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Low magnesium levels an important new prognostic parameter can be overlooked in patients with Fournier's gangrene: a multicentric study

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Abstract
Introduction We evaluated low magnesium levels and three different scoring systems including the Fournier's Gangrene Severity Index (FGSI), the Uludag Fournier's Gangrene Severity Index (UFGSI), and the Charlson Comorbidity Index (CCI) for predicting mortality in a multicentric, large patient population with FG.
Methods The medical records of 99 FG patients who were treated and followed up in different clinics were reviewed. The biochemical, hematological, and bacteriological results from the admission evaluation were recorded. The CCI, FGSI, and UFGSI were evaluated and stratified by survival.
Results The results were evaluated for the following patients: the survivors ($n = 82$) and the nonsurvivors ($n = 17$). The magnesium level for the survivors and nonsurvivors was 2.09 ± 0.28 and 1.68 ± 0.23, respectively (p 0.004). The admission FGSI, UFGSI, and CCI scores were significantly higher in nonsurvivors (p 0.001, p 0.001, $p < 0.001$, respectively). The receiver operating characteristics analysis revealed that the UFGSI was more powerful than the FGSI. The hypomagnesemia, low hemoglobin and hematocrit, low albumin and HCO_3 levels; high alkaline phosphatase; and the high heart and respiratory rates, an FGSI >9, rectal involvement, and a high CCI were associated with a worse prognosis.
Conclusion Low magnesium levels might be an important parameter for a worse FG prognosis. Monitoring the serum magnesium levels might have prognostic and therapeutic implications in patients with FG. High CCI, FGSI, and UFGSI scores might be associated with a worse prognosis in patients with FG. The UFGSI might be more powerful scoring system than the FGSI.

Keywords Fournier's Gangrene · Magnesium · Index · Prognosis

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Introduction

Fournier's gangrene (FG), a life-threatening necrotizing fasciitis of the male genitourinary tract, was first described as a pathology localized to the scrotum that might extend through the fascial layer to the groin, perineum, and even the abdominal wall [1–3].

Mortality has been reported in different series to range from 16 to 40 % [3–8]. Identification of prognostic factors might help to determine patients with a high risk of mortality.

Various scoring systems have been used to predict the severity of FG and patient survival. The Fournier's Gangrene Severity Index (FGSI) was developed to assign a

Table 1 Clinical and comorbidity admission parameters

	Survivors ($n = 82$) Mean ± SD	Nonsurvivors ($n = 17$) Mean ± SD	p
Age	60.90 ± 13.08	68.05 ± 12.58	0.052
Hearth rate	83.93 ± 13.41	118.00 ± 17.52	**<0.001**
Respiratory rate	20.79 ± 2.18	26.50 ± 5.70	**0.003**
Temperature	37.17 ± 1.18	37.48 ± 1.45	0.543
Mean total body surface area (TBSA)	2.58 ± 1.86 %	4.43 ± 2.30	**0.005**
Microbial culture (yes–no) (%)	72 (87.8 %)/10 (12.2 %)	17 (100 %)/–	0.427
DM (yes–no) (%)	43 (52.4 %)/39 (47.6 %)	8 (47.0 %)/9 (53 %)	0.382
HT (yes–no) (%)	22 (26.8 %)/60 (73.2 %)	6 (35.3%)/11 (64.7%)	0.485
CRF (yes–no) (%)	6 (7.3 %)/76 (92.7 %)	3 (17.6 %)/14 (82.4 %)	0.554

Boldface type indicates that the differences are significant

DM diabetes mellitus, *HT* hypertension, *CRF* chronic renal failure, *SD* standard deviation

numerical score that describes the severity of the disease. The scoring system is based on the physiological and metabolic status [2]. Uludag Fournier's Gangrene Severity Index (UFGSI) adds age and dissemination of the disease scores to the FGSI [6]. The Charlson Comorbidity Index (CCI) is a general scoring system for comorbid conditions described by Charlson et al. [7]. Additionally, our previous study demonstrated that low magnesium (Mg) levels might be used as a new parameter indicating a worse prognosis [8].

In this study, we reviewed 99 FG patients to identify the prognostic factors and evaluate low serum Mg levels and the three scoring systems for predicting mortality in patients with FG.

Materials and methods

The medical records of 99 patients with Fournier's gangrene, who were treated and followed up between December 2006 and December 2014 in various clinics, were reviewed. The collected data comprised the medical history, symptoms, and physical examination findings. The biochemical, hematological, and bacteriological study results at the admission evaluation, the physical examination findings, and the timing and extent of surgical debridement were recorded.

The extent of gangrene was calculated for the modified body surface area nomograms routinely used to assess the extent of burn injuries as follows: the penis, scrotum, and perineum each accounted for 1 % of the surface area and each ischiorectal fossa for 2.5 % [2].

All the patients underwent immediate aggressive debridement, with resection of all the necrotic skin, subcutaneous tissue, fascia, and muscle until viable tissue was identified.

All the patients received preoperative supportive fluid resuscitation and were treated with broad spectrum parenteral antibiotics until the culture results dictated the individualized therapy.

As the standard of care, the patients were returned to the operating room 24–48 h for repeat wound exploration and debridement, except in cases of hemodynamic instability or if the wound margins were clearly uninvolved on bedside examination. A colostomy was performed for infection of perirectal origin with anal sphincter involvement in conjunction with the general surgery team. A suprapubic cystostomy was performed in cases of periurethral origin with evidence of urinary extravasation. Wound closure and reconstruction (split thickness skin grafting, rotational flaps, and negative pressure wound therapy) were performed by a plastic and reconstructive surgery team in cases in which healthy, viable tissue as well as the clinical status allowed for reapproximation.

The FGSI scale provides a numerical score obtained from a combination of the physiological hospital admission parameters including the temperature, heart rate, respiration rate, sodium, potassium, creatinine, leukocytes, hematocrit, and bicarbonate. These nine parameters are measured on the FGSI, and the degree of deviation from normal is graded from 0 to 4. The sum of the individual values is then tallied to arrive at the FGSI score. The data were assessed according to whether the patient survived or died (Tables 1, 2).

To calculate the UFGSI, the nine parameters used in the FGSI are measured, and the degree of deviation from normal is graded from 0 to 4. One and zero points are added for the patient age of at least 60 years and less than 60 years, respectively. One, 2, and 6 points are added for disease dissemination grades of 1, 2, and 3, respectively. The sum of the individual values is then tallied to arrive at the UFGSI score.

The comorbidities were abstracted from the inpatient databases, and the CCI was calculated using 17 weighted

Table 2 Laboratory admission parameters, FGSI, UFGSI, and CCI for survivors and nonsurvivors (mean ± SD)

	Normal values		Survivors ($n = 82$) Mean ± SD	Nonsurvivors ($n = 17$) Mean ± SD	p
Hemoglobin (g dL^{-1})	12–18	Admission	11.81 ± 2.45	10.02 ± 1.59	**0.002**
Hematocrit (%)	37–52	Admission	34.93 ± 7.21	29.98 ± 4.78	**0.003**
WBC (total/$mm^3 \times 10^3$)	4.8–10.8	Admission	14.29 ± 6.73	22.03 ± 16.94	0.073
RDV		Admission	15.65 ± 2.67	19.16 ± 2.62	0.055
MPV		Admission	8.32 ± 1.84	6.52 ± 0.64	**0.049**
Albumin (g dL^{-1})	3.4–5.4	Admission	2.96 ± 0.67	2.60 ± 0.65	**0.036**
Total protein (g dL^{-1})	6.4–8.3	Admission	6.01 ± 0.97	5.75 ± 0.98	0.359
AST (IU L^{-1})	<40	Admission	28.50 ± 20.06	46.94 ± 50.49	0.536
ALT (IU L^{-1})	<40	Admission	24.01 ± 21.12	34.21 ± 39.00	0.861
ALP (IU L^{-1})	40–129	Admission	106.12 ± 56.28	173.35 ± 99.84	**0.001**
BUN (mg dL^{-1})	10–50	Admission	63.60 ± 51.87	86.22 ± 49.59	**0.028**
Creatinine (mg dL^{-1})	0.5–1.2	Admission	1.62 ± 1.61	2.33 ± 2.29	0.072
HCO_3, venous (mmol L^{-1})	22–32	Admission	22.53 ± 4.29	16.13 ± 6.56	**0.009**
Sodium (mmol L^{-1})	136–157	Admission	136.41 ± 5.54	137.17 ± 3.86	0.488
Potassium (mmol L^{-1})	3.5–5.5	Admission	4.23 ± 0.69	4.52 ± 1.04	0.169
Calcium (mg dL^{-1})	8.4–10.2	Admission	8.32 ± 0.68	7.55 ± 1.31	0.128
Chlor (mmol L^{-1})	98–110	Admission	102.17 ± 6.94	105.33 ± 7.44	0.721
Magnesium (mg dL^{-1})	1.7–2.5	Admission	2.09 ± 0.28	1.68 ± 0.23	**0.004**
UFGSI score	0–15	Admission	4.62 ± 2.90	10.00 ± 4.88	**0.001**
FGSI score	0–14	Admission	3.65 ± 2.90	8.29 ± 4.95	**0.001**
CCI		Admission	3.16 ± 2.06	5.47 ± 2.03	**<0.001**

Boldface type indicates that the differences are significant

WBC white blood cell count, *AST* aspartate transaminase, *ALT* alanine transaminase, *ALP* alkaline phosphatase, *BUN* blood urea nitrogen, HCO_3 serum bicarbonate, *FGSI* Fournier's Gangrene Severity Index, *UFGSI* Uludag Fournier's Gangrene Severity Index, *CCI* Charlson Comorbidity Index, *SD* standard deviation, *min* minimum, *max* maximum

indicators of coexisting conditions [7]. All the data were assessed according to whether the patient survived or died.

The statistical analysis was performed with the Statistical Package for Social Science for Windows (SPSS, Chicago, IL, USA) version 15.0. A comparison of the mean age and the mean extent of the body surface area involved in the necrotizing process and the admission metabolic parameters, heart rate, respiration rate, FGSI, CCI and UFGSI scores between the survivors and nonsurvivors were performed using the Mann–Whitney *U* test. The FGSI and UFGSI were compared using ROC analysis (Fig. 1).

The admission parameters in each group were compared using the Wilcoxon signed-rank test. The Chi-square test was used to compare the frequency of data such as that for accompanying diseases.

The Pearson's correlation analysis was used for the correlation between the clinical and laboratory admission parameters, age, CCI, FGSI, FGSI > 9, UFGSI, total body surface area (TBSA), rectal involvement, a diverting colostomy, and mortality (Table 3). Additionally, $p < 0.05$ was considered statistically significant.

Results

Of the 99 evaluated patients, 17 died (17.1 %) and 82 survived (82.9 %). The difference in age between the survivors (mean 60.90 ± 13.08; range 27–83 years) and nonsurvivors (mean 68.05 ± 12.58; range 51–84 years) was not significant ($p = 0.052$).

The patients were evaluated using the onset of symptoms. The first symptom had appeared in the scrotum in 80 survivors and 15 nonsurvivors and in the perineum in 2 survivors and 2 nonsurvivors.

The mean extent of the body surface area involved in the necrotizing process in those who survived and those who did not was 2.58 and 4.43 %, respectively (p 0.005). The predisposing factors were evaluated in these patients. Diabetes mellitus was found in 51 patients (51.5 %), hypertension was present in 28 patients (28.2 %), and chronic renal failure occurred in 9 patients (10 %). The comorbidities and predisposing factors were similar between the survivors and nonsurvivors. A comparison of the *clinical and comorbidity admission parameters* in the survivors and nonsurvivors is shown in Table 1.

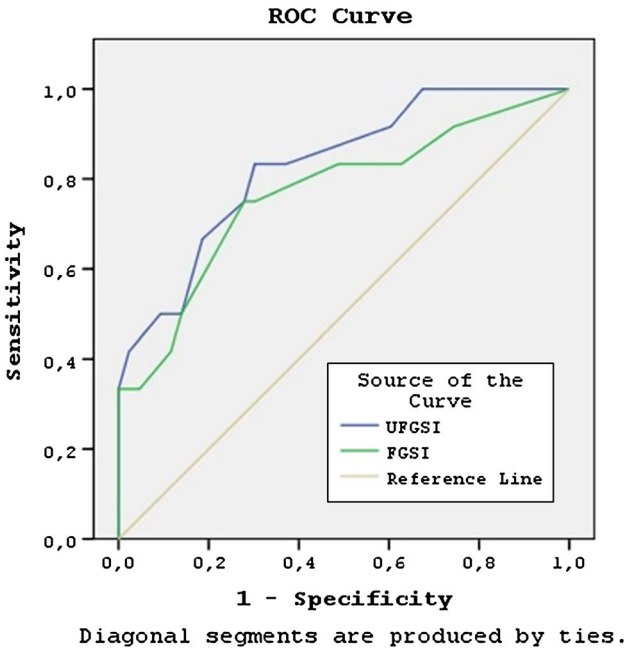

Fig. 1 Comparison of the Fournier's Gangrene Severity Index (FGSI) and Uludag Fournier's Gangrene Severity Index (UFGSI) ROC curves. Difference between areas: 0.062, SE: 0.019

Table 3 Correlation between the clinical and laboratory parameters and mortality

Variable	Pearson's correlation	*p* value*
Age	0.205	0.042
CCI	0.394	<0.001
Hemoglobin	−0.280	0.005
Hematocrit	−0.265	0.008
WBC	0.304	0.002
RDV	0.515	0.020
MPV	−0.445	0.049
Magnesium	−0.534	0.004
Calcium	−0.340	0.013
Albumin	−0.210	0.045
ALP	0.378	<0.001
HCO_3	−0.494	0.003
TBSA	0.369	0.001
UFGSI	0.553	<0.001
FGSI	0.470	<0.001
FGSI > 9	0.457	<0.001
Heart rate	0.673	<0.001
Respiratory rate	0.605	<0.001
Rectal involvement	0.640	<0.001
Diverting colostomy	0.598	0.009

CCI Charlson Comorbidity Index, *FGSI* Fournier's Gangrene Severity Index, *ALP* alkaline phosphatase, *TBSA* total body surface area

* Pearson correlation test

The median admission time was 4.3 days and was similar between the survivors and nonsurvivors.

All the patients underwent radical surgical FG debridement. The necrotizing tissues were completely removed, and abscess drainage was performed, if necessary. The pathological features were localized to the genital region in 80 survivors during surgery and had spread beyond the genital region to the umbilicus in 2 survivors and 17 nonsurvivors ($p < 0.001$) and to the rectum in 7 survivors and 10 nonsurvivors. Seven survivors and ten nonsurvivors underwent a diverting colostomy. Two orchiectomies were performed in the nonsurviving group, and a cystostomy was needed in ten patients in the nonsurviving group ($p > 0.05$).

Various organisms were cultured from necrotic tissue or pus during surgery or at the bedside. The culture results revealed a microbial infection in 89 patients (89.8 %). In ten patients (10.2 %), the wound cultures were negative. The culture results were positive for a microbial infection in 100 % of the nonsurvivors and positive in 72 (87.8 %) of the survivors (Table 1).

A polymicrobial infection was observed in 27 of 89 patients (30.3 %). The organisms most commonly isolated from the wound were *Escherichia coli* in 25 patients (28 %), enterococcus in 18 patients (20 %), staphylococcus in 16 patients (17.9 %), streptococcus in 9 patients (10.1 %), proteus in 4 patients (4.4 %), and acinetobacter in 4 patients (4.4 %). Mortality was not related to a specific isolated organism. Anaerobes were not harvested in our patients.

The etiological factors for mortality in the nonsurvivors were congestive heart insufficiency ($n = 4$), a pulmonary embolism ($n = 4$), pneumonia and acute renal failure ($n = 4$), severe septic shock, and multiple organ dysfunction syndrome ($n = 5$).

The mean admission FGSI scores for the survivors and nonsurvivors were 3.65 ± 2.90 (0–10) and 8.29 ± 4.95 [8–14], respectively (p 0.001). The mean admission UFGSI scores for the survivors and nonsurvivors were 4.62 ± 2.90 (0–10) and 10.0 ± 4.88 (0–15), respectively (p 0.001).

The following laboratory and clinical parameters differed between the survivors and nonsurvivors: the serum magnesium, blood urea nitrogen (BUN), hemoglobin, hematocrit, albumin, alkaline phosphatase (ALP) levels; the heart and respiration rates; the TBSA%; and the FGSI and UFGSI levels (Tables 1, 2).

The CCI score was 3.16 ± 2.06 in the survivors and 5.47 ± 2.03 in the nonsurvivors ($p < 0.001$).

Additionally, the hemoglobin, hematocrit, ALP, TBSA%, CCI, UFGSI, FGSI, FGSI > 9, MPV (the middle platelet volume), heart and respiratory rates, rectal involvement, colostomy diversion, and low magnesium levels were associated with a worse prognosis (Table 3).

Table 4 Comparison of ROC curves of FGSI and UFGSI

Scoring system	Area under ROC curve	SE	95 % CI	*p*
FGSI	0.767	0.085	0.600–0.935	0.001
UFGSI	0.829	0.066	0.699–0.960	0.005

Difference between areas: 0.062, SE: 0.019

ROC receiver operator characteristics, *SE* standard error, *CI* confidence interval, *FGSI* Fournier's Gangrene Severity Index, *UFGSI* Uludag Fournier's Gangrene Severity Index

The performances of the FGSI and UFGSI were compared, and the ROC analysis results are shown in Fig. 1 and Table 4.

Discussion

FG is a necrotizing fasciitis of the genital, perineal, and perianal region that leads to thrombosis of the small subcutaneous vessels and results in the development of gangrene of the overlying skin. Several studies have shown the effect of the extent of necrotizing tissue infection on a negative outcome of patients with the disease [6, 8, 9]. Some studies have suggested that the extent of the disease was not predictive of the outcome [2, 4]. In this study, the extent of the body surface area involved in the necrotizing process was significantly higher in the non-survivors (Table 1). The authors hypothesize that the extent of necrotizing tissue infection is one of the most important risk factors for mortality in patients with FG.

Despite the increasing knowledge regarding the etiology, diagnosis, treatment, and intensive care techniques in FG, the mortality rate remains high. In this study, the mortality rate was 17.1 %.

Laor et al. [2] described the FGSI, which is useful for evaluating the prognosis and stratifying the risks in FG patients; it remains an objective method of quantifying at presentation the extent of the metabolic status in patients with FG. Laor established that an FGSI score above 9 is sensitive and specific as a mortality predictor in FG patients [2]. There is a debate regarding the utility and cutoff values in the current literature. A recent published study demonstrated no associated between FGSI and mortality [10]. The author reported that the FGSI did not reflect the disease severity and treatment outcome in these patients [10]. However, this cutoff point has been validated in other small retrospective series, our previous and present studies [8, 9, 11]. We reported a high mortality rate, with a significant difference in the FGSI values (3.65 survivors vs. 8.29 non-survivors, *p* 0.001) between the two groups.

The UFGSI is a powerful scoring system combining age and disease dissemination with the FGSI score. It was described by Yilmazlar et al. [6] in 2010 as a novel scoring system that could be used for predicting mortality in patients with FG. It is also a matter of debate; there are some reports comparing FGSI and UFGSI.

Roghmann et al. [12] retrospectively compared these scoring systems, and the authors reported that the UFGSI does not seem to be more powerful than the FGSI.

Yilmazlar et al. concluded that the UFGSI scoring system was more powerful than the FGSI, which has been validated in our study (Fig. 1; Table 4). Based on this scoring system, patients with wide disease dissemination and age over 60 years are in a high-risk group. In this study, the admission UFGSI score was significantly lower in the survivors than in the non-survivors (4.6 and 10.0, *p* 0.001, respectively). Similarly, Tuncel et al. reported that the median admission UFGSI score was significantly lower in survivors than in non-survivors (4 and 7.5, respectively) [10]. Further large prospective studies are needed to compare UFGSI and FGSI in patient with FG.

Many predisposing factors have been reported in FG, including systemic diseases such as diabetes mellitus (DM), chronic renal failure, and malignancy [4, 13, 14]. Tuncel et al. [10] demonstrated that comorbid disorders, particularly DM, were related to mortality in FG. Corcoran et al. [15] did not find comorbid conditions to be significantly associated with mortality. In the previous and present study, the comorbidities were similarly distributed among the patients in both groups, and DM did not affect the outcome [8]. These findings correlated with the published data [2, 13, 14]. However, a high CCI might be associated with a worse outcome and was likely responsible for mortality. CCI might be useful for evaluating the outcomes of FG. In contrast to the findings of Corcoran et al. [15], our previous study and the present series and others have reported that a primary colorectal source [8, 16, 17] and creation of a diverting colostomy [17, 18] were associated with increased mortality.

Some studies [10, 15] have demonstrated that specific metabolic parameters such as the serum creatinine, bicarbonate, lactate, and calcium levels were important prognostic findings. When we evaluated the admission laboratory parameters, we found that those who died had greater BUN and ALP levels and lower magnesium, hematocrit, hemoglobin, and albumin levels.

The high BUN, low hematocrit and hemoglobin, increased ALP, low calcium and albumin levels reflected debilitation and were associated with mortality. These findings correlated with the classical and recent studies [2, 8–10, 15].

Serum albumin is an objective parameter that closely correlates with the critical diseases. Hypoalbuminemia has been reported as a negative prognostic factor for survival in patients with severe disease such as cancer and chronic

renal disease [19]. FG is also a severe disease, and in our study the albumin levels were significantly decreased in nonsurvivors.

In this study, we confirmed that high ALP levels were associated with mortality in FG patients. Since ALP is expressed in the liver, kidneys, intestines, bones, and leukocytes, high ALP levels may be a prognostic parameter in FG [20]. We believe that an increase in ALP itself probably reflects an increase in the extent of the body surface area involved in the necrotizing process. Prospective studies are needed to confirm the role of ALP as a prognostic factor in FG. This may become the topic of a future study.

Our previous study was the first to demonstrate the prognostic value of the serum Mg level in patients with FG [8]. Several studies have suggested that Mg plays an important protective role in the development of cardiovascular diseases, infectious diseases, and malignant neoplasia and that lower serum Mg levels are associated with vascular calcification and cardiovascular mortality among patients with end-stage renal disease [21, 22]. Our four patients had low serum Mg levels and acute renal failure, which could be explained by the impaired intestinal absorption or intracellular shift of Mg.

Many studies have shown that low Mg levels were associated with impairment of myocardial contractility [23, 24]. Mg treatment suppresses ventricular arrhythmias in acute myocardial infarction and possibly affects mortality after an infarction. The reduced arrhythmicity by Mg is closely linked to enhancement in the homogeneity of repolarization [25]. Our four patients had low Mg levels and congestive heart failure with arrhythmia.

Some studies have demonstrated that low serum Mg levels on admission are closely related to the mortality rate in critically ill patients [26]. FG is a critical disease, and greater attention should be paid to the occurrence of hypomagnesemia in FG patients.

Some studies have revealed that Mg could play a strong role in wound restoration and that Mg supplementation improves the outcome of wound healing and the postoperative quality of the recovery period [27–30]. Similarly, Mg treatment might be a promising candidate for accelerating wound healing in FG patients.

In conclusion, low magnesium levels might be a new and important parameter for a worse prognosis in FG. Monitoring the serum Mg levels might have prognostic and therapeutic implications in patients with FG. Mg supplementation in FG patients might prevent progression of the disease. This study is one of the largest series in the literature that has investigated the effect of a low Mg level on the prognosis of FG. However, the retrospective and multicentric nature of the study, major limitations are standardization of the laboratory evaluation and selection bias. This new prognostic parameter should be validated through other prospective studies and independent observations.

Compliance with ethical standards

Conflict of interest None.

Ethical standard The Ethics Committee of our faculty approved the study protocol (2015/0069).

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