Preoperative and Postoperative Renin Levels in Coarctation of the Aorta

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SUMMARY We studied plasma renin activity (PRA) in eight children before and after surgical correction of aortic coarctation. These eight children underwent a combination of low-sodium diet and diuresis before surgery, and PRA was measured shortly thereafter. Thirty-two to 51 months after successful surgical correction, PRA was measured again. The mean PRA was 21.4 ± 1.3 ng/ml/hour (± sd) preoperatively and 5.5 ± 1.5 ng/ml/hour postoperatively. These findings provide further evidence of the significance of increased renin-angiotensin activity in patients with aortic coarctation.

THE EXACT MECHANISMS of hypertension accompanying coarctation of the thoracic aorta are not known. Of the several theories advanced, decreased blood flow to the renal mass with resulting hyperactivity of the renin-angiotensin system has received renewed interest. Previous studies of this theory have shown equivocal results. Recently, depletion of the extracellular volume by sodium restriction and loop diuretics has identified excessive plasma renin activity (PRA) in several hypertensive conditions secondary to decreased renal blood flow.1 Using this technique, we found excessive renin-angiotensin activity in a group of preoperative aortic coarctation patients.2 We restudied these patients to determine the effect of surgical correction of aortic coarctation on PRA.

Methods

Eight children, ages 5–16 years, who had coarctation of the thoracic aorta were admitted to the Clinical Research Center of our hospital 4 days before surgical correction. Six were hypertensive and two were normotensive. Hypertension was defined in accordance with standards for children's blood pressure developed from studies supported by the National Heart, Lung, and Blood Institute.3 The mean preoperative gradient across the coarctation was 55.3 mm Hg. Six patients had gradients measured at cardiac catheterization; the other two gradients were obtained by difference in systolic blood pressure between the right arm and left leg. All patients were placed on a 10-mEq sodium diet for 36–48 hours. Baseline serum electrolytes and 24-hour urine values for vanilmandelic acid, catecholamines and metanephrine were normal. On the morning of the third hospital day, i.v. furosemide was administered (20 mg for patients under 60 kg and 40 mg for patients over 60 kg). Three hours later, after the patients had been upright for 2 hours, blood was drawn for measurement of PRA by radioimmunoassay.4 Corrective repair, which involved excision of the coarcted segment and end-to-end anastomosis, was undertaken the next day.

All patients had a successful postoperative course and were readmitted to the Clinical Research Unit 32–51 months (mean 41.8 months) after repair. Seven patients were normotensive, but one patient was still hypertensive. None of the seven patients who were normotensive postoperatively had a systolic gradient between the right arm and right leg as measured by sphygmomanometer. The hypertensive patient had a persistent 30-mm gradient, later confirmed at cardiac catheterization. The patients were again placed on a 10-mEq sodium diet and were given a similar dose of furosemide on the third hospital day; 3 hours later, after the patients had been in the upright position for 2 hours, blood was drawn for measurement of PRA.

The statistical analyses were performed using the t test for paired data. Values are expressed as mean ± sd.

The experimental procedure was approved by the Institutional Review Board for Protection of Human Subjects in our hospital. Informed consent was obtained from the parents of each patient.

Results

Preoperative Studies

After diuresis and standing for 2 hours, the coarctation patients had a mean PRA of 21.4 ± 11.3 ng/ml/hour. In our hospital, the PRA level in normal adults on an unrestricted salt diet is 0.2–3.6 ng/ml/hour, and in those who are given 40 mg of i.v. furosemide and remain upright for 2 hours, 1.7–8.5 ng/ml/hour. In five control children pretreated like the coarctation patients, the PRA was 2.3–8.3 ng/ml/hour.

Postoperative Studies

In the late postoperative period, the stimulated PRA was 5.5 ± 1.5 ng/ml/hour. The one patient who remained hypertensive after repair also had a normal PRA. These values are significantly different (p < 0.005) from the preoperative values (table 1).

Discussion

The hypothesis relating the hypertension of coarctation to underperfusion of the renal mass remains attractive. Shortly after it became possible to measure the activity of the renin-angiotensin system, attention was focused on coarctation. Early findings were equivocal,5–7 and most authors found no significant increase

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in PRA in these patients. Recently, it has become evident that abnormalities of PRA in patients with decreased blood flow to the kidneys can be shown more clearly by depleting the extracellular volume.1 When intake of salt is unrestricted, these patients may have a relative hypervolemic state. The increased blood volume may cause an elevation in renal blood flow. Only after extracellular volume depletion can the elevated PRA be recognized. One might postulate that in coarctation, an initial decrease in renal blood flow increases renin secretion from the juxtaglomerular apparatus of the kidney. This in turn increases production of angiotensin I and II, which stimulate vasoconstriction and aldosterone hypersecretion. The resultant sodium and water retention expands extracellular volume, which increases renal blood flow and by feedback inhibition to the kidney decreases renin secretion. The patient remains hypertensive due to hypervolemia.

Recent studies using sodium restriction or diuretics have demonstrated increased PRA in hypertensive coarctation patients. Alpert et al.8 demonstrated normal PRA during unrestricted sodium intake in 12 patients whose plasma volume and extracellular fluid volume were increased above normal values. After furosemide, all patients had an excessive increase in PRA. Bagby et al.9 created coarctation in 1-week-old puppies. At 2 years, the dogs were placed on a markedly sodium-restricted diet. The PRA levels were significantly higher in these dogs than in controls. Evidence of increased blood volume in these dogs with coarctation has also been reported.10

We previously found excessive PRA in coarctation patients after salt restriction and diuresis.2 We hypothesized that if excessive PRA plays a role in the pathogenesis of hypertension in coarctation, surgical correction of the coarctation should return the PRA and the blood pressure to normal levels. Seven of our eight patients had increased PRA preoperatively, and PRA returned to normal in the late postoperative period in all seven. The preoperative hypertension was correct-

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**Table 1. Pre- and Late Postoperative Plasma Renin Activity**

<table>
<thead>
<tr>
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<th>Preop</th>
<th>Late postop</th>
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<tr>
<td>RV</td>
<td>18.5</td>
<td>5.3</td>
</tr>
<tr>
<td>AW</td>
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<td>6.3</td>
</tr>
<tr>
<td>WH</td>
<td>13.4</td>
<td>3.7</td>
</tr>
<tr>
<td>ML</td>
<td>26.9</td>
<td>6.6</td>
</tr>
<tr>
<td>DS</td>
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<td>8.3</td>
</tr>
<tr>
<td>DC</td>
<td>10.2</td>
<td>4.5</td>
</tr>
<tr>
<td>JB</td>
<td>23.1</td>
<td>4.9</td>
</tr>
<tr>
<td>RB</td>
<td>7.4</td>
<td>4.3</td>
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</tbody>
</table>

Abbreviation: PRA = plasma renin activity.

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References

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