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Factors Affecting the Prognosis and Outcome of Fournier's Gangrene

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ABSTRACT

Background: The purpose of the present study was to prospectively analyze the data of patients data presenting with Fournier's gangrene (FG) and to compare the obtained data with the literature in an effort to find out the various factors affecting the prognosis and outcome in Fournier's gangrene (FG). **Methods:** A prospective study was conducted on 32 patients (all males) with Fournier's gangrene (FG) who attended the Department of General Surgery, Assam Medical College and Hospital, Dibrugarh over a period of 1 year from July 2013 to June 2014. **Results:** The mean age of the patients was 52.56 +/-14.5 years. The most common presentation was swelling (n=30; 93.75%). Scrotum has been shown to be the most commonly affected area in the patients (n=31; 96.88%). Alcohol consumption on regular basis was the leading predisposing factor (n=18; 56.25%) and apart from idiopathic cuases, trauma was the leading incidental cause for Fournier's gangrene (FG). Polymicrobial growth pattern was seen in 68.75% of wound swab culture with Escherichia coli as the most frequently identified microorganism (28.8%). Primary closure was the most common technique used for all patients. Eight patients exhibited a mortal course due to multi-organ failure following sepsis. **Conclusion:** In conclusion Fournier's gangrene is a rapidly progressive fulminant infection and represents a surgical emergency. Rapid and correct diagnosis of the disease can avoid inappropriate or delayed treatment and may prevent death of the patient. With late hospital presentation or delayed diagnosis, mortality remains high.

Keywords: Fournier's gangrene (FG); Polymicrobial; Sepsis; Multi-Organ Failure Syndrome.

INTRODUCTION

Fournier's gangrene (FG) is an uncommon and nasty condition of infective origin that is characterized by scrotal inflammation, with rapid onset of gangrene leading to exposure of scrotal contents.[1] FG is a vascular disaster of infective origin and obliterative endarteritis plays a key role in its pathogenesis.[2]

The entity is no longer restricted to the young males and it may affect a wide range of population of both sexes.[3] Now-a-days in approximately 95% of the cases, a source can be identified.[4] The infection is frequently polymicrobial and synergistic with several aerobic, or anaerobic microorganisms.[5,6]

Risk factors for FG includes diabetes mellitus (DM), alcoholism, malnutrition, low socioeconomic status, neoplasm, chronic glucocorticoid therapy, immune-compromised states, Human immunodeficiency virus (HIV) infection, chemotherapy, radiotherapy, Crohn's disease and infected hydrocele.[7,8]

Apart from parameters of Fournier's Gangrene Severity Index (FGSI); chronic renal failure, pre-hospital delay time, extent of the affected area, serum-blood urea nitrogen and creatinine level are some of the factors that affected the prognosis of the disease.[9] Mortality rate has been shown to range from 7.5–8.8%, depending upon co-morbidities and severity of the disease.[10] FG is recognized in International Classification of disease as diagnosis code 49.3 according to ICD 10.[11]

The purpose of the present study was to prospectively analyze the data of patients presenting with FG so as to compare obtained data with the literature regarding the various factors affecting the prognosis and outcomes in patients with FG including FGSI score.

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MATERIALS AND METHODS

A one year prospective study was conducted on 32 patients with FG who attended the Department of General Surgery, Assam Medical College and Hospital, Dibrugarh from July 2013 to June 2014. The diagnosis of FG was made on the basis of clinical findings.

Inclusion Criteria: All patients with cellulitis, erythemas, skin necrosis, ulcer, discharge, necrotizing fasciitis of perineal and perianal region were included in this study.

Exclusion Criteria: All patients with long standing diabetes mellitus [>10 years duration], immune-compromised states, steroid therapy, chemotherapy, radiotherapy as well as female patients were excluded from the study.

The cases after being stabilized hemodynamically were subjected to detail clinical examination, culture

and antibiotic sensitivity test from the wound swabs routine and special blood examinations and urine examinations,.

Patients' data regarding age, sex, hospital presentation, anatomic distribution, pre-hospital delay time, predisposing factors, etiologic causes, treatment modalities, hospitalization time, and mortality rate were evaluated prospectively. Pre-hospital delay was defined as the time from the onset of symptoms until hospital admission. Clustered data were analyzed statistically by paired T test (two tailed) and chi-square test with and without Yale's correction.

The cases were treated with medical therapy and surgically where required depending upon the clinical condition of the patient.

RESULTS

Age: The age of the patients ranged from 25 to 82 years. The highest incidence of FG was observed in the age group of 41-60 years (n=14, 43.74%) (Fig. 1) and the mean age was 55.06+/-15.52 years. In this study the mean age of survivors and non-survivors were 51.17 +/- 14.12 and 66.88 +/- 14.10 years respectively

Table 1A: Presenting Features at Hospital Admission:

Sl No	Clinical Features	Number Of Patients [N]	Percentage (N/Total Cases)X 100
1	Genital oedema	30	93.75
2	pain	27	84.38
3	hyperemia	25	78.13
4	Skin necrosis	20	62.5
5	discharge	18	56.25
6	pyrexia	18	56.25
7	crepitus	17	53.13
8	sepsis	15	46.88
9	Necrotic ulcer	10	31.25
10	hypotension	8	25
11	gangrene	4	12.5
12	Urinary retention	2	6.25
13	Faecal incontinence	1	3.13

Anatomic Distribution: The scrotum was found to be the most commonly affected area in the patients (n=31, 96.88%). Other affected areas, in decreasing order of frequency were penis (n=16, 50.00%), perineum (n=13, 40.63%), inguinal region (n=10, 31.25%), thigh (n=5, 15.63%), abdomen (n=3, 9.38%), and chest (n=1, 3.13%). The TSBA involved was calculated by rule of nine (used for assessing the bur injury), in survivors it was 4.63 +/- 0.92% and in non-survivors 1.75 +/- 0.79%.

Pre-hospital Delay Time: The mean pre-hospital delay time of the patients was 5.19 ± 2.29 days (range of 2–10 days); in survivors it was 4.12 +/- 1.64% and in non-survivors 8.13 +/- 1.13%. The duration of symptoms before hospital admission wasless than 3 days in 8 patients(25.00%), 4-6 days in 13 patients (40.63%), 7-9 days in 10 patients (31.24 %), and more than 9 days in 1 patient(3.13%).

Predisposing Factors: Of the patients, 22 had more than one predisposing factor for FG. Frequent alcohol consumption 56.25%, smoking 53.13% and diabetes 37.50% were the leading factors. They were followed by cardiovascular diseases 25.00%, obesity12.50% and COPD9.38%.

Etiologic Causes: Etiologic causes were identified in 20 patients (62.5%) they were local trauma (n=7; 21.88%), perianal source (n=5,15.63%), dermatological causes (n=4, 12.50%), previous surgery (n=3, 9.38%). Previous surgery included removal of sebaceous cyst, lipoma and granuloma from urogenital region especially scrotum. Ruptured urethra following an attempt of urological instrumentation for urethral stricture leading to FG was seen in a single case (3.13%). Rest 12 patients (37.5%) were Idiopathic

Microbiology and Antibiotic therapy: Positive bacteriologic cultures were obtained in 28(87.5%) patients and the infection was polymicrobial in 22 patients (68.75%) E. coli was the most frequently identified microorganism (n=15, 28.85%) that was followed by Streptococcus species, Staphylococcus species, Pseudomonas species, Enterobacter species, Proteus species, Klebsiella species and Bacteroides species respectively. Monomicrobial growth was found in 6 patients (18.75%) all Streptococcus spp.

Table 1B: Microbiology of Wound Swab Culture

Growth Type	Number (N = 32)	Percentage (%)	Organism	Number (N = 52)	Percentage (%)
Polymicrobial	22	68.75	E.coli	15	28.85
			Streptococcus	9	17.31
			Staphylococcus	8	15.38
			Pseudomonas	7	13.46
			Enterobacter	5	9.62
			Proteus	4	7.69
			Klebsiella	3	5.77
			Bacteroides	1	1.92
			Total	52	100.00
Monomicrobial	6	18.75	Streptococcus	6	
No Growth	4	12.50			

[Table 1] Above table shows the microbiological characteristics of different wound swab cultures of patients with Fournier's gangrene in present studty. The table also shows the number and percentage of

the different micro-organisms isolated from monomicrobial and polymicrobial cultures in the present study.

Based upon wound swab culture and senstivity results patients received empirical intravenous antibiotic regimen, the most common antibiotic used was imipenem-cilastin + ciprofloxacin (n=12, 42.9%). Followed by piperacillin-tazobactum (n=6, 21.4%), piperacillin-tazobactum + ciprofloxacin (n=5, 17.86%) Cefipime + Ciprofloxacin (n=3, 10.71%), Cefipime + Linezolod (n= 2, 7.14%).

Surgical Management: All patients underwent aggressive surgical debridement of the necrotic tissues after initial hemodynamic stabilization, averaging 3.41 ± 1.13 with a range of 1 to 4 debridements, 68.75% (n= 22) patients were debrided 3 - 4 times, 18.75% (n=6) patients were debrided 1-2 times and 12.50% (n=4) patients were debrided 5 – 6 times. The mean debridement in survivor was 3.88 +/- 0.68 times and non-survivors was 2 +/- 1.07 times.

Debridement was performed in the same day of hospital admission in 29 patients (90.6%), 23 patients (71.88%) were debrided within 8 hrs of hospital admission and 6 patients (18.74%) were debrided 8 – 24 hrs. Urinary diversion was done in all cases by Foley's Catheter expect one who presented with FG after rupture urethra tha patient was subjected to supra-pubic cystostomy was done in it. None of our patients needed orchidectomy or colostomy for fecal-diversion.

In the survivor group (n=24) secondary suturing (n=11, 45.83%) was most common reconstruction procedure performed. Cases involving both scrotum and penis were managed with secondary suturing of wound of the scrotum and split thickness skin graft of penile lesion (n=7, 29.1%). Six cases (25%) were managed with split thickness skin graft only. None of the patients required reconstructive flap procedure.

Hospitalization Time: The mean hospitalization time of the patient's was25.69 [± 12.8 days.] days (range 7 - 46 days). The mean hospitalization time for survivor was 30.54 ± 10.94 days (range 17- 47 days) and non-survivor was 11.13 ±3.14 days (range 7-15 days).

PROGNOSIS AND OUTCOMES: The mortality rate in this study was 25% (n=8). The mean FGSI score was 7.66 ± 3.25, range, 6 – 14). The average FGSI score in the survivor group was 5.92 ± 1.19 and the non-survivor group was 12.88 ± 0.64. (FGSI score >9 = 10 cases and <9 = 22 cases). The mean serum albumin values were 2.68 +/- 1.07 gms/dl (range, 1 – 4.5 gm/dl), mean albumin levels in survivors and non-survivors were 3.15 +/- 0.78 and 1.28 +/- 0.21 gms/dl respectively. Most of the patients were anemic with mean hemoglobin values of 8.12 +/- 1.65gms /dl (survivor – 8.91 +/- 0.99 and non-survivor – 5.74 +/- 0.36).

In this study a number of various complication were encountered e.g graft failure (n= 4, 12.5%), cosmetic deformity of the penis and scrotum (n= 3, 9.38%) and decreased sexual satisfaction in due course of time (n= 2, 6.25%). Of the eight cases who didn't survive, acute renal failure leading to Multi Organ Dysfunction syndrome (n=3, 9.38%), ARDS with Septicemia (n=2, 6.25%) and Diabetic Ketoacidosis with Severe Dyselectrolemia (n=2, 6.25%) were the leading causes.

Results Summary A & B

Table 2 A: Table showing the Mean and standard deviation of variable influencing the morbidity and mortality in our along with survivor and non-survivor group.

Sl no	Result	Mean (n=32)	Survivors	Non-survivor s	P value
1	Age (in years)	55.06+/-15.52	66.88 +/- 14.10	51.17 +/- 14.12	P = 0.104
2	Albumin (gm/dl)	2.68+/-1.07	3.15 =/- 0.78	1.28+/-0.21	P=0.0001
3	Hemoglobin (gm/dl)	8.12+/-1.65	8.91+/-0.99	5.74+/-0.36	P=0.0001
4	TBSA involved (%)	2.47+/-1.5	1.75+/-0.79	4.63+/-0.92	P<0.05
5	Serum urea	70.31+/-17.56	61.88+/-10.43	95.63+/-5.68	P<0.0001
6	Pre hospital delay (in days)	5.19+/-2.29	4.12+/-1.64	8.13+/-1.13	p= < 0.0001
7	Number of debridements	3.14+/-1.13	3.86+/-0.68	2.0+/-1.07	P<0.0001
8	FGSI Score	7.66+/-3.25	5.92 ± 1.19	12.88 ± 0.64.	P < 0.0001
a	Heart rate	113.44 +/- 13.62	105.83+/- 2.76	136.14+/-2.04	P<0.0001
b	TLC	2116.56+/-4418.40	18954.17+/-1983.62	27603.75+/-3065.93	P<0.0001
c	Temperature in Celsius	37.65+/-1.47	38.38+/-0.31	35.49+/-1.43	P<0.0001
d	Serum bicarbonate	22.15+/-4.89	24.29+/-3.61	15.75+/-0.60	P<0.0001
e	Serum sodium	133.17=/-4.91	132.15+/-1.76	129.00+/-1.41	P<0.0001
f	Serum potassium	3.92+/-0.94	3.53+/-0.65	5.06+/-0.70	P<0.0001
g	Serum creatinine	2.25+/-0.75	2.10+/-0.71	2.73+/-0.69	P=0.0366
h	Serum haematocrit	29.25+/-4.73	24.96+/-5.09	21.75+/-1.39	P=0.0913
i	Respiratory rate	27.00+/-4.81	25.13+/-3.98	32.63+/-1.51	P=0.0012

Table 2 B: Table showing variables influencing morbidity and mortality in FG along with the P value.

Sl no	Result	survivor	Non-survivor	P value
1	DM present	10/24 (41.66%)	2/8 (25%)	P=0.6757
2	Alcohol consumers	13/24 (54.1%)	6/8 (75%)	P=0.4203
3	Sepsis present at admission	7/24 (29.16%)	8/8 (100%)	P=0.0006
4	Debridement <8hrs of admission	23/24 (95.83%)	(0/8) =(0%)	P=0.0001

DISCUSSION

Fournier's gangrene is a rapidly progressing necrotizing fasciitis involving the external genitals and perineum in both males and females. It is usually a polymicrobial infection with synergistic action of both aerobic and anaerobic organisms. Despite aggressive treatment, the mortality rate in these patients remains quite high, ranging from 18% to 36%.[12] Considering rapidity of the spread of the gangrenous area that is reported to be up to 2cm/h to 3 cm/h,[13] we must consider it as a surgical emergency because if not treated quickly mortality of this condition is usually very high.[14]

FGSI score: Certain factors influencing the survival of these patients, primarily relating to the patient's metabolic status and the extent of the disease, were evaluated by Laor and others from which the Fournier's gangrene severity index (FGSI) was formulated, which assesses 9 clinical parameters and the extent of deviation from normal. They determined that a score of 9 or higher combined with advanced age correlated with increased mortality.[15]

FGSI Score has been utilized by various studies for assessing the prognosis in cases with FG. Loar et al. [1995] suggested a score greater than 9 is suggestive of a 75% probability of death and an index score 9 or less is associated with 78% survival.[16] Lin E, and others suggested that a FGSI score cutoff of 9 was an excellent predictor of the outcome of cases.[12] Corcoran AT, Smaldone MC, Gibbons EP, et al.[2008] reported that FGSI SCORE of less than or equal to 9 had a 96% survival rate and a 46% mortality rate in those with a FGSI Score of 9 or greater (p = 0.001, OR 22, 95% CI 3.5-139.7).[17] Khush Muhammad Sohu et al [2013] has reported a mortality rate was 84.6% in the group of patients with FGSI >9 (22/26 patients) and 14.3% in patients with FGSI < 9 (8/56) (p=0.0001) but, Kara E et al [2009] suggested that the FGSI scores > or = 7 can be considered as factor affecting mortality rates with statistical significance (p < 0.05) according to their study.[18,19] A study conducted by Tuncel et al. [2006] on FG patients concluded that FGSI score did not predict the disease severity and the patient survival.[14]

In our study the FGSI score was an significant predictor of mortality [FGSI score >/= 9 (n=10 cases) survivor 2/10, non-survivor 8/10and FGSI Score <9(n=22), survivor 22/22, non-survivor 0/22, p < 0.0001). Mortality rate in those with a FGSI Score of 9 or greater was 80% in our study. The average FGSI score was 7.66 ± 3.25. The average FGSI Score in the survivor group was 5.92 ± 1.19 and the non-survivor group was 12.88 ± 0.64 (p=<0.0001). Except for hematocrit values rest all components of FGSI Score were significant in our study.

Various studies have utilized this score to assess the outcome in patients of Fournier's gangrene. Out of the nine parameters described by Laor et al. temperature, heart rate and respiratory rate were considered to be the most important by some authors. FGSI Score can hence be termed with as objective and simple method to quantify the extent of metabolic aberration at presentation in patients with Fournier's gangrene.[12]

FGSI Score in various studies[12,16,17, 20-22,24]

Table 3: The above table shows the mean FGSI Score in survivors and non-survivor and the significance level (p value) [calculated by unpaired t test in various studies]

Sl No	Study	Year	FGSI Score		P Value
			Survivors	Non-survivor s	
1	Laor et al.[16]	1995	6.9 ± 0.9	13.5 ± 1.5	p = 0.005
2	Yeniyol CO et al,[28]	2004	3.0 +/- 1.8	12 +/- 2.4	P < /=0.0001
3	Lin E et at	2005	4.41+/-2.45	12.75+/-2.82	P<0.0001
4	Corcoran AT et al[17]	2008	5.4 +/- 3.5	10.9 +/- 4.7	p = 0.006
5	Ik Young Kim et al,[21]	2011	4.7 +/- 0.4	9.3 +/- 3.2	P < 0.0001
6	Longwang Wang et al,[22]	2012	5.63±1.89	13.6±3.64	P < 0.0001
7	Rohan Khandelwal et al,[12]	2013	3.8	9.4	NA
8	Andrés García Marínet al,[23]	2014	4	7	P=0.002
9	El-Shazly et al.[24]	2014	6	10.26	P < 0.001
10	AMCH (present study)	2014	5.92 ± 1.19	12.88 ± 0.64.	P < 0.0001

Sl No	Study	Year	FGSI Score		P Value
			Survivors	Non-survivor s	
1	Corcoran AT et al,[17]	2008	5.4 +/- 3.5	10.9 +/- 4.7	p = 0.006
2	Ik Young Kim et al,[21]	2011	4.7 +/- 0.4	9.3 +/- 3.2	P < 0.0001
3	Longwang Wang et al,[22]	2012	5.63±1.89	13.6±3.64	P < 0.0001
4	Andrés García Marínet al,[23]	2014	4	7	P=0.002
5	El-Shazly et al.[24]	2014	6	10.26	P < 0.001
6	AMCH (present study)	2014	5.92 ± 1.19	12.88 ± 0.64.	P < 0.0001

Other Factors:

Higher mortality rates are found in diabetics, alcoholics, and those with colorectal sources of infection who often have a less typical presentation, greater delay in diagnosis, and more widespread extension.[25] Some authors have considered sepsis at

presentation, interval between hospital admission and surgical intervention,[21] lower serum albumin and total protein levels,[12] extent of body surface involvement (more than 5 per cent body surface5 or more than 24 square meters 19), serum glucose level >140 at the time of admission,[19] repeated debridements and low hemoglobin levels as predictors of poor prognosis in patients with FG.[26,27]

TBSA Involved: The extent of involvement i.e total body surface area is calculated using charts routinely used to assess the extent of burn injuries. The penis, scrotum and perineum each account for 1% surface area and each ischiorectal fossa accounts for 2.5%.[18] Yeniyol CO, Suelozgen T, Arslan M, et al [2004] The greater mean extent of body surface area involved among patients who died was significantly different statistically from that of those who survived (5.4% and 2.1%, P < or =0.0001).[28] Corcoran AT, Smaldone MC, Gibbons EP et al.[2008] reported an association between mean total body surface area (p = 0.169), abdominal wall (p = 0.004) or lower extremity (p = 0.005) involvement was associated with increased mortality.[17] Kara E et al. [2009] reported extent of involvement with BSA > or = 24 square centimeter to be a factor affecting mortality in FG with a statistical significance (p < 0.05).[19] Toru Sugiharaet al [2012] concluded that debridement range ≥3000 cm2 (OR 5.22, compared with other operations)was significantly associated with a higher case fatality rate.[30] El BachirBenjelloun et al [2013] found that the extension of the infection to the abdominal wall was a predictor of mortality (p < 0.003; 50% in the non-survivor s compared to 7% in the survivors).[26] Hari Gopal Vyas, Anup Kumar, Vimal Bhandari et al [2013] in their study reported mortality rate of 9.09% in pts with scrotal involvement, 0% in scrotal and penile involvement and 80% in anterior abd. wall and thigh involvement with p value of < 0.01 and considered the area of involvement as imp predictor of poor prognosis (Hazard Ratio of 4.9, 3.81 – 6.32 as95 % Confidence Interval and p value < 0.001).[31] M EL Shazy et al. [2014] reported the BSA involvement in survivor and non- survivor group to be 4.6% and 8 % respectively with p < 0.05.[24] In our study the average TBSA involved was significant for prediction of poor prognosis (over all mean=2.47 +/- 1.5 survivor group- 4.63 +/-0.92 and non-survivor group- 1.75 +/- 0.79 with p value < 0.05).

Laor E et al [1995] reported that mean extent of body surface area involved among patients who died was not statistically different from that of those who lived (7.16 and 4.32%, respectively, p = 0.1),[16] this finding was supported by the study of Mehmet Uluğ et al [2009].[29] Some authors have reported no linear relationship between TBSA involved in FG and mortality prognosis and concluded that local involvement was associated with a reduced mortality rate, compared to extensive body involvement, corroborating the findings of Clayton et al. [1990],[5]

AGE: was considered as significant predictor of poor prognosis by Laor E et al [1995] as survivors were significantly younger (53 years old, range 23 to 90) than non-survivors (71 years old, range 53 to 83, p = 0.004) in his study.16Sorensen et al [2009] found that an increasing patient age was the strongest independent predictor of mortality (Odds Ratio-4.0 to 15.0, p <0.0001)10. El Bachir Benjelloun et al [2013] and Lin E et al. also reported similar results in their study.

Age in Survivor and Non-Survivor

Table 4: The above table shows the mean age in survivor and non-survivor group in various studies and significance of age as predictor of poor prognosis

Sl No	Study	Year	AGE		P Value
			Survivors	Non-survivors	
1	Lin E et at	2005	53.8+/-18.3	59.9+/-10.2	P < 0.05
2	Dimitrios Koukouras et al.[35]	2011	49.8 +/- 17.2	52.28 +/-13.2	P = 0.45
3	El BachirBenjelloun et al.[26]	2013	44.36 +16.05	57.5 + 19.24	P= 0.0225
4	AMCH (present study)	2014	66.88 +/- 14.10	51.17 +/- 14.12	P = 0.104

Age was reported to be insignificant by Satyajeet Verma et al. [2012] (survivor group 56.5% (39/69) cases and non-survivor group 65.3% (17/26) cases age > 50 yrs in both groups),[32] Yeniyol CO, Suelozgen T, Arslan M, et al [2004].[28] Ik Yong Kimalso considered age to be insignificant in their study of 27 patients, 19 patients with < 65 yrs, non-survivors 3 cases (15.8%) and 8 patients with > 65 yrs, non-survivors 1 case (12.5%); Odds ratio-0.762; 95%, CI – 0.067-8.665.[21] Even in our study the age was not a significant predictor of mortality.

BLOOD INVESTIGATIONS: Lin E et at [2005] reported in their study that non-survival group of patients had lower serum hematocrit (mean 28.9, p=0.019) and albumin (mean 1.93, p=0.024) levels and are associated with poor prognosis. Concentration of serum creatinine >1.4 mg/dL, and haemoglobin <10 g/dL, in whole blood were reported by Jaime Ruiz-Tovar et al [2012] to be associated with higher mortality rates.[27]

Andrés García Marínet al [2014].Reported haemoglobin (S 13; D 11; P=.014) and serum urea (S 58; D 102; P<.001) to be significant predictors of poor prognosis and increased mortality.[33] Sallami S, Maalla R, Gammoudi A, et al.[2012]reported hematocrit (p=0.003) and serum sodium (p<0.05) to be significant predictor of poor prognosis.[34]

El Bachir Benjelloun et al [2013] found renal failure on admission (blood urea >0.5 g/l) was higher among the patients who died when compared to the

survival group (p < 0.001) and was considered to be important in predicting unfavourable outcome in FG.[26]

In our study the serum albumin levels, haemoglobin levels and serum urea levels was found significant in predicting unfavourable prognosis [mean serum albumin levels (n=32) was 2.68 +/- 1.07 survivor- 3.13 +/- 0.78 andnon-survivor - 1.28 +/- 0.21, p< 0.0001 ; mean haemoglobin levels 8.12 +/- 1.65, survivor- 8.91 +/- 0.99and non-survivor - 5.74 +/- 0.36, p= < 0.0001, mean urea levels- 70.31 +/- 17.56, survivor- 61.88 +/- 10.43 and non-survivor - 95.63 +/- 5.68, p< 0.0001]

Pre-Hospital Delay Time: Most of the authors have considered pre hospital delay to be insignificant predictors of mortality in FG. Dimitrios Koukouras et al [2009] [average pre-hospital delay of 5.3 +/- 2.8, survivor- 5.3 +/- 2.8 and non-survivor 5.3 +/- 2.6 days; p=1],[35] and El BachirBenjelloun et al [2013] [survival group 11 days, non-survival group= 11.3 days; p < 0.83] reported pre hospital delay to be insignificant in predicting mortality in FG cases.[26] This result was similar to study of Ik Yong Kim [(n=27 cases) 9 patients had Spre-hospital delay of < 48 hours with mortality rate of 11.1% (1 case), 18 cases had a pre-hospital delay of > 48 hours with mortality rate of 16.7% (3 cases) (OR-0.762; 95% CI- 0.067-8.665; p =1)].[21]

But, M El-Shazly et al [2014] reported the mean duration of symptoms before admission to be significantly longer in the mortality group (3.86 days versus 1.96 days in survival group) (p < 0.05). In our study the pre hospital delay was longer in non-survival group and significant predictor of increased mortality. (meanpre hospital delay- 5.19 +/- 2.29, survivor- 4.12 +/- 1.64 and non-survivor-8.13 +/- 1.13, p= < 0.0001)

Sepsis: Most of the authors has considered shock or sepsis on initial presentation as significant predictor of increased mortality in patients with FG.[36] In the study conducted by Ik Yong Kim (27 cases) sepsis was present in 7 cases with mortality of 42.9% (3 cases) and 20 cases presented without sepsis at admission, mortalitry rate was 5% (1case) [OR-14.250; 95% CI-1.162-174.801 p=0.042] and was considered significant21. Even Kara E et al [2009] reported similar results. In our study 47.88% (15/32) cases had signs of sepsis on admission and it was found to be very significant predictor of mortality. (Survivor group; sepsis present=7, sepsis absent= 17 and non-survivor group; sepsis present= 8, sepsis absent nil case, p=0.0006). But, Satyajeet Verma et al. [2012] reported that sepsis at admission was not a predictor of the poor prognosis (survivor group 35/69 cases had sepsis and non-survivor group 15/26 cases had sepsis at admission, p=0.646).[32]

Co-Morbid Factors: Most of the authors have considered DM not affecting the outcome in FG although it has been one of the most common predisposing factors of FG. Arshad Mehmood Malik et al [2010] (n=73 cases, DM= 44/73, survivor-34, non-survivor- 10, p=0.2221)37and El Bachir Benjelloun et al [2013] (non-survivor group with DM= 41%, without DM= 49% , p=0.3) reported that neither DM affects the mortality rate neither influence hospital stay or number of debridments26. Even Ik Yong Kim reported DM to be insignificant predictor of increased mortality in FG cases [n= 27 cases, non-survivor with DM 25%, i.e 3/12, non-survivor without DM 6.7% i.e 1/15; OR- 4.667; 95% CI- 0.418-52.121; p=0.294]. In our study the results were similar to above [survivor group (n=24), with DM=10, without DM= 14; Non-survivor group (n=8) with DM=2, without DM=6, p = 0.675].

S. Aliyu et al [2013] reported a high mortality rate in this study among diabetic patients and considered a poor prognosis, when FG is associated with systemic diseases such as uncontrolled diabetes.[38] This was supported by Mehmet Uluğ et al [2009] the reported that patients with DM are more susceptible to FG.[29]

Time of Surgery: Affect of duration between hospitaladmission and 1st debridement upon the prognosis and outcomes has not been much emphasized in literatures. M El-Shazly et al [2014] has reported the mean duration of symptoms between hospital admission and first debridement to be significantly longer in the mortality group (4.39 days versus 2.35 days in survival group, p < 0.05) and found it be a significant factor of increased mortality. In our study time of surgery was a significant prognostic factor (debridement in survivor group (n=24) <8 hrs= 23 cases, > 8 hrs=1 case, Non-survivor group (n=8) <8 hrs=0 cases, >8 hrs= 8 cases; p<0.0001).

Number of Debridements: Literature have mentioned surgery to be of paramount importance should be aggressive and early. Aggressive surgical debridements always suggest a positive effect on survival.[5] Satyajeet Verma et al. [2012] reported number of debridements as a significant predictor of the poor prognosis in their study (survivor group - 7% and non-survivor group-58.8% underwent >1 debridements after admission, p <0.0532). In our study number of debridements was a significant factor in predicting poor prognosis (average debridements – 3.41 +/- 1.13, survivor- 3.86 +/- 0.68, non-survivor- 2 +/- 1.07, p<0.0001).

However it was considered insignificant in studies of Mehmet Uluğ et al [2009] and Ik Yong Kim [n= 27 cases; mortality in <2 debridements group 3/9 cases,33% and mortality rate >/= 2 debridements, 1/18 cases, 5.6%, OR-0.118; 95% CI- 0.010-1.359; p=0.093].[21,29] El Bachir Benjelloun et al [2013] reported similar results (mortality rate of 52.63% in the single-debridement group and 66.66% in repeated debridements; p = 0.08).[26]

Hospital Stay: The mean duration of the hospital stay (DOHS) has been considered as important predictor of poor prognosis by most authors. El Bachir Benjelloun et al [2013] (median

hospitalization time (MHT) 21 days, range, 4–66, MHT for the survivor- 26.00 days, non-survivors 8 days, P <0.001),[26] M El-Shazly et al [2014] (median hospitalization time (MHT) survivor- 22.24 days, non-survivors 14.28 days, P < 0.01),[24] and Eskita\csc\io\uglu et al.[2014] (mean DOHS survivors= 33.73±17.30; non-survivor=61.6±38.9; p= 0.0110). Our study too showed similar results (mean DOHS survivors= 30.54±10.94; non-survivor= 11.13±3.14; p=<0.0001). However Satyajeet Verma et al. [2012] reported hospital stay to be non-significant predictor of the poor prognosis (>30 days of DOHS survivor group 41.7% cases and non-survivor group 52.9% cases, p=0.639832).[32] Gutiérrez-Ochoa J et al. has reported in his workup that aggressive therapy, age, co-morbidities and time of presentation do not affect prognosis and there is no consensus on clinical variables for predicting FG results.[39]

CONCLUSION

In conclusion Fournier's gangrene is a rapidly progressive fulminant infection and represents a surgical emergency. Understanding the physiopathology and predisposing factors is essential for early diagnosis. Hemodynamic stabilization, aggressive surgical debridement and broad-spectrum antibiotic therapy, is the key to good treatment. The mean age of our study population coincides with other studies; we did not have any female patients in our study. We have found that the FGSI score, increased age, TBSA involved, pre-hospital delay time, time between admission, sepsis at admission and first debridement to be important in predicting poor prognosis.. When we co-related laboratory and clinical findings, it was found that elevated heart and respiratory rates, increased total leukocyte count, rise of serum creatinine, urea and potassium levels, decreased serum sodium, albumin, serum bicarbonate and also anemia were associated with a bad evolution.

Frequent alcohol consumption, smoking and DM were found to be predominant predisposing factors but they did not influence the mortality. The treatment offered was similar to other studies. Emergency debridement and irrigation of the wound showed good post-operative results and a remarkable decline in deaths, mortality rate was 25%, a value which compared well with other studies.40 MODS due to ARF was the leading cause of mortality.

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