The pathology of aortic-iliac thrombosis (AIT) in two adult Thoroughbred horses from the same breeding farm is reported. No. 1 showed sudden falls with moaning and profuse sweating at mating and during hard exercise repeatedly for more than two years. The symptoms subsided within several tens of minutes. No. 2 showed intermittent lameness of the left hind leg progressively for more than three years. In both horses, thromboembolism was observed in the abdominal aorta, terminal quadrifurcation, and branchial arteries bilaterally. Arterial intima without adherent thrombi had formed intimal plaques frequently. Other noticeable changes were parasitic aneurysm and Hemomelasma ilei. From these findings of the two cases it is suggested that AIT is one of the pathological conditions caused by Strongylus vulgaris infection.

Key words: aortic-iliac thrombosis, intimal plaque, Strongylus vulgaris

Thromboembolism occurring in the abdominal aorta and its branch of the iliac artery is called aortic-iliac thrombosis (AIT), and is considered to develop secondary to thrombotic endocarditis when fragmented thrombi that have formed at cardiac valves occlude terminal arteries [28]. AIT is known to occur often in cows, dogs and cats [22, 27]. Also in humans, there is a similar condition called Buerger’s disease, characterized by endoarteritis obliterans of the small terminal arteries [6]. A similar disease occurs in horses as well. It was first reported by Merillat & Merillat in 1907 [23], followed by Tutt (1913) [32], then Innes & Whittick (1940) [15], Azzie (1969) [2], and most recently by McDonnel et al. (1976) [21], Crawford & Smythe (1982) [7], Maxie & Physick-Sheard (1985) [19], and Brama et al. (1996) [4]. Azzie estimated its prevalence as 1.0–1.5% of all Thoroughbred horses in South Africa at the time of reporting [19]. While bacterial infection, parasitism, mechanical injury of the arteries and stress have all been considered as possible causes of AIT [2, 19], the prevailing theory today is that AIT is caused by Strongylus vulgaris parasitism [9, 17, 26]. Despite frequent reports of AIT in foreign countries, there have been as yet no reports of the condition in Japan. Therefore, we decided to prove the existence of this pathological condition in two cases of equine AIT and discuss the mechanism of thrombi formation.

The two Thoroughbred horses with AIT came from the same breeding farm. Case No. 1 was a 12-year-old stallion with a history of sudden falls, moaning in agony and profuse sweating of the trunk at mating and during hard exercise for more than two years. These symptoms occurred repeatedly but on each occasion they subsided within several tens of minutes. During mild exercise, the horse showed slow movement in the hind limbs and unstable gait. These clinical signs progressively worsened and an abnormal swelling 20 cm in diameter appeared on the left flank immediately after exercise. Several tumorous nodules were also found on the skin of the left inner thigh. Because rectal examination revealed a firm nodule on the upper pelvic cavity, the horse was euthanized based on a suspicion of multiple tumor growth in the abdominal cavity.
cavity. Case No. 2 was a 14-year-old mare. She had not been subjected to hard exercise nor did she have any severe clinical signs as in Case No. 1; however, she had showed intermittent lameness of the left hind leg, which progressively worsened over 3 years. She was euthanized two months after Case No. 1. Both cases shared a long history of intermittent lameness or falling down of the left hind legs.

Macroscopically, old thrombi were found in the abdominal aorta caudal from the kidneys and also in many of its branches in both cases (Fig. 1). In Case No. 1, a brownish-yellow fibrin thrombus, which was about 18 cm long with a maximum diameter of 4.5 cm, embolized the abdominal aorta, 5 cm caudal to the cranial mesenteric artery to the terminal quadrifurcation. The dorsal part of the thrombus strongly adhered to the aortic wall. Furthermore, the thrombi were found to extend into the branches of the iliac circumflex artery, external iliac artery, femoral artery, popliteal artery, and internal iliac artery bilaterally as well as symmetrically (Fig. 2). In Case No. 2, not only were thrombi found from 2 cm caudal from the caudal mesenteric artery to the quadrifurcation, but the iliac circumflex artery, external iliac artery, femoral artery, internal iliac artery and popliteal sacral artery were all embolized bilaterally and symmetrically (Fig. 3). All thrombi in these arterial branches became almost completely organized, making the remaining lumen extremely narrow. A few recanalized vessels were observed in the organized thrombi. On the other hand, arterial intima without adherent thrombi had formed transverse folds in all areas and had a rough appearance (Fig. 3, arrows). In Case No. 2, organized thrombi were formed in the peripheral femoral artery of the right hind leg from the popliteal sacral artery, the cranial tibial artery, and further to the caudal tibial artery. Due to the thromboembolism, multiple foci of muscle degeneration and fibrosis were also found around the right tibia such as the digital flexor muscle, posterior tibial muscle, and lateral digital extensor muscle.

Other common changes were seen in the spleen: multiple hematomas and echymotic hyperplastic trabeculae were formed on almost all areas of the splenic parenchyma. The swelling on the left flank observed immediately after exercise was confirmed to
have been caused by splenic swelling due to acute hyperemia. Other findings included small parasitic aneurysms in the cranial mesenteric artery (Fig. 2, arrow), *Haemomelasma ilei* in the serosa of the small intestine (20 found in Case No. 1, 10 in Case No. 2), parasitism of a number of *Anoplocephala perfoliata* in the cecum (500 found in Case No. 1, 100 in Case No. 2) and *Gastrophilus intestinalis* in the stomach (Case No. 2), concentric cardiac hypertrophy (Case No. 1), and adhesive and partially fibrous testicular vaginalitis (Case No. 1). Nodules observed on the left inner thigh in Case No. 1 were benign neurofibroma of the subcutaneous tissue. The major autopsy findings of both cases are summarized in Table 1.

The large thrombus formed in the abdominal aorta and iliac quadrifurcation was fibrinous (Fig. 4) and a small number of leucocytes, erythrocytes and cellular debris were contained in the fibrin net. However, no parasites were found. At the dorsal portion of the abdominal aorta, the thrombus become extensively organized and adhered strongly to the artery wall. Foci of calcification or bone metaplasia with osteoclast-like giant cells were found in the areas of from the organized intima to the upper media. These were also accompanied by severe hemorrhage, large amount of hemosiderin deposition, and infiltration of round cells such as hemosiderin-laden macrophages, lymphocytes, and plasma cells. In the deep media, increased smooth muscle cells caused frequent rupture and loss of elastic fiber; however, elastosis was also observed in some areas. On the other hand, the abdominal aorta, where folds were formed without adhesion of thrombi, underwent collagenous thickening in the media and

<table>
<thead>
<tr>
<th>Change</th>
<th>Case No.</th>
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<tbody>
<tr>
<td>1. aortic-iliac thrombosis &amp; thromboembolism in other branchial arteries</td>
<td>1 &amp; 2</td>
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<tr>
<td>2. aneurysm of cranial mesenteric artery</td>
<td>1 &amp; 2</td>
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<tr>
<td>3. <em>Haemomelasma ilei</em></td>
<td>1 &amp; 2</td>
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<td>4. splenic hematoma</td>
<td>1 &amp; 2</td>
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<td>5. parenchymal fibrosis (spleen, heart, liver)</td>
<td>1 &amp; 2</td>
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<td>6. other parasitism in the alimentary tract</td>
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<tr>
<td>7. hypertrophy of heart</td>
<td>1</td>
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Fig. 2. Case No. 1. Large thrombi in abdominal aorta and its quadrifurcation are exposed by cutting along their ventral surface (asterisks). An arrow indicates parasitic aneurysm in the cranial mesenteric artery.

Fig. 3. Case No. 2. Dark-red and friable thrombi (asterisks) are embolized in the aorta, its quadrifurcation, and arterial branches. Two arrows show intimal plaque in the femoral artery where the embolized thrombus was removed.

Table 1. Main pathologic changes
hyperplasia of smooth muscle cells with loss/localized increase of elastic fibers (Fig. 5). Peripheral arteries, such as the external iliac, internal iliac, iliac circumflex and femoral arteries, showed similar changes to the abdominal aorta (Fig. 6). None of these arterial branches revealed the existence of parasites or the infiltration of inflammatory cells. Compared to Case No. 1, in Case No. 2 peripheral thrombi became almost completely organized. Furthermore, the skeletal muscles around the tibia, which are supplied by these arteries, showed prominent necrosis, regeneration, and fibrosis due to old infarction.

The aneurysm at the cranial mesenteric artery had embedded *Strongylus vulgaris*, and chronic thrombotic arteritis had developed with severe infiltration by chronic inflammatory cells including eosinophils and fibrosis (Fig. 7). In many arteries of general organs, edematous or fibrous thickening of the intima as well as the formation of intimal bodies was frequently recognized, but no inflammatory reaction accompanied them. As a result of the congestive circulatory disturbance caused by AIT, the spleen developed remarkable collagenous hyperplasia from the trabeculae to red pulp with many foci of old thrombi, necrosis with calcification, or hemosiderin deposition. Fibrotic foci were also scattered in the parenchyma of the heart, liver and kidneys. Severe parasitic catarrhal enteritis accompanied by infiltration

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**Fig. 4.** Case No. 2. Cross section of thrombosed abdominal aorta. Hematoxylin and eosin stain.

**Fig. 5.** Case No. 2. Intimal thickening with irregular fibrosis (intimal plaque) of non-thrombotic aorta. Elastica van Gieson stain.

**Fig. 6.** Case No. 2. Cross-section of the left femoral artery. Almost all of the thrombus is organized. Hematoxylin and eosin stain.

**Fig. 7.** Case No. 1. Parasitic aneurysm of the cranial mesenteric artery. Two parasites (arrows) are embedded in the fibrin thrombus (asterisk). Hematoxylin and eosin stain.
of eosinophils and round cells and formation of intimal bodies in the small arteries were observed in the small intestine.

Among the many frequent reports of equine AIT before the 1990s, Azzie and Maxie & Physick-Sheard observed 38 and 17 cases, respectively, and described the clinical signs, epidemiology, histopathology and treatment [2, 19]. Maxie & Physick-Sheard concluded that AIT occurs more often in older horses, in males rather than in females, and in heavy horses rather than light breeds [19]. Azzie ruled out the involvement of parasitism based on the fact that there was no indication of parasitic lesions at the sites where thrombi adhered [2]. However, the high incidence of intimal edematous/fibrous hyperplasia, hyperplasia of the medial smooth muscle cells and intimal bodies in the abdominal aorta and iliac quadrifurcation [2, 5, 14, 19, 20, 24, 29, 30]. Angiography and ultrasonography have also been shown to be effective at diagnosing AIT in recent years [14, 25, 30]. In Case No. 1, a firm mass palpated on the upper wall of the pelvic cavity at rectal examination was considered a tumor in the abdominal cavity; however, this was actually an old thrombus in the abdominal aorta. About Case No. 2, if the mare had been subjected hard exercise, she would also have presented not only intermittent lameness but also dramatic clinical signs such as Case No. 1. A better knowledge of AIT could have resulted in a correct diagnosis. When correctly diagnosed as AIT and treated, some horses showed improvement in the clinical signs or successfully recovered by developing collateral circulation to maintain the volume of peripheral blood in conjunction with thrombolysis and reduced workload [4, 5, 24, 29, 30].

The condition of thromboembolism in both cases, corresponded closely to what has been described previously. Although identifying the primary site was not easy with thromboembolism already having spread to a large area, the evidence suggests that the lesions in the abdominal aorta were the primary site of thrombosis, because the thickness of the organized layer where the thrombi adhered was larger in the abdominal aorta than in the terminal arteries, and because calcification/ossification developed in the aortic intima.

Intimal plaques, i.e., folds caused by fibrous hyperplasia of the intimal layer seen in the terminal quadrifurcation or terminal arteries, are pathological changes which trigger blood coagulation [17, 19]. However, the high incidence of intimal edematous/fibrous hyperplasia, hyperplasia of the medial smooth muscle cells and intimal bodies in the abdominal arteries has also been shown in Strongylus infection; therefore, intimal plaques are also likely to be parasite-related lesions caused by Strongylus vulgaris infection.
[11, 13]. Roony & Robertson concluded in their report that histopathological findings implied no causes other than parasites [26]. Larvae of Strongylus vulgaris are known to migrate to the abdominal aorta. This is an example of aberrant parasitism [8, 16, 18], and the authors have also witnessed this form of parasitism in the aortic arch and the abdominal aorta in a young horse. AIT is one of the pathological conditions caused by Strongylus infection, and intimal plaques of the terminal arteries should be considered changes that develop secondary to AIT over a long period of time.

References


