

# Successful Radiofrequency Ablation of Bilateral Macronodular Adrenal Cortical Disease for Cushing's Syndrome in a 6-Year-Old Girl: Preservation of Adrenal Function.

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## AUTHORS' CONTRIBUTIONS

All authors participated in the study's design, as well as its review and editing processes. SHS and WHL were responsible for data collection, while DMN and YHL took the lead in drafting the manuscript. SHS and HLT provided oversight of the manuscript. All authors have reviewed and approved the final version of the manuscript.

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## DISCLOSURES

The authors declare no conflicts of interest.

## CONSENT

Yes. Written informed consent was obtained from the patient's family for the publication of this manuscript.

## HUMAN RIGHTS

This case report was conducted in accordance with the ethical principles set out in the Helsinki Declaration of 1975, as revised in 2000. No experimental procedures were performed; the patient underwent routine clinical examinations and conventional treatment. Informed consent was obtained from the patient and their family for the publication of photographs and medical records for academic purposes, with full awareness of the study's aims and potential uses of the provided information.

## ABSTRACT

**Background:** Bilateral macronodular adrenal cortical disease (BMACD) has not been reported in children. In adults with overt CS due to BMACD, bilateral adrenalectomy is a common intervention but typically leads to lifelong dependency on glucocorticoid and mineralocorticoid replacement, increasing the risk of adrenal crises. This highlights the need for adrenal-sparing treatment alternatives.

**Case Presentation:** A 6-year-old girl presented with growth delay, moon facies, hirsutism, hypertension, short stature, and obesity. Biochemical tests confirmed corticotropin-independent CS, with elevated midnight cortisol, high 24-hour urinary cortisol, and undetectable ACTH. An abdominal CT revealed multiple adrenal nodules, consistent with BMACD.

**Results:** CT-guided radiofrequency ablation (RFA) of bilateral adrenal adenomas led to complete resolution of CS symptoms and normal adrenal function at one-year follow-up.

**Conclusion:** CT-guided RFA is an effective adrenal-sparing alternative to bilateral adrenalectomy for corticotropin-independent CS, eliminating the need for lifelong glucocorticoid replacement while minimizing adrenal crisis risk in pediatric patients.

## CASE REPORT

## CASE REPORT

A 6-year-1-month-old girl from Kinmen, a small island in Taiwan, presented with hypertension, lack of growth in height, and rapid weight gain beginning around age 4. Her anthropometric measurements were 100 cm (<3rd percentile) and 26.3 kg (85-97th percentile). Over the past year, she had developed a moon face, a fatty hump on the back of her neck, pubic hair, and striae on the medial sides of both thighs. Hypertension (systolic blood pressure ~150 mmHg) was noted, and she was started on captopril (7.5 mg BID) and amlodipine (2.5 mg QD) to maintain her blood pressure within an acceptable range. Physical examination revealed distinct Cushingoid features, including central obesity, facial plethora, buffalo hump, and purple striae on the medial thighs and left axilla. Early pubertal signs, with Tanner stage 2 breast budding and pubic hair, as well as axillary hair growth, were noted. Skin examination was otherwise unremarkable, with no lentigines, blue nevi, or café au lait spots. The family history was negative for genetic or endocrine disorders.

Under suspicion of Cushing syndrome, further evaluation confirmed hypercortisolism. Both midnight (27.9 µg/dL; reference <7.5 µg/dL) and early morning (23.1 µg/dL; reference 4.8-19.5 µg/dL) serum cortisol levels were elevated, with a loss of the typical circadian rhythm. A 24-hour urinary free cortisol (UFC) level was markedly elevated at 1395 µg/24 h (reference 21-111 µg/24 h). Plasma ACTH levels were undetectable (<5 pg/mL; reference <46 pg/mL), suggesting ACTH-independent Cushing syndrome. Additional laboratory results showed a normal 17-hydroxyprogesterone level, but elevated levels of dehydroepiandrosterone sulfate (DHEA-S, 4.8 µmol/L; reference 0.08-2.3 µmol/L), androstenedione (ASD, 3.5 ng/mL; reference 0.06-1.15 ng/mL), and testosterone (0.66 ng/mL; reference <0.4 ng/mL for age). Aldosterone (41.7 pg/mL; reference 40-310 pg/mL) and plasma renin activity (PRA, 1.1 ng/mL/hr; reference 0.3-1.9 ng/mL/hr) were within normal ranges, but the aldosterone-to-renin ratio (ARR) was elevated at 39.7 (reference <30). Genetic testing through whole exome sequencing (WES) on peripheral blood leukocytes did not reveal germline mutations in the ARMC5 gene or other related genes. Tumor tissue was unavailable for somatic mutation analysis.

**Imaging findings**

Abdominal contrast-enhanced computed tomographic (CT) imaging revealed multiple nodular lesions in both adrenal glands, with at least five nodules on each side. The largest nodules measured 1.4 cm on the right and 1.3 cm on the left, consistent with bilateral macronodular adrenal cortical disease (Figure 1).

**Management and Follow-up**

Given the patient's young age and the invasive nature of adrenalectomy, along with its potential lifelong complications, her parents opted for CT-guided radiofrequency ablation (RFA) of the bilateral adrenal tumors. The ablation procedure

was performed under intravenous general sedation using fentanyl. With the patient in the prone position, a 17-gauge V-Tip™ (Variable Exposure Length Tip, RF Medical Co., Seoul, Korea) electrode was utilized to ablate the bilateral adrenal tumors under CT guidance. The exposure length of the electrode was determined based on the tumor size. A total of four needle trajectories (three on the right side and one on the left side) were employed, with two instances of needle repositioning, resulting in total six ablation zones. The mean ablation time was 6 minutes and 24 seconds, and the average final temperature reached 77.67°C. Complete lesion coverage with five breaks was achieved for all targeted areas. During the procedure, the patient was managed with propranolol (20 mg TID) to mitigate a transient hypertensive crisis caused by the release of catecholamines from the injured adrenal adenomas. The RFA procedure was successfully completed without any immediate complications such as hematoma, pneumothorax, or other adverse events. High-dose hydrocortisone (2 mg/kg/day) was applied to counter adrenal insufficiency resulting from suppression of the hypothalamic-pituitary-adrenal (HPA) axis due to chronic hypercortisolism. Propranolol was tapered and discontinued within one week, and the high dose of hydrocortisone was gradually reduced to a physiologic replacement dose (12-15 mg/m<sup>2</sup>/day, divided three times daily) within one week, with complete discontinuation achieved by two months postoperatively. Considering that the average time for HPA axis recovery after surgical cure of Cushing's syndrome in children is approximately 12.6 ± 3 months [1], we remain vigilant about the risk of adrenal crises due to incomplete HPA axis recovery, despite the early discontinuation of hydrocortisone. Therefore, hydrocortisone remains prescribed with explicit instructions for parents to administer stress dosing in response to acute stress, such as during episodes of illness, trauma, or surgical procedures.

The patient's recovery was rapid, with normalization of cortisol levels (morning cortisol 14 µg/dL; reference range 4.82-19.5 µg/dL) and an increase in ACTH levels (7.06 pg/mL) one-month post-RFA. At her six-month follow-up, afternoon serum cortisol levels remained within the normal range (4.37 µg/dL; reference range for afternoon 2.47-11.9 µg/dL), and afternoon ACTH levels (5.47 pg/mL) were also normal. Other hormones, including testosterone, DHEA-S, and androstenedione (ASD), and the aldosterone-to-renin ratio (ARR), were all within normal ranges. She exhibited significant weight loss and resumed normal growth in height. One year after treatment, her physical appearance had returned to normal (Figure 2).

## DISCUSSION

**Etiology & demographics**

Cushing syndrome (CS) is a rare endocrine disorder characterized by prolonged exposure to elevated glucocorticoid levels, particularly cortisol. Its rarity and potential post-treatment complications make CS particularly challenging for clinical diagnosis and management. The estimated incidence of

endogenous CS ranges from 0.7 to 2.4 cases per million people annually, with only 10% of cases occurring in the pediatric population [2,3]. In pediatric patients, Cushing syndrome (CS) most commonly results from an adrenocorticotrophic hormone (ACTH)-secreting pituitary adenoma, known as Cushing disease (CD), which accounts for approximately 75–90% of cases. CS is categorized as either ACTH-dependent, which includes CD and, rarely, ectopic ACTH syndrome (<1%), or ACTH-independent. ACTH-independent CS can arise from cortisol excess due to primary adrenal abnormalities, such as unilateral adrenal adenomas (10–22%) or, less commonly, adrenal carcinomas (<5%). Rare causes of ACTH-independent CS include primary bilateral macronodular adrenal cortical disease (BMACD) and primary pigmented nodular adrenocortical disease (PPNAD), each representing about 2% of cases [2,4]. The new WHO classification has refined the classification of adrenal cortical nodular disease to include (a) sporadic nodular adrenocortical disease, (b) bilateral micronodular adrenal cortical disease (miBACD), and (c) bilateral macronodular adrenal cortical disease (BMACD), formerly called bilateral macronodular adrenal hyperplasia [5]. BMACD is extremely rare, constituting less than 2% of all CS cases and is almost exclusively documented in adults. To our knowledge, after literature review, we have not found any previously reported cases in young children of this age.

### Clinical & imaging findings

Bilateral macronodular adrenal cortical disease (BMACD) is an extremely rare cause of Cushing syndrome (CS), accounting for less than 2% of cases [6]. Although BMACD can present with overt CS, it more commonly appears as a milder, subclinical form known as autonomous cortisol secretion (ACS). BMACD primarily affects individuals over 50 years old, with pediatric cases being exceedingly rare [4]. Only a minority of BMACD cases exhibit clinically overt CS. Typically, hypercortisolism follows a slow, insidious course, with gradual increases in both tumor size and cortisol levels, often delaying diagnosis for years or even decades [7,8]. Our patient, a six-year-old with overt CS symptoms, represents a unique case given her young age and pronounced clinical presentation, distinguishing her from the typical BMACD profile.

Cushing's syndrome (CS) secondary to bilateral adrenal cortical disease may be caused by bilateral macronodular adrenal cortical disease (BMACD) or bilateral micronodular adrenal cortical disease (miBACD). The size of adrenal nodules is a key factor for differentiating between these two entities (>1 cm for BMACD and <1 cm for miBACD) [9]. Cross section imaging such as CT or magnetic resonance imaging (MRI) can demonstrate multiple nodules in bilateral adrenal glands with visible normal adrenal tissues.

The bilateral nature of adrenal lesions and documented familial cases of BMACD support the hypothesis of a germline genetic predisposition [10]. Pathogenic mutations in the armadillo repeat-containing protein 5 (ARMC5) gene are

currently recognized as a major cause of BMACD, accounting for approximately 20–55% of cases [11–13]. The ARMC5 gene typically follows a "two-hit" model, whereby a somatic "second hit" in addition to a germline inactivating mutation of ARMC5 gene drives BMACD development [14]. Patients with ARMC5 mutations generally present with earlier onset, larger nodules, higher cortisol levels, and more severe clinical manifestations [12], with certain symptoms appearing specific to ARMC5 mutations [15]. Due to the more severe presentation, patients with ARMC5 mutations more frequently require surgical intervention [10]. Thus, identifying ARMC5 mutations in BMACD patients may be important for guiding treatment decisions [16]. In our patient, no germline mutation in ARMC5 or related genes was detected through whole-exome sequencing (WES), and no tumor sample was available to assess potential somatic mutations in these genes.

### Treatment & prognosis

The optimal therapeutic approach for bilateral macronodular adrenal cortical disease (BMACD) remains controversial. In cases of overt Cushing's Syndrome (CS), intervention is imperative. Historically, bilateral adrenalectomy has been considered the standard surgical approach, offering minimal recurrence risk with complete resection [17]. However, this procedure necessitates lifelong glucocorticoid and mineralocorticoid replacement therapy and carries associated risks of adrenal crisis. Contemporary practice has shifted toward unilateral adrenalectomy, targeting the larger adrenal gland or the one demonstrating higher iodo-cholesterol uptake. This approach significantly reduces the risk of adrenal insufficiency [18]. Initial studies reported promising outcomes, with initial hypercortisolism control rates of up to 100% [19]. However, a recent series of 39 BMACD patients demonstrated less favorable long-term results, with only 32% achieving sustained hypercortisolism control. In this cohort, 12% received contralateral adrenalectomy, and a concerning hypercortisolism-related mortality rate of 15% was observed. Despite this approach, approximately 68% of patients experience a recurrence of CS after long-term follow-up [20]. Notably, no pediatric cases of BMACD treatment and outcomes have been reported to date.

Image-guided ablation is a minimally invasive technique used to destroy diseased tissue by direct applying heat, cold, or other energy sources. This approach relies on guidance by advanced imaging modalities, such as ultrasound, CT, or MRI, to precisely target the affected area while avoiding damage to surrounding healthy tissue. Application of image-guided ablation has been applied on unilateral or bilateral adrenocortical tumors with Cushing syndrome and satisfactory results have been achieved [21]. RFA offers distinct advantages in treating BMACD through its ability to selectively target cortical nodules while preserving normal adrenal tissue architecture and function, which is a major concern in pediatric population. In our patient, CT-guided ablation enabled precise targeting and successful treatment of all visible lesions while maintaining

physiological adrenal function—an outcome distinctly different from both bilateral adrenalectomy, which inevitably results in adrenal insufficiency, and unilateral adrenalectomy, which carries risks of disease persistence or recurrence. The favorable outcome achieved with this targeted approach suggests that adrenal thermal ablation represents a promising alternative to traditional adrenalectomy in selected cases of BMACD and potentially other adrenal adenomas.

### Differential Diagnoses

When performing image-guided ablation for patients with BMACD, the primary goal is to precisely target hyperfunctioning adenomas while preserving as much normal adrenal tissue as possible. This approach helps prevent adrenal insufficiency, control clinical symptoms, and reduce the risk of relapse. Cross-sectional imaging, including CT and MRI, is the most effective method for identifying prominent adenomas. Additionally, adrenal scintigraphy, such as  $^{18}\text{F}$ -FDG PET or  $^{131}\text{I}$ -MIBG, may assist in differentiating hyperfunctioning nodules from non-functional ones. Although studies suggest that in BMACD, the most prominent nodules on CT often correspond to hyperfunctioning nodules on scintigraphy [19], identifying non-secreting nodules among all lesions in PBMAH patients could potentially reduce the ablation area, enabling more precise and targeted treatment.

### TEACHING POINT

Bilateral macronodular adrenal cortical disease (BMACD) in pediatric patients can present with clinical features of Cushing syndrome, including rapid weight gain, hypertension, and growth failure. Imaging typically reveals multiple nodular lesions in both adrenal glands, and a minimally invasive treatment approach like CT-guided radiofrequency ablation (RFA) may effectively manage this condition while preserving adrenal function, offering a potential alternative to adrenalectomy.

### QUESTIONS

**Question 1:** Which of the following symptoms is not typically associated with Cushing's syndrome?

- A. Central obesity with a rounded "moon face" appearance
- B. Purple striae on the skin, particularly the abdomen
- C. Hypoglycemia (low blood sugar)
- D. Proximal muscle weakness

**Answer:** C. Hypoglycemia.

Cushing's syndrome is characterized by hypercortisolism, which often causes hyperglycemia (high blood sugar) due to cortisol's effects on glucose metabolism. Hypoglycemia is not a typical symptom of Cushing's syndrome. Other common features include central obesity, purple striae, and proximal muscle weakness.

**Question 2:** Which of the following distinguishes bilateral macronodular adrenal cortical disease (BMACD) from bilateral micronodular adrenal cortical disease (miBACD) on imaging?

- A. Absence of adrenal nodules

- B. Nodules >1 cm in size
- C. Irregular adrenal gland atrophy
- D. Hypodense adrenal lesions with calcifications

**Answer:** B. Nodules >1 cm in size

BMACD is characterized by nodules larger than 1 cm, whereas miBACD typically presents with smaller nodules (<1 cm).

**Question 3:** Which of the following is characteristic of Bilateral Macronodular Adrenal Cortical Disease (BMA)?

- A. Hypersecretion of cortisol due to bilateral adrenal nodules
- B. Excessive aldosterone production leading to hypertension
- C. Decreased production of androgens
- D. Primary hyperparathyroidism

**Answer:** A. Hypersecretion of cortisol due to bilateral adrenal nodules.

Bilateral Macronodular Adrenal Cortical Disease (BMA) is characterized by the presence of large adrenal nodules in both adrenal glands, leading to excess cortisol production. This condition can cause features of Cushing's syndrome. It is not primarily associated with aldosterone or androgen overproduction, nor is it related to hyperparathyroidism.

**Question 4:** What potential complication during radiofrequency ablation (RFA) of adrenal nodules must be closely monitored and managed?

- A. Severe adrenal insufficiency
- B. Catecholamine release causing hypertensive crisis
- C. Tumor rupture and peritoneal bleeding
- D. Prolonged cortisol deficiency after ablation

**Answer 4:** B. Catecholamine release causing hypertensive crisis.

During RFA, catecholamines can be released from injured adrenal tissue, leading to transient hypertensive crises, which require management with medications like propranolol.

**Question 5:** What is a potential advantage of CT-guided radiofrequency ablation (RFA) compared to bilateral adrenalectomy for BMACD?

- A. Lower risk of recurrence
- B. Complete prevention of future adrenal nodules
- C. Immediate cessation of hypercortisolism
- D. No need for postoperative monitoring
- E. Preservation of adrenal function (applies)

**Answer:** E. Preservation of adrenal function.

RFA offers an adrenal-sparing treatment, reducing the need for lifelong glucocorticoid and mineralocorticoid replacement therapy, which is typically required after bilateral adrenalectomy.

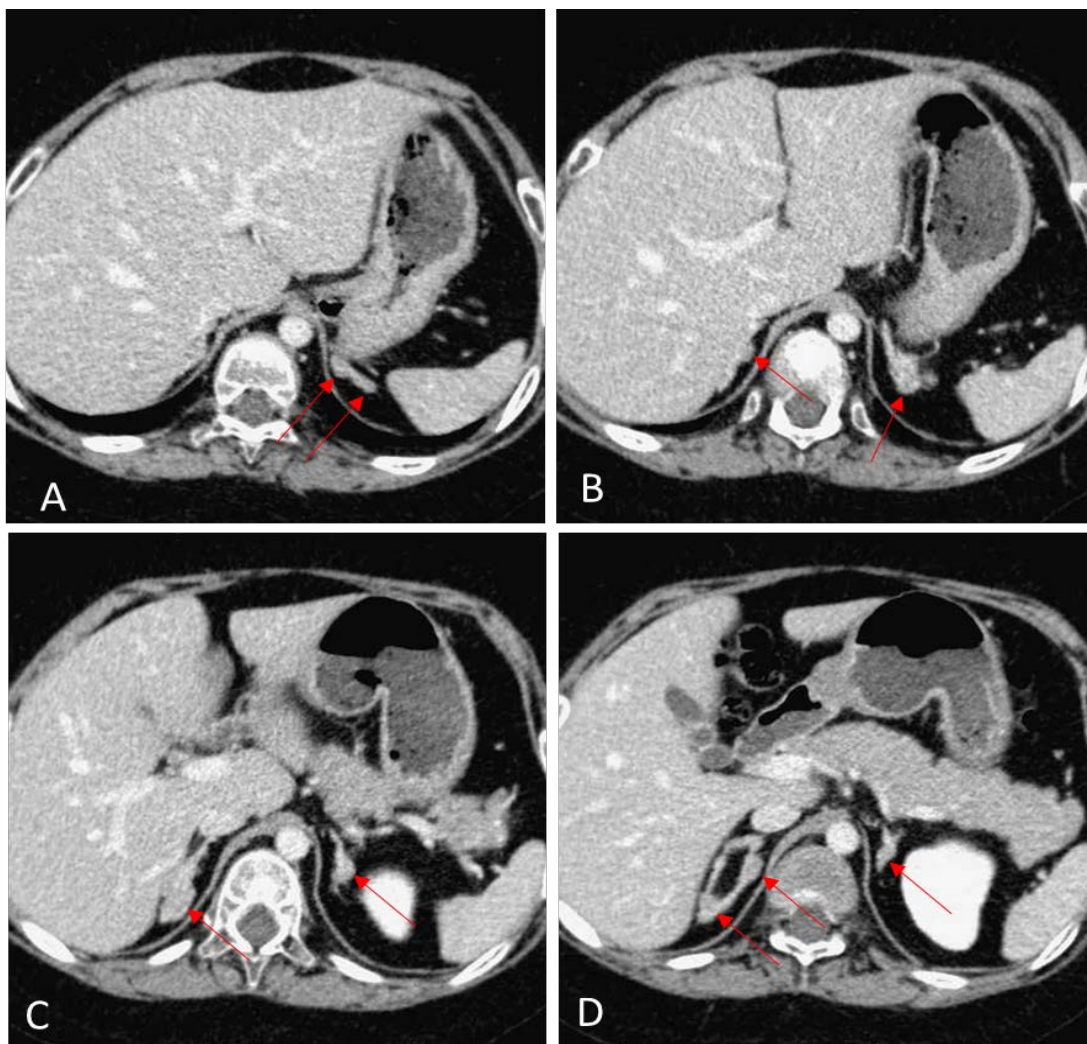
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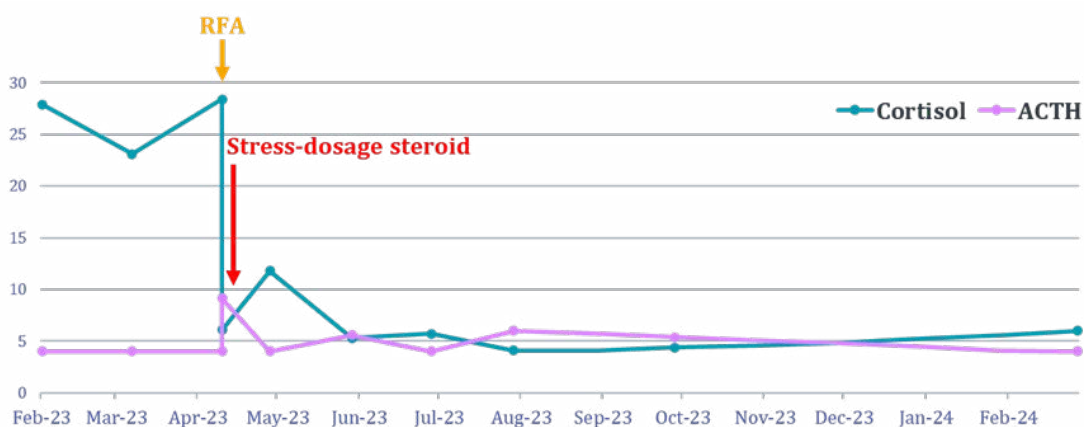


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## FIGURES



**Figure 1:** Contrast enhanced CT (A-D, sequential axial scans from cranial to caudal) shows multiple nodular lesions in bilateral adrenal glands (at least 5 on each side, red arrows), with the most prominent ones 1.4 cm on right side and 1.3 cm on left side, compatible with bilateral macronodular adrenal cortical disease.



**Figure 2:** This figure illustrates the temporal progression of cortisol and ACTH laboratory values, alongside photographs that capture the gradual normalization of the patient's physical appearance throughout the follow-up period. RFA: time of receiving radiofrequency ablation.

## KEYWORDS

*Bilateral macronodular adrenal cortical disease, BMACD, bilateral adrenalectomy, Cushing's Syndrome, radiofrequency ablation*

## ABBREVIATIONS

ACTH = Adrenocorticotrophic Hormone  
BMACD = Bilateral Macronodular Adrenal Cortical Disease  
CS = Cushing's Syndrome  
CT = Computed Tomography  
HPA = Hypothalamic-Pituitary-Adrenal  
miBACD = Bilateral Micronodular Adrenal Cortical Disease  
RFA = Radiofrequency Ablation  
UFC = URINARY FREE CORTISOL

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