Original Research Article

Effect of mast cell blocker Ebastin in male infertility due to oligo-zoospermia and asthenozoospermia

Deeba F[1*], Banu J[2], Ishrat S[3], Anawary S A[4], Begum N[5]

[1,3,5]Associate Professor. [2]Chairman, [4]Assistant Professor, Department of Reproductive Endocrinology and Infertility, Bangabandhu Sheikh Mujib Medical University, Dhaka, BANGLADESH.
Email: deeba_51@yahoo.com

Abstract

Background: Approximately 50% of infertility is due to male factors, with a 36% prevalence of semen abnormalities. Mast cells (MC) are effectors cells of the immune system and play a crucial role in anaphylactic reactions, they are also critically involved in the pathophysiology of fibrotic disorders. Sperm count below 15 x 10^6 sperm/ml is called oligozoospermia and less than 40% sperm with forward progression or less than 32% with rapid forward progression is called asthenozoospermia. The right treatment of male infertility is relatively costly procedure. In our country due to ignorance of the patients, lack of sharp cut technology in every level sometime people are misguided by various type of treatment procedures. **Aim of the study:** The aim of this study was to find out the Mast cell blocker (Ebastin) is effective in improvement of oligozoospermia and asthenozoospermia in infertile male. **Methods:** This was a longitudinal clinical trial study and was conducted in the Infertility Unit outdoor, Department of obstetrics and gynecology, Bangabandhu Sheikh Mujib Medical University, Dhaka. Bangladesh during the period from January 2015 to December 2015. Finally, 334 cases were enrolled in this study. Among the male with abnormal semen parameter 168 patients were Asthenozospermic, 106 patients were Oligospermic and 60 patients were combined Oligo-asthenozospermic. Statistical analysis was carried out by using the Statistical Package for Social Sciences version 16.0 and MS-Excel 2016. **Result:** From 334patients in group II, mean sperm count was 10.19±1.75 million/ml in pretreatment and 10.18±1.95 million/ml in post treatment. 63.9% oligospermic male showed improvement in sperm count in ebastin group and 18.1% in placebo group. Which was statistically significant ($p<0.05$). In group I, mean sperm total mortality was 24.86±12.02% in pretreatment and 34.29±9.35% in post treatment. The difference was statistically significant ($p<0.05$). In group II, mean sperm total mortality was 21.96±6.17% in pretreatment and 22.44±4.81% in post treatment. The difference was not statistically significant ($p>0.05$). Improved sperm motility was 75.4% of asthenozospermic male in ebastin group and 19.3% in placebo group. Which was statistically significant ($p<0.05$). **Conclusion:** sperm count, Sperm motility and rapid progress had significantly improved after three months' treatment period with Ebastin. Therefore, this study suggested that mast cell blocker ebastin can be helpful to improve the patients' sperm count and sperm motility with oligospermia and asthenospermia.

Keywords: Male infertility, Mast cells, oligozoospermia, asthenozoospermia, Ebastin

[*]**Address for Correspondence:**
Dr Farzana Deeda, Associate Professor, Department of Reproductive Endocrinology and Infertility, Bangabandhu Sheikh Mujib Medical University, Dhaka, BANGLADESH.
Email: deeba_51@yahoo.com

Received Date: 22/02/2021 Revised Date: 12/03/2021 Accepted Date: 06/04/2021
DOI: https://doi.org/10.26611/10121823

Access this article online	
Quick Response Code:	Website: www.medpulse.in
	Accessed Date: 15 May 2021

INTRODUCTION

Male infertility has been shown to be a widespread problem all round the world with an increasing trend in incidence. Approximately 50% of infertility is due to male factors, with a 36% prevalence of semen abnormalities.[1] Infections and inflammatory disorders of the male genital tract represent a major cause of male infertility. Mast cells (MC) are effectors cells of the immune system and play a crucial role in anaphylactic reactions, they are also critically involved in the pathophysiology of fibrotic

How to cite this article: Deeba F, Banu J, Ishrat S, Anawary S A, Begum N. Effect of mast cell blocker Ebastin in male infertility due to oligo-zoospermia and asthenozoospermia. *MedPulse International Journal of Gynaecology.* May 2021; 18(2): 27-34.
http://medpulse.in/Gynacology/index.php

disorders.[2] The release of tryptase by MCs in the testis has been associated with fibrotic remodeling in defective spermatogenesis of infertile men and sperm number and quality decrease under chronically testicular inflammatory conditions.[3-4] Sperm count below 15 x 10^6 sperm/ml is called oligozoospermia and less than 40% sperm with forward progression or less than 32% with rapid forward progression is called asthenozoospermia.[5] 50% improvement of baseline sperm count and or motility regarded as improvement. So, the treatment of patients suffering from oligo-asthenozoospermia with MC blockers were found to have some improved semen parameters by many authors.[5-7] The right treatment of male infertility is relatively costly procedure. In our country due to ignorance of the patients, lack of sharp cut technology in every level sometime people are misguided by various type of treatment procedures. Maximum people of our country lives below the standard level economical parameter. So we should sincerer about the treatment procedure to evaluate the role of mast cell blocker ebastin in male infertility for the sake of the wellbeing of our people. This study will help our people to save their hard earnings as well as infertility service provider to take proper action in improvement of abnormal semen parameters in the population of low resource country. The study aims Mast cell blocker (Ebastin) is effective in improvement of oligozoospermia and asthenozoospermia in infertile male.

II Objectives

a) General objective:

- To evaluate the effect of mast cell blocker ebastin in infertile male with oligozoospermia and asthenozoospermia.

b) Specific Objectives:

- To describe a demographic chart, personal and family history of the study people.
- To determine the effect of ebastin upon sperm count.
- To assess the effect of ebastin upon sperm motility.

III Methodology and Materials

This was a longitudinal clinical trial study and was conducted in the Infertility Unit outdoor, Department of obstetrics and gynecology, Bangabandhu Sheikh Mujib Medical University, Dhaka. Bangladesh during the period from January 2015 to December 2015. This study was conducted among the male patients presented to the study place during study period with infertility which is not attributable to any endocrine or urological abnormality and followed Purposive sampling technique. A total of 340 infertility patients were included in this study with maintaining inclusion criteria, and among them 6 patients were excluded from the study due to dropout (not came during follow-up). Finally, 167 cases were given Ebastin as group I and 167 patients were given only Placebo as group II.

Inclusion criteria:

All the male patients with infertility with following criteria:

- Age 25-50 years
- Abnormal semen parameters of oligozoospermia, asthenozoospermia.

Exclusion criteria:

- Age less than 25 years or more than 50 years
- Abnormalities in reproductive organ
- Known Hormonal disorder
- Known Medical disorder
- Pscho-sexual abnormalities
- Urological abnormality

Finally, 334 cases were enrolled in this study. Among the male with abnormal semen parameter 168 patients were Asthenozospermic, 106 patients were Oligospermic and 60 patients were combined Oligo-asthenozospermic. Alternate patient was administered, Tab. Ebastin 10 mg and Placebo twice daily and follow up semen analysis was done at pre fixed schedule after 3 months to analyze the changes that is achieved. Ethical clearance was taken from the local Ethical Committee to perform investigation and study. Statistical analysis was carried out by using the Statistical Package for Social Sciences version 16.0 for Windows (SPSS Inc., Chicago, Illinois, USA) and MS-Excel 2016. The mean values were calculated by frequencies and percentages. The quantitative observations were indicated by frequencies and percentages. Chi square test was used for categorical variables. Unpaired and Paired t-test was used for continuous variables. P values <0.05 was considered as statistically significant.

RESULT

Majority of the patients (n=168) had isolated asthenospermia and divided into group I (n=84) and group II (n=84). Isolated oligospermia was found (n=106) patients and divided into group I (n=53) and group II (n=53). Combined (oligospermia+ asthenospermia) was found in 60 patients in group I (n=30) and group II (n=30)[Figure-I]. Demographic variable of the patients. It was observed that majority patients belonged to age 31-40 years in both groups, which was 106(63.5%) in group I and 108(64.7%) in group II. The mean age was found 35.8±5.8 years in group I and 34.9±6.1 years in group II. The difference was not statistically significant (p>0.05). Majority patients were private job in both groups, which was 92(55.1%) in group I and 101(60.5%) in group II. The difference was not statistically significant (p>0.05) when compare between the groups. Regarding monthly income, 82(49.1%) were earn monthly 6000-15000 Tk in group I

and 84(50.3%) in group II and their average monthly income was 22034.8±19727.9 Tk in group I and 18220.3±17961.5 Tk in group II. The difference was not statistically significant (p>0.05). Majority patients came from urban area in both groups, which was 145(86.8%) in group I and 136(81.4%) in group II. The difference was not statistically significant (p>0.05)[Table-I]. Regarding sexual dysfunction, it was observed that no sexual dysfunction was found 155(92.8%) in group I and 160(95.8%) in group II. Mean staging together was found 5.8±4.1 years in group I and 5.6±3.9 years in group II. Mean coital frequency was found 2.8±0.9 years in group I and 2.6±1.1 years in group II. Primary infertility was found 130(77.8%) in group I and 115(68.9%) in group II. Mean duration of infertility was found 149(89.2%) in group I and 161(96.4%) in group II. Majority 135(80.8%) patients non smokers in group I and 141(84.4%) in group II. Smoker was found 32(19.2%) in group I and 26(15.6%) in group II. Mean duration of smoking was found 12.0±6.7 years in group I and 5.9±4.7 years in group II. Majority 164(98.2%) patients in group I and 166(99.4%) in group II were not taken alcohol. The difference was not statistically significant (p>0.05)[Table-II]. It was observed that majority 115(68.9%) patients in group I and 128(76.6%) patients in group II patients had no family history. 34(20.4%) patients had DM in group I and 26(15.6%) in group II. 13(7.8%) and 9(5.4%) had infertility in group I and group II respectively. The difference was not statistically significant (p>0.05)[Table-III]. It was observed that mean serum TSH was found 2.3±1.9 miu/ml in group I and 2.4±2.0 miu/ml in group II. Mean prolactine was 8.0±4.0 ng/dl in group I and 7.8±4.1 ng/dl in group II. Mean serum free testostosterone was found 12.3±8.5 ng/dl and 12.1±8.4 ng/dl in group I and group II respectively. The difference was not statistically significant (p>0.05)[Table-IV]. In group I, mean sperm count was 10.32±2.71 million/ml in pretreatment and 18.87±9.56 million/ml in post treatment. The difference was statistically significant (p<0.05). In group II, mean sperm count was 10.19±1.75 million/ml in pretreatment and 10.18±1.95 million/ml in post treatment. The difference was not statistically significant (p>0.05)[Table-V]. 63.9% oligospermic male showed improvement in sperm count in ebastin group and 18.1% in placebo group. Which was statistically significant (p<0.05)[Figure-II]. In group I, mean sperm total mortality was 24.86±12.02% in pretreatment and 34.29±9.35% in post treatment. The difference was statistically significant (p<0.05). In group II, mean sperm total mortality was 21.96±6.17% in pretreatment and 22.44±4.81% in post treatment. The difference was not statistically significant (p>0.05)[Table-VI]. Improved sperm motility was 75.4% of asthenozospermic male in ebastin group and 19.3% in placebo group. Which was statistically significant (p<0.05)[Figure-III]. In group I, mean rapid progress was found 11.81±7.76 % in pretreatment group and 19.99±11.31 % in post treatment. The difference was statistically significant (p<0.05). In group II, mean rapid progress was found 11.25±1.67% in pretreatment group and 11.4±1.59% in post treatment. The difference was not statistically significant (p>0.05)[Table-VII].

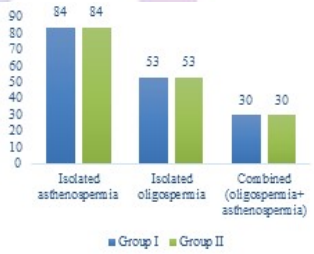

Figure 1: Distribution of study subject according to semen parameters (n=334)

Table-I: Distribution of the study patients by demographic variable (N=334)

Demographic parameter		Group I		Group II		P value
		n	%	n	%	
Age	<30	35	21	41	24.6	0.168
	31-40	106	63.5	108	64.7	
	41-50	26	15.6	18	10.8	
	Mean±SD	35.8±5.8		34.9±6.1		
	Range (min-max)	25-50		26-45		
Occupation	Businessman	40	24	35	21	0.208
	Private job	92	55.1	101	60.5	
	Govt. service	18	10.7	19	11.4	
	Aboard	6	3.6	8	4.8	
	Farmer	5	3	4	2.4	

	Other	6	3.6	0	0	
Monthly income (Tk)	<6000	3	1.8	1	0.6	0.067
	6000-15000	82	49.1	84	50.3	
	15001-30000	64	38.3	62	37.1	
	>30000	18	10.8	20	12	
	Mean±SD	22034.8±19727.9		18220.3±17961.5		
	Range (min-max)	4000-150000		5500-120000		
Habitat	Urban	145	86.8	136	81.4	0.178
	Rural	22	13.2	31	18.6	

Table-II: Distribution of the study patients by personal history (N=334)

Personal History		Group I		Group II		P value
		n	%	n	%	
Sexual dysfunction	Impotency	5	3	3	1.8	0.497
	Premature ejaculation	7	4.7	4	2.4	
	No	155	92.8	160	95.8	
Staying together (in years	Mean±SD	5.8±4.1		5.6±3.9		0.648
	Range (min-max)	0.2-19		0.5-11		
Coital frequency (in weeks)	Mean±SD	2.8±0.9		2.6±1.1		0.070
	Range (min-max)	1-5		1-5		
Type of infertility	Primary	130	77.8	115	68.9	0.063
	Secondary	37	22.2	52	31.1	
Duration of infertility (in years)	≤10	149	89.2	161	96.4	
	>10	18	10.8	6	3.6	
	Mean±SD	5.15±3.83		5.1±4.1		0.908
	Range (min-max)	0.5-18		1-15		
Smoking	Yes	32	19.2	26	15.6	0.386
	No	135	80.8	141	84.4	
Stick per day	Mean±SD	6.4±6.97		5.9±4.7		0.443
	Range (min-max)	1-30		0.5-24		
Duration of smoking (in years)	Mean±SD	12±6.7		10.9±5.9		0.112
	Range (min-max)	0.2-30		0.5-24		
Alcohol	No	164	98.2	166	99.4	0.311
	Occasional	3	1.8	1	0.6	
Other drugs	Yes	0	0	0	0	
	No	167	100	167	100	

Table-III: Distribution of the study patients by family history (N=334)

Family History	Group I		Group II		P value
	n	%	n	%	
DM	34	20.4	26	15.6	0.457
Infertility	13	7.8	9	5.4	
TB	5	3	4	2.4	
No family history	115	68.8	128	76.6	

Table 4: Distribution of the study patients by endocrine evaluation (N=334)

Endocrine evaluation		Group I(n=167)	Group II(n=167)	P value
S.TSH (miu/ml)	Mean±SD	2.3±1.9	2.4±2.0	0.640
	Range (min-max)	0.26-8.95	0.31-9.2	
Prolactine (ng/dl)	Mean±SD	8±4.0	7.8±4.1	0.652
	Range (min-max)	2.2-29.8	2.1-16.2	
S.free testostosterone (ng/dl)	Mean±SD	12.3±8.5	12.1±8.4	0.829
	Range (min-max)	2.5-39.6	2.4-31.2	

Table 5: Pretreatment and post treatment difference in sperm count of isolated oligospermia (N=334)

Sperm count (million/ml)		Pretreatment	Post treatment	P value
Group I (n=53)	Mean±SD	10.32±2.71	18.87±9.56	0.001[s]
	Range (min-max)	3-14	7-60	
Group II (n=53)	Mean±SD	10.19±1.75	10.81±1.95	[a]0.715[ns]
	Range (min-max)	3-11	6-12	
p value		b0.770ns	b0.001s	

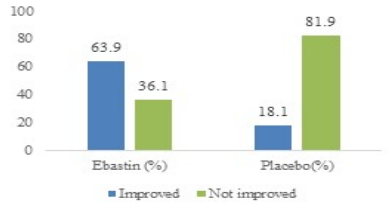

Figure-II: Bar diagram showing improvement of sperm count (Ebastin N=84); (Placebo N=84)

Table 6: Pretreatment and post treatment difference in sperm motility of isolated asthenospermia (N=334).

Sperm motility		Pretreatment	Post treatment	P value
Group I (n=84)	Mean±SD	24.86±12.02	34.29±9.35	[a]0.001[s]
	Range (min-max)	0-39	10-82	
Group I (n=84)	Mean±SD	21.96±6.17	22.44±4.81	[a]0.121[ns]
	Range (min-max)	0-31	10-35	
P value		b0.051ns	b0.001s	

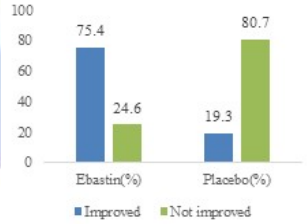

Figure 3: Bar diagram showing improvement of sperm motility (Ebastin N=114); (Placebo N=114)

Table 7: Pretreatment and post treatment difference in rapid progress of isolated asthenospermia (N=334)

Rapid progress (%)		Pretreatment	Post treatment	P value
Group I (n=84)	Mean±SD	11.81±7.76	19.99±11.31	[a]0.001[s]
	Range (min-max)	0-32	10-60	
Group II (n=84)	Mean±SD	11.25±1.67	11.4±1.59	[a]0.052[ns]
	Range (min-max)	7-13	8-16	
P value		b0.519ns	b0.001s	

DISCUSSION

This longitudinal clinical trial was carried out with an aim to determine the effect of ebastin upon sperm count and to assess the effect of ebastin upon sperm motility. A total of 334 male patients with infertility presented in Gynaecology and Obstetrics department of Bangabandhu Sheikh Mujib Medical University, Dhaka, between January 2015 to December 2015 were included in this study. All the male patients with infertility with age belonged to 25–50 years, abnormal semen parameters i.e. Oligospermia, asthenozospermia and not attributable to any endocrine or urological abnormality were enrolled in this study. Age less than 25 years or more than 50 years, abnormalities in reproductive organ, known Hormonal disorder, known Medical disorder, pscho-sexual abnormalities and urological abnormality were excluded from the study. Mast cells (MCs), play a key role in the inflammation, hypersensitivity and fibrosis, are as well

normally present in the testes.[4,8] Roaiah[9] study suggest that there is a prominent increase in the number of testicular MCs in the testes of infertile men which may disrupt spermatogenesis. Also, it was reported that increased numbers of MCs have been associated with different types of infertility, including asthenospermia.[9-10] Therefore some of male factor infertility problems could possibly diminished if such pathogenesis agent would greatly reduce using drug that block MC mediator release. The present study findings were discussed and compared with previously published relevant studies. We found majority of the patients (n=168) had isolated asthenospermia and divided into group I (n=84) and group II (n=84). Isolated oligospermia was found (n=106) patients and divided into group I (n=53) and group II (n=53). Combined (oligospermia+ asthenospermia) was found in 60 patients in group I (n=30) and group II (n=30). In this present study it was observed that majority patients belonged to age 31-40 years in both groups, which was 106(63.5%) in group I and 108(64.7%) in group II. The mean age was found 35.8±5.8 years in group I and 34.9±6.1 years in group II. The difference was not statistically significant (p>0.05). Saharkhiz[11] found the male were aged 23-40 years of mean age 32.8 years, which is consistent with the current study. Similarly, Multigner[12] and Cayan[13] showed the mean age of the infertile male patients were 32.0 years varied from 22 to 47 years and 34.5±7.2 years varied from 24 to 49 years respectively. Similar, age range also observed by Roaiah[7] and Hussein[14] in their respective studies. On the other hand, El-Karaksy[15] showed 64.0% were in more than 40 years and 36.0% were in under 40 years, which is higher with the current study. The higher age range maybe due to geographical variations, racial, ethnic differences and different lifestyle may have significant influence to identified their infertility. Prostatitis causes substantial morbidity to men, through associated urinary symptoms, sexual dysfunction, and pelvic pain; however, 90% to 95% of cases have an unknown etiology obtained by Ellem.[16] Regarding sexual dysfunction, no sexual dysfunction was found 155(92.8%) in group I and 160(95.8%) in group II. Mean staging together was found 5.8±4.1 years in group I and 5.6±3.9 years in group II. Mean coital frequency was found 2.8±0.9 years in group I and 2.6±1.1 years in group II. The difference was not statistically significant (p>0.05). In this current study it was observed that majority 135(80.8%) patients non smokers in group I and 141(84.4%) in group II. Smoker was found 32(19.2%) in group I and 26(15.6%) in group II. Mean duration of smoking was found 12.0±6.7 years in group I and 5.9±4.7 years in group II. Majority 164(98.2%) patients in group I and 166(99.4%) in group II were not taken alcohol. The difference was not statistically significant (p>0.05). Seminal mast cells showed higher frequency among smokers compared with nonsmokers, suggesting an aetiological relationship between smoking and mast cell abundance in infertile patients, and therefore an indirect relationship between smoking and infertility. This supports the studies that establish smoking as having an adverse effect on fertility, especially on progressive sperm motility, irrespective of total amount of cigarettes smoked per day.[17-18] In this study it was observed that majority 115(68.9%) patients in group I and 128(76.6%) patients in group II patients had no family history. 34(20.4%) patients had DM in group I and 26(15.6%) in group II. 13(7.8%) and 9(5.4%) had infertility in group I and group II respectively. The difference was not statistically significant (p>0.05). In this present study it was observed that mean serum TSH was found 2.3±1.9 miu/ml in group I and 2.4±2.0 miu/ml in group II. Mean prolactine was 8.0±4.0 ng/dl in group I and 7.8±4.1 ng/dl in group II. Mean serum free testostosterone was found 12.3±8.5 ng/dl and 12.1±8.4 ng/dl in group I and group II respectively. The difference was not statistically significant (p>0.05). Cayan[13] showed the serum testosterone values was 5.5±2.6 ng/mL varied from 1.7 to 12.6 ng/mL, which is lesser with the present study. Ketotifen's (Mast cell blocker) androgenic effects on male infertility, associated with oligospermia and asthenospermia, have been reported by Olivia and Multigner[19] Studies have also shown that it may counteract the ability of MCs to trigger an inflammatory response. It is worth mentioning that the number of MCs sometimes increases more than normal in the testicular tissue of infertile men.[20] In another study Schill[5] also suggested that ketotifen (Mast cell blocker) can be more helpful to improve the patients' sperm count and sperm motility with idiopathic oligo/asthenospermia. In this current study it was observed that in group I, mean sperm count was 10.32±2.71 million/ml in pretreatment and 18.87±9.56 million/ml in post treatment. The difference was statistically significant (p<0.05). In group II, mean sperm count was 10.19±1.75 million/ml in pretreatment and 10.18±1.95 million/ml in post treatment. The difference was not statistically significant (p>0.05). In this present study it was observed that in group I, mean sperm total mortality was 24.86±12.02% in pretreatment and 34.29±9.35% in post treatment. The difference was statistically significant (p<0.05). In group II, mean sperm total mortality was 21.96±6.17% in pretreatment and 22.44±4.81% in post treatment. The difference was not statistically significant (p>0.05). Saharkhiz[11] showed the mean sperm motility increased significantly from 16.7% to 21.4% after ketotifen (Mast cell blocker) treatment (p<0.001). This sperm motility improvement was more pronounced in the primary infertility cases (p<0.05). In 52.0% of infertile men's semen, the percentage of sperm motility was increased from 5% to 35% and this sperm

motility improvement was also observed in 33% of necrospermia (0% motility) cases. In group I, mean rapid progress was found 11.81±7.76 % in pretreatment group and 19.99±11.31 % in post treatment. The difference was statistically significant ($p<0.05$). In group II, mean rapid progress was found 11.25±1.67% in pretreatment group and 11.4±1.59% in post treatment. The difference was not statistically significant ($p>0.05$). In this study 63.9% oligospermic male showed improvement in sperm count in ebastin group and 18.1% in placebo group. Which was statistically significant ($p<0.05$). Improved sperm motility was 75.4% of asthenozospermic male in ebastin group and 19.3% in placebo group. Which was statistically significant ($p<0.05$). Saharkhiz[11] found 52% of infertile men's semen, the percentage of sperm motility was increased from 5% to 35% and this sperm motility improvement was also observed in 33% of necrospermia (0% motility) cases. Multigner (2006) observed 23.6% patients presented a sperm concentration of $<20 \times 10^6$ /mL and a sperm motility of <50% in 65.4%, which is comparable with the current study. Matsuki[21] investigated the effect of ebastine, a mast cell blocker, on semen quality in idiopathic oligozoospermic men reporting 66.7% of the patients had improved semen quality. Olivia and Multigner[19] carried out a study to assess the efficacy of daily administration of ketotifen (Mast cell blocker), on the semen quality of men with leukocytospermia and unexplained infertility. They noticed that ketotifen diminished the white blood cell count in semen and so the sperm motility has been dramatically improved. Moreover, the number of morphologically normal spermatozoa was more pronounced at 8 weeks of treatment and these changes remained until at least 4 weeks after stopped of ketotifen (Mast cell blocker) treatment. Ketotifen (Mast cell blocker) was used several years ago for treating patients with idiopathic oligozoospermia and asthenozoospermia (independent of the presence of leukocytospermia). This led to a very moderate but statistically significant improvement in sperm count and sperm motility.[5] Matsuki[21] showed that the mast cell blocker ebastine significantly improved semen quality in idiopathic oligozoospermia. Hibi[7] reported that tranilast improved semen parameters in severe oligoasthenozoospermia, but not for long-term administration. Matsuki[21] observed the effect of mast cell blocker (ebastine) on semen quality was evaluated in 15 idiopathic oligozoospermic males and 66.7% showed definite improvement in the semen quality. It would appear that mast cell blocker (ebastine) significantly improves semen quality in men with idiopathic oligozoospermia. Nagai[22] had shown that the number of mast cells in testicular tissue was increased and the ratio of mast cell subtypes (chymase and tryptase) was changed in idiopathic azoospermia and oligozoospermia. Yamamoto[23] prescribed randomly mast cell blocker (tranilast) or placebo for 3 months infertile males with severe idiopathic oligozoospermia, showing significant improvement in semen parameters.

Limitations of the study

The study population was selected from one selected tertiary hospital in Dhaka city, so that the results of the study may not reflect the exact picture of the community. The present study was conducted at a very short period of time. Small sample size was also a limitation of the present study. Therefore, in future further study may be under taken with large sample size. Although the present study had a number of restrictions; first, it was not double blind. Sample size and power to detect clinically essential outcomes were small. The trial was not designed with sufficient power to address important basic outcomes. Nevertheless, it was shown that it seems the failure rate of infertility treatments can be decreased if MC blockers such as Ebastin and Placebo can be given to the oligospermic and asthenozospermic men before intrauterine insemination (IUI) or ART cycles of treatment. Indeed, it seems that further research is necessary to identify the precise capacity of Ebastin to improve quality of semen parameters and whether this MC blocker has effects on the cumulative outcome of reproduction.

CONCLUSION AND RECOMMENDATIONS

From this paper we found that sperm count, Sperm motility and rapid progress had significantly improved after three months' treatment period with Ebastin. Therefore, this study suggested that mast cell blocker ebastin can be helpful to improve the patients' sperm count and sperm motility with oligospermia and asthenospermia. Further community based or multicentre, double blind placebo controlled studies can be undertaken by including large number of patients and Ebastin can be given to the oligospermic and asthenozospermic men before intrauterine insemination (IUI) or ART cycles of treatment.

REFERENCES

1. Bhasin S, de Kretser DM, Baker H.W 1994. clinical review 64: pathophysiology and natural history of male infertility. J Clin Endocrinal Metab, vol. 79, no. 6, pp. 1525-1529.
2. Frungieri MB, Calandra RS, Lustig L 2002. Number, distribution pattern, and identification of macrophages in the testes of infertile men. Fertil. Steril, vol. 78, no.2, pp. 298–306.
3. Pejler G, Abrink M, Ringvall M, Wernersson S 2007. Mast cell proteases. Adv. Immunol, vol. 95, pp. 167–255.

4. Pejler G, Ronnberg E, Waern I, Wernersson S 2010. Mast cell proteases: multifaceted regulators of inflammatory disease. Blood, vol. 115, no. 24, pp. 4981–4990.
5. Schill WB, Schneider J, Ring J 1986. The use of ketotifen, a mast cell blocker, for treatment of oligo-and asthenozoospermia. Andrologia, vol. 18, no. 6, pp. 570–573.
6. Yamamoto M, Hibi H, Miyake K 1994. Appearance of spermatozoon after administration of mast cell blocker to a patient with azoospermia. Hinyokika Kiyo, vol. 40, no. 6, pp. 541–543.
7. Hibi H, Kato K, Mitsui K 2001. The treatment with tranilast, a mast cell blocker, for idiopathic oligozoospermia. Arch. Androl, vol. 47, no. 2, pp. 107–111.
8. Chakraborty S, Bonthu N, Swanson BJ, Batra SK 2011. Role of mucins in the skin during benign and malignant conditions. Cancer Lett, vol. 301, pp. 127–41.
9. Roaiah MM, Khatab H, Mostafa T 2007. Mast cells in testicular biopsies of azoospermic men. Andrologia, vol. 39, no. 5, pp. 185–189.
10. Haidl G, Duan YG, Chen SJ, Kohn FM, Schuppe HC, Allam JP 2011. The role of mast cells in ale infertility. Expert Rev Clin Immunol, vol. 7, pp. 627–34.
11. Saharkhiz N, Nikbakht R and Hemadi M 2013. Ketotifen, a mast cell blocker improves sperm motility in asthenospermic infertile men. J Hum Reprod Sci, vol. 6, no. 1, pp. 19–22.
12. Multigner, L 2006, 'Ketotifen improves sperm motility and sperm morphology in male patients with leukocytospermia and unexplained infertility', Fertil Steril, vol. 85, pp. 240 –3.
13. Cayan S, Apa DD, Akbay E 2002. Effect of fexofenadine, a mast cell blocker, in infertile men with significantly increased testicular mast cells. Asian J. Androl, vol. 4, no. 4, pp. 291–294.
14. Hussein MR, Abou-Deif ES, Bedaiwy MA 2005. Phenotypic characterization of the immune and mast cell infiltrates in the human testis shows normal and abnormal spermatogenesis. Fertil. Steril, vol. 83, no. 5, pp. 1447–1453.
15. El-Karaksy A, Mostafa T, Shaeer OK, Bahgat DR, Samir N 2007. Seminal mast cells in infertile asthenozoospermic males. Andrologia, vol. 39, no. 6, pp. 244–247.
16. Ellem SJ, Wang H, Poutanen M, Risbridger GP 2009. Increased endogenous estrogen synthesis leads to the sequential induction of prostatic inflammation (prostatitis) and prostatic pre-malignancy. Am. J. Pathol, vol. 175, no. 3, pp. 1187–1199.
17. Hassa H, Yildirim A, Can C, Turgut M 2006. Effect of smoking on semen parameters of men attending an infertility clinic. Clin Exp Obstet Gynecol, vol. 33, pp.19–22.
18. Mostafa T, Tawadrous G, Roaih MMF, Amer M, Ashour S, Aziz A 2006. Effect of smoking on seminal plasma ascorbic acid in infertile and fertile males. Andrologia, vol. 38, pp. 221–224.
19. Oliva A, Multigner L 2006. Ketotifen improves sperm motility and sperm morphology in male patients with leukocytospermia and unexplained infertility. Fertil. Steril, vol. 85, no. 1, pp. 240–243.
20. Moskovtsev SI, Willis J, White J, Mullen JB 2007. Leukocytospermia: Relationship to sperm deoxyribonucleic acid integrity in patients evaluated for male factor infertility. Fertil Steril, vol. 88, pp. 737–40.
21. Matsuki S, Sasagawa I, Suzuki Y 2000. The use of ebastine, a mast cell blocker, for treatment of oligozoospermia. Arch. Androl, vol. 44, no. 2, pp. 129–132.
22. Nagai T, Takaba H, Miyake K, Hirabayashi Y, Yamada K 1992. Testicular mast cell heterogeneity in idiopathic male infertility. Fertil. Steril, vol. 57, no. 6, pp. 1331–1336.
23. Yamamoto M, Hibi H, Miyake K 1995. New treatment of idiopathic severe oligozoospermia with mast cell blocker: results of a single-blind study. Fertil. Steril, vol. 64, no. 6, pp. 1221–1223.
24. Winters BR, Walsh TJ 2014. The Epidemiology of Male Infertility. Urol Clin N Am, vol. 41, pp.195–204.

Source of Support: None Declared
Conflict of Interest: None Declared