A presumptive case of vomiting and wasting disease in a swine nucleus herd

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Summary
An outbreak of vomiting and wasting disease was presumptively diagnosed in a 650-sow genetic nucleus herd in January 2002. Clinical signs included inappetence, coughing, and pyrexia in nursing sows, and vomiting, huddling, and pyrexia in piglets. More than 500 weaned pigs were euthanized because of anorexia and wasting. No clinical signs were observed in gestating sows or in pigs in the finishing barns. The clinical diagnosis was presumptively confirmed by signs in piglets and weaned piglets. However, the cause of the outbreak was not determined, as the herd had been seropositive for hemagglutinating encephalomyelitis virus (HEV) prior to the onset of clinical signs. Since February 18, 2002, there have been no clinical signs of HEV in the herd.

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vey dehydrated and die. Older nursing or weaned piglets become cachectic due to inadequate feed intake and may persist in a wasting state for several weeks before dying. The abdomen may be distended due to gaseous distension of the stomach and intestines.\textsuperscript{11}

The encephalomyelitic form may begin as a VWD outbreak.\textsuperscript{2} The first signs may include vomiting at 4 to 7 days of age, but this is rarely severe and pigs do not become dehydrated. Alternatively, piglets may show acute depression and huddling. Coughing and sneezing may be observed and the piglets rapidly lose weight.\textsuperscript{12} After 1 to 3 days, more severe clinical signs occur, including generalized muscle tremors, hyperesthesia, posterior paresis, and convulsions.\textsuperscript{2} Mortality may reach 100% in neonatal piglets. The clinical course in a herd is usually 2 to 3 weeks, coinciding with the time required for sows to develop immunity and pass this on to their offspring through colostral antibodies.\textsuperscript{2}

**Clinical report**

In January 2002, sows and piglets in a recently established high-health, 650-sow production nucleus herd (pursbred Landrace and Large White × Landrace) in southwestern Ontario began to exhibit anorexia, vomiting, fever, and coughing. The first piglets had been farrowed in October 2001.

The herd receives monthly veterinary visits. The sows are not routinely blood tested; serological testing is carried out only if there are clinical signs of disease. Each month, pigs in the finishing barn are serologically monitored for PRRS virus (PRRSV) (Idexx HerdChek ELISA; Idexx Laboratories, Westbrook, Maine), *Mycoplasma hyopneumoniae* (DAKO monoclonal blocking ELISA), *Actinobacillus pleuropneumoniae* (serotypes 1, 5a, and 5b; ELISA) and toxigenic *Pasturella multocida* (ELISA). At the time of the outbreak, the sow herd was seronegative for *M hyopneumoniae*, *Actinobacillus pleuropneumoniae* (serotypes 1, 5a, and 5b), TGE virus (TGEV), porcine respiratory coronavirus (PRCV), toxigenic *Pasturella multocida*, and swine influenza virus (SIV) H1N1, and seropositive for PRRSV (vaccine strain), *Lassoia intracellularis*, *Streptococcus suis*, SIV H3N2, and porcine circovirus type 2 (PCV2). The breeding animals in the herd are vaccinated against PRRSV, parvovirus, six *Leptospira* serovars, *Erysipelothrix rhusiopathiae*, and *Escherichia coli* during each parity period. The sow barn is located in a wooded area 2 km from the nearest swine unit. Farrowing rooms are operated on an all-in-all out basis. Piglets receive no vaccines, and are weaned at 14 to 17 days of age and transferred to a 2250-head oosite nursery located 14 km away. After approximately 8 weeks in the nursery, they are transferred to one of two 2000-head finishing barns located 18 km from the sow herd and approximately 12 km from other unrelated swine facilities.

Biosecurity is excellent. Replacement animals are housed in an isolation facility for 6 to 8 weeks and tested serologically (PRRS ELISA) before entering the main herd. Personnel entry is restricted: all staff and visitors must shower and change clothing before entering the facilities. Transport trailers delivering or collecting animals are washed, disinfected, and dried before arrival. Supplies and equipment are removed from exterior packaging, disinfected, or vomiting. Approximately 50% of the nursing sows were severely anorexic, and 15% to 20% were coughing and febrile, with rectal temperatures of 40°C to 41°C. Gestating sows appeared unaffected. On the same day, similar signs were observed in nursery pigs that had been weaned on January 24. Four weaned piglets exhibiting clinical signs were euthanized, and tissue samples and sera were submitted to AHL.

On the basis of the respiratory signs and anorexia in the sows and the vomiting and fever in the piglets, the differential diagnoses at this time included TGE, swine influenza, and PRRS. Lung tissue was negative for influenza virus type A by fluorescent antibody testing, and both lung and serum were negative for PRRSV by reverse transcriptase-polymerase chain reaction (PCR). Pooled lung samples were weakly positive for PCV2 by PCR. There was no significant bacterial growth in lung, liver, spleen, or kidney. Histologically, there was mild chronic interstitial pneumonia suggestive of viral infection, and renal tubular necrosis secondary to dehydration. There was no histologic evidence of infection with SIV or of bacterial pneumonia-septicemia. No virus was isolated from lung samples.

Breeding-stock sales from the nursery and finishing barns ceased until the etiology of the problem could be determined. Between January 21 and 28, approximately 50% of the affected sows and piglets were treated with procaine penicillin G (30 mg per kg, IM, once daily) and the remainder were injected with oxytetracycline HCl (10 mg per kg, IM, once daily), with no response to either treatment. On January 29, acetyl-
salicylic acid (ASA) was administered to anorexic sows in the drinking water, and their piglets were injected with flunixin meglumine (8.3 mg, IM). Within 24 hours, pyrexia had resolved in pigs and sows. Some sows were already agalactic and were weaned within 3 to 8 days of farrowing. Affected litters were supplemented with liquid milk replacer and creep feed.

On February 4, the herd veterinarian visited the sow herd and nursery. Coughing in the nursing sows had resolved. The only affected litter in the newly farrowed piglets was in a room into which older affected piglets had been fostered onto newly farrowed sows: this was not the normal practice. Nursery pigs weaned after January 24 had received ASA in the drinking water between January 30 and February 2. Piglets were thin but bright and alert. Postweaning feed intake was much lower than normal.

On February 18, 510 weaned piglets in the nursery were euthanized because of anorexia and irreversible wasting, and blood samples for convalescent titres were collected from 30 sows that had exhibited clinical signs. Sera collected on January 7 were used for acute titres. Because of the severe clinical signs in the weaned piglets, HEV infection was added to the list of differential diagnoses. Sows were seronegative for TGE-PRCV (differential ELISA), M. hyopneumoniae, and SIV H1N1, and seropositive for PRRSV and SIV H3N2. Reciprocal HI titers for HEV ranged from 16 to 4096 in the sow tested on January 7, with titers of 1024 or greater in 11 of the 14 sows tested (78.6%). In the February 18 samples, titers ranged from 32 to 16,384, with titers of 2048 or greater in 21 of the 30 sows tested (70%) (Figure 1). Statistical analysis (paired t test; STATISTIX 7.0; Analytical Software, Tallahassee, Florida) revealed no significant difference in sow HEV HI titres before and after the outbreak.

The lactation-feed sample tested negative for vomitoxin.

On the basis of clinical signs, the presumptive diagnosis was VWD caused by HEV. No further clinical signs have occurred in the herd. Clinical signs related to the outbreak were never observed in animals in the breeding-gestation barn on the same site as the farrowing barn or in the finishing barns. Breeding-stock sales resumed in March 2002.

**Discussion**

In this case, two confounding issues complicated the diagnosis of HEV. First, the initial clinical sign, anorexia in lactating sows, was consistent with the anorexia that had been occurring intermittently since farrowings had begun in October. Second, the mild cough heard in the nursing sows was attributed to the adverse environment caused by malfunction of the ventilation system. It was only when piglets also began to exhibit clinical signs that it became obvious that the problem was caused by an infectious disease rather than a nutritional or environmental problem.

Hemagglutinating encephalomyelitis virus is transmitted via nasal secretions and replicates in epithelial cells of the nasal mucosa, tonsil, lungs, and small intestine. After local replication, the virus spreads from the peripheral nervous system to the central nervous system. Vomiting associated with the disease is presumed to result from altered function of vagal and gastric intramural plexus neurons secondary to viral infection. Persistence of VWD is believed to be due to viral-induced neuronal death and delayed stomach emptying probably plays an important role in development of wasting.

Virus can be isolated from the tonsils and respiratory tract during the incubation period, which lasts for approximately 5 days. Isolation of HEV may be attempted on tonsil, brain, and lung samples from affected piglets. However, sensitivity is low unless samples are collected from acute cases within 1 or 2 days of the onset of clinical signs. It is suspected that hemagglutination inhibiting and seroneutralizing antibodies preclude virus isolation at later stages of the disease. In this case, the piglets sampled were no longer in the acute stage of the disease, and even if they had been infected with HEV, the virus might not have been recovered from the lungs.

Because of the widespread subclinical nature of infection, paired sera must be collected to demonstrate a fourfold increase in titer in convalescent samples. Acute sera must be collected as soon as possible after the onset of clinical signs, as high antibody titers develop rapidly. In this case, paired sera were not collected. The initial samples were collected randomly from gestating sows for routine monitoring, whereas the follow-up samples were collected specifically from sows that had been clini-
cally affected during the outbreak. Although some sows tested in the second set of sera had HEV titers higher than those in the first set of samples, this could be explained by laboratory variation or small sample size. Since the clinical signs in the weaned piglets were consistent with HEV infection, a presumptive diagnosis was made.

Respiratory signs in sows related to an outbreak of VWD have not been reported in the literature, although Greig and Girard observed anorexia in sows in affected herds. However, coughing was also observed in sows as an initial sign of a VWD outbreak in another Ontario herd in 1986 (K. Richardson, written communication, 2005).

The positive PCV2 PCR on lung tissue from nursery pigs was not unexpected. The gilt in this herd had been purchased from a PCV2-positive herd, and many pig herds in Ontario are seropositive. Porcine circovirus type 2 has been implicated in postweaning multisystemic wasting syndrome (PMWS), in which clinical signs, including weight loss, emaciation, and CNS disturbances, resemble those of HEV. The literature clearly supports enhanced disease in growing pigs concurrently infected with PCV2 and other pathogens, and PCV2 might have been a cofactor in this case. However, nursing piglets in this herd showed clinical signs, and nursing piglets rarely are affected by PMWS, no additional tests for circovirus were performed.

The reason for the apparent outbreak of VWD has not been determined. Serological results from January 7 indicated that the sows were already HEV-seropositive prior to the outbreak. Since the breeding herd was very young at this stage (all gilts or parity-one animals), it may have included both seropositive and subclinically infected animals and naive animals, even though all had been purchased from the same source. Replacement gilts entered the herd on November 7, 2001, and it is possible that the animals might have been recently infected with a more virulent strain of HEV. However, as the incubation period of HEV is only 4 to 7 days, the timing of the outbreak is not likely to have been associated with new additions to the herd. Another possibility is that the virus was transmitted by fomites. In common with other coronaviruses, HEV is most stable at low temperatures, and in winter, the virus can survive for extended periods of time on boots, clothing, transport trailers, and other fomites. However, because of the excellent biosecurity on the farm, fomite transmission is unlikely. The third possibility is that the ventilation breakdown triggered the outbreak, and this may explain why the nursing sows, and not the gestating sows, exhibited clinical signs. Whether the environmental changes acted as a stressor and cofactor to induce clinical disease is difficult to prove or refute.

The duration of clinical signs (3 weeks) was consistent with that reported in the literature. Despite the severity of the problem, preweaning mortality did not exceed 12% during the outbreak. This differs from reports in the literature of significantly higher mortality, approaching 100% in affected litters. The excellent nursing care provided by barn personnel (eg, antipyretics and milk supplementation) contributed to the low mortality in the nursing piglets. However, due to the nature of the disease and its impact on gastric function, it was necessary to euthanize a large number of affected piglets in the postweaning period. Approximately half of these animals were potential breeding stock; therefore, the economic loss to the farm was significant.

Implications

- Hemagglutinating encephalomyelitis virus should be included as a differential diagnosis when piglets exhibit vomiting and pyrexia.
- When unusual clinical signs suggesting HEV infection occur in a swine population, diagnostic sampling should be initiated during the acute phase of the disease, as a definitive diagnosis cannot be made without paired serum samples.

References

* Non-refereed references.