Adult Spermatic Cord Inflammatory Myofibroblastic Tumor: A Case-Based Narrative Review of The Literature

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ABSTRACT

Purpose: Inflammatory myofibroblastic tumors are exceptionally rare mesenchymal neoplasms that often mimic malignancies clinically and radiologically, frequently leading to radical orchiectomy because of diagnostic uncertainty. The existing literature is limited to isolated reports with inconsistent follow-up and little discussion of conservative surgical approaches. ***Materials and methods:*** We present a case of a 40-year-old man diagnosed with an anaplastic lymphoma kinase-negative inflammatory myofibroblastic tumor arising from the spermatic cord, successfully treated with testis-sparing excision. In addition, a focused literature review was performed using PubMed, Scopus, and Web of Science to identify previously reported adult cases of spermatic cord inflammatory myofibroblastic tumor. Clinicopathological features, immunohistochemical findings, treatment approaches, and outcomes were analyzed. ***Results:*** Including the present case, 12 adult patients with inflammatory myofibroblastic tumors of the spermatic cord were identified. The median age was 42.5 years (interquartile range, 32.5–54.0). Anaplastic lymphoma kinase status was reported, with negativity observed in 7 cases (58%) and positivity in 1 case (8%), while status was not assessed in the remaining 4 patients (33%). Radical orchiectomy was performed in 9 patients (75%), whereas testis-sparing mass excision was performed in 3 cases (25%). No local recurrence or distant metastasis was documented during a median follow-up of 36 months (interquartile range, 30–46) among the 11 cases with available follow-up data. ***Conclusion:*** Adult spermatic cord inflammatory myofibroblastic tumors appear to follow an

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indolent clinical course after complete excision. Organ-preserving management may be considered in carefully selected patients, highlighting the importance of accurate pathological assessment and multidisciplinary decision-making.

INTRODUCTION

Paratesticular masses represent a heterogeneous group of lesions arising from extratesticular scrotal structures, including the tunica vaginalis, epididymis, and spermatic cord.[1] Malignancies in this region are predominantly sarcomas, with rhabdomyosarcoma accounting for approximately 24% of adult cases.[2] Benign lesions are more diverse, with lipomas, adenomatoid tumors, and inflammatory myofibroblastic tumors (IMTs) being among the most common.[2]

IMT is a rare mesenchymal neoplasm characterized by spindle-shaped myofibroblasts and a dense inflammatory infiltrate rich in plasma cells and lymphocytes, typically demonstrating immunohistochemical positivity for vimentin and smooth muscle actin (SMA), whereas S100 protein and cytokeratin are usually negative.[3-5] Initially described as an inflammatory pseudotumor, IMT has been reclassified by the World Health Organization (WHO) as a neoplasm of intermediate malignant potential because of its capacity for local recurrence and, in rare cases, metastasis.[4] Although the lung is the most common site, extrapulmonary IMTs are increasingly reported, with genitourinary involvement accounting for approximately 9.5% of cases.[6]

Spermatic cord IMTs typically present as painless scrotal masses and often mimic malignancies on imaging and physical examination.[7] This diagnostic ambiguity frequently leads to radical orchiectomy, despite the generally benign nature of these tumors. The etiology remains poorly understood but has been associated with trauma, infection, or immune dysregulation.[8] Anaplastic lymphoma kinase (ALK) gene rearrangements, more common in pediatric IMTs, are rarely seen in adults.[9]

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In adults, spermatic cord IMTs are exceptionally rare, and published reports are largely limited to single cases. Long-term follow-up and organ-sparing surgical approaches are rarely described.[10] Complete surgical excision with negative margins remains the cornerstone of management and the key prognostic factor.[11]

Herein, we report a rare case of spermatic cord IMT in a 40-year-old man treated with testis-sparing surgery. We also present a comparative analysis of 11 previously reported adult cases to better characterize this tumor and to assess the potential for conservative management in select patients.

MATERIALS AND METHODS

A focused literature review was conducted using the PubMed, Scopus, and Web of Science databases to identify previously reported adult cases of inflammatory myofibroblastic tumor arising from the spermatic cord. The search was performed using combinations of the terms "inflammatory myofibroblastic tumor," "paratesticular," and "spermatic cord." Only studies reporting adult patients with tumors originating from the spermatic cord were included. Cases arising from other paratesticular structures (such as the epididymis or tunica vaginalis), pediatric cases, and articles lacking sufficient clinicopathological data were excluded. No language restrictions were applied. Duplicate records retrieved from different databases were identified and removed before analysis. Data regarding patient demographics, tumor characteristics, immunohistochemical findings (including ALK status), treatment modalities, and outcomes were extracted and summarized descriptively. Descriptive statistical analysis was performed. Continuous variables were summarized as mean ± standard deviation or as median with interquartile range (IQR) and range, as appropriate. Categorical variables were expressed as frequencies and percentages. Cases with missing data for specific variables were excluded from the corresponding analyses, and no data imputation was performed.

CASE PRESENTATION

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A 40-year-old man with no known comorbidities presented with a 3-month history of painless swelling in the left hemiscrotum, accompanied by mild discomfort. He denied constitutional symptoms such as fever, weight loss, anorexia, hematuria, or dysuria. His medical history included blunt trauma to the scrotum and perineal region from a motor vehicle accident 10 years earlier. There was no history of tuberculosis, recurrent urinary tract infections, or sexually transmitted infections. Family history was notable for lung cancer.

On physical examination, a firm, mildly tender, nodular mass was palpated in the upper pole of the left scrotum. The lesion was distinct from the testicular parenchyma. No inguinal lymphadenopathy was appreciated, and the remainder of the genital and abdominal examinations were unremarkable. Serum tumor markers, including alpha-fetoprotein (AFP), beta-human chorionic gonadotropin (β-hCG), and lactate dehydrogenase (LDH), were within normal limits. HIV testing was negative, and routine laboratory and biochemical investigations were unremarkable.

Scrotal Doppler ultrasonography demonstrated a well-defined, hypoechoic, moderately vascularized solid lesion measuring 30 × 20 mm in the upper left hemiscrotum, clearly separated from the testis and epididymis. Bilateral testes were normal in size and echotexture. No hydrocele or inguinal lymphadenopathy was noted.

The patient underwent left inguinal surgical exploration. Intraoperatively, the mass was identified as arising from the spermatic cord and was adjacent to, but not invading, the testis or epididymis. Given the well-circumscribed and clearly extratesticular appearance on preoperative imaging and the intraoperative finding that the mass was readily separable from the testis and epididymis without evidence of local invasion, frozen section analysis was not considered necessary, and testis-sparing excision was performed. Complete excision of the lesion was achieved with clear surgical margins, and the testis was preserved.

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Gross examination revealed a firm, encapsulated, yellow-gray mass measuring 35 × 20 × 17 mm. Histopathological analysis showed a well-circumscribed fibroinflammatory nodular proliferation composed of spindle-shaped myofibroblasts within a dense inflammatory background rich in plasma cells and lymphocytes, with focal central necrosis **(Figure 1)**. Higher magnification showed myofibroblastic cells with vesicular nuclei, variable cytoplasm, and mixed inflammatory infiltrates **(Figure 2)**. No significant atypia or increased mitotic activity was observed.

Immunohistochemistry (IHC) demonstrated strong positivity for vimentin and SMA, with focal weak staining for actin **(Figure 3)**. The tumor cells were negative for ALK, CD34, S100, desmin, cytokeratin, caldesmon, and CD21 **(Figure 4)**. ALK negativity was further confirmed by fluorescence in situ hybridization (FISH) analysis. Next-generation sequencing (NGS) was attempted; however, molecular analysis could not be completed because of insufficient DNA and RNA material. The IgG4/IgG ratio was low, and serum IgG4 levels were within the normal range, effectively excluding IgG4-related sclerosing disease. Based on histopathological and immunophenotypic features, a diagnosis of ALK-negative IMT of the spermatic cord was confirmed.

Postoperative staging with computed tomography (CT) of the chest, abdomen, and pelvis showed no evidence of metastasis. The patient was managed without adjuvant therapy. At 30 months of follow-up, he remained disease-free, with no signs of local recurrence or distant spread.

DISCUSSION

Spermatic cord inflammatory myofibroblastic tumors remain exceptionally rare entities within the spectrum of genitourinary neoplasms. Although their etiology is not fully understood, several possible triggers have been suggested in the literature, including prior trauma, chronic inflammation, and infections such as Epstein-Barr virus, human herpesvirus

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8 (HHV-8), and *Mycobacterium avium-intracellulare*.[6] In our case, a history of scrotal and perineal trauma was noted, which may have contributed to tumor development. Although IMTs can occur at any age, they are more frequently seen in children and young adults.[10] In our series of 12 adult patients, including the present case, the age range was broad (17–82 years), with a median age of 42.5 years (IQR, 32.5–54.0). The index case aligns with this median, reinforcing the tendency of adult-onset IMTs to present in early to middle adulthood. Clinically, spermatic cord IMTs typically manifest as painless scrotal masses, although pain or mild discomfort was reported in 2 of the 12 reviewed cases, including the present case. This symptom variability may reflect differences in tumor size, location, or the degree of local inflammation. The average tumor diameter in our cohort was 6.7 ± 3.0 cm, and the present case (3.5 × 2.0 × 1.7 cm) falls below the 6.5-cm threshold previously associated with increased recurrence risk.[11]

Diagnostically, spermatic cord IMTs present a challenge because of their nonspecific clinical and radiologic features, often mimicking both benign and malignant pathologies. Ultrasonography is the preferred initial imaging modality to determine the lesion's solid or cystic nature and its intra- or extratesticular origin.[12] The sonographic appearance of IMTs, initially described by Kapur et al., typically includes heterogeneous echotexture, lobulated contours, and hypervascularity.[10] In our case, ultrasonography demonstrated a well-defined, hypoechoic, moderately vascularized, solid extratesticular lesion clearly separated from the testis and epididymis. In contrast, Megremis et al. described a complex paratesticular mass that compressed and deformed the ipsilateral testis, illustrating the variable sonographic appearance of paratesticular IMTs.[13] However, because imaging findings are not diagnostic, confirmation depends on histopathological and immunohistochemical evaluation of excised or biopsied tissue.[14] Macroscopically, spermatic cord IMTs generally present as well-circumscribed, gray-white, solid nodular lesions, occasionally exhibiting areas of cystic

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degeneration or hemorrhage. Histologically, they are composed of spindle-shaped myofibroblasts within a background of dense lymphoplasmacytic infiltration. Immunohistochemically, these tumors most frequently express vimentin and SMA, whereas S100 and cytokeratin are typically negative.[8,12] In our series, all cases with available IHC data (100%) were positive for vimentin or SMA. S100 and cytokeratin were negative in all cases in which these markers were assessed **(Table 1)**. The index case exhibited an immunophenotype consistent with these findings, aligning with the typical profiles described in previous reports. Of note, necrosis—present in our case—has been suggested as a potential adverse prognostic feature in IMT,[7] warranting close postoperative monitoring despite otherwise benign characteristics.

ALK gene rearrangements, which activate oncogenic pathways such as JAK-STAT and PI3K-Akt, are present in about half of IMTs, predominantly in pediatric patients, and are associated with aggressive behavior.[15] Among the 12 adult paratesticular IMT cases in our study, ALK status was available in 8. Only 1 patient, reported by Rico et al., was ALK-positive, whereas the remaining 7 cases, including our own, were ALK-negative.[16] In the present case, ALK negativity was confirmed by both immunohistochemistry and FISH, supporting the classification as ALK-negative IMT. This low rate of ALK expression is consistent with adult-onset IMTs and may also reflect historical limitations in testing before the widespread adoption of ALK immunohistochemistry. Although ALK immunohistochemistry and molecular testing can support diagnosis and guide targeted therapy in selected cases, current guidelines do not mandate routine molecular testing for all adult inflammatory myofibroblastic tumors, particularly in resectable localized disease.

Because of its nonspecific presentation, the differential diagnosis of spermatic cord IMT includes several benign and malignant lesions. Malignant mimics include rhabdomyosarcoma, liposarcoma, sarcomatoid carcinoma, and inflammatory fibrosarcoma,

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whereas benign conditions such as spermatocele or tuberculous epididymitis may appear similar.[17] IgG4-related disease should also be considered because of overlapping histological features, including IgG4-positive plasma cell infiltration and storiform fibrosis.[18] In our case, the absence of these features and normal serum IgG4 levels effectively ruled out IgG4-related disease.

The cornerstone of treatment for spermatic cord IMT is complete surgical excision with negative margins.[19] Testis-sparing surgery is preferred when feasible to preserve organ function, whereas radical orchiectomy is reserved for large, invasive, or indeterminate lesions.[17] In such scenarios, preoperative counseling should include discussion of the potential need for orchiectomy. Although intraoperative frozen section analysis has been advocated as a useful adjunct to avoid unnecessary orchiectomy in benign paratesticular lesions, its application in adult spermatic cord inflammatory myofibroblastic tumors has been inconsistently reported in the literature.[12] In the present case, frozen section was not performed; instead, the decision to proceed with testis-sparing excision was based on preoperative imaging findings and intraoperative assessment demonstrating the absence of local invasion.

In several published paratesticular cases, radical orchiectomy was performed because of strong suspicion of malignancy or intraoperative uncertainty, as in those described by Chakrabarti and Shetty and by Yee et al.[20,21] Overall, 25% of patients underwent testis-sparing surgery and 75% underwent radical orchiectomy **(see Table 1)**. This preference for radical surgery appears to be largely driven by diagnostic uncertainty and the rarity of IMTs, which often precludes confident preoperative differentiation from malignant paratesticular tumors. Conversely, in a limited number of carefully selected patients, including the present case, testis-sparing surgery could be performed safely when the lesion was clearly separable from the testis, preoperative imaging suggested benign behavior, and no evidence of local

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invasion was observed intraoperatively. These findings support the feasibility of organ-preserving surgery in selected adult patients with spermatic cord IMT, emphasizing the importance of meticulous preoperative assessment and careful intraoperative decision-making.[4]

Although distant metastases have been observed more frequently in ALK-negative tumors, local recurrence can occur irrespective of ALK status.[6] Therefore, long-term surveillance with periodic clinical and imaging evaluations is essential after surgery.[14] Among the 11 cases with available follow-up data, the median follow-up duration was 36 months (IQR, 30–46; range, 6–48 months). No local recurrence or distant metastasis was observed in the ALK-negative cases, including the present one, and the single ALK-positive case also showed no evidence of recurrence during follow-up. This may suggest that paratesticular IMTs, particularly ALK-negative ones, follow a more indolent clinical course than IMTs at other anatomical sites.

Although IMTs generally display benign histological features, their biological behavior is not fully understood. At present, there is no high-level evidence supporting the routine use of adjuvant chemotherapy or radiotherapy. However, such therapies may be considered in select cases with incomplete resection, histological features of malignant transformation, or recurrent disease.[22]

This case is novel because it represents one of the very few reported adult spermatic cord inflammatory myofibroblastic tumors successfully managed with testis-sparing surgery without the use of intraoperative frozen section analysis, supported by comprehensive immunohistochemical and molecular evaluation, including ALK FISH confirmation, and long-term follow-up.

This study provides a narrative comparative analysis of reported adult spermatic cord inflammatory myofibroblastic tumors. Several limitations should be acknowledged, including

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the retrospective nature of the literature review, heterogeneity in follow-up duration, and incomplete immunohistochemical data in some cases. Nevertheless, the absence of local recurrence or distant metastasis among cases with available follow-up data, with a median follow-up of 36 months (IQR, 30–46), supports complete surgical excision as the cornerstone of durable disease control in adult spermatic cord IMTs.

CONCLUSIONS

Spermatic cord inflammatory myofibroblastic tumors are rare lesions with variable biological behavior that may exhibit clinical and radiological features overlapping with those of malignant neoplasms. Comparative evaluation of the present case and reported adult cases suggests that testis-sparing surgery can be a safe and effective option in carefully selected patients, particularly in ALK-negative tumors. Careful preoperative imaging, meticulous intraoperative assessment, and multidisciplinary collaboration are essential to guide organ-preserving decision-making, with intraoperative frozen section considered when diagnostic uncertainty persists. Complete excision with negative margins remains the cornerstone of management, and long-term clinical and imaging surveillance is recommended due to the potential risk of local recurrence.

SUMMARY

Inflammatory myofibroblastic tumors of the spermatic cord (the structure that suspends the testicle) are extremely rare. In 12 adult cases reviewed, removing the tumor while keeping the testicle appeared safe, with good long-term outcomes.

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CONFLICT OF INTEREST

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The authors declare that there are no conflicts of interest regarding the publication of this article.

DECLARATIONS

Ethical Approval

This case report did not require institutional ethical approval in accordance with applicable regulations.

Informed Consent

Written informed consent was obtained from the patient for publication of this case report, including the use of clinical and histopathological images. All identifying information has been removed to ensure confidentiality.

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Author Contributions

İ.B. (lead author): Responsible for case conceptualization, clinical evaluation, study design, data acquisition, literature review, and drafting of the initial manuscript.

İ.G.: Contributed to the revision of the manuscript, ensuring scientific accuracy and intellectual content.

E.M.D.: Performed the histopathological evaluation, contributed to image selection, and assisted in figure preparation.

D.D.: Provided English language editing and contributed to the linguistic refinement of the manuscript.

M.B.H.: Evaluated the study comprehensively, contributed to overall scientific validity, and served as supervisor.

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Table 1. Clinicopathological features, immunohistochemical profile, treatment approaches, and outcomes of reported adult spermatic cord inflammatory myofibroblastic tumor cases.

Reference	Age (years)	Presentation	Size (cm)	Positive IHC markers	Negative IHC markers	Treatment	Follow-up (months)/outcome
Megremis et al. (2007)[13]	45	Left scrotal mass, gradually enlarging, painless	2 × 2.5 × 4	SMA, Desmin	CD34, S100, CK (AE1/AE3), ALK, Muscle-specific actin, Inhibin	Radical orchiectomy	36 months, no recurrence
Yee et al. (2009)[21]	40	Left lower quadrant mass with undescended testis	10 × 8 × 7	SMA	Desmin, ALK, CD34, CD21, CK, S100, EBV	En bloc excision with undescended testis	30 months, no recurrence
Chakrabarti and Shetty (2010)[20]	64	Painless right upper scrotal mass for 6 months, separate from testis	8 × 5	Vimentin, SMA, Desmin	Myoglobin, S100 (ALK not assessed)	Radical orchiectomy	36 months, no recurrence
Tunuguntla et al. (2011)[17]	17	Right scrotal mass for 2 months	6 × 2.2 × 1.8	SMA, Vimentin, Desmin	Myoglobin, S100, ALK	Orchiectomy plus mass resection	48 months, no recurrence
Rafeek et al. (2012)[23]	22	Painless left scrotal mass for 7 months	4.5 × 3.5 × 2	Vimentin, CD34, EMA	ALK, Desmin, SMA, S100, CK	Testis-sparing mass excision	8 months, no recurrence
Rico et al. (2017)[16]	46	Left inguinoscrotal mass with mild discomfort	Not stated	ALK, Vimentin, SMA	Desmin, PAX-8, CK	Radical orchiectomy	48 months, no recurrence
Aydemir et al. (2020), case 1[24]	82	Left inguinal mass for 1 year, painless,	13 × 8	Actin, Desmin, Vimentin, CD68	CD30, CD15, S100, CK (AE1/AE3), ALK	Radical orchiectomy	46 months, no recurrence

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Reference	Age (years)	Presentation	Size (cm)	Positive IHC markers	Negative IHC markers	Treatment	Follow-up (months)/outcome
		separate from testis					
Aydemir et al. (2020), case 2[24]	56	Right scrotal mass for 4 years, painless, separate from testis	3.5 × 3 × 2.7	Vimentin, Actin, CD34	Desmin, CK (AE1/AE3), ALK	Radical orchiectomy	36 months, no recurrence
Javid et al. (2023)[19]	32	Painless left scrotal mass for 2 years	7.5 × 7 × 3.5	Not reported	(ALK not assessed)	Radical orchiectomy	6 months, no recurrence
Purnomo et al. (2023)[14]	33	Painless right scrotal mass for 2 years	8 × 8 × 7	CD68, S100, Vimentin	Not reported (ALK not assessed)	Testis-sparing mass excision	36 months, no recurrence
Shamseldin et al. (2025)[12]	52	Left scrotal mass	6 × 5 × 4	Vimentin, SMA, Actin	S100, CK, Desmin (ALK not assessed)	Radical orchiectomy	Not specified
Our case	40	Left scrotal mass with mild discomfort for 3 months; history of scrotal trauma	3.5 × 2 × 1.7	Vimentin, SMA, Actin	ALK, CD34, S100, Desmin, CK, Caldesmon, CD21	Testis-sparing mass excision	30 months, no recurrence

Abbreviations: ALK, anaplastic lymphoma kinase; CK, cytokeratin; EBV, Epstein-Barr virus; EMA, epithelial membrane antigen; IHC, immunohistochemistry; SMA, smooth muscle actin.

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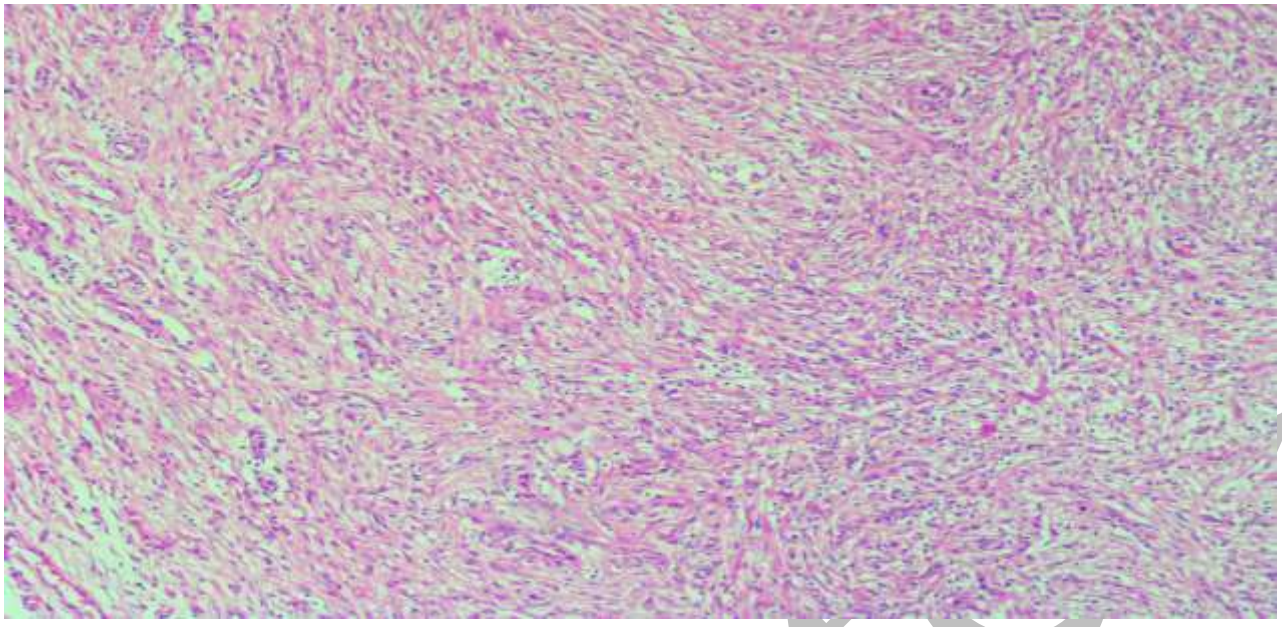


Figure 1. Spindle cell tumor with a fasciitis-like growth pattern (hematoxylin and eosin stain, original magnification ×100).

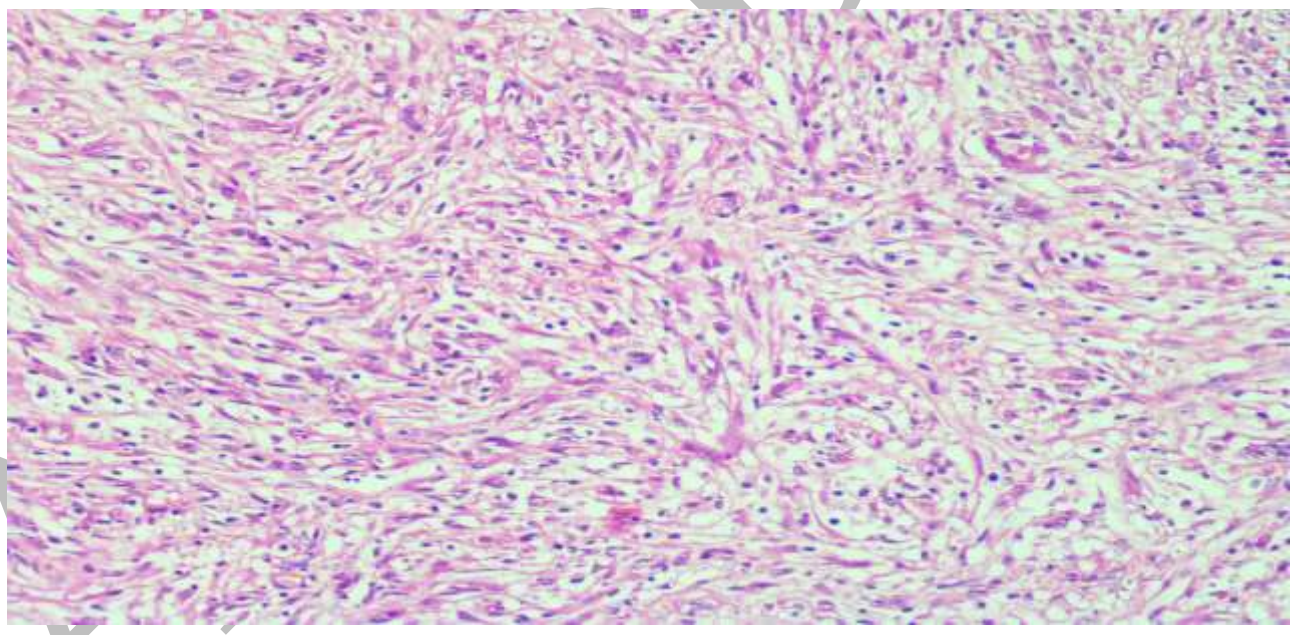


Figure 2. Myofibroblastic cells with vesicular nuclei and variable cytoplasm, with a mixed inflammatory infiltrate (plasma cells, lymphocytes, eosinophils, and neutrophils) (hematoxylin and eosin stain, original magnification ×200).

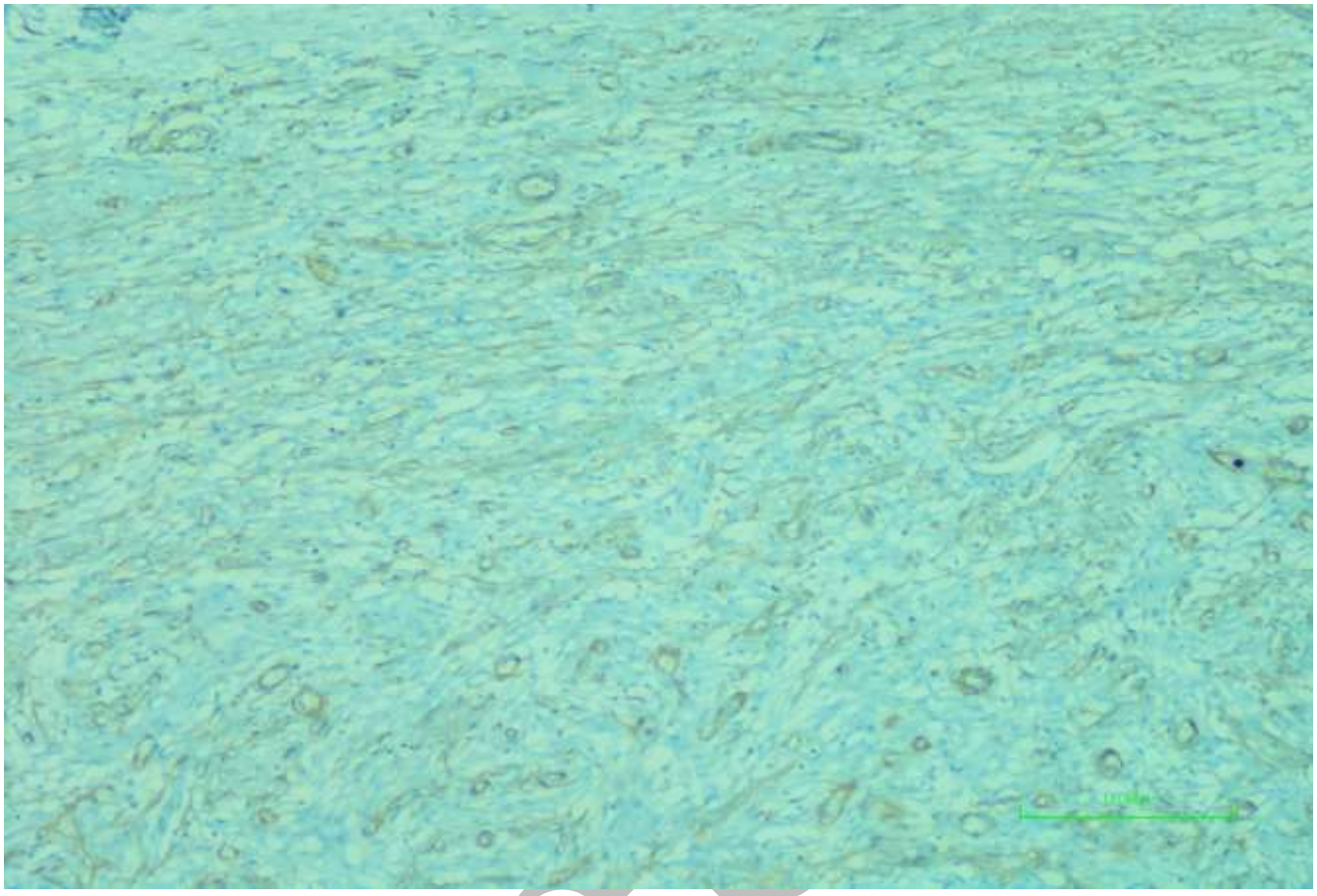


Figure 3. SMA highlights cytoplasmic "tram-track" staining in tumor cells (immunoperoxidase, original magnification ×100).

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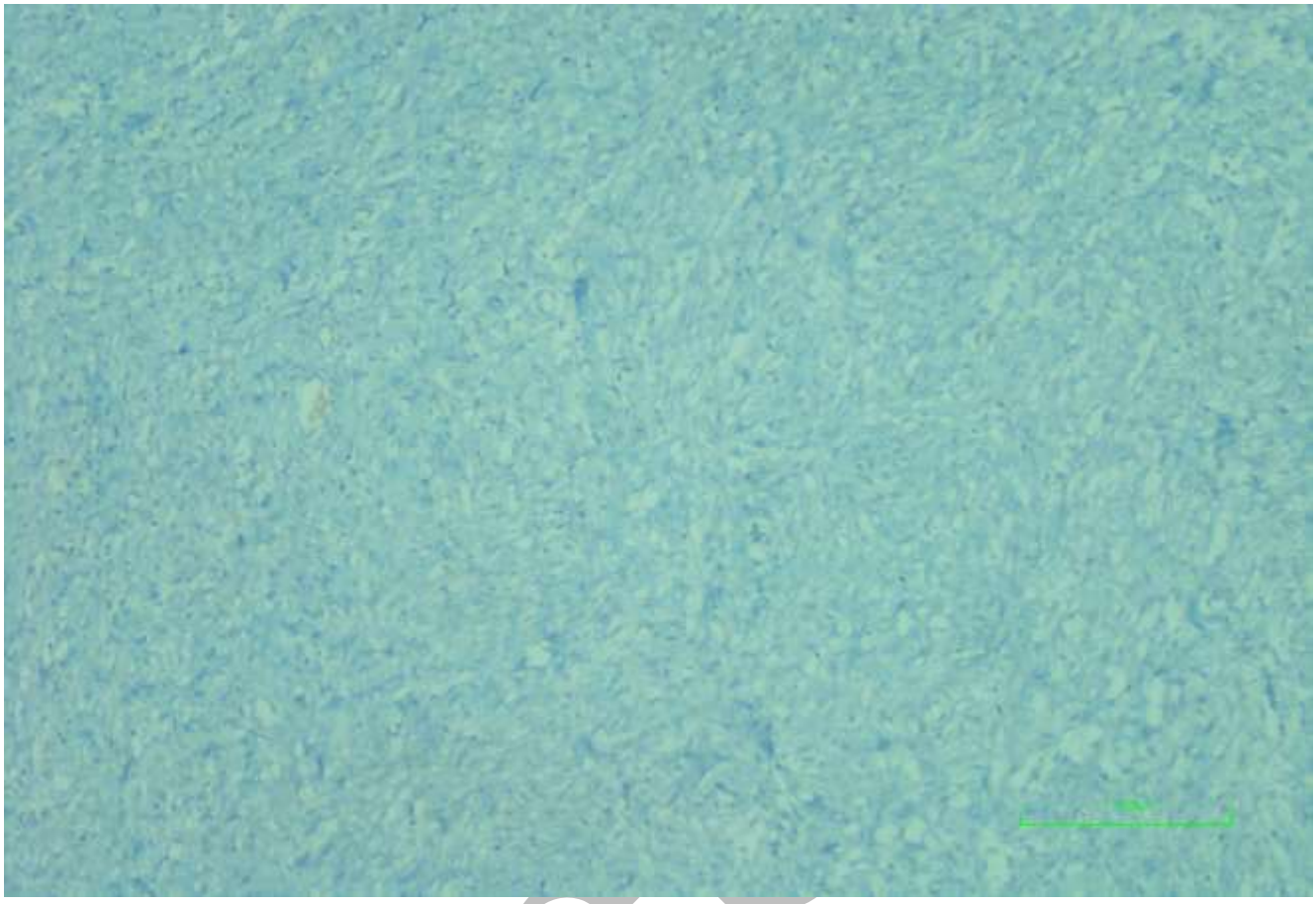


Figure 4. No staining was observed with ALK (immunoperoxidase, original magnification ×100).

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