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Short communication

Outbreak of aflatoxicosis on a local cattle farm in Pakistan

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Abstract

Mycotoxins are the metabolites produced by some fungi and their consumption instigate deleterious effects on human and animals health. This report describes 45 cases of aflatoxicosis in a bovine herd, which were being fed corn rich forage. Clinical signs were anorexia, depression, photosensitization and diarrhea. Fifteen animals died and were operated for necropsy. Postmortem lesions included, hemorrhages on viscera, blood exudation from natural orifices and prolapse. Gross lesions were mainly on the hepatic portal system and generalized anasarca was evident. Laboratory culturing of feed samples showed presence of *Aspergillus flavus* and *Aspergillus parasitticus*, while the toxin level of aflatoxin B1 (AFB1) was as high as 33,500 ppb. In conclusion, moldy and suspected diet must be tested for aflatoxin. Differential diagnosis of aflatoxicosis requires consciousness of geographical location, past events, provender testing and clinical signs. Feed withdrawal and supportive therapy helped in the disappearance of signs and mortality. This report is first field outbreak report of aflatoxicosis in the bovine herd from Pakistan.

Key words: Aflatoxin B1, Cattle, Treatment, Fatty liver, Hematuria, Acute death

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Introduction

Livestock is an important sector contributing in Gross Domestic Production [1]. Ever-present stresses that affect animal productivity include, environmental extremities, low quality nosh, toxin contaminated foodstuffs, diseases and poor management. Cattle provisions possibly will be contaminated owing to preen of microbes i.e. viruses, bacteria, molds and yeast etc. and their toxins i.e. endotoxins, exotoxins, mycotoxins etc. Mycotoxins are ubiquitous and different genera of fungi produce them under optimum conditions of humidity, temperature and suitable substrate [2]. Not all but some mycotoxins induce acute and chronic toxicity in host depending upon the extent, concentration and duration of exposure, age, health and nutritional status of animal [2, 3]. Back in 1953, a disease was suspected to cause death in cattle and swine by ingestion of moldy corn. The cause of death was a toxic substance from *Aspergillus* and *Penicillium* fungi. This toxic substance was purified and later named as Aflatoxin [4-6]. Previously the effect of mycotoxins was well studied on monogastrics i.e. pigs and poultry, but today its importance for bovines is well recognized [1]. Some mocotoxins become inactive in rumen because microflora can degrade them [7-9]; but few of the degraded metabolites of ingested toxins are more toxic than parental toxin [10]. Aflatoxins produced by molds of Aspergillus species, are of most interest because of acute toxicity and oncogenic effects in susceptible host, however aflatoxin B1 (AFB1) is most prevalent causing aflatoxicosis [11]. Aflatoxin causes direct animal losses, production losses and trade limitations, eventually affecting the economy [7].

The objective of this study is to present salient clinical signs, postmortem lesions and treatment strategies to enhance early diagnosis and management of aflatoxicosis. Keeping in the view above mentioned importance, the present report is being presented to find out the common cause of aflatoxicosis in local bovine herds. Determination of type and level of aflatoxin has also been considered as part of this investigation. Literature review has been presented to increase the suspicion index of this toxicity. Although, there is published data on aflatoxicosis in ruminants, this report is the first of its type from Pakistan.

Materials and Methods

In the present case, death of 15 animals have been narrated out of 45 cattle, including calves and heifers, present on a local farm in Okara, Punjab, Pakistan. Animals were routinely being fed ¾ green fodder and ¼ dry wheat straw. It was conveyed that animals

have received total mixed ration (TMR), containing corn and showed signs of diarrhea and acute death was evident in 5 animals within 3 hours of feeding, 10 died afterwards. Clinical signs varied with age, younger animals exhibited acute signs and all under the age of 1 died; death was reported in older animals too but they looked slightly resistant. Generally, animals exhibited signs of depression, staggering gait, anorexia, inappetence, colic, epistaxis, diarrhea, prolapse and finally death. Owner fed acidic vegetable pickle to lower the effect of toxins, probably by increasing the rumen acetic acid level. Autopsies of dead and slaughtered animals were performed, and pathological findings of carcass were recorded. Animals were suspected to be died due to aflatoxin ingestion because mold was found in provender and it was later confirmed by laboratory test. Feed samples were taken and *Aspergillus flavus* and parasiticus Agar (AFPA) was used as a growth medium [12]. Isolated samples were further analyzed for estimation of Aflatoxin via thin layer chromatography and spectrophotometry as recommended [13]. Treatment included fluid therapy and feeding of toxin binders, multivitamins and protein concentrate, antibiotic therapy was also used in remaining animals to diminish the threat of secondary infection in immunosuppressive conditions.

Results and discussion

The death was declared in 5 calves within 3 hours of feedstuff ingestion. Due to this peracute effect owner suspected mold toxicity and withdrawn feed and replaced it with fresh fodder. Animals were reluctant to eat and in the next 12 hours 7 more animals died. Animals were treated with fluid therapy, antibiotics (to avoid secondary infection), multivitamins and minerals as immunobooster. Three more animals showing severe condition died during treatment. Remaining animals were successfully recovered later.

Clinically affected animals affirmed anorexia, depression, inappetence, photosensitization, lethargy, diarrhea and mild rectal prolapse (Fig. 1). Analysis of feed specimens confirmed growth of *Asprgillus flavus* and *Aspergillus parasiticus* with 33,500 ppb of AFB1 as a major toxin. Similar findings were stated from South Africa, where 7 out of 25 Hereford calves died of this toxicity following ingestion of contaminated peanut hay and maize [14, 15].

Epistaxis (Fig. 2), depression, apathy and neurological signs were observed in younger fattening calves [16, 17]. Affected animals showed pyrexia, changes in the pulse and respiration rate as described by previous study [18].

Fig. 1: Rectal prolapse in aflatoxicosis effected cattle

Fig. 2: Epistaxis in affected cattle

Rumen motility was generally low in affected animals. Earlier experimental findings also described altered amplitude and frequency of rumen contractions [18, 19]. This change in rumen motility is probably due to alternation in rumen microflora and volatile fatty acids. Although, the significant change in production and the proportion of volatile fatty acids has been published under the influence of aflatoxicosis, their relation with rumen motility has not been established [8, 18]. Therefore, further studies are needed to address this relation. Elevated toxin doses were detected in feed in present scenario, at lower doses of aflatoxin (800 ppb AFB1) for 15-18 weeks did not seem to have acute effects on animals [20]. A postmortem of intoxicated animals generally revealed anasarca and congestion (Fig. 3). There were hemorrhages on visceral organs and diaphragm

(Fig. 4, 5). This study reveals that aflatoxin had a generalized effect on visceral organs [21].

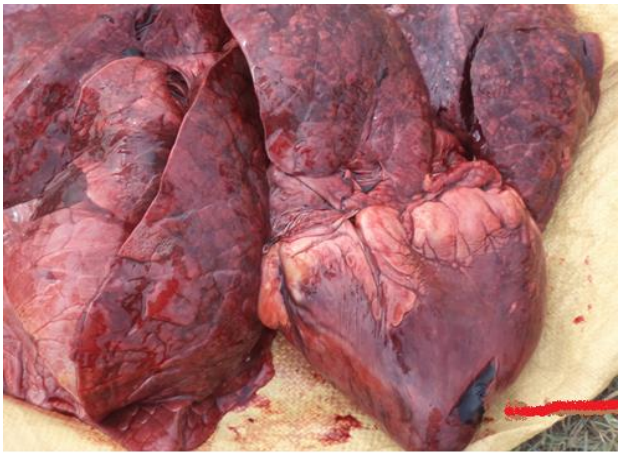

Fig. 3: Congested liver and heart

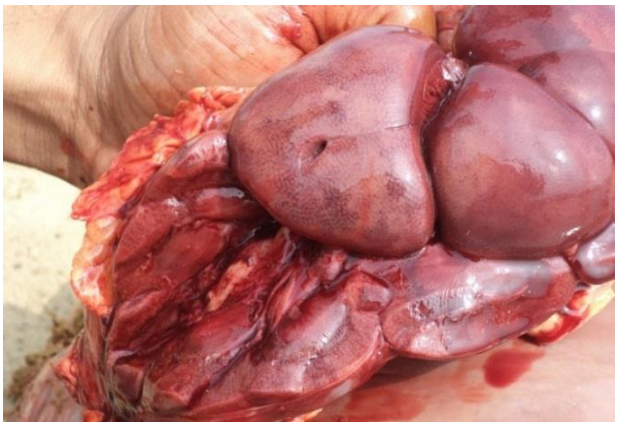

Fig. 4: Hemorrhagic kidney of aflatoxicosis effected cattle

Usually, liver is the target organ of aflatoxicosis and in present case liver was enlarged, congested, light tan colored; more lesions included bile duct hyperplasia and mild periportal fibrosis (Fig. 5, 6). Anasarca and pale tan liver were the main findings in cattle pastured on sweet corn field which had aflatoxin level 2,365 ppb [22]. With upper dose of aflatoxin i.e. 5,136 ppb of AFB1 in corn, similar lesions were observed in Holstein male calves [23].

At even higher aflatoxin level i.e. 77,000 ppb AFB1, abortion and acute death was reported in crossbred cows [24]. Another author gave an account of these lesions in Holstein calves experimentally fed 0.008-0.08 mg AFB1 / day, but no death was witnessed at this dose level [25-27]; in one more study, calves received a single dose of AFB1 1,800 ppb death was declared within 23 days [28]. Mild fibrosis was observed in fattening calves and feedlot cattle when they ingested grains containing 4,600-8,200 ppb AFB1 [16, 17]. At upper doses, 11,790 ppb, of aflatoxin, liver fibrosis and bile duct proliferation becomes clearly evident [14]. At even more dose, 12,000 ppb, acute human hepatitis (aflatoxicosis) has been put out from Kenya, where 12 of 20 patients died [29].

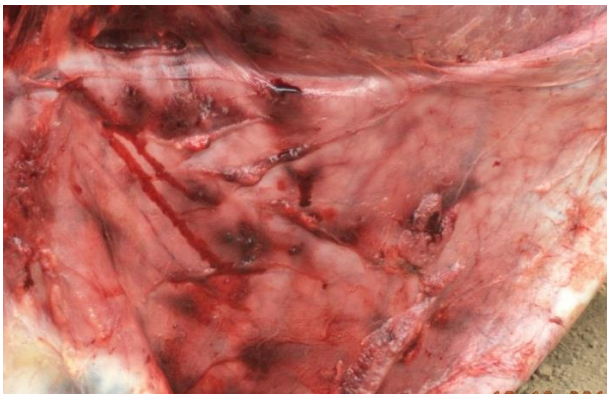

Fig. 5: Hemorrhages on diaphragm

Urinary system of ruminants is also affected by aflotoxin because of its involvement in toxin excretion process. In the present case, urine was apparently brown and hemorrhagic kidneys were congested (Fig. 4). More pronounced effects are seen when toxin dose is greater. Aflatoxicosis in lambs at the dose of 2500 ppb AFB1 was studied for 21 days, which lead to the presence of AFB1 in liver, kidney, urine and feces. Kidneys were congested and urine of affected animals was brownish giving indication of hematuria [17, 30]. A study of cattle with 200 mg AFB1, confirmed pronounced of aflatoxin on kidneys, resulting in proteinuria, glycosuria, ketonuria and haematuria in affected animals [18]; similar findings were professed in experimentally poisoned goats, where necrosis of tubular epithelial cells and proteinaceous exudation in the glomerular space was obvious [31]. Although the immunosuppression was not the subject of present study, certain erstwhile studies have avowed such findings [32-34].

Conclusions

Field outbreaks of aflatoxicosis are common in Pakistan, but in author's knowledge this is first one to be published. Aflatoxin mainly effects liver and kidney but other organs are also influenced. Ruminants have mild tolerance against aflatoxins and usually there are chronic cases that cause production losses acute and per acute toxicity cause animal losses. Younger animals are more susceptible to

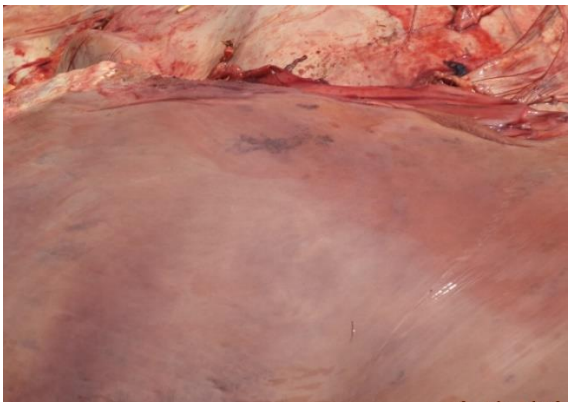

Fig. 6: Pale tan texture of liver

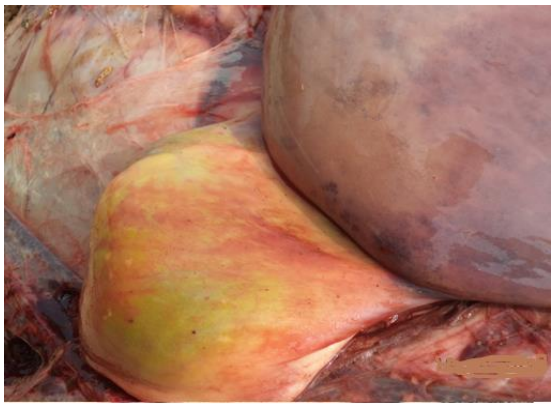

Fig. 7: Bile stasis and hemorrhagic liver

toxicity. All in nutshell, provender contaminated with aflatoxin must be avoided. Outbreak management of aflatoxin include: feed withdrawal and supportive therapy to affected animals.

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