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Unsuspected renal artery stenosis in peripheral vascular disease

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An appreciable proportion of patients with heart failure or hypertension requiring treatment with an angiotensin converting enzyme inhibitor have widespread atherosclerotic disease. Angiotensin converting enzyme inhibitors have recently been proposed as the treatment of choice in patients with hypertension and peripheral vascular disease as they are free of many of the side effects of other antihypertensive agents such as β blockers.[1] They can, however, induce acute renal failure in patients with renal artery stenosis.[2] Retrospective studies in the 1960s suggested a high incidence of coexisting renal artery stenosis and peripheral vascular disease.[3] In a prospective study, therefore, we carried out renal arteriography in 100 patients referred for investigation of their peripheral vascular disease to determine the prevalence of anatomical renal artery stenosis in this population.

Patients, methods, and results

We performed renal and peripheral angiography in 100 consecutive patients (70 men, 30 women; mean age 67 (range 38-87)). All the patients had been referred for peripheral angiography to assess symptoms of intermittent claudication or pain of vascular origin in the legs at rest. The renal arteries were examined by intra-arterial digital subtraction angiography. Patients were defined as having hypertension if they were receiving antihypertensive treatment (information obtained from the clinical folders) or a blood pressure of >150 mm Hg systolic or >95 mm Hg diastolic was recorded in the ward before angiography. Serum creatinine concentration was measured before angiography in 83 patients and found to be raised in 30 (>125 μmol/l).

Both renal arteries were normal in 41 patients; 24 patients had bilateral stenoses. Seven patients had an occluded renal artery, of whom four had a stenosis of >50% of the contralateral artery. Hypertension, abnormal renal function, and male sex did not predict the presence of renal artery stenosis in this population (table).

Comment

We showed that most patients with peripheral vascular disease referred for angiography have either stenosis or occlusion of the renal arteries. A high percentage of these patients had bilateral stenoses. Furthermore, seven of these patients had a single functioning kidney.

Angiotensin converting enzyme inhibitors may induce reversible renal failure, which may be a specific effect of reducing production of angiotensin II. Efferent arteriolar dilatation may lead to a fall in glomerular hydrostatic pressure, resulting in a glomerulus with a blood supply but no filtration. Such an effect is likely to occur only when the glomerular pressure falls to very low levels, but this may occur in renal artery stenosis. In a patient with unilateral disease this may not be disastrous, but in those with only one kidney or bilateral disease acute reversible renal failure may ensue.[2] Angiotensin converting enzyme inhibitors may also, however, induce acute irreversible renal failure. This is not specific to this group of agents but due to a reduction in perfusion pressure leading to thrombosis in the stenosed renal artery.[4,5] During the course of this study two patients (not included in the study) who had been treated with angiotensin converting enzyme inhibitors for hypertension and peripheral vascular disease were admitted to our hospital with acute renal failure. In both cases this proved to be reversible, although haemodialysis was required. Subsequent angiography showed bilateral renal artery stenoses in both cases.

Peripheral vascular disease seems to be the best clinical marker for the presence of anatomical renal artery stenosis. Normal renal function has been assumed by other workers to preclude appreciable renal artery

Results of renal and peripheral angiography in 100 patients with peripheral vascular disease. Figures are numbers (percentages) of patients

Risk factor	Normal arteries	Stenosis of one artery		Bilateral stenosis	Occluded artery
		≤50%	>50%		
Sex:					
Male (n=70)	31 (44)	12 (17)	7 (10)	13 (19)	7 (10)
Female (n=30),	10 (33)	5 (17)	4 (13)	11 (37)	
Blood pressure:					
Hypertension (n=38)	18 (47)	7 (18)	2 (5)	10 (26)	1 (3)
Normotension (n=62)	23 (37)	10 (16)	9 (15)	14 (23)	6 (10)
Renal function:					
Normal (n=53)	22 (42)	12 (23)	7 (13)	12 (23)	
Abnormal (n=30)	10 (33)	5 (17)	2 (7)	7 (23)	6 (20)

stenosis in these patients,[1] but we found this not to be the case. Our findings accord with those of Dustan *et al*, who found that most patients with angiographic evidence of renal artery disease did not have hypertension.[3] It could be argued that angiographic evidence of renal artery disease does not necessarily indicate a functionally important stenosis, hence the poor relation between angiographic findings and hypertension in our study. Nevertheless, evidence of renal artery stenosis probably indicates those patients at high risk of renal complications during treatment with angiotensin converting enzyme inhibitors. We believe that in patients with evidence of peripheral vascular disease angiotensin converting enzyme inhibitors should be used with caution. Consideration should be given to the possibility of underlying renal artery stenosis.

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Recording diastolic blood pressure in pregnancy

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Obstetricians are guided mainly by diastolic blood pressure in diagnosing and managing hypertensive disorders in pregnancy.[1] Unfortunately, there has been confusion and uncertainty as to how diastolic pressure should be measured in pregnancy, whether it should be recorded at the point of muffling of Korotkoff sounds (phase IV) or at the point of disappearance of sounds (phase V). Interobserver variation in detecting phase V is less than that associated with phase IV and in subjects who are not pregnant the difference in diastolic pressure between the two phases is small.[2] Accordingly, phase V diastolic pressure is recommended for non-obstetric practice.[3] In pregnancy (as in other states characterised by increased cardiac output), however, a large difference has been reported between the onset of muffling and the disappearance of sounds, with muffling of sounds commonly heard down to zero.[4] Phase IV is therefore used to record diastolic pressure. The size and the distribution of the difference in diastolic pressure between phases IV and V in pregnancy, however, have not been systematically measured. We therefore conducted this study.

Subjects, methods, and results

We recorded the difference in diastolic pressure between phase IV and V Korotkoff sounds in 197 women who were pregnant (mean age 25 (range 16 to 40)) and 197 control women who were not pregnant (26 (14 to 45)). The mean arm circumference of the pregnant women was 25·7 (range 18 to 43) cm and of the controls 24·9 (19 to 34) cm. Arm circumference was greater than 33 cm (requiring a large cuff) in six pregnant women and one control. Blood pressure (mean of three recordings) was measured in the right arm with the women seated by using a Hawksley random zero sphygmomanometer. Two observers were trained to recognise the muffling and disappearance of Korotkoff sounds using a London School of Hygiene audiotape recording and a videotape recording developed in the department. The normal approximation to the binomial was used to compare proportions and Student's *t* test to compare means when the data were parametric.

The gap between phase IV and V diastolic pressure was wider in pregnant women than in control women, but the difference between the groups was small. The median gap in the pregnant group was 2·7 (interquartile range 0·7 to 4·7 mm Hg compared with 0·7 (0·0 to 3·3) mm Hg in the control group. Muffling was detected in 150 pregnant women and 118 controls (table).

As expected mean (SD) blood pressure was lower in pregnant women (systolic 106 (13·9), phase IV 68 (11·7), phase V 65 (12·4) mm Hg) than in controls (112 (13·6), 73 (10·8), 71 (11·4) mm Hg; all $p<0·0001$), and mean pulse rate was higher in pregnant women than in controls (92 (15·1) *v* 77 (12·8) beats/min; $p<0·0001$). Surprisingly, the mean arm circumference in the pregnant group was significantly higher than that in the control group ($p<0·0001$). When pregnant and control women with a phase IV-V gap >0 mm Hg and those with no gap were compared those with a gap >0 mm Hg had lower blood pressure, higher pulse rate, and greater arm circumference, though the differences were not significant. The two observers detected muffling in a similar number of women: 129 and 139.

Distribution of difference in diastolic blood pressure (mm Hg) measured at phase IV and V Korotkoff sounds among women who were and were not pregnant

	0	-2	-5	-10	>10
No of pregnant women (n=197)	47*	44	64	32	10
No of control women (n=197)	79	55	42	19	2

*Difference between groups −32; 95% confidence interval −14 to −50.

Comment

Though previous studies have described the distribution of diastolic pressure in phase IV and V in pregnancy,[4] this is the first study designed specifically to compare the interphase gap in women who were and were not pregnant. The data suggest that the gap between fourth and fifth phase diastolic pressure in pregnancy is somewhat wider on average than in the non-pregnant state, though the gap's size has been overestimated in previous reports. The principle justification for measuring phase IV diastolic pressure in obstetric practice is the belief that in many pregnant women phase IV is audible to zero and phase V is not detected. Our study does not support this view.

Assuming that this finding is replicated, we suggest that adopting phase V diastolic pressure in obstetric practice merits consideration.

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