Vascular Alterations and Degranulation of Eosinophil Leucocytes in Bovine Eosinophilic Myositis

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Abstract. Histopathological examination was made on affected skeletal muscles from 10 cases of spontaneous eosinophilic myositis in Japanese Black cattle. Prominent regressive lesions with increased connective or adipose tissues were observed in affected skeletal muscles of all cases and also in the tongue, diaphragm and myocardium of a few cases. Small arteries or arterioles in muscular lesions showed edematous swelling of the intima, degeneration of the media and cellular infiltration. Infiltration of eosinophils, lymphocytes and plasma cells was remarkable with some neutrophils, while neither basophils nor mast cells appeared. Interestingly, degranulation of infiltrated eosinophils was encountered frequently accompanying eosinophilia in local lymph nodes, suggesting allergic basis in pathogenesis of the disease. Neither parasites, common bacteria, mycoplasmas nor viruses were detected, while a few sarcosporidial cysts without any reactions were seen in muscle fibers of 4 cases.

A myopathy characterized by prominent infiltration of eosinophils in skeletal muscles has been described on cattle, dogs, sheep, horses, pigs and a monkey [15, 38].

Bovine eosinophilic myositis has been known in many countries including Japan since the latter half of the 19th century [20]. No clinical findings were revealed because most cases were detected at meat inspections [19, 35]. Really many cases of eosinophilic myositis were reported to be parasitized with sarcosporidia, but there were no evidences for this protozoa to be

a causal agent [15, 19]. Neither helminths nor microorganisms suspected of causal agents were detected from the lesions in any cases.

We have experienced 10 cases of spontaneous eosinophilic myositis in Japanese Black cattle, and the present paper deals with histopathological findings of these cases, in particular, alterations of blood vessels in muscular lesions and degranulation of eosinophil leucocytes suggesting an allergic basis of the disease.

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Table 1. Cases examined

Case No.	Breed	Sex	Age	Feedlot	Detection
1	J.B.*	Steer	2.5**	Fukushima	Oct. 1971
2	J.B.	Female	?	Tokushima	Apr. 1972
3	J.B.	Female	?	Miyagi	Apr. 1972
4	J.B.	Steer	1.5	Fukushima	July 1972
5	J.B.	Female	3.0	Gumma	Nov. 1972
6	J.B.	Female	5.0	Kagoshima	Sep. 1971
7	J.B.	Female	3.0	Miyazaki	July 1972
8	J.B.	Female	5.0	Tochigi	Feb. 1975
9	J.B.	Steer	?	Fukushima	Mar. 1975
10	J.B.	Female	2.0	Kagoshima	July 1976

Remarks.

* : Japanese Black.

** : Age in years.

Materials and Methods

Affected skeletal muscles and other organs from a total 10 cases of Japanese Black cattle were examined. They were sampled at meat inspecting at abattoirs in Tokyo and Kagoshima districts. Among 10 cases, 7 were females and 3 were steers aged from 1.5 to 5 years (Table 1). No special prevalence of feedlots or seasons were found.

Clinically, only 1 case (No. 6) was emaciated, while others had neither clinical abnormalities nor notable history and feeding.

Microbiological examination: Aerobic cultures were made on blood agar plates and DHL agar plates with affected skeletal muscles and main internal organs of all cases. Attempts at isolating mycoplasmas and viruses were made on 2 cases (Nos. 8 and 10), using PPLO broth (Difco Lab.) containing horse serum (20%) and yeast extract (10%) and cultures of bovine kidney cells or Vero cells, respectively.

Pathological examination: Materials were fixed in 10% phosphate-buffered formalin and 5 to 6 μ m paraffin sections were prepared. They were stained with hematoxylin and eosin (HE), Masson's trichrome, elastica van Gieson, periodic acid Schiff (PAS), phosphotungstic acid-hematoxylin (PTAH), Giemsa, Goodpasture and thionin.

Electron microscopy was made on muscular lesions from only 1 case (No. 8). Fresh materials were fixed in 5% glutaraldehyde for 2 hr, transferred into 14% saccharose and then incubated at 4°C overnight. The samples were washed with phosphate-buffered saline, pH 7.2 cut into a block about 1 mm³ in size, and post-fixed in 1% OsO₄ for 1.5 hr. After dehydration with graded ethanol

and acetone, the tissue blocks were embedded into Epon 812. Ultrathin sections were made by an ultramicrotome (LKB, Sweden), and stained with lead nitrate and uranyl acetate. Observation was made using an electron microscope (HU-12, Hitachi at 75 kv and HVEM, J.E.M. at 1000 kv).

Results

1. Gross observations

Gross lesions were observed in skeletal muscles of the whole body, especially Mm. thoracis, Mm. abdominis, Mm. dorsi and Mm. membri pelvini. The lesions of skeletal muscles of all cases were irregular-shaped, recognized as diffuse grayish-green areas or white patches with increased connective or adipose tissues. The diaphragm and tongue muscles were slightly affected in Case Nos. 4, 5 and 7, and focal necrosis was seen in the myocardium of Case No. 5. Lesions of the diaphragm, tongue and heart showed similar appearances but they were milder than those of skeletal muscles.

Since no cases were found to have gross changes in internal organs at meat inspections, samples from these organs for microscopic examinations were not taken in most cases, except for most organs of Cases Nos. 5 and 7, and lymph nodes (L. axillares and L. poplitei) and costal bone marrows of

Table 2. Histopathology of most severely affected skeletal muscles

	Case No.									
	1	2	3	4	5	6	7	8	9	10
Atrophy	++*	+++	+++	+++	+	+++	+++	+++	+++	++
Degeneration	+	+++	+++	++	+	+	+	++	+	+
Necrosis	+++	++	+++	+++	+	+	++	+++	+	++
Edema	—	+	+	+	++	—	+	+	++	+
Fibrosis	+	+++	++	+++	++	++	+++	+++	++	+++
Steatosis	+++	+++	+++	+++	+++	+	++	++	+++	+
Cellular reaction	+++	+++	+++	+++	++	+++	+++	+++	+++	+++
Neutrophil	+	+	+	+	—	+	+	+	+	+
Eosinophil	++	+++	+++	++	+	+++	+++	+++	+++	+++
Basophil	—	—	—	—	—	—	—	—	—	—
Lymphocyte	+++	++	+	++	+++	+++	+++	++	++	+
Plasma cell	+	++	++	++	+	++	++	++	++	+
Mast cell	—	—	—	—	—	—	—	—	—	—
Others	+++	+++	++	+++	+++	++	++	+++	+++	+++
Degranulation of eosinophil	+	+	+	+	++	++	+	+++	++	+
Vascular lesions	+	+	+	+	+	+	+	+	+	+
Sarcosporidia	+	—	+	—	—	+	+	—	—	—

Remarks.

*: +++ very severe, +++ severe, ++ moderate, + slight, — negative.

Case No. 8.

2. Microbiological examinations

From neither affected skeletal muscle tissues of all cases examined nor internal organs of Case Nos. 5 and 7, any common bacteria were detected on blood agar or DHL agar culture. From affected muscle tissues examined of Case Nos. 8 and 10, no mycoplasmas were detected. Cytopathogenic viruses were not detected on bovine kidney cell as well as Vero cell cultures after inoculation with homogenates of affected muscle tissues from Case No. 8.

3. Histopathological observations

(1) Skeletal muscles

Histopathological changes of most severely affected skeletal muscles and diaphragm were summarized in Table 2. Prominent atrophy and degeneration were seen in muscle fibers (Figs. 1 and 2), and severely atrophied and rarefied fibers showed wav-

ing, hyalinization, vacuolation or degeneration showing homogeneous basophilia (Figs. 4–6). Some fibers were necrotized and calcified frequently with phagocytosis by macrophages or myoblasts and infiltration of a considerable number of eosinophils (Figs. 7 and 8). Increased nuclei of muscle cells showed centralization or swelling, occasionally, having enlarged and strongly eosinophilic nucleoli (Fig. 9). Sometimes myogenic giant cells were seen (Fig. 10).

In the interstitium, showing edema in some areas (Fig. 4), connective or adipose tissues increased prominently forming un-circumscribed granulomatous lesions associated with proliferation of fibroblasts and endothelial cells (Figs. 2 and 3).

In most lesions, diffuse or focal eosinophil infiltration was characteristic (Fig. 1). Most eosinophils had a bilobular nucleus, but some were mononuclear. Neither mitosis nor proliferation of infiltrated eosino-

phils was observed. Interestingly, frequent degranulation of those infiltrated eosinophils was observed in all cases (Fig. 11), and electron microscopy revealed disseminated granules among collagen fibers of the interstitium (Figs. 12-14).

Infiltration of lymphocytes and plasma cells was also seen and more predominant rather than eosinophil infiltration depending upon regions or cases (Fig. 15). A few neutrophils were also seen in necrotic lesions, but basophils and mast cells did not exist in every case. In general, more severe degenerative lesions in the muscle parenchyma accompanied more severe inflammatory response in the interstitium, while mild atrophy or degeneration of muscle fibers was associated only with edema or mild cellular infiltration (Fig. 4).

Within muscular lesions of all cases small arteries or arterioles showed conspicuous changes, such as edematous swelling of the intima with cuboidal metaplasia of endothelial cells. Also hyalinization and swelling were seen in the media with edema of perivascular connective tissues, while no fibrinoid degeneration of the media was demonstrable. In some cases, eosinophils or erythrocytes were infiltrated in all layers of vascular walls (Fig. 16), and vessels contained many leucocytes rich in eosinophils. Only slightly increased connective tissues or a few inflammatory cells were recognized around peripheral nerve fibers and muscle spindles (Fig. 17).

By Giemsa, Goodpasture or thionin stain, neither bacteria nor parasites such as toxoplasmas or trichinella were detected in muscular lesions. In 4 cases (Nos. 1, 3, 6 and 7), a few sarcosporidial cysts were observed in muscle fibers without any tissue damages or inflammatory reactions (Fig. 18).

(2) Others than skeletal muscles

In the myocardium of 2 cases (Nos. 5 and 7) which could be examined, there were mild edema and degeneration in Case No. 7, and a necrotic focus about 1 mm in diameter with calcification and infiltration of plasma cells, lymphocytes, eosinophils and neutrophils, giant cells and fibroblasts in Case No. 5 (Figs. 19 and 20). Dilated lymphatic vessels and diffuse infiltration of lymphocytes were seen in the interstitium near the necrotic focus. Sarcosporidial cysts were seen in muscle fibers without any tissue damages or inflammatory reactions.

In 3 cases (Nos. 4, 5 and 7) in which the tongue was available, degenerative and inflammatory lesions were limited to the musculature without involvement of the mucosa. In Case No. 4, mild edema and infiltration of a few eosinophils and lymphocytes were observed in the muscle interstitium (Fig. 21). Muscle atrophy, edema and infiltration of lymphocytes were seen in Case No. 5 (Fig. 22). In Case No. 7, there was hyalinization or rarefaction of muscle fibers, but no inflammatory cells.

In 2 cases (Nos. 5 and 7) in which the spleen could be examined, follicles were decreased in number of cells but did not change in size, while a number of eosinophils and plasma cells were present in the red pulp.

Lymph nodes (L. ruber in Case No. 5 and L. axillares and L. poplitei in Case No. 8) were sampled only from 2 cases. There existed a number of eosinophils either in medullary sinuses or around small blood vessels of medullary cords. The medullary cords themselves were rich in plasma cells.

In Case No. 8, marrows of costal bones were examined revealing the presence of many eosinophils.

The liver of 2 cases examined (Nos. 5

and 7) had markedly enlarged Kupffer cells, and accumulation of neutrophils, eosinophils or lymphoid cells was seen in sinusoids as well as interlobular connective tissues.

Mild interstitial nephritis with infiltration of lymphocytes, plasma cells and eosinophils was seen in 2 cases examined (Nos. 5 and 7).

In the cortex of cerebrum of Case No. 5, a few eosinophils and lymphoid cells were seen around small veins.

Neither lesions nor parasites were detected in other organs which could be examined.

Discussion

Bovine eosinophilic myositis is known to occur mostly in beef cattle and occasionally in dairy cows without sex or age dependency [19, 31]. The present cases examined were of young steer or female beef cattle, suggesting that the disease might have a trend of higher incidence in beef cattle. The role of some vectors for transmitting the disease has been discussed because of the prevalence in summer time [31], but we could not find out any seasonal prevalence nor correlation between the occurrence and feeding.

Most of the present cases had no apparent clinical signs, as had been described [19, 35]. However, the histopathology revealed that leucocytes in blood vessels in muscular lesions contained a number of eosinophils and that these kinds of cells were also scattered around blood vessels of the cerebrum and bone marrows, indicating that there may be some cases of bovine eosinophilic myositis having peripheral eosinophilia as was reported in canine eosinophilic myositis [4, 13, 27, 28, 33, 42, 43, 45].

Lesions demonstrated in affected skeletal

muscles of the present cases were analogous to most of those described already, and they were characterized by chronic myositis accompanying regressive lesions of muscle fibers, increase of connective or adipose tissues and remarkable infiltration of eosinophils [11, 15, 35]. Besides, we noticed, (1) prominent infiltration of lymphocytes and plasma cells, (2) degranulation of infiltrated eosinophils and (3) changes of small arteries or arterioles in diseased muscles.

The characteristic lesions of bovine eosinophilic myositis may be different from those of muscular dystrophy of other species of animals [30, 36, 37], because of absence of changes in either peripheral nerve fibers or muscle spindles in muscular lesions or other organs except for one bovine case having some lesions in smooth muscle layers of alimentary tracts [19], which were not seen in our cases.

Depending upon regions or cases, infiltration of lymphocytes and plasma cells was more predominant than that of eosinophils which sometimes seemed to be secondary participants in the inflammatory process. These suggest involvement of some immune processes. The strong eosinophil reaction may represent a stage of variegated inflammatory process [10, 14], and there were also a small number of neutrophils, but neither basophils nor mast cells.

Interestingly, in all cases examined, infiltrated eosinophils were shown to undergo degranulation that was confirmed by electron microscopy. Such findings have never been described in literatures, but may be of importance for considering the significance of eosinophil reactions in this disease entity. Eosinophil granules are known to have a large amount of peroxidase [2, 41] and may be released into phagosomes and then outside the cells [6,

48] in the process of phagocytizing mycoplasmas [47], mast cell granules [39] or immune complexes [18, 23]. In the present cases, many necrotized muscle fibers were phagocytized by macrophages or myoblasts with the response of a considerable number of degranulating eosinophils, suggesting some relationships between muscle degeneration and eosinophil degranulation. These findings associated with eosinophilia may suggest allergic basis in pathogenesis of this disease. Eosinophilia was also found in lymph nodes, which might reflect an antibody-forming process [24, 25].

Another most important findings with the present cases were alteration in small arteries or arterioles in muscular lesions, that is, edematous swelling of the intima with cuboidal metaplasia of endothelial cells, degeneration of the media and cellular infiltration. Such vascular changes were limited to diseased muscles and were never found in other organs. Vascular lesions in myopathies of animals and its significance have been described [29, 30, 36, 37, 46], but those in the present cases may be different in hyalinization and cellular infiltration in vascular walls, suggesting also allergic changes.

There have been no significant evidences indicating that this disease is infectious one, while some microorganisms or parasites may play some role as triggers. From the present cases, common bacteria and viruses were not detected, and eosinopenia rather than eosinophilia has been described to occur in bacterial or viral infection [18]. Isolation of *Mycoplasma gallisepticum* from lesions of bovine eosinophilic myositis was described [22], suggesting specific relation between mycoplasma infection and eosinophilic myositis. From our cases, however, no mycoplasmas were detected. Although bactericidal activity of eosinophils was

described to be lower than that of neutrophils or macrophages [10, 18], it was reported that ability of this kind of cells to phagocytize mycoplasmas is comparable to that of neutrophils [47]. In most cases of parasitic infection with eosinophilia, parasites were detected in tissues [1, 3, 5, 7, 18]. There have been no cases of bovine eosinophilic myositis having *Trichinella spiralis* or other parasites [11]. According to Kennedy [19], there is some difference in eosinophil reaction in the skeletal muscles between bovine eosinophilic myositis and experimental infection with *Trichinella spiralis* in cattle.

In contrast, sarcosporidia have been frequently detectable from muscle tissues of different species of animals, though not always associated with eosinophil reaction [8, 9, 21, 26, 32, 34]. The role of sarcosporidia was discussed in a case of chronic eosinophilic myositis of the rhesus monkey [38]. In bovine eosinophilic myositis also, sarcosporidial parasites were detected frequently [11, 19, 21]. In 4 of the 10 present cases, a few sarcosporidial cysts were seen without any reactions. In lesions with this protozoa, however, eosinophil reaction is not always present, and in experimental sarcocystis infection in calves [16], severe necrotic lesions were observed without eosinophil reactions. There have been many cases of eosinophilic myositis having no sarcosporidia [15, 19]. Relationships between sarcosporidia and eosinophilic myositis should be studied further.

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Explanation of Figures

All figures except for Figs. 12 to 14 were taken from preparations stained with hematoxylin and eosin.

- Fig. 1. Atrophy of muscle fibers with severe eosinophil infiltration. Case No. 8.
- Fig. 2. Atrophy of muscle fibers and fibrosis. Case No. 2.
- Fig. 3. Steatosis and fibrosis with cellular infiltration. Case No. 5.
- Fig. 4. Atrophied and waved muscle fibers with interstitial edema. Case No. 4.
- Fig. 5. Degeneration of muscle fibers including vacuolation (A), rarefaction (B) and hyalinization (C). Case No. 2.
- Fig. 6. Basophilic degeneration of a muscle fiber. Case No. 3.
- Fig. 7. Necrosis of a muscle fiber with phagocytosis and infiltration of eosinophils. Case No. 8.
- Fig. 8. Necrosis of a muscle fiber with calcification. Case No. 3.
- Fig. 9. Enlarged nucleolus of a muscle fiber (arrow). Case No. 2.
- Fig. 10. Myogenic multinuclear giant cells. Case No. 3.
- Fig. 11. Degranulation of many infiltrated eosinophils (arrows). Case No. 9.
- Fig. 12. Eosinophils infiltrated in the interstitium of muscle tissues. Case No. 8. Electron microscopy (75 kv).
- Fig. 13. Degranulation of an eosinophil infiltrated

in the interstitium of muscle tissues. Case No. 8. Electron microscopy (75 kv).

- Fig. 14. Degranulation of eosinophils infiltrated in the interstitium of muscle tissues. Many eosinophil granules were dispersed among collagen fibers. Case No. 8. High voltage electron microscopy (1000 kv).
 - Fig. 15. Infiltration of lymphoid cells in the interstitium of muscle tissues. Case No. 5.
 - Fig. 16. Edematous swelling of the intima and degeneration of the media with eosinophil infiltration in a small artery of a muscle lesion. Case No. 4.
 - Fig. 17. Intact peripheral nerve fiber in a muscle lesion with a few inflammatory cells around the tissue. Case No. 4.
 - Fig. 18. Sarcosporidial cyst in a muscle fiber without inflammatory reactions. Case No. 1.
 - Fig. 19. Necrotic focus with calcification and cellular infiltration in the myocardium. Case No. 5.
 - Fig. 20. Infiltration of plasma cells and lymphocytes around the necrotic focus in the myocardium. High power magnification of Fig. 19.
 - Fig. 21. Eosinophil infiltration in the interstitium of tongue muscles. Case No. 4.
 - Fig. 22. Infiltration of lymphoid cells in the interstitium of tongue muscles. Case No. 5.
- Bar=100 μ m (Figs. 1-11 and 15-26) or 2 μ m (Figs. 12-14)