Table 1 (*continued*)

Adjuvant chemotherapy	Administered: 79% (15/19)
	Not Administered: 21% (4/19)
Type chemotherapy	BEP: 87% (13/15)
	VIP: 13% (2/15)
	None: 21% (4/19)
Recurrence/Progression (after initial treatment, including adjuvant chemotherapy)	53% (10/19)
Site of recurrence/progression	Retroperitoneum: 70% (7/10)
	Lung: 20% (2/10)
	Mediastinum: 30% (3/10)
	Liver: 10% (1/10)
	Adrenal: 10% (1/10)
Clinical follow-up	
Time to recurrence	2-87 months (range), 12 months (median)
Alive at last follow up	84% (16/19)
Dead at last follow up	16% (3/19)

Conclusions: Selected patients with DISC and absence of concurrent metastases kept under surveillance after radical orchiectomy did not experience recurrences/progression in extratesticular sites. This is incompatible with the natural history of untreated CSIII disease. Therefore, DISC does not in and of itself merit pM1a stage, as this practice may result in overtreatment. Instead, we recommend that isolated DISC (i.e., DISC in otherwise CSI patients) be specifically mentioned in pathology reports and considered a risk factor for retroperitoneal recurrences until more data are generated.

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790 | Molecular Characterization of Mixed Epithelial and Stromal Tumor of the Kidney

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Background: Mixed epithelial and stromal tumor (MEST), including the purely cystic subtype, adult cystic nephroma (CN), is an uncommon benign renal neoplasm that occurs mostly in middle-aged women. Prior studies have shown that adult MEST lack the characteristic molecular findings of pediatric CN and congenital mesoblastic nephroma. To date, comprehensive molecular genetic investigation of MEST has not been performed; its pathogenesis and any recurrent genomic alterations remain unknown.

Design: Archival cases of MEST were retrieved. Available clinicopathologic features were recorded. All available hematoxylin and eosin (H&E) and immunohistochemistry (IHC) slides were re-reviewed. Representative formalin fixed paraffin embedded tissue blocks were selected for targeted DNA next generation sequencing (NGS) panel.

Results: Fourteen cases of MEST were identified. All arose in female patients (median 49 y) and showed variable admixtures of bland epithelial glands, tubules, and cysts within an ovarian-type spindle cell stroma (Figure 1). No features of malignancy were identified. Of 13 cases with follow-up, all are without recurrence. Additional clinicopathologic features are listed in Table 1. Relative whole-chromosome gains of chromosome 12 were identified in 5/14 (35.7%) MESTs based on NGS (N=4/13) and cytogenetic analysis (N=1). Additional non-recurrent copy changes were observed in 4 cases, including 1 case without chromosome 12 gain (Figure 2). No clinically actionable alterations were identified, and there were no alterations in renal neoplasia related genes (e.g. *VHL*, *FH*, *SDHA/B*, *BAP1*, *NF2*, *TCEB1/ELOC*, *TFEB*, *TFE3*, *DICER1*, *MTOR*, *TSC1*, *TSC2*).

Case	Age	Sex	Laterality, Location	Gross Size (cm)	Follow Up Period (months)	Disease Status	Chromosome 12 alteration
1	73	F	L , interpolar	17.1	26	NED	Gain
2	51	F	R , interpolar	4.5	51	NED	None
3	82	F	R, inferior	20.0	77	NED	Gain
4	65	F	R, superior	8.3	4	NED	Gain
5	40	F	L, interpolar	3.6	99	NED	None
6	47	F	R, interpolar	3.3	60	NED	None
7	54	F	L, interpolar	7.2	172	NED	None
8	46	F	R, unspecified	5.4	NA	Unknown	Gain
9	37	F	R, superior	8.5	72	NED	None
10	35	F	R, superior	10.5	48	NED	None
11	40	F	R, superior	4.0	48	NED	None
12	42	F	R, inferior	5.0	24	NED	None
13	57	F	R, superior	5.5	144	NED	None
14	60	F	R, superior	7.5	231	NED	Trisomy 12

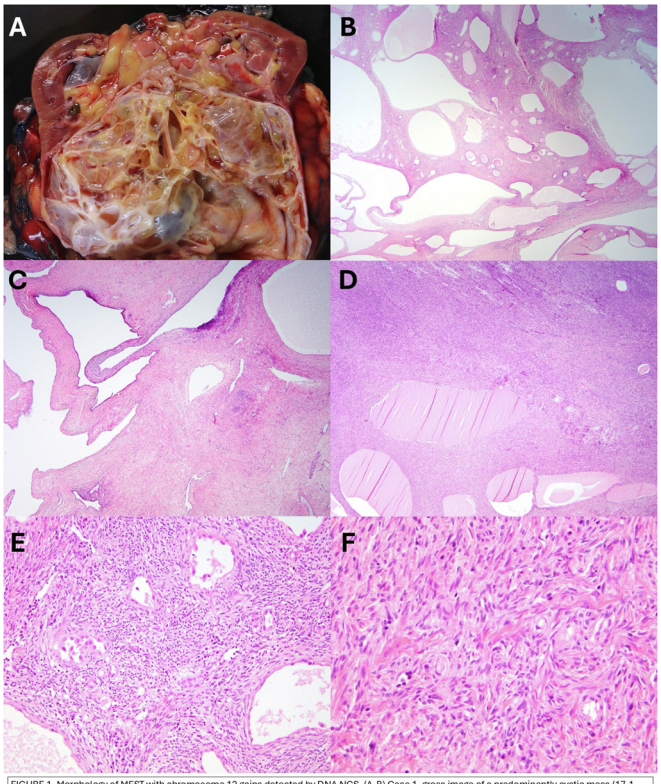


FIGURE 1. Morphology of MEST with chromosome 12 gains detected by DNA NGS. (A-B) Case 1, gross image of a predominantly cystic mass (17.1 cm) arising from the kidney parenchyma, and low power view of characteristic biphasic morphology with variably sized cysts and tubules within an ovarian-type stroma. (C-D) Case 3 and Case 4 showed more extensive, variably cellular stromal components. (E-F) Case 8, cysts and tubules with flat to hobnail epithelial lining and stroma with prominent collagen.

Figure 1 - 790.

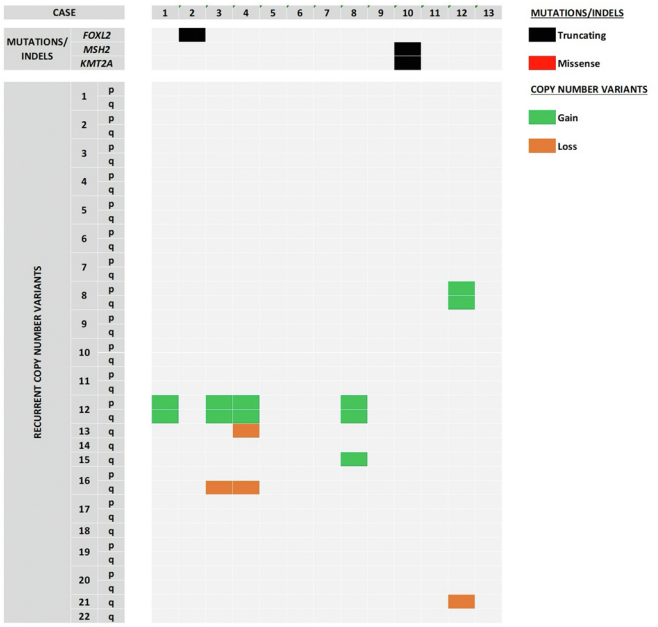


Figure 2. Molecular features of mixed epithelial and stromal tumor (MEST). Previous cytogenetic analysis performed on case 14 (not shown) identified trisomy 12.

Figure 2 - 790.

Conclusions: This is the first evidence of recurrent alterations in MEST. Partial or complete gains involving chromosome 12 is not specific, and is described in a range of human neoplasia, including testicular germ cell tumors associated with germ cell neoplasia in situ (isochromosome 12p), a subset of chronic lymphocytic leukemias (trisomy 12), and multiple sarcoma subtypes (gains/amplifications of 12q12-q15). Perhaps most relevant to MEST, trisomy 12 is the most common alteration seen in ovarian fibromas, and multiple arm and whole-chromosome level gains, including chromosome 12, are characteristic of testicular sex-cord stromal tumors. Lack of significant genomic findings in 6 MESTs may be artifactual, given the relatively low tumor content in these cystic lesions. Nevertheless, copy number variations in MEST is supportive of a neoplastic process.

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791 | Granulomatous Prostatitis as a Cause of Elevated PI-RADS Scores on Multiparametric MRI: Insights from a Single Institution Cohort

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Background: Granulomatous inflammation of the prostate (GP) can often mimic prostate cancer on multiparametric MRI. GP frequently leads to elevated PI-RADS scores suggestive of clinically significant disease, resulting in unnecessary biopsies, and management challenges. While intravesical Bacillus Calmette-Guerin (BCG) therapy can accentuate MRI findings, elevated PI-RADS scores also occur in GP without BCG. Herein, we correlated radiologic and histopathologic findings in a contemporary cohort of patients with GP.

Design: Our institutional cohort was interrogated to identify patients diagnosed with GP on prostate biopsy between 2019 and 2025 with paired MRI evaluation. Patients with any evidence of concurrent prostate cancer were excluded. This cohort was retrospectively reviewed for age, PSA, imaging year, highest PI-RADS score, percentage of biopsy parts involved by GP (PPI), and history of BCG therapy. Descriptive statistics summarized the cohort.

Results: Fifty-seven patients met inclusion criteria. Median age was 66 years (range 41–80). Median PSA was 6.1 ng/mL (range 0.1-26.4); median number of biopsy parts evaluated was 13.5 (range 2-17), and the median PPI for the cohort was 33% (range 6%-100%). PI-RADS distribution was: 2 (n=7, 12 %), 3 (n=12, 21%), 4 (n=14, 25%), and 5 (n=24, 42%). When comparing patients with low (19 patients, 33%) versus high (38 patients, 66%) PI-RADS scores, the latter category had a higher median PSA (4.8 ng/mL vs 6.4 ng/mL; p=0.26) and median PPI (16% vs 45%; p=0.01). Among BCG-treated patients (n=19, 33%), 10 (52%) had PI-RADS 4–5 lesions with a median PPI of 56% and a median PSA of 7.0 ng/mL, whereas non-BCG high PI-RADS cases (n=28, 49%) had a lower median PPI of 39% (p=0.32) and a lower median PSA of 5.6 ng/mL (p=0.77). Notably, 74% of high PI-RADS lesions occurred in patients without BCG exposure, supporting that GP alone can elevate PI-RADS scores. One patient had sarcoidosis, presenting with a PI-RADS 5 lesion, PPI of 93%, and PSA of 5.5 ng/mL.

Conclusions: GP can produce a broad range of PI-RADS scores, from high scores that mimic clinically significant prostate cancer to lower scores with limited zonal involvement and modest PSA elevations. PI-RADS elevation may be further accentuated by BCG