Peritoneal fluid volume parameters in infertile patients

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Summary: The peritoneal fluid volume (PFV) and its cellular and acellular components have been repeatedly associated with infertility. The PFV from 88 infertile women was analyzed in relation to endometriosis, pelvic adhesions, tubal patency, luteinization, endometrial cells and macrophages.

The independent existence of parameters such as the onset of luteinization, the increased population of macrophages and the presence of endometriosis increase statistically significantly the PFV. In the presence of peritoneal adhesions or/and obstructed tubes the PFV was significantly decreased.

INTRODUCTION

The presence of peritoneal fluid (PF) in cul-de-sac in normal women has been recognized from many years. It is acceptable that the PF is the natural environment for the tubes and the ovaries. There are many indications that the cellular (macrophages) and acellular (prostaglandins, hormones) components of PF influence the ovulatory and corpus luteum function, gamete transport or survival, sperm-oocyte interaction and early embryonic development ([1, 2, 3]).

It is well known that there is an increase of the peritoneal fluid volume (PFV) after the ovulation in normal cycles but there is a controversy for the PFV changes in the different pathophysiological status. In endometriosis the PFV was found decreased, normal or increased ([4, 5, 6]).

This disagreement was due to the fact that the control groups were not homogeneous and other pathological conditions such as adhesions or tubal patency were ignored and finally there was no reference to the cycle phase.

The purpose of this study was to examine the PFV of patients with primary or secondary infertility in relation with many parameters as: a) stage of endometriosis; b) pelvic adhesions; c) tubal patency; d) ovulation; e) populations of macrophages and f) population of endometrial cells.

MATERIAL AND METHODS

Eighty eight patients with primary or secondary infertility underwent laparoscopy. Prior to the procedure a routine infertility work up was done including hysterosalpingogram, semen analysis, post coital test, serial ultrasound examinations, basal body temperature and intrauterine biopsy one or two days prior to the menses.

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Infertile couples due to male factors and uterine abnormalities were excluded from the analysis. Patients with hyperprolactinaemia or thyroid disorders were appropriately treated. Patients with polycystic ovarian disease received clomiphene citrate, human gonadotrophins, and/or dexamethasone, and no pregnancy was achieved.

Laparoscopy was done in 5 (5.6%) patients before ovulation (3rd to 12th cycle day), in 23 (26.2%) periovulatory (13th to 15th cycle day) and in 60 (68.2%) postovulatory (16th to 29th cycle day).

The laparoscopy was done under general anesthesia. The PF was aspirated under direct visual control from the posterior cul-de-sac and the anterior uterovesical compartment, through a needle. Aspiration was done before the patient was placed into the Trendeleburg position in order to be sure that all the PF was collected. The PFV was recorded and centrifuged at 1500 rads/min. for 20 min. Three slides were also prepared for cytological examination. The test of PF was anticoagulated with 10 I.U. sodium heparin and chilled on ice.

The peritoneal cavity was inspected for: a) adhesions, fibromas, preovulatory follicles, ovulation stigma, and corpus luteum; b) presence of endometriosis and classification according to the revised classification of the American Fertility Society; c) tubal patency using blue de methylene.

The macrophages were examined in three slides. A lot of macrophages was characteristic when there were more than 70%, enough when the percentage was 30% to 70%, few, between 10% to 30% and rare when the percentage was lower than 10%. Statistical analysis was done using the Student-t-test.

RESULTS

Endometriosis was found in 32 (36.4%) of the patients. The mean age of the patients was 30.26 years (range 18-40 years). Sixteen (50%) of them were Stage I, eight (25%) Stage II, seven (21.8) Stage III and one (0.13%) Stage IV. From 82 patients PF was aspirated and the mean volume was 22.66 ± 2.38 ml ($\bar{x} \pm SE$).

In patients with endometriosis the PFV was higher but not statistically significant. On the contrary in the patients with peritoneal adhesions or obstructed tubes the PFV was significantly lower ($p < 0.01$ and $p < 0.05$ respectively) (Table 1).

Table 1. – *Relation of PFV with endometriosis, pelvic adhesions and tubal pregnancy.*

Pathology	No.	PFV ml ($\bar{x} \pm SE$)	P
Endometriosis	31	26.74 ± 4.23	NS
No Endometriosis	51	20.35 ± 2.99	
Adhesions	24	13.33 ± 2.54	<0.001
No Adhesions	58	26.52 ± 3.21	
Both tubes obstructed	23	15.74 ± 2.83	<0.05
Tube/Tubes patent	59	25.62 ± 3.44	
Adhesions + both tubes obstructed	29	14.72 ± 2.40	<0.001
No adhesions + tube/tubes patent	53	26.50 ± 3.47	

For the detection of luteinization we used the following criteria: 1) The observation of a stigma on the ovary through the laparoscope. 2) Levels of serum-P higher than 5 ng/ml. 3) Levels of PFV-P higher than 30 ng/ml. 4) Consecutive rise of basal body temperature for thirteen days at least 0.5° C. According to the above criteria in 74 patients, luteinization was found in 54 (72.97%) patients and in 20 (27.03%) patients the cycle was anovulatory.

The percentage of luteinization in cases with endometriosis was lower (55% versus 64%).

In all the patients with luteinization the PFV was statistically significantly higher ($p = 0.01$). The same was found in patients with no endometriosis and luteinization ($p < 0,0001$). In the ovulatory patients with endometriosis the PFV was significantly higher ($p < 0.001$) than in cases without endometriosis. Between the ovulatory and anovulatory patients with endometriosis no statistical significant in the PFV was found (Table 2).

In patients without pelvic adhesions and/or patent tubes the percentage of lu-

Table 2. – *The influence of luteinization on the PFV in patients with and without endometriosis* * *ml* ($\bar{x}\pm SE$), (1) $p=0.01$, (2) $p<0.001$, (3) $p<0.0001$.

Luteinization	n.	%	PFV	with endometriosis n.	%	PFV*	without endometriosis n.	%	PFV*
Yes	54	72.97	27.09±3.39 (1)	19	35.19	30.1 ±6.29 (2)	35	64.81	25.4±3.92 (2) (3)
No	20	27.03	14.6 ±3.26 (1)	9	45.0	23.89±5.51 (3)	11	55.0	6.09±2.7 (3)

Table 3. – *The influence of the luteinization on the PFV in patients with and without adhesions or patent-obstructed tubes.* * *ml* ($\bar{x}\pm SE$), (1) $p<0.01$, (2) $p<0.001$.

Luteinization	with adhesions n.	%	PFV*	without adhesions n.	%	PVF*
Yes (n=54)	16	29.63	13.43±2.86	38	70.37	30.47±3.99 (1)
No (n=20)	7	35.0	12.14±6.06	13	65.0	15.92±3.95 (1)
	Both tubes obstructed			Tube/tubes patent		
Yes (n=54)	15	27.77	16.33±3.08	39	72.23	28.92±4.02 (2)
No (n=20)	5	25.0	19.0 ±9.14	15	75.0	13.33±3.26 (2)

teinization was higher (70.37% and 72.23% respectively).

The postovulatory PFV in the patients with pelvic adhesions and/or obstructed tubes was not significantly increased. On the contrary the postovulatory PFV was significantly higher in patients without adhesions ($p<0.01$) and/or obstructed tubes ($p<0.001$) than those with anovulatory cycles (Table 3).

No statistically significant difference was found in PFV between the different stages of endometriosis. In patients in Stage I (n=16) the PFV was 23.06±7.15 ml ($\bar{x}\pm SE$), in Stage II (n=8) 31.25± 4.6 ml, in Stage III (n=7) 29.85±10.21 ml ($\bar{x}\pm SE$) and in Stage IV (n=1) 25.0 ml.

No statistical difference was found in the PFV in the subgroups "plenty/enough" macrophages in all the patients, on the contrary in the subgroup "few-rare" macrophages, the PFV was significantly higher in patients with endometriosis ($p<0.05$) or in patients without adhesions ($p=0.01$). The statistically significant reduction of PFV in patients with pelvic adhesions and/or obstructed tubes did not exist in the subgroup of macrophages "plenty/enough" (Table 4).

The PFV with the presence of endometrial cells was significantly higher in pa-

Table 4. – *The PFV in populations of macrophages "plenty/enough" and "few/rare" in patients with or without endometriosis and with or without pelvic adhesions.* *$ml(\bar{x}\pm SE)$, (1) $p<0.05$, (2) $p=0.01$.

Macrophages	with endometriosis n.	PFV*	without endometriosis n.	PFV*	with adhesions n.	PFV*	without adhesions n.	PFV*
Plenty/enough	6	28.33±12.63	5	20.0 ±3.53	2	25.0 ±5.01 (1)	9	24.44±8.47
Few/rare	20	30.25± 5.72 (1)	38	19.31±3.17 (1)	19	14.73±2.71 (1) (2)	39	27.15±3.76 (2)

Table 5. – *The influence of the presence of the endometrial cells in women with and without endometriosis and with and without adhesions.* *$ml(\bar{x}\pm SE)$, (1) $p<0.05$, (2) $p<0.001$.

Endometrial cells	with endometriosis n.	PFV*	without endometriosis n.	PFV*	with adhesions n.	PFV*	without adhesions n.	PFV*
Yes	18	25.16±5.58 (1)	18	12.83±2.26	11	20.63±3.88	35	19.8 ±3.2
No	4	28.25±5.96	23	25.13±5.96 (1)	8	11.62±2.29 (1) (2)	27	25.59±4.07 (2)

tients with endometriosis ($p<0.05$). The wellknown statistically, significant reduction of PFV in patients with adhesions ($p<0.001$) did not exist with the presence of endometrial cells (Table 5).

DISCUSSION

The PFV changes in response to the ovarian activity during the normal menstrual cycle. Pathological processes such as endometriosis, peritoneal adhesions, tubal patency etc. resulted in alteration in PFV or its cellular components ([6, 7, 8]).

The main source of PFV is from the rupture of the follicles and the mean value is 4.67±1.48 ml, but the increased size of PFV cannot be explained by the fluid content of the preovulatory follicle or by fallopian tube production ([9, 10]). Most of the PF has its origin in the increased permeability of peritoneum and ovarian surface. There is an increase in permeability in cases of high serum steroid and high serum prostaglandins ([4, 5, 11]).

The mean value of PFV in all the patients of our study was higher (22.6± 2.3 ml, $\bar{x}\pm SE$) than in the normal population but in agreement with other reports with specific populations of women ([12]). In patients with endometriosis the PFV was higher but not statistically significant. On the contrary in patients with pelvic adhesions and/or obstructed tubes the PFV was, significantly lower ($p<0.01$ and $p<0.05$ respectively). This is in agreement of a report of Syrop ([13]) but in disagreement with others ([12]).

In our study the PFV was significantly higher in patients with luteinization ($p=0.01$), in patients with luteinization without endometriosis ($p<0.0001$) and in patients with anovulatory cycles and endometriosis ($p<0.0001$). On the contrary in the patients with endometriosis, luteinization did not play any role in the quantity of PFV. The significantly higher PFV was due to independent action of endometriosis or luteinization. On the contrary, when those two parameters coexist, the PFV was higher but not statistically significant (Table 2).

The same was found in the relation of PFV with pelvic adhesions and/or obstructed tubes. The PFV was significantly higher after luteinization and significantly lower in patients with pelvic adhesions. When these two parameters coexist there was no more statistical significant difference in the PFV (Table 3).

Findings and differences in PF parameters, particularly differences found in the postovulatory period, lend support to their proposed pathophysiological roles. Patients with endometriosis who achieved pregnancy had a significantly lower mean PFV than those remaining not-pregnant. Also the time required for the occurrence of pregnancy in patients with endometriosis appears influenced by the PFV.

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