

RESULTS

Clinical data:

Fifty infertile couples were attending the IVF unit from April 2008 till October 2009. Twenty five patients were stimulated by GnRH agonist long protocol and twenty five patients were stimulated by GnRH antagonist protocol.

The two groups did not differ significantly with respect to base-line characteristics. Mean patient age, (BMI), cause of infertility and basal FSH, LH and E2 concentration was similar in the two groups (Tables 3, 4, 5).

Table (3): Comparison between agonist & antagonist regarding maternal Parameters

Parameters	Agonist G (n=25) X± SD	Antagonist group (n=25) X± SD	t-test	P-value
Maternal age (years)	30.8± 3.4	29.1± 3.5	1.74	> 0.05
BMI (Kg/ m ²)	20.4± 1.8	20.7± 1.3	0.68	> 0.05
Duration of infertility (years)	4.7± 3.4	4.9± 3.5	0.85	> 0.05

Table (3): Shows no significant difference between agonist & antagonist groups regarding maternal age, BMI & duration of infertility (P> 0.05).

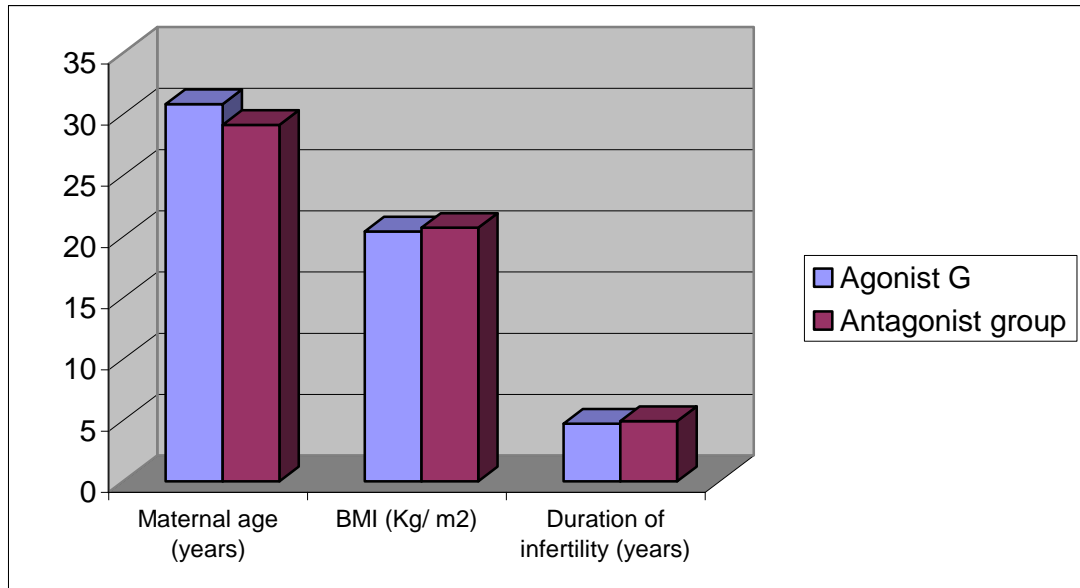


Fig (5): Comparison between agonist & antagonist regarding maternal Parameters

Table (4): Comparison between agonist & antagonist group regarding infertility causes.

Infertility causes	Agonist group (n=25)		Antagonist group (n=25)		X ²	P-Value
	No	%	No	%		
Tubal Factor	8	32	7	28	0.1	> 0.05
Male factor	7	28	5	20	0.44	> 0.05
Endometriosis	3	12	5	20	0.6	> 0.05
Idiopathic	3	12	6	24	1.22	> 0.05
Both male & female factor	2	8	1	4	0.35	> 0.05
Others	2	8	1	4	0.35	> 0.05

Table (4): Shows no significant difference between agonist & antagonist groups regarding causes of infertility ($P > 0.05$)

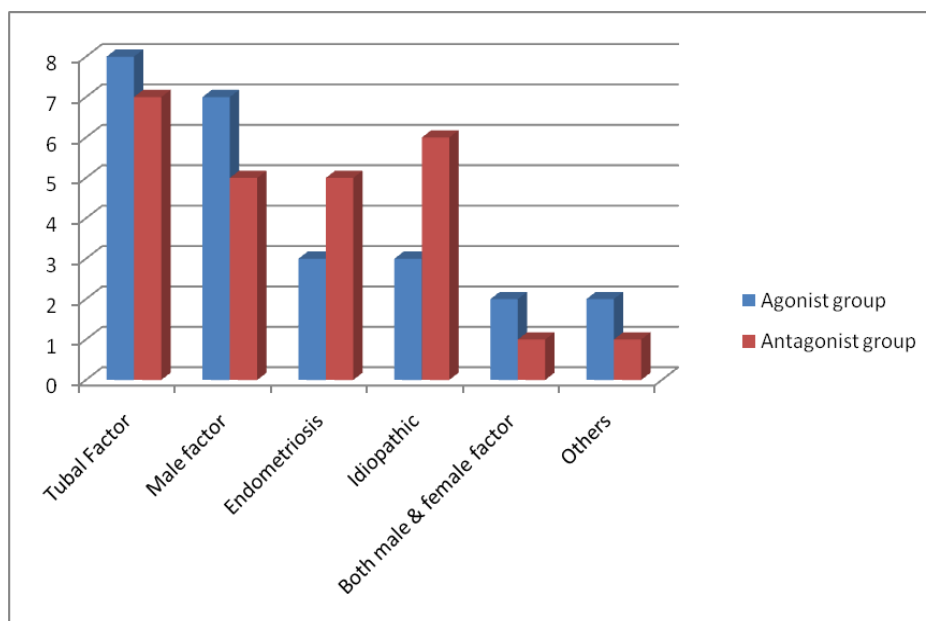


Fig (6): Comparison between agonist & antagonist group regarding infertility causes.

Table (5): Comparison between agonist & antagonist regarding basic hormonal assay.

Hormonal assay	Agonist G (n=25) X \pm SD	Antagonist group (n=25) mean \pm SD	t-test	P=-value
FSH(mlu/ml)	4.1 \pm 1.3	3.8 \pm 1.2	0.85	> 0.05
LH(mlu/ml)	2.91 \pm 1.6	3.21 \pm 1.1	0.80	> 0.05
E2(pg/ml)	82.41 \pm 4.4	84.56 \pm 6.3	1.65	> 0.05

Table (5): Shows that there is non significant difference between agonist & antagonist groups as regard basic hormonal assay ($P > 0.05$)

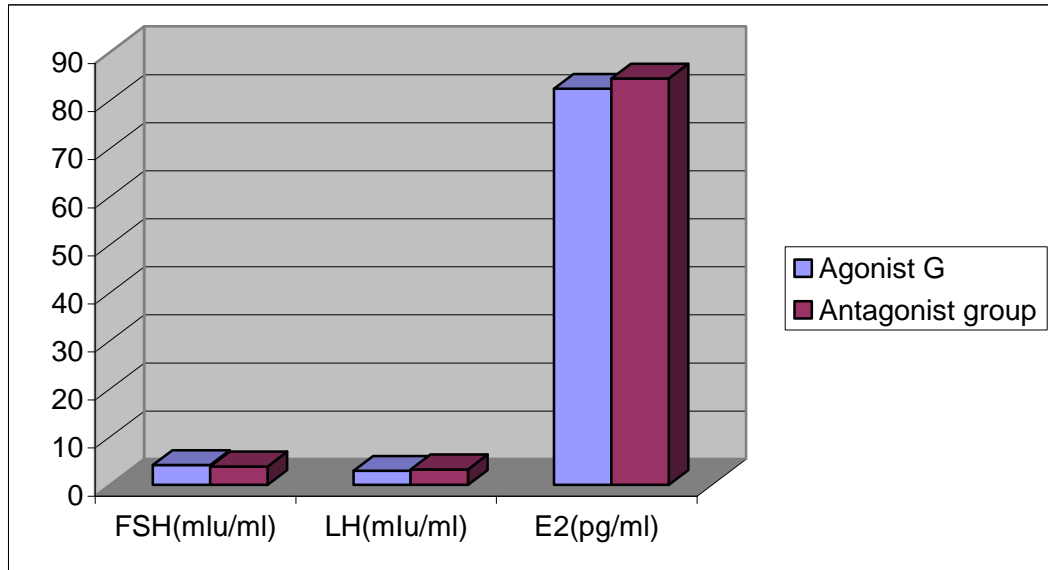


Fig (7): Comparison between agonist & antagonist regarding basal hormonal assay.

The type of gonadotrophin used for ovarian stimulation was similar in the two groups. The clinical parameters of ovarian stimulation showed that significantly less ampoules of r-FSH were needed in the antagonist group than in agonist group and the length of stimulation was significantly longer in the agonist group ($P < 0.05$) (Table 6).

Table (6): Comparison between agonist & antagonist regarding ovarian stimulation.

	Agonist G (n=25) mean \pm SD	Antagonist group (n=25) X \pm SD	t-test	P= value
Number of r-FSH ampoules	31.8 \pm 10.2	26.7 \pm 7.3	2.03	< 0.05
Length of stimulation(days) (days)	10.4 \pm 1.8	8.2 \pm 1.6	4.57	< 0.001

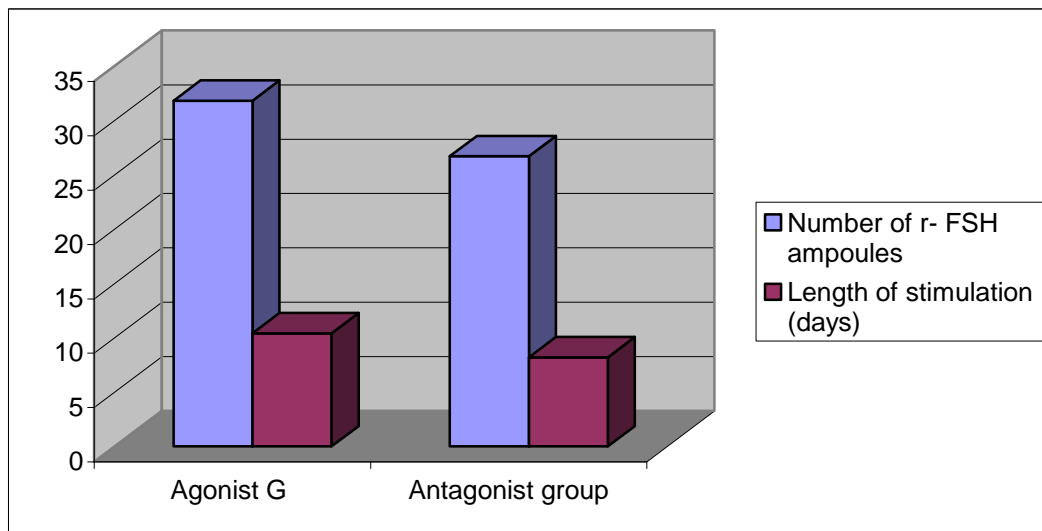


Fig (8): Comparison between agonist & antagonist regarding ovarian stimulation.

The number of follicles aspirated and oocytes retrieved was significantly lower in the antagonist group than with the use of the agonist. There was one cycle in the antagonist group and three cycles in the agonist group where we could not find oocytes. Rate of mature (MII) oocytes, cytoplasmic abnormalities in retrieved oocytes were higher in the agonist group than in the antagonist cycles but non significant (Table 7).

Table (7): Comparison between agonist & antagonist regarding oocyte characteristics.

Oocyte characteristics	Agonist group (n=25)		Antagonist group (n=25)		X ²	P-Value
	No	%	No	%		
Cycles with no oocytes	3	12	1	4	1.09	>0.05
Follicle aspiration	11.3±2.6		9.2±2.1		3.14*	<0.05
Retrieved oocytes	8.1±2.3		6.4±1.9		2.85*	<0.05
Mature oocytes% (metaphase II)	88%		84%		0.00**	>0.05
	7.1±2.2		5.4±1.6		3.12*	<0.001
Oocytes with cytoplasmic abnormalities%	64%		52%		0.57**	>0.05
	4.4±1.06		2.7±0.9		6.11*	<0.001

* t-test

** z-test

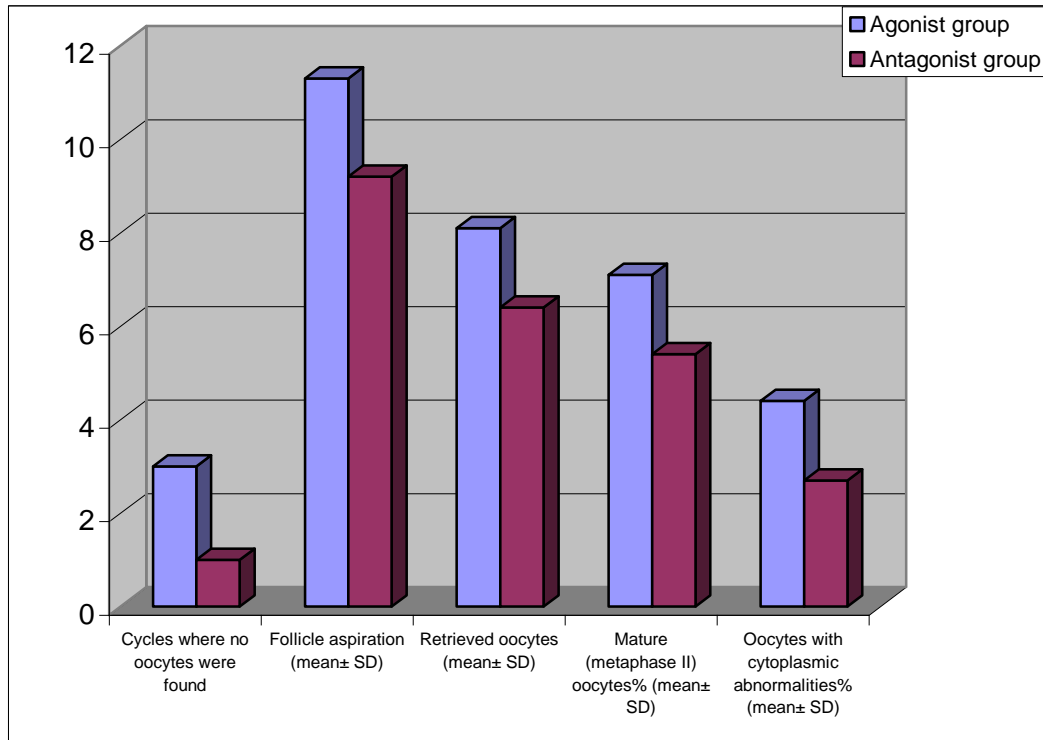


Fig (9): Comparison between agonist & antagonist regarding oocyte characteristics.

The method of fertilization was similar in both groups. The percentage of normal fertilized oocytes (2PN zygotes) was similar in both groups. Having significant less oocytes available for fertilization in the antagonist group. We observed non significant higher rate of zygotes showing normal nucleolar distribution with the use of GnRH agonist analogues ($P > 0.05$) (Table 8).

Table (8): Comparison between agonist & antagonist regarding fertilization.

Fertilization	Agonist group (n=25)	Antagonist group (n=25)	t-test	P-Value
Number of fertilized oocytes/ cycle	5.1±1.4	4.6±1.1	1.4	> 0.05
Normal pronuclear morphology%	68%	52%	0,87**	> 0.05
(X±SD)	3.1±1.2	2.7±0.8	1.56	> 0.05

** z-test

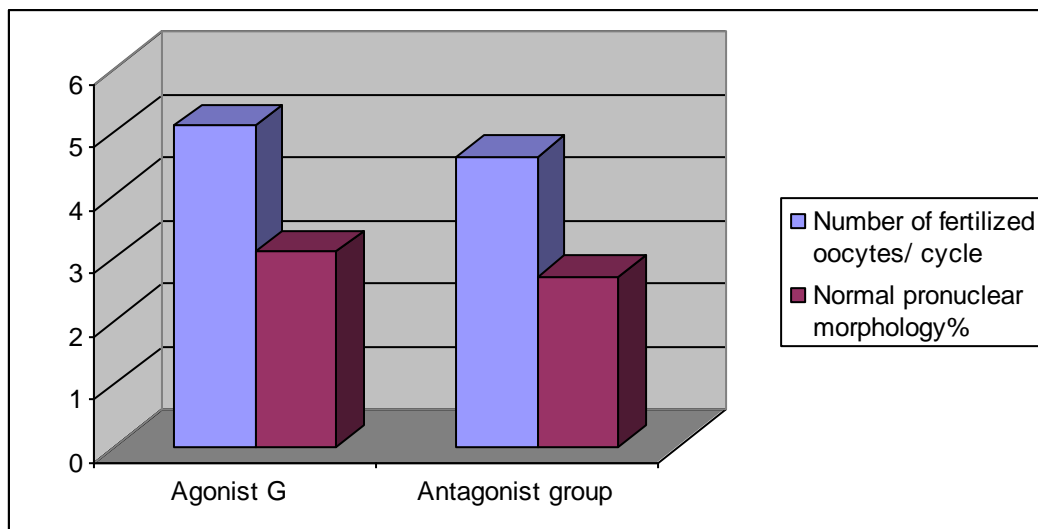


Fig (10): Comparison between agonist and antagonist regarding fertilization

Clinical pregnancy rate were intendancy lower in the agonist group than in the antagonist group, although this difference did not reach statistical significance ($P > 0.05$) (table 9).

Table (9): Comparison between agonist & antagonist regarding clinical outcome.

Clinical outcome	Agonist group (n=25)		Antagonist group (n=25)		X ²	P-Value
	No	%	No	%		
Embryo transfer performed	21	84	23	88	0.76	> 0.05
Clinical pregnancy rate	8	32	10	40	0.35	> 0.05

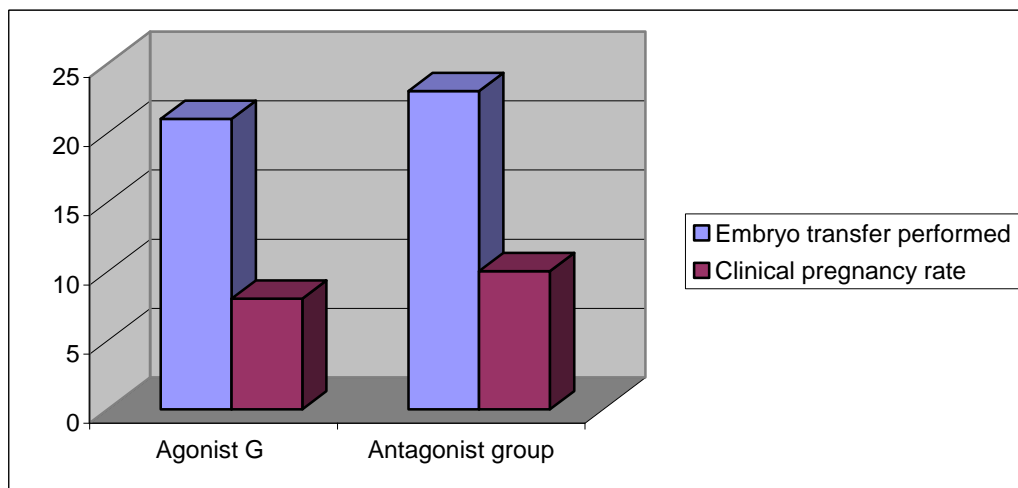


Fig (11): Comparison between agonist & antagonist regarding clinical outcome.