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Case report

Two cases of primary leiomyosarcoma of sigmoid colon treated with laparoscopic surgery: A case report and a review of literature

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ABSTRACT

Introduction and importance: Leiomyosarcoma (LMS) of the colon is an extremely rare and highly invasive tumor arising from the muscularis propria of the gastrointestinal tract. After the introduction of oncogenic role of KIT by immunohistochemistry (IHC), the reported cases of gastrointestinal leiomyosarcoma were highly limited. True LMS of the colon is such a rare disorder that there isn't much description of its nature.

Case presentation: We reported two very rare cases of primary leiomyosarcoma of sigmoid colon, which referred to our institution with symptoms of abdominal pain, lower GI bleeding and fatigue. After the initial investigations, both patients were diagnosed with primary LMS of sigmoid colon that underwent laparoscopic tumor resection.

Clinical discussion: The classical colonic LMS presents with a vast majority of non-specific symptoms including mild abdominal pain, fresh/obscure rectal bleeding, and weight loss. The most common location for colonic LMS is the sigmoid colon, and ascending colon. The prognostic factors for the disease outcome have not been established properly; however, patient age, tumor size/grade, and local/distant dissemination are of great importance.

Conclusion: Herein, we reported two rare cases of primary leiomyosarcoma of sigmoid colon that was treated with laparoscopic surgery.

1. Background

Leiomyosarcoma (LMS) of the colon is an extremely rare and highly invasive tumor arising from the muscularis propria of the gastrointestinal tract [1]. Before 1998, every neoplasm raised from mesenchymal cells was mistakenly classified as LMS; However, after the introduction of the oncogenic role of KIT by immunohistochemistry (IHC), only a very small number of true gastrointestinal leiomyosarcomas were reported [2]. Immunohistochemically, true LMS expresses actin (SMA) and desmin without the expression of GIST markers (CD117, CD34, and DOG1.1) and KIT mutations, which allow distinguishing LMS from other GI mesenchymal neoplasms [1,3]. We herein describe two rare cases of Leiomyosarcoma of sigmoid colon that were treated with laparoscopic resection. This study was reported in line with the SCARE criteria [4].

2. Case presentation

2.1. Case 1

We reported a case of a 48-year-old man referred to our surgical outpatient clinic in December 2019. He presented with mild intermittent abdominal pain and occasional rectal bleeding for eight months without any history of weight loss, fatigue or night sweating. Furthermore, He had no family history of malignant neoplasia. General examination and vital signs were normal. Abdominal examinations revealed an ill-defined, round, fixed mass at the left lower quadrant. Digital rectal examination was normal. Routine lab data were unremarkable, except mild microcytic anemia that was due to his thalassemia trait (Mentzer index = 13.46). Carcinoembryonic antigen (CEA) test was normal. After the initial investigation patient underwent colonoscopy that was suggestive of a large polypoid mass with hard texture in 15 cm from the anal

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verge; rest of the colon were unremarkable.

2.1.1. Treatment

Before the operation, patient was scheduled for a routine metastatic workup with Contrast-enhanced computed tomography (CT) of thoracic and abdomen and pelvis, and liver function test (LFT). The abdominal CT showed diffuse wall thickening of the sigmoid colon and distal part of the left colon associated with enhancing soft tissue mass measuring about 72 mm × 40 mm in sigmoid colon that was suggestive of a malignant process (Fig. 1). Also, evidence of multiple regional lymph nodes was seen adjacent to the sigmoid colon. Chest CT and Liver function test (LFT) were unremarkable. The surgery was conducted under general anesthesia with the purpose of complete resection of the tumor and involved lymph nodes.

Laparoscopic anterior resection was performed with resection of sigmoid colon and upper part of rectum. Colorectal anastomosis was performed with no diverting ileostomy. Tumor dimensions were 8 × 6 × 4.5 cm with a smooth surface and the cross-section showed homogenous white appearance. The specimen was sent for IHC profiling and histologic evaluation (Fig. 2). Histopathologic examinations revealed a neoplasm consists of spindle cells with hyperchromatic nuclei and mild pleomorphism. Subsequent immunohistochemistry showed immunoreactivity for smooth muscle actin and desmin but the tumor cells were negative for S100, CD34, C-KIT and DOG-1. The Ki-67 index was 15–20%. KIT mutation study also showed no mutation in exon 9, 11, 13 and 17. Also, histologic evaluations of resected lymph nodes showed reactive sinus histiocytosis with no evidence of tumoral involvement.

2.1.2. Outcome and follow-up

The post-operative course was uneventful without any signs of short-term complications. The patient discharged four days after surgery. The patient was scheduled for Contrast-enhanced computed tomography (CT) of thoracic and abdomino-pelvic every 6 months, and colonoscopy every 12 months. Patient has no signs of local recurrence or distant metastasis Up-to-date. We will follow the patient for at least 5 years of close surveillance.

2.2. Case 2

On April 29th 2020, a 49-year-old man came with 3-month history of fatigue, melena, and unintentional weight loss. Family history was negative for any previous disease. General examination revealed

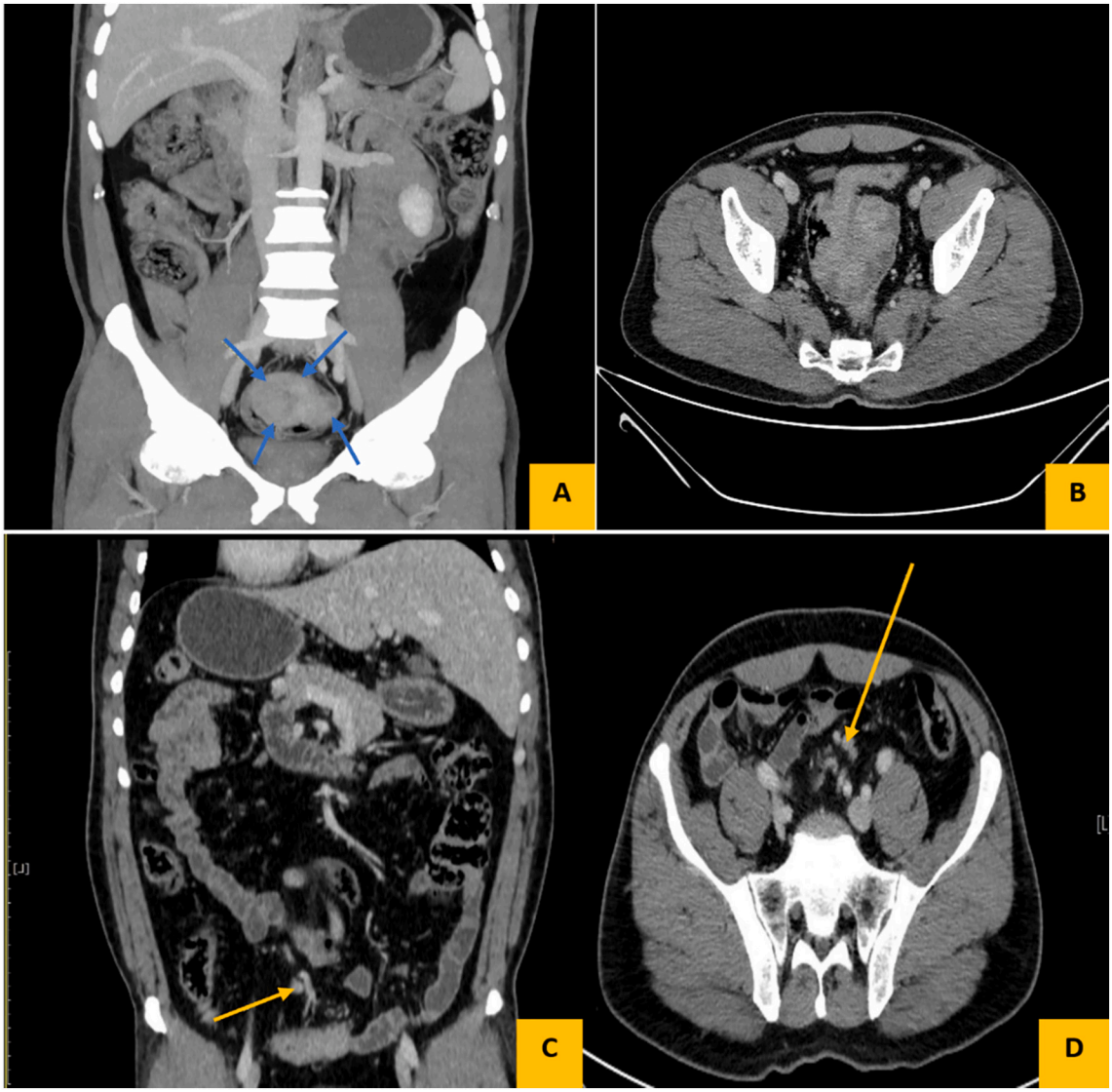

Fig. 1. The abdominal CT showed diffuse wall thickening of the sigmoid colon and distal part of the left colon (blue arrows in A), associated with enhancing soft tissue mass measuring about 72 mm × 40 mm in sigmoid colon that was suggestive of a malignant process. Evidence of multiple regional lymph nodes was seen adjacent to the sigmoid colon (yellow arrows).

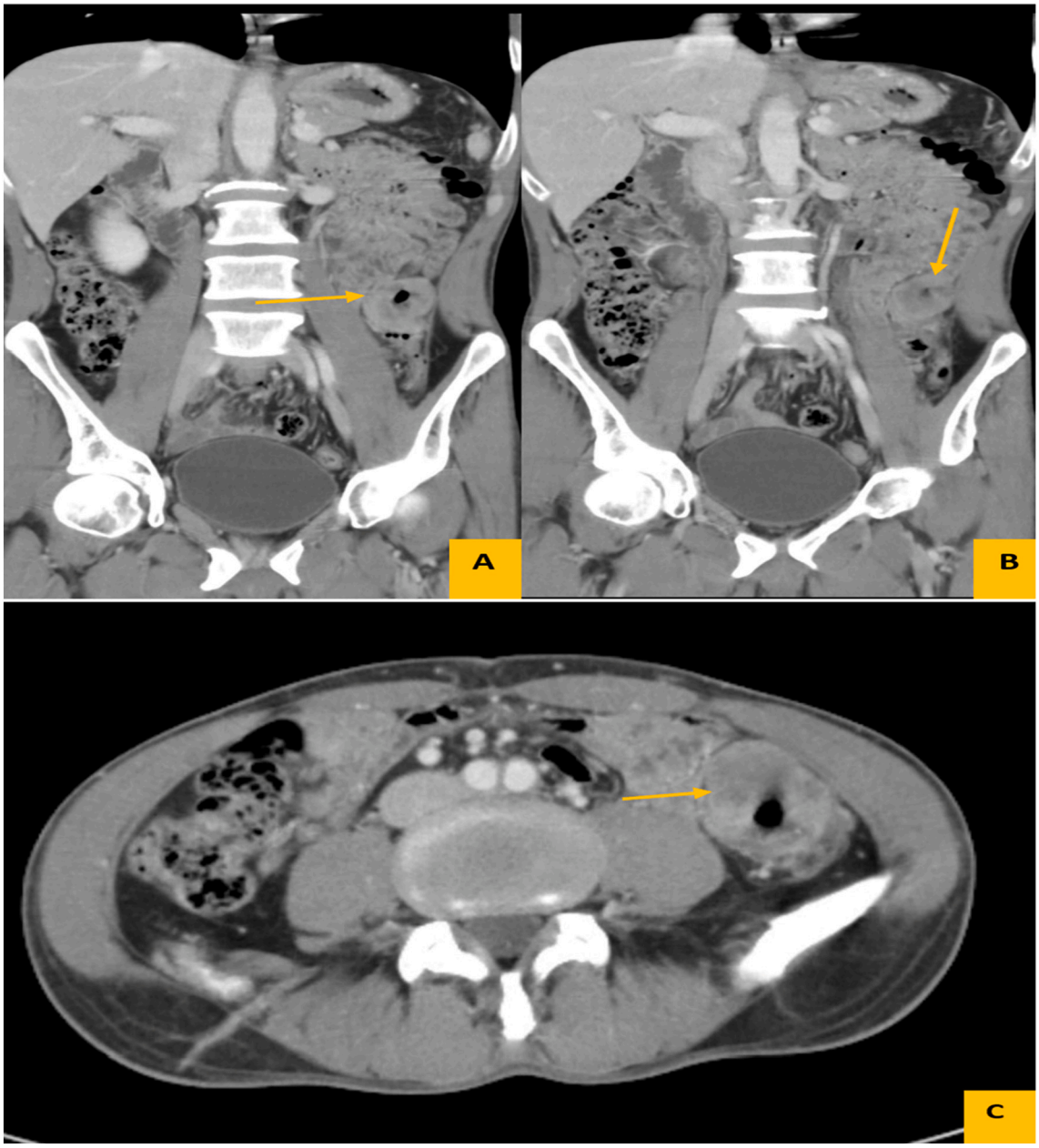

Fig. 2. Wall thickening of distal descending colon in Lt. lower quadrant with maximum wall thickness of 24 mm and length of involvement about 43 mm. Also, thickening of adjacent peritoneum was demonstrated.

temporal wasting and cachexia. Vital signs and abdominal examinations were unremarkable. Digital rectal examination showed grade-II internal hemorrhoids. Lab data was in favor of microcytic-hypochromic anemia (HB = 11.7). Carcinoembryonic antigen (CEA) test was normal. Colonoscopy revealed a large 3 * 4 * 3.5 cm circumferential mass with deep central ulceration in sigmoid colon 40 cm from anal verge.

Prior to surgery, Abdomino-perineal CT showed circumferential wall thickening of distal descending colon in Lt. lower quadrant with maximum wall thickness of 24 mm and length of involvement about 43 mm. Also, thickening of adjacent peritoneum without signs of lymph node metastasis were demonstrated (Fig. 2). Pre-op Chest CT was normal.

2.2.1. Treatment

Laparoscopic left hemicolectomy was conducted under general anesthesia with the purpose of complete resection of the tumor and involved lymph nodes. The specimen was extracted via a midline incision and extra-corporal anastomosis was performed. The specimen was sent for IHC profiling and histologic evaluation.

2.2.2. Outcome and follow-up

Surgery was successfully performed without any short-time complications and patient was discharged 4 days after the operation. Histologic evaluations were suggestive of spindle cells tumor with hyperchromatic nuclei and moderate pleomorphism (mitotic activity>5/50 HPF). IHC results were positive for desmin and SMA. Ki-67 was positive in 15–20% of neoplastic cells. C-KIT, CD34, DOG1, and S100 were negative. Unfortunately, histologic evaluations obtained from surgery revealed peritoneal and abdominal wall involvement by high grade sarcoma. He was referred to an oncologist for chemotherapy initiation with Adriamycin, and Ifosfamide. Also, the patient was scheduled for Contrast-enhanced computed tomography (CT) of Thoracic and Abdomino-

pelvis every 6 months, and colonoscopy every 12 months. Follow-up CT scan revealed multiple small nodules in both lungs with the largest being 4-mm in lateral segment of right lower lobe in favor of metastatic lesions. Abdominal CT showed multiple malignant-looking lesion in peritoneal cavity with the largest being 38 * 39 mm. Patient is alive and is under close follow up by an oncologist (Fig. 3).

3. Discussion

GISTs are the most common mesenchymal GI malignancies with the incidence of 1–3% of all GI malignancies. Moreover, LMS is an extremely rare cancer representing 3–6% of all GI mesenchymal tumors. They arise from muscularis mucosae or propria occurring mostly in middle-aged males [3,5]. During the pre-KIT area, most of the GI mesenchymal malignancies were wrongfully diagnosed as leiomyoma, LMS, or leiomyoblastomas. However, their incidence declined after the diagnosis of KIT mutations and the immunohistochemical differences between LMS and other GI mesenchymal tumors, particularly GISTs [6].

In 1998, Hirota et al. first introduced the presence of activating KIT-mutations in 94% of GISTs [7]. Kit gene, a tyrosine kinase receptor proto-oncogene, causes increased cellular proliferation. Subsequently, this mutation can lead to cellular atypia and neoplasia. Later studies confirmed that 95% of GISTs expressed CD34, CD137, and DOG1.1 [8]. On the contrary, the LMS is negative for kit-mutations and mostly positive for desmin, SMA, h-caldesmon, and vimentin [9].

Differentiation between LMS and GISTs is paramount importance since they have very similar clinical presentations but require radically different courses of treatment. The cellular origin of GISTs, the interstitial cells of Cajal, was first introduced in mid-1990s. However, LMS originates from muscle fibers of the muscularis mucosae and muscularis propria [10]. Also, most of the colonic LMS are polypoid, while esophageal LMS is mainly intramural [11].

The most common location of GI involvement by GISTS is the stomach (55%), small intestine (29%), colon (2.9%), and rectum (2.7%) [12]. However, GI LMS mainly involves stomach followed by small intestines, rarely colon and rectum [13]. True LMS of the colon is such a rare disorder that there isn't enough description of its nature. The classical colon LMS presents with a vast majority of non-specific symptoms including mild abdominal pain, fresh/obscure rectal bleeding, intra-abdominal hemorrhage, weight loss, changes in bowel habits, bowel obstruction, and tenesmus [14]. Diagnosis is based on colonoscopy and histologic evaluation and IHC profiling.

Based on our survey of the four main online databases (EMBASE, PUBMED, MEDLINE, and Scholar), there are only thirty-four previous cases of published colonic LMS after the pre-kit era that was confirmed by immunohistochemistry (Table 1). Sigmoid colon, followed by the ascending colon, is most commonly involved by primary LMS. The prognostic factors for the disease outcome have not been established properly, however patient age, tumor size/grade, and local/distant dissemination are of great importance [15]. Yamamoto et al., concluded that the only negative predictive factor for survival is tumor size of more than 5 cm [16]. However, based on previous case studies local and distal recurrence occurred even with favorable tumor features [14]. Compared with adults, infantile LMS has a better prognosis even with poor histologic features [17].

According to the recent survey by Faraj et al. lymph node involvement is very unlikely and distant metastasis is mostly by hematogenous spread [18]. Liver is the most common site of secondary tumor metastasis followed by lungs and peritoneum [18]. Due to the paucity of data, there is not enough evidence to establish reliable mortality estimates. However, based on a study by Aggarwal et al. in 2012 only 2 of 11 cases of colon LMS survived in 5-year surveillance [1]. The main cause of death was spreading of the primary tumor and multiple organ failure [1].

Medical therapy is the main course of treatment in mesenchymal GI malignancies, particularly GISTs. However, Due to absence of KIT-mutations in LMS, tyrosine kinase inhibitors (TKI) are not effective in tumor treatment. Therefore, Surgery is considered as the gold standard treatment for LMS [19]. Since, there are only a few numbers of true LMS cases reported, no standard therapeutic strategy has been established. Radical excision is the most reasonable option since even with low-grade tumors recurrences occurred [20]. According to YTNM Lee et al. all cases of smooth muscle sarcomas, should undergo wide excisional surgery with 4 cm tumor margin involving mesentery [21].

Although lymph node involvement is very uncommon in LMS, lymph node dissection is recommended due to its highly invasive nature [3,16]. Anthracyclines, first-line conventional chemotherapy regimen for soft tissue sarcomas, have minimal to no effect on LMS. A multi-drug regimen of Doxorubicin plus dacarbazine may have some clinical response in treatment. In conclusion, Adjuvant chemotherapy is unnecessary when tumors are completely resected [19]. However, neoadjuvant chemotherapy may decrease the risk of local recurrence in some cases of rectal sarcomas [22]. Furthermore, radiotherapy is completely unbeneficial since LMS is highly radio-resistant [23].

4. Conclusion

Herein, we described two rare cases of colon LMS which was confirmed with IHC. The tumors were surgically removed via laparoscopic approach, with wide, tumor-free margins. Due to the rarity of disease, there is not enough information about tumor characteristics. Overall, colonic LMS is a highly invasive neoplasm with poor oncologic outcome.

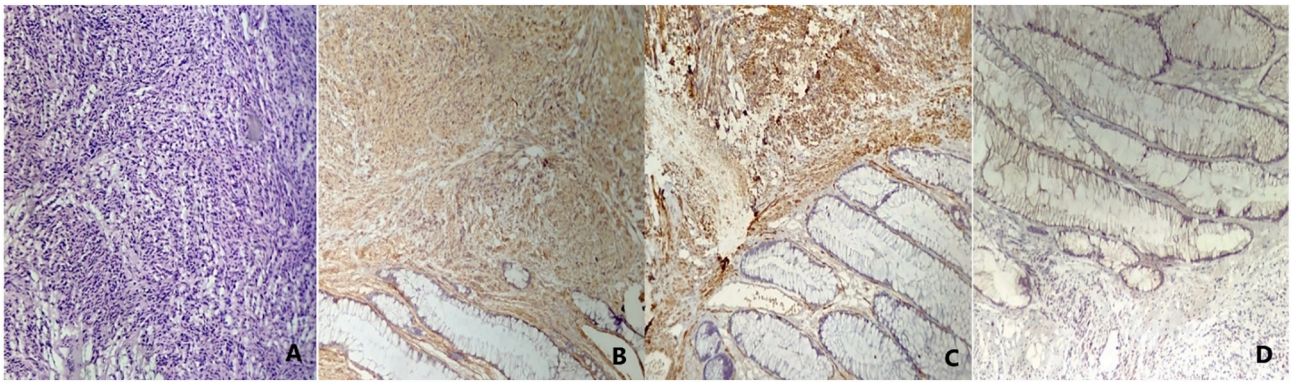

Fig. 3. Spindle cell neoplasm with nuclear hyperchromasia and mild pleomorphism, H&E, ×200 (a), Desmin immunoreactivity. Note the negative colonic crypts and positive tumoral cells for desmin, ×400 (b), Smooth muscle actin, Negative colonic crypts and positive tumoral cells for C-KIT, ×400 (d).

Table 1
All cases of published colonic LMS after the pre-kit era.

Case	Year	Age	Sex	site	Size (CM)	Local recurrence	Metastasis	Survival	c-kit	α-SMA	Desmin	CD-34
1[24]	2000	54	M	D	3.2	U	U	Dead	Neg	Pos+	Pos+	Neg
2[24]	2000	61	M	A	4.2	None	None	Alive	Neg	Pos+	U	Neg
3[24]	2000	75	M	A	6.5	U	U	Dead	Neg	Pos+	U	Neg
4[24]	2000	76	F	C	7.8	U	U	Dead	Neg	Pos+	U	Neg
5[24]	2000	36	F	S	6.5	None	Lung	Dead	Neg	Pos+	Pos+	Neg
6[24]	2000	66	M	A	U	None	Liver	Dead	Neg	Pos+	U	Neg
7[24]	2000	41	M	C	7.5	None	Humerus	Alive	Neg	Pos+	Pos+	Neg
8[25]	2004	65	M	D	10	None	Positive (U)	Dead	Neg	Pos+	Pos+	Neg
9[26]	2004	67	F	T	5.7	None	None	Alive	Neg	Pos+	U	Neg
10[11]	2007	77	F	S	U	Positive	None	U	Neg	Pos+	Pos+	Neg
11[11]	2007	52	M	S	U	None	Liver	U	Neg	Pos+	Pos+	Neg
12[27]	2009	74	F	A	6	None	Lung	Dead	Neg	Pos+	U	U
13[28]	2011	70	F	S	3.7	None	None	Dead	Neg	Pos+	Pos+	Neg
14[28]	2011	56	M	C	U	None	Liver	Alive	Neg	Pos+	Pos+	Neg
15[29]	2012	66	F	S	3	None	Liver	Dead	Neg	Pos+	Pos+	Neg
16[16]	2013	94	F	D	25	None	Liver	Dead	Neg	Pos+	U	Neg
17[16]	2013	56	M	S	1	None	LN	Alive	Neg	Pos+	U	Neg
18[16]	2013	78	F	S	8.5	None	Lung	Dead	Neg	Pos+	U	Neg
19[16]	2013	87	M	T	11	None	None	Dead	Neg	Pos+	U	Neg
20[30]	2013	65	M	S	U	None	None	Alive	Neg	Pos+	U	U
21[31]	2014	66	F	T	4	None	None	Alive	Neg	Pos+	U	Neg
22[30]	2014	65	M	S	U	None	None	Alive	Neg	Pos+	U	U
23[6]	2015	46	M	T	11.8	Positive	None	Alive	Neg	Pos+	U	Neg
24[32]	2015	89	F	A	4.5	None	Liver	U	Neg	Pos+	U	U
25[5]	2015	54	M	A	13	Positive	None	Alive	Neg	Pos+	Pos+	Neg
26[33]	2015	59	M	A	10	None	None	Alive	Neg	Pos+	Pos+	Neg
27[34]	2016	89	M	C	2.2	None	None	Alive	Neg	Pos+	Pos+	U
28[35]	2016	51	F	D	4	None	None	Alive	Neg	Pos+	Pos+	Neg
29[36]	2016	44	M	SF	8.5	None	None	Alive	Neg	Pos+	Pos+	Neg
30[9]	2017	55	F	A	8	None	None	Alive	Neg	Pos+	Pos+	Neg
31[14]	2018	57	F	S	U	U	U	Alive	Neg	Pos+	U	Neg
32[14]	2018	88	M	A	6.5	None	Liver	Dead	Neg	Pos+	U	U
33[37]	2019	53	M	S	3.5	None	None	Alive	Neg	Pos+	Pos+	Neg
34[3]	2019	46	M	S	4.2	None	None	Alive	Neg	Pos+	Pos+	Neg
35(case1)	2019	48	M	S	7.2	None	None	Alive	Neg	Pos+	Pos+	Neg
36(case2)	2020	49	M	S	4	None	Peritoneum Lungs	Alive	Neg	Pos+	Pos+	Neg

Ethics approval and consent to participate

The purpose of this research was completely explained to the patient, and was assured that their information will be kept confidential by the researchers. The present study was approved by the medical ethics committee of the academy.

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CRediT authorship contribution statement

AB – drafted the manuscript and provided images. MS- helped with the draft and reviewed the literature. MM- provided histologic evaluations. RS- Supervisor, provided initial feedback and reviewed the final manuscript (corresponding author). The authors read and approved the final manuscript.

Guarantor

Ramin Shekouhi M.D

Consent for publication

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Availability of data and materials

All data generated or analyzed during this study are included in this published article. A preprint copy of the article is available in research square (https://doi.org/10.21203/rs.3.rs-214702/v1).

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Declaration of competing interest

The authors declare that they have no competing interests.

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