Table (3) : Pus cells : Classificationaccording to sperm count/ml Through Five orders of pus cells Prevalence From (265 Seminal Fluid)

Pus Cells /H.P.F.	0 - 5	10 - 20	20 - 50	Over 50	Over 1oo
Total= 265	60	157	19	9	20
Normospermia (Normal) = 146 40 – 150 million)	20	93	13	5	15
(Normospermia (Moderate) =26 (20 – 40 million)	10	10	3	2	1
Olegospermia (Weak) =73 (1 – 20 million)	16	51	2	1	3
20 =Asospermia (no sperm)	14	3	1	1	1

Table(4): Classification of Sperm count cases according toViability in four Orders of sperm count/ ml (265 Sample)

Viability% / Count Sperm ranges	Active = 167 (50– 100%)	Sluggish = 48 (10 – 50 %)	Dead = 30
Normospermia (Normal) (40 – 150 million) = 146	110	20	16
Normospermia (Moderate) (20 – 40 million)=26	15	8	3
Olegospermia (Weak) ((1 – 20 million) = 73	42	20	11
Azospermia (no sperm) = 20	Zero	Zero	Zero

Table (5): Number of Seminal Fluid Infection in relation to Sperm Density and Percentage (500 Sample). Tested .:

Sperm	Sperm Density ($x10^6$ cell/ml)	No.of patients	No.of Infected	Percentage(%)
Normospermia	>20	209	31	14.8 %
Oligospermia	2-19	145	51	35.2%
Oligospermia	<2.0	59	26	44.1%
Azoospermia	NIL	87	66	75.8 %
Total		500	174	34.8%

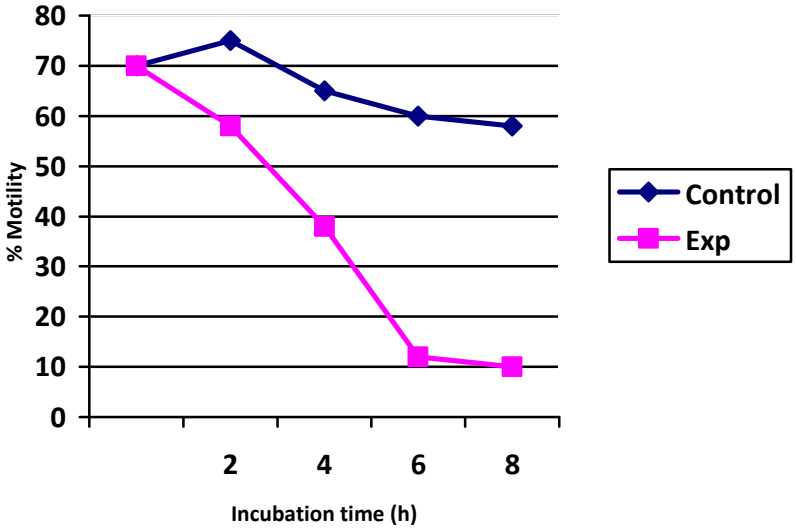
Pathogenic Organisms Isolated From Seminal Fluid :(68 pure Culture)

Table (6) Identification and percentage of bacteria as etiological agents of seminal tract infection:-

Organism	No. of isolates	Percentage of isolates
1)Gram negative microorganism		
Escherichia coli	31	45.6%
Proteus vuligars	3	4.4%
Pseudomonas aeruginosa	3	4.4%
2)Gram positive microorganism		
Streptococcus faecalis	16	23.5%
Streptococcus pyogenes	2	2.9%
Staphylococcus aureus	10	14.7%
Staphylococcus epidrmids	3	4.4%

Table(8) MBC (Minimal bactericidal Concentration) of antimicrobial agents

Antimicrobial agent :	MBC(µg/ml)
Amoxycillin	15 ug/ml
Ciprofloxacin	33 ug/ml
Erythromycin	10 ug/ml
Gentamicin	4 ug/ml
Penicillin G	10 ug/ml



DISCUSSION

The present study demonstrated that human sperm motility and viability was affected by E. coli. Also E. coli isolated from the semen of infertile male produced profound depression in the motility of human spermatozoa by agglutination in vitro. Schirren & Zander (1966) in past reported a negative influence of E. coli on sperm motility after mixing sperm and bacteria in vitro. This negative influence was confirmed by several authors (Auroux, et al., 1991)., (Teague, et al., 1971) were the first to claim high numbers of bacteria as causative for this effect. After addition of antibiotics to the incubation medium, all bacterial effects on sperm motility disappeared. Bacterial growth may be one cause of contradictory conclusions concerning the effective sperm/bacteria ratio. The absence of sperm agglutination was also observed after heat treatment of bacteria. In one of the earlier reports on effect of heat killed bacteria on spermatozoa characteristics (Kaur, et al., 2010) had studied that the live pathogenic S. aureus obtained from cervical cultures decreases in motility and viability of human spermatozoa which was absent in the sets in which the bacteria had been killed by boiling for 30 min before mixing the ejaculate. Another study by (Hosseinzadeh et al., 2010) reported that elementary bodies of Chlamydia trachomatis Serovar E can induce premature sperm death upon co-incubation with human sperm but this effect is abolished when these EBs have been previously killed by heat treatment. Alternatively, effect of antimicrobial agents on bacteria was also observed. Treatment of cultured bacteria with different groups of antibiotics abolished the effect. This indicates that there must be some adhesion sites on spermatozoa to which E. coli binds. The mechanisms by which E. coli affect sperm functions have not yet been identified. Interference of E. coli with these receptors may influence the motility and viability. Other authors reported similar observations concerning sperm motility and agglutination after incubation with E. coli (Auroux, et al., 1991).

Thus, we suggest that E. coli/spermatozoa-interaction may be a two-step process: i.e. adhesion to and subsequent destruction of the sperm membrane. This mechanism may account for any inhibitory effects of E. coli infection on

male fertility. It is speculative whether the same mechanisms are also responsible for impaired male fertility in cases of genitourinary infection

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