

Case Report

Prostatic Adenocarcinoma Metastatic to Pleomorphic Liposarcoma, a “Collision Phenomenon”: Report of a Case with Review of Pelvic Collision Tumors

Somak Roy,¹ Ronald L. Hrebinko,² Kathleen M. Cieply,³
Anil V. Parwani,⁴ and Uma N. M. Rao⁵

¹ Department of Pathology, University of Pittsburgh Medical Center, A615, Scaife Hall, 3550 Terrace Street, Pittsburgh, PA 15261, USA

² Department of Urology, University of Pittsburgh Medical Center, Suite 209, Shadyside Medical Building, 5200 Centre Avenue, Pittsburgh, PA 15232, USA

³ Department of Pathology, University of Pittsburgh Medical Center (UPMC), 631E Montefiore Hospital, Pittsburgh, PA 15261, USA

⁴ Department of Pathology, UPMC Shadyside Hospital, Room Wing 02.10, 5230 Centre Avenue, Pittsburgh, PA 15232, USA

⁵ Department of Pathology, University of Pittsburgh Medical Center (UPMC), Room 2.9 West Wing, Shadyside, Pittsburgh, PA 15232, USA

Correspondence should be addressed to Somak Roy, roys911@gmail.com

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“Collision tumor” is an uncommon phenomenon characterized by coexistence of two completely distinct and independent tumors at the same site. Collision tumors have been reported in different sites in the body; however, these are particularly uncommon in the pelvic cavity. A 70-year-old man, with prior history of urothelial and prostate cancer, presented with a large pelvic mass detected on imaging studies. Pathological examination revealed a large liposarcoma with prostatic carcinoma embedded in it. Immunohistochemistry and fluorescence in situ hybridization studies were performed to reach to a conclusive diagnosis. To the best of our knowledge, this is the second case reported till date. We present the challenges encountered in the diagnosis of this case and review of pelvic collision tumors.

1. Introduction

“Collision tumor” is a well-documented but uncommon phenomenon characterized by coexistence of two completely distinct and independent tumors at the same site [1–11]. The two morphologically separate tumors, which are genetically different, are sharply demarcated from each other. This entity is distinct from neoplasms demonstrating heterologous elements, as a result of divergent differentiation [1, 4].

The previously documented cases of collision tumor are predominantly synchronous epithelial tumors with different lineages such as melanoma occurring in a basal cell carcinoma [10] or gastric adenocarcinoma within a gastrointestinal stromal tumor [11], tumor to tumor metastasis [1, 2, 4, 12, 13], tumors coexisting as part of a systemic process (small

cell lymphoma/chronic lymphocytic leukemia with pelvic myelolipoma and renal cell carcinoma with intravascular lymphomatosis) [5, 6], and two coexistent metastatic tumors in a single lymph node [14–21]. The last category is rare and has also been referred to as “collision metastasis” [14, 16]. The various collision tumors have been documented in different regions of the body including head and neck, thorax, abdomen, and pelvic cavity. The incidence of carcinoma metastasizing to mesenchymal tumor, especially in the pelvis, is extremely rare [3, 7, 9, 12, 22]. We present a case of a patient with both urothelial and prostate cancers where in the prostatic carcinoma metastasized to a retroperitoneal pleomorphic liposarcoma. The lineages of both tumors were confirmed by immunohistochemistry and fluorescence in situ hybridization (FISH).

2. Clinical History

The patient was a 70-year-old man with a surgical history significant for high-grade urothelial carcinoma of urinary bladder. In 2003 he presented with painless hematuria and was found to have a tumor confined entirely inside a large urinary bladder diverticulum. Cystoscopy confirmed no tumor in the bladder cavity, and a pelvic CT did not demonstrate any evidence of extravesical tumor spread or metastatic disease. Microscopic examination of the prior bladder tumor demonstrated mixture of highly atypical malignant epithelioid and spindle cells (Figure 1), which demonstrated diffuse immunorepression of pancytokeratin and cytokeratin 20. The tumor cells were negative for vimentin and CD68. The features were diagnostic of high-grade sarcomatoid urothelial carcinoma. He underwent partial cystectomy followed by adjuvant chemotherapy and intravesical BCG. In 2006, he had radical retropubic prostatectomy and radiotherapy for a prostate adenocarcinoma, Gleason's score $4 + 4 = 8$ (Figure 2). Extracapsular extension, seminal vesicle, and perineural and angiolymphatic invasion were found on pathological examination. There were no palpable lymph nodes; computed tomography did not show any evidence of lymph node metastasis, and there was no recurrence of urothelial carcinoma.

Forty-five months later, a steady rise in serum prostate-specific antigen (PSA) from 1.9 ng/mL to 8.9 ng/mL was noted over the last 4 months. A surveillance cystoscopy showed evidence of extrinsic bladder compression which on digital rectal examination was felt to be a firm mass. Computed tomography with contrast of the pelvis and abdomen showed a large, 18×12.7 cm heterogeneous solid-cystic mass with irregular internal enhancement. The mass, extended from the pelvis up to the transverse segment of the duodenum, involved the dome of the bladder, left pelvic side wall, left ureterovesical junction, and rectosigmoid colon. There was evidence of left-sided obstructive hydronephrosis due to the tumor. No evidence of metastasis was seen in thorax and upper abdomen.

The patient underwent resection of the tumor with en bloc sigmoid colectomy and ureteroneocystostomy. Intraoperatively the large mass was visualized which appeared to be arising from pelvis with definite invasion into the sigmoid colon, involving lower portion of left ureter and portion of the dome of bladder. A small irregular lesion was noted in the lower retrovesical region which was found to be well-differentiated prostatic adenocarcinoma on intraoperative frozen section. The mass was entirely resected and sent for pathological examination.

3. Pathologic Findings

3.1. Gross Findings. The resected specimen was a large, well circumscribed, nonencapsulated, and yellow-pink multinodular $18.5 \times 16.0 \times 11.5$ cm mass with an attached segment 32.5 cm of large intestine (Figure 3(a)). The cut surfaces of the mass were firm with multiloculated cysts, focally hemorrhagic, and variegated tan-brown to red. The cystic

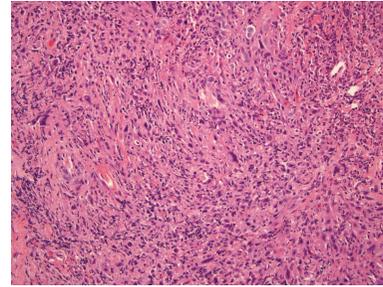


FIGURE 1: Sarcomatoid urothelial carcinoma. Tumor comprised of sheets of epithelioid and plump spindle cells which are highly pleomorphic with nuclear hyperchromasia. There is prominent stromal inflammatory response to the invading tumor with destruction of the bladder wall. (Hematoxylin & eosin, original magnification $\times 100$).

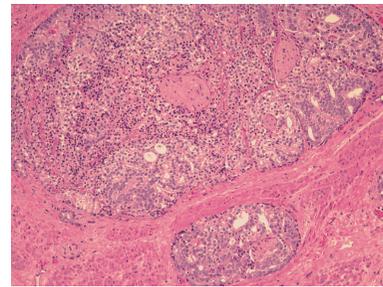


FIGURE 2: Prostatic adenocarcinoma: The large nodules of tumor cells, invading the stroma, are arranged mainly as sheets of cells and fused acini. Prominent perineural invasion is identified. (Hematoxylin & eosin, original magnification $\times 100$).

structures were smooth-walled, filled with serosanguinous fluid, and some had ragged and nodular tan-yellow lining.

3.2. Microscopic Findings. The mass was extensively sampled, and subsequent histologic examination demonstrated a malignant mesenchymal tumor with variably cellular stroma. The cellular areas were comprised of large cells with moderate to abundant clear to eosinophilic cytoplasm, enlarged, highly pleomorphic, and vesicular nuclei with distinct nucleolus, admixed with scattered pleomorphic and bizarre multinucleated giant tumor cells (Figure 3(b)). Scattered lipoblasts were noted in the cellular areas with prominent plexiform vasculature (Figures 3(c) and 3(d)). The stroma in the hypocellular areas was sclerotic to focally myxoid with few scattered spindle cells. Also seen dispersed in the hypocellular areas were collection of glandular tumor, morphologically similar to the prostate carcinoma, lined by cuboidal to columnar cells with eosinophilic to clear cytoplasm and enlarged vesicular nuclei with prominent nucleoli (Figures 3(e), 3(f)). Tumor was also noted in the retrovesical tissue.

Immunohistochemical analysis revealed immunorepression of cytokeratin AE1/AE3 (Monoclonal Mouse, DAKO, 1:100) (Figure 4(a)), CAM5.2 (Monoclonal Mouse, BD, 1:10), focal and weak P501S (Monoclonal Mouse, DAKO,

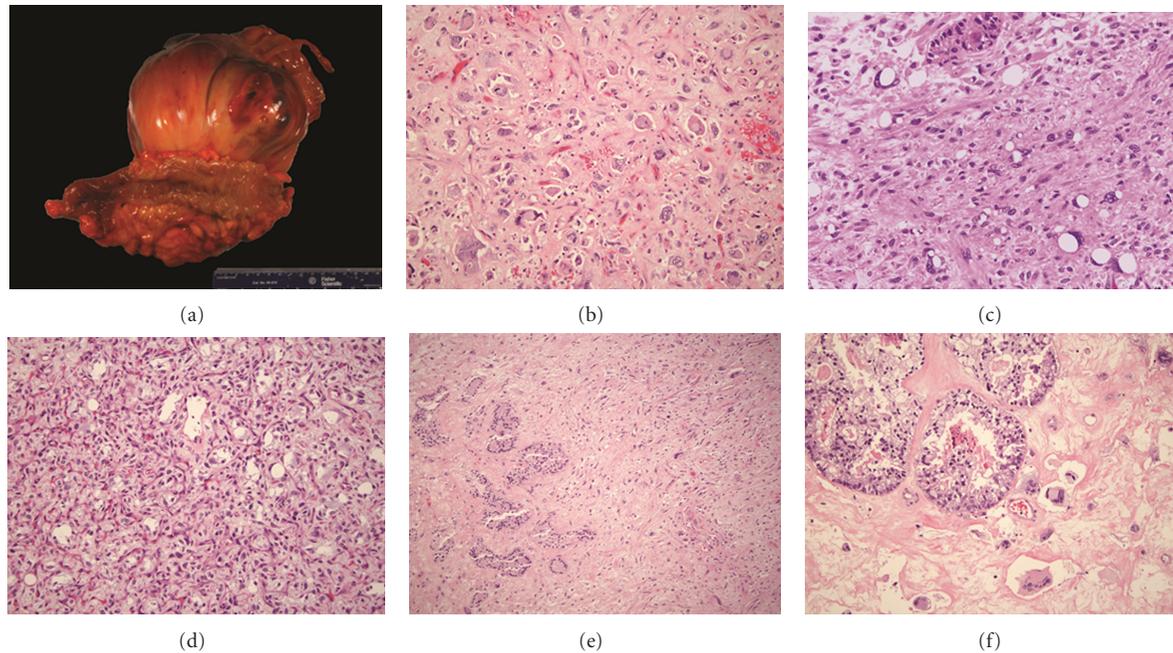


FIGURE 3: Image Plate I. (a) Gross image of the pelvic mass resection. The mass was large, well circumscribed, nonencapsulated and yellow-pink with an attached segment of large intestine. (b) Cellular area of the tumor, comprised of large cells with enlarged, highly pleomorphic, and vesicular nuclei with distinct nucleoli and abundant, clear to eosinophilic cytoplasm, with scattered bizarre multinucleated tumor giant cells. The stroma appears hyalinized with delicate branching capillary network. (Hematoxylin & eosin, original magnification $\times 200$.) (c) Scattered lipoblasts in a variably cellular background. (Hematoxylin & eosin, original magnification $\times 400$.) (d) Highly cellular area of tumor showing plexiform network of delicate capillaries, admixed tumor cells, and rare lipoblast. (Hematoxylin & eosin, original magnification $\times 200$.) (e) Glandular tumor aggregate comprised of variably sized acini (left), scattered in a hyalinized stroma. Interface with the liposarcoma component (right) is clearly seen. (Hematoxylin & eosin, original magnification $\times 40$.) (f) Close-up view of the prostatic carcinoma showing well formed acini and inspissated eosinophilic luminal secretion. The acini are intricately admixed with the bizarre tumor giant cells of the liposarcoma in a myxoid to hyalinized stroma.

1:40), and androgen receptor (AR) (Monoclonal Mouse, DAKO, 1:100) in the glandular component. The myxoid and pleomorphic component was strongly positive for vimentin (Monoclonal Mouse, Ventana, Prediluted) (Figure 4(b)) and negative for several cytokeratin markers, S-100 (Polyclonal rabbit, DAKO, 1:500), HMB45 (Monoclonal Mouse, Ventana, Prediluted), Melan-A (Monoclonal Mouse, DAKO, 1:100), demsin (Monoclonal Mouse, Ventana, Prediluted), smooth muscle actin (SMA) (Monoclonal Mouse, Cell Marque, Prediluted), myogenin (Monoclonal Mouse, Cell Marque, Prediluted), and CD117 (Polyclonal rabbit, DAKO, 1:100). CD34 (Monoclonal Mouse, Ventana, Prediluted) highlighted the prominent plexiform vasculature (Figure 4(c)).

Florescence in situ hybridization (FISH) analysis, performed using dual-color break-apart probe for *CHOP* (*CHOP* dual-color break-apart probe, Abbott Molecular, Des Plaines, Ill) gene (Figure 4(d)), was positive for the translocation pattern and negative for *MDM2* amplification (Figure 4(e)) (*MDM2* clones RP11-450G15, RP11-775J10 (*CHORI*)/CEP12 Abbott Molecular, Des Plaines, Ill). FISH analysis using dual-color double-fusion probes for *TMPRSS2* (clone RP11-24A11 (*CHORI*, Oakland, Calif)) and *ERG* (clone RP11-35C4 (*CHORI*), Oakland, Calif), demonstrated the fusion transcript in the focus harboring prostatic adenocarcinoma (Figure 4(f)).

The patient is alive at 7 months followup, but has a rising PSA from 6.1 to 8.6 ng/mL. Magnetic resonance imaging revealed a 4.2 cm left pelvic cavity mass concerning for recurrent/residual tumor.

4. Discussion

Collision tumors represent an uncommon coexistence of tumors with distinct morphology and biology. These tumors occurring in the pelvic cavity are rare with only 8 cases being reported till date [2–5, 7, 9, 12, 22] (Table 1) of which prostate adenocarcinoma was the most frequent component tumor. There are 4 reported cases with soft-tissue tumor as one of the components (two leiomyosarcoma [3, 9], one leiomyoma [22], and one liposarcoma [7]). Only 3 cases of collision tumors with liposarcoma as one of the components have been reported [1, 7, 24]. The collision tumor of prostate adenocarcinoma and liposarcoma has been documented previously as a single case report in 1978 by Juhasz and Kiss, the current case being the second one reported till date.

In our current case, the patient had past history of a urothelial and a prostate carcinoma, followed by the liposarcoma (Table 2). Each epithelial tumor was treated surgically followed by chemotherapy and radiation. There was an approximate three-year interval between occurrence

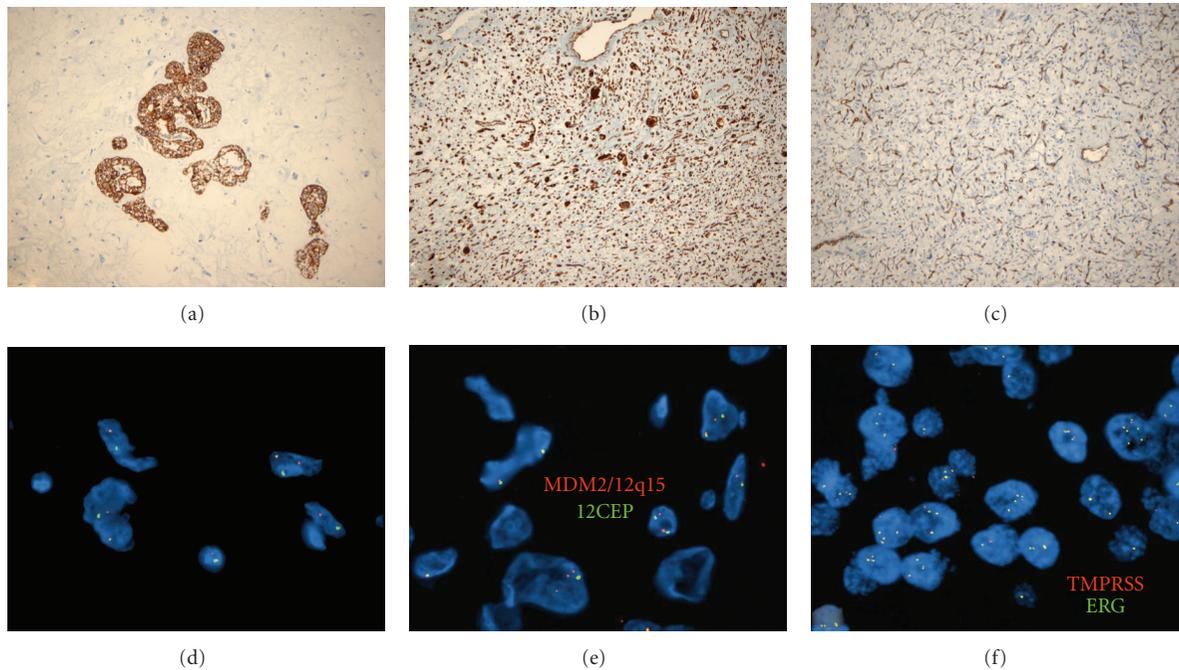


FIGURE 4: Image Plate II. (a) Malignant prostatic acini are strongly positive for AE1/AE3. The spindle cells are negative. (Original magnification $\times 100$.) (b) Strong expression of vimentin in the liposarcoma component. (Original magnification $\times 40$.) (c) CD34 highlights the plexiform network of capillaries in the tumor. (Original magnification $\times 40$.) (d) FISH analysis, dual-color break-apart probe for CHOP is positive for the translocation, represented by a cell with one yellow signal (normal), one red (part of CHOP gene), and one green signal (part of CHOP gene). (e) FISH analysis, amplification probe for MDM2, is negative (not amplified). (f) FISH analysis, dual-color fusion probes for TMPRSS (red) and ERG (green), is positive, represented by cells with at least one fused yellow signal (red + green).

TABLE 1: List of collision tumors in the pelvis.

Case no.	Source	Tumor 1	Tumor 2	Site
(1)	Juhasz and Kiss/1978 [7]	Liposarcoma	Pr ACa	Right seminal vesicle
(2)	Erler/1978 [22]	Pleomorphic carcinoma	Leiomyoma	True pelvis
(3)	Palma et al./1983 [9]	Leiomyosarcoma	Pr ACa	Prostate
(4)	Oda et al./1984 [23]	TCC bladder	Colon ACa	Vesicocolic fistula
(5)	Paveli et al./2000 [3]	Rectal leiomyosarcoma	Pr ACa	Prostate & rectum
(6)	Roh et al./2006 [2]	Rectal ACa	Gastric ACa	Rectum
(7)	Brahmania et al./2007 [4]	Colon ACa	Granulosa cell tumor, ovary	Pelvic mass
(8)	Gheith et al./2009 [5]	Myelolipoma	CLL/SLL	Pelvic mass

PrACa: prostatic adenocarcinoma, ACa: adenocarcinoma, TCC: transitional cell carcinoma, CLL/SLL: chronic lymphocytic leukemia/small lymphocytic lymphoma.

of the tumors, each of which was extensively studied, and diagnosis confirmed by immunohistochemistry.

It is not certain whether the liposarcoma had been present during the initial or subsequent surgeries, since retroperitoneal sarcomas, especially liposarcomas, can manifest when they attain large size, but in this patient, each epithelial malignancy was staged appropriately, and at diagnosis, no other intra-abdominal masses were detected. It is possible that the liposarcoma was smaller and differentiated at inception and had a growth spurt when dedifferentiation occurred.

Pathological examination revealed a liposarcoma with embedded prostatic adenocarcinoma. The diagnosis in

this case was challenging due to paucity of characteristic lipoblasts in the high-grade sarcoma and some morphologic similarity with the patient's prior bladder tumor, thus necessitating the use of ancillary tests, especially FISH, for the liposarcoma-related gene targets.

Collision metastasis to pelvic lymph nodes is a rare event and most frequently involved by prostate adenocarcinoma in combination with colonic adenocarcinoma and bladder carcinoma [15–20]. In this case, we describe that prostatic adenocarcinoma was scattered throughout the liposarcoma, which suggests that this best represents a tumor (prostate adenocarcinoma) to tumor (liposarcoma) metastasis, since no residual lymph node architecture was found.

TABLE 2: Sequence of events.

Year	Site	Morphology	IHC	FISH	Diagnosis	Treatment
5/2003	Urinary bladder diverticulum	Biphasic (epithelial and spindle cells) tumor with high-grade cytologic features. ALI-, PNI-, perivesical soft-tissue invasion	panCK+, CK20+, Vimentin-, CD68-, desmin-, SMA-	NA	Sarcomatoid urothelial carcinoma	Partial cystectomy, gemcitabine and carboplatin (4 cycles), intravesical BCG
10/2006	Prostate	Prostate adenocarcinoma, Gleason's Score 4+4=8, ALI+, PNI+, ECE+, Seminal vesicle involvement.	NA	NA	High-grade prostatic adenocarcinoma	Radical prostatectomy and radiotherapy
4/2010	Pelvic mass	Myxoid liposarcoma with pleomorphic areas, characteristic lipoblasts and hyalinized stroma mingled with well-differentiated prostatic adenocarcinoma	Liposarcoma: vimentin+, S100-, SMA-, desmin-, HMB45-, melan A-, myogenin-, CD117- PCa: AE1/AE3+, CAM5.2+, weak P501S+, AR+	Liposarcoma: <i>CHOP</i> +, <i>MDM2</i> - PCa: <i>TMPRSS2</i> - <i>ERG</i> +	Dedifferentiated myxoid liposarcoma with embedded prostatic adenocarcinoma	Tumor resection, en bloc sigmoid colectomy, and ureteroneocystostomy

ALI: angiolymphatic invasion, AR: androgen receptor, CK: Cytokeratin, ECE: extracapsular extension, FISH: fluorescence in situ hybridization, IHC: immunohistochemistry, PCa: prostatic adenocarcinoma; PNI: perineural invasion, SMA: smooth muscle actin.

Use of immunohistochemical markers helped in establishing the diagnosis of prostate adenocarcinoma based on immunoreactivity for pancytokeratin, CAM5.2, P501S, and AR. The sarcomatous component expressed vimentin only and was negative for an entire battery of immunostains.

Determination of loss of heterozygosity (LOH) and polymerase chain reaction (PCR) can be used to help differentiate the components in a tumor to tumor metastasis [2, 3, 5]. We used dual-color FISH to confirm diagnosis of liposarcoma, because of the presence of pleomorphic areas rather than the round cell component generally associated with high-grade myxoid liposarcoma. *CHOP* gene, mapped to the long arm of chromosome (12q23) is also known as *DDIT3*, encodes a member of the CCAAT/enhancer-binding protein (C/EBP) family of transcription factors which is implicated in adipogenesis and erythropoiesis. The *FUS* gene, present on chromosome 16, encodes for an RNA-binding protein [25, 26]. Chromosomal translocation *t*(12;16)(q13;p11) leads to fusion of 5' end of *FUS* gene (exons 1–5, promoter region) and complete coding region of *CHOP* which causes stable expression of the fusion protein leading to tumor formation [26, 27]. This translocation is characteristic of myxoid liposarcomas and seen in 95% of the cases. Three types of fusion transcripts have been reported based on the length of the promoter portion of *FUS* gene, type II being the most common [26]. The remainder 5% of myxoid liposarcomas have another translocation, *t*(12;22)(q13;q12), involving *CHOP* and *EWSR1* genes [26]. FISH analysis using *CHOP* dual-color, break-apart probe has been reported to be the most efficient and sensitive method for diagnosis of myxoid liposarcomas [25]. In our case, the above FISH analysis helped in the diagnosis of dedifferentiated liposarcoma and

also ruled out the possibility of metastasis from prior bladder sarcomatoid carcinoma.

MDM2 gene region amplification, characterized by the presence of giant marker/ring chromosome 12 containing *MDM2* sequences, is seen in atypical lipomatous tumor/well-differentiated liposarcoma and dedifferentiated liposarcomas [28]. However, the specificity of the *MDM2* FISH analysis is lower in the later case, as other pleomorphic sarcomas may also show *MDM2* amplification [25, 26]. In the current case FISH for *MDM2* analysis was negative.

ERG, a member of the *ETG* family of transcription factors, functions in conjunction with other transcription factors in regulation of many cellular functions, proliferation, differentiation, oncogenesis, and angiogenesis [29]. *TMPRSS2* is predominantly expressed in the luminal epithelial cells of prostatic acini. Both the genes are located 3 Mb apart on the long arm of chromosome 21 (q22.2-22.3). Interstitial deletion of the intervening intronic DNA in this region is the most common mechanism for formation of the *TMPRSS2-ERG* fusion transcript, which is seen in about half of prostate cancers [29–31]. This rearrangement can be detected either using FISH or reverse transcriptase PCR (RT-PCR). We performed FISH analysis using dual-fusion probes for *TMPRSS2* and *ERG*, which demonstrated the fusion transcript in the foci of prostatic adenocarcinoma. Although prostate carcinomas associated with this gene rearrangement have been reported to have a poor clinical outcome and decreased overall survival, the data available till date is still controversial [29].

The underlying pathobiology of collision tumors is still uncertain; however, various hypotheses have been postulated [1] as follows.

- (1) Occurrence of two different tumor types in one site is coincidental.
- (2) A single carcinogenic stimulus alters the environment of the region conducive for occurrence of two different tumors.
- (3) Presence of first tumors alters the microenvironment favorable for development of the second tumor.

In this multiple cancer-prone individual with no history of familial cancers, several intriguing possibilities exist. (a) Most of the high-grade retroperitoneal sarcomas are dedifferentiated liposarcomas that are frequently de novo or have dedifferentiated from a preexisting low-grade liposarcoma; the latter is most likely a temporal event [26, 32, 33]. Liposarcomas for practical purposes do not arise from pre-existing lipomas [26]. Radiation-induced dedifferentiation in liposarcomas is unlikely and has not been reported for prostate carcinomas also. (b) All three neoplasms in the current case must have been synchronous tumors that became clinically manifested at different time frames. (c) In majority of tumor to tumor metastases, the recipient is usually a richly vascular tumor as argued in previous reports [34, 35]. Myxoid liposarcoma are also richly vascular and contain distinct plexiform vasculature and, therefore, can be considered as a recipient of the prostate adenocarcinoma since no residual lymph node architecture was found.

In conclusion we have documented a rare case of prostate adenocarcinoma that most likely metastasized to a liposarcoma and grew undetected in a large dedifferentiated or pleomorphic myxoid liposarcoma. It is also of interest that this *CHOP FUS*-positive poorly differentiated myxoid liposarcoma did not contain the round cell component associated with myxoid liposarcoma, but this has been described to occur in some myxoid liposarcomas [36].

The use of morphological features, immunohistochemistry, and appropriate cytogenetic tests helped in reaching a correct diagnosis. The emphasis is on awareness amongst clinician, and pathologists of existence of such a collision tumor and role of extensive tumor sampling and appropriate use of ancillary tests to arrive at a diagnosis.

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