Prognostic Value of Nephrography in Atherosclerotic Occlusion of the Renal Artery

To the Editor:

Atherosclerotic lesions of the renal artery are a common cause of renovascular hypertension and may ultimately lead to complete occlusion. Since renal viability can be maintained in this condition through collateral blood flow, the basic issue is how to differentiate clinically between a kidney that is viable and one that has irreversible ischemic lesions. The former should be revascularized, and the latter should be explanted.

The proposed criteria for discriminating between these two conditions, including serum creatinine levels, split renal function tests, plasma renin activity, renal length and morphology at intravenous pyelography, arteriography, renal biopsy, intraoperative angiography, and gross aspect of the kidney, are either misleading or unfeasible. In fact, in patients in whom revascularization was indicated, we observed a high recurrence rate of hypertension and no improvement in renal function.

We, and others, have discovered that the finding of a markedly delayed and reduced nephrogram during both intravenous pyelography and arteriography is the most valuable indicator of irreversible vascular and parenchymal damage. Conversely, a normal nephrogram implies that kidney function may return after revascularization.

To illustrate, we report on three severely hypertensive patients (aged 54, 57, and 64 years) with a totally occluded, atherosclerotic renal artery who had normal or slightly raised serum urea nitrogen and creatinine levels, hypersecretion of renin on the affected side plus contralateral suppression, and a slightly shrunken kidney (longitudinal length, 8.5, 9.5, and 9 cm, respectively) with poor (Patient 1) or absent radioisotope uptake. Revascularization was indicated on the basis of some of the commonly accepted criteria, but nephrectomy was performed because of an abnormal nephrogram at intravenous pyelography and in vivo angiography. Collateral blood supply was evident at in vivo angiography only in Patient 1.

In vitro microangiography and histological studies were performed in the three explanted kidneys to investigate the microvascular and parenchymal lesions responsible for the abnormal in vivo nephrogram. In all three patients, microangiographic analysis showed that the kidneys were inhomogeneously opacified for the presence of multiple infarcted areas in the cortex, which was reduced in thickness. The interlobar, arcuate, and interlobular arteries showed pruning and marked tortuosity with corkscrew aspects (Figure 1A). The glomeruli were smaller, incompletely opacified, and markedly decreased in number (Figure 2A). The number of aglomerular afferent arterioles was increased, indicating partial occlusion of afferent arterioles and irreversible glomerular ischemic damage. A conspicuous and inhomogeneous reduction in perfusion of the postglomerular peritubular network was noted. The reduction in perfusion of vasa recta was less pronounced than that of the peritubular network, possibly because of the presence of a collateral circulation bypassing the outer cortex.

The histological findings were similar in all the kidneys, and the lesions were irregularly distributed. Tiny cortical infarcts and subcapsular hemorrhages were present. Fibrosis of the intima and media and splitting of the intimal elastic membrane were seen in the interlobar and interlobular arteries. Severe intimal fibrocellular proliferation and hyalinosis of the wall were predominant in the afferent arterioles. Inhomogeneous ischemic damage secondarily involved the glomeruli, the majority of which were hyalinized. Finally, interstitial fibrosis and tubular atrophy were noted.

The microangiographic and histological findings provide substantial evidence against the traditionally accepted criteria of renal salvageability in total atherosclerotic occlusion of the renal artery. Preoperative serum urea nitrogen and creatinine levels do not predict the potential improvement of renal function with revascularization. Lateralization of renin secretion is a marker of renal ischemia and a predictor of the effect on blood pressure but not of renal salvageability. The three kidneys were of a size that would have been acceptable for revascularization, while the histological and microangiographic findings clearly demonstrated irreversible ischemic lesions. This discrepancy may depend on the length of the kidney, which varies considerably from patient to patient in relation to body build, age, and sex. Moreover, decreased renal size may reflect several factors, ranging from reversible ischemic "atrophy" with reduction of intrarenal blood volume to progressive cellular damage leading to irreversible fibrosis. Preoperative biopsy, recommended by some and not by others, gives limited information that is not representative of the entire kidney; in our three patients, the infarcted areas on the cortex were interspaced with less severe lesions. Furthermore, it may be difficult to predict whether or not these lesions are reversible. Lack of function demonstrated by split renal tests does not necessarily exclude renal salvageability because subfiltration pressure has been shown to adequately preserve renal tissue. Intraoperative
Figure 1. Pathological (A) and normal (B) microangiographic appearance through two adjacent renal lobes in 500-μm sections (original magnification ×5). In A, note the reduction of cortical thickness, an infarcted area (white arrow), and the tapering, angulation, pruning, and marked tortuosity of arcuate (a) and interlobular arteries. The number of glomeruli in the cortex and arterioles rectae vera in the medulla (m) is reduced. In B, note the normal thickness of the cortex, the regular aspect of the arcuate arteries (a), and the normal number of glomeruli in the cortex and vasa rectae in the medulla (m). s = spiral pelvic arteries.

Figure 2. Pathological (A) and normal (B) microangiographic appearance of interlobular arteries in 500-μm sections (original magnification ×67). In A, note the tortuosity of the arteries (i); the number of afferent arterioles and glomeruli (g) is reduced, while the peritubular capillaries are poorly visualized. The normal kidney (B) shows uniform opacification of glomeruli (g) and peritubular vascular network (p) and the usual number of glomeruli.
renal arteriography with cannulation of the renal artery distal to the occlusion can provide valuable information but is seldom feasible.

Our microangiographic and histological findings show that severe damage of the microvascular bed was present even in kidneys that would have been judged salvageable on the basis of renal size, overall results of renal function tests, and a single histological specimen. Thus, we suggest that an abnormal (i.e., marked filling defects) nephrogram during intravenous pyelography and in vivo angiography plus a poor collateral circulation may be a better indicator of severe, irreversible lesions than the generally accepted criteria and may aid in choosing the most appropriate treatment in atherosclerotic renovascular occlusion.

References


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(Hypertension 8: 962-964, 1986)
Prognostic value of nephrography in atherosclerotic occlusion of the renal artery.
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