Endovascular aneurysmal models at the external iliac artery of dogs

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Introduction: **Establishing an aneurysm model using simple and easy operative techniques is desirable to develop new endovascular treatment devices such as stent grafts. We developed an aneurysm model using the external iliac arteries (EIAs) of adult Beagles, a relatively large animal that we thought would be easy to handle, using simple and less complicated endovascular procedures. In addition, we evaluated the generated aneurysm model histologically and determined the factors that were necessary for creating more dilated aneurysms.**

Methods: **Experimental animals consisted of 16 beagles (average weight, 14.0 kg). The animals were divided into four groups (S, E, B+S, and B+E). Eight Beagles were in the S and E groups, without balloon dilation. S group Beagles were injected with normal saline into the right EIA and served as a control group. Elastase was injected into the left EIA of the same Beagles (E group). Eight Beagles were in the B+S and B+E groups with balloon dilation. After balloon dilation, normal saline was injected into right EIA of the B+S group. Elastase was injected into the left EIA of the same Beagles (B+E). After 4 weeks, we measured the EIA diameter using abdominal ultrasound imaging from a body surface. Both sides of the EIA were harvested. We evaluated the dilation rate of the EIA diameter, and histologically, evaluated the disappearance of the internal elastic lamina, degeneration and disappearance of medial smooth muscle and the external elastic lamina, and neointimal thickening.**

Results: **Inner diameters were dilated more in the B+E group vs the other groups. The B+E group internal elastic lamina had almost disappeared, with significantly more severe degeneration and disappearance of external elastic lamina.**

Conclusions: **We developed a muscular artery aneurysm model using the EIA arteries of adult Beagles and a simple endovascular procedure. Histologically, internal and external elastic lamina degeneration was an important factor to create significantly dilated aneurysms in this muscular artery model. (J Vasc Surg 2012;55:1742-8.)**

Clinical Relevance: **We tried to develop an aneurysm model using the external iliac arteries of adult Beagle dogs, a relatively large animal that we thought would be easy to handle, and a simple and less complicated endovascular procedure. Establishing an aneurysm model using simple and easy operative techniques is desirable for the development of new endovascular devices.**

Many kinds of stent grafts have been developed for the endovascular treatment of aortic aneurysms. Many new devices have been examined using an aneurysm model in experimental studies in which a stent graft is placed at the site of the aneurysm and then biologic responses are examined over time.[1-3] To date, reports have described experimental aneurysm models in mice,[4] rats,[5] rabbits,[6] dogs,[7] and pigs.[8] The most popular experimental aneurysm types are sidewall aneurysms using a vein patch, and bifurcation aneurysms using blood vessel branch ligation, possibly with intraluminal injection of elastase, a protease that denatures the elastin of elastic fibers.[9]

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Small-animal models, such as some varieties of mice, are not appropriate for endovascular treatment studies because they are too small to deliver endovascular devices into their vessels. Many peripheral arteries of dogs have adequate-diameter vessels for introducing human endovascular devices.[10] Strindberg et al[3] developed an aneurysm model at the abdominal aorta of dogs in which the aorta was surgically exposed and the short segment was isolated by temporary occlusion. Within that range, every lumbar artery was ligated except for one, from which elastase was injected through a catheter to create an aneurysm. However, generating these models requires surgical stresses, such as an abdominal operation or blood vessel sutures and other complicated procedures.[11]

Establishing an aneurysm model using simple and easy operative techniques is desirable to develop new endovascular treatment devices such as stent grafts. We developed an aneurysm model using the external iliac arteries (EIAs) of adult Beagles, a relatively large animal that we thought would be easy to handle, using simple and less-complicated endovascular procedures. In addition, we evaluated the generated aneurysm model histologically and determined the factors that were necessary for creating more dilated aneurysms.

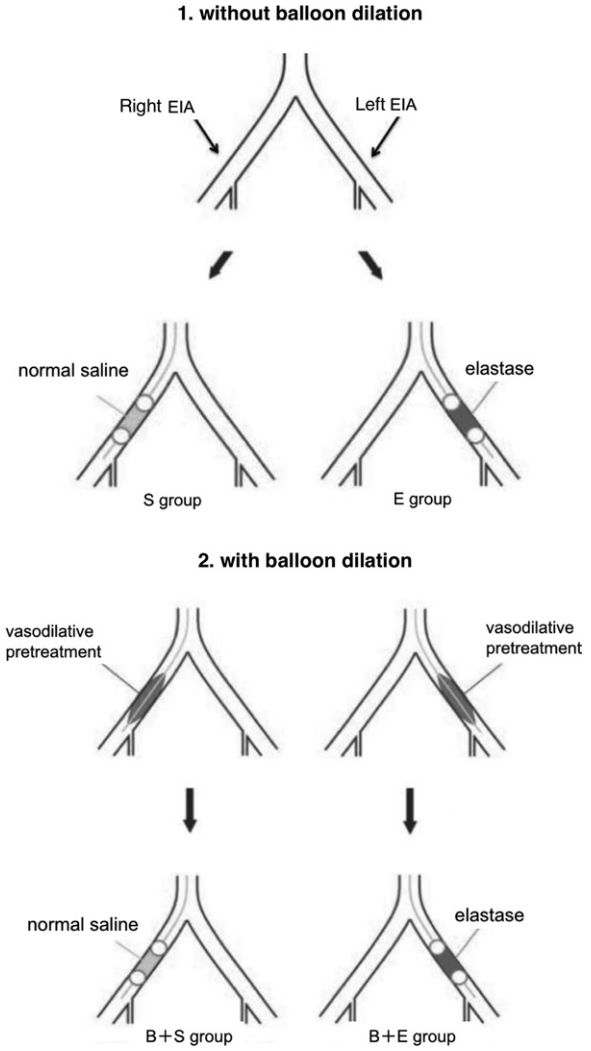

Fig 1. Diagrams illustrate the division of the dogs into S, E, B+S, and B+E groups. In all groups, a double-balloon catheter was used to create a closed cavity by the external iliac artery (*EIA*). *1,* In groups without balloon dilation (n = 8), *S* group: The closed cavity at the right EIA was filled with saline. *E* group: The closed cavity at the left EIA was filled with elastase. *2,* With balloon dilation (n = 8). *B+S* group: The right EIA was expanded with a balloon catheter and the closed cavity filled with saline. *B+E* group: The left EIA was expanded with a balloon catheter and the closed cavity was filled with elastase.

METHODS

This study was approved by the Tohoku University Animal Experiment Committee. Experimental animals consisted of 16 adult Beagles that weighed an average of 14.0 kg (range, 13.5-15.3 kg). The dogs were anesthetized with an intramuscular injection of ketamine (20 mg/kg body weight) and atropine sulfate (0.1 mg). Sodium pentobarbital (25 mg/kg; Nembutal, Abbott Laboratories, Chicago, Ill) was used as a supplemental anesthetic.

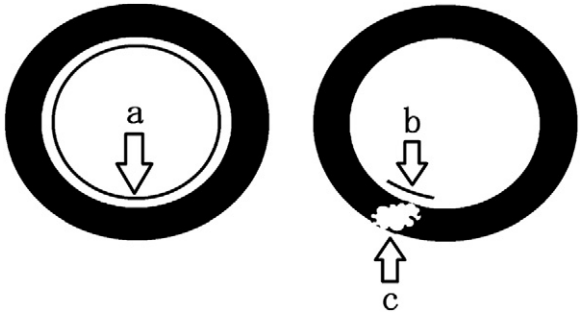

Fig 2. Diagrams show (*a*) vessel inner circumference, (*b*) positive finding length, (*c*) and disappearance or degeneration of the normal structure. Above vessel inner circumference and positive finding length of a tissue specimen, Elastica-Masson staining, were measured using ImageJ. Positive findings were calculated using the following formula: b/a.

We measured the inner diameter of the bilateral external iliac artery (EIA), 1.5 cm distal to the first branch, using abdominal ultrasound imaging. We placed the dog face-up on an operating table. Anesthesia was provided. After surgically exposing the right internal carotid artery, an 8F introducer sheath (Medikit, Tokyo, Japan) was inserted into the descending aorta, followed by an intravenous loading dose of heparin (50 U/kg body weight; with a maintenance dose, 300 U/h).

The animals were divided into two groups, with and without balloon dilation, both of which were subdivided into the following four groups according to whether elastase was used (Fig 1):

1. Without balloon dilation (n = 8). S group: normal saline injection into the right EIA. E group: elastase injection into the left EIA.
2. With balloon dilation (n = 8). B+S group: balloon dilation plus normal saline injection into the right EIA. B+E group: balloon dilation plus elastase injection into the left EIA.

The procedures used in each group were as follows:

1. Without balloon dilation (n = 8): We created a closed cavity with a double-balloon catheter (Selecon MP Catheter, Clinical Supply, Gifu, Japan) at the left EIA. A total of 2.5 mL elastase (106 U/mL; Worthington Biochemical Corporation, Lakewood, NJ) was injected into the closed cavity, which was left unattended for 30 minutes (E group). As a control, we created a similar closed cavity with a double-balloon catheter at the right EIA, and 2.5 mL normal saline was injected into the cavity, which was also left unattended for 30 minutes (S group).
2. With balloon dilation (n = 8): We expanded the left EIA with a 2-cm-long UltraThin Diamond balloon catheter (Boston Scientific/Medi-Tech, Natick, Mass) for vasodilatation. That balloon diameter was about 1.5 times that of the left EIA inner diameter measured on abdominal ultrasound imaging. The balloon was expanded 10 times at 4

		disappearance of the internal elastic lamina(%)	degeneration and disappearance of medial smooth muscle(%)	degeneration and disappearance of the external elastic lamina(%)	neointimal thickening(%)	**The inner vasodilation ratio**
S	no.1	0	0	0	0	0.98
	no.2	0	0	0	9.2	0.96
	no.3	0	0	0	0	1
	no.4	0	0	0	0	1
	no.5	0	0	0	0	1
	no.6	0	0	0	0	1
	no.7	0	0	0	0	1
	no.8	0	0	0	0	1
E	no.1	77.2	0	18.2	64.4	1.02
	no.2	35.9	0	17.1	29.0	1
	no.3	56.6	0	2.6	47.5	1
	no.4	26.5	0	0	0	1
	no.5	13.7	0	0	10.0	1
	no.6	43.8	0	43.6	0	1
	no.7	51.8	0	0	75.8	1
	no.8	46.6	5.6	0	56.6	1
B+S	no.1	18	0	0	12.1	0.95
	no.2	38.3	23.9	0	100.0	1
	no.3	0	0	0	0.0	0.94
	no.4	0	0	0	6.5	0.98
	no.5	0	0	0	0.0	0.93
	no.6	54.5	40.5	0	100.0	1
	no.7	12	13.4	0	9.2	0.93
	no.8	0	0	0	0.0	0.98
B+E	no.1	66.4	0	59.9	51.4	1.11
	no.2	93.7	2.7	41.0	92.4	1.2
	no.3	11.9	9.3	92.7	36.9	1.18
	no.4	30.1	0	11.8	33.4	1.19
	no.5	5.7	0	3.7	100.0	1.09
	no.6	100.0	61.5	69.1	100.0	1.24
	no.7	100.0	3.4	50.1	100.0	1.15
	no.8	100.0	24.2	30.2	100.0	0.93

Fig 3. Histologic findings percentage and the inner vasodilation ratio are shown for each dog of each group.

atm, each for 10 seconds, followed by another 10 times of expansion at 6 atm, each for 10 seconds. Then, we created a closed cavity with the double-balloon catheter at the site of the expansion, into which 2.5 mL of elastase was injected, and the animals were left unattended for 30 minutes (B+E group). Likewise, we expanded the right EIA and created a closed cavity, into which 2.5 mL of normal saline was injected, and the animals were left unattended for 30 minutes (B+S group).

In both groups, we performed abdominal aortography with a 5F Cobra-type catheter, withdrew the sheath, and ligated the right internal carotid artery. After adequate hemostasis, the wound was closed. Abdominal aortography was performed 4 weeks later, after which each dog was euthanized with Nembutal and then exsanguinated. Both sides of the EIA were harvested. The EIA was immediately fixed in 10% formalin and dehydrated in alcohol 1 week later.

The ratio of the postoperative/preoperative inner diameters was calculated to evaluate the degree of expansion. The inner diameter of both sides of the EIA was measured using abdominal ultrasound imaging from the surface of the body under anesthesia preoperatively and postoperatively, and the ratio of the postoperative/preoperative diameters was calculated. The outer diameter was not obtainable in vivo. Finally, hematoxylin-eosin staining, Elastica-Masson staining, and immunostaining with CD3 and CD20 were performed for the histologic examination, and the following four items were evaluated:

1. Disappearance of the internal elastic lamina;
2. Degeneration and disappearance of medial smooth muscle;
3. Degeneration and disappearance of the external elastic lamina; and
4. Neointimal thickening.

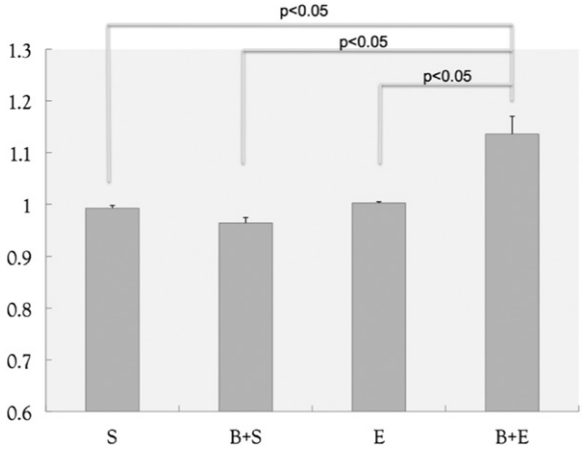

Fig 4. The graph shows the inner vasodilation ratio of the external iliac artery (EIA), defined as the ratio of postoperative to preoperative inner diameters in each group. In the B+E group, the EIA was significantly dilated compared with that in other groups ($P < .05$). *S* group, A closed cavity made by a double-balloon catheter at the EIA is filled with saline. *B+S* group, The EIA is expanded with a balloon catheter and a closed cavity made by a double-balloon catheter is filled with saline. *E* group, A closed cavity made by a double-balloon catheter at the EIA is filled with elastase. *B+E* group, The EIA is expanded with a balloon catheter and a closed cavity made by a double-balloon catheter is filled with elastase.

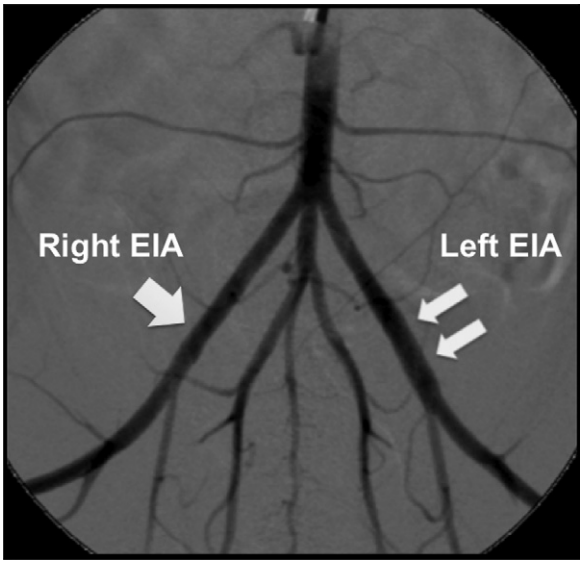

Fig 5. Digital subtraction angiography at 4 weeks after balloon dilation and elastase or saline injection shows enlargement of the external iliac artery (*EIA*) that received elastase (*left*) but no significant dilation in the artery that received saline (*right*).

The percentage of positive findings along the vessel circumference was measured using ImageJ software (National Institutes of Health, Bethesda, Md). Positive findings were judged to be present in the layers of the medial smooth muscle and the external elastic lamina when observed in >50% of the thickness. We measured the area of the findings for neointimal thickening. The area with positive findings was measured for neointimal thickening (Fig 2). We evaluated the presence of CD20-positive or CD3-positive lymphocytes by immunostaining, using CD3 as a marker of B cells and CD20 as a marker of T cells.

Statistical analysis was performed using SPSS 11.0 software (SPSS Inc, Chicago, Ill). We performed the Scheffe *F* test for multiple comparisons after conducting a Kruskal-Wallis analysis on the rate of inner diameter dilation and the percentage of the histologic characteristics at a significance level of $P < .05$. Mean data are shown with the standard deviation.

RESULTS

During the follow-up period, no rupture of the EIA, death, or other major complications occurred. The histologic percentages and the inner vasodilation ratio are shown for each dog in every group (Fig 3).

Vasodilation ratio of the external iliac arteries. The inner vasodilation ratios, or the ratio of postoperative/preoperative inner diameters for the S, B+S, E, and B+E groups, were 0.99 ± 0.01, 1.00 ± 0.01, 0.96 ± 0.03, and 1.13 ± 0.09, respectively (Fig 4). Expansion of the artery diameter was confirmed by angiography and macroscopic examination (Figs 5 and 6).

Histologic examination. Disappearance of the internal elastic lamina was significantly greater in the E group than that in the S group ($P < .05$) and in the B+E group than in the B+S or S group ($P < .05$; Fig 7, *A*). In addition, degeneration and disappearance of medial smooth muscle were observed in the B+S and B+E groups but was not significantly different from those in other groups (Fig 7, *B*). Degeneration and disappearance of external elastic lamina was observed only in groups that received elastase (E and B+E group). Compared with other groups, the B+E group demonstrated significantly more severe degeneration and disappearance of the external elastic lamina ($P < .05$; Fig 7, *C*). Neointimal thickening was observed in the E, B+S, and B+E groups, but no significant difference was noted among them (Fig 7, *D*). Finally, immunohistochemical staining for CD3 and CD20 showed a lack of significant CD20-positive or CD3-positive lymphocytic infiltration.

Histologic findings in each group. The S group showed no histologic changes without vessel dilatation (Fig 8, *A*). The B+S group showed no change in the external elastic lamina (Fig 8, *B*). Mild changes were noted in the disappearance of the internal elastic lamina, degeneration, and disappearance of medial smooth muscle and neointimal thickening. The E group showed moderate disappearance of the internal elastic lamina (Fig 8, *C*). A tendency for degeneration and disappearance of the external elastic lamina and neointimal thickening was also seen, but no change was observed for the medial smooth muscle. In the B+E group (Fig 8, *D*), the internal elastic lamina almost disappeared, with considerable degeneration and disappearance

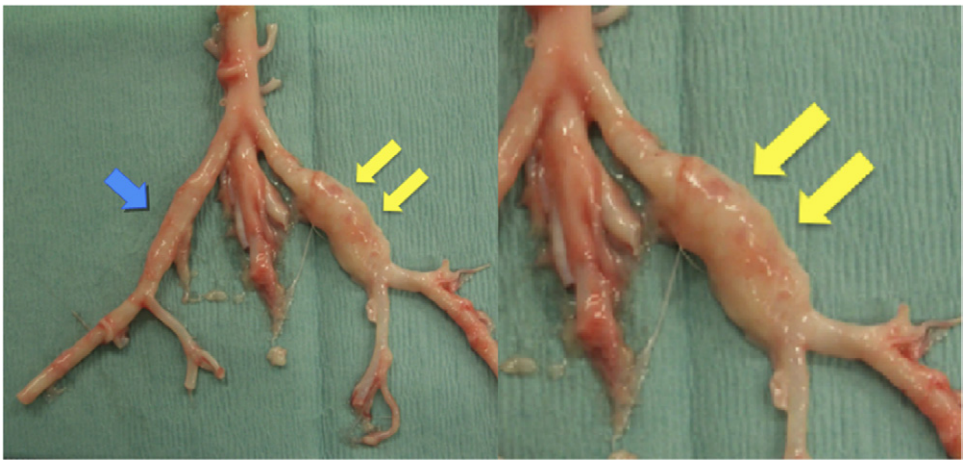

Fig 6. Macroscopic evaluation of the external iliac artery (EIA) in the balloon-dilation groups. The EIA in dogs that received elastase injection group (*left: yellow arrow*) is obviously dilated, whereas the EIA in those that received saline injection (*right: blue arrow*) is only minimally dilated.

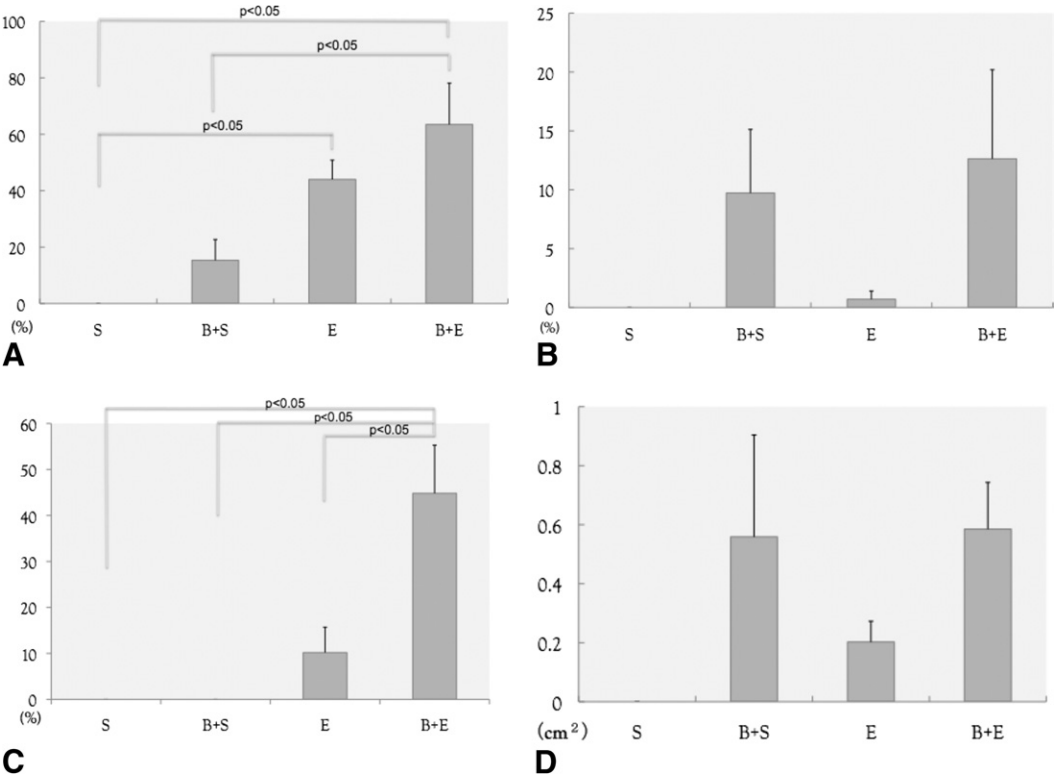

Fig 7. Histologic findings in each group. **A,** Disappearance of the internal elastic lamina is significantly pronounced in the E group compared with the S group ($P < .05$). Disappearance of the internal elastic lamina is significantly pronounced in the B+E group compared with the B+S and S groups ($P < .05$). **B,** Degeneration and disappearance of medial smooth muscle is observed in the B+S and B+E groups, which is not significantly different from the other groups. **C,** Degeneration and disappearance of the external elastic lamina is significantly pronounced in the B+E group compared with the other groups ($P < .05$). **D,** Neointimal thickening is observed in the E, B+S, and B+E groups, but no significant difference is noted among them. *S* group, A closed cavity is created at the external iliac artery (EIA) and is filled with saline. *B+S* group, The EIA undergoes balloon dilation and a closed cavity made by a double-balloon catheter is filled with saline. *E* group, A closed cavity is created at the EIA and is filled with elastase. *B+E* group, The EIA undergoes balloon dilation and a closed cavity made by a double-balloon catheter is filled with elastase.

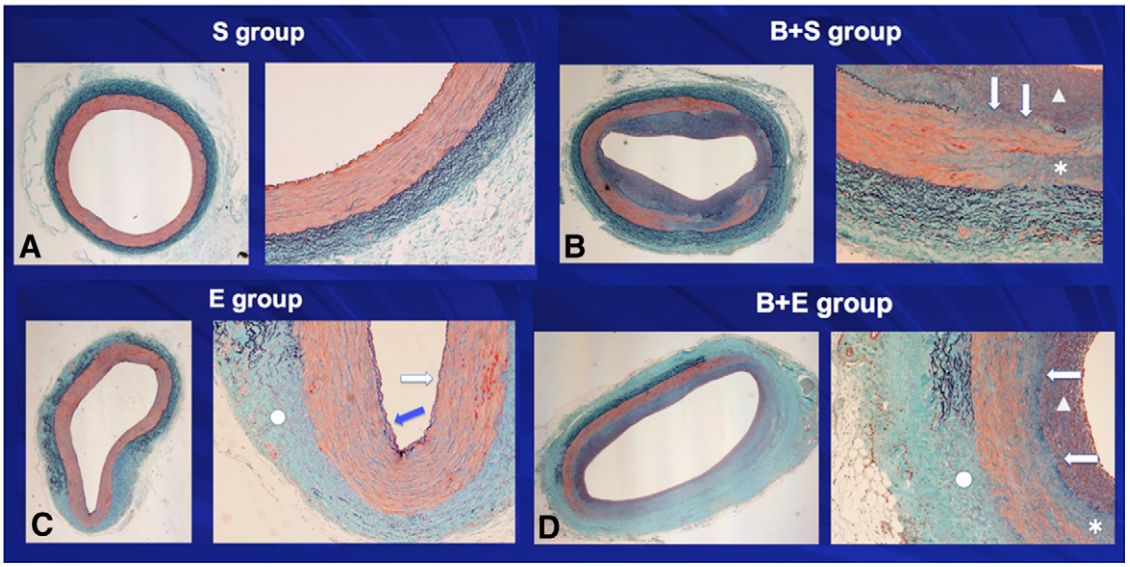

Fig 8. Histologic findings at Elastica-Mason staining of each group (*left* is original magnification, × 20 and *right* is original magnification, × 100) regarding the disappearance of the internal elastic lamina, degeneration and disappearance of medial smooth muscle and the external elastic lamina, and neointimal thickening in the external iliac artery (EIA). **A,** *S* group, received saline injection and EIA shows normal histologic findings. **B,** *B+S* group, The EIA undergoes balloon dilation and a closed cavity made by a double-balloon catheter is filled with saline. The EIA shows disappearance of the internal elastic lamina (*white arrows*), degeneration and disappearance of medial smooth muscle (*), and neointimal thickening (*arrowhead*) but no degeneration and disappearance of the external elastic lamina. **C,** *E* group, The EIA undergoes balloon dilation and a closed cavity made by a double-balloon catheter is injected with elastase. The EIA shows disappearance of the internal elastic lamina (*white arrow*), degeneration and disappearance of the external elastic lamina (*circle*), and neointimal thickening (*blue arrow*), but no degeneration and disappearance of medial smooth muscle. **D,** *B+E* group, The EIA undergoes balloon dilation and a closed cavity made by a double-balloon catheter is filled with elastase at the left EIA. The EIA shows disappearance of the internal elastic lamina (*white arrows*), degeneration and disappearance of the external elastic lamina (*circle*), degeneration and disappearance of medial smooth muscle (*), and neointimal thickening (*arrowhead*).

of external elastic lamina. A tendency for degeneration and disappearance of medial smooth muscle and neointimal thickening was also seen.

DISCUSSION

In a previous study, we documented the histopathology of aneurysm in elastic arteries, such as the abdominal aorta, including the disappearance of smooth muscle cells and the internal elastic lamina.[11] In addition, aneurysms in muscular arteries, such as the popliteal artery, include the disappearance of the internal elastic lamina, external elastic lamina, and smooth muscle cells, as well as neointimal thickening.[12]

In the present study using the EIA, which is a muscular artery, only the B+E group showed significant dilatation of the arterial diameter. Furthermore, the dilated segment in the B+E group histologically showed the disappearance of the internal elastic lamina, medial smooth muscle, and the external elastic lamina, similar to the histology reported for other aneurysms. Therefore, we believe that we achieved a successful aneurysm model in the B+E group. In contrast, the other groups showed no significant arterial diameter dilatation or wall degeneration, and disappearance was less complete.

We used an intraluminal elastase infusion to develop the aneurysm model. For the development of an aneurysm model in vitro and in vivo, the initial event probably includes destruction of elastin, from which destruction of collagen results.[13] Elastase is a proteolytic enzyme distributed in the pancreas and white blood cells that denatures elastin and elastic fibers. That is why many aneurysm model studies commonly use an intraluminal infusion of elastase, as we did. In other studies on mice, calcium chloride has been applied to the surgically exposed thoracic aorta to create an aneurysm,[14] which we used in our endovascular approach.

In addition, we used a vasodilative pretreatment with a balloon catheter before infusion of elastase in the B+E group, which successfully generated an aneurysm. This procedure may be needed due to the morphologic characteristics of the muscular arterial wall; the elastic fibers are distributed over the internal elastic lamina in the intima and external elastic lamina in the adventitia. In a muscular artery, there are few elastic fibers in the internal elastic lamina, whereas the external elastic lamina has a larger number of elastic fibers. Without balloon dilation (E group), the internal elastic lamina disappeared, whereas the external elastic lamina remained, which might have inhibited sufficient artery dilatation. In contrast, the internal and

external elastic laminae both disappeared significantly in the balloon dilation (B+E group), with resulting artery dilatation. In our preliminary experiment, a torn balloon-dilated EIA was observed in both the internal elastic lamina and medial smooth muscle. We suspect that balloon dilation is an essential procedure to help elastase reach the external elastic lamina and to generate the aneurysm model successfully.

The resulting aneurysm model had the following two characteristics. First, an aneurysm can be formed using an endovascular approach with only a simple surgical procedure of exposing the internal carotid artery. Second, our method is applicable to any artery where a closed intraluminal cavity can be created using a double-balloon catheter.

We did not use the abdominal aorta (elastic artery) but rather the EIA (muscular artery), because we thought the former vessel, with many branches, was less suitable for making a closed cavity. However, this may have caused a low vasodilation rate of the generated aneurysm in this study (13%) compared with a previous aneurysm model that used the infrarenal aorta of the pig.[8] The limited rate of vasodilation could be related to the quantity and distribution of the elastic fibers in the EIA or muscular artery wall. A smaller number of elastic fibers is present in a muscular artery wall than in an elastic artery, such as the abdominal aorta,[15] which could explain why elastase affects the muscular artery less than the elastic artery. Elastic fibers are present mainly in the adventitia of the muscular artery, whereas they are distributed mainly in the media of elastic arteries. Therefore, it may be more difficult to locate intraluminal elastase in the elastic fibers of a muscular artery than in an elastic artery, which, in turn, may limit the degree of vasodilation via the denaturation of elastic fibers in the muscular artery aneurysm model.

In future aneurysm model experiments, increasing the vasodilation rate may be accomplished by increasing the elastase dose, changing the pressure or frequency of the balloon dilation, or both, and observing for a longer follow-up period. Alternatively, using elastic arteries, such as the abdominal aorta, might improve the vasodilation rate. For that purpose, developing a double-balloon catheter that can create a shorter, closed cavity would be desirable for application to the aorta, which has many branches.

CONCLUSIONS

We developed an aneurysm model using the arteries of adult Beagles and a simple endovascular procedure. Internal and external elastic lamina degeneration was an important factor to create significantly dilated aneurysms in the muscular artery model. We tried to develop a more dilated aneurysm model using the arteries of adult Beagles, a relatively large animal that we thought would be easy to handle, with a simple and less-complicated endovascular procedure. Establishing an aneurysm model using simple and easy operative techniques is desirable for developing new endovascular devices.

AUTHOR CONTRIBUTIONS

Conception and design: KM, CT, KS, TM, KT
Analysis and interpretation: KM, KS, ST
Data collection: KM, CT, KS, TM
Writing the article: KM, KS, TM, ST
Critical revision of the article: KM, KS, TM, ST
Final approval of the article: KM, KS, TM, ST
Statistical analysis: KM, KS
Obtained funding: KS
Overall responsibility: KM

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