ABSTRACT. A jejunal nodular mass was identified in an aging rat. Histologically, the boundaries between the lesion and surrounding normal tissue as well as between the inner circular muscle and outer longitudinal muscle were indistinct. The lesion consisted of abundant eosinophilic matrix and cells with a large round to oval nucleus and indistinct cytoplasm. There was no characteristic proliferating pattern, nuclear polymorphism and a low mitotic figure count. Masson’s trichrome stain revealed that the intestinal smooth muscles were replaced by the abundant collagen fiber. Immunohistochemistry revealed that the cells within the lesion were labeled with anti-vimentin antibody and not with anti-α smooth muscle actin antibody, suggesting that these cells were fibroblasts. The mass was diagnosed as jejunal fibroplasia.

KEY WORDS: fibroplasia, jejunum, rat
peroxidase conjugate. Mouse monoclonal anti-porcine vimentin antibody (Dako, Carpinteria, CA, U.S.A.), rabbit monoclonal anti-human alpha smooth muscle actin (αSMA) antibody (Abcam, Tokyo, Japan) and rabbit polyclonal anti-rat ionized calcium binding adapter molecule 1 (Iba1) antibody (Wako Pure Chemical Industries, Osaka, Japan) were used as the primary antibodies. Negative controls were performed by omitting the primary antibody. IHC revealed that the cells with a large round to oval nucleus were labeled with anti-vimentin antibody (Fig. 8) and not with anti-αSMA antibody (Fig. 9), suggesting that these cells were fibroblasts. On the other hand, many cells with a small, irregular and chromatin-rich nucleus were also observed in the lesion (Fig. 4). IHC revealed that the cells with a small nucleus were labeled with anti-Iba1 antibody (Fig. 10), suggesting that these cells were macrophages.

It is considered that fibrogenic factors, such as major basic protein, transforming growth factor (TGF)-β, interleukin (IL)-1β and IL-13 secreted by infiltrated eosinophils and mast cells into the lesion, are related to the proliferation of the fibroblasts and the formation of the sclerosing fibers as the pathogenesis of the FGESF [1–4, 7, 9]. In present case, many macrophages labeled with anti-Iba1 antibody and a few eosinophils and mast cells were observed compared with that in the lesion of FGESF. There were no descriptions of a relation of macrophage to the formation of FGESF lesions; however, it is known that fibrogenic factors, such as TGF-β and monocyte chemoattractant protein-1 (MCP-1), are also secreted by macrophages [11]. TGF-β1, a well-known fibrogenic factor, is produced mainly by infiltrated macrophages and related to fibrosis in the intestinal tract [10]. MCP-1, a chemokine associated with the migration of macrophages, is related to fibrosis in the peritoneum [6]. Therefore, it may be possible...
that the fibrogenic factors were secreted not only by the eosinophils and mast cells but also by macrophages, and could be related to the proliferation of the fibroblasts and the deposition of the extracellular matrix and collagen fibers.

Pathogenesis of this lesion may be similar to that of FGESF on the basis of the possible relation of the fibrogenic factor. However, the histologic feature of this lesion did not match to that of FGESF [3]. In present case, the infiltration of eosinophils and mast cells was mild, and there was no formation of the trabecular sclerosing collagen fiber. Therefore, we diagnosed this lesion as ‘fibroplasia’ in the rat jejunum.

Fig. 5. Most of the smooth muscles are replaced by the abundant collagen fiber stained blue, and there are a few residual inner circular smooth muscles stained red. MT. Bar, 50 µm.

Fig. 6. Argyrophilic fibers stained black surrounding the smooth muscle cells disappear in the lesion. Watanabe’s Silver. Bar, 50 µm.

Fig. 7. A few eosinophils with red granules and mast cells with blue granules are infiltrated into the lesion. EMB. Bar, 20 µm.

Fig. 8. The cells with a large round to oval nucleus are labeled with anti-vimentin antibody. IHC. Bar, 20 µm.

Fig. 9. The cells with a large round to oval nucleus are not labeled with anti-αSMA antibody. Most of the smooth muscles disappear, and a few residual inner circular smooth muscles are labeled with anti-αSMA antibody. IHC. Bar, 50 µm.

Fig. 10. The cells with a small, irregular and chromatin-rich nucleus are labeled with anti-Iba1 antibody. IHC. Bar, 20 µm.

Fig. 11. A few residual inner circular smooth muscles are stained red. EMB. Bar, 20 µm.

Fig. 12. The cells with a large round to oval nucleus are not labeled with anti-αSMA antibody. Most of the smooth muscles disappear, and a few residual inner circular smooth muscles are labeled with anti-αSMA antibody. IHC. Bar, 50 µm.

To our knowledge, the definition of the term ‘fibroplasia’ in the small intestine has not been clarified properly. In the small intestine, it is very difficult to define and differentiate fibrosis and fibroplasia as we consider it possible that fibroplasia represents different histologic futures depending on the tissues and organs in which the term is used. In the skin, fibroplasia and fibrosis are defined as sequential reactions of wound healing. Fibroplasia is often used synonymously with granulation tissue, and fibrosis is a late stage of fibroplasia [12]. When we follow the definition of this field, the lesion in this case is considered to be fibrosis. On the other hand, in the medium-sized muscular artery, fibroplasia of the intima, media and adventitia is described as fibromuscular dysplasia [5]. In this disease, medial fibroplasia is classified into one of the medial dysplasia and defined as thickening of the media caused by the replacement of the medial smooth muscle by collagen [8]. Therefore, it is reasonable to define that fibroplasia is the lesion that the original constituents of the organs and tissues are replaced by excessive collagen. When we follow the definition of this field, the lesion in this case is considered to be fibroplasia. Although there is a difference between the digestive system and the cardiovascular system, we diagnosed the lesion in this case as fibroplasia from the similarities of the lesion in that the smooth muscle is replaced by collagen in the tubular tissue, in which the smooth muscle is one of the main components.
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