Prognostic factors for overall survival in malignant ureteral obstruction

J. Bradley Mason, MD,[1]* Michael Creswell, MD,[2]* Jillian Egan, MD,[1] Christopher Dall, MD,[1] Tamir Sholklapper, MD,[2] Lan Anh Galloway, MD,[2] Joanna Orzel, MD,[2] Harry Lee, MD,[2] Sameer Desale, MS,[3] Lambros Stamatakis, MD[2,4]

[1]Department of Urology, MedStar Georgetown University Hospital, Washington, DC, USA
[2]Georgetown University School of Medicine, Washington, DC, USA
[3]Biostatistics and Biomedical Informatics Department, MedStar Health Research Institute, Hyattsville, Maryland, USA
[4]Department of Urology, MedStar Washington Hospital Center, Washington, DC, USA

MASON JB, CRESWELL M, EGAN J, DALL C, SHOLKLAPPER T, GALLOWAY LA, ORZEL J, LEE H, DESALE S, STAMATAKIS L. Prognostic factors for overall survival in malignant ureteral obstruction. *Can J Urol* 2022;29(3):11162-11169.

Introduction: *To identify prognostic factors for overall survival (OS) in patients with malignant ureteral obstruction (MUO) from gynecologic malignancy (GM), with the goal of improving patient selection for urinary diversion.*

Materials and methods: *Retrospective review of 126 patients with MUO from GM at two academic centers from 2011-2019. Factors related to OS identified by Cox regression proportional hazard model. In patients with incomplete survival data (n = 30), hospice was used as a surrogate for death. Multivariate models and receivers operating characteristics (ROC) curves were created for hemoglobin and albumin values.*

Results: *Overall median survival was 6.2 months. On univariate analysis, age at diagnosis, Charlson Comorbidity Index (CCI) ≥ 8, advanced clinical stage, ascites, pleural effusion, albumin, and hemoglobin were associated with poor OS. OS was higher for those receiving ureteral stenting as compared with no intervention. There was no survival difference based on hydronephrosis grade, stent failure (SF), or creatinine at the time of intervention. On multivariate analysis, albumin < 2.85 g/dL and hemoglobin < 9.6 g/dL were predictive of poor OS.*

Conclusions: *OS in patients with MUO due to GM is poor. Several prognostic factors for poor survival including low serum albumin and hemoglobin were identified. Ureteral stenting was associated with improved OS compared to observation, but selection bias likely contributed to this result. Additional studies are needed to clarify this finding. These data can be utilized to counsel patients regarding outcomes after urinary diversion in the setting of MUO and perhaps avoid additional procedures in some of these patients who will not derive meaningful benefit.*

Key Words: ureteral obstruction, urinary diversion, hydronephrosis, malignant obstruction, ureteral stent, percutaneous nephrostomy

Introduction

In patients with malignant ureteral obstruction (MUO), overall prognosis is poor with median overall survival (OS) reported as < 180 days from the time of diagnosis.[1-3] Gynecologic malignancies (GM) in particular account for approximately 15%-30% of all non-urologic cases of MUO.[2,3] Urinary diversion with ureteral stenting or percutaneous nephrostomy (PCN) has been used to preserve renal function for additional treatments (i.e. chemotherapy) or for palliative purposes if symptomatic, however the optimal treatment for MUO is not yet fully understood. Despite relieving the obstruction, early reports suggest that urinary diversion may reduce quality of life in some of these patients due to bothersome urinary symptoms from stents and increased self-care

Accepted for publication April 2022

*co-first authors

Address correspondence to Dr. Lambros Stamatakis, 110 Irving St. NW, Ste 315, Washington, DC 20010 USA

needs for PCNs.[4,5] Although more recent data puts this assertion into question,[6,7] patients that undergo urinary diversion with either stent or PCN still have a poor prognosis and high complication rate.[3] This is especially true for patients who fail ureteral stenting and ultimately undergo PCN placement. Therefore, it is necessary to identify patients who will benefit from urinary diversion prior to intervention in order to counsel patients appropriately and better select the subset who will benefit. Recent studies from Ishioka et al, Cordiero et al, and Leinert et al have reported prognostic factors for OS in patients with MUO,[1,2,8] but overall data are sparse. These factors include events related to malignant dissemination (metastases, ascites, pleural effusion), poor ECOG status, mild grade hydronephrosis, low serum albumin levels, and hyponatremia.[1,2,8] In this study, we attempted to further identify prognostic factors for OS in patients with MUO from GM.

Material and methods

A retrospective review was performed of patients with GM and MUO. These patients were 18 years or older and treated at one of two academic medical centers between 2011 and 2019. Several factors were collected to study possible prognostic factors for OS in this patient population including demographics, TNM stage, presence of ascites, presence of pleural effusion, comorbid conditions, grade of hydronephrosis, type of intervention for hydronephrosis, laboratory values at time of intervention, stent failure (SF), laboratory values at time of SF, date of death, date of hospice, and laboratory values at death or hospice. Hydronephrosis was identified on abdominal imaging and were graded based on radiologist interpretation. Treatments for MUO included 44 (36.1%) patients where no intervention occurred, 50 (41.0%) underwent retrograde ureteral stenting, and 28 (23.0%) who underwent percutaneous nephrostomy tube placement. Patients were fully consented by the treating physician before treatment, which included a conversation of risks, benefits, and alternatives. Treatment decisions were patient-driven and considered both physician and patient factors. Fifty-three (42.1%) patients had bilateral hydronephrosis and were analyzed on a single patient basis rather than a renal unit basis. In 30 patients (23.8%), hospice data was used as a surrogate for death.

Descriptive statistics was presented using mean and standard deviation (SD) for continuous variables and using median and interquartile range (IQR) for non-normal continuous variables. Comparison of continuous variables between groups was conducted using a two-sample t test or Kruskal Wallis test if variables were not normally distributed. Data for categorical variables was presented using frequency and percentages. Comparison of percentage distribution between two groups was conducted using Fisher's exact test. Cox proportional hazard ratios (HR) were calculated to compare time to death/hospice. Two multivariate models were built and included inputs from existing literature and our univariate analysis that were predictive of OS.[1,2,8] Albumin and hemoglobin were significantly correlated and thus were included in two separate models. Additionally, age at diagnosis and clinical stage were not included in the models because they exhibited multicollinearity with Charlson Comorbidity Index (CCI). Model one included: intervention type, CCI, grade of hydronephrosis and hemoglobin at time of intervention. Model two was identical to model one but included albumin at time of intervention rather than hemoglobin.

In survival analysis, median survival time is defined as time taken for survival probability to reach 0.5. In some subgroups, survival probabilities do not reach 0.5 and median survival is not available. Variables Creatinine (Cr) and white blood cell count (WBC) were skewed, hence log transformed variables were used in the cox regression. Receiver operating characteristics (ROC) curves were plotted for albumin and hemoglobin (Hgb) and the best cut off point was determined by using Youden method, which maximizes the sum of sensitivity and specificity.

Results

A total of 126 patients with MUO and GM were assessed. Eighty-six patients (68.3%) identified as African American, 24 (19.0%) as Caucasian, and 16 (12.7%) as other race. Mean age at diagnosis of malignancy was 59.9 (SD 13.2) years old. The majority of patients had advanced disease (64 [53.3%] stage IV; 32 [26.7%] stage III), Table 1. At the time of chart review 37 patients had died and 30 initiated hospice with incomplete survival data. OS for the entire cohort was 36.5% with a median survival of 6.2 months. OS was 29.1% for African American patients and 52.5% for non-African American patients. At the time of MUO diagnosis, 29 (24.8%) patients had mild hydronephrosis, 60 (51.3%) patients had moderate hydronephrosis, and 28 (23.9%) patients had severe hydronephrosis. Fifty (41.0%) patients received a stent, 28 (23.0%) underwent PCN placement, and 36 (29.5%) received no intervention. Eleven patients (25.0%) who received stents experienced

TABLE 1. **Patient characteristics**

Variables	Overall 126	Alive 46	Died/hospice 67	p value
Race (%)				0.044
African American	86 (68.3)	25 (54.3)	51 (76.1)	
Other	16 (12.7)	8 (17.4)	5 (7.5)	
Caucasian	24 (19.0)	13 (28.3)	11 (16.4)	
Type of intervention (%)				0.969
Stent	50 (41.0)	20 (45.5)	28 (42.4)	
PCN	28 (23.0)	11 (25.0)	17 (25.8)	
No intervention	44 (36.1)	13 (29.5)	21 (31.8)	
Age group for CCI (%)				0.150
0	23 (18.4)	12 (26.1)	8 (12.1)	
1	34 (27.2)	14 (30.4)	16 (24.2)	
2	43 (34.4)	14 (30.4)	27 (40.9)	
3	25 (20.0)	6 (13.0)	15 (22.7)	
CCI total (%)				0.002
< 8	49 (39.5)	25 (55.6)	17 (25.4)	
≥ 8	75 (60.5)	20 (44.4)	50 (74.6)	
Localized vs. metastatic (%)				0.001
Localized	34 (28.3)	21 (47.7)	11 (16.7)	
Metastatic	86 (71.7)	23 (52.3)	55 (83.3)	
Ascites (%)				0.054
Yes	27 (23.1)	5 (11.4)	18 (28.1)	
No	90 (76.9)	39 (88.6)	46 (71.9)	
Pleural effusion (%)				0.093
Yes	18 (15.5)	3 (6.8)	12 (19.0)	
No	98 (84.5)	41 (93.2)	51 (81.0)	
Stent failure (%)				0.733
Yes	11 (25.0)	5 (29.4)	6 (24.0)	
No	33 (75.0)	12 (70.6)	19 (76.0)	
Grade of hydronephrosis (%)				0.277
Mild	29 (24.8)	12 (30.0)	15 (23.1)	
Moderate	60 (51.3)	17 (42.5)	38 (58.5)	
Severe	28 (23.9)	11 (27.5)	12 (18.5)	
Clinical stage at hydronephrosis (%)				< 0.001
I	18 (15.0)	14 (31.8)	4 (6.1)	
II	6 (5.0)	1 (2.3)	3 (4.5)	
III	32 (26.7)	14 (31.8)	15 (22.7)	
IV	64 (53.3)	15 (34.1)	44 (66.7)	
Age at cancer diagnosis (mean (SD))	59.94 (13.24)	56.13 (12.70)	62.61 (12.65)	0.009
Albumin at hydronephrosis (mean (SD))	2.93 (0.84)	3.30 (0.68)	2.70 (0.83)	0.003
Creatinine at hydronephrosis (median [IQR])	1.40 [0.83, 2.51]	1.31 [0.79, 2.70]	1.56 [0.89, 2.54]	0.331
WBC at hydronephrosis (median [IQR])	8.60 [6.00, 12.90]	9.40 [6.40, 13.50]	8.50 [5.90, 12.35]	0.523
Hemoglobin at hydronephrosis (mean (SD))	9.70 (1.86)	10.45 (1.82)	9.30 (1.78)	0.006

PCN = percutaneous nephrostomy; CCI = Charlson Comorbidity Index; SD= standard deviation; IQR= interquartile range; WBC = white blood cell

TABLE 2. **Hazard ratios and median survival**

Variables	n (%)	Median	HR (95% CI)	p value
Race				
African American	86 (68.3%)	5.5	1.644 (0.856, 3.157)	0.135
Other	16 (12.7%)	NA	0.858 (0.298, 2.47)	0.776
Caucasian	24 (19%)	41.5		
Type of intervention				
Stent	50 (41%)	17.6	0.665 (0.377, 1.172)	0.158
PCN	28 (23%)	5.1	0.89 (0.469, 1.689)	0.722
No intervention	44 (36.1%)	2.9		
Age group for CCI				
1	34 (27.2%)	15.2	1.794 (0.766, 4.201)	0.178
2	43 (34.4%)	5.1	2.473 (1.117, 5.476)	0.026
3	25 (20%)	3.7	2.923 (1.233, 6.93)	0.015
0	23 (18.4%)	NA		
CCI total				
≥ 8	75 (60.5%)	3.7	3.046 (1.738, 5.339)	< 0.001
< 8	49 (39.5%)	NA		
Metastatic				
Metastatic	86 (71.7%)	4	3.605 (1.868, 6.959)	< 0.001
Localized	34 (28.3%)	NA		
Ascites				
Yes	27 (23.1%)	1.5	2.194 (1.264, 3.807)	0.005
No	90 (76.9%)	12		
Pleural effusion				
Yes	18 (15.5%)	1.5	2.27 (1.202, 4.287)	0.011
No	98 (84.5%)	12		
Grade of hydronephrosis				
Moderate	60 (51.3%)	4.9	1.208 (0.664, 2.198)	0.535
Severe	28 (23.9%)	41.5	0.703 (0.329, 1.504)	0.364
Mild	29 (24.8%)	5.6		
Clinical stage at hydronephrosis				
II	6 (5%)	42.9	2.839 (0.635, 12.696)	0.172
III	32 (26.7%)	15.2	3.399 (1.126, 10.256)	0.03
IV	64 (53.3%)	2.9	6.554 (2.333, 18.407)	< 0.001
I	18 (15%)	NA		

Variable	Mean	SD/IQR	HR (95% CI)	p value
Age at cancer diagnosis	59.94	13.24	1.035 (1.014, 1.056)	< 0.001
Albumin at hydronephrosis	2.93	0.84	0.346 (0.238, 0.503)	< 0.001
Creatinine at hydronephrosis	1.40	0.83, 2.51	1.336 (0.962, 1.855)*	0.084
WBC at hydronephrosis	8.60	6.00, 12.90	1.414 (0.81, 2.469)*	0.223
Hemoglobin at hydronephrosis	9.70	1.86	0.797 (0.692, 0.918)	0.002

PCN = percutaneous nephrostomy; CCI = Charlson Comorbidity Index;
SD= standard deviation; IQR= interquartile range;
WBC = white blood cell

a stent failure, of which 9 (81.8%) subsequently received PCN placement. Of the patients who received PCN, 3 (10.7%) became blocked or dislodged requiring replacement. Laboratory values including albumin, serum Cr, WBC, and Hgb were reported from values drawn at MUO diagnosis. Overall laboratory values showed a mean (SD) albumin level of 2.93 g/dL (0.84) and Hgb of 9.70 g/dL (1.86). WBC and Cr were not normally distributed and had a median of 8.60 k/uL (IQR 6.00, 12.90) and 1.40 mg/dL (IQR 0.83, 2.51), respectively.

The group of patients who died or went on hospice were more likely to be older (p = 0.009), African American (p = 0.044), have a CCI ≥ 8 (p = 0.002), and have metastases (p < 0.001) compared with the alive group. Differences in ascites and pleural effusion trended toward significance (p = 0.054, p = 0.093 respectively). Patients who died or were on hospice were more likely to have lower serum albumin (p = 0.003) and hemoglobin (p = 0.006) levels at the time of intervention compared with patients who survived. There were no differences between groups in terms of the grade of hydronephrosis (p = 0.277), type of intervention (p = 0.472), or stent failure (p = 0.733). There were also no differences in Cr and WBC levels at diagnosis of MUO.

Predictors of OS were analyzed by patient characteristics using univariate Cox proportional hazard ratios with a 95% confidence interval (CI), Table 2. Patients with locally advanced and metastatic cancers

TABLE 3. **Multivariate models for overall survival. Model one (A) utilized hemoglobin at time of hydronephrosis; n = 81, number of events = 54, c-statistic = 0.71. Model two (B) used albumin as the laboratory variable; n = 74, number of events = 50, c-statistic = 0.77**

Model one (A) **Variable**	**HR**	**95% CI lower**	**95% CI upper**	**p value**
Type of intervention: stent vs. no intervention	0.462	0.239	0.893	0.022
Type of intervention: PCN vs. no intervention	0.974	0.463	2.049	0.944
CCI total: ≥ 8 vs. < 8	1.717	0.917	3.214	0.091
Hydronephrosis grade: moderate vs. mild	0.758	0.371	1.548	0.446
Hydronephrosis grade: severe vs. mild	0.426	0.161	1.126	0.085
Hemoglobin at hydronephrosis	0.760	0.644	0.896	0.001
Model two (B) **Variable**	**HR**	**95% CI lower**	**95% CI upper**	**p value**
Type of intervention: stent vs. no intervention	0.634	0.314	1.278	0.203
Type of intervention: PCN vs. no intervention	1.847	0.82	4.158	0.139
CCI total: ≥ 8 vs. < 8	1.651	0.843	3.234	0.144
Hydronephrosis grade: moderate vs. mild	1.003	0.481	2.091	0.993
Hydronephrosis grade: severe vs. mild	0.692	0.270	1.772	0.443
Albumin at hydronephrosis	0.312	0.206	0.471	< 0.001

HR = hazard ratio; PCN = percutaneous nephrostomy; CCI = Charlson Comorbidity Index

fared worse than those with local cancers. Patients with stage III disease had a HR of 3.399 [1.126, 10.256] (p = 0.03) and those with stage IV disease had a HR of 6.554 [2.333, 18.407] (p < 0.001). Similarly, events related to metastatic disease were predictive of poor OS. Patients with malignant ascites were 2.194 [1.264, 3.807] (p = 0.005) times more likely to die or go on hospice compared with those without ascites. Patients with pleural effusion had a HR of 2.27 [1.202, 4.287] (p = 0.011) relative to those without pleural effusion. Charlson Comorbidity Index was also a predictor of death/hospice in this cohort. Patients with CCI ≥ 8 had a HR of 3.046 [1.738, 5.339] (p < 0.001) compared to the reference group, CCI < 8. The grade of hydronephrosis at diagnosis was not predictive of poor outcomes.

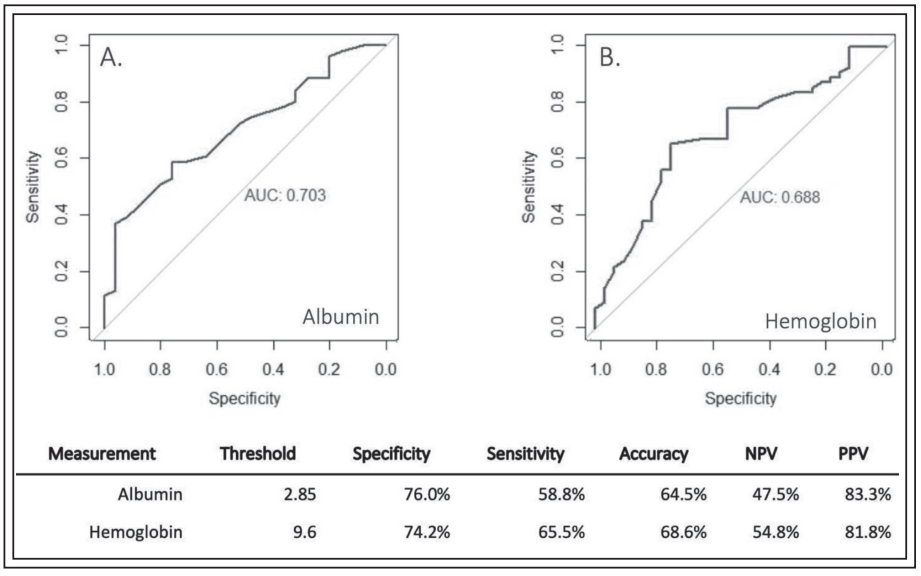
Two multivariate models were constructed using the following inputs: type of intervention, CCI total, grade of hydronephrosis at diagnosis, and either hemoglobin or albumin at time of intervention, Table 3. The model including hemoglobin was moderately predictive of death/hospice (c-statistic = 0.71), with type of intervention (stent versus no intervention) (p = 0.022), and hemoglobin (p = 0.001) as statistically significant inputs. The model including albumin showed moderately predictive power (c-statistic = 0.77), however, only albumin at time of intervention was statistically significant (p = <0.001).

Compared to patients without intervention, patients with stents were less likely to die or enter hospice (HR: 0.554 [0.309, 0.994]) (p = 0.048). Outcomes in patients who received PCN versus no intervention were no different in terms of survival (HR: 0.743 [0.386, 1.431]) (p = 0.374). Compared to the overall cohort, stented patients had less severe disease with lower rates of metastasis, malignant ascites, and pleural effusion at 65.9%, 16.7%, 9.5%, respectively. Mean (SD) albumin levels were also increased at 3.19 g/dL (0.77).

On laboratory analysis, higher albumin and Hgb levels were protective against death or hospice (HR: 0.346 [0.238, 0.503]) (p < 0.001), (HR: 0.797 [0.692, 0.918]) (p = 0.002), respectively. Albumin and hemoglobin were chosen to analyze further using ROC curves, Figure 1. The albumin area under the curve (AUC) showed moderate discriminatory power between the alive and the died/hospice groups (AUC: 0.703). A threshold of 2.85 g/dL produced a specificity of 76.0% and positive predictive value of 83.3%. On the hemoglobin ROC, hemoglobin demonstrated an AUC of 0.688. At a threshold of 9.60 g/dL, specificity was 74.2% and positive predictive value was 81.8%.

Discussion

This multicenter retrospective cohort study was performed to validate previous findings and expand on factors related to OS in patients with MUO from GM. Our results suggest several prognostic factors for shorter survival time in patients with MUO from GM, including serum albumin < 2.85 g/dL, hemoglobin < 9.6 g/dL, high CCI, and events related to malignancy (metastases, pleural effusion, ascites, advanced clinical stage). Much of this corroborates previous work,[1,2,8] though it does highlight new factors of low hemoglobin and high CCI as predictors of poor survival. Low serum albumin has been studied previously and our results are consistent with the Ishioka et al cutoff value of < 3 g/dL.[1] Hemoglobin levels < 9.6 g/dL as a predictor of OS are a novel finding in our study. A recent meta-analysis has shown similar findings with low hemoglobin associated with worse survival



Measurement	Threshold	Specificity	Sensitivity	Accuracy	NPV	PPV
Albumin	2.85	76.0%	58.8%	64.5%	47.5%	83.3%
Hemoglobin	9.6	74.2%	65.5%	68.6%	54.8%	81.8%

Figure 1. Receiver Operating Characteristic Curves. Albumin **(A)** and hemoglobin **(B)** analyzed with regard to overall survival. Albumin and hemoglobin thresholds reported in gram per deciliter.
NPV = negative predictive value; PPV = positive predictive value.

in patients with bladder cancer, however, no cutoff value could be obtained from current data.[9] No such data has been found for hemoglobin and MUO to our knowledge. The relationship of CCI to OS is likely related to the health of our overall cohort. A CCI ≥ 8 has a 0% estimated 10-year OS, therefore it is not surprising that patients with higher scores will have a lower OS in a shorter period of time.[10] The age of diagnosis and clinical stage are similarly expected to be associated with poor OS as these are nested components of the CCI.

The grade of hydronephrosis in our data did not significantly differ between the alive and deceased/hospice group (p = 0.277). Previous data on this point in particular has been conflicting.[11,12] This demonstrates the need for additional studies on symptomatic patients, including the use of functional studies (i.e. nuclear renal scans) to better assess renal obstruction and determine proper indications for urinary diversion with MUO.

Data are also conflicting on the predictive utility of preoperative serum creatinine values and OS.[1,2,8,13,14] Our study demonstrated no such association between preoperative Cr and survival. It is unclear from current data if improvement in renal function (as determined by serum Cr) after intervention is associated with improved OS. To further complicate this point, we observed that placement of a stent was protective (HR 0.554, 95% CI 0.309-0.994) and median survival was 17.6 months after stent placement compared to 5.1 months after PCN placement and 2.9 months without intervention. However, these findings may be subject to selection bias. In our study, patients who received stents were generally healthier than the overall cohort with lower rates of metastatic disease, events related to metastasis, and higher albumin levels, each of which are independently predictive of survival.[1,2,8] Stented patients may also undergo additional interventions such as chemotherapy or radiation, which could plausibly extend their life. Patients who receive retrograde stenting and subsequent treatment have an improved prognosis than those who are stented but decline additional treatment.[13,14] Further studies are needed to elucidate the independent prognostic utility of stenting in patients with MUO.

This study has several limitations. First, the investigators were limited to analyze only existing data in patient charts due to the retrospective study design. Notably, missing data of interest included standardized patient symptoms, quality of life surveys, factors influencing choice of intervention, and renal function data such as MAG3 scans pre- and post-intervention. Additionally, the start of hospice was used as a surrogate endpoint for patient death when date of death was unavaialble. This likely underestimated OS as patients may continue to live for weeks or months while on hospice. Our cohort was also exclusively female patients with GM which limits the generalizability of our results. Future studies in other malignancies that create MUO are needed to help expand our findings and create better selection criteria for intervention in such patients.

In addition to the prognostic factors identified in this study, future research should correlate patients' symptoms and functional renal imaging (i.e. MAG3 scan) with OS and endpoints. Further studies are needed to investigate the prognostic value of stenting in order to inform care and should also incorporate Quality-Adjusted Life Years and the presence of symptoms related to MUO on presentation to measure the burden of disease faced by patients with MUO. Additionally, future studies are needed to confirm the predictive value of low hemoglobin for poor OS that was demonstrated in our study.

Conclusions

In patients with advanced GM and MUO, OS is poor. This study identified several prognostic factors of poor survival including serum albumin < 2.85 g/dL, hemoglobin < 9.6 g/dL, CCI ≥ 8, and events related to malignancy. Low hemoglobin level as a predictor of poor survival was a novel finding in this study. Ureteral stenting was associated with improved OS, however, was subject to selection bias. These prognostic factors can be used to risk stratify patients and counsel women with MUO and GM on expected outcomes. In particular, studies like these can help support the urologist in avoiding intervention in those patients who will likely not derive meaningful benefit from urinary diversion. Additional studies are needed to clarify these findings and develop firmer guidelines in this often-encountered clinical scenario. □

References

1. Ishioka J, Kageyama Y, Inoue M, Higashi Y, Kihara K. Prognostic model for predicting survival after palliative urinary diversion for ureteral obstruction: analysis of 140 cases. *J Urol* 2008;180(2):618-621.
2. Cordeiro MD, Coelho RF, Chade DC et al. A prognostic model for survival after palliative urinary diversion for malignant ureteric obstruction: a prospective study of 208 patients. *BJU Int* 2016;117:266-271.

3. Shekarriz B, Shekarriz H, Upadhyay J et al. Outcome of palliative urinary diversion in the treatment of advanced malignancies. *Cancer* 1999;85(4):998-1003.
4. Zadra JA, Jewett MA, Keresteci AG et al. Nonoperative urinary diversion for malignant ureteral obstruction. *Cancer* 1987;60(6):1353-1357.
5. Wilson JR, Urwin GH, Stower MJ. The role of percutaneous nephrostomy in malignant ureteric obstruction. *Ann R Coll Surg Engl* 2005;87(1):21-24.
6. Joshi HB, Adams S, Obadeyi OO, Rao PN. Nephrostomy tube or "JJ" ureteric stent in ureteric obstruction: assessment of patient perspectives using quality-of-life survey and utility analysis. *Eur Urol* 2001;39(6):695-701.
7. Monsky WL, Molloy C, Jin B et al. Quality-of-life assessment after palliative interventions to manage malignant ureteral obstruction. *Cardiovasc Intervent Radiol* 2013;36(5):1355-1363.
8. Lienert A, Ing A, Mark S. Prognostic factors in malignant ureteric obstruction. *BJU Int* 2009;104(7):938-941.
9. Mori K, Miura N, Mostafaei H et al. Prognostic value of preoperative hematologic biomarkers in urothelial carcinoma of the bladder treated with radical cystectomy: a systematic review and meta-analysis. *Int J Clin Oncol* 2020;25(8):1459-1474.
10. Charlson ME, Pompei P, Ales KL, MacKenzie CR. A new method of classifying prognostic comorbidity in longitudinal studies: development and validation. *J Chronic Dis* 1987;40(5):373-383.
11. Matsuura H, Arase S, Hori Y. Ureteral stents for malignant extrinsic ureteral obstruction: outcomes and factors predicting stent failure. *Int J Clin Oncol* 2019;24(3):306-312.
12. Alawneh A, Tuqan W, Innabi A et al. Clinical factors associated with a short survival time after percutaneous nephrostomy for ureteric obstruction in cancer patients: an updated model. *J Pain Symptom Manage* 2016;51(2):255-261.
13. Izumi K, Mizokami A, Maeda Y, Koh E, Namiki M. Current outcome of patients with ureteral stents for the management of malignant ureteral obstruction. *J Urol* 2011;185(2):556-561.
14. Izumi K, Shima T, Shigehara K et al. A novel risk classification score for malignant ureteral obstruction: a multicenter prospective validation study. *Sci Rep* 2021;11(1):1-9.