Clinical and experimental studies on *Narthecium asiaticum* poisoning in grazing heifers

AKITOTO

Summary

I. Natural occurrence

The disease occurred in a pasture 650 to 850 meters above sea level which consisted of 59 hectares of artificially seeded grassland and 20 hectares of wild grassland. In May, 1975, 39 Holstein heifers less than 12 months old were grazed in the pasture. The rainfall in July and August, 1975, was about one-quarter of that in the same months of an ordinary year, with poor growth of pasture grass. As there was no sufficient amount of green grass in the artificially seeded grassland, the herd was transferred to the neighboring wild grassland on August 22. When the herd were again transferred to the artificially seeded grassland on September 12, one of them died about 2 hours later. After that, 28 heifers were affected over a period of 21 days up to October 2. Of them, 25 died within 2 to 13 days after the first clinical signs were noticed.

Clinical findings: The patients showed depression and a marked anorexia. A cessation of rumination, hyperemia in nasal membrane, low skin temperature, weak heart sound, abdominal distension and bloody feces were often observed. At last, they lay down and fell into coma, and died.

Clinico-pathological findings: The increase in total white blood cell count with a mild left shift of neutrophils and serum urea nitrogen level, and the decrease in serum calcium concentration were distinctive feature. Urine was strongly positive for protein and glucose.

Pathological findings: The major gross changes in 16 dead heifers were turbid swelling of the kidney (16 cases), ascites (9), edema of the perirenal adipose tissue (8) and subcutaneous tissue (5). In some cases, the hemorrhage of various visceral organs and tissues was observed. Histologically, the extensive epithelial degeneration and necrosis of the proximal convoluted and straight uriniferous tubules were detected in all of 16 heifers. Epithelial cells sloughed off and the basement membrane was exposed to the tubular lumen. The
fibrosis in interstitial tissue was recognized in many cases. The hematopoietic tissue of sternal bone marrow was hypoplastic in 11 cases. In addition, hemorrhage and edema were usually found in various visceral organs and tissues. Edema was particularly outstanding in the lung and the submucosal tissue of the gastrointestinal tract.

The microbiological investigation of the disease was performed and failed to detect any significant bacteria and virus. It was suggested that the affected animals might have ingested a large quantity of leaves from Narthecium asiaticum Polygonum sachalinense and Vitis coignetiae by plant flora survey in the wild grassland. The poisoning was presumed to be caused by these plants or drinking water in the pasture.

II. Experimental studies

1. Three Holstein calves 4 to 8 months old weighing 120 to 170kg were used. Their feeding program was as follow:

Calf A
V. coignetiae 9 days → P. sachalinense 25 days → N. asiaticum 5 days → P. sachalinense 2 days

Calf B
Hay 1 day → N. asiaticum 5 days → grass and hay 10 days

Calf C
Hay and water from the wild grassland 6 days → N. asiaticum 3 days → grass and hay 15 days

N. asiaticum was impalatable for calves and ingested it on the first day but not on the following day. When they fed it, however, blood urea nitrogen levels began to increase and urine glucose became positive after 1 to 2 days. Calf A and B were sacrificed. The macro- and microscopic changes were essentially similar to those in the natural cases. When calf A fed V. coignetiae and P. sachalinense, his body temperature, heart rate and serum calcium concentration were lowered. As for drinking water, 46 L of water derived from the wild grassland was given to calf C for 6 days without any noticeable results.

2. In September, 1976, second trial was performed. The calves and feeding program were as follow:

Calf No. 1
P. sachalinense 7 days → soiling grass 3 days → N. asiaticum 2 days → P. sachalinense 2 days

Calf No. 2
P. sachalinense 7 days → soiling grass 3 days → N. asiaticum 2 days → P. sachalinense 8 days

Calf No. 3
P. sachalinense 7 days → soiling grass 3 days → P. sachalinense 10 days

Calf No. 4
Soiling grass 10 days → N. asiaticum 2 days → soiling grass 2 days

Calf No. 5
Soiling grass 20 days

Hypothermia, decrease in heart rate and lowered serum calcium concentration were
again showed after fed P. sachalinense. When the animals fed N. asiaticum, depression, anorexia and elevation in blood urea nitrogen developed. Urinalysis revealed proteinuria, glucosuria and positive occult blood. In calf No.1,2 and 3, increased ascites were found. Histopathologically, the three calves fed N. asiaticum showed the same renal changes. In the proximal convoluted and straight uriniferous tubules, the epithelium was so extensively degenerative and necrotic it began to slough off.

Two experiments described above suggested that the main cause of the disease was N. asiaticum.

3. The juice from N. asiaticum also produced a similar pathological changes in kidneys of goats and dogs. Their blood urea nitrogen and serum creatinine concentration elevated. Urinalysis in dogs revealed that urine γ-GTP activities increased after fed N. asiaticum. The juice from N. asiaticum in post-blooming stage produced more severe clinical and pathological changes in goats than those in pre-blooming and blooming stage.

III. Chemical analysis of N. asiaticum

Colchicine, phenol compounds and aconitine were evidenced in N. asiaticum. However, isolation for these substances remained unsuccessfully. The oxalate content was 0.1% in dry weight in N. asiaticum.

The author concluded that the pathophysiological entity of N. asiaticum poisoning is acute tubular necrosis.