Table 1

(Per mm^2) $P < 0.01$	P	EL	ML	LL	M	Oestradiol/NET
% Vascular space area	2.3	1.9*	2.8	2.5	2.8	2.7
Mean vascular space diameter	15.0	13.1	16.6	15.4	14.9	12.8*
Number of Vascular Space	114	110	103	113	128	167*

P, proliferative; EL, early luteal; ML, mid luteal; LL, late luteal; M, menstrual.

culature to the same progestogen. Endometrial blood vessels undergo different morphological changes under the effect of different sex steroids.

2. Objectives

We examined the effect of a trimegestone-based HRT regimen upon the expression of cell determinant (CD) 34 (a glycoprotein expressed by endothelial cells), compared with the natural cycle. To try and explain the mechanism underlying the abnormal bleeding patterns in postmenopausal women on this HRT regimen.

3. Patients and methods

Endometrial biopsies were obtained from 26 postmenopausal women 48–80 years (mean: ±standard deviation, 62.9±9.3 years) who completed 3 months treatment of 2 mg of oestradiol valerate daily with 1 mg norethisterone from days 17 to 28. Included in the study were healthy women aged over 50 years with an intact uterus who were at least 6 months postmenopausal. The control samples ($n = 34$), were healthy, regularly menstruating women, aged 27–50 years (mean age: ±standard deviation, 38±6.1 years). These women were given urinary luteinising hormone (LH) surge detection kit tests during the month preceeding the endometrial biopsy or hysterectomy. Endometrial samples were immunohistochemically stained for CD34.

4. Results

Ten random fields ($\times 200/0.121$ mm^2) were examined per biopsy to evaluate the total vascular space area, diameter and number per field and analysed using the KS-300 image analysis programme. Non-parametric Kruskal–Wallis and Mann–Whitney tests were used for statistical analysis (Table 1).

5. Conclusion

Norethisterone induced larger vascular area, smaller diameter and higher number of vascular spaces, unlike other progestogens. This may indicate that norethisterone induces a different angiogenic stimulus to the endometrium.

Abstract: P15

The prevalence of Ki 67 and oestrogen receptor β antigens in the carotid arteries with atheromatous plaques compared with controls

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1. Introduction

The anti-atheromatous effects of oestrogen are well recognised but the underlying mechanisms are not well understood. We have previously documented the regression of carotid artery atheromas using the unopposed oral oestrogen. The classical oestrogen receptor (ERα) is expressed on blood vessels and may, at least in part, be directly responsible for oestrogen action. Atheromatous plaques are surrounded by proliferating smooth muscle cells and oestrogen is known to inhibit smooth muscle proliferation.

In the ERα- knock-out (ERKO) mouse, the administration of oestradiol was just as effective in the protection against carotid artery injury as in the wild-type. This suggests the presence of an ERα-independent mechanism of action of oestradiol. ERβ has been described, along with other isoforms, and may be a potential mechanism for the vascular effects of oestrogen.

2. Aim

To evaluate the characteristics of the vascular wall of atheromatous carotid arteries in terms of ERβ and Ki-67 expression, compared with controls.

3. Material and methods

Segments of carotid arteries with atheromatous plaques were obtained from female patients undergoing carotid endarterectomy. Control carotid artery specimens were obtained from cadavers, and were morphologically identified as not bearing atheromas. Two other specimens were obtained from the internal mammary artery to serve as additional controls. Paraffin-embedded sections (5 μm) were stained for Ki-67 and ERβ antigens. Controls included non-specific mouse IgG (Sigma, Dorset, UK), and normal rabbit serum for anti-ERβ antibodies. The sections were incubated with species-specific biotin-linked secondary antibody, vectastain ABC peroxidase and DAB substrate. Slides stained with MiB-1 were counterstained with haematoxylin. The pattern of the distribution of the positively stained cells for all the antigens was assessed in the sections, and then 10 randomly selected fields per slide (×200) were captured to evaluate the Ki-67 and ERβ cells. Images were captured using Axioplan microscope and a colour video camera and the positive cells were counted using the KS300 image analysis programme.

4. Results

Table 1

	No. of positive cells (count mm^3)		
	Controls ($n=13$)	Internal mammary	Arteries with atheroma
MiB-1	0 ($n=9$) 48 ($n=4$)	9	8
ERβ cells	1011	2462	1165

5. Conclusions

The higher expression of MiB-1 cells in the carotid arteries with atheromas compared with controls is concordant with the published literature. There was, however, no apparent difference in the expression of ERβ between atheromatous carotid arteries and controls, which may be due to the number of specimens examined.

It is difficult to explain the marked difference in the expression of ERβ in sections of atheromatous and healthy carotid arteries compared with the internal mammary artery; a blood vessel renowned for its resistance to atheroma formation.