A Case of Renovascular Hypertension Caused by an Unintentional Clipping of Renal Artery with Subsequent Development of Collateral Pathway through the Renal Capsular Artery

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Dear Sir,

Renovascular hypertension occurs with the increased renin secretion induced by the decrease of blood circulatory pressure in the kidney. The pathogenic lesions leading to the renovascular human hypertension are various. But they are mostly associated with the narrowing of the unilateral renal artery. Our case of renovascular hypertension is attributed to a peculiar pathogenesis; i.e. complete obstruction of left renal artery due to unintentionally leftover clipping at the artery in surgical procedure performed for testicular tumor, which was followed by the development of collateral pathway through renal capsular artery.

Case Report
TN; a 31-year-old male engineer, was hospitalized because of hypertension in October, 1986. His family history and a past history provided no clues to the conventional pathogenesis of human hypertension.
In 1980, at another hospital at the age of 25, he underwent a orchidectomy of the left testis with a dissection of abdominal lymph nodes because of left testricular tumor. Several months thereafter he was noted to have hypertension (150/130 mm Hg).
Laboratory examination on admission revealed normal serum electrolyte levels of Na 139 mEq/l, K 3.7 mEq/l and Cl 103/mEq/l. PRA was as high as 7.2 ΔAI ng/ml/h (normal range: 1.4–2.0) and plasma aldosterone concentration was slightly elevated (26 ng/dl, normal range: 3–15). Urine specimen showed no abnormality. Cate-cholamine levels in the blood and urine were normal. 24 h creatinine clearance was 93 ml/day. Glomerular filtration rate (GFR) and renal plasma flow (RPF) were 69 ml/min and 287 ml/min, respectively.
Computerized tomography showed the left contracted kidney and the slight hypertrophied contralateral kidney.
Aortography showed normal right renal artery including the vasculature in the kidney, but complete obstruction of the left renal artery by clipping. Just above occluded renal artery, the left renal capsular artery was opacified. Therefore, selective angiography of the left renal capsular artery was performed. It gave a staining in the
Fig. 1. Selective angiography of the left renal capsular artery. It gave a staining in the kidney and the renal vein in venous phase.

Table 1. PRA provocation test (captopril 25 ml p.o. renal-vein catheterization)

<table>
<thead>
<tr>
<th>Time</th>
<th>Right Renal Vein</th>
<th>Left Renal Vein</th>
</tr>
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<tbody>
<tr>
<td>Pre</td>
<td>4.4</td>
<td>56.3</td>
</tr>
<tr>
<td>1 h</td>
<td>10.8</td>
<td>86.0</td>
</tr>
<tr>
<td>2 h</td>
<td>7.9</td>
<td>160</td>
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Values are given as ΔAI ng/ml/h.

kidney and renal vein in venous phase indicating the left renal capsular artery supplying the blood to the kidney (fig. 1).

PRA provocation test was studied for a diagnosis of renovascular hypertension using angiotensin I-converting enzyme (ACE) inhibitor [1].

The blood sample for measurement of PRA from each renal vein was obtained by renal-vein catheterization after administration of ACE inhibitor (captopril 25 mg p.o.) in supine position. In table I, pre-ACE inhibitor PRA level of the left renal vein is significantly higher than that in the right renal vein. The post-ACE inhibitor PRA levels of the left renal vein became much higher than that of right vein in 1 and 2 h.

Renovascular Hypertension by the Renal Capsular Artery

Discussion

In our patient, selective angiography suggested that the blood supply of the left kidney was carried out by the collateral pathway served by left capsular artery. It is easy to speculate, however, that arteries failed to transmit adequate blood pressure to preserve appropriate renin secretion. Indeed, characteristic feature of hypertension of our case, i.e. diastolic hypertension, coincides with that of two-kidney one clip type renovascular hypertension.

Silas et al. [2] and Hall et al. [3] have both stated in the review of their cases that GFR are significantly lowered, which was attributed to the perturbation of autoregulation of the renal circulation. Our case likewise showed moderately decreased GFR and RPF in the face of slightly hypertrophied contralateral kidney, suggesting the same mechanisms may be operating as the above authors have proposed.

As expected, dosing of 5 mg/day of enalapril normalized the blood pressure without further deterioration of renal function.

References
