Rarefaction of skin capillaries in normotensive offspring of individuals with essential hypertension

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Background: Rarefaction of skin capillaries in people with intermittent borderline essential hypertension suggests a primary or an early abnormality that may antedate the onset of sustained hypertension.

Objective: To compare skin capillary density in subjects with and without a family history of essential hypertension.

Subjects: 21 normotensive individuals, one or both of whose parents had essential hypertension (mean age 39.3 years; blood pressure 124/79 mm Hg); 21 normotensive controls with no family history of hypertension (age 46.3 years; blood pressure 124/78 mm Hg).

Methods: The skin of the dorsum of the fingers was examined by intravital capillary microscopy before and after venous congestion at 60 mm Hg for two minutes.

Results: By analysis of variance, both baseline and maximum skin capillary density were lower in subjects with a family history of essential hypertension than in those with no family history (baseline: 67 v 79 capillaries per field, p = 0.008; maximum: 74 v 93 capillaries per field, p < 0.0005).

Conclusions: Capillary rarefaction in essential hypertension may occur before the increase in blood pressure and could, at least in part, reflect a primary rather than a secondary abnormality.
was counted by hand from these acetate sheets. On the acetate sheet, the total number of visible capillaries could then be marked. Additional intermittently perfused capillaries which were not visible on the initial still frame images were traced. The same acetate sheet was then used with the video monitor during live playback of the recorded tapes. Reproducibility was first assessed by examining an identical area of skin marked by a microtattoo (by implanting a drop of sterile methylene blue ink into the epidermis with a 23 gauge hypodermic needle) to act as a reference point. Intraobserver repeatability of data analysis was assessed by reading the same prints in a blinded manner on two separate occasions (n = 20; coefficient of variability 4.3%). To assess interobserver repeatability, a second observer independently assessed capillary density in the same prints (n = 20; coefficient of variability 5.9%). Skin temperature was monitored throughout the study with a temperature probe on the dorsum of the left index finger (YSI Tele-Thermometers).

Maximisation of visualised skin capillaries

We have recently shown that venous congestion maximises the number of visible capillaries much more than reactive hyperaemia. In this study a miniature blood pressure cuff was applied to the base of the left middle finger and the cuff was then inflated and maintained at 60 mm Hg for two minutes; further images were then recorded using one of the four microscopic fields chosen at random.

Blood pressure and heart rate

Blood pressure was measured with an automatic oscillometric device (Omron PEN705CP, Omron Healthcare, Henfield, West Sussex, UK) with appropriate cuff size. Supine and standing blood pressure measurements were taken as the mean of three readings observed at one to two minute intervals with the individual in the corresponding position.

Blood and urine analysis

Venous blood was taken without stasis after the patient had been sitting upright for 10 minutes. Variables measured included serum electrolytes, urea, creatinine, uric acid, glucose, total cholesterol, triglycerides, and full blood count.

Statistical analysis

All results are given as mean (SEM). The data were processed by StatView 5.0 (SAS Institute Inc, Cary, North Carolina, USA). Analysis of variance (ANOVA) and Bonferroni’s post hoc tests were used to compare the groups.

RESULTS

Table 1 shows baseline clinical and laboratory characteristics and capillaroscopic data at baseline and after two minutes of venous congestion at 60 mm Hg in the study individuals. As the index and control subjects were matched for blood pressure, it was necessary to recruit slightly older individuals in the control group (39.3 (2.8) vs 46.3 (2.1) years, p = 0.052 by ANOVA), because people with a family history of hypertension have higher blood pressures (albeit in the normal range) than age and weight matched people with no family history of hypertension.

There was a significant (15%) lower mean capillary density in the index subjects at baseline than in the controls (67 (2) vs 79 (4) capillaries per field (0.68 mm²); p = 0.008). After two minutes of venous congestion, maximum capillary density remained significantly lower (by 20%) in the index group than in the controls (74 (2) vs 93 (4) capillaries per field (0.68 mm²); p = 0.0005) (fig 1).
DISCUSSION

Our major finding in this study was that normotensive subjects whose parents have essential hypertension have reduced skin capillary density. This reduction in capillary density in comparison with controls with no family history of hypertension was of similar degree before and after maximisation of capillary visibility. This suggests that the rarefaction is likely to reflect the structural absence of capillaries rather than a functional abnormality. This does not, of course, rule out the presence of additional functional abnormalities in the microcirculation. As essential hypertension is, as least in part, an inherited condition, our results indicate that capillary rarefaction occurs early in essential hypertension independently of the blood pressure (that is, it is a primary structural abnormality), or that it is associated with whatever eventually causes the rise in blood pressure. It is doubtful whether the skin capillaries have a major regulatory role in blood pressure regulation. However, it is widely believed that capillary rarefaction in essential hypertension is not limited to the skin but is a more generalised abnormality that affects different vascular beds.2-11

Our results are in agreement with previous studies that reported structural abnormalities of the vasculature in normotensive individuals with a parental history of essential hypertension. Noon and colleagues reported that offspring with raised blood pressure whose parents also had a high blood pressure had fewer capillaries on the dorsum of their fingers than those whose parents had a lower blood pressure.12,13 However, as blood pressure was raised in these individuals, a secondary decrease in capillary density cannot be excluded. In our study, the index and control groups were carefully matched for both systolic and diastolic blood pressure, but reflects a decreased angiogenic capacity of the microcirculation of individuals predisposed to hypertension. This reduction in vascular growth may affect different organs and be expressed in divergent ways in distinct phenotypes of the cardiovascular risk syndrome.2

REFERENCES


ACKNOWLEDGEMENTS

This research was supported by the British Heart Foundation.

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A 79 year old woman with a history of good health presented with sudden cardiac arrest caused by ventricular fibrillation. She was successfully resuscitated and admitted into the coronary care unit. An ECG showed sinus rhythm with left ventricular hypertrophy with T wave inversion over V4–V6. Serial cardiac enzymes were normal. An echocardiogram showed asymmetric septal hypertrophy, midventricular obstruction, and an apical aneurysm. Cardiac catheterisation showed angiographically normal coronary arteries. Left ventriculogram revealed severe left ventricular hypertrophy with systolic midventricular total obstruction and apical aneurysm (below left and right). A peak-to-peak intraventricular pressure gradient of 110 mm Hg was documented during pullback from the apical high pressure chamber (270 mm Hg) to the subaortic low pressure chamber in the left ventricle (160 mm Hg). The patient was subsequently treated with a β blocker and an implantable cardioverter-defibrillator was implanted.

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Left ventriculogram in right anterior oblique [30°] projection during systole showing nearly complete midventricular obstruction with apical aneurysm.

Left ventriculogram in right anterior oblique [30°] projection during diastole showing severe midventricular hypertrophy with apical aneurysm.
Sudden cardiac death caused by hypertrophic cardiomyopathy associated with midventricular obstruction and apical aneurysm
H-F Tse and H-H Ho

Heart 2003 89: 178
doi: 10.1136/heart.89.2.178

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