPE and differentiate HBS or volvulus in acute cases.

The role of intestinal accidents

History describes HBS as infrequent outbreaks of relatively high (up to 20%) mortality. In contrast, HBS
as currently perceived is a common cause of mortality in herds in the US. Production systems attribute
mortality rates from 0.5% to 7% to HBS in the grow-finish phase. Lower mortality and widespread occur-
rrence, with lack of association with major changes in feeding practices suggest there may other factors
to consider. Recent work strongly implicates intestinal accident (twisted intestine, volvulus, torsion) as
a major contributor to grow-finish mortality that may be wrongly referred to as hemorrhagic bowel syn-
drome. Straw, et. al. [7-8] have conducted several prospective studies, carefully examining sudden deaths
and risk factors. In one conducted at a test station with individual daily feed consumption records, six of 6
pigs with features of HBS were confirmed as intestinal volvulus. Moreover, five of the six had disruption of
feeding behavior (disruption of feeding schedule by handling, weighing, sorting or a feeder not working)
within one or two days prior to death. In this author's experience in test facilities, it is not unusual to find
occasional pigs dead the day following a disruptive event, consistent with observations of Straw. In an
other investigation of commercial grow-finish mortality, Straw found relatively higher incidence of sudden
deaths with summer placement, pigs approaching finish weight, and pigs fed no antibiotics but found no
relationship to other concurrent causes of mortality, to gender, or to pen density,

Mechanisms proposed for volvulus help to explain relatively high prevalence across herds and low in-
cidence within herds. Pigs usually eat several (7-13) discrete meals during daylight hours with relatively
long inter-meal intervals. It is speculated that pigs consume larger, fewer meals as they get older and are
predisposed to volvulus by a combination of mechanical and physiologic mechanisms. Larger meals, ex-
acerbated by feeding disruptions, may alter motility to increase the likelihood of volvulus. Altered feeding
behavior disrupts the pattern of neurologic control of intestinal movement. Body type conformation of longer and deeper carcasses may also contribute, along with pure mechanical effects of “pig play.” It is likely that many cases of (partial) volvulus are wrongly diagnosed as HBS in modern production systems.

**Laboratory assistance and other differentials**

Autolysis occurs rapidly in cases of HBS. Necropsy should be performed as quickly as possible and tissues immediately preserved in formalin and on ice. Microscopic examination of intestinal sections reveals profound transmural congestion, affecting serosa, intestinal wall, and mucosa. Antemortem necrosis and inflammation are not usually features but there may be mild superficial necrosis of mucosa or mild cellular infiltrates in some sections. Crypt hyperplasia is not present nor is *Lawsonia intracellularis* detectable by IHC. Occasionally there will be increased numbers of eosinophils in the lamina propria but significance of this finding is dubious in pigs. Morphologically distinct populations of bacteria are not a consistent finding with HBS. Microscopic lesions are similar and not specific for either volvulus or HBS.

Bacterial isolation attempts generally yield *E. coli* or *Clostridium perfringens* from intestines of dead pigs, including those with HBS. Most authors have not associated infectious causation with HBS yet it is believed by some that antimicrobial therapy often alters the incidence of disease. It is important to rule out infectious causes of blood in intestine that may threaten health of the group. These diseases include: porcine proliferative enteritis (ileitis, *Lawsonia intracellularis*); salmonellosis; coccidiosis; whipworms; swine dysentery (*Brachyspira hyodysenteriae*); *E. coli* by virtue of enterotoxin and endotoxin; and systemic bacterial diseases (*Erysipelas, Actinobacillus*, etc). *Clostridium perfringens* type C causes hemorrhagic necrosis but is limited to suckling pigs and not a true differential for HBS.

Differentiating postmortem autolysis, intestinal congestion, and circumstances of death requires experience and knowledge. Autolysis and intestinal congestion may yield blood-tinged intestinal contents and can be present with many other sudden deaths, including trauma from fighting, porcine stress syndrome, bacterial septicemia and meningitis, or bacterial enteritis.

**Other diagnostic considerations**

Hypersensitivities are occasionally implicated in cases of explosive outbreaks of sudden deaths and diarrhea in a group of pigs but is usually related to an abrupt feed change or the administration of a particular drug or antibiotic. One might speculate allergy, hypersensitivity, or direct irritation of intestinal tract by the agent or its metabolites. Diarrhea is a feature in some of the pigs in this situation and not all affected pigs are found dead with the lesions typical for HBS. The original descriptions of HBS were associated with feeding of whey and, indeed, still occur. Scrutiny of the history often reveals a change in the whey processing, dehydration, or storage of the material. Feeding of whey is considered a major risk factor for an increased incidence of true HBS cases. Several authors have investigated HBS associated with whey feeding but mechanism remains unknown.

A final diagnosis of HBS is made only after ruling out other causes of death. Because HBS cannot be experimentally reproduced, it is best viewed as a default diagnosis. Since the cause of HBS is not known and the literature is often confusing, it is useful to consider other causes by careful examination of carcasses, environment, and laboratory testing. The list of rule-outs is long, including: autolysis, infectious agents of enteritis or sudden death, and noninfectious conditions such as volvulus and gastric ulcers all previously mentioned. Other, less likely noninfectious causes of sudden deaths that may have intestinal congestion and / or blood in intestine include:

1. anticoagulant toxicity or vitamin K deficiency
2. nasopharyngeal hemorrhage, tumor, abscess, erosion
3. ulceration of glandular stomach from parasites, mycotic, nonsteroidal anti-inflammatory drugs;
4. inguinal or umbilical hernia with incarceration of intestine
5. ectopic mesenteric ossification with strangulation of a portion of intestine
6. rectal trauma usually does not cause sudden death
7. acute vitamin D toxicosis
8. mycotoxins are an unlikely cause and are poorly documented.
Risk factors and control

The risk factors most frequently mentioned by veterinarians involve disruptions of feed intake, feed or water quality, or change in social status. Factors contributing to inconsistent feed consumption include: feed system failure; out of feed; limited access to feed; limited access to water; with effects magnified in hot summer periods. Changes in the pig’s environment or social status might include: moving a group or change in pen location; social stresses of domination and competition; sorting or topping off pens at market; mixing pigs in the same pen; or perhaps a pig jumps a pen. Speculative feed or water constituents that may affect microflora or intestinal physiology include: high iron in water; high copper in rations; high (rancid) fat content; poor water quality; poor protein quality; abrupt changes in feed ingredients or ratios; mixing errors (mineral, salt, protein, etc); pelleted rations; and high protein rations. There are undoubtedly other “risk factors.”

There is little opportunity for therapeutic intervention for individual pigs with HBS because of the rapid clinical course. Outbreaks of classic HBS responds to removal of the suspected offending feedstuff. In current production systems, HBS must be carefully differentiated from intestinal accident or other causes of death. Because incidence is usually low, intervention with management and husbandry is attempted first. A cost/benefit analysis is warranted in most herds before aggressive intervention is pursued. Empirical methods of control have included the use of feed-grade antimicrobials. Evaluation of antimicrobial efficacy is difficult since in most herds, HBS tends to be transient and seasonal. Management changes to address the perceived risk factors is usually not expensive and often is beneficial. Incidence may be reduced by simply assuring a consistent, plentiful, accessible source of palatable feed and water, avoiding mixing of pigs, and providing additional floor space.

Summary

Historical reports of hemorrhagic bowel syndrome (HBS) in swine describe infrequent, explosive outbreaks of sudden deaths with intestinal hemorrhage and no apparent infectious cause. Hemorrhagic bowel syndrome has since become a default diagnosis applied to previously healthy grow-finish swine found dead with bloated abdomen and pallor, with bloody, thin-walled small intestines at necropsy, and death is attributable to no other known cause. It is an economic concern since such deaths are common across production systems, occur in healthy pigs late in the grow-finish phase, and incur intervention expenses. Clinical signs are rarely observed since death occurs rapidly. Diarrhea is not a feature of this condition.

Hemorrhagic bowel syndrome as currently defined does not have a single, known etiology nor are specific risk factors consistently associated with the deaths. A diagnosis of HBS is applied only after thorough efforts to rule out other causes of rapid death and intestinal hemorrhage have been completed. The most common differential diagnoses are intestinal volvulus (twisted gut), the hemorrhagic form of porcine proliferative enteritis (PPE, ileitis), gastric ulcers, bacterial toxemia (e.g. acute infections with *Salmonella* or hemolytic *E. coli*), or other causes of sudden death.

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References

Additional Reading