Mesenchymal tumors of the prostate

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Mesenchymal tumors of the prostate are rare but often cause considerable diagnostic difficulty when encountered. These may be either benign or malignant and can arise within the prostate gland or in adjacent tissues. This review covers prostatic stromal proliferations (prostatic stromal tumor of uncertain malignant potential and stromal sarcoma), solitary fibrous tumor, myofibroblastic proliferations, smooth muscle neoplasms (leiomyoma and leiomyosarcoma), gastrointestinal stromal tumor, schwannoma, rhabdomyosarcoma, sarcomatoid carcinoma, postradiation sarcoma, and mixed epithelial stromal tumor of the seminal vesicle. Differential diagnostic considerations are emphasized, including descriptions of helpful histological and/or clinical clues and the use of adjuvant diagnostic techniques.

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True mesenchymal neoplasms of the prostate make up < 1% of the tumors occurring in this anatomical location. Considerable diagnostic difficulties may be encountered with such lesions due to their rarity, which leads to a very limited experience for most pathologists. This is further complicated by the fact that these lesions are sampled by relatively small core biopsy specimens and that the diagnosis is often unexpected. Moreover, the histological differences between distinct spindle cell lesions are generally more subtle than for epithelial neoplasms. Although prostatic stromal proliferations/neoplasms represent the most commonly considered mesenchymal tumor at this site, solitary fibrous tumor, myofibroblastic proliferations, smooth muscle neoplasms, gastrointestinal stromal tumors, schwannomas, rhabdomyosarcomas, postradiation sarcomas, and mixed epithelial stromal tumors of the seminal vesicle may all be encountered in 'prostate biopsies'. Sarcomatoid carcinoma, while not of mesenchymal derivation, is also discussed due to its considerable histological overlap with sarcomas. This review details the diagnostic features of each entity to include detailed differential diagnostic considerations and helpful adjunctive diagnostic tests.

Prostatic stromal proliferations

Benign prostatic hyperplasia (BPH), which is often stromal predominant, is a fairly common finding in transurethral resection specimens of the prostate. As florid BPH must be differentiated from prostate stromal neoplasia, the latter is probably the most frequently considered spindle cell neoplasm of the prostate gland. The current WHO classification includes two diagnostic categories: stromal tumor of uncertain malignant potential (STUMP) and stromal sarcoma.¹

Prostatic STUMPs may have significant histological overlap with florid stromal hyperplasia. Several histological patterns of STUMP are described, including cellular spindled, eosinophilic pattern (closely mimicking smooth muscle), a pattern with scattered atypical stromal cells, phyllodes-like pattern, and myxoid pattern (Figures 1a-d); these patterns may be intermixed.^{2–4} Another recently described histological pattern has 'round cell' morphology.⁵ The hypercellular stromal pattern consists of bland fusiform stromal cells, often admixed with benign prostatic glands. In general, the cytologically bland spindle cell pattern has the most overlap with stromal hyperplasia. Stromal hyperplasia generally contains small blood vessels, often with some degree of hyalinization, which is distinctive and helps in distinction from a true prostatic stromal neoplasm (Figures 2a and b). On low power evaluation, florid stromal hyperplasia often has a more lobulated or nodular architecture, while a true prostatic stromal neoplasm (STUMP) should have a more diffuse growth pattern. It should be noted that BPH can occasionally have stromal

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Figure 1 Prostatic stromal neoplasm of uncertain malignant potential (STUMP) with (a) cellular spindled pattern and invasion of extraprostatic soft tissue. (b) The individual neoplastic cells are cytologically bland. Other patterns include (c) tumors with scattered atypical stromal cells between glands and (d) an eosinophilic pattern mimicking smooth muscle.

Figure 2 Benign prostatic hyperplasia (BPH) may be distinguished from STUMP by (a) lobular/nodular architecture and (b) a characteristic vascular pattern with small round well-formed blood vessels.

indentation into a glandular space creating a ‘phyllodes-like’ or ‘fibroadenoma-like’ architecture (Figure 3a). In such cases, the hyperplastic stroma does not show cellular condensation adjacent to the

epithelium, and this ‘phyllodes-like’ pattern is often very focal in the background context of prototypical BPH. Myxoid patterns of prostatic STUMP may mimic a variety of other myxoid mesenchymal

Figure 3 (a) Benign prostatic hyperplasia (BPH) may have foci that mimic a 'phyllodes-like' pattern, but other surrounding features of BPH are typical. (b) Prostatic stromal sarcoma has marked nuclear atypia and mitotic activity is often brisk.

lesions, including inflammatory myofibroblastic tumor and myxoid variant of solitary fibrous tumor. The 'round cell' pattern of STUMP may be more readily mistaken for a gastrointestinal stromal tumor (GIST), or possibly a prostatic adenocarcinoma, but immunostains should easily resolve those considerations (discussed in separate sections below).

Epithelial proliferations are also described in association with prostatic STUMPs and include: glandular crowding, prominent basal cells, papillary infolding, cystic dilatation, basal cell hyperplasia, urothelial metaplasia, squamous metaplasia, and cribriform hyperplasia.⁶ Of these features, glandular crowding and prominent basal cells are most common.

Prostatic stromal sarcomas are much easier to diagnosis as neoplastic because their overtly malignant features are more easily distinguished from florid stromal hyperplasia. Stromal sarcomas often show greater cellularity, greater cytological atypia, easily identifiable mitotic activity, and tumor cell necrosis (Figure 3b). When phyllodes-like patterns are present, there is often marked stromal condensation around the glands, a pattern similar to a malignant phyllodes tumor of the breast or adenocarcinoma of the uterus.

Immunophenotypically, prostatic stromal tumors commonly show expression of CD34 and progesterone receptor. Although some actin and desmin reactivity may be seen in STUMPs, this is commonly lost in stromal sarcomas. Additionally, while not commonly used for diagnosis, common chromosomal aberrations are reported and include loss of chromosome 10, 13, and 14.⁷

Prostatic STUMPs are typically indolent and are generally cured with complete resection. They may be treated more conservatively in carefully considered patients; however, there are rare cases associated with sarcomatous dedifferentiation, warranting very careful clinical follow-up if resection is not attempted. Sarcomas have fully metastatic

potential and generally require surgical resection and consideration for adjuvant therapy.

Solitary fibrous tumor

Solitary fibrous tumor represents a distinct fibroblastic-type mesenchymal neoplasm that may have significant histological overlap with prostatic stromal neoplasia and florid stromal hyperplasia. In the older literature, many of these tumors were diagnosed and reported as 'hemangiopericytoma', but they are now designated as solitary fibrous tumor.⁸ Given the common expression of CD34 by solitary fibrous tumors at any anatomical site, there is significant overlap with prostatic stromal proliferations. In fact, there was original skepticism about true solitary fibrous tumors occurring in the prostate. However, identification of a novel recurrent in *NAB2-STAT6* gene fusion has confirmed that solitary fibrous tumors do occur in the prostate (and it also provides a very useful diagnostic signature).^{9,10} These tumors appear identical to their description in other anatomical sites, with bland fibroblast-like cells arranged in a very disorganized non-repetitive pattern (ie, 'patternless' pattern), scattered angulated hemangiopericytic-like vessels, variable stromal collagenization, and occasional cellular and myxoid forms (Figures 4a and b). Occasionally, a cord-like growth pattern may be seen and could mimic a smooth muscle neoplasm or even a plasmacytoid carcinoma from the urothelium. Rare solitary fibrous tumors may contain lipid-rich areas. In superficial biopsies obtained by a transurethral method, solitary fibrous tumor can be difficult to recognize and may closely mimic benign subepithelial connective tissue or BPH. A novel monoclonal antibody for STAT6 is now available, which shows strong and diffuse nuclear reactivity in solitary fibrous tumors and is generally negative in most morphological mimics.¹¹⁻¹³ Although some immunophenotypic

Figure 4 Solitary fibrous tumor (a) may extend to the urethral surface where it could mimic lamina propria or BPH. (b) Some foci may show more characteristic intercellular collagen.

overlap is reported between solitary fibrous tumor and prostatic stromal neoplasms using the polyclonal STAT6 antibody,¹⁴ it is our experience that the monoclonal antibody is more specific. However, this warrants further study, and fluorescence *in situ* hybridization (FISH) or PCR confirmation of the *NAB2-STAT6* fusion is available for challenging cases. It should also be remembered that STAT6 may be amplified in a subset of dedifferentiated liposarcomas; therefore, those tumors may also show nuclear reactivity with the STAT6 antibody.^{15,16} Finally, it is important for genitourinary pathologists to be aware that solitary fibrous tumor expresses PAX-8 in 40–45% of cases.¹⁷

Solitary fibrous tumors, when completely excised, most commonly follow a benign clinical course, but more aggressive behavior can occur.¹⁸ There are several prognostic models provided for risk assessment in solitary fibrous tumors and they generally include features such as tumor size, anatomical location, mitotic activity, nuclear pleomorphism, necrosis, and patient age.^{19–21}

Smooth muscle neoplasms

Smooth muscle neoplasms in the prostate are similar to those that occur elsewhere, such as somatic soft tissue and the uterus. Leiomyomas are rare and their distinction from prostatic stromal hyperplasia is arbitrary in some cases. They are comprised of a well-circumscribed proliferation of cytologically bland spindle cells with prominent eosinophilic cytoplasm and a fascicular architecture. Leiomyosarcomas may be infiltrative or have a pushing border but show greater nuclear variability, nuclear hyperchromasia (Figure 5), mitotic activity, and (in some cases) necrosis. As in other sites, leiomyosarcomas are aggressive with potential for distant metastasis.

Figure 5 Leiomyosarcoma typically has marked nuclear atypia.

There are some reported cases of ‘atypical’ leiomyomas in the urinary tract, analogous to those in the uterus.^{22,23} Although we acknowledge that rare smooth muscle neoplasms of the urinary tract, including the prostate gland, may have morphological features similar to ‘atypical’ (also known as ‘symplastic’ or ‘bizarre’) leiomyoma, that diagnosis should be rendered with extreme caution. These tumors may have significant overlap with the atypical cell pattern of prostatic STUMP. For a smooth muscle tumor with cytological atypia, the presence of mitotic activity would favor a leiomyosarcoma.

Myofibroblastic proliferations

Myofibroblastic proliferations may occur in the prostate but are much less common than those seen in the urinary bladder. These have been described under a variety of different names, such as post-operative spindle cell nodule, inflammatory

myofibroblastic tumor, and pseudosarcomatous myofibroblastic proliferation.^{24–27} Some occur *de novo*, while others follow a prior injury (including biopsy). Cases described as inflammatory myofibroblastic tumor have heterogeneous findings, but up to 75% have an *ALK* re-arrangement by FISH and *ALK* protein expression by immunohistochemistry.^{24,28} Although nomenclature is still debated for myofibroblastic proliferations in the urinary tract, cases with typical histological features have all behaved in a benign manner regardless of prior instrumentation or presence/absence of *ALK* re-arrangement. Immunophenotypically, these myofibroblastic proliferations often express low-molecular-weight cytokeratin, which may cause confusion with sarcomatoid carcinoma; however, they do not express high-molecular-weight cytokeratins. They also commonly express smooth muscle actin and desmin.

The most important differential diagnostic distinction is with a malignant neoplasm, such as leiomyosarcoma (or other sarcoma) and sarcomatoid carcinoma with myxoid stroma. Myofibroblasts may have significant nuclear variability in size and shape, and prominent nucleoli (sometimes multiple) are not uncommon, but the chromatin is evenly dispersed (Figures 6a and b). The presence of irregular, hyperchromatic chromatin is the key feature that suggests a malignant neoplasm.²⁹ In patients aged <40 years of age, we would recommend careful exclusion of embryonal rhabdomyosarcoma before a myofibroblastic lesion is diagnosed (see below).

Gastrointestinal stromal tumor

Very rarely, GISTs may be sampled on transrectal prostate biopsies.³⁰ GISTs discovered in prostate needle biopsies often represent rectal based tumors that compress and displace the prostate gland. These

GISTs, similar to other sites of origin, may have a variety of histological patterns, including spindled and epithelioid morphology (Figure 7).³¹ The spindle cell morphology may have significant overlap with that of prostatic stromal proliferations, which are more common. Immunophenotypically, GISTs characteristically express CD34, CD117, and DOG-1. Although not exhaustively studied, DOG1 staining has been reported in prostatic stromal sarcoma.³² As with many tumors presenting in unusual locations, the key to diagnosis is routinely considering the diagnostic possibility of GIST in this location when faced with a spindle cell lesion. Although not specifically evaluated in the prostatic region, we would utilize the typical GIST prognostic stratification recommend by the WHO.³¹

Figure 7 Gastrointestinal stromal tumor in a prostate needle core biopsy. One must remember to consider this diagnostic possibility in prostate biopsies.

Figure 6 Myofibroblastic proliferation ('inflammatory myofibroblastic tumor') of the prostate. (a) The cells have typical myofibroblastic features with elongated or 'stretched-out' spindle cells, often with a loose myxoid background. (b) The nuclei may have significant variation in size, and nucleoli may be quite prominent or multiple; however, nuclear hyperchromasia is not seen.

Schwannoma

The diagnosis of schwannoma is typically straightforward. However, when sampled unexpectedly in a needle core biopsy of the 'prostate' or 'seminal vesicle', the diagnostic possibility of schwannoma may not be considered. On imaging, a pelvic-based schwannoma may compress the prostate and seminal vesicles, creating confusion as to the site of origin.³³ There may be significant histological overlap with a prostatic stromal proliferation or solitary fibrous tumor, but helpful histological clues for schwannoma include large caliber blood vessels with mural hyalinization, nuclear palisading, a 'wavy' character of the spindle cells, and variation from hypercellular to hypocellular zones (Figure 8a). Once the diagnosis is considered, immunohistochemical demonstration of diffuse S-100 protein (or SOX-10) expression can confirm the diagnosis. Some schwannomas may appear macroscopically cystic,

which causes further overlap with prostatic or seminal vesicle tumors.^{34,35} Histologically, these cystic schwannomas can contain 'pseudoglandular' spaces that are lined by elongated cells resembling pseudostratified columnar epithelium (Figure 8b).³⁶ These cyst-lining cells also express S-100 protein and are negative for keratin. One other immunohistochemical pitfall is the expression of PSMA by schwannomas, which may also be detected by PSMA imaging methodologies.^{37–39}

Rhabdomyosarcoma

Rhabdomyosarcoma involving the prostate gland is typically of the embryonal subtype and occurs predominantly in the pediatric population; only rare cases are described in adults. Alveolar, spindle cell/sclerosing, and pleomorphic variants of rhabdomyosarcoma are extremely rare in this location. These

Figure 8 Schwannoma presenting as a 'prostate' mass. (a) The large hyalinized blood vessels are a helpful clue to consider schwannoma. (b) Rare cases show 'pseudoglands' that are not epithelial but may cause consideration of a mixed glandular and stromal lesion.

Figure 9 Embryonal rhabdomyosarcoma of the urinary tract (a) may mimic a variety of spindle cell lesions and must be carefully considered in young patients. (b) Cartilaginous differentiation may also be seen.

tumors often have increased cellularity underneath the prostatic urethra, which may be urothelial or squamous-lined in children; however, this classic cambium layer is not always present because significant morphological heterogeneity is typical. The neoplastic cells are often spindled, but fusiform or more rounded cells may be seen and well-developed rhabdomyoblasts are frequently identified (Figure 9a). Some foci may be quite hypocellular and cytological atypia may not be pronounced (Figure 9b); therefore, it is imperative to consider the diagnosis of a rhabdomyosarcoma in young patients when a spindle cell proliferation is encountered on biopsy. Another unusual feature of embryonal rhabdomyosarcoma is the occasional presence of cartilaginous differentiation (Figure 9b), which we have seen in the prostate where it mimics a sarcomatoid carcinoma.^{40,41} Immunohistochemical markers of skeletal muscle differentiation, myogenin and MyoD1, are most helpful for this diagnosis. Correct diagnosis is critical because pediatric rhabdomyosarcoma of the genitourinary tract has excellent cure rates with appropriate standard therapy, which is frequently updated by the Children's Oncology Group.^{42,43}

Sarcomatoid carcinoma

Sarcomatoid carcinoma of the prostate is very rare and may occur *de novo* or as an aggressive recurrence following a history of typical acinar adenocarcinoma.^{44–46} Histologically, a mixture of frank epithelial and sarcomatous components are seen in most cases (Figure 10a). The epithelial components often have a high-grade acinar morphology, but ductal, small cell, or squamous differentiation may also be seen. The epithelium expresses markers typical of their differentiation (eg, keratin, NKX3.1, PSA). The sarcomatous component is often

'undifferentiated' with bizarre giant cells (Figure 10b) or pleomorphic spindle cells, but heterologous differentiation in the form of osteosarcoma, chondrosarcoma, and rhabdomyosarcoma are described. In rare cases, epithelial differentiation may not be recognizable histologically and is only documented by immunohistochemistry (in the clinical context of a prior prostate cancer). Immunophenotypically, the sarcomatoid component of most cases will show at least focal expression with broad spectrum keratins, but PSA expression is typically absent.⁴⁴ The main differential diagnostic consideration in a *de novo* setting is a primary sarcoma. In the setting of prior prostate cancer, a postradiation sarcoma should be considered (see below). Rarely, sclerosing adenosis might mimic a sarcomatoid carcinoma.^{47–49}

Postradiation sarcoma

Postradiation sarcomas often occur close to 10 years following radiation treatment and may show a variety of morphological patterns, most commonly osteosarcoma, pleomorphic undifferentiated sarcoma, and angiosarcoma.^{50,51} The pathological distinction between a sarcomatoid carcinoma and a postradiation sarcoma may be impossible in some cases. Late progression to an aggressive prostatic carcinoma variant may occur (eg, sarcomatoid carcinoma or 'pleomorphic giant cell carcinoma'), which can follow radiation in some cases and often has significant histological overlap with sarcoma.⁵² Obviously, the presence of a more typical carcinomatous component (described above) makes the diagnosis straightforward, but in tumors with a pure sarcomatous pattern adjunctive testing may be needed. Unfortunately, the spindle cell component of sarcomatoid carcinoma may lose the expression of epithelial- and prostatic-specific markers. In the

Figure 10 Sarcomatoid carcinoma of the prostate often shows (a) frank carcinomatous and sarcomatous components. The carcinomatous component shows squamous differentiation in this example. (b) Some cases may show markedly pleomorphic tumor giant cells.

Figure 11 (a) Benign mixed epithelial and stromal tumor of the seminal vesicle shows an admixture of cytologically bland stromal cells and simple epithelial glands, while (b) malignant mixed epithelial and stromal tumor has more marked cytological atypia in the stromal cells.

absence of any histological or immunophenotypic evidence of epithelial differentiation, a postradiation sarcoma may be favored. This distinction makes little clinical difference as both tumors follow a rapidly progressive course despite therapy.

Mixed epithelial–stromal tumor of seminal vesicle

Mixed epithelial and stromal tumors of the seminal vesicle are rare biphasic neoplasms comprised of benign epithelial glands and more variable stromal components (Figure 11a). They have historically been described under a variety of names, including phyllodes tumor, cystadenoma, cystomyoma, and müllerian adenosarcoma-like tumor.⁵³ These may be small incidental lesions seen at radical prostatectomy or may present as large cystic masses. Histologically, benign tumors have a cytologically bland spindle cell stroma separated by cystically dilated, often branching glands. The epithelium is immunoreactive for keratins and negative for PSA, while the stroma commonly expresses smooth muscle actin. Malignant mixed epithelial and stromal tumors are more cellular (often condensing under the epithelium), have more cytological atypia, and have increased mitotic activity (Figure 11b). These malignant tumors may also have areas of sarcomatous stromal overgrowth very similar to adenosarcomas of the gynecological tract. Benign tumors are cured by excision, but the rare malignant variant has potential for an aggressive course with metastatic spread.

In our experience, the most significant differential diagnosis on biopsy is prostatic cystadenocarcinoma.⁵⁴ These may also present as a large cystic masses and may distort the seminal vesicle, causing confusion as to the site of origin. On biopsy, the epithelium is quite heterogenous and may be difficult to recognize as neoplastic. If intracystic

papillary excrescences are present (often lined by pseudostratified columnar epithelium), a cystadenocarcinoma of prostatic origin is likely; this may be confirmed with prostatic epithelial markers, such as NKX3.1 or PSA. In contrast, seminal vesicle epithelium lacks this expression and commonly shows nuclear PAX-8 reactivity.⁵⁵ Pseudoglandular schwannomas may also closely mimic a mixed epithelial and stromal tumor of the seminal vesicle (discussed separately above).^{34,35}

Other lesions

A variety of other mesenchymal and spindle cell lesions have been reported to occur very rarely in the prostate. These include: angiosarcoma,⁵⁶ hemangioma,^{57,58} hemangioendothelioma,⁵⁹ pleomorphic undifferentiated sarcoma,^{60,61} synovial sarcoma,⁶² malignant peripheral nerve sheath tumor,^{63,64} neurofibroma,⁶⁵ and granular cell tumor.⁶⁶

Conclusions

Mesenchymal neoplasms of the prostate may present significant diagnostic difficulty because their morphological features have a significant degree of overlap. Moreover, they are quite rare and often encountered unexpectedly in biopsy or transurethral resection samples. Therefore, pathologists have less experience (and comfort) diagnosing such lesions. Awareness of the diagnostic possibilities for this spectrum of mesenchymal lesions encountered in the prostate provides a significant step toward arriving at the correct diagnosis.

Disclosure/conflict of interest

The author declare no conflict of interest.

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