Unique Arterial Collaterals in the Presence of Renal and Aortic Occlusions

A.K.		Salahudeen
A.		Pingle
Z.		Barri

Department of Nephrology, Central Hospital, Abu Dhabi, United Arab Emirates

A.K. Salahudeen, MRCP, Department of Nephrology, Central Hospital, PO Box 233, Abu Dhabi (United Arab Emirates)

Dear Sir,

We like to report a case of severe hypertension in whom, despite the occlusion of the abdominal aorta and the renal artery to the solitary kidney, perfusion of the vital organs was maintained through the development of numerous and unique arterial collaterals. The case highlights the critical importance of normal but trivial and usually invisible arterial anastomotic channels that take over the blood supply to vital organs in the presence of major arterial occlusions.

A 35-year-old man, who was admitted with acute renal failure and severe hypertension, recovered renal function following 4 weeks of dialysis. 15 years ago he underwent right nephrectomy for unknown reasons. Hypertension was noted to persist after the acute renal failure. He complained of recurrent headaches but denied symptoms of lower limb ischemia or sexual weakness. The femoral pulses were equally palpable and no abdominal bruit was heard. The kidney measured 12 cm on IVU and showed good perfusion on a DTPA scan. Blood pressure (mean 218/127 mm Hg) control was difficult during the next 6 months despite quadruple antihypertensive therapy.

An arteriogram showed aortic obstruction at the level of the third lumbar vertebra with absence of its distal part and branches (fig. 1). Although the left renal artery was occluded, the kidney was still opacified. Numerous vascular collaterals were present. The marginal branch, normally an inconspicuous anastomotic channel between the superior and inferior mesenteric arteries, from the superior mesenteric artery became grossly enlarged to occupy a strikingly large space in the arteriogram (fig. 1, 2). The marginal artery was also found running along the colon and branching in the pelvis. Arterial collaterals were seen on either side of the pelvis running down towards the thighs (fig. 2).

Any attempt at renovascular surgery was felt to pose a substantial threat to the existing blood supply and was deferred. Addition of captopril 150 mg to his existing antihypertensive treatment induced, as expected, moderate renal failure which persisted (average serum creatinine of 295 mol/l) with the continuation of captopril for the next 24 months. Captopril was replaced by nifedipine and the renal function returned to pretreatment level within a few days.

Surgical intervention to improve renal vascularity became unavoidable with the appearance of diuretic-resistant cardiac failure.

Fig. 1. Arteriogram showing occlusion of the aorta and the left renal artery (1), faintly opacified left kidney (2), and a branch of superior mesenteric artery developed into the marginal artery (3).

An autogenous venous bypass from the mesenteric marginal artery collateral to the left renal artery was carried out with significant improvement in the control of blood pressure and cardiac failure.

This patient, with occlusion of the distal part of the aorta and its branches, did not have impotence or symptoms of lower-limb or colonic ischemia, due to the development of excellent vascular collaterals. Although the inferior mesenteric artery was absent, the anastomotic channel between the branches of the superior mesenteric artery, namely the marginal artery [1], had developed enormously, maintaining the vascularity to the rectum

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Fig. 2. Arteriogram showing continuation of the marginal artery along the colon and its branched termination in the pelvis (black arrows) and arterial collaterals most likely between inferior hypo-gastric and superior epigastric arteries, supplying the lower limbs (white arrows). and transverse, descending and sigmoid colon. Femoral arteries were patent through the engorgement of otherwise minor anastomotic channel between superior epigastric and inferior hypogastric arteries, shown in the arteriogram as arteries running down on either side of the pelvis (fig. 2). The kidney remained viable and functioning due to the perfusion maintained by the lumbar and ureteric collaterals.

The fact that captopril induced renal dysfunction in this case, details of whom are reported elsewhere [2], returned to normal at the end of 2 years of continuous treatment, is of interest. This suggests that no organic damage occurs to glomerular arterioles following chronic Angiotensin Converting Enzyme inhibition.

References

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