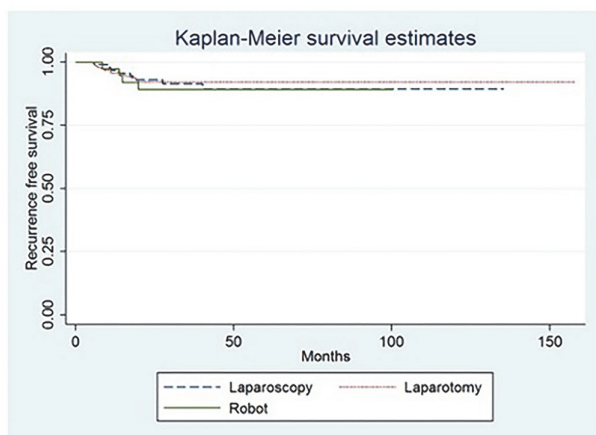
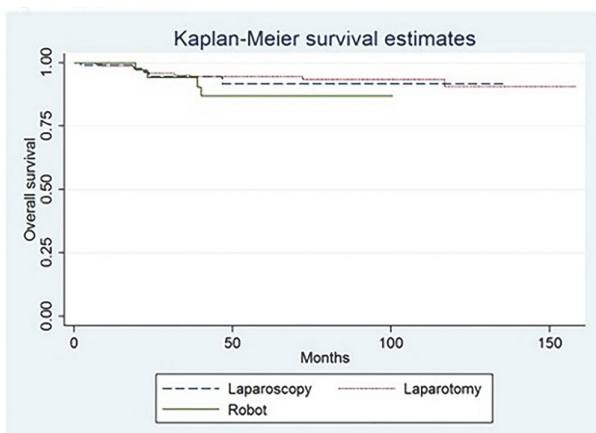


the statistical differences in the survival curves were estimated with the log-rank test.

Results A total of 334 cases were reviewed. Baseline characteristics were similar, despite a higher rate of sentinel lymph node mapping in the laparoscopy group (5% - laparotomy vs. 42% - laparoscopy vs. 21% - robot, $p < 0.001$, table 1). The total number of dissected lymph nodes was higher in the laparotomy group, compared with the laparoscopic and robotic groups ($p < 0.0001$, table 1); however, the positive lymph node rate did not differ between groups (8.6% - laparotomy vs. 3.8% - laparoscopy vs. 11.9% - robot, $p = 0.15$, table 1). The recurrence-free survival and overall survival also did not differ between groups (log-rank test, $p = 0.85$ and 0.50 , respectively, figures 1 and 2).



Abstract EP129/#919 Figure 1 Recurrence free survival



Abstract EP129/#919 Figure 2 Overall survival

Conclusion/Implications Less numbers of lymph nodes were dissected in the laparoscopy and robot groups, compared with laparotomy. However, recurrence-free survival and overall survival seem similar between the laparotomy, laparoscopy and robot surgical groups in women with uterine-confined endometrial cancer.

EP130/#706

IGFBP2 REGULATES GLUCOSE METABOLISM REPROGRAMMING THROUGH PKM2 TO PROMOTE ENDOMETRIAL ADENOCARCINOMA PROGRESSION

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Introduction Endometrial cancer (EC) is one of the most common malignant tumors in the female reproductive system. The incidence and mortality of EC have been increasing in the past 40 years. As an important member of the insulin-like growth factor (IGF) family, IGF binding protein 2 (IGFBP2) is an important molecular target in many cancers, also plays a pivotal role in metabolic diseases, such as obesity and diabetes. However, whether IGFBP2 in EC can affect the metabolism of tumor cells and participate in the metabolic reprogramming of endometrial adenocarcinoma cells and consequently affect tumorigenesis and progression, these questions are still unclear and the specific mechanisms have not been elucidated.

Methods Cancer and paracancer tissue specimens were collected from 80 patients with EC, and the expression of IGFBP2, PKM2 and glycolytic enzymes in the tissues were examined. The expression of IGFBP2 in EC cells was altered to detect the effect of IGFBP2 on cellular glycolysis, and the expression of key enzymes of intracellular glycolysis was examined.

Results IGFBP2 and PKM2 were highly expressed in tumor tissues of EC patients and correlated with tumor stage and differentiation. After knockdown of IGFBP2 expression in EC cells, cell proliferation capacity was significantly reduced, tumorigenic capacity in vivo was decreased, cellular glycolytic function, glucose uptake, lactate production and ATP production were significantly reduced, the expression levels of various key enzymes of glucose metabolism were significantly reduced, and the nuclear plasma ratio of PKM2 was decreased.

Conclusion/Implications We found IGFBP2 regulates glucose metabolism reprogramming through PKM2 to promote endometrial adenocarcinoma progression.

EP131/#702

CLINICAL CHARACTERISTICS AND PROGNOSIS ANALYSIS OF UTERINE SARCOMA: MULTI-CENTER RETROSPECTIVE STUDY

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Introduction To examine the histopathological features and treatment modalities in patients with uterine sarcoma according to subgroups (uterine leiomyosarcoma, low grade/high grade endometrial stromal sarcoma, adenosarcoma, undifferentiated uterine sarcoma) and to determine the factors affecting mortality rates.

Methods We retrospectively evaluated patients diagnosed with uterine sarcoma in our eight multicenter institutions between March 2012 and December 2021. We compared the clinicopathological characteristics and treatment modalities of the

subgroups and investigated the factors affecting mortality rates using logistic regression analysis.

Results In the entire US group, the rate of 5-year OS was 51.2% and the rate of DFS was 39.9%. There was no difference between the subgroups in terms of age, body mass index, menopausal status, comorbidity, presenting complaint, primary diagnosis, surgical treatment protocol, adnexal and lymph node involvement and tumor size ($p > 0.01$). High NLR was significantly associated with worse DFS ($p = 0.007$) and OS ($p = 0.039$). Advanced stage ($p = 0.017$) and high mitotic index ($p = 0.036$) retained their prognostic significance for DFS. Other clinical variables, including PLR, CA125, and lactate dehydrogenase (LDH) failed to show significant impact.

Conclusion/Implications Uterine sarcoma is an aggressive cancer with poorer survival in this specific cohort than has been described in other contemporary cohorts. Despite their different histopathological features, subgroups do not have distinctive features such as demographic features, presenting complaints, primary diagnosis and surgical treatment protocols. Therefore, prospective randomized clinical studies should be performed to evaluate the prognostic influencing factor and the value of adjuvant treatments for patients with uterine sarcoma.

Abstract EP131/#702 Table 1

N (%) [±]	ALL sarcomas [±] (N=99) [±]	LMS [±] (N=57) [±]	ESS [±] (N=29) [±]	UUS [±] (N=7) [±]	Adenosarcoma [±] (N=6) [±]	P [±]	[±]
Age (years) [±]							
≥60 [±]	21 (21.2%) [±]	13 (22.8%) [±]	4 (13.8%) [±]	1 (14.3%) [±]	3 (50.0%) [±]	0.224 [±]	
<60 [±]	78 (78.8%) [±]	44 (77.2%) [±]	25 (86.2%) [±]	6 (85.7%) [±]	3 (50.0%) [±]		
BMI (kg/m2) [±]							
Normal 18.5-22.9 [±]	41 (41.4%) [±]	22 (38.6%) [±]	14 (48.3%) [±]	2 (28.6%) [±]	3 (50.0%) [±]	0.718 [±]	
Overweight-obese ≥23 [±]	58 (58.6%) [±]	35 (61.4%) [±]	15 (51.7%) [±]	5 (71.4%) [±]	3 (50.0%) [±]		
History of hypertension [±]							
Yes [±]	25 (25.3%) [±]	16 (28.1%) [±]	4 (13.8%) [±]	3 (42.9%) [±]	2 (33.3%) [±]	0.238 [±]	
No [±]	74 (74.7%) [±]	41 (71.9%) [±]	25 (86.2%) [±]	4 (57.1%) [±]	4 (66.7%) [±]		
History of diabetes [±]							
Yes [±]	14 (14.1%) [±]	8 (14.0%) [±]	2 (6.9%) [±]	2 (28.6%) [±]	2 (33.3%) [±]	0.158 [±]	
No [±]	85 (85.9%) [±]	49 (86.0%) [±]	27 (93.1%) [±]	5 (71.4%) [±]	4 (66.7%) [±]		
Stage [±]							
I [±]	73 (73.7%) [±]	37 (64.9%) [±]	23 (79.3%) [±]	7 (100.0%) [±]	6 (100%) [±]	0.834 [±]	
II [±]	7 (7.1%) [±]	5 (8.8%) [±]	2 (6.9%) [±]	0 (0.0%) [±]	0 (0.0%) [±]		
III [±]	8 (8.1%) [±]	7 (12.3%) [±]	1 (3.4%) [±]	0 (0.0%) [±]	0 (0.0%) [±]		
IV [±]	11 (11.1%) [±]	8 (14.0%) [±]	3 (10.3%) [±]	0 (0.0%) [±]	0 (0.0%) [±]		
Tumor size (cm) [±]							
≥10 [±]	36 (36.4%) [±]	24 (42.1%) [±]	6 (20.7%) [±]	3 (42.9%) [±]	3 (50.0%) [±]	0.189 [±]	
<10 [±]	63 (63.6%) [±]	33 (57.9%) [±]	23 (79.3%) [±]	4 (57.1%) [±]	3 (50.0%) [±]		
Mitotic index (MF/10HPF) [±]							
≥15 [±]	44 (44.4%) [±]	33 (57.9%) [±]	7 (24.1%) [±]	2 (28.6%) [±]	2 (33.3%) [±]	0.015 [±]	
<15 [±]	55 (55.6%) [±]	24 (42.1%) [±]	22 (75.9%) [±]	5 (71.4%) [±]	4 (66.7%) [±]		
Nuclear atypia [±]							
≥severe [±]	20 (20.2%) [±]	16 (28.1%) [±]	2 (6.9%) [±]	2 (28.6%) [±]	0 (0.0%) [±]	0.050 [±]	
<severe [±]	79 (79.8%) [±]	41 (71.9%) [±]	27 (93.1%) [±]	5 (71.4%) [±]	6 (100.0%) [±]		
Vascular invasion [±]							
Yes [±]	24 (24.2%) [±]	12 (21.1%) [±]	7 (24.1%) [±]	4 (57.1%) [±]	1 (16.7%) [±]	0.220 [±]	
No [±]	75 (75.8%) [±]	45 (78.9%) [±]	22 (75.9%) [±]	3 (42.9%) [±]	5 (83.3%) [±]		
Tumor necrosis [±]							
Yes [±]	74 (74.7%) [±]	50 (87.7%) [±]	13 (44.8%) [±]	6 (85.7%) [±]	5 (83.3%) [±]	<0.001 [±]	
No [±]	25 (25.3%) [±]	7 (12.3%) [±]	16 (55.2%) [±]	1 (14.3%) [±]	1 (16.7%) [±]		
Lymphadenectomy [±]							
Yes [±]	29 (29.3%) [±]	21 (36.8%) [±]	5 (17.2%) [±]	1 (14.3%) [±]	2 (33.3%) [±]	0.219 [±]	
No [±]	70 (70.7%) [±]	36 (63.2%) [±]	24 (82.8%) [±]	6 (85.7%) [±]	4 (66.7%) [±]		
Recurrence [±]							
Yes [±]	30 (30.3%) [±]	21 (36.8%) [±]	6 (20.7%) [±]	1 (14.3%) [±]	2 (33.3%) [±]	0.373 [±]	
No [±]	69 (69.7%) [±]	36 (63.2%) [±]	23 (79.3%) [±]	6 (85.7%) [±]	4 (66.7%) [±]		

* p value between different histologic type. Abbreviations: LMS: leiomyosarcoma, ESS: endometrial stromal sarcoma, UUS: undifferentiated uterine sarcoma[±]