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Persistence of the Central Endometrial Echo During the Midluteal Phase of the Cycle

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Abstract

Objective: To find factors possibly related to the persistence of a midluteal central endometrial echo usually associated with lower pregnancy rates.

Methodology: 2 groups of women with regular menstrual cycles were followed by transvaginal ultrasound scan examinations during the follicular and luteal phases of the cycle. One group consisted of 31 parous women who requested assisted reproduction treatment and gender selection for family completion. The other group consisted of 79 nulliparous women monitored during infertility investigations. There was no history of prior uterine surgery or recent hormonal medication. The age and body mass index of all women were recorded beforehand. Presence of polycystic ovaries, midcycle endometrial thickness, uterine arteries pulsatility index and ovulation time were recorded. Endometrial texture and serum progesterone level were examined 7-9 days after ovulation. All monitored indices were assessed in relation to the two studied groups and to the presence or absence of a central endometrial echo.

Results: No differences were detected between the two groups regarding age, body mass index, polycystic ovaries prevalence, midcycle endometrial thickness, midcycle uterine arteries pulsatility index or midluteal serum progesterone. A central midluteal endometrial echo was evident in 6 of 31 women in the parous group (19.4%) and 26 of 79 (32.9%) in the nulliparous group (p=0.119). It was significantly more common in women with polycystic ovaries (19 of 34, 55.9%) than the non-polycystic ovaries group (13 of 76, 17.1%); p<0.001. All other parameters examined had no significant correlation with the luteal phase persistent central endometrial echo.

Conclusion: Local uterine factors might be responsible for the persistent midluteal central endometrial echo as it had no significant correlation to the uterine arteries blood perfusion or midluteal serum progesterone. It might be corelated to endometrial hyperandrogenisation because of its significant correlation to polycystic ovaries. Other local possibilities need to be explored.

Keywords: Central endometrial echo; Polycystic ovaries; Endometrial hyper androgenisation

Introduction

Changes in endometrial thickness and echogenicity during the cycle have been studied intensively during infertility investigations. Relevance of endometrial thickness to conception rates has been a controversial subject. Few studies showed no pregnancies when the endometrium thickness was less than 6 mm, while others showed no such correlation. Furthermore, no correlation has been shown between endometrial thickness and endometrial histology during natural or assisted reproduction treatment cycles [1,2]. At the same time, no association could be found between endometrial thickness and endometrial vascularization which is a factor indicative of endometrial receptivity [3]. Accordingly, endometrial thickness may not correlate with function.

On the other hand, ultrasonic endometrial texture may have a better prognostic value of implantation. The trilaminar midcycle endometrial pattern has been considered a good prognostic factor for pregnancy during gonadotrophin stimulated cycles [4,5]. At the same time, higher pregnancy rates were reported in cases with midluteal phase homogeneous hyperechogenic endometrium compared to non-homogeneous patterns [6,7]. Persistence of the midline echo during the midluteal phase was reported in 39% of women investigated before having *in vitro* fertilization treatment [8]. Yet again, such pattern was not associated with the histological dating of the endometrium [9] despite its clinical relevance. This study was designed to find factors possibly related to the persistence of such pattern during the midluteal phase of the cycle.

Materials and Methods

A total of 110 women with regular menstruation had monitored cycles during this study. Group 1 included 31 parous women who requested assisted reproduction treatment with gender selection for family completion. Group 2 included 79 nulliparous patients who were monitored during their infertility investigations. None of the patients had any previous uterine surgery or recent history of hormonal medication. Each patient had multiple serial transvaginal ultrasound scan examinations, with at least one examination done 7-9 days after ovulation. Parameters recorded included endometrial thickness, ovulation time, midcycle uterine arteries pulsatility index, midluteal endometrial echogenicity and persistence of a central endometrial echo when present. Serum progesterone was also measured 7-9 days after ovulation. Patients' age, body mass index (BMI), parity, and presence or absence of polycystic ovaries as documented by transvaginal ultrasound scan examinations were documented beforehand. All examinations were conducted using Samsung ultrasound machine, model UGEO WS80A with the intracavity probe V5-9. One-way analysis of variance (ANOVA) was used to test the differences between patients in the two groups and those who did and did not show a central midluteal endometrial echo. Parameters tested included age, BMI, midcycle endometrial thickness and uterine arteries pulsatility index and serum

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Parameter	Parous group (31 women)	Nulliparous group (79 women)	Significance
Age (years)	31.75 (30.19-33.31)	33.26 (32.43-34.08)	p=0.066
BMI (KG/M^2)	28.51 (27.28-29.75)	29.89 (29.15-30.63)	p=0.053
Endometrium (mm)	09.28 (08.64-09.93)	09.52 (07.80-11.20)	p=0.865
Pulsatility index	03.20 (02.96-03.45)	03.29 (03.14-03.43)	p=0.544
Progesterone (ng/ml)	05.98 (05.41-06.56)	05.52 (05.21-05.83)	p=0.135

Table 1: Shows different data given as the mean with the 95% confidence interval. There were no significant differences in the age or BMI of the parous and nulliparous groups. There was also no difference in the 3 other tested parameters between the two groups with p>0.05.

Parameter	Present central echo	Absent central echo	Significance
Age (years)	31.75 (30.19-33.31)	33.26 (32.43-34.08)	p=0.066
BMI (KG/M^2)	28.91 (27.73-30.08)	29.75 (28.98-30.52)	p=0.236
Endometrium (mm)	10.22 (05.94-14.49)	09.14 (08.78-09.50)	p=0.430
Pulsatility index	03.41 (03.20-03.62)	03.20 (03.10-03.40)	p=0.125
Progesterone (ng/ml)	05.41 (04.79-06.03)	05.57 (05.45-06.05)	p=0.265

Table 2: Shows no significant correlation between the central endometrial echo in relation to age, BMI, endometrial thickness, pulsatility index or serum progesterone level. Figures are shown as the means with the 95% confidence intervals. In all cases p>0.05.

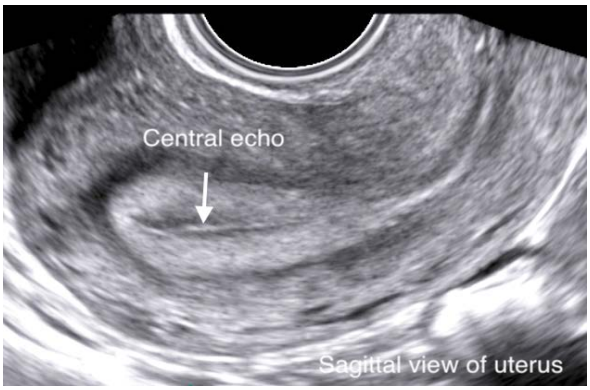

Figure 1: Shows sagittal transvaginal ultrasound scan view of a uterus during the midluteal phase of the cycle. A central echo is clearly seen in the middle of the otherwise echogenic endometrium.

progesterone. The means of the data were used with 95% confidence intervals to check for any significant differences. Crosstabulation with chi square test was also used to test categorical data. $p \leq 0.05$ indicated statistical significance. The Statistical Package for Social Sciences (IBM SPSS) was used for data analysis.

Results

Table 1 shows the personal characteristics of the two groups. There were no differences in age, BMI, midcycle endometrial thickness, uterine arteries PI or midluteal serum progesterone. At the same time there was no significant difference in the number of women with PCO between the two groups. In the nulliparous group 26 of 79 (32.9%) women showed PCO compared to 8 of 31 (25.8%) in the parous group (p=0.314).

There was also no significant difference between the two groups regarding the presence of a central endometrial echo during the midluteal phase (Figure 1). This was evident in 6 of 31 women in the parous group (19.4%) and 26 of 79 (32.9%) in the nulliparous group (p=0.119). At the same time, Table 2 shows that presence of a central endometrial echo was not related to age, BMI, endometrial thickness or serum progesterone level. However, it was significantly more common in women with PCO than others with normal looking ovaries. In the PCO group 19 of 34 patients showed a central endometrial echo (55.9%) compared to 13 of 76 women with no PCO (17.1%), p<0.001.

Discussion

This study showed that the presence of a central endometrial echo during the midluteal phase was not affected by age, BMI or parity status. It also had no significant correlation with the midluteal serum progesterone or uterine arteries pulsatility index. Accordingly, such texture could not be consequent to lower progesterone levels or lower endometrial blood perfusion but could have resulted from local endometrial factors. The strong correlation with the presence of PCO is interesting. Polycystic ovaries are known to be associated with higher production of androgens with a high androgen gradient between the ovaries and peripheral blood. Such androgenic environment may affect the endometrium because of the direct communication between the ovarian and uterine blood vessels. High androgens have negative effects on endometrial cells growth and secretory activity [10]. They can downregulate endometrial oestrogen receptors, alter endometrial pregnancy specific protein and reduce endometrial receptivity and pregnancy rates. These may be affected through elevated endometrial androgen receptors and decrease in biomarkers of endometrial receptivity, the $\alpha_v\beta_3$ integrin [11]. On the other hand, patients with PCO may also have deranged prolactin secretion and thyroid problems. This may reflect a deficiency of hypothalamic dopamine, which is manifested by the basal elevations of both prolactin and thyrotropin levels [12,13]. These may have detrimental effects on steroidogenesis which may affect endometrial integrity and the prostaglandins cascade. There is no direct proof to support this hypothesis but research on endometrial receptors and micro-endocrinology may offer some clues about this problem.

Conclusion

Persistence of a central endometrial echo during the midluteal phase of the cycle needs further investigations. Lack of correlation to serum progesterone and uterine arteries blood flow should direct attention to local uterine factors. Since PCO is significantly correlated to this phenomenon, a hyperandrogenic endometrial environment could be one likely explanation. Other local possibilities need to be explored to improve endometrial receptivity and pregnancy rates.

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