Primary aldosteronism is a potentially curable cause of hypertension, especially when caused by an adrenal adenoma. Aldosteronomas because of their small size often elude techniques to locate them. This case illustrates the advantages, disadvantages and complications of noninvasive and invasive techniques used for their diagnosis.

A patient with hypertension and hypokalemia underwent an adrenal venous effluent sampling for measurement of aldosterone concentrations. This procedure was complicated by an injury to the right adrenal gland. Subsequently, it was difficult to control the patient's hypertension and hypokalemia with medical therapy alone. A re-assessment years after his initial diagnosis included a CT scan, which now visualized a left adrenal tumor. The functional status of this tumor and lack of function of the previously injured right adrenal gland were demonstrated by NP-59 scintigraphy. This information modified the surgical intervention (adnectomry rather than total adrenalectomy) and the residual left sided adrenal tissue prevented adrenocortical insufficiency. A year later the patient remains euadrenal.

Key Words: aldosteronoma; adrenal infarction; NP-59 scintigraphy

CASE REPORT

A 61-yr-old patient presented more than a decade after onset of initial symptoms for reevaluation of a left adrenal aldosteronoma. The patient had been hypertensive since the early 1970s and subsequently developed episodic anginal chest pain, atrial fibrillation, congestive heart failure and presyncope. Multiple hospital admissions later, the patient showed unprovoked hypokalemia. His interim medications included quinidine, diazepam, verapamil, enalapril, digoxin, nitroglycerine and lorazepam. In 1987, frustrated by repeated failures to respond to medical therapy, he sought further consultation. This evaluation included a normal coronary angiogram, while the concurrent hypertension and unprovoked hypokalemia initiated a search for aldosteronism.

Data obtained prior to his evaluation in our clinic included a low plasma renin of 0.05 ng/l/sec (0.18 ng/ml/hr) and high aldosterone of 3.2 nmole/liter (116 ng/dlter). An initial abdominal CT scan was equivocal (Table 1) and was followed by an adrenal venous effluent sampling procedure for aldosterone (Table 2) which was complicated by contrast medium extravasation into the right adrenal gland. The injury caused no symptoms, and since the patient had demonstrated an allergic reaction to spironolactone, amiloride and potassium supplements were added to the medical regimen. These drugs reduced the episodes of uncontrolled hypertension and atrial fibrillation.

At the initial visit to our clinic in 1993, the patient was asymptomatic. The blood pressure on optimum therapy was 160/100 mmHg, pulse 62 beats/min, regular with no apex pulse deficit. The pedal pulses were intact and there was no edema. The optic fundi manifested tortuous and narrow arterioles with strioventricular nicking. The lungs were clear and the cardiac examination was normal. No abdominal organomegaly or bruits were present and the patient was neurologically intact.

A CT scan depicted a 1.8 cm mass in the left adrenal, while the right gland was morphologically normal (Fig. 1A). One millicurie...
of NP-59 was injected without dexamethasone suppression and clearly visualized the left adrenal gland but not the right 5 and 8 days postinjection (Fig. 1B). This allowed us to conclude that the aldosteronism arose from the left adrenal gland, probably from an adenoma, and that the right adrenal had no function. The patient was advised to undergo selective surgical resection for the aldosteronoma. A left partial adrenalectomy was performed. Normal adrenal tissue conserved on the left side allowed for preservation of adrenocortical function. After surgery, the serum potassium normalized and the blood pressure was well-controlled with verapamil alone (mean BP-140/70). Assessment of the adrenal secretory reserve 6 wk after surgery, demonstrated a normal baseline cortisol at 0.34 μmole/liter (12 μg/dl) which rose to 0.53 μmole/liter (19 μg/dl) following cortrosyn stimulation. A year later the patient remains normokalemic and a stimulated cortisol value was 0.66 μmole/liter (23 μg/dl).

**DISCUSSION**

The patient’s initial biochemical parameters were diagnostic of primary aldosteronism and treatment with multiple medical regimens was unsuccessful.

![Image 1](https://example.com/image1.png)

![Image 2](https://example.com/image2.png)

**TABLE 1**

Results of Serial CT Scans of the Adrenals

<table>
<thead>
<tr>
<th>Date</th>
<th>Left adrenal</th>
<th>Right adrenal</th>
</tr>
</thead>
<tbody>
<tr>
<td>5/87 (prior to venous sampling)</td>
<td>Mild thickening of proximal portion of lateral limb, with lobulation approximately 6 mm diameter</td>
<td>Normal</td>
</tr>
<tr>
<td>6/87 (soon after venous sampling)</td>
<td>Unchanged 2 cm soft tissue density consistent with hemorrhage into the gland resulting from adrenal venography</td>
<td>Normal</td>
</tr>
<tr>
<td>9/87</td>
<td>Unchanged</td>
<td>Reduction in size of soft tissue mass</td>
</tr>
<tr>
<td>6/88</td>
<td>Fullness in lateral limb of left adrenal unchanged since 5/87</td>
<td>Complete resolution of right soft tissue mass</td>
</tr>
<tr>
<td>9/89</td>
<td>1.2 cm mass in left adrenal not definitely visualized on earlier studies</td>
<td>Normal</td>
</tr>
<tr>
<td>9/91</td>
<td>Unchanged since 1989</td>
<td>Normal</td>
</tr>
<tr>
<td>9/93 (at our institution)</td>
<td>1.8 cm left adrenal mass</td>
<td>Normal</td>
</tr>
</tbody>
</table>

**TABLE 2**

Selective Venous Catheterization of Adrenal Glands (5/87)

<table>
<thead>
<tr>
<th>Region</th>
<th>Aldosterone (nmole/liter)</th>
<th>Norepinephrine/epinephrine (pmole/liter)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peripheral plasma</td>
<td>0.56</td>
<td>191/113</td>
</tr>
<tr>
<td>IVC</td>
<td>0</td>
<td>1426/5455</td>
</tr>
<tr>
<td>Left adrenal vein</td>
<td>271</td>
<td>14270/57750</td>
</tr>
<tr>
<td>Right adrenal vein</td>
<td>35</td>
<td>14270/57750</td>
</tr>
</tbody>
</table>

Initial CT scans revealed left adrenal fullness, and follow-up scans demonstrated the development of an adrenal mass. Small aldosteronomas are not always readily visualized by CT. When larger than 0.8 cm, localizing accuracies range between 60% and 80%, depending upon the type of scanner and the technique used (13,14).

Adrenal effluent sampling performed early in the patient’s course, revealed high aldosterone levels bilaterally (Table 1). The left-to-right aldosterone ratio although not diagnostic was very suggestive of a left sided aldosteronoma. Additionally, the extremely high epinephrine and norepinephrine levels on the right suggest hormone release due to adrenal injury of that side. We were unaware as to the reasons for the patient’s protracted medical course, since the biochemical and localizing studies would have resulted in unilateral adrenalectomy at most centers. We presume it was due to the concern for adrenal insufficiency in the setting of contralateral adrenal injury.

This case illustrates one of the potential complications encountered with venography and venous sampling. The most serious complication, namely hemorrhage and infarction which, if bilateral, may lead to complete loss of adrenal function. There are several reports of bilateral adrenal infarction, adrenal hemorrhage (8,15) with hypocortisolism (16,17) and even death (18) related to this procedure.

NP-59 scintigraphy for aldosteronism is usually performed under dexamethasone suppression, but in this case, an unsuppressed NP-59 scan better served to demonstrate the function (or lack of function) of the morphologically normal right adrenal gland (19). The scintigraphic results permitted the surgeon to choose tumor resection (adenectomy) rather than total adrenalectomy, which is the usual and easier surgical approach. Had it been present, normal NP-59 uptake on the right side, would predict a low risk of hypoadrenalizm following a complete left adrenalectomy. Absence of NP-59 uptake, and by inference adrenal function, depicted by scintigraphy with normal morphology on CT scan could be due to organi-
zation of a hemorrhage secondary to contrast extravasation at the
time of venous sampling. If performed appropriately, as in this
case, adrenal scintigraphy has the advantage of being noninvasive,
cost-effective and accurate in defining functionality of an adrenal
tumor. When performed under dexamethasone suppression, its
efficacy exceeds that of CT and MRI especially in the diagnosis of
bilateral adrenal hyperplasia. Series of NP-59 scintigraphy yield
varied results of efficacy due either to differences in techniques,
dexamethasone suppression protocols, and variation in interpreter
experience. In our experience an accuracy as high as 94% and
sensitivity of 96% has been noted (14).

When aldosteronism is biochemically confirmed (with high
plasma or urinary aldosterone levels and suppressed plasma
renin activity), an evaluation for the site(s) of aldosterone
hypersecretion must be initiated. CT scanning or adrenal
scintigraphy usually correctly lateralize an aldosteronoma. CT
scanning is used first as it is widely available and may be
sufficient when it yields an unequivocal result. NP-59 is most
useful when CT results are equivocal, for the positive identifi-
cation of bilateral hyperplasia and in problem cases such as this
one. Adrenal venous sampling is reserved for cases where both
the above tests fail to identify the adrenal abnormality.

Sampling in addition to being invasive, requires the admin-
istration of iodinated contrast and the skills of a highly
experienced invasive radiologist. NP-59 scintigraphy plays an
important role in aldosteronism for tumor localization and the
differentiation from bilateral adrenal hyperplasia. Additionally
as this case illustrates, scintigraphy can locate sites of residual
adrenocortical function when there has been injury to one or
both adrenal glands. Adrenocortical radiopharmaceuticals are
widely available around the world, but in the United States
NP-59 remains an Investigational New Drug and thus must be
utilized as part of an experimental protocol which is a signifi-
cant logistic hurdle in its widespread use.

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Gastric Antral Vascular Ectasia: A Case Report and
Review of the Literature

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Hospital and Health Center, Billings, Montana; Department of Radiology and Nuclear Medicine, National Naval Medical
Center, Bethesda, Maryland; and Office of the Attending Physician, U.S. Capitol, Washington, D.C.

We present an 83-yr-old woman with a history of renal insufficiency,
diabetes and idiopathic thrombocytopenic purpura (ITP) who expe-
rienced recurrent hemorrhage from gastric antral vascular ectasias
(GAVE). Methods: Extensive evaluation consisting of barium small
bowel series, colonoscopy, abdominal CT scan and visceral angio-
ography excluded other causes of bleeding. Results: After 99mTc-
labeled red cell imaging to localize the bleeding to the antrum,
an antrectomy was performed. Seven months post-surgery, the patient
experienced no further hemorrhage. Conclusion: 99mTc-labeled red cell scans can be used for the diagnosis of GAVE.

Key Words: gastric antral vascular ectasias; technetium-99m-RBCs

Gastric antral vascular ectasia (GAVE) is a rare condition
associated with chronic iron deficiency anemia (1-5). GAVE
usually occurs in elderly women and has been reported in
association with achlorhydria (4) and cirrhosis (2). In 1984, Jahbari et al. (5) coined the condition with the term “water-
melon” stomach (5) due to the characteristic linear antral
streaking seen endoscopically.

Hemorrhage from GAVE (6) is rare. We describe a case
of GAVE with recurrent hemorrhage documented by a 99mTc-
labeled red cell scan and reviewed the reported cases of this
entity.

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The opinions and assertions contained herein are the private ones of the authors and
are not to be construed as official policy or as reflecting of the Department of Defense.
Simultaneous Scintigraphic Depiction of Aldosteronoma and Adrenal Infarction

Roma Y. Gianchandani, Gregory A. Quin, Roger J. Grekin, Milton D. Gross, James C. Sisson, Norman W. Thompson and Brahms Shapiro


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