Case report: Systemic granulomatous disease with vasculitis in a bull due to hairy vetch (*Vicia villosa*) toxicosis

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Abstract

A 5-year-old Angus bull presented for generalized alopecia, thickened pleated skin and profuse malodorous watery diarrhea. This bull, along with 6 other beef bulls, had been grazing in a pasture containing hairy vetch (*Vicia villosa*) for 5-6 months. The owner had treated the bull 2-3 days prior to presentation with injectable oxytetracycline, injectable trace minerals and pour-on parasiticide with no improvement. Diagnostic samples included blood for CBC and chemistry, skin biopsy and scrapings, fecal sample and liver biopsy. Laboratory results included elevated fibrinogen and creatinine kinase. Urinalysis, fecal culture and fecal floatation revealed no significant findings. PCR for Johne's disease was negative. Biopsy of skin revealed a chronic, moderate, perivascular and perifollicular, eosinophilic to histiocytic-lymphocytic dermatitis with moderate acanthosis and hyperkeratosis indicative of an ongoing hypersensitivity reaction. The bull did not respond to treatment and was euthanized due to poor prognosis. At necropsy, disseminated systemic granulomatous disease with variable numbers of multinucleated giant cells and eosinophils was observed in multiple organs indicative of systemic granulomatous disease, along with vasculitis in the kidney, liver, and adrenal gland, indicative that vasculitis is involved in the pathogenesis of the lesions. Microorganisms were not identified in the histologic sections of skin biopsy and necropsy tissues. Mineral analysis of liver revealed increased zinc levels. Viral diseases were ruled out by immunohistochemistry of sections of kidney (bovine viral diarrhea virus and infectious bovine rhinotracheitis), and by *in situ* hybridization on kidney sections (ovine herpesvirus-2 [malignant catarrhal fever]). CD3, CD20, and CD79a lymphocyte markers immunostaining of sections of kidney revealed predominance of CD3 positive lymphocytes within inflammation and blood vessel walls, indicative of predominance of T-cell lymphocytes supporting a type-IV hypersensitivity. The vasculitis observed prompted formulating a list of differential diagnoses for infectious diseases that have evidence of vasculitis, including bovine virus diarrhea virus and malignant catarrhal fever. Vasculitis, although previously described in cases of hairy vetch poisoning, is not observed in every single case of the disease. The remaining bulls were not removed from the pasture and none developed clinical signs.

Key words: cattle, granulomatous disease, hairy vetch, *Vicia villosa*, vasculitis, toxicosis

Introduction

Ingestion of *Vicia villosa* (hairy vetch) has been recognized in the U.S. since 1955 as a culprit for clinical disease and mortality associated with systemic granulomatous disease in cattle.[1-4] This condition has also been described in horses in the United States associated with ingestion of *Vicia villosa* (hairy vetch)[5] and *V. benghalensis* (purple vetch).[6] Outbreaks and isolated cases of systemic granulomatous disease in cattle attributed to ingestion of *Vicia* spp. have been identified in several countries including Argentina, [7,8] Australia,[9,10] Brazil,[11-13] France[14] and South Africa.[15] The following report describes an isolated case of systemic granulomatous disease due to hairy vetch toxicity in a beef bull.

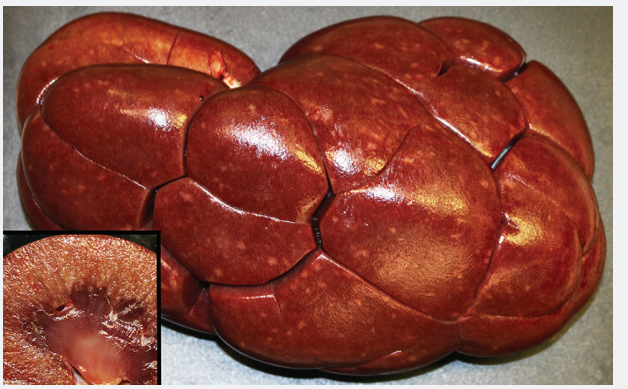
Case description

A 5-year-old registered Angus bull presented in the spring of 2016 for pruritus, alopecia, and weight loss of 2-3 weeks duration. This bull was one out of 7 in a bull herd on winter pasture. The remaining bulls were in normal condition without evidence of clinical disease. Pasture forage consisted of native grasses, Bahia grass and some residual winter seeded forage of rye, and crimson clover. All bulls were examined 5 weeks prior to development of clinical signs and passed a thorough breeding soundness examination (BSE) with a recorded body condition score of 5 (beef scale 1 to 9) on the Society for Theriogenology BSE form. At that time, the bull appeared normal with no record of hair loss or any abnormality noted. Bulls were not treated with any topical parasiticide at the time of BSE.

Physical examination of the bull revealed generalized alopecia with thickened, pleated skin (Figure 1) and profuse, malodorous, watery diarrhea. Rectal temperature was 103°F (39.4°C) (ambient temperature at the time was approx. 85°F [29.4°C]), heart rate was 55 bpm, and respiration was 60 breaths pm. Heart and lung auscultation was normal. There was a small amount of mucopurulent discharge from the right nostril and mucous membranes were pink. Capillary refill test was not performed. The owner had treated the bull 2-3 days prior to presentation with injectable oxytetracycline, injectable trace minerals and pour-on parasiticide with no improvement. Blood and serum samples were collected for complete blood count and blood chemistry respectively. A free catch urine sample was collected (for urinalysis). Skin scrapings were taken for parasite or fungal identification and

Figure 1: Affected bull with skin lesions of presumptive hairy vetch toxicosis.

Figure 2: Kidney with lesions of presumptive hairy vetch toxicosis. Multifocal to coalescing disseminated white foci throughout capsular surface. Insert (bottom left), cross section of kidney showing irregular white foci and streaks throughout the renal cortex.



skin punch biopsies for histopathology. A liver biopsy was taken for mineral analysis and a fecal sample was collected for culture and parasitology. The bull was treated with a topical macrocyclic lactone[a] by the owner after physical examination. Following the physical exam, additional history provided by the owner indicated all bulls had been foraging hairy vetch (*Vicia villosa*) over the past 5-6 months.

Diagnostic findings

Results of blood count, serum chemistry, and urinalysis are provided in Tables 1A-C. The only abnormalities noted were elevated fibrinogen and creatinine kinase. Fecal culture yielded heavy growth of *Escherichia coli*. No parasitic ova were noted on fecal flotation, and PCR for *Mycobacterium avium* ss *paratuberculosis* (Johne's disease) was negative.

Histopathology on the skin biopsies revealed chronic, moderate, perivascular and perifollicular, eosinophilic to histiocytic-lymphocytic dermatitis with moderate acanthosis and hyperkeratosis indicative of an ongoing hypersensitivity reaction. No microorganisms, fungi or ectoparasites were identified in examined histologic sections.

With the skin biopsy findings, evidence of systemic disease, extensive alopecia, and additional history, hairy vetch toxicosis was strongly suspected. Due to continued deterioration of clinical signs and poor prognosis associated with the presumptive diagnosis of hairy vetch toxicosis, the owner elected not to attempt treatment and the animal was euthanized and submitted to the Tifton Veterinary Diagnostic and Investigational Laboratory, University of Georgia, for necropsy.

At post-mortem exam the bull was in poor body condition (BCS 3). There was generalized alopecia and skin thickening throughout the body with multifocal 1 cm diameter crusts affecting mainly the distal limbs and ears. Both kidneys were enlarged with multifocal to coalescing irregular 2 to 5 mm in diameter white foci in the cortex; these foci extended throughout the renal cortex as white streaks (Figure 2). Multiple lymph nodes were enlarged with prominent cortices. Both adrenal glands were enlarged and there was a white rim delineating the cortex and medullary junction. The spleen was enlarged with a bulging parenchyma and numerous white 2 to 3 mm foci on section. There was a dry yellow exudate on the mucosal surface of the upper larynx, and greenish yellow exudate on the mucosal surface of the trachea and distal bronchi. On sections of lung, there was thick green exudate within the lumen of bronchioles and bronchi. There were multifocal, up to 1 cm in diameter ulcerations on the abomasal mucosa. No significant lesions were observed in the oral cavity, esophagus, heart, liver, gallbladder, pre-stomachs, small and large intestines, and urinary bladder.

Mineral analysis was performed in a liver sample collected at necropsy instead of the liver biopsy obtained from the live animal because the liver biopsy sample was too small. Mineral analysis results are provided in Table 2 with the only abnormality being increased zinc levels.

Organs collected for histopathology included brain, trigeminal ganglion, *rete mirabile*, pituitary gland, kidney, liver, spleen, lung, adrenal gland, heart, lymph nodes, thyroid gland, small and large intestines, abomasum, urinary bladder, trachea, esophagus, forestomachs, testicle, skin, and bone marrow. In the kidney, infiltrates of variable numbers of lymphocytes, macrophages, eosinophils, few plasma cells, and occasional multinucleated giant cells were observed in the renal interstitium, indicative of granulomatous disease (Figure 3). Multifocally, inflammatory cells extended within the wall of blood vessels and intima, mainly arterioles and arteries. These blood vessels had reactive hypertrophied endothelial cells, marginated leukocytes, and occasional fibrinonecrotic cell debris deposited within the blood vessel walls, which are indicative of vasculitis (Figure 4). Within or nearby these foci of inflammation, scattered renal tubules had intratubular cell debris with neutrophils and eosinophils and were lined by flattened epithelium. Differential diagnoses for granulomatous lesions with multinucleated giant cells include systemic mycosis or mycobacterial diseases thus special stains in sections of kidney were used to rule those out. Microorganisms were not observed with Ziehl-Neelsen acid-fast stain for mycobacteria and Gomori's methenamine silver stain for fungi.

Infiltrates of lymphocytes, macrophages, eosinophils, fewer plasma cells and variable numbers of multinucleated giant cells were observed in the adrenal gland, liver, myocardium, spleen, intestines and bone marrow. Similar inflammatory

Table 1A: Results of blood chemistry in a bull with hairy vetch toxicosis.

Test	Result	Reading	Unit	Reference range
Total protein	6.7		g/dl	6.4-9.5
Albumin	3.5		g/dl	2.5-4.3
Globulin	3.2		g/dl	2.6-6.5
A/G ratio	1.1			None
Urea nitrogen	72	H	mg/dl	7-20
Creatinine	1.5		mg/dl	1-1.8
BUN/ creatinine ratio	48			None
Total bilirubin	0.30		mg/dl	0-0.4
Glucose	52	L	mg/dl	55-95
Phosphorous	6.1		mg/dl	4-8.6
Calcium	8.6		mg/dl	7.6-10.2
Sodium	131	L	mEg/L	136-147
Potassium	3.5		mEg/L	4-5
Magnesium	2.0		mg/dl	1.6-3.6
Chloride	94	L	mEg/L	95-105
AST	389		U/L	0-350
Biocarbonate	20		mmol/L	20-30
Creatine kinase	411	H	U/L	13-20
Anion gap	21	H	mmol/L	13-20
GGT	25		IU/L	None

Table 1B: Results of CBC with differential cell count in a bull with hairy vetch toxicosis.

Test	Result	Reading	Unit	Reference range
WBC	6.2		× 10^ 3/µl	4-12
RBC	5.39		× 10^ 6/µl	5-10
HGB	10.3		g/dl	8-15
HCT	26.4		%	24-46
MCV	48.9		fl	40-60
MCH	19.1	H	pg	11-17
MCHC	39.2	H	g/dl	30-36
Platelets count	291		× 10^ 3/µl	100-800
MPV	9.3		fl	3.5-6.5
Plasma protein	7.9		g/gl	6-8
Fibrinogen	1000	H	mg/dl	100-600
Segs	1.800		× 10^ 3/µl	0.6-4
Lymphocytes	3.200		× 10^ 3/µl	2.5-7.5
Monocytes	0.4		× 10^ 3/µl	0-0.9
Eosinophils	0.6		× 10^ 3/µl	0-2.4
Basophils	0.2		× 10^ 3/µl	0-0.2

Table 1C: Results of urinalysis in a bull with hairy vetch toxicosis

Test	Result
RBC	Few/hpf
Casts	1 hyaline cast/hpf
Color	yellow
Turbidity	clear
Specific gravity	1.021
pH	6.5
Protein	2 +
Glucose	negative
Ketone	negative
Bilirubin	negative
Blood	trace

infiltrates without multinucleated giant cells were observed in lymph nodes, leptomeninges, choroid plexus, trigeminal ganglion, thyroid gland, abomasum, and urinary bladder. In the spleen, multinucleated giant cells were prominent and in bone marrow some multinucleated giant cells had cytoplasmic bright eosinophilic material or light eosinophilic cleft-like material. The presence of multinucleated giant cells is indicative of granulomatous disease. Microscopic skin lesions were similar to those observed in the biopsy specimen (Figure 5).

Vasculitis was also observed in the liver and adrenal gland. In the kidneys and adrenal glands, lesions in blood vessels were affecting primarily arteries (arteritis) and in the liver primarily veins (phlebitis). In the lungs, bronchi and scattered bronchioles were dilated, lined by hyperplastic epithelium, had large intraluminal accumulations of cell debris, and intraepithelial infiltrates of eosinophils and lymphocytes. There was a peri-bronchial and peri-bronchiolar infiltrate of lymphocytes, plasma cells, eosinophils, and macrophages that extended to and effaced bronchial glands. Gram-positive bacteria were observed in one bronchus with tissue Gram stain. The tracheal epithelium was hyperplastic with loss of cilia although not expected to have clinical relevance in this animal; there were occasional intraepithelial neutrophils and eosinophils, along with a mild submucosal and periglandular

Table 2: Mineral analysis results in a liver sample of a bull with hairy vetch toxicosis. All values and reference ranges are on a dry tissue basis.

Mineral	Concentration in liver	Interpretation	Reference range (µg/g dry)
Arsenic	< 0.09		≤ 9.00
Lead	< 0.09		≤ 3.00
Mercury	< 1.43		No reference range
Thallium	<0.09		No reference range
Cadmium	0.16		No reference range
Selenium	1.43		0.60-3.30
Iron	326.18		170-750
Copper	136.79		40-650
Zinc	631.06	H	90-500
Molybdenum	2.42		1.80-4.70
Manganese	7.66		5.50-15
Cobalt	0.14		0.10-0.40

infiltrate of plasma cells, fewer lymphocytes, neutrophils and occasional eosinophils. A few inflammatory cells and necrotic debris were observed in the lumen of submucosal tracheal glands. Bacterial culture of lungs yielded moderate growth of *Trueperella* (*Arcanobacterium*) *pyogenes.* No *Mycoplasma* spp. was isolated.

The vasculitis observed prompted formulating a list of differential diagnoses for infectious diseases that show evidence of vasculitis including bovine virus diarrhea and malignant catarrhal fever. Immunohistochemistry to better characterize the population of lymphocytes present in the sections was performed to aid in supporting a diagnosis of hypersensitivity and poisoning due to hairy vetch, suspected due to the history of the animal. Immunohistochemistry (IHC) for bovine virus diarrhea virus (BVD), infectious bovine rhinotracheitis (IBR), and lymphocytes markers including CD3 (T-cell marker) and CD20, and CD79a (B-cell markers) were performed in sections of kidney by the Department of Pathology, College of Veterinary Medicine, University of Georgia. There was no evidence of immunostaining for BVD or IBR in areas of vasculitis and inflammation. CD3, CD20, and CD79a immunostaining of sections of kidney revealed predominance of CD3 positive T-cell lymphocytes within inflammation and within walls and intima of blood vessels (Figure 6) supporting a type-IV hypersensitivity reaction. Within the inflammatory infiltrate, rare cells expressed CD20 and only a few cells expressed CD79a suggesting that very few B-cell lymphocytes were in the areas of inflammation.

In situ hybridization (ISH) for malignant catarrhal fever (ovine herpesvirus-2 [OHV-2]) was performed in sections of kidney by the Washington Animal Disease Diagnostic Laboratory (WADDL), Washington State University. OHV-2 genome was not detected.

Discussion

Outbreaks and isolated cases of systemic granulomatous disease in cattle attributed to ingestion of *Vicia* spp. have been identified in several countries.[7-15] Most cases are attributed to ingestion of *V. villosa* (hairy vetch); [7,8,11-13] however, other species have also been identified in several cases including *V. sativa* (common vetch),[14] *V. dasycarpa* (wooly-pod vetch),[9,10] and *V. benghalensis* (purple vetch).[9] In some outbreaks, a mixture of vetches are found in the same pasture[9,11,13] or a hybrid vetch has been identified.[15] Despite some cases finding *V. villosa* and *V. sativa* in the same pasture, the disease has been mostly attributed to *V. villosa*.[11,13] Most outbreaks have been reported in cattle in mixed pastures containing vetch and other grasses or cereals. Disease usually is not seen in a herd until at least 2 weeks after cattle have access to pastures containing the plant.[2] In most cases, 6 or more weeks have passed before clinical signs are observed, and some outbreaks occur after 2 to 5 months of grazing in pastures containing the plant.[9,13-15] Clinical signs have even been observed after the animals have been removed from the offending pasture.[2]

Disease seems to be more prevalent in animals older than 3 years.[2] It seems there is increased prevalence of the disease in certain breeds of cattle, including Holstein and Angus.[3] Outbreaks of the disease occur mainly in the spring, with some outbreaks also occurring during the summer or winter.[1,2,4,5,8,9] Morbidity can be as low as 2.8-2.9%, or as high as 33%.[2,8,9,11,12] Mortality ranges from 50% to up to 100% of the clinically affected animals.[2,8,9,12]

Common clinical signs include dermatitis with variable pruritus and alopecia, conjunctivitis, catarrhal or serous rhinitis, weight loss, fever, reduced milk production, diarrhea, lymphadenopathy, weakness, tachypnea and tachycardia.[2,4,7-9,12,13] Histopathology shows disseminated granulomatous inflammation characterized by infiltrates of macrophages, lymphocytes, and plasma cells with variable numbers of multinucleated giant cells and eosinophils. [1,2,7-9,11,13,15] Lesions can be

Figure 3: Infiltrates of variable numbers of lymphocytes, macrophages, eosinophils, fewer plasma cells and occasional multinucleated giant cells (arrow) are observed in the renal interstitium. Hematoxylin eosin stain of a kidney sample. Renal tubule (*); glomerulus (arrowhead).

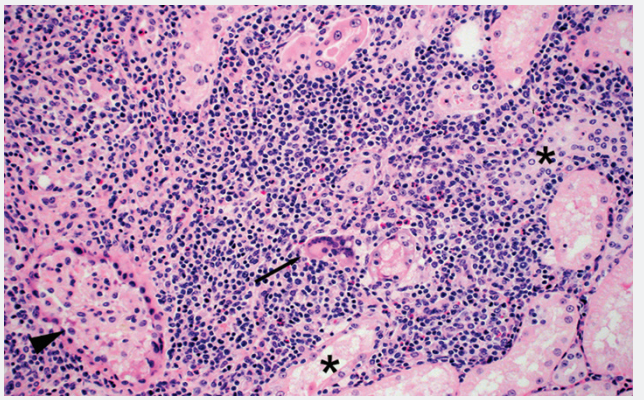
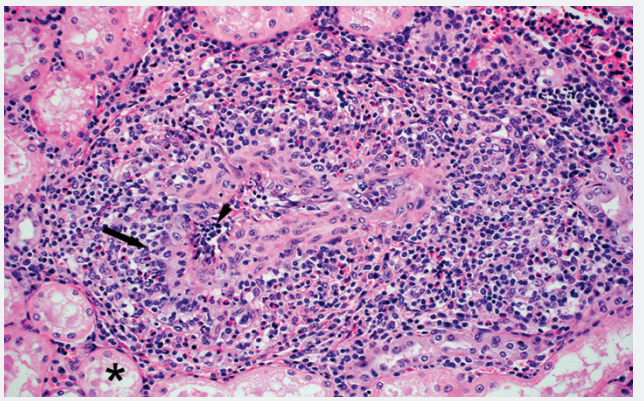


Figure 4: Marked perivascular granulomatous inflammation with infiltrates of lymphocytes, occasional macrophages and eosinophils within the smooth muscle (arrow) and intima (arrowhead). Hematoxylin eosin stain of a kidney sample. Renal tubule (*)



seen in any organ; however, the main organs affected include skin, kidney, adrenal gland, lymphoid tissues, heart, liver and thyroid glands.[1,2,7-9,11,13,15]

A syndrome of pyrexia with dermatitis, pruritus, and sometimes hemorrhage in dairy cows with similar systemic granulomatous inflammation has been described by several authors.[16-22] This syndrome was initially suspected to be due to di-ureido-isobutane in the pelleted concentrate[16] and use of a commercial additive containing sulphuric acid and formaldehyde in silage.[18] Later, a syndrome of pyrexia with dermatitis, pruritus, hemorrhage, and granulomatous inflammation was also associated with consumption of citrus pulp containing citrinin.[17] Other authors have described a similar disease of systemic granulomatous inflammation in cows associated with consumption of citrus pulp.[23,24] Systemic granulomatous disease in dairy cattle has also been induced by dicyandiamide in a feeding trial.[25]

Figure 5: Skin. Chronic, moderate, perivascular and perifollicular, hitiocytic-lymphocytic dermatitis with eosinophils (arrow). Hematoxylin eosin stain of a skin sample. Hair follicle (arrowhead).

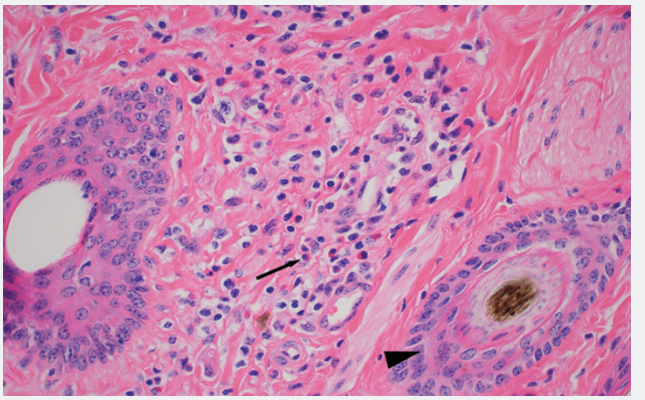
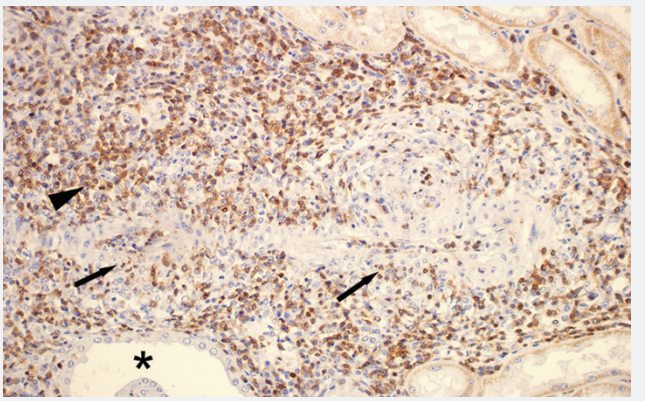


Figure 6: Immunohistochemical staining for CD3. T lymphocytes are observed within the renal interstitium (arrowhead) and infiltrating the vascular walls (arrows). Renal tubule (*).



The presumptive diagnosis of vetch toxicosis or vetch-like disease is a diagnosis of exclusion. It is based on review of herd history, in conjunction with the character and distribution of lesions.[26] Granulomatous inflammation affecting several organs is considered a fairly common pathologic finding of vetch toxicosis; however, diagnosis can only be made if other diseases are ruled out and the affected animals are known to be grazing in pastures containing the plant.[26] The bull in this study was reported to be foraging on pasture containing hairy vetch for 5-6 months, which is consistent with the literature.

The presence of vasculitis was an unusual finding; it was most prominent in the kidney, but also observed in adrenal gland and liver. Vasculitis, although described in cases of hairy vetch toxicosis, is not observed in every case of the disease and, based on the reviewed literature, is not well documented. The vasculitis observed is highlighted in this paper also to emphasize the importance of formulating a list of differential diagnoses for specific lesions observed and the use of molecular tests to rule out these diseases. IHC for BVDV and ISH for OHV-2 were negative in kidney samples ruling out these viral diseases as potential causes of vasculitis and inflammation. Moreover, granulomatous inflammation with

multinucleated giant cells and the presence of eosinophils are not typical lesions seen in internal organs of cattle with infections due to BVDV and malignant catarrhal fever.[27,28] Although an unusual finding, vascular lesions have been previously described in cases of vetch toxicosis both in cows[9,14,15] and horses.[6] Variable amounts of vasculitis in dermal, hepatic portal, and renal lesions were observed in cattle along with submucosal vasculitis in small intestine in a previous report.[9] Hyaline degeneration or fibrinoid necrosis with prominent endothelial cells were described in dermal lesions of a dairy cow.[14] Swollen, hyperplastic endothelial cells and swollen, vacuolated smooth muscle cells, along with variable numbers of luminal mononuclear cells were observed in renal vessels of a cow.[15] Fibrinoid necrosis and invasion of the vascular smooth muscle by the inflammatory infiltrate were observed in arterioles, arteries, and veins (vasculitis) in the lung of a horse affected by the disease.[6] Similarly, phlebitis of portal veins with infiltration of the intima by lymphocytes and plasma cells has also been observed in liver samples of cows with systemic granulomatous disease attributed to citrus pulp ingestion.[24] In the only experimental reproduction of the disease in a cow previously exposed to hairy-vetch, swollen vascular endothelial cells were prominent in many of the areas containing inflammation in a skin biopsy. [3] At necropsy, there was prominent perivascular dermatitis of the superficial and deep dermis, but it was considered less prominent when compared with the biopsy sample, which could suggest that part of the treatment was helpful.

The pathogenesis of the granulomatous disease associated with vetch toxicosis and vetch-like diseases remains unclear.[26] An allergic reaction was suspected in early reports of the syndrome of pyrexia with dermatitis in dairy cows[18,21] with a type III allergic reaction suspected by one of the authors of one of the studies but the reason was not further discussed.[21] Later, the pathogenesis of hairy vetch granulomatous disease was thought to involve a hypersensitivity reaction, with the histologic lesions indicating that a type-IV hypersensitivity reaction was the major component of the host response.[3] Supporting evidence for a hypersensitivity reaction for vetch associated and vetch-like diseases is based on history of exposure and the difficulty in experimentally reproducing the disease because only some animals consuming the plant will develop a hypersensitivity reaction. The low morbidity, the fact that the disease is observed predominantly in animals older than 3 years of age, that certain breeds are described as more susceptible, and that long-term or repeated exposure to the plant is required supports the pathophysiology associated with a hypersensitivity reaction.[3] Hairy vetch toxicosis was experimentally attempted in animals without previous exposure to the plant without success by using concentrated diets for several weeks.[3,10] The disease has only been experimentally reproduced in a cow that had recovered from the natural disease a year earlier.[3] Attempts to reproduce the syndrome of pyrexia with dermatitis that is not linked to ingestion of hairy vetch but has similar lesions and is believed to have similar pathogenesis were successful when feeding a concentrated ration only in dairy cows that had previously recovered from the syndrome of pyrexia with dermatitis.[16] These cows were without symptoms for half a year after the diet was discontinued, but showed clinical signs 1 month after the concentrated diet was reintroduced.

The pathogenesis of vetch and vetch-like associated granulomatous disease is not completely understood. It was suggested that substances within the plant that are ingested or its derivatives are absorbed as haptens or as complete antigens. Antigen-specific lymphocytes are distributed throughout the body and subsequent exposures to the antigen(s) evoke a type-IV hypersensitivity reaction. The presence of multinucleated giant cells was an indication that some constituents of the hypersensitivity process acted as persistent foreign material.[3] An alternative pathogenesis also postulated that vetch lectins directly bind to and activate T lymphocytes, leading to lymphokine production, cytotoxicity and granulomatous reaction.[3]

Further supporting evidence for a type-IV hypersensitivity reaction is the predominance of T lymphocytes within the lesions. CD3 lymphocytes are consistently the main population of lymphocytes observed within the inflammatory infiltrates in cases of systemic granulomatous disease associated with citrus pulp[23] and hairy vetch in cows.[13] Similarly, CD3 positive lymphocytes were observed in the present case within the areas of inflammation and within the inflamed blood vessel walls (vasculitis). It is possible that the vascular lesions observed in this bull were induced by T-lymphocytes and were part of the type-IV hypersensitivity reaction that has been proposed for the pathogenesis of such lesions.[3] As vasculitis could be an early lesion in the disease pathogenesis, the time-frame of no more than 5 weeks from no clinical signs to euthanasia may explain the prominent vasculitis observed in this particular bull. In the study that attained experimental reproduction of the disease, vascular lesions at necropsy were much smaller than those observed in biopsies obtained during clinical disease.[3] In other reports where vascular lesions were observed, some animals were euthanized,[6,14] other animals died naturally,[9] or information was not available if the animals died or were euthanized.[9,24] The pathophysiology leading to natural death has not been documented.

Conclusion

We describe a case of presumptive hairy vetch toxicosis in a bull with disseminated granulomatous disease affecting multiple organs and vasculitis in kidney, liver and adrenal gland. Vasculitis is not a common lesion described with vetch toxicosis, although it has been observed sporadically. A systematic review of natural cases of the disease may help evaluate if this is a common yet overlooked finding, or if it is a rare finding in vetch toxicosis. It also requires more study to determine whether vasculitis is implicated in the pathogenesis of granulomatous lesions observed in cattle, especially in animals that develop hemorrhagic syndrome. This report highlights ancillary testing that can be performed to rule out infectious diseases associated with vasculitis. This case report also should remind practitioners of the importance of histopathology for the diagnosis of skin pathology in cattle and, the importance of thorough epidemiological, clinical observations and laboratorial analyses.

Acknowledgements

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Endnote

[a] moxidectin, Cydectinâ, Boehringer Ingelheim, St. Joseph, MO

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