



Original contribution

## Pseudosarcomatous and sarcomatous proliferations of the bladder

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**Summary** Pseudosarcomatous fibromyxoid tumor (PFT), postoperative spindle cell nodule (PSN), sarcoma, and sarcomatoid carcinoma of the bladder are frequently difficult to distinguish histopathologically with significant differences in disease-related outcomes. A retrospective review of our pathology registry over the last 25 years identified 7 PFT, 10 PSN, 18 primary bladder sarcomas, and 17 sarcomatoid carcinomas. Most patients with PFT, PSN, sarcoma, and sarcomatoid carcinoma were diagnosed between the ages of 50 to 60 years with PFT and PSN most commonly detected in women. A previous history of urological instrumentation and bladder cancer was present in all patients with PSN but none of the patients with PFT. Pseudosarcomatous fibromyxoid tumors were characterized by a tissue culture-like proliferation of myofibroblastic cells with focal atypia and overall cytoarchitectural features mimicking nodular fasciitis. Sarcomas and sarcomatoid carcinomas exhibited cellular atypia, mitotic activity with atypical mitosis, and the presence of necrosis. Transurethral resection was sufficient to control all PFT and PSN with no evidence of distant metastatic spread. In contrast, local recurrences and distant metastases frequently occurred in patients with primary sarcoma and sarcomatoid carcinoma despite aggressive surgical management, which was often combined with neoadjuvant chemotherapy (50% and 65% disease-specific mortality, respectively). Pseudosarcomatous fibromyxoid tumor and PSN have unique clinical and pathologic features that allow their distinction from primary bladder sarcoma and sarcomatoid carcinoma.

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### 1. Introduction

Pseudosarcomatous spindle cell proliferations of the bladder comprise 2 groups of lesions with overlapping microscopic features, referred to as pseudosarcomatous fibromyxoid tumor (PFT) and postoperative spindle cell

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nodule (PSN). Both of these lesions represent florid proliferations of spindle cells that may be confused with sarcomas. These lesions have overlapping microscopic features but are believed to represent pathogenetically distinct lesions.

Pseudosarcomatous fibromyxoid tumor of the urinary bladder is a rare reactive proliferation of myofibroblasts [1]. This lesion was first described by Roth [2] as a pseudosarcomatous myofibroblastic tumor, and numerous designations have since been used to describe it, including inflammatory pseudotumor [3,4], nodular fasciitis [5], and pseudosarcomatous myofibroblastic proliferation [6]. Postoperative spindle cell nodule has a very similar gross and histologic appearance consisting of a reactive proliferation of spindle cells, which usually occurs several months following a lower genitourinary surgical procedure such as a transurethral resection (TUR) or a biopsy [7-10]. Despite these previous studies, it remains unclear whether these lesions are reactive or neoplastic in origin [11-13]. Both PFT and PSN of the urinary bladder portend an excellent prognosis, with some of these tumors exhibiting spontaneous regression [11,14-16]. However, these benign lesions are frequently composed of atypical spindle cells, making their potential misdiagnosis as a bladder sarcoma or sarcomatoid carcinoma an important concern for the pathologist and treating physician [6,16]. We previously described 2 cases of PFT that were initially diagnosed as bladder rhabdomyosarcoma [17].

The purpose of the present study was to define the clinical and pathologic features that distinguish these lesions to aid pathologists and treating physicians in establishing the correct diagnosis and provide appropriate management.

## 2. Materials and methods

### 2.1. Pathologic specimens

We searched our tumor registry over the last 25 years (January 1980 to January 2005) and identified 7 patients with PFT, 10 patients with PSN, 18 patients with primary sarcoma, and 17 patients with sarcomatoid carcinoma of the urinary bladder. The pathologic slides and pathology reports were reviewed for all cases. Cases of PFT and PSN treated at our institution and previously reported [15,17] were not included in this report. Pseudosarcomatous proliferations, primary bladder sarcomas, and sarcomatoid carcinomas were classified and graded, when appropriate, according to the World Health Organization Classification and Grading Systems (1999) [18]. The associated urothelial carcinomas were classified according to the World Health Organization /International Society of Urological Pathology Consensus Grading Scheme [19]. Of those patients with primary sarcoma, the sarcoma subtypes included leiomyosarcoma (n = 11), unclassified sarcoma (n = 4), and angiosarcoma (n = 3).

### 2.2. Clinical data

We reviewed patient medical records to determine their clinical presentation, treatment, and disease-related outcomes. The follow-up information for PFT and PSN was available for all cases, with median follow-ups of 3 years (8 months to 13.1 years) and 3.1 years (2 months to 5.6 years), respectively. The median lengths of follow-up for primary bladder sarcoma and sarcomatoid carcinoma were 2.6 years (2 months to 11.9 years) and 1.4 years (2 months to 5.6 years), respectively. These data were used to calculate disease-specific mortalities defined as the number of years between the date of diagnosis and the date of death from disease. All pathologic specimens and clinical data were analyzed under institutional review board-approved protocols. All patient identifiers were removed so that patient confidentiality could be maintained throughout the study.

## 3. Results

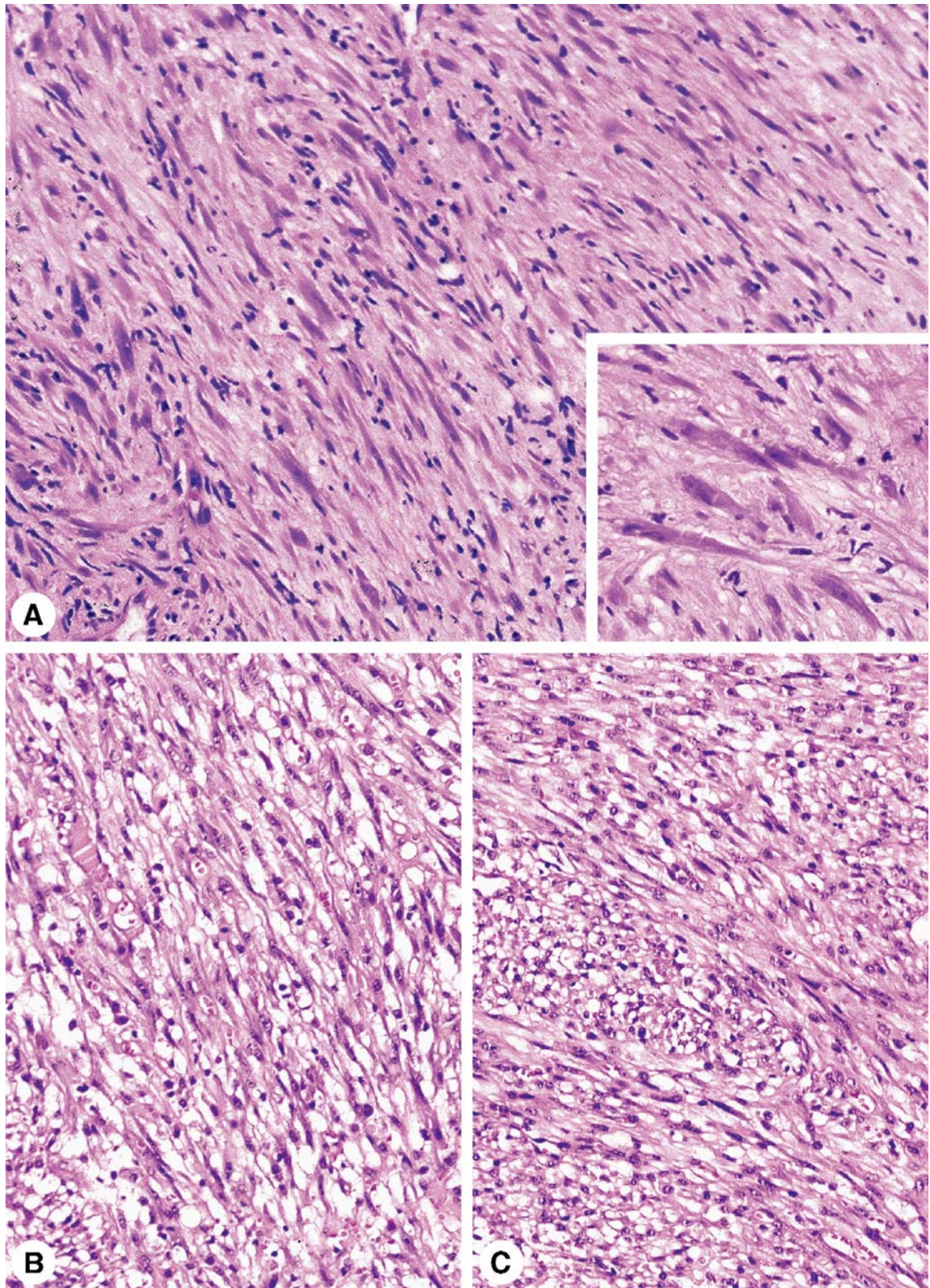
### 3.1. Microscopic features

#### 3.1.1. Pseudosarcomatous fibromyxoid tumor

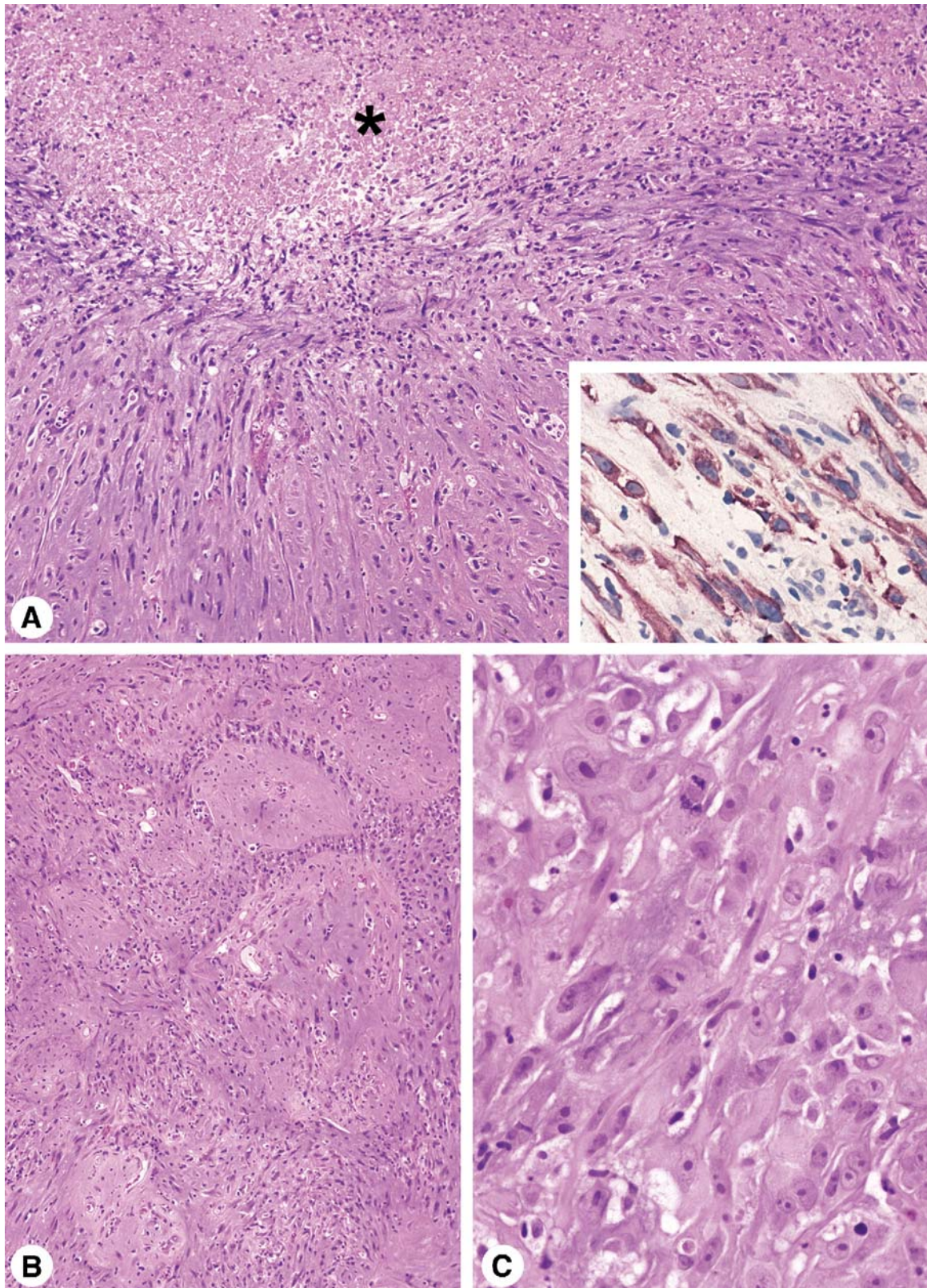
All 7 PFTs were characterized microscopically by proliferation of tissue culture-like spindled myofibroblastic cells within a myxoid stroma (Fig. 1). Scattered cells with enlarged atypical nuclei were nearly always present. The myofibroblastic cells were admixed with lymphocytic inflammatory cells. Typically, spindle cells were loosely arranged within the myxoid stroma. In some cases, a more compact arrangement of interlacing bundles mimicking the herringbone pattern, typically seen in fibrosarcoma, was present. A network of radiating capillary channels was prominent in peripheral parts of the lesion. Scattered mitoses (2-4 per 10 high-power fields) were present, but no atypical mitotic figures could be identified. In 3 PFT cases, paraffin blocks or unstained slides were available for immunohistochemical studies, with the spindle cells staining positive for smooth muscle actin. Scattered positive staining for cytokeratin was present within myofibroblastic cells in all 3 cases; however, some of the areas showed diffuse, strong positive staining for cytokeratin (AE1/AE3). Pseudosarcomatous fibromyxoid tumors were typically located within the submucosal stromal tissue. The involvement of the muscularis propria could be documented in all cases in which the biopsy material involved the deeper portion of the bladder wall.

#### 3.1.2. Postoperative spindle cell nodule

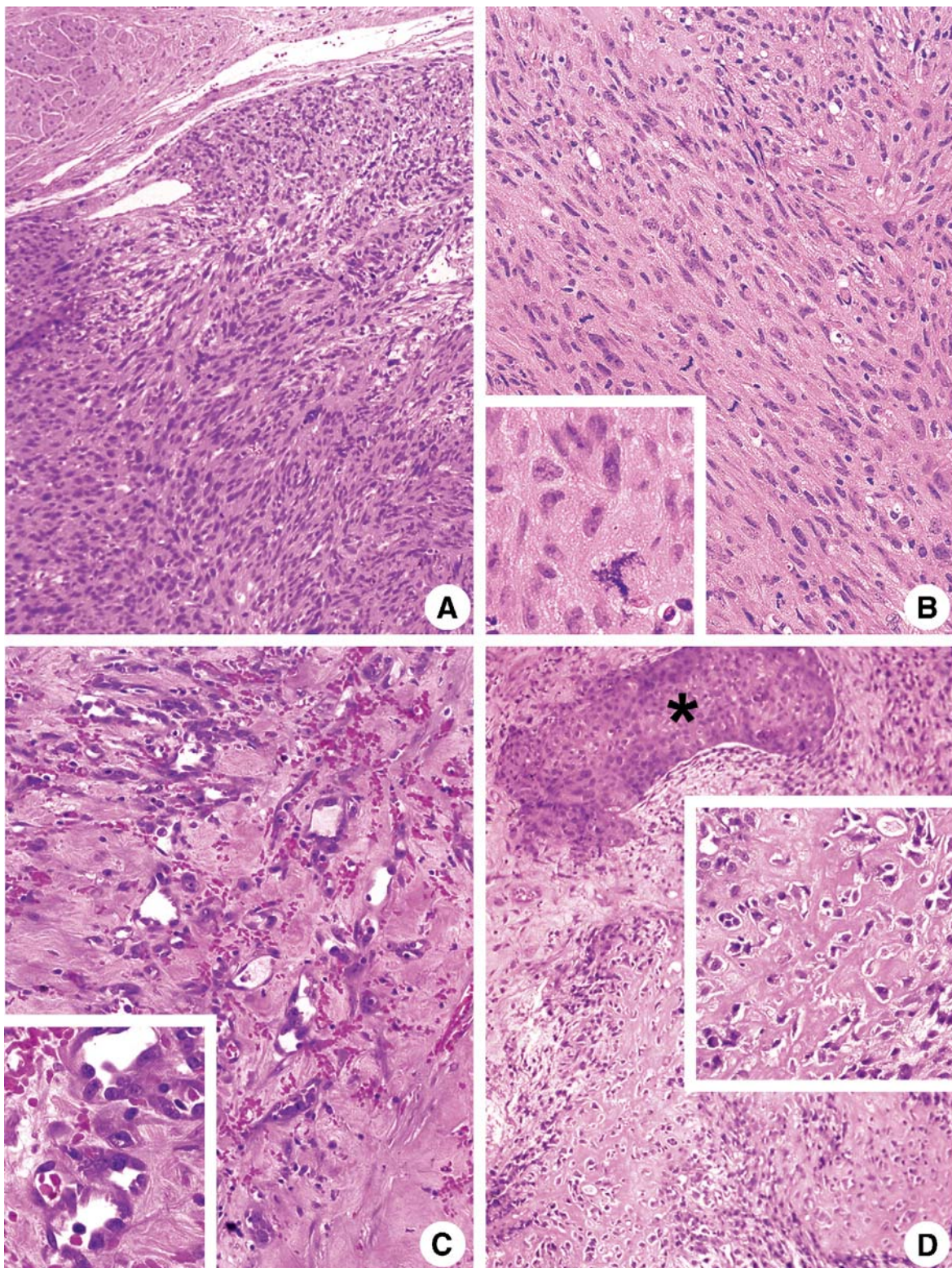
Some degree of reactive spindle cell proliferation follows every biopsy or TUR of the bladder; however, PSN is distinct in that it consists of a florid spindle cell proliferation forming a cystoscopically identifiable lesion or constituting a major component of the surgical specimen. Typically, PSNs are identified on follow-up cystoscopy as nodular mucosal protrusions measuring 1 cm or less. Consistent with a prior



**Fig. 1** Pseudosarcomatous fibromyxoid tumor microscopic features. A, Proliferation of spindled myofibroblastic cells admixed with inflammatory cells (H&E, original magnification  $\times 100$ ). Inset, Loose proliferation of tissue culture-like myofibroblastic cells (H&E, original magnification  $\times 200$ ). B, Loose fascicular arrangement of myofibroblastic cell (H&E, original magnification  $\times 100$ ). C, Compact fascicular arrangement of myofibroblastic cells (H&E, original magnification  $\times 100$ ).



**Fig. 2** Postoperative spindle cell nodule microscopic features. A, Proliferation of spindled myofibroblastic cells and areas of necrosis indicated by an asterisk related to a prior resection (H&E, original magnification  $\times 80$ ). Inset, Expression of cytokeratin (AE1/AE3) in myofibroblastic cells (H&E original magnification  $\times 200$ ). B, Low power view showing pseudo-organoid growth pattern produced by spindle and epithelioid myofibroblastic cells (H&E, original magnification  $\times 80$ ). C, Myofibroblastic cells with epithelioid features. Note nuclear atypia (H&E, original magnification  $\times 200$ ).



**Fig. 3** Sarcoma and sarcomatoid carcinoma microscopic features. A, High-grade unclassified spindle cell sarcoma (H&E, original magnification  $\times 100$ ). B, Leiomyosarcoma showing proliferation of spindled smooth muscle cells (H&E, original magnification  $\times 100$ ). Inset, Pronounced atypia and atypical mitosis (H&E, original magnification  $\times 200$ ). C, Angiosarcoma composed of atypical spindled and epithelioid cells forming vascular channels (H&E, original magnification  $\times 100$ ). Inset, Vascular channels composed of highly atypical endothelial cells (H&E, original magnification  $\times 200$ ). D, Sarcomatoid carcinoma showing phenotypic switch to osteosarcoma with prominent tumor osteoid deposition. Note well-demarcated epithelial nest (H&E, original magnification  $\times 80$ ). Inset, Atypical osteoblastic cells and tumor osteoid deposition (H&E, original magnification  $\times 200$ ).

**Table 1** Clinical characteristics of PSN, PFT, sarcoma, and sarcomatoid carcinoma

	PSN (n = 10)	PFT (n = 7)	Sarcoma (n = 18)	Sarcomatoid carcinoma (n = 17)
Mean age at presentation (y)	66 (40-85)	52 (37-76)	56 (22-94)	69 (40-88)
Ratio of men to women	4 : 6 (0.7)	2 : 5 (0.4)	11 : 7 (1.6)	11 : 6 (1.8)
Previous instrumentation	10 (100%)	0 (0%)	1 (5%)	4 (24%)
Previous pelvic surgery	1 (10%)	0 (0%)	2 (11%)	0 (0%)
Previous pregnancy	3 (30%)	5 (71%)	9 (50%)	1 (6%)
Previous radiotherapy	0 (0%)	1 (14%)	3 (17%)	0 (0%)
Previous history of bladder cancer	10 (100%)	0 (0%)	0 (0%)	4 (24%)

history of bladder instrumentation, PSNs were often in direct contact with necrotic areas associated with prior resection sites (Fig. 2). In TUR specimens tangentially cut, these prior resection sites could mimic geographic areas of necrosis similar to PFT, with the lesion characterized by a proliferation of tissue culture-like spindled cells within a myxoid stroma. Focally, cells showed epithelioid features with atypical nuclei varying in size and containing prominent nucleoli. These cells resembled the ganglion-like cells typically seen in proliferative myositis. The epithelioid cells were forming ill-defined areas or occasionally produced a pseudo-organoid pattern. A polymorphous inflammatory infiltrate was focally present and had a tendency to be more prominent in the vicinity of the necrotic areas. Although in many areas the spindle cells were sparsely arranged in a myxoid stroma, a focal compact arrangement of interlacing spindle cell fascicles could also be present. Both spindle and epithelioid cells showed coexpression of vimentin, smooth muscle actin, cytokeratin AE1/AE3, and CD68.

### 3.1.3. Sarcoma

The most frequent sarcoma of the bladder was leiomyosarcoma. Among 11 leiomyosarcomas of the bladder,

10 tumors were classified as high-grade, and the remaining case was of low histologic grade. High-grade unclassified spindle cell sarcomas resembled malignant fibrous histiocytoma of the soft tissue and consisted of 4 cases. All 3 cases of angiosarcoma were of high histologic grade composed of spindle and epithelioid cells forming vascular channels. In 9 cases, the specific lineages of sarcoma were identified with the help of immunohistochemical markers (smooth muscle actin, desmin, vimentin, CD34, CD31, and cytokeratin AE1/AE3). In the remaining 9 cases, the diagnosis of bladder sarcoma was based on hematoxylin and eosin (H&E) staining only. The presence of microscopically recognizable conventional urothelial carcinoma and/or positive staining for keratin were considered diagnostic of sarcomatoid carcinoma.

### 3.1.4. Sarcomatoid carcinoma

Among 17 sarcomatoid carcinomas, the epithelial component of 12 tumors was a conventional transitional cell carcinoma (TCC), and in 1 case, it showed prominent squamous differentiation. In the remaining 4 cases, no obvious microscopically recognizable precursor epithelial lesion could be identified, but strong positivity of compact

**Table 2** Patient characteristics of PSN (n = 10) and PFT (n = 7) groups

Case	Diagnosis	Age at diagnosis (y) (sex)	Presentation (history)	Primary treatment	Disease status (FU [mo])
1	PSN	75 (F)	GH (Hx of UC, T1G3)	TUR	NED (27)
2	PSN	49 (F)	SC (Hx of UC, TAG2)	TUR	NED (13)
3	PSN	78 (F)	SC (Hx of UC, TAG3)	TUR	NED (2)
4	PSN	71 (M)	GH (Hx of UC, T2G3)	TUR <sup>a</sup>	NED (67)
5	PSN	40 (M)	SC (Hx of UC, T1G3)	TUR	NED (45)
6	PSN	85 (M)	SC (Hx of UC, T1G3)	TUR	NED (46)
7	PSN	76 (F)	GH (Hx of UC, T1G3)	TUR	DOD (48)
8	PSN	62 (M)	SC (Hx of UC, TAG3)	TUR	NED (63)
9	PSN	72 (F)	SC (Hx of UC, T1G3)	TUR	DOD (48)
10	PSN	66 (F)	SC (Hx of UC, T1G3)	TUR	NED (34)
11	PFT	37 (F)	GH	TUR	NED (31)
12	PFT	76 (F)	GH	TUR	NED (30)
13	PFT	39 (F)	GH	TUR	NED (26)
14	PFT	53 (F)	GH	TUR	NED (157)
15	PFT	60 (M)	Dysuria	TUR	NED (42)
16	PFT	41 (F)	GH	TUR	NED (36)
17	PFT	61 (M)	GH	TUR	NED (32)

Abbreviations: GH, gross hematuria; UC, urothelial carcinoma; SC, Surveillance cystoscopy; PC, partial cystectomy; FU, follow-up; UC, urothelial carcinoma; Hx, history; NED, no evidence of disease; DOD, died of other disease; F, female; M, male.

<sup>a</sup> One patient underwent partial cystectomy as part of his treatment for UC.

**Table 3** Clinical management of PSN, PFT, sarcoma, and sarcomatoid carcinoma

	PSN (n = 10)	PFT (n = 7)	Sarcoma (n = 18)	Sarcomatoid carcinoma (n = 17)
Surgical management				
TUR	10 (100%)	7 (100%)	3 (17%)	6 (35%)
Partial cystectomy	0 (0%)	0 (0%)	3 (17%)	1 (6%)
Radical cystectomy and urinary diversion	0 (0%)	0 (0%)	7 (39%)	3 (18%)
Salvage cystectomy and urinary diversion	0 (0%)	0 (0%)	2 (11%)	7 (41%)
Chemotherapy	0 (0%)	0 (0%)	2 (11%)	0 (0%)
Palliation	0 (0%)	0 (0%)	1 (6%)	0 (0%)
Neoadjuvant chemotherapy	0 (0%)	0 (0%)	2 (11%)	7 (41%)

cell clusters for cytokeratin implicated sarcomatoid carcinoma rather than sarcoma. In 3 cases of sarcomatoid carcinoma, a sarcoma component showed a phenotypic switch to an osteoblastic lineage with prominent osteoid deposition recapitulating osteosarcoma. One of these cases, in addition to osteoblastic differentiation, contained areas of chondroblastic cells producing chondroid matrix. The most prominent feature differentiating all sarcomas and sarcomatoid carcinomas from PFT was a confluent growth of highly atypical sarcomatoid cells and the presence of atypical mitosis in high-grade sarcomas and sarcomatoid carcinomas (Fig. 3).

## 3.2. Clinical features

### 3.2.1. Pseudosarcomatous fibromyxoid tumor

The mean age of the 7 patients presenting with PFT was 52 years (37-76 years) with 5 women and 2 men (Table 1). All 5 female patients had a remote history of pregnancy (13-45 years ago) with 1 patient having received prior radiotherapy. None of the male patients had previous urological instrumentation. The most common clinical presentation of PFT was gross, painless hematuria (n = 6), as shown in Table 2. All patients were treated initially by TUR (Table 3). After definitive treatment, there were no local recurrences or metastatic spread in any of the patients at a median follow-up of 3 years (0.7-13.1 years), and none of the patients died of disease (Table 4).

### 3.2.2. Postoperative spindle cell nodule

The mean age at diagnosis of patients with PSN was 66 years (40-85 years) with 6 women and 4 men. All 10 patients had a history of bladder cancer and bladder instrumentation. In addition, 1 patient with PSN had a previous history of pelvic surgery. Most PSNs (n = 7) were detected on surveillance cystoscopy for bladder cancer. All

patients underwent a TUR as an initial treatment, and 2 patients had residual disease evident 2 to 3 months after the initial procedure, which was successfully treated by repeat TUR. In 1 patient with a prior history of TCC, partial cystectomy was performed. At a median follow-up of 3 years (0.1-5.6 years), there were no local recurrences in any of the patients with PSN, with all patients being alive without disease.

### 3.2.3. Sarcoma

The mean age of patients presenting with sarcoma was 57 years (22-94 years, 7 women and 11 men). The patient characteristics and treatment outcomes by sarcoma subtype are shown in Table 5. Most sarcomas had no prior history of instrumentation, pelvic surgery, or bladder cancer. Three patients with bladder sarcoma had previously received external beam radiotherapy (1 case 10 years ago for colon cancer, 1 case for prostate cancer 5 years ago, and 1 for cervical cancer 35 years ago), with all 3 cases being angiosarcomas.

Aggressive surgical therapy was used in the majority of these tumors consisting of radical cystectomy with or without neoadjuvant chemotherapy in 50%. Neoadjuvant chemotherapy was administered in 2 patients with sarcoma, with the neoadjuvant chemotherapeutic regimen consisting of vincristine, adriamycin, and cisplatin. The primary treatment modality in the remaining 3 patients with bladder sarcoma consisted of chemotherapy in 2 patients (doxorubicin and dacarbazine in 1 patient and vincristine, doxorubicin, and cisplatin in the other) and of palliation in 1 patient with advanced disease and a low performance status. Local recurrences and distant metastases frequently developed in primary sarcomas, with death from disease occurring in 50% patients. Of those still alive at last follow-up, 3 patients (17%) had sarcoma.

**Table 4** Disease-related outcomes of PSN, PFT, sarcoma, and sarcomatoid carcinoma

	PSN (n = 10)	PFT (n = 7)	Sarcoma (n = 18)	Sarcomatoid carcinoma (n = 17)
Rate of local recurrence	0%	0%	17%	65%
Rate of distant recurrence	0%	0%	56%	59%
Died of disease	0 (0%)	0 (0%)	9 (50%)	11 (65%)
Median DSM in years (range)	— <sup>a</sup>	— <sup>a</sup>	2.9 (0.2-14.6)	1.4 (0.2-7.5)
Median FU in years (range)	3.1 (0.2-5.6)	3.0 (0.7-13.1)	2.6 (0.2-11.9)	1.4 (0.2-5.6)

Abbreviation: DSM, disease-specific mortality.

<sup>a</sup> No disease-specific deaths occurred in the PSN and PFT groups; therefore, median disease-specific mortality could not be calculated.

**Table 5** Patient characteristics of primary bladder sarcomas and sarcomatoid carcinomas

	Mean age at diagnosis (y) (range)	Disease specific deaths (%)	Mean length of FU (mo) (range)
Bladder sarcoma (n = 18)			
Leiomyosarcoma (n = 11)	55 (30-75)	5/11 (55%)	39 (3-144)
Unclassified sarcoma (n = 4)	57 (22-94)	2/4 (50%)	11 (3-33)
Angiosarcoma (n = 3)	67 (66-69)	2/3 (67%)	29 (3-69)
Sarcomatoid carcinoma (n = 17)			
Without phenotypic shift (n = 14)	69 (40-88)	10/14 (71%)	17 (1-68)
With phenotypic shift (n = 3)	73 (42-83)	1/3 (33%)	39 (12-91)

### 3.2.4. Sarcomatoid carcinoma

The mean age of those with sarcomatoid carcinoma was 69 years (40-88 years, 6 women and 11 men), with none having previously received radiotherapy. Most patients with sarcomatoid carcinoma were managed by neoadjuvant chemotherapy, followed by salvage surgery (41%) or TUR (35%). The neoadjuvant chemotherapeutic regimen selected for these patients consisted of (1) cisplatin, gemcitabine, and ifosfamide in 4 patients, (2) ifosfamide and adriamycin in 2; and (3) methotrexate, vinblastine, adriamycin, and cisplatin in 1. The disease-specific mortality rate of patients with sarcomatoid carcinoma was 65%, with an additional 2 patients alive with persistent disease.

## 4. Discussion

This and other case series [1,5,12] have described some of the clinical and histopathologic features of bladder PFT, PSN, sarcoma, and sarcomatoid carcinoma. However, accurate diagnostic criteria are not clearly defined, and their optimal treatment remains a frequent clinical dilemma.

Our data show that PFT and PSN have some overlapping microscopic features but most likely represent pathogenetically distinct lesions that develop in distinct clinical settings. Postoperative spindle cell nodule shows a clear association with recent bladder instrumentation, such as biopsy or TUR, often with a history of prior resection of TCC. Consistent with this association, the florid myofibroblastic proliferation of PSN shows a clear association with areas of necrosis at prior biopsy sites, and they typically occur in an older patient population (>60 years of age). Microscopically, these lesions are similar to PFT but often contain areas of epithelioid histiocytic cells that may form pseudo-organoid patterns. These lesions typically do not show confluent bundles of spindle cells typically seen in PFT. Our series of PFTs shows a clear predilection for females below the age of 45 years. The association with potential bladder injury by instrumentation or other procedures appears to be remote and uncertain. We believe that the development of PFT in association with pregnancy may be due to a subclinical trauma to the bladder, but again, such association is remote and uncertain.

Microscopically, these lesions are characterized by more compact spindle cell proliferation admixed with inflamma-

tory cells that may form interlacing bundles mimicking the herringbone pattern seen in fibrosarcoma. Loose proliferations of spindle cells in myxoid stroma are less prominent than in PSN, and epithelioid histiocytic cells forming focal organoid patterns as well as areas of necrosis are not present. Overall, PFT has a microscopic resemblance to nodular fasciitis.

Since first described by Roth [2], there have been 100 urogenital PFTs described in the scientific literature, of which 72 have involved the urinary bladder [1]. In addition, a significant proportion of the bladder PFT and PSN was initially diagnosed as primary sarcomas or sarcomatoid carcinomas [11,17]. Establishing the correct diagnosis has important clinical implications because the misdiagnosis of a PFT or PSN as a malignant neoplasm places the patient at risk of unnecessary radical surgery with its inherent potential complications and morbidities. In the present study, we have outlined some of the key clinical and pathologic features that distinguish benign from malignant spindle cell lesions of the bladder. Pathologists and urologists should take these characteristics under consideration when establishing the diagnosis of one of these histologic entities. Many of the clinical features characteristic of PFT and PSN have been mentioned in a recent review of 42 cases by Harik et al [20]. In this series, the authors also noted the limited growth potential of these bladder lesions, with none of the patients developing metastases.

Our findings are consistent with previous reports suggesting that PFT occurs in younger female patients [1,15,21]. A previous history of urologic instrumentation is characteristic of PSN and was present in all patients. This finding points to the importance of reviewing the patient's medical history, which could provide important clues in making a clinical and pathologic correlation. This has been previously cited as a salient clinical feature of PSN [7,9,22]. Other important components of the clinical history include a previous history of pregnancy and prior radiotherapy. Three patients with primary sarcoma (all angiosarcomas) had received prior radiotherapy, with this association previously reported by others [23].

In the study by Iczkowski et al [24], the presence of necrosis and nuclear atypia were the features that best differentiated primary sarcoma from PFT. Although muscularis propria invasion is suggestive of invasive disease for TCC, this is not so with PFTs, as exemplified in this and

other studies [1]. Positivity of PFT and PSN for epithelial markers such as cytokeratin is misleading and may contribute to the misdiagnosis of these conditions as sarcomatoid carcinomas [12,15]. However, in a recent study by Hirsch et al [25], the authors noted the use of immunostaining for anaplastic lymphoma kinase in the diagnosis of PFT, thereby potentially allowing its distinction from sarcomatoid carcinoma.

In summary, conservative excision of PFT and PSN by TUR is sufficient to eradicate these lesions, with very few patients requiring further additional resection. This further underlines the importance of establishing a correct diagnosis among these lesions, which have varying biologic potentials with its important therapeutic and prognostic implications [24].

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