An acute case of iron toxicity on newborn piglets from Vitamin E/Se deficient sows

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Summary

A 270 sow herd cut down feed costs by lowering Vitamin E and Selenium that was changed from an organic to an inorganic form. Several months later two week newborn batches were affected by a deadly hemorragic clinical entity developed 6 hours after a normal iron dextran injection. The brand of the iron came from a 2 year supplier, a world renown manufacturer. A therapeutic diagnosis was applied satisfactorily.

Key words: *vitamin E, selenium, toxicity, piglets, deficient, sows.*

Case of interest

In the most intensive swine producing region of Colombia, a sudden 80% mortality outbreak occurred on one day old piglets from a two site farrow-to-finish production system of 270 sows, that normally produce week batches close to 110 pigs. This herd uses a cross of American Danish Landrace boars (fast growth genetic breed) on Dekalb® 45 females. The diet tended to fit Kansas State University suggested requirements for gestating and lactating sows. The farm was under preventive measures applied elsewhere for enzootic diseases like parvovirus, *E. coli* and *Mycoplasma hyopneumoniae*. Mandatory vaccination against Foot and Mouth Disease-FMD and Classical Swine Fever-CSF were properly implemented as it is ruled by animal health regulations issued by the colombian government. Also, because the region has recently had outbreaks of vesicular stomatitis, the vaccination against this virus was introduced one year before this clinical case. The herd had a negative status to PRRS virus, *Actinobacillus pleuropneumoniae*, and five serovars of *Leptospira*, under the Colombian Association of pork producers health serologic monitoring program, sponsored by the National Pork Producers Council Colombian-Like-Agency fund. The pig processing included an injection of 200 mg Iron dextran product as it is used elsewere. The presence of Aujeszky, TGE and Porcine multisystemic wasting syndrome-PMWS are not declared to date, in Colombia.

Clinical findings

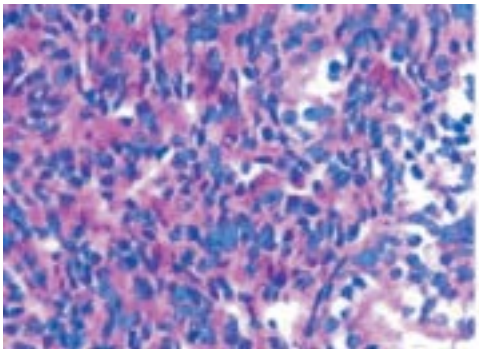
The farrowings were synchronized, so there were batch farrowing and the piglets developed these clinical signs 6 hours following iron injection. Initially the piglets showed dysnea, turned pale and many of them got icteric and there were no fever or abnormal secretions. Also, those signs described on iron toxicosis like, hypothermia, anorexia and oliguria before dead (1), were observed. Histopathologically, a vascular congestion syndrome was seen in most organs.

Pathological findings (significant lesions).

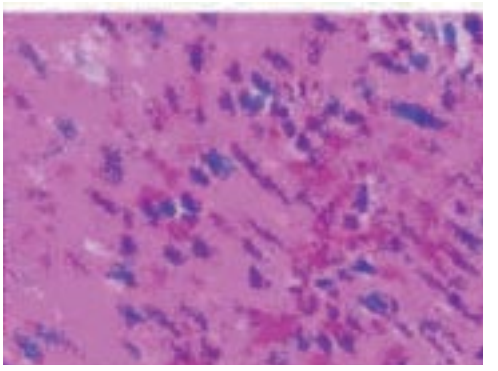
Gross lesions

The carcasses were jaundiced as well as the lymph nodes and muscles. Petechiation and ecchymosis were wide spread on most organs, even the lumen of the gall bladder and the stomach. A large inflammation was seen on the iron site of injection, despite the fact that the dose was properly given (2ml, IM) and the product met the normal and legal requisites to be used in Colombia and worldwide.

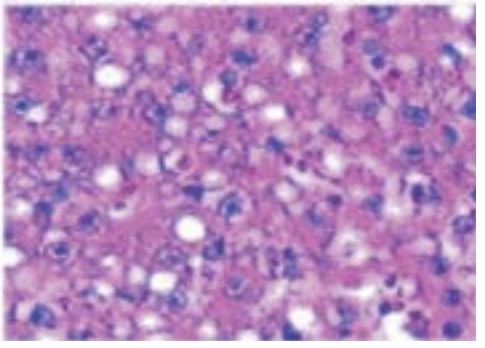
Microscopic lesions



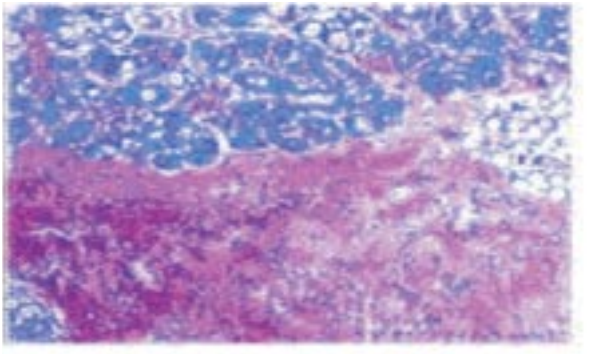
Lungs. Subacute instersticial pneumonia with a thickening of alveoli septae, capillary congestion and variable degree of necrosis of pneumocytes type 1 and proliferation of pneumocytes type 2.



Heart. Hemorrhages in miocardio and pericardio with hemosiderin and a lymphocitary mononuclear infiltrate, severe degree of degeneration of miocardial fibres, where even, there was evidence of fragmentation. In the miocardio and the pericardio there were inflammatory mononuclear infiltrates and very active macrophages, which is a finding compatible with vit E and selenium deficiency.



Liver. Difuse hepatocitic tumefaction as a consequence of a change on fat degeneration with intracytoplasmic vacuoli of variable size. Very often were seen accumulation of foci made up of biliar pigment, that induce a process named as intra and extra hepatocitic cholestasis. This findings are compatible with a difuse fatty change with cholestasis.



Páncreas. Intersticial areas of multifocal hemorrhages which means pancreatic hemorrhagic necrosis 6 hours after iron injection during the first day after birth.

Diagnosis

This diagnose was emitted as *Iron toxicity in piglets born from vitamin E and selenium deficient sows*, based on indirect evidence as follows: 1) there were clinical signs compatible with those seen on iron toxicity, 2) It was observed compatibility of microscopic tissue lesions with those described for vitamin E and selenium deficiency, specially on the cardiac muscle, 3) It occurred even by using the same dose and brand of iron dextran complex that was being used as a routinely procedure on this farm. Taking together the above reasons, these observations provided enough ground to eliminate the commercial product labeled and registered in Colombia like iron dextran, as a causal agent. To test this hypothetical or presumptive diagnosis, pigs were injected with the expected iron dextran dose split in three aliquots, to complete 200 mg within a total volume of 2 ml every three days. Like a proven evidence, none of the treated piglets with the split dose, developed symptoms, which means that all piglets started to born during the outbreak, unable to assimilate a full dose of iron dextran that were being routinely applied at this herd and worldwide.

One fact that probably contribute to explain this toxicity was a sudden reduction on the breeding herd diet of vitamin E and selenium as follows: 1) vitamin E, from 120 gr/ton at 50% to 80gr/ton at 50%, which means a reduction of 60 mg/kg to 40 mg/kg and, also, 2) a half reduction of a level of selenium proteinate (600mg/ton), replaced by inorganic selenium, as a way to lower dietary costs.

Mahan DC, and Vallet JL (3), recalled the knowledge about low levels of vit E and Se on diets of gestating sows, and previously reported by Tollez (4) that stated that there are susceptible pig progenies when the dams had low content of µ-tocoferol and selenium on their colostrum causing iron toxicosis, especially on piglets from old sows.

Treatment

Most animal species including swine are tolerant to high iron levels, and toxicity could occur by minerals interactions or by concentration of free radicals processes when oxidative reactions are interfered (2). Based on a report that vitamin E gave protection against iron toxicosis in mice and young pigs (4), several prescription measures were adopted like 1) bringing back the same dietary regime applied and changed 4 months before, 2) All sows were injected with vitamin E/selenium preparation by IM route, 1 week before parturition, during it, and 1 week after farrowing, 3) The split iron dextran dose application on piglets was added as a controlling measure during the outbreak, and besides, every piglet was injected at birth and at weaning with a vitamin E/selenium commercial preparation.

Conclusions

It was feasible that a change on the breeding herd dietary regime was a major cause to start up this outbreak. The correction of the diet brought back this herd to normality. It wasn't determined what was the contribution of selenium as a mineral and vitamin E, neither genetics, on this clinical case. The treatment and preventive measures were appropriate, according with the consistent disappearance of the clinical signs during the following 12 months.

Resumen

Un caso agudo de toxicidad por hierro en lechones hijos de cerdas deficientes en vitaminaE/ selenio.

Un hato de 270 cerdas bajó el costo de alimento, mediante el cambio por reducción vitamina E y selenio de forma orgánica a una inorgánica. Varios meses después, los lechones recién nacidos de dos semanas del parto, se afectaron de una entidad mortal hemorrágica, 6 horas después de la inyección con hierro dextran. El producto provenía de un proveedor de dos años, reconocido mundialmente. Se aplicó un diagnóstico terapéutico con resultados satisfactorios.

Palabras clave: *cerdas, deficiente, lechones, selenio, toxicidad, vitamina E.*

References

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