

Hypertension Secondary to a Pheochromocytoma in the Context of a Multiple Adrenal Tumor: A Case Report

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Abstract

Pheochromocytoma is a rare but curable cause of hypertension. When it is not discovered incidentally during a morphological examination, it can cause variable, fluctuating, nonspecific clinical manifestations, and sometimes even no symptoms at all. We report the case of a 42-year-old man who was being treated for uncontrolled hypertension with dual therapy; investigation of an adrenal incidentaloma, discovered during a workup for hypertension, led to the diagnosis of an isolated right adrenal pheochromocytoma. The patient underwent laparoscopic adrenalectomy, with a postoperative course marked by normalization of blood pressure and the subsequent development of adrenocortical insufficiency. Pathological examination confirmed the pheochromocytoma, with loss of fumarate hydratase expression and multiple adjacent adrenocortical nodules secreting cortisol and aldosterone. Our case illustrates the need to consider a secondary cause when evaluating hypertension in a young patient or in cases of de novo hypertension, the value of abdominal imaging in the workup, the complexity of mixed secretions (catecholamines and cortisol), and the importance of postoperative endocrine follow-up for the early detection of adrenal insufficiency. Finally, the discovery of fumarate hydratase (FH) deficiency requires genetic evaluation and personalized follow-up.

Keywords

Secondary Hypertension, Pheochromocytoma, Multiple Adrenal Tumors

1. Introduction

High blood pressure remains the leading risk factor for cardiovascular disease worldwide [1]. It is estimated that 1.39 billion people worldwide have high blood pressure, resulting in approximately 9 million deaths annually [2].

By 2025, researchers estimate that 29.2% of the adult population will have hypertension—1.56 billion people—representing a 60% increase over 25 years. It therefore appears that hypertension is a major public health problem worldwide, and one that is here to stay [2].

Although the majority of cases are classified as essential, meaning no specific cause is identified (90% of cases), approximately 10% of hypertension cases are secondary to specific conditions, particularly endocrine disorders [3], which are the most common and account for more than half of the secondary causes of hypertension [4].

Among these endocrine causes is pheochromocytoma, a rare neuroendocrine tumor that develops from the chromaffin cells of the adrenal medulla and produces an excess of catecholamines [5]. With an annual incidence of approximately 3 to 8 new cases per million inhabitants, its prevalence among patients with hypertension is 0.1% - 0.6% [6]. Autopsy studies, however, report a higher incidence of 250 to 1,300 cases per million inhabitants, suggesting that the majority of cases go undiagnosed because their symptoms are variable and nonspecific [7]. Hypertension is a key sign but is not constant. It is particularly suggestive when associated with Ménard's clinical triad: headaches, sweating, and palpitations.

Pheochromocytoma is one of the leading causes of treatable hypertension, even though its diagnosis and management can be challenging due to the wide variability in clinical presentations, clinical forms, silent or atypical forms, and the need for specific hormonal and radiological evaluations [8]. It is therefore important to diagnose and treat this condition due to a significant increase in cardiovascular morbidity and mortality [9].

In 70% of cases, pheochromocytomas are sporadic, while the remaining 30% are associated with hereditary syndromes such as multiple endocrine neoplasia type 2 (MEN 2), Von Hippel-Lindau disease (VHL), neurofibromatosis type 1 (NF1), and familial paraganglioma syndrome [10]. This requires systematic genetic screening for both the patient and their family. Diagnosis is based on measurement of plasma or urinary catecholamines and localization imaging. Management is primarily surgical, preceded by meticulous medical preparation to prevent serious intraoperative hemodynamic complications. Long-term follow-up is recommended to detect any recurrence [11].

This study aims to improve the management of hypertension secondary to pheochromocytoma by exploring the clinical and diagnostic characteristics as well as the challenges posed by its management.

2. Clinical Case

1) We report the case of a 42-year-old male patient born on April 3, 1983.

He has no known family history of hypertension and has been living with hy-

pertension since November 2022, treated with 5 mg of bisoprolol and 10 mg of amlodipine daily; his blood pressure remains uncontrolled on this regimen.

He was referred to us for evaluation of an adrenal incidentaloma following a renal artery Doppler ultrasound performed as part of his hypertension workup. The evaluation revealed a 29-mm right adrenal nodule.

Clinical examination findings are as follows:

On questioning, episodes of palpitations without headache or sweating; no gastrointestinal disturbances; no signs of hypercortisolism (muscle weakness, skin disorders, decreased libido, sleep disturbances).

The patient weighed 81 kg and was 165 cm tall, with a BMI of 29.8 kg/m². Blood pressure was 158/89 mmHg, and heart rate was 57 beats per minute. Body surface area was 1.9 m².

2) Examination of the thyroid revealed no thyroid nodules.

The ENT examination revealed no cervical mass or signs of compression (dysphonia, dysphagia, dyspnea). No palpable supraclavicular mass.

On examination of the digestive system, no suspicious abdominal or pelvic mass or signs of compression (urinary problems, pelvic pain, constipation) were noted.

Cardiovascular findings: heart sounds were clearly audible, without additional murmurs, and synchronous with peripheral pulses; no periumbilical murmur was detected. Examination of the supra-aortic trunks for a vascular murmur—particularly if a paraganglioma were located at the carotid bifurcation—was unremarkable.

The skin and mucous membrane examination revealed no skin lesions or nodules, and no findings consistent with Cushing's syndrome.

3) Examination of the spine revealed no localized tender points.

Elsewhere, the pulmonary, ophthalmological, and neurological examinations were unremarkable. In biology, the following was noted in **Table 1**.

Table 1. Biological investigation of the case.

Exams	Results	Normal Ranges
Fasting Blood Glucose (mg/dL)	0.83	0.7 - 1
HbA1c (%)	4.9	4 - 6
Total Cholesterol (g/L)	1.57	1.58 - 2.01
HDL Cholesterol (g/L)	0.41	0.43 - 0.77
Triglycerides (g/L)	1.02	0.44 - 1.84
LDL Cholesterol (g/L)	0.95	0 - 1.58
Uric Acid (μmol/L)	361	200 - 420
Sodium Level (mmol/L)	142	136 - 146
Serum Potassium (mmol/L)	3.6	3.5 - 4.5

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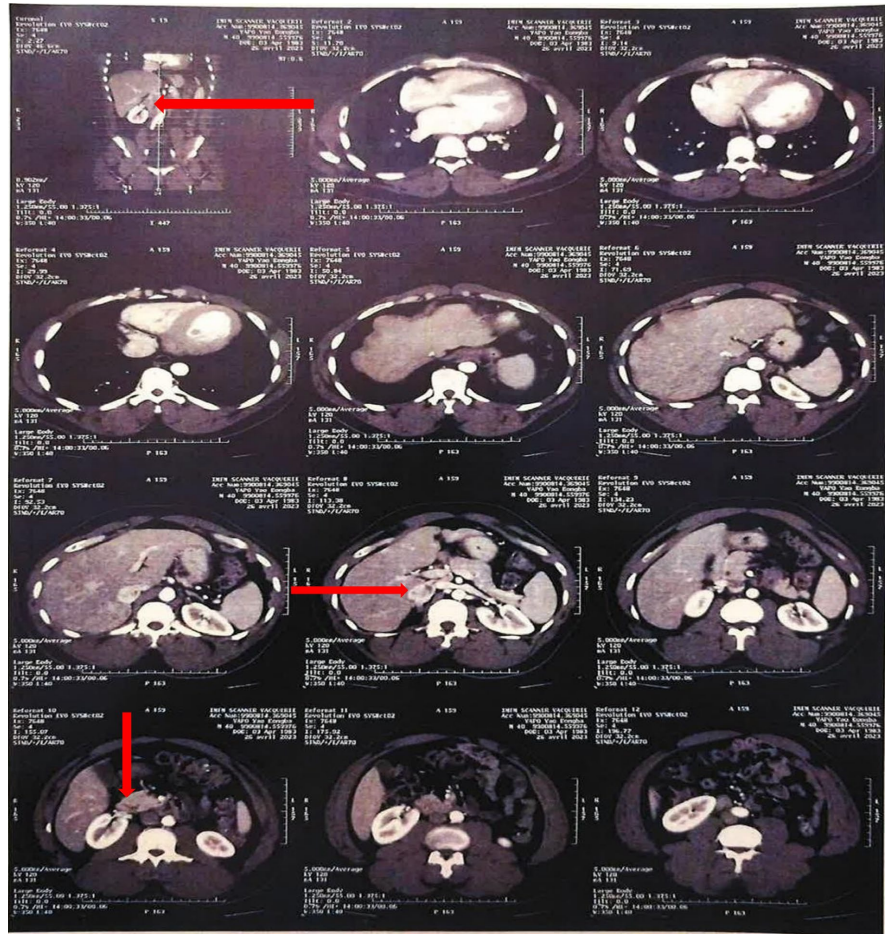
Chlorides (mmol/L)	106	95 - 106
Bicarbonate (mmol/L)	26.9	22 - 29
Proteins (g/L)	72.1	65 - 82
Serum Creatinine (μmol/L)	94	55 - 110
Creatine Kinase (U/L)	99	<190
ASAT (U/L)	30.1	10 - 45
ALAT (U/L)	28.5	7 - 50
CRP (mg/L)	1.7	<5.0
gGT (U/L)	51	<50
PAL (U/L)	56	35 - 120
Total Bilirubin (μmol/L)	7.4	<17
Markers		
Chromogranin A (μg/L)	253	<102
Serum Calcium (nmol/L)	2.29	2.2 - 2.6
Phosphorus (nmol/L)	1.07	0.8 - 1.45
Vitamin D (nmol/L)	32.9	87.5 - 138
PTH (pmol/L)	3.47	0.48 - 4.20
Urinary Ionogram		
Creatinine Excretion (mmol/24 h)	12.3	9 - 18
Urinary Sodium (mmol/24 h)	132	40 - 220
Urinary Potassium (mmol/24 h)	32	25 - 125
Urinary Chloride (mmol/24 h)	118	25 - 150
Numbering		
White Blood Cells: 10 ⁹ /L	4.67	4 - 11
White Blood Cells. Neutrophils: 10 ⁹ /L	2.45	1.8 - 6.8
Polymorphonuclear Cells. Eosinophils: 9 × 10 ⁹ /L	0.09	0.1 - 0.6
Poly. Basophils (10 ⁹ /L)	0.04	0 - 0.1
Lymphocytes	1.6	1.3 - 3.8
Monocytes	0.49	0.2 - 0.7
Hemoglobin (g/dL)	14.8	13 - 17
VGM	86.3	80 - 95
Brake Pads (10.3/L)	174	171 - 397
VS	36	1 - 15
Synacthen Test		
Plasma Cortisol at Time 0 (T0) (nmol/L)	221	> 510
Plasma Cortisol T60 (nmol/L)	964	

Continued

Pituitary Bases		
TSH (mIU/L)	1.46	0.4 - 4.0
Free T4 (pmol/L)	17.3	12 - 22
Estradiol (pmol/L)	182.3	21 - 184
17-OH Progesterone (nmol/L)	3.3	1.8 - 10.4
Delta-A-androstene-3,17-dione (nmol/L)	3.5	1.75 - 12.22
DHA Sulfate (μmol/L)	1.91	2.63 - 10.63
Low ACTH (pmol/L)	5.2	<13
Low Plasma Cortisol (nmol/L)	233	166 - 507
Compound S (nmol/L)	1.41	0.30 - 2.30
Midnight Salivary Cortisol		<4.3 nmol/L
Cortisol T1 (nmol/L)	2.4	
Cortisol T2 (nmol/L)	2.6	
Cortisol T3 (nmol/L)	11.1	
Plasma cortisol after dexamethasone suppression test: <10 μg/dL or <28 nmol/L (normal)		
Postural Test		
Aldosterone (Lying Down) (pmol/L)	293	32.5 - 655
Aldosterone at Bedtime (pmol/L)	402	61.3 - 979
Renin (Measured) (μIU/mL)	< 1	3 - 40
Renin-C (μIU/mL)	< 1	4 - 46
FLU		
Creatinine/24 h (mmol/24 h)	13.5	9 - 18
24-Hour Urine Output	1.8 L	
24-Hour Cortisoluria	158	<142
Urinary Metanephrine		
Normetanephrines (nmol/24 h)	2052	677 - 2642
Metanephrines (nmol/24 h)	378	218 - 1318
Normetanephrines/Creatinine (nmol/mmol)	152	33 - 228
Metanephrines/Creatinine (nmol/mmol)	28	17 - 91
Blood Metanephrines		
Normetanephrines (nmol/L)	1.05	0.17 - 0.79

The imaging findings were as follows:

a) CT scan of the urinary tract (**Figure 1**): A well-defined right adrenal mass measuring 33×26 mm; Spontaneous Density 55 HU, Washout 86%. No fatty component.



Source: Imaging Department, Scanner, Vacquerie, Paris (2023).

Figure 1. Contrast-enhanced abdominal CT scan with serial axial sections, showing a right adrenal mass consistent with a pheochromocytoma.

b) Abdominal MRI (**Figure 2**): A well-defined right adrenal mass measuring 33×26 mm, causing heterogeneous T2 hyperintensity and T1 hypointensity without a significant drop in signal on the T1-weighted in-phase sequence. It appears as diffusion hyperintensity with a decrease in the diffusion coefficient. After gadolinium injection, intense heterogeneous contrast enhancement is noted as early as the arterial phase, with visualization of central hypointense areas in the late phase. The left adrenal gland is thin.

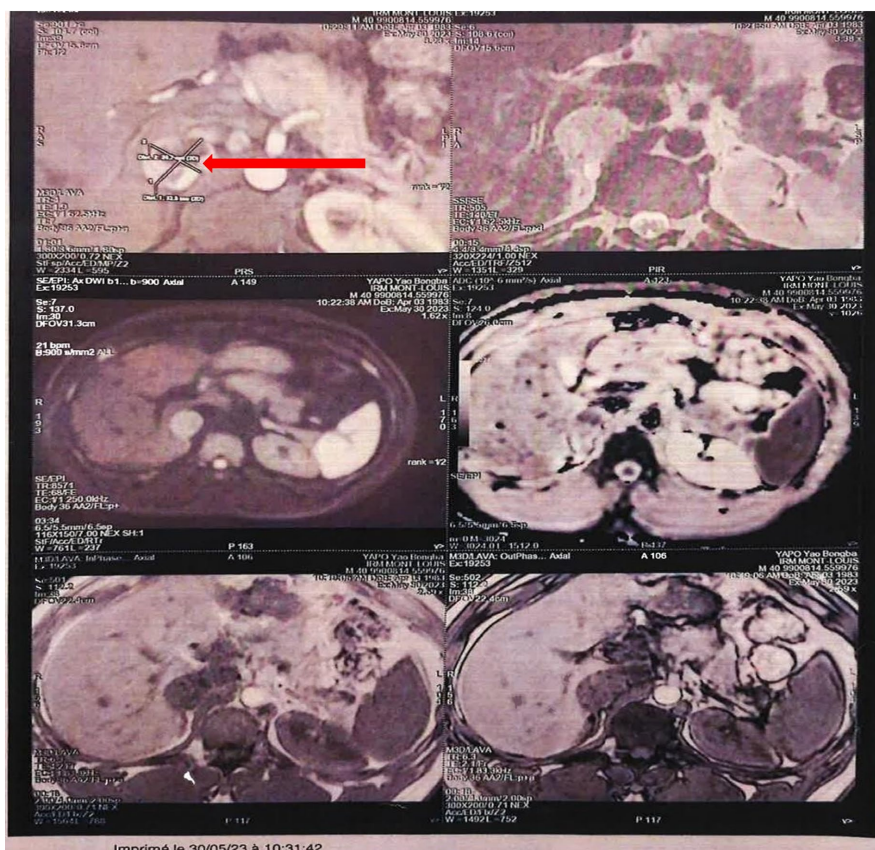
Overall, this was a case of suspected pheochromocytoma based on:

- a) A 33×26 mm right adrenal mass, atypical (Spontaneous Density 55 HU, Washout 86% on CT/no signal loss on MRI);
- b) A history of hypertension very recently treated and moderately controlled with dual therapy, accompanied by palpitations;

c) Elevated chromogranin A at 2.5 nmol/L; plasma normetanephrines modestly elevated at 1.05 nmol/L (normal < 0.79); normal urinary methoxylated metabolites (**Table 1**).

The patient was being treated with 5 mg of bisoprolol and 10 mg of amlodipine daily.

Since all pheochromocytomas are potentially syndromic, a workup to investigate other paragangliomas or a NEM was performed, including a DOPA-PET scan.



Source: Department of Medical Imaging, MRI, Mont-Louis, Paris (2023).

Figure 2. Abdominal MRI in axial and coronal sections showing a right adrenal mass consistent with a pheochromocytoma.

DOPA-PET Results:

a) Cerebral and cervical region: physiological distribution of the tracer in the basal ganglia. No suspicious foci in the cervical and supraclavicular lymph node regions. Small hyper-uptake focus in the left upper dental arch and a second focus in the left lower dental arch, likely corresponding to dental inflammatory foci.

b) Thoracic region: no suspicious pleuro-parenchymal pulmonary foci. No suspicious mediastinal lymph node foci.

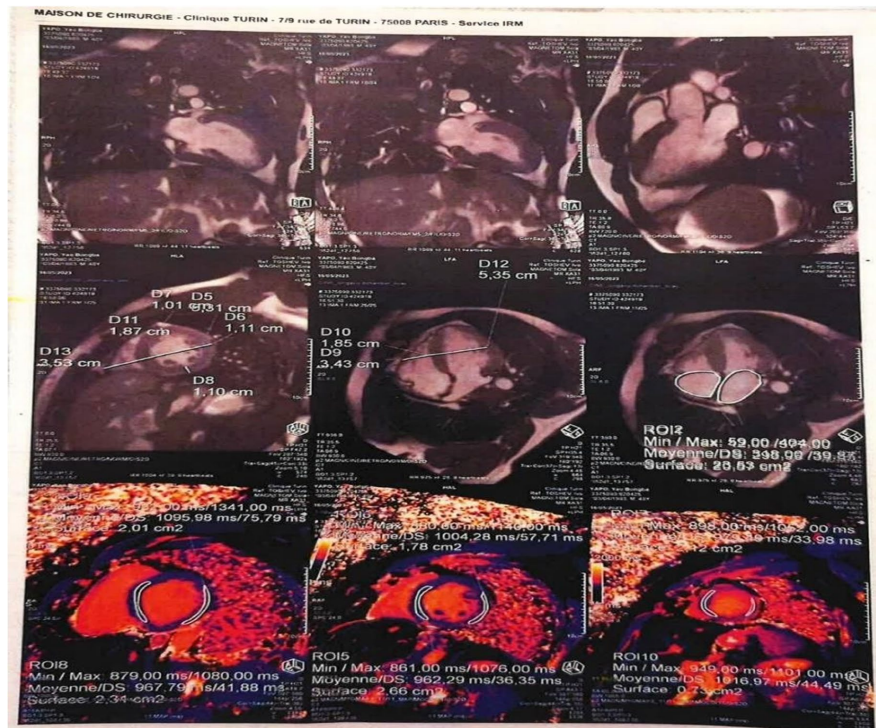
c) Abdominal-pelvic region: intense hypermetabolism of the 23-mm right adrenal mass: SUVmax 20.8. No suspicious foci in the liver or spleen. No suspicious foci in the lumbosacral and pelvic lymph node regions. Physiological distribution

of the tracer in the pancreas with no suspicious foci. Biliary and urinary excretion.

d) Bone structures: no suspicious foci in the bone structures.

Conclusion of the DOPA-PET scan: intense hypermetabolism of the 23-mm right adrenal mass, suggestive of pheochromocytoma. No other hyperfixating foci suggestive of secondary distant involvement.

Following these investigations, the diagnosis of isolated right pheochromocytoma was established. **Figure 3** shows the Cardiac MRI with multi-axial morphological sequences.



Source: Imaging Department, Turin Clinic, Paris (2023).

Figure 3. Cardiac MRI with multi-axial morphological sequences, consistent with type 2 hypertrophic cardiomyopathy (takotsubo cardiomyopathy).

The patient underwent right adrenalectomy via laparoscopy under general anesthesia in the left lateral decubitus position, following adequate medical preparation with an alpha-blocker.

Pathology of the resected specimen: a 4-cm hemorrhagic pheochromocytoma with loss of fumarate hydratase (FH). Complete resection.

PASS score = 2 (<4 indicates benignity).

GAPP score = 3 (0 - 2 = well-differentiated/3 - 6 = moderately differentiated/7 - 10 = poorly differentiated).

COPPS score = 0 (<3 indicates benignity).

The adjacent adrenal gland contains multiple corticosurrenal nodules corresponding to:

- Multiple small cortisol-producing adenomas

- One aldosterone-producing nodule
- A few aldosterone-producing micronodules

The postoperative course was marked by gastrointestinal symptoms consistent with adrenocortical insufficiency three weeks after surgery, requiring the reintroduction of hydrocortisone.

The course was favorable, with resolution of symptoms and normalization of blood pressure after discontinuation of antihypertensive medications.

Biologically, chromogranin A levels normalized, blood and urine methoxylated metabolites were negative, and the cortisol cycle was normal.

The abdominal CT scan 6 months postoperatively was normal, with no signs of local or distant recurrence.

Additionally, HbA1c was 4.8%, and LDL cholesterol was 1.10 g/L.

3. Clarification of the Diagnosis of Pheochromocytoma

The diagnosis of pheochromocytoma is straightforward when metanephrines are more than 3 or 4 times the upper limit of normal (ULN). However, where urinary and plasma metanephrines are only slightly or moderately elevated (between 1 and 3 times the ULN), the situation is described as a “grey area” or equivocal. The diagnostic reasoning then relies on a rigorous approach to rule out false positives and confirm genuine tumour autonomy.

Here are the key steps in this process:

We have rigorously ruled out false positives.

Before any imaging was carried out, we ensured that this slight elevation in plasma metanephrines was not due to physiological stress or pharmacological interference.

- **Strict sampling conditions were observed:** Plasma metanephrine levels were measured after 20 to 30 minutes of complete rest in the supine position and whilst fasting. Sampling whilst seated can double normetanephrine levels due to sympathetic activation.
- **Exclusion of drug-related interference:** Many treatments cause moderate elevations. Consideration should be given to discontinuing (if clinically safe) antidepressants (tricyclics, serotonin and noradrenaline reuptake inhibitors), alpha/beta-blockers, or decongestants.
- **Management of stress and stimulants:** Eliminate the consumption of coffee, tobacco, alcohol, and chocolate prior to the test.

Suspicious incidental adrenal mass: The contrast-enhanced CT scan of the abdomen (figure) revealed a well-defined right adrenal mass measuring 33 × 26 mm, with a spontaneous density of 55 HU and a wash-out of 86 per cent. No fatty component, consistent with a pheochromocytoma. Furthermore, abdominal MRI (figure) showed a right adrenal mass consistent with a pheochromocytoma. Overall, this was a suspected pheochromocytoma based on: a right adrenal mass measuring 33 × 26 mm, atypical (spontaneous density 55 UH, washout 86% on CT scan/no signal loss on MRI); a history of hypertension, very recently treated and

moderately controlled on dual therapy, with palpitations; elevated chromogranin A at 2.5 N; plasma normetanephrines modestly elevated at 1.05 nmol/L ($N < 0.79$); and normal urinary methoxylated derivatives (table).

Findings from the DOPA PET scan: intense hypermetabolism of a 23 mm mass in the right adrenal gland, suggestive of a pheochromocytoma. No other areas of increased uptake suggestive of secondary distant involvement.

Following these investigations, a diagnosis of isolated right-sided pheochromocytoma was made. Pathological examination of the surgical specimen confirmed a 4 cm haemorrhagic pheochromocytoma with loss of fumarate hydratase (FH). Complete resection.

PASS score = 2 (<4 indicates benignity).

GAPP score = 3 (0 - 2 = well-differentiated/3 - 6 = moderately differentiated/7 - 10 = poorly differentiated).

COPPS score = 0 (<3 indicates benignity).

In the decision-making process regarding surgery, it is generally the imaging findings that have been the main factor justifying adrenalectomy.

Abdominal ultrasound (**Figure 1**), abdominal MRI (**Figure 2**), and DOPA-PET provided visual and anatomical evidence of an adrenal mass consistent with a pheochromocytoma, and the histopathological examination of the surgical specimen following adrenalectomy confirmed the diagnosis of pheochromocytoma.

- **Repeated sampling and pre-analytical conditions:** These factors have influenced the quality and reliability of laboratory biological analyses, particularly the measurement of plasma metanephrines and chromogranin A. Poor pre-analytical conditions (e.g., a poorly stored tube) or repeat blood sampling are used to adjust or confirm a biological diagnosis, but they do not, on their own, dictate the need for surgery.
- **The effects of medication:** These are essential for perioperative safety (e.g., the administration of hydrocortisone to prevent adrenal insufficiency), but they represent factors for adjusting or managing risk rather than a justification for the surgery itself.

Confirmation of autonomous secretion of cortisol and aldosterone is based on demonstrating that a tumour or hyperplasia of the adrenal gland produces these hormones independently, bypassing the body's regulatory and feedback mechanisms.

4. Explanation of the Autonomous Secretion of Cortisol and Aldosterone

1) Establishing Cortisol Autonomy

In a healthy individual, the hypothalamus and the pituitary gland (via ACTH) stimulate cortisol production, which naturally declines at night. Autonomy is established when production persists despite the absence of a physiological signal or during an attempt at artificial blockade.

- **Midnight salivary cortisol:** This test assesses whether the circadian rhythm is

maintained or disrupted. Normally, cortisol levels reach their lowest point (nadir) at midnight. A high level at this precise time indicates a disruption of the circadian rhythm, an early sign that the adrenal glands are secreting cortisol continuously, independently of the central biological clock. In this patient, the salivary cortisol level remains consistently above 11.1 nmol/L, whereas the normal range is below 4.3 nmol/L.

- **24-hour cortisol excretion:** This test reflects the overall tissue exposure to free (active) cortisol over a full day. An elevated level (158 micrograms/24 h, compared with a normal level of less than 142 micrograms/24 h) confirms the presence of genuine and persistent hypercortisolism, ruling out stress-related fluctuations seen in a single blood test.
- **Dexamethasone suppression test (1 mg or rapid suppression):** A potent synthetic corticosteroid is administered at 11.00 pm to mimic excess cortisol and force the pituitary gland to cease its secretion of ACTH. The following morning, if the cortisol level does not drop sharply (absence of suppression), this confirms the independence of adrenal secretion (autonomous cortisol secretion), which ignores the “stop” signal from the central axis.

2) Determining Aldosterone Autonomy

Under normal circumstances, aldosterone is regulated by the renin-angiotensin system in response to blood pressure and blood volume. Autonomy (primary hyperaldosteronism) is confirmed when aldosterone production persists even though its natural stimulator (renin) has been completely suppressed.

- **Aldosterone/renin profile (or ARR ratio):** This profile indicates a disruption of the normal hormonal balance.
- Elevated or inappropriate aldosterone levels cause water and salt retention, which raises blood pressure.
- In response, the kidneys block renin production in an attempt to lower blood pressure. Renin levels are consequently severely reduced (suppressed).

A high ratio (high aldosterone/low renin) clearly demonstrates the autonomy of the adrenal glomerular zone: the adrenal gland continues to produce aldosterone indiscriminately, even though the kidney is signalling, through the absence of renin, that the body has no need for it.

The results of the laboratory tests following adrenalectomy are presented in **Table 2**.

Table 2. Postoperative biological tests at Day 7 and Day 34.

Exams	Postoperative Results		Normal Ranges
	07/08/2023 (J7 Post Op.)	09/04/2024 (J34 Post Op.)	
Fasting Blood Glucose (mg/dL)	0.85	0.9	0.7 - 1
HbA1c (%)	4.7	4.8	4 - 6
Total Cholesterol (g/L)	1.83	1.75	1.58 - 2.01

Continued

HDL Cholesterol (g/L)	0.46	0.44	0.43 - 0.77
Triglycerides (g/L)	1.32	0.99	0.44 - 1.84
LDL Cholesterol (g/L)	1.11	1.11	0 - 1.58
Uric Acid (μmol/L)		428	200 - 420
Uric Acid (μmol/L)	137	142	136 - 146
Serum Potassium (mmol/L)	3.6	4.5	3.5 - 4.5
Chlorides (mmol/L)	102	102	95 - 106
Bicarbonate (mmol/L)	23	27.5	22 - 29
Proteins (g/L)	77.4	77.7	65 - 82
Serum Creatinine (μmol/L)	89	102	55 - 110
Creatine Kinase (U/L)		183	<190
ASAT (U/L)	56.4	34	10 - 45
ALAT (U/L)	68.4	34.7	7 - 50
CRP (mg/L)	4.8	1.3	<5.0
gGT (U/L)	84	77	<50
PAL (U/L)	59	62	35 - 120
Total Bilirubin (μmol/L)	7.3	7	<17
Markers			
Chromogranin A (μg/L)	76.8	80.2	<102
Serum Calcium (nmol/L)	2.41	2.47	2.2 - 2.6
Phosphorus (nmol/L)	0.92	1.04	0.8 - 1.45
Vitamin D (nmol/L)	64.8	61.9	87.5 - 138
PTH (pmol/L)		2.45	0.48 - 4.20
Urinary Ionogram			
Creatinine Excretion (mmol/24 h)	13.9	17	9 - 18
Urinary Sodium (mmol/24 h)	140		40 - 220
Urinary Potassium (mmol/24 h)	47		25 - 125
Urinary Chloride (mmol/24 h)	130		25 - 150
Numbering			
White Blood Cells: $9 \times 10^9/L$	5.55	3.83	4 - 11
White Blood Cells. Neutrophils: $10^9/L$	2.82	1.76	1.8 - 6.8
Polymorphonuclear Cells. Eosinophils: $9 \times 10^9/L$	0.2	0.07	0.1 - 0.6
Poly. Basophils ($10^9/L$)	0.04	0.02	0 - 0.1
Lymphocytes	1.79	1.6	1.3 - 3.8
Monocytes	0.7	0.38	0.2 - 0.7
Hemoglobin (g/dL)	15.8	16.9	13 - 17
VGM	85.5	87.2	80 - 95

Continued

Brake Pads (10.9/L)	176	155	171 - 397
VS		6	1 - 15
Synacthen Test			
Plasma Cortisol at Time 0 (T0) (nmol/L)	371	172	>510
Plasma Cortisol T60 (nmol/L)	556	1106	
Pituitary Bases			
TSH (mIU/L)		1.33	0.4 - 4.0
Free T4 (pmol/L)		17	12 - 22
Estradiol (pmol/L)		173	21 - 184
17-OH Progesterone (nmol/L)		1.48	1.8 - 10.4
Delta-A-androstene-3.17-dione (nmol/L)		2.29	1.75 - 12.22
DHA Sulfate (μmol/L)		1.57	2.63 - 10.63
Low ACTH (pmol/L)		3.2	<13
Low Plasma Cortisol (nmol/L)	393	195	166 - 507
Compound S (nmol/L)		1	0.30 - 2.30
Postural Test			
Aldosterone (Lying Down) (pmol/L)		229	32.5 - 655
Aldosterone free (pmol/L)		319	61.3 - 979
Renin (Measured) (μIU/mL)		1.8	3 - 40
Renin-A (μIU/mL)		1.8	4 - 46
FLU			
Creatinine/24 h (mmol/24 h)	10.2		9 - 18
24-Hour Cortisoluria	52		<142
Urinary Metanephrine			
Normetanephrines (nmol/24 h)	678	1690.5	677 - 2642
Metanephrines (nmol/24 h)	184	374.9	218 - 1318
Normetanephrines/Creatinine (nmol/mmol)	66.6	99.10	33 - 228
Metanephrines/Creatinine (nmol/mmol)	18.07	22	17 - 91
Blood Metanephrines			
Metanephrines (nmol/L)	<0.1	0.126	0.068 - 0.28
Normetanephrines (nmol/L)	0.277	0.630	0.17 - 0.79
3-Methoxytyramine (nmol/L)	< 0.1	< 0.1	<0.1
Neurotransmitters in the Blood			

Continued

Norepinephrine		3.511	1 - 2.7
Adrenaline	< 0.1	< 0.1	0.2 - 0.5
Dopamine	0.172	< 0.1	

5. Reconciling the Diagnosis of Post-Operative Adrenal Insufficiency with the Presented Hormonal Data Shown

The diagnosis of secondary post-operative adrenal insufficiency is fully consistent with severely reduced levels of ACTH and cortisol, indicating suppression or damage to the corticotropic axis following surgery.

Consistency with hormonal findings:

- Ablated corticotropic axis: Baseline levels of ACTH and plasma cortisol at 8 am are abnormally low.
- Adrenal cortical atrophy: The lack of response to Synacthen confirms that the adrenal glands, deprived of their natural stimulant (ACTH) since the operation, have atrophied and can no longer produce cortisol.
- Timing of symptom onset.
- The symptoms appeared 30 days after the resection of the pheochromocytoma.
- They manifested as severe asthenia and orthostatic hypotension.

Treatment with hydrocortisone had not yet been initiated prior to the samples being taken.

Reason: Taking hydrocortisone immediately before the test would have distorted the results by artificially raising the plasma cortisol level measured by the laboratory.

Explanation of the Synacthen test results:

- Principle: Synacthen is a synthetic form of ACTH which should normally stimulate the immediate production of cortisol.
- Result supporting the diagnosis: A cortisol peak of less than 180 µg/L (or 500 nmol/L) following injection demonstrates the adrenal glands’ inability to respond.
- Confirmation of the mechanism: This supports a diagnosis of secondary adrenal insufficiency, where a chronic lack of endogenous ACTH has led to functional atrophy of the adrenal gland.

In our patient, following adrenalectomy, the cortisol level at 8 am was 371 nmol/L and 172 nmol/L; 34 days post-operatively, and following the Synacthen load test, the cortisol level remained low at 171 nmol/L, compared with a normal range above 510 nmol/.

6. The Chronological Timeline of the Case

In summary, this is a 42-year-old patient who has had hypertension since 2022 and is being treated with amlodipine and bisoprolol. During evaluation for hypertension, a pheochromocytoma was discovered, accompanied by type 2 hypertrophic

cardiomyopathy, in a clinical context of palpitations without headache or sweating, and without signs of hypercortisolism (**Table 3**).

Table 3. Case history.

14 November 2022	Palpitations, uncontrolled high blood pressure despite taking 5 mg of bisoprolol and 10 mg of amlodipine daily.
4 January 2023 Urological CT scan Imaging Department, CT Scanner, Vacquerie, Paris, 2023	Incidentaloma; Well-defined mass in the right adrenal gland measuring 33 × 26 mm; Spontaneous density 55 UH, wash-out 86 per cent. No fatty component.
6 February 2023 Abdominal CT scan; Imaging Department, CT Scanner, Vacquerie, Paris, 2023	Abdominal CT scan with contrast enhancement, showing serial axial sections of a mass on the right adrenal gland consistent with a pheochromocytoma.
20 February 2023 Abdominal MRI Medical Imaging Department, MRI, Mont-Louis, Paris, 2023	A well-defined right adrenal mass measuring 33 × 26 mm, showing heterogeneous T2 hyperintensity and T1 hypointensity without a significant drop in signal on the T1 in-phase sequence. It appears as hyperintensity on diffusion-weighted imaging with a reduction in the diffusion coefficient. Following gadolinium injection, intense, heterogeneous contrast enhancement is observed from the arterial phase onwards, with central hypointense areas visible in the late phase. The left adrenal gland is thin. Abdominal MRI in axial and coronal sections shows a right adrenal mass consistent with a pheochromocytoma.
25 February 2023 Biology Laboratory, Mont-Louis, Paris	Elevated chromogranin A at 2.5 N; plasma normetanephrines modestly elevated at 1.05 nmol/L (N < 0.79); normal urinary methoxylated derivatives.
10 March 2023 DOPA PET Medical Imaging Department, MRI, Mont-Louis, Paris 2023	Findings from the DOPA PET scan: marked hypermetabolism of a 23 mm mass in the right adrenal gland, suggestive of a pheochromocytoma. No other areas of increased uptake suggestive of secondary distant involvement.
16 March 2023 Cardiac MRI Source: Imaging Department, Turin Clinic, Paris, 2023	Following these investigations, a diagnosis of isolated right-sided pheochromocytoma was made. Cardiac MRI using multi-axial morphological sequences, consistent with type 2 hypertrophic cardiomyopathy (Takotsubo cardiomyopathy).
2 April 2023 Paris Hospital	Medical preparation using an alpha-blocker, followed by right adrenalectomy.
15 April 2023 Department of Pathology, Turin Clinic, Paris	Pathological examination of the surgical specimen: a 4 cm haemorrhagic pheochromocytoma, with loss of fumarate hydratase (FH). Complete resection. PASS score = 2 (<4 suggests benignity) GAPP score = 3 (0 - 2 = well-differentiated/3 - 6 = moderately differentiated/7 - 10 = poorly differentiated) COPPS score = 0 (<3 suggests benignity)

Since all pheochromocytomas are potentially syndromic, an evaluation was con-

ducted to investigate associations with hereditary syndromes, such as multiple endocrine neoplasia type 2, neurofibromatosis type 1, and Von Hippel-Lindau syndrome. The evaluation concluded that the patient had an isolated right-sided pheochromocytoma.

Treatment consisted of a right laparoscopic adrenalectomy. Pathological examination of the surgical specimen confirmed complete resection of a 4-cm, hemorrhagic, benign, moderately differentiated pheochromocytoma with loss of fumarate hydratase (FH) and multiple adrenocortical nodules.

7. Perioperative Management

Perioperative management of pheochromocytoma is crucial to prevent life-threatening intraoperative hypertensive crises and post-resection haemodynamic collapse. It is based on blood pressure control and correction of volume status.

7.1. Alpha-Blockers Are Mandatory as First-Line Treatment

The introduction of a beta-blocker before effective alpha-blockade is strictly contraindicated (risk of a hypertensive crisis due to unopposed alpha-adrenergic stimulation). Table 4 details the prazosin protocol.

Table 4. Prazosin protocol.

Active Ingredient	Prazosin
Dose	Initial dose: 0.5 - 1 mg Increase the dose every 12 hours if well tolerated
Usual Dosage	0.5 to 1 mg per day
Maximum Dosage	20 mg
Start	7 to 15 days before adrenalectomy
Route of Administration	Oral
Monitoring	Patient seated or lying down Monitor blood pressure whilst seated and standing at 15, 30, 60 and 120 minutes

Note: Combined with fluid and sodium replacement of 1 to 2 liters over 24 hours.

7.2. Pre-Operative Saline and Volume Expansion

Pheochromocytoma causes significant chronic vasoconstriction, leading to a reduction (contraction) in intravascular volume. Relieving this constriction with an alpha-blocker without first fluidising the patient puts the patient at risk of severe vasoplegic shock following tumour resection.

A saline and water infusion of 1 to 2 litres over 24 hours was required.

The postoperative course was marked by adrenocortical insufficiency three weeks after surgery. The patient’s condition improved with resolution of symptoms and normalization of blood pressure. Genetic follow-up following the detection of a loss of FH (fumarate hydratase) expression requires specialised and structured care.

8. Genetic Follow-Up and Germline Status

A loss of FH expression (detected by immunohistochemistry or molecular biology in a pheochromocytoma) strongly suggests hereditary leiomyomatosis and renal cell carcinoma (HLRCC).

1) **Constitutional (germline) testing:** It is essential to confirm whether the loss of FH is of hereditary origin. A germline genetic test (using blood or saliva) must be scheduled or is currently underway to screen for a pathogenic constitutional variant of the FH gene.

2) Interpretation:

a) If the germline test is positive, the diagnosis of HLRCC syndrome is confirmed.

b) If the germline test is negative, the loss of FH in the pheochromocytoma is considered an isolated somatic (non-hereditary) event.

3) Family screening strategy:

If a pathogenic germline variant is identified in a patient with pheochromocytoma, the family screening strategy is organised as follows:

4) **Cascade screening:** This process involves offering targeted genetic testing to first-degree relatives (parents, siblings, children), and then progressively to members of the extended family as soon as a new carrier is identified.

5) **Objective:** To identify at-risk family members so that they can be offered early and appropriate clinical monitoring (particularly for the risk of aggressive renal cell carcinoma and leiomyomas), and to reassure non-carriers, who can then return to the general population screening programme.

In our patient's case, the yield of the germline test is not yet known.

9. Discussion

Pheochromocytoma is a rare neuroendocrine tumor [12] with a prevalence of 0.1% - 0.6% among patients with hypertension [6]. It should be considered in cases of treatment-resistant hypertension or hypertension occurring in young individuals [7]. The evaluation for secondary hypertension is warranted in any case of newly diagnosed hypertension. The case of this 42-year-old patient, who has had uncontrolled hypertension for two years despite dual therapy and has no significant personal or family history, fits this scenario perfectly. The detection of an adrenal nodule on Doppler ultrasound of the renal arteries, in the context of hypertrophic cardiomyopathy confirmed by MRI, led to the investigation of a secondary endocrine cause of hypertension.

The symptoms of PPGL may be related to catecholamine hypersecretion and/or the tumor syndrome. They are variable, fluctuating, nonspecific, and sometimes even completely absent [13]. As a result, diagnostic delay is common, and the average time between the onset of the first symptoms and diagnosis is estimated to be more than 3 years [14]. The classic triad of symptoms, known as Ménard's triad, includes headaches, profuse sweating, and palpitations. However, these symptoms are not mandatory, and other clinical signs may be present: blood pres-

sure abnormalities (persistent or paroxysmal hypertension, orthostatic hypotension), chest pain, electrocardiographic abnormalities, visual disturbances, weight loss, heat intolerance, hyperglycemia, nausea, vomiting, and psychiatric disorders (anxiety) [15]. All of these symptoms may occur in isolation and are nonspecific, making clinical diagnosis difficult in some cases. Furthermore, the condition remains completely asymptomatic in approximately 10% of cases, although it is sometimes secretory [16]. This was the case with our 42-year-old patient, who had been hypertensive for two years with minimal symptoms consisting only of episodes of palpitations.

The diagnosis is based on the detection of hormonal hypersecretion through the measurement of plasma or urinary MN and NMN levels, as well as on conventional imaging and nuclear medicine studies [11]. In our case, plasma NMN levels were slightly elevated, as was chromogranin A (CGA), which was moderately elevated at approximately 2.5 times the normal level—a biomarker often used to support the diagnosis of neuroendocrine tumors in general [17]. Indeed, blood tests offer greater sensitivity, particularly when combined with chromogranin A testing [18]. The measurement of chromogranin A is increasingly requested and will help to account for false negatives in metanephrine testing [19]. CGA has a role in the long-term monitoring of pheochromocytomas, in combination with the measurement of plasma and/or urinary metanephrines, as a marker of recurrence or progression [16].

It should be noted that some pheochromocytomas may exhibit fluctuating or partially active secretion, making their detection more subtle. Following a laboratory diagnosis, imaging to determine the tumor's location is essential. In this regard, computed tomography (CT) or magnetic resonance imaging (MRI) are the first-line tests for locating the tumor [20]. In our case, this imaging confirmed the presence of an atypical right adrenal mass measuring 33×26 mm (Spontaneous Density 55 HU, Washout 86% on CT/no signal loss on MRI).

Approximately 40% of patients with pheochromocytoma have an associated hereditary syndrome. Therefore, once a diagnosis of pheochromocytoma has been established, it is important to rule out any signs of a hereditary syndrome such as Multiple Endocrine Neoplasia type 2, Neurofibromatosis type 1, and Von Hippel-Lindau syndrome. Our patient did not present any clinical manifestations associated with hereditary syndromes, and nuclear imaging, particularly DOPA-PET, which is essential for detecting multifocal involvement and/or secondary lesions that may be present at the time of diagnosis, also supports the diagnosis of an isolated pheochromocytoma.

The treatment of pheochromocytoma is strictly surgical. In our case, it consisted of right adrenalectomy via laparoscopy in accordance with recommendations. Laparoscopy is the preferred approach for small tumors (≤ 8 cm), as in this case, and following medical preparation with α -blockers, although this point was not detailed here [7].

Pathological examination of the surgical specimen, which remains the definitive test for confirming or ruling out pheochromocytoma [21], confirmed the complete

resection of a 4 cm pheochromocytoma, hemorrhagic, benign, poorly differentiated tumor with loss of fumarate hydratase (FH), suggesting a possible underlying genetic mutation and warranting further genetic evaluation and close monitoring with family screening even in the absence of a family history. Up to 40% of pheochromocytomas and paragangliomas are hereditary [22].

The postoperative course was favorable, with resolution of symptoms and normalization of blood pressure following discontinuation of antihypertensive medications. This rapid response is typical of properly resected pheochromocytomas. However, the onset of adrenocortical insufficiency three weeks after surgery, likely related to prolonged inhibition of the hypothalamic-pituitary-adrenal axis due to preoperative hypercortisolism, underscores the importance of rigorous hormonal monitoring following adrenalectomy. The initiation of hydrocortisone replacement therapy was essential, followed by a subsequent reassessment of corticotropic function.

The distinctive feature of our case lies in its association with primary hypercortisolism, as evidenced by elevated midnight salivary cortisol levels and 24-hour cortisoluria. Pathological examination of the surgical specimen revealed, in addition to the pheochromocytoma, multiple adrenocortical nodules secreting cortisol and aldosterone. This coexistence of a mixed cortical-medullary tumor, although rare, has been reported in the literature [23]. The presence of multiple nodules could also suggest spontaneous or genetic adrenal cortical hyperplasia, particularly in the context of Carney syndrome. This is not the case in our patient.

Finally, the type 2 hypertrophic cardiomyopathy observed on cardiac MRI could be explained by the chronic toxic effect of catecholamines on the myocardium, known as Tako-tsubo adrenergic cardiomyopathy. Although this condition is reversible after resection, it remains serious due to the complications it can cause in the acute phase (cardiogenic shock, heart failure, mitral regurgitation, left ventricular gradient, associated right ventricular dysfunction, formation of a left intraventricular thrombus with its thromboembolic complications, and the onset of arrhythmias) [24], justifying prolonged cardiological follow-up. Pheochromocytoma complicated by Takotsubo syndrome is a rare but increasingly diagnosed condition [25]. The overall prognosis is good, but endocrine monitoring is necessary in the medium and long term to prevent the risk of recurrence, contralateral involvement, or underlying genetic disease.

10. Conclusions

Although rare, pheochromocytoma is a curable cause of hypertension. The case presented here illustrates the importance of a thorough etiological evaluation in patients with hypertension, particularly in young individuals. The diagnosis should be systematically considered in this context, especially when there are suggestive symptoms or an incidental finding of an adrenal nodule.

The favorable outcome following adrenalectomy, marked by normalization of blood pressure and discontinuation of antihypertensive medications, confirms the

efficacy of surgical management.

The development of adrenocortical insufficiency in the postoperative period underscores the importance of regular endocrine follow-up after adrenalectomy. Finally, the loss of fumarate hydratase expression observed on pathological examination raises the possibility of an underlying genetic predisposition, which warrants systematic genetic testing in accordance with current recommendations.

This case highlights the multiple clinical, biological, and therapeutic dimensions of pheochromocytoma, a rare condition with significant diagnostic implications.

Ethical Considerations and Consent

Written informed consent was obtained from the patient for the publication of this case report and the associated images. In accordance with institutional guidelines, approval from a research ethics committee was not required for the presentation of this single clinical case.

Author Contributions

Pierre Koffi Dago, Assita Yao, Fagnan Levy Gouh, Serges Olivier Kouame, Emma Attiegoua, Anselme Yao N'Guessan, Jocelyne Danho, Jacko Abodo, Franck Kouassi, Adelaide Hué, Jocelyne Lecadou, Seydou Koné, and Mory Taoré: Conceptualization, Validation, Writing, Original Draft Preparation, Review, and Editing of the Manuscript; Pierre Koffi Dago, Assita Yao, Fagnan Levy Gouh, Serges Olivier Kouame, Emma Attiegoua, Anselme Yao N'Guessan, Jocelyne Danho, and Jacko Abodo: Validation; Pierre Koffi Dago, Assita Yao, Fagnan Levy Gouh, Serges Olivier Kouame, Emma Attiegoua: Formal Analysis and Data Curation; Pierre Koffi Dago, Assita Yao, and Fagnan Levy Gouh: Funding Acquisition.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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