Am J Clin Exp Urol 2026;14(4):97-105
www.ajceu.us /ISSN:2330-1910/AJCEU0172880

Original Article

Testicular endocrine function recovers following Fournier's gangrene

Yurong Shao[1], Marie-Therese Valovska[2], Claire C Yang[2], George E Koch[2,3], Judith C Hagedorn[2]

[1]University of Washington, School of Medicine, Seattle, WA, USA; [2]Department of Urology, University of Washington, Seattle, WA, USA; [3]Department of Urology, The Ohio State University, Columbus, OH, USA

Received March 25, 2026; Accepted July 8, 2026; Epub August 15, 2026; Published August 30, 2026

Abstract: Objectives: Between 5.6-21% of Fournier's Gangrene (FG) patients undergo orchiectomy, despite testicular viability. Arguments for this practice center around the presumed lack of functional recovery following critical illness as well as improved ease of wound closure. We aimed to study the effects of FG on the hypothalamic-pituitary-gonadal (HPG) axis and characterize hormonal recovery patterns. Methods: We retrospectively studied male FG patients at a quaternary center from July 2023-September 2024. Total testosterone (TT), free testosterone (FT), luteinizing hormone (LH), follicle-stimulating hormone (FSH), sex hormone-binding globulin (SHBG), and estradiol (E2) at admission, discharge, and follow-up (1-9 months) were examined. Two-sided Wilcoxon signed-rank tests were used to compare paired hormone levels across time points. A validation analysis using two-sided Mann-Whitney U tests was performed to compare hormone distributions between patients with complete vs. incomplete data at all three time points. Results: Of 31 FG patients, 20 had hormone measurements at multiple time points; 12 had complete data at all time points. All patients presented with castrate to near-castrate levels of TT. TT and FT increased significantly throughout recovery (both $P \leq 0.011$). E2 decreased significantly from admission to discharge and from admission to follow-up ($P = 0.014$ and $P = 0.008$, respectively). All reconstruction types showed similar recovery patterns. Conclusions: FG transiently suppresses testicular hormone production to castrate levels via the HPG axis. However, the HPG axis has the capacity to recover to physiologic ranges, with testicular steroidogenesis following suit. Without clear evidence of testicular necrosis, efforts should prioritize testicular preservation during debridement and reconstruction.

Keywords: Fournier's gangrene, hypothalamic pituitary gonadal axis, hypogonadism

Introduction

Fournier's Gangrene (FG) is a rapidly progressive necrotizing soft tissue infection of the external genitalia that primarily affects men [1, 2]. Extensive tissue necrosis can rapidly progress to sepsis or septic shock [3]. Diagnosis relies largely on clinical examination with soft tissue necrosis of the scrotal and perineal tissues being pathognomonic. Treatment requires urgent broad-spectrum antibiotic therapy combined with emergent and aggressive debridement of necrotic tissues [1]. In contemporary cohorts, the mortality rate of FG has declined to 5-10%, justifying a closer examination of survivorship outcomes [1, 4, 5]. Scrotal skin infection and the associated critical illness expose the testicles to physiologic dysregulation, while surgical debridement disrupts local thermoregulation. While testicular insult likely stems from a combination of local inflammation, systemic critical illness, and a loss of local protective soft tissue, this gonadal injury remains poorly understood.

Current literature on FG has primarily focused on identifying risk factors for mortality, improving reconstructive techniques, and optimizing the timing of surgical debridement [1, 6]. However, the hormonal function of the testes throughout the infection, surgical debridement and reconstruction, and post-hospitalization recovery has not been explored. Furthermore, no established guidelines exist for endocrine follow-up in these patients, despite the potential for significant disruption to hormonal homeostasis. Between 5.6% and 21% of FG patients ultimately undergo orchiectomy. This is

https://doi.org/10.62347/YOLG9225

most often due to concern for testicular necrosis or irreversible ischemia. However, orchiectomies are occasionally performed to facilitate wound closure or reconstruction in the setting of skin loss [7-9]. This practice remains controversial as the testicles are typically viable, even in severe infections [10]. Orchiectomy may carry long-term consequences for hormonal regulation and fertility. A better understanding of the physiological impact of FG on the hypothalamic-pituitary-gonadal (HPG) axis is therefore necessary to establish the value of testicular preservation over orchiectomy.

This study aims to characterize changes in testicular endocrine function in men with FG involving the scrotum who did not undergo orchiectomy, from hospital admission through follow-up. We hypothesized that the systemic inflammatory response associated with the severe infection would transiently suppress the HPG axis at presentation, with recovery of endocrine function over time in most patients.

Methods

Data collection

A retrospective cohort study was performed on adult male patients treated for FG at a single, high-volume center from July 2023 to September 2024. This study was reviewed and approved by the Institutional Review Board at the University of Washington and a formal waiver of informed consent was granted (approval number: STUDY00018856).

Patients were prospectively identified on admission and treated via our institutional FG clinical care pathway. All clinical data, including serum studies of interest (total testosterone (TT), free testosterone (FT), follicle-stimulating hormone (FSH), luteinizing hormone (LH), sex hormone-binding globulin (SHBG) and estradiol (E2)) and follow-up data were collected from institutional records following the completion of each patient's care.

Demographic and clinical variables included age, sex, race, body mass index (BMI), diabetes, hemoglobin A1c, hypertension, presence of coronary artery disease, and smoking history. Hospitalization details included length of stay, reconstruction type, and number of surgical debridements. Hormonal profiles were obtained at admission and discharge. Repeat hormone profiles were collected at any follow-up visit occurring within twelve months of discharge. Follow-up time points varied due to differences in patient access to care. Discharge labs were consistently collected in the morning, whereas admission and follow-up samples were collected throughout the day based on timing of presentation and outpatient visit schedules. Hormone levels were reported in the following units: total testosterone in ng/dL, free testosterone in pg/mL, LH and FSH in mIU/mL, SHBG in nmol/L and E2 in pg/mL. Laboratory reference ranges were defined per institutional standards.

Statistical analysis

The Shapiro-Wilk test confirmed non-normal distributions of demographic data. Therefore, continuous variables are presented as median and interquartile range (IQR, Q1-Q3) using descriptive statistics. For the primary analysis, all available hormone measurements at each time point were utilized for the expanded cohort (n = 20 total). One patient lacked admission hormone measurements but had measurements at subsequent time points and was therefore included in the cohort but excluded from the admission time point analysis. Missing data were handled through pairwise deletion. Two-sided Wilcoxon signed-rank tests were used to compare paired hormone levels across time points, with each pairwise comparison limited to patients with non-missing values at both time points. A Bonferroni-adjusted significance threshold of $\alpha = 0.017$ was applied to account for three pairwise comparisons. A validation analysis using two-sided Mann-Whitney U tests was performed to compare hormone distributions between patients with complete data at all three time points (n = 12) and the expanded cohort, with statistical significance defined as $\alpha = 0.05$. Descriptive analyses were performed for subgroups stratified by reconstruction type, with values presented as median (IQR, Q1-Q3) when multiple measurements were available. Single measurements and missing data were indicated accordingly. No formal statistical comparisons were made within reconstruction subgroups due to limited sample sizes. All statistical analyses were performed using R, version 4.5.1.

Table 1. Baseline patient characteristics of expanded cohort including patients with partial data (n = 20)

Characteristics	Total (n = 20)
Age, median (IQR)	57 (48.5-63.5)
Race, n (%)	
White	14 (70)
Black	2 (10)
Asian	1 (5)
Hispanic	2 (10)
Other	1 (5)
BMI kg/m^2, median (IQR)	30.8 (26.8-37.9)
Diabetes Mellitus, n (%)	12 (60)
Hypertension, n (%)	13 (65)
Coronary Artery Disease, n (%)	4 (20)
Smoking Status, n (%)	
Current	7 (35)
Former	6 (30)
Never/Unknown	7 (35)
Hemoglobin A1c, median (IQR)	7.35 (5.7-10.3)
Closure Type, n (%)	
Primary with scrotum	12 (60)
Primary with one thigh pouch	3 (15)
Primary with two thigh pouches	2 (10)
Secondary Intent	2 (10)
Split-thickness Skin Graft	1 (5)
Median number of debridements, n (range)	3 (2-16)
Median time to discharge labs, days (range)	13 (6-50)
Median time to follow-up labs, days (range)	48 (31-274)
Median length of stay, days (range)	14.5 (4-70)

IQR, interquartile range, Q1-Q3; BMI, body mass index.

Results

Between July 2023 and September 2024, 31 male patients were treated for FG. Twenty patients (65%) had hormone measurements for at least two of three time points (admission, discharge and follow-up) and comprised the expanded cohort for primary analysis. Of these, 12 patients (39%) had complete hormone panels at all three time points (admission, discharge, and follow-up).

Patient characteristics of the expanded cohort are shown in **Table 1**. Patients underwent a median of three surgical debridements (range 2-16) prior to reconstruction. Post-discharge complications occurred in five patients at one month (urinary tract infection, wound dehiscence, superficial skin separation, and thigh pouch pain), and in three patients at six months (urethral stricture, urinary retention, and delayed ejaculation). No patients had hormone measurements at one-year of follow-up.

Hormone levels of the expanded cohort at each time point are summarized in **Table 2**, with longitudinal trajectories shown in **Figure 1**. TT and FT increased significantly across all three time points ($P \leq 0.011$). Gonadotropin responses differed by hormone. LH did not increase across time points after Bonferroni correction, while FSH remained stable during hospitalization but increased from discharge to follow-up ($P = 0.014$). SHBG increased significantly during hospitalization ($P = 0.004$) but did not maintain significance at follow-up. E2 decreased significantly from admission to discharge and from admission to follow-up ($P = 0.014$ and $P = 0.008$, respectively). Comparison of the complete cohort (n = 12) to the expanded cohort revealed no significant differences in median hormone values at any time point (**Table 3**, all $P > 0.05$). When stratified by reconstruction type, all surgical closure groups demonstrated similar hormonal recovery trajectories (**Table 4**).

Discussion

To our knowledge, this is the first study to examine the endocrine impact of FG on the HPG axis. Our results showed that testosterone levels were markedly suppressed at admission, increased steadily by discharge, and continued to rise to physiological range at disease resolution within one year of follow-up, with gonadotropin levels following a similar trend. The recovery of testosterone alongside gonadotropin recovery suggests that local inflammation and disrupted scrotal thermoregulation from FG and the subsequent surgical interventions did not permanently damage the testicular steroidogenic capacity. Rather, these findings are

Table 2. Changes in serum hormone levels measured at three time points following treatment in the expanded cohort

Hormone	Admission Median (IQR)	Discharge Median (IQR)	Follow-Up Median (IQR)	*p*-value
TT (ng/dL)	25 (14-37.8)	131 (49.5-172.5)	251.8 (192.2-377.8)	Ad vs. Dc: **<0.001** Dc vs. Fu: **0.003** Ad vs. Fu: **0.001**
FT (pg/mL)	7.9 (3.8-9.2)	27.5 (10.1-35.4)	55.5 (37.6-74.9)	Ad vs. Dc: **<0.001** Dc vs. Fu: **0.011** Ad vs. Fu: **0.003**
LH (mIU/mL)	4.5 (3.5-7.2)	6.5 (5.2-9.8)	8 (5.4-9.8)	Ad vs. Dc: 0.029 Dc vs. Fu: 0.168 Ad vs. Fu: 0.038
FSH (mIU/mL)	7.5 (5-12.5)	8 (6-10.8)	14 (10.8-17.4)	Ad vs. Dc: 0.459 Dc vs. Fu: **0.014** Ad vs. Fu: 0.054
SHBG (nmol/L)	20 (12-27)	32.5 (28.2-47.2)	30 (21.2-41)	Ad vs. Dc: **0.004** Dc vs. Fu: 1.000 Ad vs. Fu: 0.120
E2 (pg/mL)	38.5 (25.2-57)	25 (20-31.5)	26.5 (21.9-32.4)	Ad vs. Dc: **0.014** Dc vs. Fu: 0.759 Ad vs. Fu: **0.008**

Ad, admission; Dc, discharge; Fu, follow-up; TT, total testosterone; FT, free testosterone; LH, luteinizing hormone; FSH, follicle-stimulating hormone; SHBG, sex hormone-binding globulin; E2, estradiol; IQR, interquartile range. Bold numbers indicate significance after Bonferroni correction ($\alpha = 0.017$). Values reported as Median (IQR, Q1-Q3). *p*-values from two-sided Wilcoxon signed-rank tests. Sample sizes vary at each time point due to incomplete follow-up data, with all available measurements included in the analysis.

consistent with acute suppression of the HPG axis by the systemic illness response. As the infection resolved, the HPG axis recovered to its eugonadal range, with testicular steroidogenesis following suit.

The initial suppression of the HPG axis and testosterone production is likely multifactorial. Transient HPG axis suppression has been well-documented in the setting severe illness, and both hyper- and hypogonadotropic hypogonadism can occur in acutely ill men [11-13]. In our cohort, we observed hypogonadotropic hypogonadism throughout the disease course. Many proposed mechanisms may contribute to hypogonadism in critical illness, including reduced gonadotropin-releasing hormone (GnRH) synthesis, hypercortisolemia, and hyperestrogenemia, though the exact mechanism remains unclear [12, 13]. The decline in serum E2 levels observed in our patients over time makes hyperestrogenemia an unlikely driver of HPG axis suppression in the setting of FG. Similar to Cushing's disease, the severe systemic inflammatory response in FG likely activates the hypothalamic-pituitary-adrenal (HPA) axis, producing endogenous hypercortisolemia that inhibits GnRH synthesis and serves as the main driver of central axis inhibition [13]. Glucocorticoids may further inhibit sex steroid hormone production through glucocorticoid receptor-mediated inhibition at the level of the gonads [14]. Future research should explore this proposed mechanism of HPA-mediated HPG axis suppression in the context of FG.

The variability in admission hormone levels observed in our cohort mirror patterns reported in other critical illnesses. The degree of HPG axis suppression has been shown to correlate inversely with illness severity, and the reported mortality rate in FG ranges from 7.5% to 88% in literature, reflecting substantial heterogeneity in clinical disease severity [15, 16]. This variability in infection severity likely accounts for much of the individual variability in hormonal levels seen in **Figure 1**.

Central HPG axis suppression is likely not the only driver of the hypogonadism in patients with FG. Previous studies have found a significant negative correlation between serum

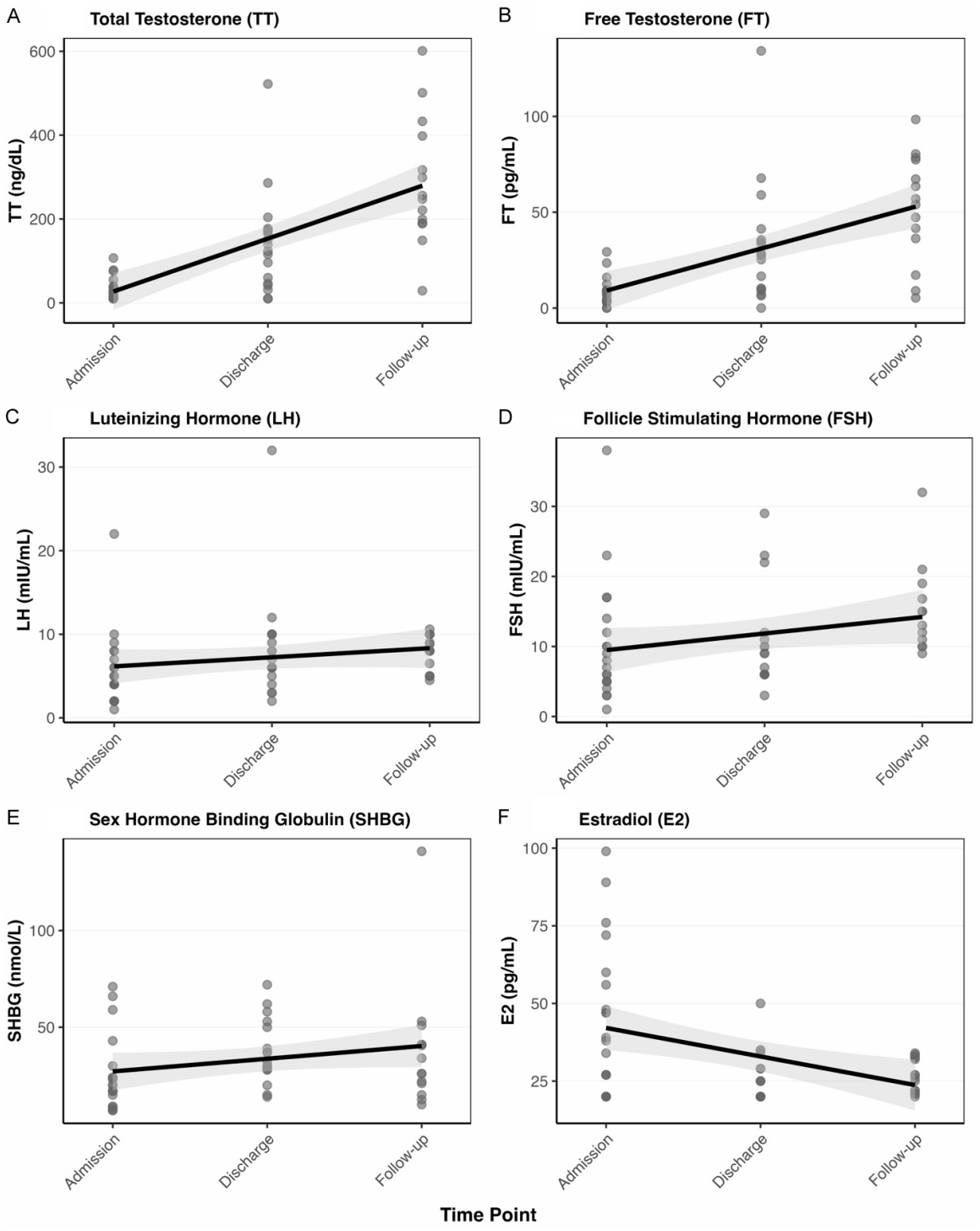

Figure 1. Individual hormone trends of the expanded cohort. This figure displays the collective trends of each individual hormone measure. Subpanels include: A. Total testosterone in ng/dL; B. Free Testosterone in pg/mL; C. Luteinizing Hormone in mIU/mL; D. Follicle-Stimulating Hormone in mIU/mL; E. Sex Hormone-Binding Globulin in nmol/L; F. Estradiol pg/mL.

inflammatory markers and free testosterone, independent of LH and FSH levels [17]. The polymicrobial nature of FG ensures substantial endotoxin exposure, which may locally impair Leydig cell function in addition to centrally suppressing the HPG axis, potentially contributing

Table 3. Comparison of serum hormone levels between patients with complete hormone data at all three time points (complete cohort, n = 12) and the expanded dataset that includes additional patients with partial data (expanded cohort, n = 19 at admission, n = 18 at discharge, n = 14 at follow-up)

Hormone	Complete Cohort Median (IQR)	Expanded Cohort Median (IQR)	*p*-value
TT (ng/dL)			
Admission	27.5 (14.8-46)	25.0 (14-37.8)	0.683
Discharge	158.5 (102.8-180.8)	131 (49.5-172.5)	0.597
Follow-Up	308 (215.5-406.8)	251.8 (192.2-377.8)	0.561
FT (pg/mL)			
Admission	8.2 (5.8-9.9)	7.9 (3.8-9.2)	0.710
Discharge	30.0 (14.7-45.7)	27.5 (10.1-35.4)	0.718
Follow-Up	62.1 (49.6-77.7)	55.5 (37.6-74.9)	0.707
LH (mIU/mL)			
Admission	4.5 (2-6)	4.5 (3.5-7.2)	0.471
Discharge	8 (5.5-9.2)	6.5 (5.2-9.8)	0.844
Follow-Up	8 (5-9.1)	8 (5.4-9.8)	0.808
FSH (mIU/mL)			
Admission	7 (5.8-11.8)	7.5 (5-12.5)	0.980
Discharge	7.5 (6-9.2)	8 (6-10.8)	0.528
Follow-Up	15.9 (10.8-19.5)	14 (10.8-17.4)	0.727
SHBG (nmol/L)			
Admission	17 (13.5-25.5)	20 (12-27)	0.831
Discharge	31.5 (29.5-41.8)	32.5 (28.2-47.2)	0.911
Follow-Up	37.3 (25-41)	30 (21.2-41)	0.784
E2 (pg/mL)			
Admission	38.5 (27-60)	38.5 (25.2-57)	0.858
Discharge	20 (20-26)	25 (20-31.5)	0.242
Follow-Up	23.5 (21.6-28.2)	26.5 (21.9-32.4)	0.538

TT, total testosterone; FT, free testosterone; LH, luteinizing hormone; FSH, follicle-stimulating hormone; SHBG, sex hormone-binding globulin; E2, estradiol; IQR, interquartile range. This validation analysis assessed whether the complete cohort was representative of the broader patient population. Values represent median with interquartile range (IQR, Q1-Q3). Two-sided Mann-Whitney U tests were used to compare hormone distributions between cohorts at each time point. Statistical significance defined as $\alpha = 0.05$. No significant differences were observed between the complete and expanded cohorts for any hormone at any time point (all $P > 0.05$).

to the castrate-range testosterone levels observed in some patients [3, 18]. Moreover, testosterone is known to decline following surgical stress for 6-7 days postoperatively, with compensatory LH elevations [19-21]. Extensive surgical debridement may therefore have compounded HPG axis suppression in our cohort.

Importantly, our findings provide the first hormonal evidence in support of the current consensus favoring testicular preservation over orchiectomy in FG management [9, 16]. Current literature emphasizes that orchiectomies should be performed only when strictly necessary, as testicular involvement is rare given the typical preservation of testicular neurovascular supply and the separate fascial compartment enclosing the testes, even in the setting of extensive perineal and scrotal infection [7, 9, 10]. To date, however, this recommendation has focused on anatomical and surgical principles as opposed to endocrinologic data. While clinical assessment of testicular viability can be difficult in the setting of severe surrounding tissue necrosis, our data demonstrate that preserved testicles not only survive the initial insult of FG but also recover physiological endocrine function within the follow-up period. The similar hormonal recovery observed across different reconstructive efforts, despite small sub-group sizes, further suggests that reconstructive efforts are worthwhile during surgical treatment [22]. Therefore, without clear evidence of testicular necrosis, effort should be undertaken to preserve the testicles at the time of both the initial debridement and closure or reconstruction. Testicular preservation may help minimize HPG axis disruption and optimize recovery of hormone production and fertility potential in FG patients, even when transient hypogonadism is present.

This study has several limitations, and its generalizability may be limited to similar patient populations. First, this is an observational study with a small sample size. Because many patients are transferred to our hospital after primary debridement elsewhere, or reside out of state, obtaining follow-up hormone data was challenging. As a result, complete hormone panels were not available for all patients

Table 4. Descriptive analysis of median serum hormone levels at three time points stratified by surgical closure type of the expanded cohort, which includes patients with partial data

Hormone	Primary Closure with Scrotum n = 12	Primary Closure with One Thigh Pouch n = 3	Primary Closure with Two Thigh Pouches n = 2	Secondary Intent n = 2	Split-Thickness Skin Graft n = 1
TT (ng/dL)					
Admission	25.5 (14.8-36.2)	55 (34.5-65.5)	25 (17.5-32.5)	22.5 (21.2-23.8)	10[a]
Discharge	123 (53-172)	152 (97-164.5)	117.5 (106.8-128.2)	10[a]	522[a]
Follow-Up	308 (196.5-450)	309.5 (265.2-353.8)	218.8 (204.4-233.1)	142.5 (85.8-199.2)	-
FT (pg/mL)					
Admission	8.4 (3.9-9.9)	7.9 (7.2-15.7)	3.5[a]	6.9 (5.7-8.2)	0[a]
Discharge	25.3 (8.6-38.5)	27.2 (18.1-31)	30.8 (29.3-32.2)	0[a]	134.2[a]
Follow-Up	62.1 (44.5-77.7)	67.2 (60.6-73.8)	52.5 (47.1-58)	13.1 (11.1-15.1)	-
LH (mIU/mL)					
Admission	4.5 (3.5-6.2)	6 (3.5-7)	3 (2.5-3.5)	15 (11.5-18.5)	4[a]
Discharge	6 (5-8.5)	10 (8-11)	6 (5.5-6.5)	3[a]	32[a]
Follow-Up	8 (6.1-9.1)	7.5 (6.2-8.8)	8.5 (8.2-8.8)	7.5 (6.2-8.8)	-
FSH (mIU/mL)					
Admission	8.5 (5.8-13.2)	6 (4.5-8)	2.5 (1.8-3.2)	18.5 (16.2-20.8)	5[a]
Discharge	9 (6-10.5)	6 (4.5-14.5)	6.5 (6.2-6.8)	10[a]	29[a]
Follow-Up	12 (10.5-17.9)	23.5 (19.2-27.8)	14 (13.5-14.5)	10[a]	-
SHBG (nmol/L)					
Admission	21.5 (13.5-25.5)	17 (12-38)	17[a]	25 (16-34)	20[a]
Discharge	32 (29.5-44.5)	33 (30.5-43)	26 (20.5-31.5)	62[a]	28[a]
Follow-Up	40.8 (22.6-43.5)	28 (25-31)	23.5 (22.2-24.8)	78 (46.5-109.5)	-
E2 (pg/mL)					
Admission	27 (20-47.2)	39 (29.5-57.5)	73 (60-86)	61.5 (47.8-75.2)	60[a]
Discharge	20 (20-25)	25 (22.5-27)	35[a]	34[a]	50[a]
Follow-Up	23.5 (21.6-32.6)	29.5 (28.2-30.8)	29.8 (27.9-31.6)	24 (22.5-25.5)	-

All data presented as median (IQR, Q1-Q3) when multiple data are available. a, Single measurements; -, Missing data; TT, Total Testosterone; FT, Free Testosterone, LH, Luteinizing Hormone; FSH, Follicle-Stimulating Hormone; SHBG, Sex Hormone-Binding Globulin; E2, Estradiol.

at each prespecified time points, limiting statistical power. Second, hormone measurements were not consistently performed in the early morning across all time points. Given the diurnal variations of testosterone levels, collection timing may have introduced variability in the observed values. Third, heterogeneity in infection severity resulted in variability in the number of debridements and reconstructive approaches performed. The small number of patients within each reconstruction type limited our ability to independently assess the effects of reconstruction approach on hormonal recovery. Finally, variable lengths of hospitalization resulted in discharge hormone measurements at different postoperative time points, potentially introducing additional variability. However, we considered discharge a reasonable time point as it uniformly represented when infection was resolved and reconstruction was complete. Future studies should focus on prospective, multicenter cohorts with standardized hormone testing protocols and larger sample sizes to further clarify FG's impact on endocrine function. A larger sample size may also allow for stratification by type of scrotal reconstruction to understand potential nuances in postoperative hormonal recovery.

Conclusions

Our findings suggest that the HPG axis is transiently disrupted during the acute phase of FG but retains the capacity to return to physiologic ranges following recovery. These results underscore the importance of testicular preservation

at the time of surgical debridement when clinically feasible.

Acknowledgements

The authors thank Alexandra Jacobs, MD for valuable contributions to the conceptualization and initial development of this project.

Disclosure of conflict of interest

None.

Address correspondence to: Dr. George E Koch, Department of Urology, The Ohio State University, 915 Olentangy River Road, Suite 2000, Columbus, OH 43212, USA. Tel: 315-651-2901; E-mail: George.Koch@osumc.edu

References

[1] El-Qushayri AE, Khalaf KM, Dahy A, Mahmoud AR, Benmelouka AY, Ghozy S, Mahmoud MU, Bin-Jumah M, Alkahtani S and Abdel-Daim MM. Fournier's gangrene mortality: a 17-year systematic review and meta-analysis. Int J Infect Dis 2020; 92: 218-225.

[2] Belinchón-Romer I, Ramos-Belinchón A, Lobato-Martínez E, Sánchez-García V and Ramos-Rincón JM. National study of fournier gangrene in Spain (2016-2021): gender/sex differences in mortality and risks. Medicina (Kaunas) 2024; 60: 1600.

[3] Yilmazlar T, Gulcu B, Isik O and Ozturk E. Microbiological aspects of Fournier's gangrene. Int J Surg 2017; 40: 135-138.

[4] Shet P, Mustafa AD, Varshney K, Rao L, Sawdagar S, McLennan F, Ansari S, Shet D, Sivathamboo N and Campbell S. Risk factors for mortality among patients with fournier gangrene: a systematic review. Surg Infect (Larchmt) 2024; 25: 261-271.

[5] Abbasi B, Hacker E, Ghaffar U, Hakam N, Li KD, Alazzawi S, Fernandez A, Patel HV and Breyer BN. Higher morbidity and mortality in females with Fournier gangrene compared with males: insights from national inpatient sample data. J Urol 2025; 213: 99-109.

[6] Lin TY, Cheng IH, Ou CH, Tsai YS, Tong YC, Cheng HL, Yang WH, Lin YM and Cheng YS. Incorporating Simplified Fournier's Gangrene Severity Index with early surgical intervention can maximize survival in high-risk Fournier's gangrene patients. Int J Urol 2019; 26: 737-743.

[7] Bowen D, Juliebø-Jones P and Somani BK. Global outcomes and lessons learned in the management of Fournier's gangrene from high-volume centres: findings from a literature review over the last two decades. World J Urol 2022; 40: 2399-2410.

[8] Sugihara T, Yasunaga H, Horiguchi H, Fujimura T, Ohe K, Matsuda S, Fushimi K and Homma Y. Impact of surgical intervention timing on the case fatality rate for Fournier's gangrene: an analysis of 379 cases. BJU Int 2012; 110(11 Pt C): E1096-E1100.

[9] Matloubieh JE, Patel RD, Fram E and Lowe FC. Factors associated with Fournier's gangrene survival in a diverse urban community: a 15-year review. BJU Int 2025; 136: 314-320.

[10] Elbakry AA, Wasef K, Pettit A, Mitchell K, Mihalo J, Kandzari S and Osterberg EC. PD12-05 testicular involvement in fournier's gangrene: is orchiectomy overutilized? J Urol 2023; 209 Suppl 4: e401.

[11] Salonia A, Pontillo M, Capogrosso P, Gregori S, Tassara M, Boeri L, Carenzi C, Abbate C, Cignoli D, Ferrara AM, Cazzaniga W, Rowe I, Ramirez GA, Tresoldi C, Mushtaq J, Locatelli M, Santoleri L, Castagna A, Zangrillo A, De Cobelli F, Tresoldi M, Landoni G, Rovere-Querini P, Ciceri F and Montorsi F. Severely low testosterone in males with COVID-19: a case-control study. Andrology 2021; 9: 1043-1052.

[12] Ma Y, Fan X, Han J, Cheng Y, Zhao J, Fang W and Gao L. Critical illness and sex hormones: response and impact of the hypothalamic-pituitary-gonadal axis. Ther Adv Endocrinol Metab 2025; 16: 20420188251328192.

[13] Basaria S. Male hypogonadism. Lancet 2014; 383: 1250-1263.

[14] Pivonello R, Isidori AM, De Martino MC, Newell-Price J, Biller BM and Colao A. Complications of Cushing's syndrome: state of the art. Lancet Diabetes Endocrinol 2016; 4: 611-629.

[15] Lanser L, Burkert FR, Thommes L, Egger A, Hoermann G, Kaser S, Pinggera GM, Anliker M, Griesmacher A, Weiss G and Bellmann-Weiler R. Testosterone deficiency is a risk factor for severe COVID-19. Front Endocrinol (Lausanne) 2021; 12: 694083.

[16] Tarasconi A, Perrone G, Davies J, Coimbra R, Moore E, Azzaroli F, Abongwa H, De Simone B, Gallo G, Rossi G, Abu-Zidan F, Agnoletti V, de'Angelis G, de'Angelis N, Ansaloni L, Baiocchi GL, Carcoforo P, Ceresoli M, Chichom-Mefire A, Di Saverio S, Gaiani F, Giuffrida M, Hecker A, Inaba K, Kelly M, Kirkpatrick A, Kluger Y, Leppäniemi A, Litvin A, Ordoñez C, Pattonieri V, Peitzman A, Pikoulis M, Sakakushev B, Sartelli M, Shelat V, Tan E, Testini M, Velmahos G, Wani I, Weber D, Biffl W, Coccolini F and Catena F. Anorectal emergencies: WSES-AAST guidelines. World J Emerg Surg 2021; 16: 48.

[17] Tremellen K, McPhee N, Pearce K, Benson S, Schedlowski M and Engler H. Endotoxin-initiated inflammation reduces testosterone produc-

tion in men of reproductive age. Am J Physiol Endocrinol Metab 2018; 314: E206-E213.
[18] Inoue T, Aoyama-Ishikawa M, Uemura M, Yamashita H, Koga Y, Terashima M, Usami M, Kotani J and Hirata J. Interleukin-18 levels and mouse Leydig cell apoptosis during lipopolysaccharide-induced acute inflammatory conditions. J Reprod Immunol 2020; 141: 103167.
[19] Thompson K, Klein D, Sreekumar S, Kenny L, Campbell K, Alaia M, Strauss EJ, Jazrawi L and Gonzalez-Lomas G. Testosterone levels before and after anterior cruciate ligament reconstruction a prospective observational study. Bull Hosp Jt Dis (2013) 2022; 80: 265-269.
[20] Foster MA, Taylor AE, Hill NE, Bentley C, Bishop J, Gilligan LC, Shaheen F, Bion JF, Fallowfield JL, Woods DR, Bancos I, Midwinter MM, Lord JM and Arlt W. Mapping the steroid response to major trauma from injury to recovery: a prospective cohort study. J Clin Endocrinol Metab 2020; 105: 925-937.
[21] Szécsi B, Tóth K, Szabó A, Eke C, Szentgróti R, Dohán O, Benke K, Radovits T, Pólos M, Merkely B, Gál J and Székely A. Hormonal changes in the first 24 postoperative hours after cardiac surgical procedures. Physiol Int 2023; 110: 251-266.
[22] Staniorski C, Myrga J, Hayden C, Sterling J and Rusilko P. Fasciocutaneous flap perineal closure with testicular thigh pouch for scrotal defects: surgical technique and initial experience. Urology 2023; 182: 231-238.