DEATHS IN CATTLE SUGGESTIVE OF SUBACUTE FLUORINE POISONING FOLLOWING INGESTION OF SUPERPHOSPHATE

R. G. CLARK, A. C. HUNTER, D. J. STEWART

INTRODUCTION

Poisoning of cattle and sheep with superphosphate where the fertilizer was stored within reach of the animals has been reported by Romanenko (1954). In New Zealand losses of pregnant and lactating ewes grazing pastures topdressed with phosphatic fertilizers have been recognized for several years, and records kept since 1965 show that 39 outbreaks have been reported to Ruakura Animal Health Laboratory (P. J. O’Hara, pers. comm.). Swan and McIntosh (1952) have described the toxicity to dairy cows of grazing pasture topdressed with North African phosphate and superphosphate. The toxic factor was later identified as fluorine (Animal Research Division of the New Zealand Department of Agriculture Reports 1952-3, p. 28; 1953-4, p. 34; 1954-5, p. 35). Phosphatic fertilizers are known to contain 1 to 4% fluorine (Caro, 1964), and the danger of poisoning stock with fluorine in untreated phosphates used as feed supplements has been described by many authors (Emmerling, 1902; Dammann and Manegold, 1904; Gardiner, 1930; Phillips et al., 1934; Hatfield et al., 1942; Udall, 1947; Fincher, 1952; Harris et al., 1952; Crampton, 1954; Dale and Crampton, 1955; Snook, 1962; Rek, 1967; Agarwala et al., 1971).

This paper reports a case in which deaths occurred in a beef cattle herd after grazing pasture freshly topdressed with superphosphate.

HISTORY

Early in November, 1973, 50 mixed-age Hereford cows in light condition with 2- to 3-month-old calves at foot were brought in from unimproved tussock pasture to a paddock of lush, rapidly growing ryegrass/clover/browntop pasture. The cows, in two groups, arrived 4 and 10 days before the paddock was topdressed with sulphurized superphosphate at 314 kg/ha. A hay supplement was fed till topdressing. The cows were not observed for 2 days following topdressing but on the third day the owner found five cows dead and one recumbent. The following day a further cow died and one cow was observed to have a stiff gait.

The animals were ravenous when introduced to the pasture. It was also noted that they had made a deliberate effort to ingest the superphosphate lying on the tyre tracks of the spreading truck or where an excess amount had accumulated during topdressing, particularly at the turning points.

CLINICAL SIGNS

When seen on the third evening following topdressing, the cattle were reluctant to move, approximately 30% were mildly bloated, and 20% were drooling. The cow in lateral recumbency was dyspnoeic and had a heart rate of about 124. It responded to intravenous calcium borogluconate and 1 kg of magnesium sulphate per os to the extent of maintaining an upright position and eating, although remaining recumbent. However, it was destroyed 5 days later when it failed to improve further. The cow that showed a stiff-legged, somewhat spastic gait on the fourth day, recovered rapidly after intravenous administration of calcium borogluconate.

NECROPSY FINDINGS

One of the cows found dead on the third day following topdressing was necropsied the same day. Its lungs were emphysematous; its rumen was filled with ingesta and distended by gas, and there were patchy haemorrhages throughout the intestine, particularly in the ileum and colon.

Necropsy of the cow that died on the fourth day showed similar gastrointestinal lesions and the ingesta contained copious quantities of superphosphate particles.
LABORATORY FINDINGS

Blood samples were taken from two of the affected cows prior to treatment with calcium borogluconate and the results of calcium, magnesium, and fluorine analyses are shown in Table 1. The fluorine concentrations in the rumen contents, livers and kidneys of the autopsied cows are given in Table 2.

The superphosphate contained 1.45% fluorine.

A smooth strain *E. coli* was isolated from the livers, abomasum and small intestines of both autopsied cows.

Soil and pasture samples from the unimproved tussock pasture were collected the following autumn. Soil phosphorus was moderately low (J. L. Grigg, pers. comm.) and pasture phosphorus (expressed as P) was 0.15% of DM which is considered phosphorus-deficient (K. J. McNaught, unpubl.).

**TABLE 1: SERUM CALCIUM, MAGNESIUM, AND FLUORINE LEVELS IN RECUMBENT AND STIFF-GAITED COWS**

<table>
<thead>
<tr>
<th></th>
<th>Calcium (mg/l)</th>
<th>Magnesium (mg/l)</th>
<th>Fluorine (mg/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stiff-gaited cow</td>
<td>46</td>
<td>16.8</td>
<td></td>
</tr>
<tr>
<td>Recumbent cow</td>
<td>41</td>
<td>9.8</td>
<td>2.8</td>
</tr>
<tr>
<td>Normals</td>
<td>80-120*</td>
<td>18-28*</td>
<td>0.05-0.20†</td>
</tr>
<tr>
<td></td>
<td>(Mean 0.09)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

†Obtained from analysis of 50 random samples of sera from clinically normal cattle.

**TABLE 2: FLUORINE LEVELS IN COWS DYING ON THE THIRD AND FOURTH DAYS AFTER TOPDRESSING WITH SUPERPHOSPHATE**

<table>
<thead>
<tr>
<th></th>
<th>Fluorine (ppm)</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>Dry Weight</td>
</tr>
<tr>
<td>Day 3 death:</td>
<td></td>
</tr>
<tr>
<td>Rumen contents</td>
<td>192</td>
</tr>
<tr>
<td>Kidney</td>
<td>1.3</td>
</tr>
<tr>
<td>Liver</td>
<td>1.4</td>
</tr>
<tr>
<td>Day 4 death:</td>
<td></td>
</tr>
<tr>
<td>Rumen contents</td>
<td>182</td>
</tr>
<tr>
<td>Kidney</td>
<td>1.4</td>
</tr>
<tr>
<td>Liver</td>
<td>2.0</td>
</tr>
</tbody>
</table>

In the cow necropsied on the third day the tissues were too autolysed to allow interpretation, other than confirmation of a severe pulmonary interstitial and alveolar emphysema. In the cow that died on the fourth day there was a mild acute enteritis with haemorrhages in the superficial portions of the mucosa. The liver had a moderately thickened fibrous capsule, moderate portal fibrosis with mild bile duct proliferation, and foci of acute inflammation in the sinusoids. The kidney showed congestion at the cortico-medullary junction, interstitial oedema surrounding the interlobular blood vessels, and flattened cortical tubular epithelium. There was also a mild focal subacute glomerulonephritis and a mild focal subacute interstitial nephritis.

**DISCUSSION**

The serum calcium levels in both the cows from which blood samples were taken were consistent with hypocalcaemia, a condition that without treatment results in death in most cases within 12 to 24 hours (Blood and Henderson, 1968). The serum magnesium level of the recumbent cow was consistent with those found in hypomagnesaemic tetany, although it is possible for serum magnesium levels to fall lower than this without clinical illness (Blood and Henderson, 1968). The favourable response to treatment confirmed the diagnosis of, at least, hypocalcaemia.

The low serum calcium and magnesium levels could have been caused by a metabolic disease such as milk fever or hypomagnesaemic tetany which are characteristically associated with a change of diet to lush pasture and lactation and have clinical signs similar to those observed. However, because large quantities of superphosphate were present in the ingesta, superphosphate poisoning was also considered.

A considerable amount is known about the metabolic disease (see, for example, Blood and Henderson, 1968, or Allcroft and Burns, 1968), but relatively little is known about superphosphate poisoning of cattle. The toxic agent in superphosphate is generally believed to be fluorine. Gründer (1964) has distinguished "acute" and "subacute" forms of severe fluorine...
intoxication as resulting, respectively, from a single ingestion of a large dose or from repeated ingestion of smaller doses over a period of days or weeks. Both acute and subacute fluorine poisoning may result in death, but in the former case the animals die in a matter of hours whereas in the latter case they become emaciated and expire within one to two weeks with signs of general cachexia, or have to be slaughtered because of increasing cardiac insufficiency.

Gruenberg (1951) has described a case of acute poisoning in dairy cows. The initial symptoms were drowsiness, loss of appetite, constipation, and paresis of the rumen, followed by prostration and colicky pains. In some cows the clinical picture approximated that observed in hypocalcaemia, with staggering gait, paresis and decubitus as prominent features. Intravenous calcium gluconate was an effective antidote. Krug (1927) reported another case in which 18 cows died within 12 to 14 hours after ingesting sodium fluorosilicate. Convulsions, exaggerated chewing motions, and frothing at the mouth were observed.

Shupe and Alther (1966) state that acute fluorine intoxication may simulate several different types of poisonings or toxicosis. Symptoms may appear in half an hour after ingestion if the amount is high, and are characteristically: excitement, clonic convulsions, incontinence of urine and faeces, high fluorine content of blood and urine, stiffness, weakness, loss of body weight, drop in milk production, excess salivation, nausea, vomiting. Severe depression in some cases, and cardiac failure. Blood and Henderson (1968) give the symptoms as dyspnoea, muscle tremor, weakness, hyperaesthesia, and constant chewing.

Gründer (1974) describes subacute fluorine poisoning as exhibiting the clinical picture of acute to subacute febrile digestive disturbances with cessation of food uptake, decreased motility of the rumen, bloating, as well as diarrhoea. However, there is unlikely to be any clear-cut distinction between the symptoms of acute and subacute fluorine poisoning, since the severity of symptoms will depend on the amount of fluorine ingested. In their grazing trials, Swan and McIntosh (1952) observed two cows on the North African phosphate-topdressed pasture to almost cease grazing; they gave practically no milk, were apparently weak and somewhat unsteady in gait, and it was deemed advisable to take them off the experiment for fear they would die.

The lowered serum magnesium and calcium levels in acute and subacute fluorine poisoning are considered to be due to the formation of fluorine complexes with calcium, magnesium, and perhaps other ions of physiological importance.

Thus since both the metabolic diseases and acute fluorine poisoning have similar clinical signs and cause similar lowering of serum calcium and magnesium levels, differential diagnosis must rely on pathological changes and fluorine analysis.

There are no significant lesions in milk fever, and lesions seen in hypomagnesaemia are variable, limited to agonal pulmonary emphysema, and extravasations of blood in the subcutaneous tissue, under the serosal surfaces and in the intestinal mucosa (Blood and Henderson, 1968). The most commonly reported lesions in humans and animals that have died from acute fluorine poisoning are gastro-intestinal hyperaemia, and oedema and congestion of the kidney (Hodge and Smith, 1965). Krug (1927) found hyperaemia of the abomasum, small intestine and kidneys in dairy cows that died after ingesting sodium fluorosilicate, and Mariakulandai and Venkataramaiah (1955) found acute inflammation of the gastro-intestinal tract, especially the small intestine, in calves that died after being fed sodium fluoride. Obel and Erne (1971) described acute gastro-enteritis and patchy atrophy of the renal tubules, whilst Shupe (1972) considers that necrosis of the mucosa of the digestive tract is generally observed.

The lesions found in the cattle in this investigation were similar to those reported for acute fluorine poisoning, and suggest that more than just a metabolic disease was involved. However, they cannot be considered as conclusive.

The clinical signs and the response of the treated cows to calcium borogluconate did not support the deaths being caused by the pathogenic E. coli. Although the foci of acute inflammation in the
hepatic sinusoids suggested a bacteræmia, it is likely that this was terminal, arising from the intestine through the portal circulation.

The chronic inflammatory changes in the liver and kidney were suggestive of a previous infection and were not likely to have been associated with the death.

Exposure to fluorine compounds does not increase the fluorine content of soft tissues to any significant extent (Hodge and Smith, 1965; Mariakulandai and Venkataramaiah, 1955), and there is considerable variation in reported values both for the normal levels of fluorine in the liver and kidney of the cow and in values associated with excessive exposure to fluorine (Hodge and Smith, 1965). Therefore, it was not possible to draw any conclusions from the fluorine concentrations found in these organs in the dead cattle (Table 2).

The concentration of 2.8 ppm fluorine found in a serum sample from the recumbent cow was well above the normal range (Table 1), but it is not known what concentrations are likely in acute fluorine poisoning.

The analysis of rumen contents in dead animals has been recommended by Egyed and Brisk (1967) as an aid in the diagnosis of fluorine poisoning. They found fluorine concentrations of 260 to 2600 ppm (wet weight basis) in the rumen contents of cows that died from acute fluorine poisoning, but do not give concentrations likely in subacute poisoning. Nor does Gründer (1974) give rumen fluorine concentrations likely in subacute poisoning, but he states that “the minimal acute toxic dose for readily soluble fluoride in cattle lies between 6 and 20 mg/kg body weight per day following oral uptake for several days.”

It is possible to estimate the daily intake of fluorine necessary to produce the concentrations of 31 and 34 ppm found in this study by using the data of Leeman and Stahel (1972). They measured the concentrations of fluorine in the rumen of cattle following daily doses of 2, 3 or 4 mg/kg body weight of sodium fluoride over a period of one week, and extrapolation suggests that the above concentrations would result from daily intakes of 12 to 26 mg/kg body weight. An intake figure of about 11 mg/kg is arrived at if it is assumed that a 250 kg cow consumes 10 kg of dry matter in a day, and that the concentration of fluorine on the pasture by the third day after topdressing would probably have been about 270 ppm (dry weight basis) for superphosphate containing 1.45% fluorine applied at 314 kg/ha (cf. Stewart et al., 1974). Similar calculations indicate that 18 and 13 mg/kg respectively, would probably have been ingested on the previous two days. All these figures are likely to be higher if the cattle were ravenous and made a deliberate attempt to eat the superphosphate. All the intake figures estimated above fall within the range that Gründer (1974) states is consistent with subacute fluorine poisoning.

The observation of cattle deliberately ingesting superphosphate is unusual. A possible explanation is that it resulted from pica due to the cattle suffering a phosphorus deficiency. The poor condition of the cows, their lactating state, and the low soil and pasture phosphorus status of the area on which they had been grazing, provide support for such a deficiency.

The clinical signs, serum calcium and magnesium levels, and the response to treatment suggested that the deaths were associated with a disease causing hypocalcaemia and hypomagnesaemia. Both acute and subacute fluorine toxicity and metabolic diseases can lower these constituents. It would appear likely, however, because of the pathological changes and the apparently toxic doses of fluorine ingested by the cattle, that the deaths were initiated by the fluorine in the superphosphate. It seems likely, also, that because the conditions favoured milk fever and/or hypomagnesaemic tetany the effect of the superphosphate was more severe than it might otherwise have been.

**SUMMARY**

Clinical and autopsy findings are reported for a case in which cattle deaths occurred following grazing of pasture freshly topdressed with superphosphate. The clinical signs, serum calcium and magnesium levels, and the response of the affected animals to treatment with
calcium borogluconate, suggested that the deaths were associated with a disease causing hypocalcaemia and hypomagnesaemia.

Both metabolic diseases, such as milk fever and hypomagnesaemic tetany, and acute fluorine poisoning can cause hypocalcaemia and hypomagnesaemia. It was concluded, however, that the illness and resulting death were probably initiated by the fluorine in the superphosphate. This was suggested by the pathological changes observed and because analysis of the rumen contents indicated the cattle had ingested sufficient fluorine to cause subacute poisoning. It was suggested that in conditions that favour milk fever and/or hypomagnesaemic tetany the intake of fluorine required to cause deaths may be lower than the normal lethal dose because of a combined effect on serum calcium and magnesium levels.

REFERENCES


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