

Asymptomatic Urinary Tract infection (UTI) among diabetic females

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Abstract:

Our study was conducted on 1000 diabetic females of variable ages without symptoms of UTI. There were both type 1 and type 2 diabetic patients.

There were both married and unmarried females in both types of DM.

In addition to 100 normal females, which are age matched with patients group. They constituted control group.

Prevalence of ASB is significantly higher ($P < 0.01$), by 4-5 folds in diabetic females than in normal ones. Several risk factors have been identified as glucosuria, proteinuria and duration of DM, whereas age, duration of marriage and sexual activity are not proven to increase prevalence of ASB in diabetic females in our study. Repeated pregnancy times may be a risk factor for ASB in type 2 diabetic females ($P < 0.01$). Staph. aureus was present in 54% of bacteriuric patients (with positive cultures) with either types of DM and E.coli was present in 30.8% of bacteriuric patients with either types of DM. Staph aureus is present in 45.9% of patients with type 1DM, while in type 2 DM, it was present in 59.1% of patients. E.Coli was isolated in 41.2% of patients with type 1 DM and it was present in 24.2% of patients with type 2 DM.

Introduction

It is well established that individuals with diabetes are at higher risk than their non-diabetic counterparts for a variety of bacterial infections. High post voidal residual volumes related to bladder dysfunction increase the likelihood of urinary tract infections (*Sherita et al., 1999*).

Urinary tract infections can be asymptomatic or symptomatic (*Gonzalez and Schaeffer, 1999*).

Patients with diabetes generally have a 2-fold to 4-fold increased incidence of bacteriuria over patients without diabetes (*Ronald and Luduring, 2001*).

In contrast with man, a higher prevalence of ASB has been found in women with diabetes than in women without the disease (*Patterson and Androle, 1997*).

In a study of risks for UTI in diabetic women, sexual intercourse in the preceding week was the most important risk factor for women with type 1DM, whereas Asymptomatic bacteriuria (ASB) was associated with the highest risk among women with type 2DM (*Geerlings et al., 2000*).

The presence of asymptomatic bacteriuria is most strongly correlated with variables consistent with duration of diabetes rather than with control of diabetes (*Zhanel, Nicolle and Marding, 1995*).

The most common cause of UTI in men and women with DM is E.Coli (*Mansen et al., 1998*).

In voided urine samples obtained from patients with urinary tract symptoms, the finding of more than 10^5 organisms of a single bacterial species is highly predictive of infection (*Schrier, 2001*).

Patients and Methods

In our study, 1000 single or married variable aged females with either types of DM without symptoms of urinary tract infection were included.

The following cases were excluded from the study: cases with renal impairment, cases with symptomatic urinary tract infection, cases with structural urinary tract abnormalities, cases with disturbed immunity or receiving corticosteroid therapy and pregnant cases.

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There was also a control group of 100 age matched nondiabetic females (Both married and Unmarried).

All cases and controls were subjected to the following investigations:

1. Full history taking
2. Complete clinical examination.
3. Complete urine analysis (PH, Protein, WBCs, RBCs, Casts, sugar and other abnormal findings)
4. Urine culture for significant bacteriuria and isolation of the infectious agents.
5. Renal function tests (Serum BUN and Serum creatinine).
6. Abdominal sonography

Methods:

- Renal function tests were performed by standard methods in Ain Shams University Hospital laboratories.
- Abdominal sonography was performed by 2-dimensional ultrasonography in Radiology Department, Ain Shams University
- Complete urine analysis and urine culture were done in Ain Shams University Hospital Laboratories.

Midstream clean freshly voided urine specimens were collected for the evaluation of bacteriuria. Urine samples were immediately refrigerated and were cultured 2 hours after collection.

Urine culture was performed according to standard procedures: urine was screened with either a Uricult dipslide (Orion Diagnostica, Espoo, Finland) or a direct preparation (i.e. some urine was put on a slide and viewed with a 40x objective microscopy). If cfu in culture were = or $>10^5$ cfu/ml, being grown on the dipslide or more than 5 leukocytes or 10 micro organisms were seen on the slide, then the urine

(stored at 4°C) was plated onto split blood agar and MacConkey plates. Lower counts as 10^3 cfu/ml or 10^2 cfu/ml are detected by inoculating in a more diluted urine.

All urine specimens were plated using quantitative loops at Bosch Medicentrum. The results were read after 24 hours. Microorganisms were identified with viteck automated identification system (Bio Merieux, denBosch, the Nether lands). Glucosuria, leukocyturia and urinary PH values were determined using a dipslide method (Combur test, Boehringer Mannheim, Almere, the Netherlands).

Results:

In our study, 1000 single or married variable aged females with either types of DM without symptoms of urinary tract infection were included, in addition to 100 age matched non diabetic single or married females as a control group. Groups of the study are:

Group 1: With type 1 DM and single (unmarried) females, (200 cases).

Group 2: with type 1 DM and married females (200 cases).

Group 3: with type 2 DM and unmarried females (100 cases).

Group 4: With type 2DM and married females (500 cases).

Group 5: normal single (unmarried) females, as control group (32 controls).

Group 6: normal married females as control group (68 controls).

In our tables, the following data was used:

- RBCs in urine were measured per high power field.
 - WBCs in urine were measured per high power field.
 - Protein in urine was measured as followed:
 - + 1 protein in urine \rightarrow $<$ or $=$ 2.5 gm/24 hours
 - + 2 protein in urine = 2.5-5gm/24 hours
 - + 3 protein in urine = 5-7.5 gm/24 hours
- Sugar in urine was measured as follows:
- + 1 sugar in urine = 200 mg/dL
 - + 2 sugar in urine = 201 mg/dL – 350 mg/dL

+ 3 sugar in urine = 351 mg/dL – 500 mg/dL

The degrees of oedema were assessed as follows:

+ 1 LL oedema: till the ankle

+ 2 LL oedema: till the knee

+ 3 LL oedema : generalized oedema

- Hypertension was considered as present if blood pressure was > 130/90.

The following tables show the different results obtained from the study:

Table (1): Prevalence of ASB among the 6 groups of the study (chi-Square test):

Group	Negative Culture	Positive Culture	P value
Group 1 (n=200)	115 (57.5%)	85 (42.2%)	<0.01
Group 2 (n = 200)	113 (56.5%)	87 (43.5%)	
Group 3 (n=100)	63 (63%)	37 (37%)	
Group 4 (n=500)	265 (53%)	235 (47%)	
Total cases (n=1000)	556(55.56%)	444(44.4%)	
Group 5 (n=32)	31 (96.9%)	1 (3%)	
Group 6 N = 68	64 (94.1%)	4 (5.9%)	
Total Control (n=100)	95 (95%)	5 (5%)	

Table (2): Comparison of urine culture results between 6 groups of the study (Chi-Square test)

Group	-ve	Staph aureus	E-Coli	Actino bacter	Klebsiella pneumonia	Proteus	Diptheroid	Strept fecalis	Staph epidermidis
1 (n=200)	115 (57.5%)	29 (14.5%)	56 (28%)	-	-	-	-	-	-
2 (n=200)	113 (56.5%)	50 (25%)	15 (7.5%)	3 (1.5%)	10 (5%)	3 (1.5%)	3 (1.5%)	3 (1.5%)	-
3 (n=100)	63 (63%)	21 (21%)	6 (6%)	-	5 (5%)	-	-	5 (5%)	-
4 (n=500)	265 (53%)	140 (28%)	60 (120%)	-	30 (6%)	-	-	-	5 (1%)
5 (n=32)	31 (96.8%)	-	1 (3%)	-	-	-	-	-	-
6 (n=68)	64 (64.2%)	2 (2.9%)	2 (2.9%)	-	-	-	-	-	-
P value	<0.01								

Table (3): Comparison between the non bacteriuric females and the bacteriuric ones in group 1 (type 1DM unmarried females) as regards clinical data and urine analysis results.

	Negative culture N=115	Positive culture N = 85	Test	P-value
Age (in years)	18.1±2.11	19.2±2.1	T-test	<0.01
Duration of DM (in years)	3.7±2.4	5.3±2.3	T-test	<0.01
PH of urine (mean ± SD)	4.9±0.5	6±1	T-test	<0.01
RBCs in urine (mean ± SD)	3.2±3.8	16.2±14.2	T-test	<0.01
WBCs in urine (mean ± SD)	5.4± 5.8	97.2±23.6	T-test	<0.01
Protein in urine -ve +1 +2	110(95.7%) 5(4.3%) -	26 (30.6%) 48 (56.5%) 11 (12.9%)	Chi-Square test	<0.01
Sugar in urine -ve +1 +2 +3	67(66.11%) 28(24.31%) 5(4.3%) 6 (5.2%)	16(18.8%) 5 (5.9%) 29(34.1%) 35(41.2%)	Chi-Square test	<0.01

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Casts in urine -ve Epith.	112 (97.4%) 3 (2.6%)	83 (97.6%) 2 (2.4%)	Chi-Square test	<0.01
Crystals in urine -ve URate Ca Ox	107 (93%) 3 (2.6%) 5 (4.3%)	84 (98.8%) - 1 (1.2%)	Chi-Square test	0.134

Table (4): Comparison of clinical data and data of urine analysis with urine culture results in subgroups of group 1 (type 1 unmarried females)

	Staph aureus subgroup 1	E.Coli Subgroup 2	-ve subgroup 3	Test	P value
Age (in years)	18.8±2.1	19.3±2.2	18.1±2.1	Anova	<0.01
Duration of DM (in years)	4.8±2.1	5.5±2.4	3.7±2.4	Anova	<0.01
PH of urine	5.57±0.97	6.34±0.99	4.95±0.57	Anova	<0.01
RBCs in urine	9.48±8.44	19.67±15.41	3.21±3.81	Anova	<0.01
WBCs in urine	26.1±17.71	43.07±24.38	5.44±5.88	Anova	<0.01
Proteins in urine -ve +1 +2 +3	5(17.2%) 2(6.9%) 17(58.6%) 5(17.2%)	11(19.6%) 3 (5.4%) 12(12.4%) 30 (53.6%)	76(66.1%) 28(24.3%) 5(4.3%) 6(5.2%)	Chi-Square	<0.01
Sugar in urine -ve +1 +2 +3	5(17.2%) 2(6.9%) 17(58.6%) 5(17.2%)	11(19.6%) 3(5.4%) 12 (12.4%) 30(53.6%)	76(66.1%) 28(24.3%) 5(4.3%) 6(5.2%)	Chi-Square	<0.01
Casts in urine -ve Epith	27(93.1%) 2 (6.9%)	56(100%) -	112(92.4%) 3(2.6%)	Chi-Square	0.154
Crystals in urine -ve Urate Ca.Ox.	29(100%) - -	55(98.2%) - 1(1.8%)	107(93%) 3(2.4%) 5(4.3%)	Chi-Square	0.06447

Table (5): Comparison between the non-bacteriuric females and the bacteriuric ones in group 2 (type 1 DM married) as regards clinical data and urine analysis results

	Negative culture	Positive culture	Test	P value
Age (in years)	38.5±9	39.7±8.7	T-test	0.353
Duration of DM (in years)	18.7±9	19.2±7.6	T-test	0.701
Marriage duration (in ys)	15.4±9	16.5±9	T-test	0.402
Pregnancy times	3.9±2.2	4.1±2.6	T-test	0.491
PH of urine	5.2±0.6	5.2±0.6	T-test	0.740
RBCs in urine	3.1±4.7	3.4±1.8	T-test	0.592
WBCs in urine	3±2.8	16.1±10.6	T-test	<0.01
Protein in urine -ve +1 +2	83(73.5%) 6(5.3%) 1(0.9%)	17(19.5%) 16(18.4%) 1(1.1%)	Chi-Square	0.017
Sugar in urine -ve +1 +2 +3	83(73.5%) 14(12.4%) 9(8%) 7(6.2%)	17(19.5%) 4(4.6%) 16*18.4%) 50(57.5%)	Chi-Square	<0.01
Casts in urine -ve hyaline granular	110(97.3%) ---- 3 (2.7%)	85(97.7%) 1(1.1%) 1(1.1%)	Chi –Square	0.395
Crystals in urine -ve urate Ca.Ox.	7(6.2%) 5(4.4%) 101 (89.4%)	27(31%) ----- 60(69%)	Chi-Square	<0.01

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Table (6): Comparison of clinical data (history and examination with urine culture results in subgroups of group 2 (type 1 DM married) (by Anova test)

	STaph. aureus (Subgp 1)	E. coli (subgp 2)	-ve culture (subgp 3)	Acti bacter (Subgp 4)	Klebsiella Pneum. (subgp 5)	Proteus (subgp 6)	Diphteroid (Subgp 7)	Strept. Fecalis (Subgp 8)	P value
Age (in years)	38.32 ± 8.71	39.2 ± 10.15	38.58 ± 9.07	44 ± 4.04	43.1 ± 9.35	43.66 ± 7.09	46 ± 3.6	43.66 ± 2.08	0.5007
Duration of DM (in years)	18.46 ± 7.59	18.06 ± 7.79	18.778 ± 9	21.66 ± 9.07	23 ± 8.11	18 ± 11.35	22 ± 3.46	21.66 ± 4.93	0.8136
Marriage duration (in years)	15.38 ± 8.64	16.4 ± 9.84	15.46 ± 9.04	17.33 ± 8.5	19.90 ± 10.59	20 ± 10	20.66 ± 8.38	17 ± 9.64	0.7829
Pregnancy Times	4.79 ± 2.61	4 ± 2.23	4.34 ± 1.99	5.33 ± 2.51	4.60 ± 2.22	4.66 ± 3.05	4 ± 1	3.66 ± 0.57	0.8879

Table (7): Comparison of urine analysis results with culture results in subgroups of group 2 (type 1 DM married)

	Stap. Aureus (subgp 1)	E.Coli (Subgp 2)	-ve culture (Subgp 3)	Actino bacter (subgp 4)	Klebsiella pneum. (subgp 5)	Proteus (Subgp 6)	Diphteroid (Subgp +)	Strept. Fecalis (Subgp 8)	P value
PH of urine	5.3 ± 0.69	5.03 ± 0.66	5.26 ± 0.67	5.0 ± 1.0	5.2 ± 0.34	5.16 ± 0.28	5.33 ± 0.57	5.16 ± 0.76	(Anova) 0.8821
RBCs in urine	3.12 ± 1.45	3.40 ± 2.16	3.15 ± 4.71	2.66 ± 0.57	4.40 ± 2.22	5.66 ± 3.78	3.66 ± 1.15	4.0 ± 1.73	(Anova) 0.923
WBCs In urine	14.74 ± 5.40	20.73 ± 22.53	3.07 ± 2.81	13.66 ± 1.52	14.50 ± 4.52	27.33 ± 4.61	13.33 ± 9.07	16.66 ± 2.88	(Anova) <0.01
Protein In urine -ve +1 +2	43(86%) 6(12%) -	9(60%) 6(40%) -	106(93.8%) 6(5.3%) 1(0.9%)	3(100%) - -	10(100%) - -	- 2(66.7%) 1(33.3%)	3(100%) - -	1(33.3%) - -	(Chi-Square) <0.01
Sugar in urine -ve +1 +2 +3	5(10%) 2 (4%) 14(28%) 29(58%)	- - - 15(100%)	83(73.5%) 14(12.4%) 12(8%) 7(6.2%)	3(100%) - - -	8(80%) 2(20%) - -	- - - 3(100%)	1(33.3%) - 2(66.7%) -	- - - -	(Chi-Square) <0.01
Casts in urine -ve Hyaline granular	49(98%) 1(2%) -	15(100%) - -	110(97.3%) - 3(2.71%)	3(100%) - -	10(100%) - -	2(66.7%) - 1(33.3%)	3(100%) - -	3(100%) - -	(Chi-Square) 0.13125
Crystals in urine -ve Urate Ca OX.	35(70%) 15(30%) -	11(73.3%) 4(26.7%) -	101(89.4%) 7(6.7%) 5(4.4%)	3(100%) - -	5(50%) 5(50%) -	2(66.7%) 1(33.3%) -	1(33.3%) 2(66.7%) -	3(100%) - -	Chi-Square <0.01

Table (8): Comparison between the non-bacteriuric females and the bacteriuric ones in group 3 (type 2DM unmarried) as regards clinical data and urine analysis results.

	Negative culture N = 63	Positive culture N=37	Test	P value
Age (in years)	49.1±4.8	49.5±4.8	T-test	0.675
Duration of DM (in years)	4.4±3.8	5.4±3.7	T-test	0.223
Pregnancy times	4.4±2.3	5.6±1.6	T-test	0.01
LL.edema -ve +1 +2 +3	38 (60.3%) 22 (34.9%) 2 (3.2%) 1 (1.6%)	20(54.1%) 9(24.3%) 8(21.6%) -	Chi-Square	0.023
Hypertension +ve -ve	12 (81%) 51 (49.2%)	5(86.5%) 32(56.8%)	Chi-Square	0.477
Treatment Insulin Drugs	31(49.2%) 32 (50.8%)	21 (56.8%) 16 (43.2%)	Chi-Square	0.466
PH of urine	5.5±0.5	5.4±0.6	t-Test	0.665
Urine RBCs	3.3±2.9	2.6±1.5	T-Test	0.199
Urine WBCs	3.3±2.1	11.1±4.2	T-test	<0.01
Protein in urine +1 -ve	19(30.2%) 44(69.8%)	8(21.6%) 29(78.4%)	Chi-Square	0.245
Sugar on Urine +1 +2 +3 -ve	17(27%) 2(3.2%) 5(7.9%) 39(61.9%)	6(16.2%) 4(10.8%) 6(16.2%) 21(56.8%)	Chi-Square	0.172
Crystals in urine Urate Ca.Ox. -ve	8(12.7%) 6(9.5%) 49(77.8%)	15(40.5%) 4(10.8%) 18(48.6%)	Chi-Square	<0.01

Table (9): Comparison of clinical data with urine culture results in subgroups of group 3 (type 2 DM unmarried).

	Staph. aureus (subgp1)	E. Coli (Subgp 2)	-ve culture (Subgp3)	Klebsiella Pneum. (Subgp4)	Strept. Fecalis (Subgp 5)	P value
Age (in ys)	49.38 ± 5.39	52.16 ± 2.85	49.14 ± 4.87	48.8 ± 4.54	48 ± 4.69	(Anova) 0.6380
Duration of DM (in ys)	6.0 ± 4.31	5.33 ± 3.61	4.49 ± 3.88	4.0 ± 2.54	4.80 ± 2.49	(Anova) 0.5983
Edema of LL +1 +2 +3 -ve	5.(23.8%) 6(28.6%) - 10(47.6%)	2(33.3%) 1(16.7%) - 3(50%)	22(34.9%) 2(3.2%) 1(1.6%) 38(60.3%)	- 1(20%) - 4(80%)	2(40%) - - 3(60%)	Chi Square) 0.22435
Hypertension +ve -ve	2(9.5%) 19(90.5%)	1(16.7%) 5(83.3%)	12(19%) 51(81%)	1(20%) 4(80%)	1(20%) 4(80%)	Chi Square 0.89691
Treatement Insulin Drug	15(71.4%) 6(28.65%)	2(33.3%) 4(66.7%)	31(49.21%) 32(50.8%)	1(20%) 4(80%)	3(60%) 2(40%)	Chi Square 0.17186

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Table (10): Comparison of urine analysis results with urine culture results in subgroups of group 3 (type 2 DM unmarried).

	Staph aureus (subgp 1)	E. coli (Subgp 2)	-ve Culture (Subgp 3)	Klebsiella pneum. (Subgp 4)	Strept. Fecalis (Subgp 5)	P value
PH of urine	5.50 ± 0.63	5.41 ± 0.38	5.50 ± 0.59	5.7 ± 0.67	5.0 ± 0.61	(Anova) 0.4046
Urine RBCs	2.42 ± 1.28	2.66 ± 1.63	3.34 ± 2.94	3.80 ± 2.49	2.60 ± 0.89	(Anova) 0.5900
Urine WBCs	10.95 ± 3.51	11.66 ± 7.11	3.31 ± 2.16	12.20 ± 5.06	10.40 ± 3.20	(Anova) <0.01
Protein in urine +1 -ve	1(4.8%) 20(95.2%)	1(16.7%) 5(83.3%)	19(30.2%) 44(69.8%)	4(80%) 1(20%)	2(40%) 3(60%)	Chi-Square <0.01
Sugar in urine +1 +2 +3 -ve	- 4(19%) 5(23.8%) 12(57.1%)	1(16.7%) - 1(16.7%) 4(66.7%)	17(27%) 2(3.2%) 5(7.9%) 39(61.9%)	3(60%) - - 2(40%)	2(40%) - - 3(60%)	(Chi – square) <0.05
Crystals in urine -ve Urate Ca OX	9(42.91%) 12(57.1%) -	4(66.7%) 1(16.7%) 1(16.7%)	49(77.8%) 8(12.7%) 6(16.7%)	3(60%) 2(40%) -	2(40%) - 3(60%)	Chi-Square <0.01

Table (11): Comparison between the non-bacteriuric females and the bacteriuric ones in group 4 (type 2 DM married) as regard clinical data and urine analysis results.

	Negative culture N=265	Positive culture N=235	Test	P value
Age (in years)	50.9±4.9	51.1±5	T-test	0.660
Duration of DM (in years)	4.4±2.9	5.8±3.5	T-test	<0.01
Marriage duration (in years)	28.9±5.7	29.2±5.3	T-test	0.457
Pregnancy times	4.5±1.9	6.4±2.7	T-test	<0.01
L.L. edema +1 +2 -ve	38(14.3%) 46(17.4%) 181(68.3%)	48(20.4%) 27(11.5%) 160(68.1%)	Chi-Square	0.060
Hypertension +ve -ve	83(31.3%) 182(68.7%)	55(23.4%) 180(76.6%)	Chi-Square	0.048
Treatment. Insulin Drugs	123(46.4%) 142(53.6%)	76(32.3%) 159(67.7%)	Chi-Square	0.048
PH of urine	5.3±0.6	5.4±0.7	T-test	0.105
Urine RBCs	3.4±2	3.5±3.7	T-test	0.630
Urine WBCs	3.8±2.1	14.5±7.5	T-test	<0.01
Protein in urine +1 +2 -ve	46(18.4%) 2(0.8%) 217(81.9%)	40(17%) 14(6%) 181(77%)	Chi-square	<0.01
Sugar in urine +1 +2 +3 -ve	44(16.6%) 38(14.3%) 72(27.2%) 111(41.9%)	26(11.1%) 32(13.6%) 75(31.9%) 102(43.4%)	Chi-Square	0.284
Crystals in urine Ca.-Ox. Urate -ve (Ca.Ox. + Urate)	35(13.2%) 49(18.5%) 181(68.3%)	15(6.4%) 54(23%) 165(70.2%) 1(0.4%)	Chi-Square	0.04
Crystals in urine -ve granular epith	253(95.5%) 2(0.8%) 10(3.8%)	215(91.5%) 10(4.3%) 10(4.31%)	Chi-Square	0.036

Table (12): Comparison of clinical data with urine culture results in subgroups of group 4 (type 2 DM married)

	Staph aureus (subgp 1)	E.coli (subgp 2)	-ve culture (subgp3)	Klebsiella pneumonia (Subgp 4)	Staph Epidermidis (subgp 5)	P value
Age (in ys)	51.25±4.95	51.46±5.19	50.95±4.98	50.36±5.55	49.40±3.78	(Anova) 0.7690
Duration of DM (in ys)	5.85±3.51	6.15±3.60	4.45±2.98	5.63±3.24	5.0±3.53	(Anova) <0.01
Marriage duration	29.27±5.39	29.55±5.28	28.91±5.76	29.10±5.92	27.80±2.16	(Anova) 0.898
Pregnancy times	6.03±2.49	7.39±2.51	4.65±1.85	7.36±3.04	7.40±2.70	(Anova) <0.01
Edema of LL +1 +2 -ve	32(22.9%) 18(12.9%) 90(64.3%)	10(16.7%) 9(15%) 41(68.3%)	38(14.3%) 46(17.4%) 181(68.3%)	5(16.7%) - 25(83.3%)	1(20%) - 4(80%)	Chi-Square 0.14
Hypertension +ve -ve	40(28.6%) 100(71.4%)	9(15%) 51(85%)	83(31.3%) 182(68.7%)	5(16.7%) 25(83.3%)	1(20%) 4(80%)	Chi Square 0.1718
Treatment Insulin Drug	40(28.6%) 100(71.4%)	20(33.31%) 40(66.7%)	123(46.4%) 142(53.6%)	15(50%) 15(50%)	1(20%) 4(80%)	Chi Square <0.01

Table (13): Comparison between urine analysis parameters and urine culture results in subgroups of group 4 (type 2 DM married)

	Staph. aureus (subgp 1)	E. coli (Subgp 2)	-ve Culture (Subgp 3)	Klebsiella pneum. (Subgp 4)	Strept. Fecalis (Subgp 5)	P value
PH of urine	5.47±0.77	5.55±0.76	5.38±0.67	5.40±0.68	5.9±0.54	(Anova) 0.2503
Urine RBCs	3.37±4.4	3.28±1.95	3.40±2.59	4.20±2.32	6.60±4.33	(Anova) 0.91
Urine WBCs	14.92±6.82	12.63±9.34	3.86±2.16	16.36±6.93	15±5.19	(Anova) <0.01
Protein in urine +1 +2 -ve	25(17.9%) 9(6.4%) 106(75.7%)	10(16.7%) 5(8.3%) 45(75%)	46(17.4%) 2(0.8%) 217(81.9%)	5(16.7%) - 25(83.3%)	- - 5(100%)	Chi-Square <0.05
Sugar in urine +1 +2 +3 -ve	- 16(11.4%) 50(35.7%) 74(52.9%)	20(33.37%) 5(8.3%) 15(25%) 20(33.3%)	44(16.6%) 30(14.3%) 72(27.2%) 111(41.9%)	5(16.7%) 10(33.3%) 10(33.3%) 5(16.7%)	1(20%) 1(20%) - 3(60%)	(Chi – square) <0.01
Casts in urine -ve Epith Granular	130(92.9%) - 10(7.1%)	55(91.7%) 5(8.3%) -	253(95.5%) 10(3.8%) 2(0.8%)	25(83.3%) 5(16.7%) -	5(100%) - -	Chi-Square <0.01
Crystals in urine -ve Ca.Ox urates (urates + CaOx.)	91(65%) 10(7.1%) 38(27.1%) 1(0.7%)	44(73.3%) 5(8.3%) 11(18.3%) -	181(68.3%) 35(13.2%) 49(18.5%) -	25(83.3%) - 5(16.7%) -	5(100%) - - -	(Chi Square) 0.4656

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Table (14): Comparison between group 1 (Type 1 diabetic unmarried females) and control group 5 (normal unmarried females) as regards clinical data, urine analysis and urine culture by using T-test

	Group 1 (n=200)	Group 5 (n=32)	(T-test) P value
Age (in ys)	18.5±2.2	33.9±13.4	<0.01
Urine analysis			
PH	5.4±0.9	5.2±0.5	0.343
Urine RBCs	8.7±11.6	3.4±2	0.01
Urine WBCs	18.9±22.4	3.2±2.7	<0.01
Urine culture results			
Staph. aureus	29(14.5%)	-	-
E.Coli	56(28%)	1(3%)	<0.01
-ve	115(57.5%)	31(96.90%)	<0.01

Table (15): Comparison between group 2 (type 1 diabetic married females) and control group 6 (normal married females) as regards clinical data, urine analysis and urine culture by using T-test

	Group 2 (n=200)	Group 6 (n=68)	T-test P value
Age (in years)	39.1±8.9	44.8±7.8	<0.01
Marriage duration (in years)	15.9±9	21.8±7.1	<0.01
Pregnancy times	4.4±2.1	4.7±1.7	0.437
Urine analysis			
PH of urine	5.2±0.6	5.1±0.4	0.073
Urine RBCs	3.2±3.7	2.5±1.6	0.127
Urine WBCs	8.7±9.8	3.2±3.4	<0.01
Urine culture results			
Staph aureus	50(25%)	2(2.9%)	<0.01
E. Coli	15(7.5%)	2(2.9%)	<0.01
-ve	113(56.5%)	64(94.1%)	<0.01
Actinobacter	3(1.5%)	-	-
Klebsiella	10(5%)	-	-
Proteus	3(1.5%)	-	-
Diptheroid	3(1.5%)	-	-
Strept. Fecalis	3(1.5%)	-	-

Table (16): Comparison between group 3 (type 2 diabetic unmarried females) and control group 5 (normal unmarried females) as regards clinical data, urine analysis and urine culture by using T-test

	Group 3 (n=100)	Group 5 (n=32)	(T-test) P value
Age (in years)	49.3±4.8	33.9±13.4	<0.01
Urine analysis			
PH of urine	5.4±0.5	5.2±0.5	0.343
Urine RBCs	3.1±2.5	3.4±2	0.456
Urine WBCs	6.2±4.9	3.2±2.7	<0.01
Urine culture results			
Staph aureus	21(21%)	-	-
E.Coli	6(6%)	1(3%)	<0.01
-ve	63(63%)	31(36.90%)	<0.01
Klebsiella	5(5%)	-	-
Strept. Fecalis	5(5%)	-	-

Table (17): Comparison between group 4 (type 2 diabetic married females) and control group 6 (normal married females) as regards clinical data, urine analysis and urine culture using T-test.

	Group 4 (n=500)	Group 6 (n=68)	(T-test) P value
Age (in years)	51±5	44.8±7.8	<0.01
Marriage duration	29±5.5	21.8±7.1	<0.01
Pregnancy times	5.5±2.4	4.6±1.7	<0.01
Urine analysis			
PH of urine	5.4±0.7	5.1±0.4	<0.01
Urine RBCs	3.4±2.9	2.5±1.6	0.14
Urine WBCs	8.8±7.6	3.2±3.4	<0.01
Urine culture results			
Staph. aureus	140(28%)	2(2.9%)	<0.01
E.Coli	60(12%)	2(2.9%)	<0.01
-ve	2.65(53%)	64(94.1%)	<0.01
Klebsiella	30(6%)	-	-
Staph. epider	5(1%)	-	-

Discussion

Diabetes has long been considered to be a predisposing factor for UTI. Many UTIs are asymptomatic, especially in women. (*Bonadio et al., 2004*).

Escherichia coli was found to be the most prevalent organism in diabetic women with asymptomatic bacteriuria (*Geerlings et al., 2001*).

In the present study, we found the prevalence of asymptomatic bacteriuria in diabetic females (study group), was 44.4% (444 of 1000 diabetic females), as shown in table (1). In the control group, we found that the prevalence of asymptomatic bacteriuria is 5% (5 of 100 normal females), as shown in table (1).

The above results were higher than that mentioned in the report of (*Foman, 2002*). He reported that the prevalence of ASB among general public is estimated at 3.5% and that patients with diabetes generally have a 2-fold to 4-fold increased incidence of bacteriuria over patients without diabetes, estimated at (7-14%). Our results were near to the results reported by *Geerlings et al. (2000(1))*, that the prevalence of ASB was 26% in diabetic women and 6% in the control normal subjects.

Nicolle (2001), also reported that there is an increased prevalence of ASB in diabetic women, but not in diabetic men. The prevalence was 3 times more than in non-diabetic women (from 2 to 5% of young, sexually active women have ASB),

but a wide range, from 0 to 29% has been reported in different diabetic population.

Ludwing, (2000) reported that the prevalence of ASB in diabetic women may be only twice than in non-diabetic women. On the other hand, (*Bonadio et al., 2004*) reported that the frequency of significant bacteriuria was 17.5% among women with diabetes and 18.5% in women in the normal control group. The higher prevalence in Egypt may be due to bad hygienic measures and poverty, lack of medical advice of the general population, negligence of women to ask for medical advice when minor symptoms appear and inadequate treatment of symptomatic UTI leading to subtle infection and ASB.

In our study, we found that prevalence of ASB in type 1 diabetic females (group 1 and 2), was 43% (172 of 400 patients), while in type 2 diabetic females (group 3 and 4) was 45.3% (273 of 600 patients), as shown in table (1). All of the above investigators didn't comment on a difference in the prevalence of ASB between the two types of D.M. and this goes with our results.

In our study, age was a risk factor ($P<0.01$) only in group 1 when comparing bacteriuric females (19.2 ± 2.1 years) with non-bacteriuric females (18.1 ± 2.1 years), while age didn't show any significant difference between bacteriuric and non-bacteriuric females in the other 3 groups. On the contrary (*Geerlings et al., 2000(1)*),

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has reported that age is a well known risk factor for bacteriuria in non-diabetic women. Age was also the most important risk factor for ASB in type 2 diabetic patients in this study. *Andriole (2002)*, didn't confirm in his study that age was a major risk factor. In our study, there was an increased prevalence of *E.coli*. ASB ($P<0.01$), specially in group 1, as shown in table (4).

In our study, duration of DM was found to be a risk factor for increased prevalence of ASB ($P<0.01$) in group 1 between bacteriuric females with mean duration of DM (5.3 ± 2.3 years) as compared to non-bacteriuric females with mean duration of DM (3.7 ± 2.4 years), (table 3). This was more associated with increased prevalence ($P<0.01$) of *E.coli* ASB, especially so with mean duration of DM of (5.5 ± 2.4 years), as shown in (table 4). Also, in group 4, duration of DM was associated with increased prevalence of ASB ($P<0.01$), with a mean of (5.8 ± 3.5 years) in bacteriuric females as compared to non-bacteriuric females with a mean of (4.4 ± 2.9 years), (table 11).

This was more evident in case of *E.Coli*. ASB ($P<0.01$) with a mean duration of DM of (6.1 ± 3.6 years) as in (table 12). In group 2 and group 3, duration of DM was not associated with increased prevalence of ASB, in our study.

In the study done by *Mendoza et al. (2002)*, women with positive cultures had a longer duration of DM than those with negative cultures *Nicolle (2001)*, reported that the presence of asymptomatic bacteriuria is most strongly correlated with variables consistent with duration of diabetes, rather than of diabetes itself.

Stapleton (2002), mentioned that studies from the Netherlands, showed that risk for UTI among women with type 1 diabetes included a longer duration of DM. *Geerlings, (2000(1))*, suggested that longer duration of DM was a risk factor for ASB in women with type 1 DM. Therefore, a longer duration of DM, with presence of complications, seemingly increases the risk of ASB in type 1 diabetic women.

In our study, duration of marriage was not associated with increased prevalence of ASB in group 2 ($P=0.402$) and group 4 ($P=0.457$) of the study, between bacteriuric females as compared to non-bacteriuric females (tables 5 and 11).

In group 2, repeated pregnancy was not associated with increased prevalence of ASB with a mean of (4.1 ± 2.6) in bacteriuric females and (3.9 ± 2.2) in non-bacteriuric females, ($P=0.491$), (table 5). Pregnancy times were found to be a risk factor for increased prevalence of ASB in group 4 ($P<0.01$) with a mean of (6.4 ± 2.7) in bacteriuric females as compared to non-bacteriuric females (table 11). This means that increased pregnancy times was associated with increased prevalence of ASB in type 2 diabetic females. No other studies suggested this finding. On the contrary, *Nicolle (1999)* reported that no association exists between parity and prevalence of ASB in diabetic women.

Insulin therapy was associated with lower prevalence of ASB than those treated by antidiabetic drugs ($P<0.01$), in group 4 (Table 11).

There was no significant difference in group 3, as regards type of treatment of DM, ($P=0.466$). Group 1 and Group 2 are regularly treated by Insulin only. No other studies found stressing on this point, as a risk factor for prevalence of ASB in diabetic females.

L.L. edema and hypertension were only present in group 3 and group 4. In our study, group 3 had a significant difference between bacteriuric and non bacteriuric females ($P=0.023$), as regards L.L. edema, but not to the point of considering L.L. edema a risk factor for ASB in diabetic females, because L.L. edema were sometimes more prevalent in bacteriuric females and sometimes more prevalent in non-bacteriuric females (Table 8). In group 4, L.L. edema was not associated with increased prevalence of ASB, ($P=0.060$) in (Table 11). In group 3, hypertension was not found to be a risk factor for development of ASB in diabetic females (Table 8), whereas there was a significant statistical difference ($P =0.048$) between

non-bacteriuric and bacteriuric females in group 4, regarding hypertension. This means that hypertension may not be considered as a risk factor for ASB in diabetic females, as non-bacteriuric females were more prone to be hypertensive (31.3%) in our study than the bacteriuric ones (23.4%) (Table 11). No studies were done regarding association between LL edema and hypertension and prevalence of ASB in diabetic females.

Increased urine PH was associated in our study with increased prevalence of ASB ($P<0.01$), only in group 1, with a mean of (6 ± 1) in bacteriuric females as compared to the non bacteriuric females with a mean of (4.9 ± 0.5), (table 3). Higher PH was recorded in cases of E.Coli ASB, with a mean of (6.3 ± 0.9) and Staph aureus ASB with a mean of (5.5 ± 0.9) in bacteriuric females than in non bacteriuric females of group 1, (Table 4), with a mean of (4.9 ± 0.5), (Table 3).

All other groups didn't show significant statistical difference between bacteriuric and non-bacteriuric females. This finding in group 1 may be due to other Comorbid conditions, environmental factors or dietary factors elevating urine PH.

The mean RBCs count in urine was significantly higher in bacteriuric females ($P<0.01$) only in group 1 with a mean of (16.2 ± 14.2 RBCs/HPF) as compared to non-bacteriuric females with a mean of (3.2 ± 3.8 RBCs/HPF), in (table 3), while in other groups, there was non significant difference between bacteriuric and non bacteriuric females. No other studies included this item as a risk factor for ASB in diabetic females.

The mean WBCs count in urine was significantly higher in bacteriuric females than in non-bacteriuric females ($P<0.01$) in the four groups (both type 1 and 2 DM, both married and unmarried), in tables (3), (5), (8) and (11), for groups 1, 2, 3 and 4 respectively. This goes in agreement with *Zhanel, Nicolle and Harding, (1995)*, who reported the mean urinary leukocyte count was significantly higher ($P<0.01$) in bacteriuric females than in non-bacteriuric ones.

In this study, bacteriuric subjects had significantly higher urinary leukocyte counts if E.Coli or streptococcus species were isolated, than they did if staphylococcus species, mixed bacteria or *Gardenerella vaginalis* were isolated ($P<0.05$). This means that pyuria is somehow affected by the type of organism causing ASB.

In our study, presence of proteinuria (macroalbuminuria, increased the prevalence of ASB in group 1 ($P<0.01$) between bacteriuric and non bacteriuric females, (table 3). Also, it was increased in group 2 ($P=0.017$) between bacteriuric and non bacteriuric females, (table 5) and in group 4 ($PH<0.01$) between bacteriuric females and non-bacteriuric females, (table 11). It was nonsignificant in group 3 ($P=0.245$), (table 8). This goes in agreement with *Geerlings et al. (2000(1))* who found that macroalbuminuria is one of the risk factors for ASB in women with type 1 diabetes, as macroalbuminuria may increase the vulnerability to bacterial attacks, resulting in an increased risk of developing ASB.

Glucosuria was associated with increased prevalence of ASB in type 1 DM groups (group 1 and 2), of our study. In group 1, bacteriuric females had significantly higher degrees of glucosuria ($P<0.01$) than non-bacteriuric females, (table 3).

In group 2, significant differences ($P<0.01$) were present in different degrees of glucosuria between bacteriuric and non bacteriuric females (table 5) there were nonsignificant differences concerning different degrees of glucosuria between bacteriuric and non bacteriuric females, in group 2 and group 4, (table 8) and table (11) respectively. *Geerlings and associates, (1999)* found that moderate and severe glucosuria (glucose concentrations between 100 and 1000 mg/dl), enhanced bacterial growth in vivo. The authors concluded that glucosuria may be a factor contributing to increased prevalence of bacteriuria in diabetic patients. *Bonadio et al. (2004)*, stated that, in vitro studies showed that glucosuria enhances the growth of different E.Coli strains. However, this was not confirmed by in vivo studies, which failed to show a higher prevalence of ASB among

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diabetic patients with glucosuria as compared with those without glucosuria.

Casts in urine showed a significant difference between bacteriuric females and non-bacteriuric females, only in group 4 ($P=0.036$), with both epithelial and granular casts (table 11). Epithelial casts in urine were associated with increased prevalence of ASB ($P<0.01$) caused by *Klebsiella pneumoniae* and *E.Coli* in group 4, (table 13).

Also, in group 4, granular casts in urine were associated with increased prevalence of ASB caused by *staph. aureus* ($P<0.01$), Table (13).

This result may be due to the presence of other factors in group 4, with the highest mean age (51 ± 5 years), may be leading to more morbidity than the other groups. No other studies commented on the significance of presence of casts in urine as regards prevalence of ASB in diabetic females.

Prevalence of crystals (mostly urate crystals) in urine was found to be associated with increased prevalence of ASB in group 2 ($P<0.01$), (Table 5), in group 3 ($P<0.01$), (table 8), and in group 4 ($P=0.04$), (table 11). The significant difference was between bacteriuric females as compared to non-bacteriuric females. No significant differences were found in group 1. Crystals of calcium Oxalate have shown similar pattern. This was not reported in other studies, but also showed that crystalluria may be a predisposing factor for ASB.

In our study, we used the history of marriage and its duration as a reflection of sexual activity. We found that prevalence of ASB in unmarried females (from either types of DM) was 37.3% (112 of 300), with higher prevalence in type 1 DM (45.2%) than in type 2 (37%), ($P<0.01$) as in (table 1). On the contrary, married diabetic females had a prevalence of ASB of 37.85% (265 of 700) as in (table 1), with higher prevalence in type 2 DM (40%) than in type 1 DM (32.5%), ($P<0.01$) as in (table 1). From these results, sexual activity (marriage in our study) is not considered a risk factor as regards the prevalence of ASB in diabetic females, although it may be so in normal ones. *MCCue (1999)*, reported that during adolescence and premenopausal adulthood, the prevalence of bacteriuria and

symptomatic UTI rises sharply in females coincident with the onset of sexual activity. *Hooton et al. (2000)*, reported observations from a study of two groups of young, sexually active women who were followed at regular intervals for six months. The prevalence in the two groups was 5% and 6%, rates consistent with those found in earlier studies. The most important behavioral risk factors for asymptomatic bacteriuria were the same as those identified for symptomatic urinary tract infection (*Geerlings et al., 2000(1)*), on the other hand, reported that studies have demonstrated in women with and without diabetes that recent sexual intercourse, the use of a diaphragm or the use of spermicide coated condoms, increases the risk of developing bacteriuria. Higher age and lower frequency of sexual intercourse of the patients in our study were probably the reasons for the absence of an association between sexual intercourse and bacteriuria.

As regards prevalence of organisms detected by cultures, we found that the prevalence of *E.Coli* causing ASB was 30.8% (137 of 444) bacteriuric females with either types of DM. (table 2). In group 1, prevalence of *E.Coli* ASB was 65.8%, in group 2 it was 17.2% in group 3 it was 16.2% and in group 4, it was 25.5%. In type 1 DM, it was 41.2% and in type 2 DM, it was 24.2%. In the study made by *Geerlings et al. (2000 (1))*, *E.Coli* was isolated in 42% of bacteriuric females with either types of DM.

Ludwing (2000), reported that the increased adherence of bacteria to uroepithelial cells in diabetic women is to blame for the increased prevalence of UTIs and asymptomatic bacteriuria. *E.Coli*, in diabetic patients as in others, is the most common uropathogen, accounting for more than half of cases.

As shown in table (2), prevalence of *Staph. aureus* ASB was 54% of bacteriuric women in either types of DM, being 45.9% in type 1 DM, while in type 2 DM it was 59.1%. This higher prevalence of *staph aureus* ASB is not recorded in any previous study, which may be due to bad hygiene in our country, especially in low socioeconomic class patients (at the endocrinology clinic of Ain Shams University Hospital).

Other microorganisms, as in (table 2), found in our study, were isolated in small percentages of no significance as compared to E.Coli and Staph aureus.

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إصابة المسالك البولية بالعدوى البكتيرية غير المصحوبة بأعراض بين الإناث المصابات بداء البول السكرى

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قمنا بدراسة 1000 أنثى مصابات بداء البول السكرى من مختلف الأعمار والآتي لا يشتكين من أعراض إصابة الجهاز البولى بالعدوى البكتيرية دون أعراض المريضات كن من نوعى البول السكرى الأول والثاني وكذلك كان هناك متزوجات وغير متزوجات من بينهن.

قمنا أيضا بدراسة 100 أنثى طبيعية من اعمار متقاربة لأعمار المريضات وكن موجودات كمجموعة ضابطة .

نسبة الاصابة بعدوى المسالك البولية البكتيرية الغير مصاحبة بأعراض كانت أعلى بمعدل 4-5 أضعاف فى مريضات البول السكرى عنها فى الصحيحات.

وجدنا عدة عوامل مساعدة لحدوث هذا منها وجود نسبة عالية من السكر فى البول ، وجود بروتينات فى البول ومدة الاصابة بداء البول السكرى ، فى حين ان العمر ومدة الزواج والنشاط الجنسى لم يثبت فى دراستنا انها تزيد من اصابة المسالك البولية بالعدوى البكتيرية الغير مصحوبة بأعراض فى مريضات البول السكرى .

تعد مرات الحمل قد يكون عامل مساعد للاصابة البكتيرية للمسالك البولية الغير مصاحبة بأعراض فى النوع الثانى من مريضات البول السكرى ميكروب بستانف اورياس كان موجود فى 54% من مريضات المصابات بالبول السكرى اللاتى أعطين نتيجة موجبة لمزرعة البول ، من النوعين الاول والثانى معاً ، وبنسبة 45.9 % فى النوع الاول ، 59.1 % فى النوع الثانى لداء البول السكرى ميكروب ايشيريشيا كولى كان موجود فى 30.8 % من مريضات البول السكرى اللاتى أعطين نتيجة موجبة لمزرعة البول من النوعين الاول والثانى معاً ، بنسبة 41.2 % فى النوع الاول ، و 24.2 % فى النوع الثانى لداء البول السكرى