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Abdominal elastotic lesions. A clinicopathologic study of 23 cases

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Abstract

Abdominal elastotic deposits are uncommon lesions that often presents as polyps. They show three histological patterns: fibroelastosis, angioelastosis, and elastofibroma. We describe 23 cases including rare locations, such as mesentery, greater omentum, hernia sac, spleen, peripancreatic fat, and hypodermal fat. The age of the patients ranged from 49 to 93 years (mean, 76.8 years). Most lesions were discovered incidentally in the microscopic study. The most frequent locations were peritoneal subserosa (43.5%) and mesentery/mesocolon/greater omentum (39.1%). The most common pattern was fibroelastosis (69.6%) followed by angioelastosis (26.1%). We observed one case of omental elastofibroma. A review of the 14 abdominal elastofibromas described including our case revealed that the age of the patients ranged from 45 to 88 years (mean, 68.5 years). Female predominance is striking (M:F, 1:12). The most common site was the stomach (50%). The greater omentum (14.3%), small intestine (7.1%), and pancreas (7.1%) are very rare sites for this lesion. Only one case before ours has been published in the greater omentum. The size of the lesions ranged from 0.7 cm to 8 cm (mean 3.2 cm). In 36.4% of the cases located in the digestive tract, the mucosa did not show alterations. Ulcerations (36.4%) or polypoid excrescences (18.2%) were mostly observed. Six (42.9%) cases were asymptomatic and six (42.9%) cases simulated a neoplasm. Two cases were associated with elastofibromas in other locations. Differential diagnosis includes amyloidoma, elastofibrolipoma, mesenteric elastic vascular sclerosis in neuroendocrine tumors, diverticular disease elastosis, pseudoxanthoma elasticum, pulse granuloma, and digestive lesions in patients treated with D-Penicillamine.

Keywords: gastrointestinal tract, abdominal cavity, elastosis, fibroelastosis, angioelastosis, elastofibroma.

Introduction

Elastotic changes of the alimentary canal are considered to be uncommon benign lesions that result in focal deposits of elastic fibers that usually are accompanied by fibro-collagenous tissue. These elastic accumulations can be observed anywhere between the oral cavity [1, 2] and anus [3–8]. They may present as polyps [5], extensive, ill-defined, non-polyp-forming deposits [8], or pseudo-neoplastic accumulations of elastic fibers [9]. Exceptionally, fibroelastotic lesions have been described in intra-abdominal solid organs, such as the liver [10] or pancreas [11, 12] with a nodular or pseudotumoral appearance. In the gastrointestinal tract (GIT), the elastotic deposits are usually seen in the mucosa, *muscularis mucosae*, or submucosa layers [3–7], occasionally in the *muscularis propria* [8], and rarely in the subserosal fat [13]. According to Lichtmannegger *et al.* [8], the lesions are more frequently found in the upper than in the lower GIT (77.8% *vs.* 22.2%). In the upper GIT, these deposits are more common in the stomach. In the lower GIT, the deposits are more frequent in the sigmoid and/or rectal region where they commonly present as polypoid lesions [8]. Polypoid lesions represent 23.5% of global elastotic deposits [8].

Histopathologically, elastotic accumulations are commonly characterized by forming amorphous masses of granular, flocculent, and/or fibrillar eosinophilic material reminiscent of simple fibrosis or amyloid deposits. Thus, the elastotic change can be misdiagnosed as amyloid [4]. Therefore, the detection requires the use of Congo Red staining and Verhoeff's method for elastic fibers. In addition, the elastotic deposits of the GIT have been classified into three histological patterns: (*i*) fibroelastosis, (*ii*) angioelastosis, and (*iii*) elastofibroma [8].

From the first description of an elastotic deposit by Enjoji *et al.*, in 1985 [14], few reports have studied this topic and very few monographs on digestive pathology include this subject. Since these lesions are rarely published, they remain to be widely unknown and may cause diagnostic problems.

Aim

In this study, we present our experience with 23 cases observed in a seven-year period of study. We report herein abdominal locations not previously described, such as mesentery, hernia sac, spleen, peripancreatic fat, and abdominal hypodermic adipose tissue. In addition, we review the literature.

Materials and Methods

We observed an index case of GI elastotic lesion in 2013 and prospectively collected 22 cases during the

following six years. The collected cases correspond to a third of the total of the GIT specimens received in the Department of Pathology during the study period. These cases were directly observed by the authors of the present work. The specimens were fixed in 4% phosphate-buffered formalin and embedded in paraffin. Sections cut at 5 µm were stained with Hematoxylin–Eosin (HE), Congo Red method, and Verhoeff's elastic procedure (VEP). An immunohistochemical (IHC) study was carried out on 10 cases using the EnVision FLEX+ Visualization System (Dako, Agilent Technologies, SL, Las Rozas, Madrid, Spain). The IHC reaction was performed using appropriate tissue controls for the antibodies utilized. Automatic staining was carried out on a Dako Omnis Autostainer (Agilent Technologies, SL). Antibodies used in the IHC study are detailed in Table 1. Clinical data from all cases were obtained from Hospital records.

Results

Clinicopathological data are summarized in Table 2. The age of the patients ranged from 49 to 93 years [mean, 76.8 years, standard deviation (SD) 11.6 years; median, 78 years]. Women were predominant (M:F, 1:1.5). One lesion manifested as a colonic polyp during routine screening. The rest of the lesions were discovered incidentally in the microscopic study of the surgical specimen. Thus, these cases were mostly found when studying large surgical specimens of interventions for processes that threatened the patient's life. The most frequent locations were peritoneal subserosa (43.5%) and the adipose tissue of the mesentery, mesocolon, or greater omentum (39.1%). The subserosa location included the hernia sacs, the appendicular subserosa, and the peritoneum in fibrous peritonitis. The most common histological pattern was fibroelastosis (69.6%) followed by angioelastosis (26.1%). In contrast, we only observed one case of true elastofibroma with similar features to the entity described in subscapular location. None of the 23 cases presented a recurrence.

Table 1 – ***Immunohistochemical antibodies used in this study***

Antibody	Source	Clone	Dilution	Retrieval solution pH (Dako)
Calponin	Dako	CALP	1:200	High
CD34	Dako	QBEnd 10	FLEX RTU	High
Factor XIIIa	Gennova Scientific, SL	AC-1A1	1:100	High
α-SMA	Dako	1A4	FLEX RTU	High

α-SMS: Alpha-smooth muscle actin; CD34: Cluster of differentiation 34; Dako (Agilent Technologies, SL, Las Rozas, Madrid, Spain); Gennova Scientific SL (Sevilla, Spain); RTU: Ready-to-use.

Table 2 – ***Clinicopathological data for cases of elastotic change***

Case No.	Age [years]/ Gender	Location	Histological pattern	Associated pathology
1.	86/F	Mesocolon	Angioelastosis	Colon infarction
2.	87/M	Left inguinal hernia sac	Fibroelastosis	Postsurgical ossifying myositis
3.	77/M	Mesocolon	Angioelastosis	Infarction of the splenic angle of the colon
4.	71/M	Mesocolon	Fibroelastosis	Unresectable tumor of the transverse mesocolon
5.	49/F	Mesocolon	Fibroelastosis	Gastric signet ring cell carcinoma
6.	86/F	Appendicular subserosa	Fibroelastosis	Appendicular mucocele
7.	57/M	Colon polyp	Angioelastosis	Colon adenocarcinoma on tubular adenoma
8.	74/F	Greater omentum	Elastofibroma	Mucinous cystadenocarcinoma of the ovary
9.	77/F	Periumbilical hernia sac	Fibroelastosis	No
10.	60/M	Sigmoid mesocolon	Fibroelastosis	Rectal adenocarcinoma treated with adjuvant chemotherapy and radiotherapy
11.	78/F	Mesocolon	Fibroelastosis	Colon adenocarcinoma, multiple tubular adenomas
12.	80/F	Mesentery	Angioelastosis	Ileo-rectal fistula
13.	89/F	Mesocolon	Angioelastosis	Sigmoid colon adenocarcinoma
14.	59/F	Lower pole of the spleen	Angioelastosis	Colon adenocarcinoma, peritoneal carcinomatosis
15.	72/F	Peripancreatic fat	Fibroelastosis	Chronic pancreatitis
16.	82/F	Hernia sac	Fibroelastosis	Abdominal eventration
17.	85/M	Abdominal hypodermic adipose tissue	Fibroelastosis	Suspected amyloidosis
18.	84/M	Sigmoid colon subserosa	Fibroelastosis	Ileocolic anastomosis
19.	76/F	Small intestine subserosa	Fibroelastosis	Intestinal ischemia
20.	83/F	Terminal ileum subserosa	Fibroelastosis	Suspected metastatic carcinoma of the breast
21.	91/M	Peritoneum	Fibroelastosis	Organized fibrous peritonitis
22.	93/M	Hernia sac	Fibroelastosis	Incarcerated femoral hernia
23.	71/F	Sigmoid colon subserosa	Fibroelastosis	Anorectal melanoma

F: Female; M: Male.

Fibroelastosis manifested as amorphous masses of granular, pale, eosinophilic to gray amphophilic material often with a fibro-collagenous component. There were scant stromal spindle cells (Figure 1A). Purely slender, wavy, fibrillar material was the component of other lesions (Figure 1B). Frequently, this fibrillar material could be observed within the granular material constituting a mixed deposit (Figure 2A). Subsequent staining with VEP revealed that the lesions were almost exclusively made up of densely arranged elastic fibers (Figure 2B).

The lesions were solitary or forming two or more focal deposits.

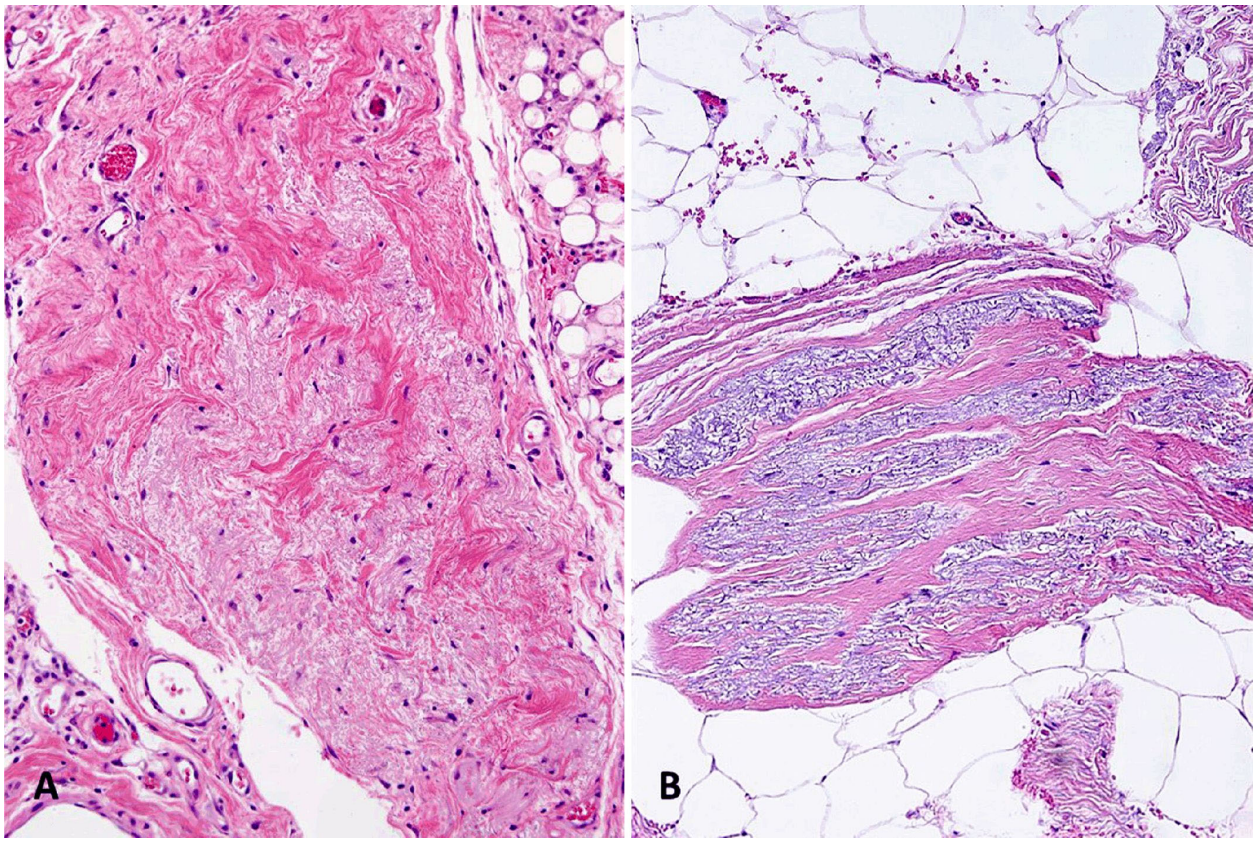

Figure 1 – ***Fibroelastosis: (A) Finely granular, pale, eosinophilic material with fibrous component. (B) Wiry fibrillar material with pseudomyxoid change. There is accompanying fibrous tissue. Hematoxylin–Eosin (HE) staining: (A and B) ×200.***

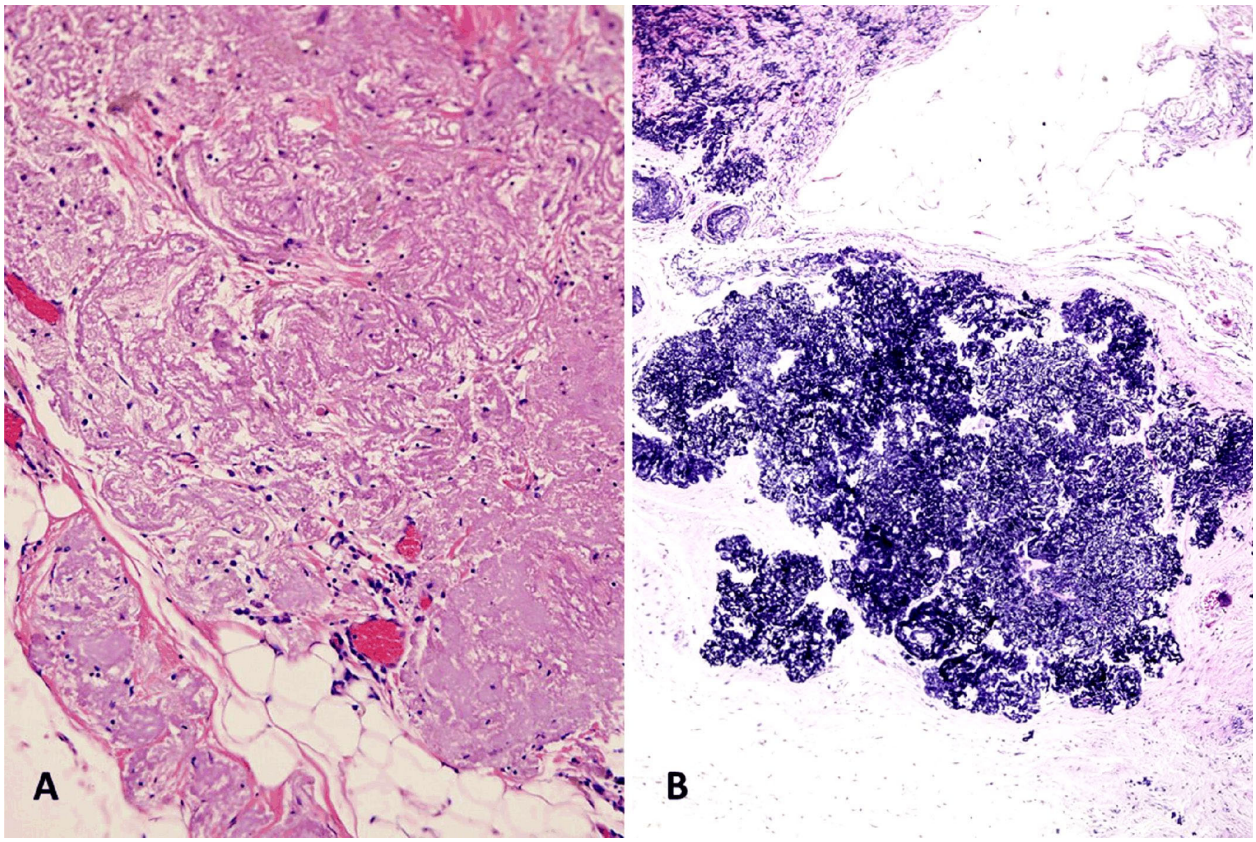

Figure 2 – ***Fibroelastosis: (A) Mixed flocculent, finely granular, and fibrillar deposit; (B) The elastic fibers that comprise the lesion are revealed with an elastic staining. Hematoxylin–Eosin (HE) staining: (A) ×200. Verhoeff's elastic procedure (VEP): (B) ×100.***

Fibroelastosis pattern was seen in cases of mesocolon (Figure 3A), hernia sacs (Figure 3, B and C), intestinal subserosa, fibrous peritonitis (Figure 3D), peripancreatic fat, and hypodermal abdominal fat. Angioelastosis was characterized by dense elastotic deposits centered around vessels (Figure 4, A–D).

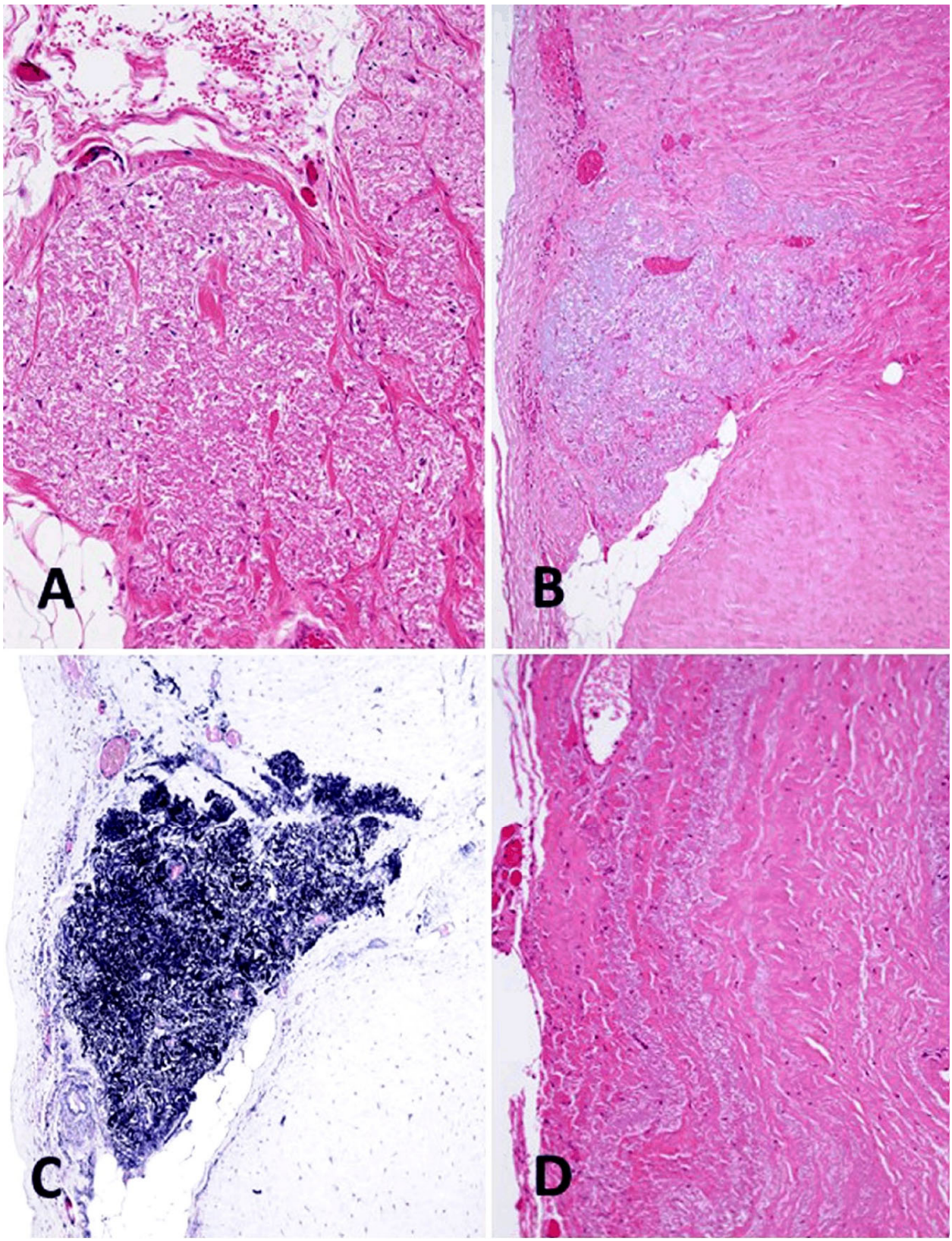

Figure 3 – ***Fibroelastosis: (A) Purely granular eosinophilic deposit in mesocolon; (B) Grayish, amphophilic, granular deposit in hernia sac; (C) The elastic special staining highlights the elastic fibers in the hernia sac; (D) Linear granular deposits in a case of fibrous peritonitis. Hematoxylin–Eosin (HE) staining: (A and D) ×200; (B) ×100. Verhoeff's elastic procedure (VEP): (C) ×100.***

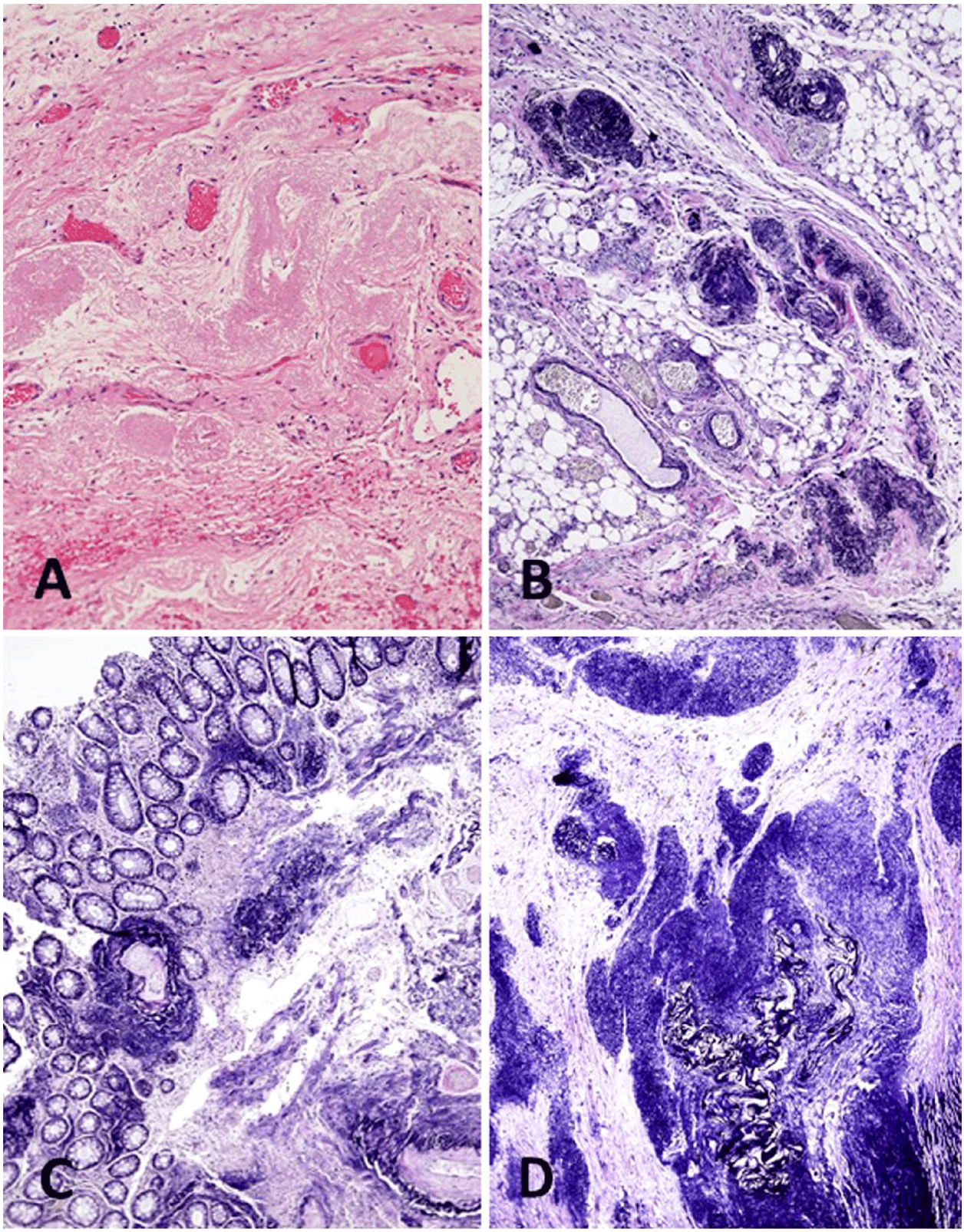

Figure 4 – ***Angioelastosis: (A) Mesentery: the finely granular eosinophilic material causes thickening of the vascular wall with lumen stenosis; the vessels are surrounded by a mantle of untruncated elastic fibers with obliteration of the lumens; (B) Mesocolon; (C) Colon polyp; (D) Spleen. Hematoxylin–Eosin (HE) staining: (A) ×200. Verhoeff's elastic procedure (VEP): (B–D) ×100.***

The vessels presented fibrous thickening of their walls and sometimes obliterated lumina. These vessels were surrounded by a broad rim of untruncated elastic VEP-positive fibers. The lesions were much more frequent in veins than in arteries. A diffuse elastotic component might also be associated. Sparse stromal spindle or stellate cells were seen among slender elastic fibers. Angioelastosis pattern was observed in cases of mesentery (Figure 4A), mesocolon (Figure 4B), colon polyp (Figure 4C), and lower pole of the spleen (Figure 4D). The only elastofibroma showed widely distributed lesions in the greater omentum. Multiple lesions were poorly demarcated. There was a

hypocellular mixture of collagen bundles and coarse elastic fibers (Figure 5A). The elastic fibers were curled and truncated with serrated outlines (Figure 5B). Besides, there were elastin globules sometimes in a linear arrangement. Perivascular elastofibromatous lesions were also present (Figure 5C). The elastic staining confirmed the characteristic thick serrated fibers and the globules (Figure 5D). Scarce spindle or stellate stromal cells were present. The characteristic features of elastofibroma were also observed in the skipped lesions.

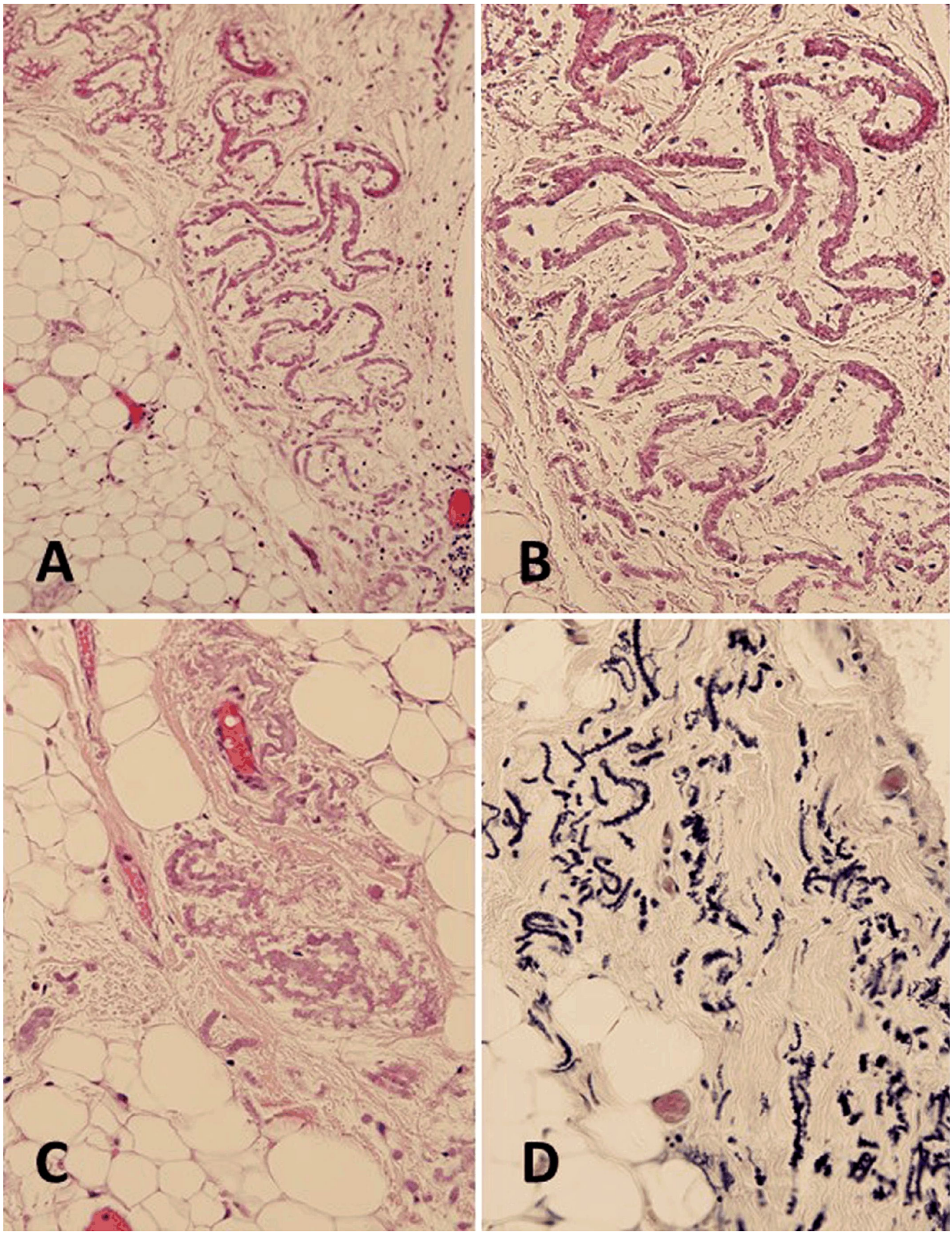

Figure 5 – ***Elastofibroma: (A) Lesion showing paucicellular collagen bundles and coarse truncated wavy elastic fibers; (B) Elastic fibers showed prominent eosinophilia and serrated outlines; (C) Skipped lesion – presence of globules and a perivascular elastofibromatous lesion; (D) VEP confirmed the presence of fragmented fibers with serrated edges, and globules. Hematoxylin–Eosin (HE) staining: (A) ×200; (B and C) ×400. Verhoeff's elastic procedure (VEP): (D) ×200.***

The IHC staining for cluster of differentiation (CD) 34, Factor XIIIa, calponin, and alpha-smooth muscle actin (α-SMA) showed in all lesions, presence in a variable quantity of a mixture of CD34, Factor XIIIa, calponin, and α-SMA-positive cells (Figure 6, A–D). These cells were spindle- or stellated-shaped. The cellular body presented either elongated or triangular morphology and contained an ovoid nucleus rich in chromatin. The cells were scattered among the elastic fibers. There was no evidence of calcification of these fibers in any of the three histological patterns. Congo Red staining was negative in all cases. Mitoses were not present in the stromal spindle cells.

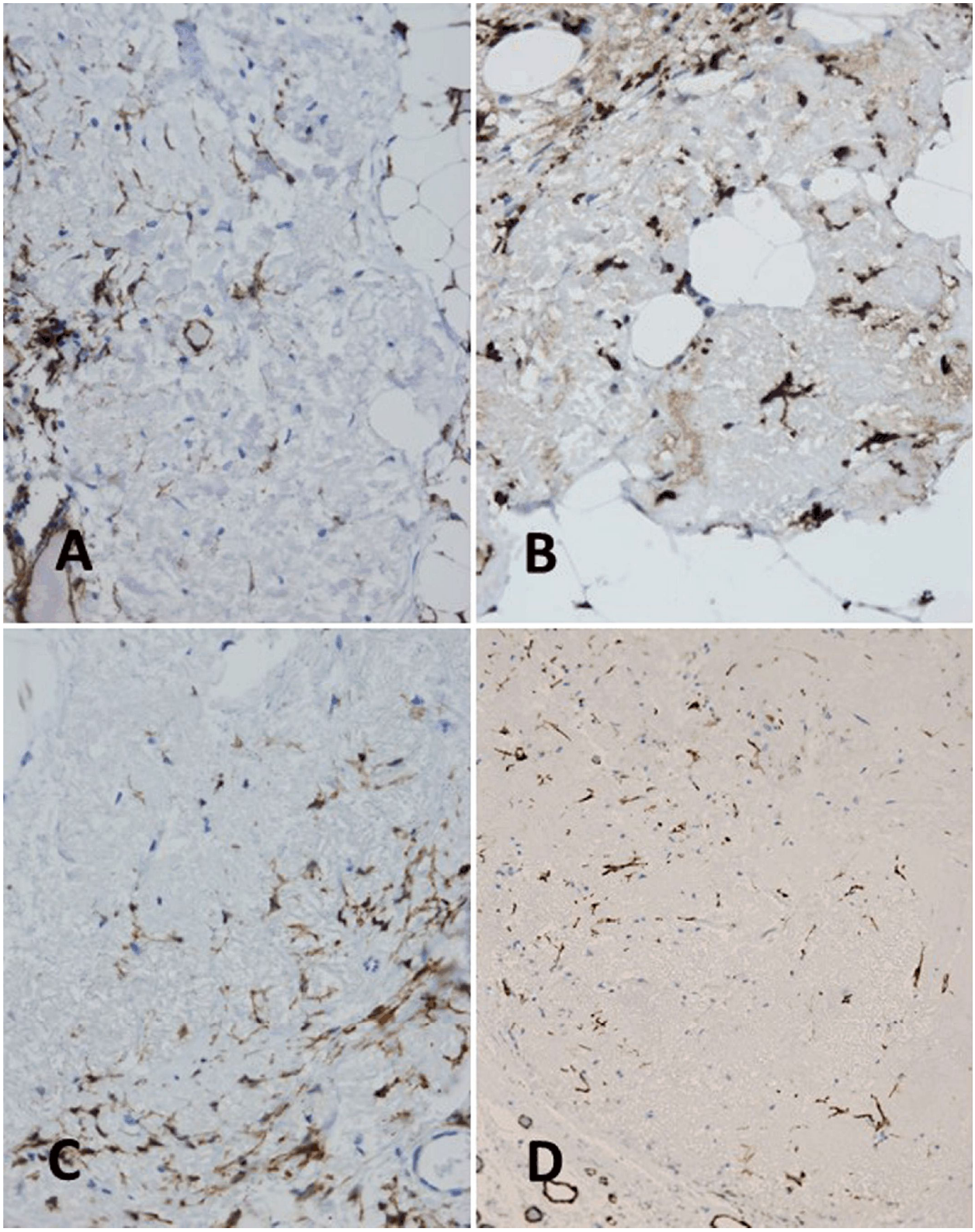

Figure 6 – ***Cellular immunohistochemistry. All lesions showed in varying amount a mixture of CD34 (A, ×400), Factor XIIIa (B, ×400), calponin (C, ×400), and α-SMA (D, ×200) positive cells. These cells were spindle- or stellated-shaped and were often predominant at the periphery of the elastotic deposits. CD34: Cluster of differentiation 34; α-SMA: Alpha-smooth muscle actin.***

Discussions

Focal accumulations of elastic fibers in the GIT are usually accompanied by a variable amount of fibrous connective tissue and show ill-defined margins. They can form focal, bifocal, or multifocal masses that may simulate polyps, ulcers, or neoplasms [3, 9, 14, 15]. All these elastotic deposits do not tend to calcify.

In this report, we describe 23 intra-abdominal elastotic lesions. These lesions were more common in women (M:F, 1:1.5) and mainly affected middle-aged to elderly people (range 49 to 93 years, mean 76.8 years) (Table 2). Among the cases, we present locations not previously described, such as mesentery, hernia sac, spleen, peri-pancreatic fat, and abdominal hypodermic adipose tissue.

The most frequent locations were peritoneal subserosa (43.5%) and the fat tissue of the mesentery, mesocolon, or greater omentum (39.1%). We have classified the cases according to the histological patterns described by Lichtmannegger *et al.* [8], as fibroelastosis (53.3%), angioelastosis (40.0%), and elastofibroma (6.7%). The elastotic lesions we are reporting were painless, incidental, and discovered microscopically.

In other series, some of the patients presented abdominal pain, obstruction, hematochezia, or anemia [3, 4, 12]. Unlike our series, other authors presented a balanced gender ratio, most lesions were found during endoscopic examination in the stomach, colon, or rectum (therefore, polypoid lesions were frequent) [6, 8], and most of the cases showed a fibroelastosis pattern [8]. We attribute this discrepancy to the different origin of the cases. Our cases were mostly found when studying large abdominal surgical specimens of interventions for serious processes that pose a threat to patient survival. On the other hand, the presence of elastotic change in the large surgical specimens can be easily overlooked, especially when they are associated with other more significant pathological processes. Elastofibroma is the least common lesion. Table 3 summarizes the clinicopathological features of the previously described 13 cases of this type of lesion [12, 14–25], as well as the present one. The age of the patients ranged from 45 to 88 years (mean, 68.5 years; SD 11.2 years; median, 70.5 years). There was an obvious predominance in women (M:F, 1:12). The most common site was the stomach (50%) followed by the sigmoid colon or rectum (21.4%). The greater omentum (14.3%), small intestine (7.1%), and pancreas (7.1%) are very rare sites for this lesion. Thus, only one case prior to ours has been published in the greater omentum [16] and in both cases the process was multifocal. The size of the lesions ranged from 0.7 cm to 8 cm (mean 3.2 cm, SD 2.2 cm, median 3 cm). In 36.4% of the cases located in the digestive tube, the mucosa did not show alterations. Ulcerations (36.4%) or polypoid excrescences (18.2%) comprised more of the 50% of cases. Six (42.9%) cases were asymptomatic (Cases Nos. 3, 4, 5, 7, 8 and 14). Six (42.9%) cases simulated a neoplasm (Cases Nos. 2, 6, 9, 10, 11 and 12) and two (14.3%) cases were associated with elastofibromas in other locations. Thus, one case was bilateral subscapular [14] and another one was bronchial [18]. Kai *et al.* [22] reported a case of an elastofibroma in the stomach with a striking association to vessels in the periphery of the lesion. We have confirmed this finding (Figure 5C).

Table 3 – ***Reported intra-abdominal elastofibromas***

Case No./ [Reference]	Age [years]/ Gender	Location	Maximum size [cm]	Mucous surface	History	Extra-abdominal elastofibroma
1/[14]	69/F	Stomach	8	Ulcerated	Peptic ulcer	Bilateral subscapular
2/[16]	76/F	Greater omentum	Multifocal, main mass 5 cm	NA	Palpable intra-abdominal mass	No
3/[17]	58/F	Rectum	NR	No	Multiple myeloma, dysphagia	No
4/[18]	88/F	Small bowel	NR	No	Bronchopneumonia	Bronchial
5/[19]	69/F	Sigmoid colon	0.7	Polypoid	Asymptomatic	No
6/[20]	76/F	Stomach	3.5	No	Acute calculous cholecystitis syndrome, gastric mass	No
7/[21]	72/M	Sigmoid colon	0.8	Polypoid	Asymptomatic	No
8/[22]	77/F	Stomach	Multifocal	No	Asymptomatic, gastric tubular adenocarcinoma	No
9/[23]	75/F	Stomach	4	Ulcerated	Epigastric pain, peptic ulcer	No
10/[24]	52/NR	Stomach	4	Mucosal bulge	Gastroesophageal reflux disease	No
11/[15]	61/F	Stomach	3	Ulcerated	Epigastric pain, nausea, vomiting	No
12/[12]	45/F	Pancreas	2.5	NA	Abdominal pain	No
13/[25]	67/F	Stomach	2	Ulcerated	Recurrent hematemesis and melena	No
14/[Our report]	74/F	Greater omentum	Multifocal	NA	Asymptomatic, ovarian carcinoma	No

F: Female; M: Male; NA: Not available; NR: Not reported.

Elastotic deposits may vary in intensity. In the series of 162 cases reported by Lichtmannegger *et al.* [8], 80% of cases were mild, 16% were moderate, and less than 4% were marked. The high rate published by these authors was because they included any case showing an increase in the elastic fibers in the mucosa [8].

This layer under normal conditions does not contain elastic fibers. In the present series, the elastotic deposits were intense in all cases. In addition, there was an increase in CD34+ fibroblastic cells suggesting a role of these cells in the production of elastic and collagen fibers. CD34+ stromal fibroblastic/fibrocytic cells (CD34+ SFCs) are the main reservoir of tissue mesenchymal cells. They are involved in several processes, such as repair,

inflammatory/immune lesions, fibrosis, and tumor stroma formation [26]. Besides, these cells are reactive for myocyte enhancer factor-2 (MEF-2), prominin 2 (CD133), and Factor XIIIa [27], periostin, and tenascin-C [28]. CD34+ SFCs can show differentiation into α-SMA+ stromal cells or calponin+ (myofibroblasts) during inflammatory or repair processes [29].

The pathogenesis of elastotic lesions of the GIT is uncertain. The association in some cases with radiotherapy, ulcerative, vascular, inflammatory, or postoperative conditions suggests a reactive process [4–6]. Fibroelastosis can also be striking in endometriosis [30]. Ischemic injury may be operative in cases of angioelastosis. However, some cases are idiopathic. Thus, these lesions have similarities with the so-called pulmonary apical (scar) cap [31]. On the other hand, a connection with systemic diseases of the connective tissue, such as *pseudoxanthoma elasticum* or Ehlers–Danlos syndrome has not been demonstrated. Otherwise, it cannot be ruled out that in some cases the elastosis is favored by an intrinsic aging process as is observed in the skin [32, 33], and suggested in the oral mucosa [1], or intestine [34]. Besides, it should be considered that a diagnostic of elastotic lesion does not exclude an underlying or adjacent malignancy [8]. The elastic component of normal tissues constitutes a complicated three-dimensional network formed during the stages of organogenesis and maturation of the organs. Injured tissues can have difficulty establishing the normal elastic pattern, frequently leading to disorganized and excessive deposits of elastic fibers [35]. Some authors suggest that elastofibroma is not a reactive change but a neoplastic process. CD34+ SFCs have an important role in the development of elastofibroma, and one publication provided evidence of clonality of these cells [36]. However, it is difficult to reconcile the existence of skip lesions in our case and other cases [16, 22] based exclusively on the neoplastic development theory. Di Vito *et al.* [28] also consider that the neoplastic nature of elastofibroma is unlikely.

The differential diagnosis of elastotic lesions includes amyloidoma, elastofibrolipoma, elastic vascular sclerosis of the mesentery in neuroendocrine (carcinoid) tumors, elastosis in diverticular disease, *pseudoxanthoma elasticum*, pulse granuloma, and digestive lesions in patients long-term treated with D-Penicillamine.

Amyloid focal deposits localized to the small or large intestine without systemic involvement (amyloidoma, tumoral amyloidosis) are rare and typically present as a polypoid protruding lesion or a large mass [37–39]. Amyloidoma may manifest as a stenosing mass, gastrointestinal bleeding, or perforation. Histological study reveals an extracellular, eosinophilic, amorphous deposit that may involve vascular walls. Giant cells can be present. The amyloid deposit is usually more uniform than the elastotic accumulation. The characteristic green color in polarized light after staining with Congo Red is diagnostic. Elastofibrolipoma is an encapsulated mass of central and peripheral adipose tissue mixed with fibrous bands, abnormal elastic fibers, and eosinophil elastic globules. The adipose tissue is mature and present in each high-power field. The lesion has been described in the anterior mediastinum and the subscapular area [40–42]. The process can be bilateral [43]. No intra-abdominal case has been published. Most authors consider the lesion a true benign neoplasm akin to a lipoma. Neuroendocrine (carcinoid) tumors of the ileum can produce occlusion of mesenteric vessels due to fibrosis and adventitial elastosis. The advanced lesions result in severe ischemia or infarction of bowel with high mortality [44–46]. This vascular lesion of neuroendocrine tumors does not form a confluent pseudoneoplastic mass as it occurs in the angioelastosis type of the elastotic lesions we are reporting. Elastosis in diverticular disease of the sigmoid colon displays an increase in elastic fibers of the *muscularis propria*. Elastin is laid down between the muscle cells with distortion of the fascicular pattern of the longitudinal external layer (*tenia coli*) [47, 48]. Therefore, the elastin deposit is in the muscularis propria and it is not confluent as seen in intra-abdominal fibroelastotic lesions. *Pseudoxanthoma elasticum*, an infrequent hereditary disease, may show submucosal yellowish nodular or linear lesions similar to the xanthoma-like skin lesions. Histologically, elastic fibers are variably described as fragmented, curled, frayed, lumpy, and raveled. These elastic fibers are intensely basophilic due to the prominent calcification. Gastric hemorrhage is an unusual complication [49–51]. In contrast, intra-abdominal fibroelastosis, angioelastosis or elastofibroma do not tend to calcify. Pulse granuloma presents as a unifocal or multifocal nodular mass that usually involves the external surface of the bowel. The lesion can show a hyaline predominant structure mimicking amyloid [52, 53]. It is a pseudotumor that results from the entrapment of food introduced through mucosal trauma. Variable presence of hyaline ribbons and rings, inflammation, foreign body giant cells, calcifications, and vegetable food may be observed [52–54]. In hyaline dominant cases with no inflammatory cells, polariscopy may fail to detect vegetable matter. D-Penicillamine is a copper chelator that has been used for the treatment of Wilson's disease, cystinuria, rheumatoid arthritis, scleroderma, and primitive biliary cirrhosis [55]. Long-term, high-dose administration of D-Penicillamine mainly causes skin lesions, kidney disease, and immune depression. This treatment rarely causes digestive complications. However, Wassef *et al.* [56] described a case of terminal ileum stenosis with numerous ulcerations that showed elastosis and fibrosis in the submucosa and *muscularis propria*. The topography of the elastosis was perivascular and diffuse. The elastic fibers were abnormal with bulgings and branchings giving a "lumpy-bumpy" or "lamp brush" appearance. The "lumpy-bumpy", "lamp brush", or "bramble-bush" elastic fiber was considered pathognomonic for Penicillamine-induced elastosis [57]. However, these features are very similar by conventional microscopy to those of the elastofibroma elastic fibers [58]. In fact, the fundamental difference between both types of elastic fibers is that those of the fibroelastoma are localized and those that appear after the administration of D-Penicillamine are widely and diffusely arranged (systemic elastotic process) including normal visceral tissues and normal skin [59].

Conclusions

Intra-abdominal elastotic lesions are assigned to different categories that are expression of a localized response to injury. They are underrecognized but have characteristic histological features. Most lesions are discovered incidentally. The elastofibroma pattern is most common in the stomach and can manifest as a pseudoneoplastic obstructive process. The differential diagnosis is broad. Recognition of these lesions is important to avoid confusion with diverse significant processes.

Conflict of interests

The authors declare that they have no conflict of interests.

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Ethical responsibilities

The authors declare that the procedures followed in this research conform to the ethical standards in accordance with the World Medical Association and the Declaration of Helsinki.

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