CASE REPORT

Acute feed-related selenium toxicosis in weaner pigs: A case report

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Summary: *Acute feed-related selenium toxicosis was observed in pigs consuming a commercial diet containing 100 ppm selenium. Clinical signs of a central nervous system disorder appeared 7 days after starting on the diet. Chemical analysis of the feed and pig tissues was necessary to diagnose the disorder. Symptomatic treatment of the affected pigs was ineffective and the group showed signs of improvement only after the offending feed was replaced.*

Clinical signs of selenium toxicosis can be similar to other disorders more commonly expected in the postweaning period. However, chemical analysis of the concentrations of selenium in feed and in porcine tissues is not a normal part of a diagnostic workup for central nervous system (CNS) signs. This case illustrates the need to go beyond the normal range of diagnostic tests when presented with CNS disorders.

Three hundred 28-day-old crossbred pigs were weaned and started on a commercially pelleted 21% protein starter diet. Seven days later four pigs developed CNS disorders. The affected pigs appeared hyperesthetic, expressing signs of muscle twitching and squealing when approached. Rectal temperatures of the affected pigs were normal to slightly below normal. The course of the illness for the affected pigs lasted 2 days. Pigs were found resting on their sternum, but progressed within 24 hours to lateral recumbency and paddling. Within another 24 hours, all pigs showing CNS signs were dead. The remainder of the group was lethargic and average daily feed consumption was less than 0.1 lb (.045 kg) per head.

We checked the waterlines, which we found to be delivering adequate amounts of water. When different feed was offered, the pigs showed increased interest and lack of appetite was not a problem with the new feed. Activity of the group in general returned to normal 2 days after the feed was changed. Selenium concentrations remained high in some pigs, however, and eight additional cases developed within 4 days of the feed change.

Necropsy of the affected pigs revealed no significant gross lesions. Histopathology of the brain tissue revealed cerebral edema with swollen astrocytes and vacuolation. No inflammatory lesions were present in the brain, liver, or intestinal tissue of the pigs showing CNS signs. Bacterial isolation of *Streptococcus suis* from the lung and brain of one pig lacked the corresponding histopathologic lesions to suggest meningitis. Additional testing ruled out pseudorabies virus, hemagglutinating encephalomyelitis virus, bacterial infections, water deprivation, organophosphate toxicosis, and chlorinated hydrocarbon insecticide toxicosis. Feed and liver tissue were analyzed for selenium. Liver samples contained 27 and 52 mg/g wet weight of selenium. Selenium concentrations in normal swine liver range from 0.2–0.6 mg/g.[1] The feed contained 100 mg/g wet weight of selenium — the maximum tolerable concentration of dietary selenium is approximately 2 mg/g.[2]

Discussion

Selenium is an essential but potentially toxic trace mineral for swine.[3] The United States Food and Drug Administration (FDA) currently limits the concentration of supplemental selenium in swine starter diets to 0.3 ppm.[4] Acute selenium toxicosis is generally not a problem under most swine feeding programs, but may occur when feed is poorly mixed, premixes are weighed inaccurately, or premix amounts are miscalculated, resulting in higher-than-normal selenium concentrations in the diet. Toxicosis due to excess selenium in the feed has been reported periodically.[1,5,–7]

The effects of ingesting a large quantity of selenium (> 20 ppm) include feed refusal, weight loss, respiratory distress, spinal paralysis, incoordination, hair loss, and death.[8] The course of the disease varies from hours to days, depending on the amount and toxicity of the selenium ingested. Extended exposure to dietary selenium values slightly greater than 5 mg/g may lead to subacute or chronic toxicosis. Acute selenium toxicosis develops at concentrations of 10–25 mg/g and pigs are not amenable to treatment.[8]

Based on the findings of excess concentrations of selenium in the liver tissue and feed sample, we concluded that the clinical CNS signs, which were consistent with selenium toxicosis, were due to excess selenium in the diet.

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Implications

- Good feed manufacturing processes are very important to avoid selenium toxicosis.
- When selenium toxicosis is suspected, it is important to differentiate the clinical signs from the more commonly suspected postweaning conditions that cause CNS disorders.
- If selenium toxicosis is suspected, establish the source and concentration of exposure to determine the prognosis and treatment regime. This includes a full chemical analysis of both the feed and liver tissues of the affected pigs.
- Chronic selenosis may be treated by feeding 40 ppm arsenic or 50-100 ppm arsenilic acid, which enhances the biliary excretion of the selenium.[8]
- The best treatment approach is to remove the offending feed.

References

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