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Case Report

Transarterial embolization of a hyperfunctioning aldosteronoma in a patient with bilateral adrenal nodules

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ABSTRACT

Primary hyperaldosteronism often results in resistant hypertension and hypokalemia, which may lead to cardiovascular and cerebrovascular complications. Although surgery is first line treatment for unilateral functioning aldosteronomas, minimally invasive therapies may be first line for certain patients such as those who cannot tolerate surgery. We present a case of transarterial embolization (TAE) of an aldosteronoma. The patient presented with a cerebrovascular accident, and subsequently developed uncontrolled hypertension, hypokalemia, and a myocardial infarction. Following TAE, potassium returned to normal levels and blood pressure control was improved. There were no postoperative complications. TAE thus may be a safe and effective alternative to surgery.

Introduction

Primary hyperaldosteronism, also known as Conn syndrome, is the excessive secretion of aldosterone from hyperfunctioning adrenal gland tissue, resulting in intractable hypertension and hypokalemia, which themselves may lead to cerebrovascular disease, myocardial infarction, congestive heart failure, and cardiac arrhythmias [1,2]. Most cases of primary hyperaldosteronism are secondary to either an adrenal adenoma or bilateral adrenal hyperplasia [1]. Surgical resection is currently the standard treatment for adrenal adenomas. However, percutaneous intervention may play a role in treatment, particularly in those patients that may not tolerate surgery. Here, we present a case of transarterial embolization (TAE) with ethanol and embospheres of an aldosteronoma in a patient with bilateral adrenal nodules and discuss the presentation, assessment, and treatment of functioning aldosterone tumors.

Competing Interests: Dr. Narayanan serves as a consultant at AngioDynamics Inc. All other authors have declared that no competing interests exist.

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Case report

The patient was a 70-year-old Hispanic male with a past medical history of an adrenal incidentaloma diagnosed 8 years ago, medically controlled hypertension, congestive heart failure, and atrial fibrillation who presented to an outside hospital for a cerebrovascular accident (CVA). Given the patient's history of adrenal nodule, the CVA was thought to be secondary to hypertension from a hyperfunctioning adrenal nodule. A contrast computer tomography (CT) scan was performed, which demonstrated a right nodule measured at 2.2 × 2.5 × 2.4 cm and a left nodule measured at 3.3 × 2.2 × 3.0 cm (Fig. 1). The right adrenal mass demonstrated approximately 88 Hounsfield units (HU) on the arterial phase, 52 HU in the portal venous phase, and 41 HU on the delayed phase, with a 21% relative washout. The left adrenal mass measured 80 HU, 59 HU, and 37 HU in the arterial, portal venous, and delayed phases, respectively, with a 37% relative washout. Both nodules were thus considered to be of indeterminate etiology based on CT criteria. The patient had refractory hypertension and while undergoing physical therapy for the CVA, his blood pressure suddenly rose to 280/110 mmHg. He was subsequently admitted to our hospital, where his blood pressure remained erratic and he developed hypokalemia with a nadir of 2.8 mEq/L.

Endocrine evaluation revealed a plasma aldosterone of 25 ng/dL and a plasma renin activity level of 0.29 ng/mL·h. The patient was scheduled for adrenal vein sampling, but he developed a non-ST elevation myocardial infarction before the procedure requiring emergent cardiac catheterization with placement of a drug-eluting stent. Subsequently, adrenal venous sampling was performed to elucidate the etiology of his primary hyperaldosteronism. Results are summarized in Table 1, which demonstrates right lateralization (aldosterone ratio > 4:1) [3]. Surgical consultation was performed and the patient was deemed a poor surgical candidate. Interventional radiology was then consulted for a minimally invasive alternative.

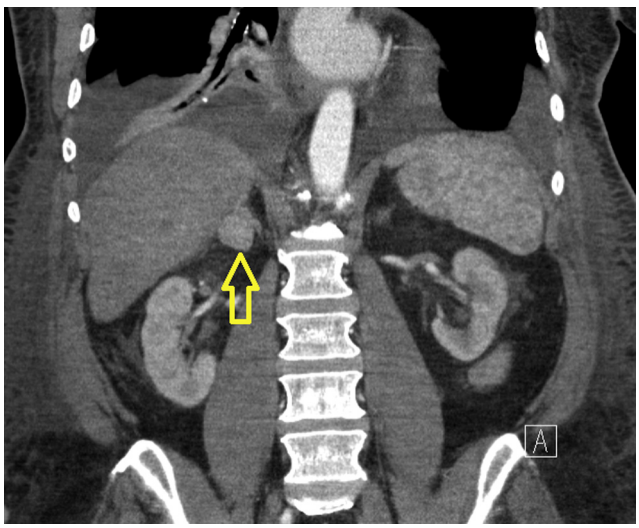

Fig. 1 – Coronal CT slice demonstrating bilateral adrenal nodules with the right nodule (arrow) measuring 2.2 × 2.5 × 2.4 cm.

Table 1 – Results of adrenal venous sampling. Adrenal-to-adrenal aldosterone-to-cortisol ratio greater than 4 indicates lateralization of disease, in this case of the right adrenal gland. Adrenal-to-IVC cortisol greater than 5 indicates correct placement of catheter in adrenal vein. Contralateral adrenal aldosterone suppression is noted.

Vein	Aldosterone (A), ng/dL	Cortisol (C), μg/dL	A:C ratio	Aldosterone ratio
Right adrenal vein	1279	483.4	2.64	22
Left adrenal vein	51	408.5	0.12	
IVC	73	61.7	1.18	

IVC, inferior vena cava.

The patient was placed under general anesthesia and intubated because of his labile blood pressures. After accessing the right common femoral artery, we cannulated the right lateral adrenal artery using the standard technique. Arteriogram showed significant adenoma blush (Fig. 2). Superselective coil embolization of an anomalous phrenic branch was then performed (Fig. 3). The catheter was next pulled back into the right lateral adrenal artery, and 3 mL of hydrated alcohol was injected. At this time, the patient experienced high blood pressures managed by the anesthesia team. Roughly 1 mL of 300-500 μm embospheres was then injected until stasis was achieved after which the proximal right lateral adrenal artery was coiled. Postembolization contrast injection demonstrated no tumor blush (Fig. 4). Right renal artery angiogram showed no evidence of adrenal capsular supply (Fig. 5). Right inferior phrenic artery angiography showed perfusion to the superior lateral limb of the adrenal gland without adenoma blush (Fig. 6).

The patient tolerated the procedure well and was discharged 5 days later. Aldosterone levels normalized and renin levels began to increase. His potassium stabilized at borderline normal levels. As of 2-month follow-up, his blood pressure has been well maintained on antihypertensive medications.

Discussion

Primary aldosteronism is the most common cause of secondary hypertension, accounting for up to 15% of all hypertension cases [4,5]. Conn syndrome, named after Dr Jerome W. Conn who first described the disease, refers specifically to primary hyperaldosteronism resulting from an adrenal aldosteronoma. Although previously considered a rare disease, it has been increasingly recognized as a secondary cause of hypertension that may improve, or resolve, with adrenalectomy [5,6].

Clinical signs of the disease result from excessive aldosterone in the renal tubules and vascular injury [1]. Aldosterone hypersecretion induces excessive renal reabsorption of sodium at the