This 76-year-old woman was diagnosed with hypertension during pregnancy. However, blood pressure (BP) returned to normal after delivery and did not recur until 1 year ago when, during an evaluation for transient visual disturbance, BP was 204/102 mm Hg. There was no evidence of stroke or transient ischemic attack by neurologic examination and computed tomographic scan of the head. Office BP was 180/84 mm Hg, on 75 mg of atenolol and 12.5 mg of hydrochlorothiazide. Daytime BP was 140/73 mm Hg and nighttime BP 149/79 mm Hg by ambulatory BP monitoring. With the addition of 10 mg of amiodipine, BP control improved. However, there was still transient high readings by home recordings. Blood chemistries were normal with potassium 4.0 mEq/L. A supine plasma renin and serum aldosterone were 0.18 ng/mL/h (normal, 0.15 to 2.33) and 72.4 ng/dL (normal, 1.0 to 16), respectively. Aldosterone to renin ratio was 40.2 (normal, <25). These findings indicated hyperaldosteronism. Magnetic resonance image of the abdomen (panel A) showed hypertrophy of the left adrenal gland (arrow). The right adrenal gland (not well visualized in this view) appeared normal. Although an aldosterone producing adenoma (APA) was not visualized, the magnetic resonance image did not clearly establish the diagnosis of bilateral primary adrenal hyperplasia (PAH) or idiopathic hyperaldosteronism (IHA). Adrenal venous sampling for aldosterone was done. Panel B shows contrast injected in the left adrenal vein. There was no tumor blush, which may outline an adenoma. Baseline aldosterone was elevated in both the right and left adrenal veins, 48.2 ng/dL and 54.2 ng/dL, respectively. After infusion of adrenocorticotropic hormone (ACTH), these values increased to 2318 ng/dL in the right adrenal vein and 5532 ng/dL in the left adrenal vein. Although higher values were noted in the left adrenal vein, there were significantly high readings bilaterally suggesting PAH or IHA. A bedside test for autonomy was performed. Baseline total cortisol, 18-OH corticosterone, and serum aldosterone were 18.2 µg/dL (normal, 4 to 22 µg/dL), 76 mg/dL (normal, 4 to 37 mg/dL), and 26 ng/dL (normal, 2 to 9 ng/dL) with an 18-OH corticosterone/cortisol ratio of 4.2. After a 2-L infusion of saline, repeat assays were 13.2 µg/dL, 32 ng/dL, and 7 ng/dL, respectively, with 18-OH corticosterone/cortisol ratio 2.42. A ratio of less than 3 is suggestive of IHA. In IHA, aldosterone and 18-OH corticosterone (which are renin dependent) decrease more than cortisol in response to saline volume expansion. Cortisol is influenced only by the circadian rhythm of ACTH, whereas aldosterone and 18-OH corticosterone have an additional decrease when saline produces exogenous suppression of the renin angiotensin system. This is not the case with adrenomas, which are more autonomous to the renin angiotensin system. IPA is characterized by micro- or macronodular hyperplasia of both adrenals and rarely as unilateral disease. The choice of therapy for this patient is medical therapy with antihypertensive medication and potassium-sparing diuretics, with spironolactone as the initial drug of choice, as amiloride may be less likely to adequately control BP. This patient responded well to a combination of spironolactone and atenolol.

References