Prevalence of ergot toxicity

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Ergot toxicity or ergotism is caused by ingestion of cereal or grass grains infected by the fungus Claviceps purpurea.

The toxic agents (alkaloids) are produced by the fungus Claviceps purprea which attacks the embryo of the developing kernel forming a sclerotium, a black tangle of vegetative fungal strands. The sclerotium may contain more than 40 different fungal alkaloids.

Ergotism is an ancient and well recognised condition in both humans and animals. In poultry, the consumption of feeds containing sufficient amounts of ergoty grains causes many signs. Signs of blackening of beaks, claws and feet, followed by necrosis of these parts are the most obvious.

Ergot poisoning had been a major problem in livestock in the past. However, due to better management and safety checks in the feed manufacturing sector, the outbreaks of ergotism have declined in recent years.

Consequently, even though the older producers may have experienced the problem first hand, the newer generation of professionals in the livestock sector are most likely to know ergotism only from textbooks.

Nonetheless, the kernels infested with ergot are still present in many commonly used feed grade grains, albeit at a low level.

In this context, the possibility of exposure to ergot with potential health consequences should not be underestimated.

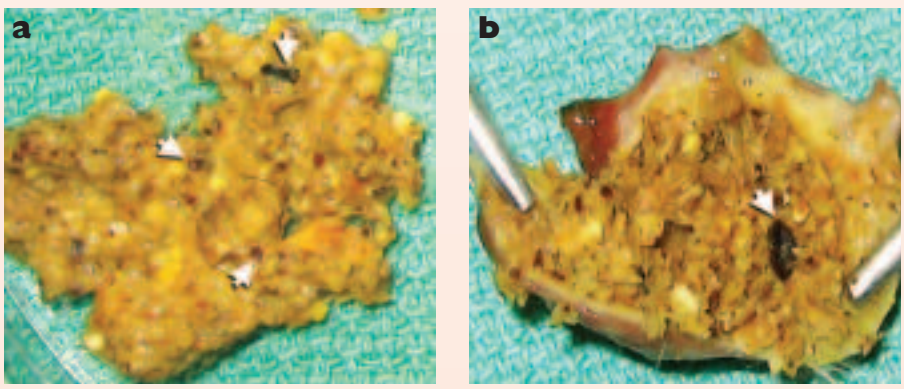
Here we report a recent experience where the cause of mortality was eventually

Fig. 1. Poultry feed. The yellow arrows point to numerous ergot infested, dark brown wheat kernels.

associated with ergot poisoning, even though initially ergot toxicity was not considered. The experience gained from these cases illustrate that even at a low level of exposure, mortality due to ergotism may occur in an otherwise normal commercial setting and an in depth investigation is worthwhile.

Furthermore, our cases demonstrate that ergotism may be more prevalent than apparent and hence, alerting the poultry growers to this seemingly dated and forgotten condition is necessary. Both cases were

Fig. 2. Ergot infested kernels (white arrows) were found in the crop (a) and gizzard (b).



submitted to the necropsy laboratory of the Prairie Diagnostic Services in Saskatoon, Saskatchewan, Canada.

Case one

In mid July of 2004 the owner of a freshly placed flock of broiler breeders noticed that the feet of several five day old chicks turned purple to black. Some birds died within the next 14 hours.

He submitted four dead and three live birds for examination. All live birds were reluctant to move. Their feet were uniformly dark reddish purple and slightly dehydrated. A few black grains, considered to be ergot's sclerotia, were found in the crops and gizzards of some birds.

No other lesions were found in the affected birds. A random feed sample, obtained a few days later, showed numerous sclerotia throughout the sample.

The sample was analysed by the reference laboratory (University of Missouri, Columbia, USA). The levels of ergopeptaine alkaloids were 8.08ppm, which was approximately 40 to 80 times higher than considered acceptable.

Case two

In mid November of 2004 the owner of a freshly placed commercial broiler flock noticed that the claws, toes, shanks and beaks of several three day old chicks were purplish-black. The feed (Fig. 1) contained many black kernels characteristic of ergot-infested wheat.

The ergot sclerotia were also found in the crops (Fig. 2a) and gizzards (Fig. 2b) of the birds investigated in the present case study. It is noteworthy that the feet of the chickens that died of causes not related to ergot (Fig. 3a) appeared normal. Those that ingested ergot infested grains had dark purple toenails (Fig. 3b) and in more advance cases necrotic toes (Fig. 3c). Similar differences were apparent in the beaks of the birds that died of ergot unrelated causes (Fig. 4a) versus those from ergot exposed birds (Fig. 4b).

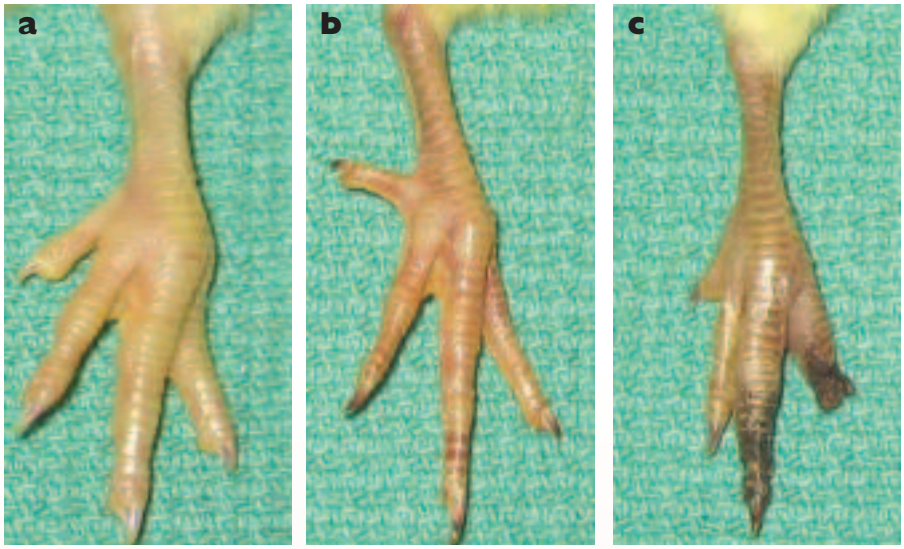


Fig. 3. A foot from a normal chick (a) is contrasted with the feet of ergot exposed birds. Moderate exposure (b) is marked by multifocal browning of the digit and toenails. Extensive and severe necrosis of the digits (c) indicates heavy exposure to ergot alkaloids.

Canadian research from the 1960s suggested that the dietary level of ergot of less than 0.3% is non-toxic to chicks.

Levels above 0.4% may cause varying degrees of growth depression and mortality. The effect of ergot toxicity on broilers was further studied in the early 1970s; this work essentially confirmed the previous findings.

Research in broilers concluded that for practical purposes no chicken feed should contain more than 0.3% of ergot.

The common practice of supplementing the poultry feeds with whole grains may, however, violate these recommendations as individual birds may easily consume a lethal dose of toxin concentrated in just a few kernels.

This is especially true for the chicks whose low body mass and natural propensity to select differently coloured grains may set the stage for acute ergot toxicity.

The cold and wet weather during the growth and maturation of cereal grains is another factor adding to the dynamic of ergotism. Such conditions lead to the proliferation of Claviceps purpurea and may have been instrumental in the cases presented here. The overabundance of inexpensive, poor quality grains and the ever present desire to economise poultry production may translate to more cases of ergot poisoning.

In view of the above dynamics it seems reasonable to consider ergot toxicity, while investigating the causes of under performance and/or mortality in chicken flocks. ■

Fig. 4. The creamy pink beak of a normal bird (a) provides a stark contrast to the purple beak of an ergot exposed bird (b).