Adrenal hemorrhage: A single center experience and literature review

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Abstract

Background. Adrenal hemorrhage (AH) is a rare condition that can lead to acute adrenal insufficiency and may be fatal. The risk factors of AH include focal adrenal lesion, abdominal trauma and anticoagulation therapy. The clinical manifestation of AH varies widely; the symptoms may be related to adrenal insufficiency or may reflect multiple organ failure. However, in many cases, the course of AH is asymptomatic.

Objectives. The study is a retrospective analysis of 23 cases of AH, whose aim is to discuss the etiology and the management of selected patients, as well as a literature review.

Material and methods. The paper presents a retrospective analysis of 23 patients with AH confirmed by radiological and/or pathological examination. Epidemiological data, the results of laboratory tests, and radiological and pathological examinations were included in the analysis.

Results. The risk factors of AH were not established in 13 patients, 5 patients had experienced a trauma prior to AH diagnosis, 1 patient was diagnosed with sepsis, 2 patients had concomitant neoplastic disease, and in 2 patients, 2 risk factors were present. Among patients who required emergency admission, 5 patients were hospitalized due to acute abdominal pain, 1 patient due to sepsis and 1 patient due to symptoms of active endocrinopathy. In the remaining patients, diagnostic procedures were prompted by the detection of adrenal incidentaloma (AI). A total of 40% of patients underwent surgical treatment due to the magnitude of AH or clinical and laboratory evidence of overt endocrinopathy. In the remaining patients, conservative treatment and further observation was recommended. In 34.8% of these patients, follow-up examinations revealed a gradual regression.

Conclusions. It seems that there is a need to distinguish patients with AH who do not require surgical intervention. Follow-up radiological examination is necessary to reassess the lesion. The patients in whom shrinkage of the tumor can be observed are likely not to require surgical treatment.

Key words: adrenal glands, hemorrhage, pseudocyst, primary adrenal insufficiency, adrenal incidentaloma

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Introduction

Adrenal hemorrhage (AH) is a rare condition that can lead to acute adrenal insufficiency and may be fatal. It is potentially life-threatening when the adrenal glands are involved bilaterally, although at least 90% of each adrenal cortex must be compromised before this is clinically evident. The incidence of spontaneous AH based on data from autopsy studies is 0.14–1.1%. The risk factors of AH include focal adrenal lesion, abdominal trauma, anticoagulation therapy, congenital or acquired bleeding disorders, sepsis, and pregnancy.[1,2] Bleeding of an adrenal gland tumor is most frequently observed in pheochromocytoma and adrenal metastases.[1,3–6] Larger lesions of adrenal myelolipoma (>5 cm) rarely present with acute retroperitoneal AH.[6] The clinical manifestation of AH varies widely; the symptoms may be related to adrenal insufficiency or may reflect multiple organ failure. However, in many cases, the course of AH is asymptomatic.[7]

Until recently, AH diagnosis was often made at postmortem examination.[8] Currently, due to the increased availability of modern imaging techniques, AH is more frequently diagnosed intravitally and in many cases lesions in the adrenal glands are detected unexpectedly (adrenal incidentaloma – AI). The features of AH on radiological imaging are specific; therefore, its diagnosis based on imaging studies is relatively simple.[8,9]

Data on AH in the literature are scarce and management standards have not been precisely established. It seems that there is a need to redefine AH risk factors and to establish guidelines for the management of high-risk patients, particularly considering that some AH cases may be associated with metastases or pheochromocytoma.

This paper summarizes data on 23 cases of AH, and discusses the etiology and the management of selected patients.

Material and methods

This paper presents a retrospective analysis of 23 patients with AH confirmed by radiological and/or pathological examination. The study group included patients treated at the Department of Endocrinology and Internal Diseases and the Outpatient Clinic of the University Clinical Center in Gdańsk from 2002 to 2016. Epidemiological data, the results of laboratory tests, and radiological and pathological examinations were included in the analysis.

Results

The study group included 23 patients; 60.8% women and 39.2% men. The mean age was 60.6 years (Table 1).

The risk factors of AH were not established in 13 patients (56.6%), 5 patients (21.8%) had experienced a trauma prior to AH diagnosis, 1 patient (4.3%) was diagnosed with sepsis, 2 patients (8.7%) had concomitant neoplastic disease, and in 2 patients (8.7%), 2 risk factors were present: anticoagulant drugs and lung cancer, and trauma and chronic oral anticoagulation.

Among patients who required emergency admission, 5 patients (21.7%) were hospitalized due to acute abdominal pain, 1 patient (4.3%) due to sepsis and 1 patient (4.3%) due to symptoms of active endocrinopathy. In the remaining 16 patients (69.7%), diagnostic procedures were prompted by AI. The symptoms of adrenal insufficiency (in the course of sepsis) were confirmed in 1 patient, and 1 patient had treatment-resistant hypertension. The rest of the patients were asymptomatic.

The results of biochemical tests and hormone assays in serum and urine – e.g., cortisol, adrenocorticotropic hormone (ACTH), androstenedione, dehydroepiandrosterone sulfate, and metoxycatecholamine (MT) – and radiological images of the study participants were analyzed. In 13 patients (56.2%), laboratory test results were within the range of normal values. The remaining 10 patients had laboratory abnormalities, including biochemical markers of adrenal insufficiency (4.3%), hypercortisolemia (8.7%), elevated urinary MTs (mainly normetanephrine, 21.8%), and disorders of both the corticotropic axis and MT secretion (8.7%). In all patients, computed tomography (CT) examination was performed. A lesion in the right adrenal gland was found in 12 patients (52.2%), a lesion in the left adrenal gland was found in 10 patients (43.5%), and 1 patient had bilateral lesions. The diameter of the AH ranged from 17 to 150 mm (mean diameter: 60.6 mm) (Table 2).

Nine patients underwent surgical treatment (40%) due to the magnitude of AH or to clinical and laboratory evidence of overt endocrinopathy. The patient in whom both an elevated MT level and hypercortisolemia were found was disqualified from surgery due to the burden of concomitant diseases. It should be stressed that the results of radiological examinations did not raise any oncological concerns. Moreover, pathological assessment unequivocally indicated AH; only in 1 specimen was AH with the presence of neoplastic cells (a metastasis from lung cancer) found. In the remaining group of patients (n = 14; 60%) conservative treatment and further observation was recommended. In this group, specific risk factors were identified (sepsis, trauma and anticoagulation) and no clinical or laboratory evidence of endocrinopathy was observed, while the adrenal lesions were explicitly described in CT reports as AH. Follow-up examinations performed in 8 patients (34.8%) revealed a gradual regression of AH, and in the patient with bilateral lesions, a complete absorption of hematomas was found. Table 2 summarizes the clinical course of AH in the study population.

Discussion

The pathophysiological mechanism of AH remains unclear. The adrenal glands have unique vasculature providing abundant blood supply, which significantly increases

Table 1. Study group characteristics

No.	Age	Gender	Risk factor	Adrenal gland	Size on CT [mm]	Laboratory tests	Intervention	Additional information
1	38	female	trauma	right	30 × 17	normal	observation	none
2	70	male	anticoagulant drugs, lung cancer	right	55 × 40	elevated normetanephrine	observation	decreased size of mass to 25 mm at 18 months
3	60	female	urosepsis	bilateral	52 × 32	adrenal insufficiency	observation	resolution of mass at 12 months
4	87	male	unknown	right	115 × 70	normal	observation	decreased size of mass to 60 mm at 3 months
5	67	female	unknown	right	32 × 28	hypercortisolemia, elevated normetanephrine	observation	decreased size of mass to 25 mm at 12 months
6	55	male	unknown	right	150 × 10	normal	adrenalectomy	histologically hematoma
7	73	female	trauma	right	85 × 70	normal	adrenalectomy	histologically hematoma
8	65	male	trauma	left	76 × 65	hypercortisolemia, elevated normetanephrine	observation	decreased size of mass to 75 mm at 2 months
9	60	male	unknown	left	60 × 60	normal	adrenalectomy	histologically hematoma
10	51	female	unknown	right	64 × 48	normal	adrenalectomy	histologically hematoma
11	58	female	unknown	left	120 × 40	hypercortisolemia, elevated normetanephrine	adrenalectomy	histologically hematoma
12	62	female	unknown	right	62 × 52	normal	adrenalectomy	histologically hematoma
13	60	male	unknown	left	97 × 78	normal	adrenalectomy	histologically hematoma
14	67	female	unknown	left	35 × 25	normal	adrenalectomy	histologically hematoma
15	53	female	unknown	left	43 × 31	hypercortisolemia	adrenalectomy	histologically hematoma
16	61	female	anticoagulant drugs, trauma	left	60 × 60	elevated normetanephrine	observation	decreased size of mass to 57 mm at 10 months
17	58	female	unknown	left	21 × 13	elevated normetanephrine	observation	decreased size of mass to 18 mm at 20 months
18	64	male	unknown	right	1 × 1	elevated normetanephrine	observation	none
19	45	male	trauma	right	18 × 9	normal	observation	none
20	63	female	unknown	right	21 × 15	normal	observation	stable size of mass
21	60	female	lung cancer	left	60 × 23	normal	observation	none
22	60	male	colon cancer	right	35 × 25	normal	observation	none
23	57	female	trauma	left	48 × 33	normal	observation	none

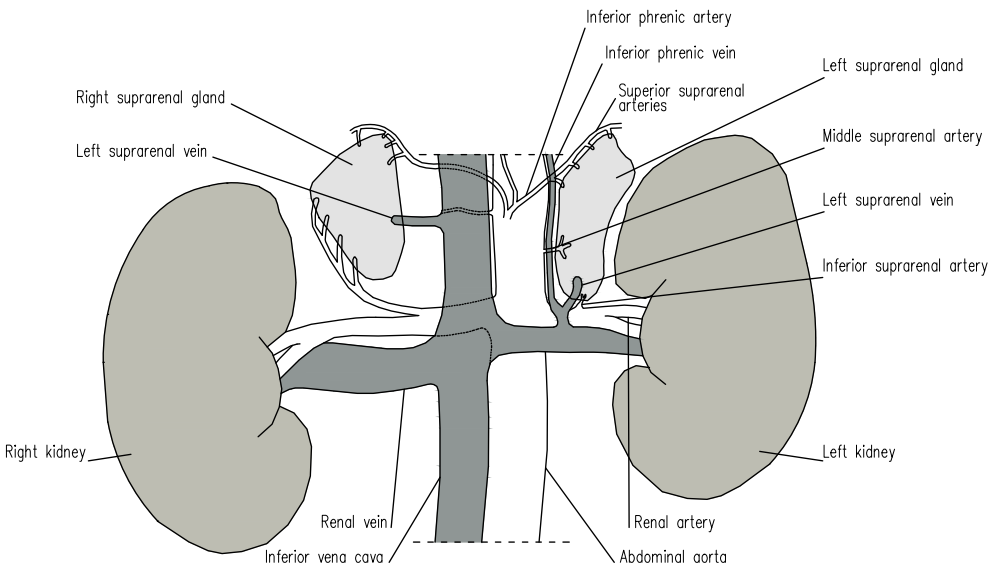

Fig. 1. Anatomical structure and blood supply of the adrenal glands

Table 2. Study group characteristics

Feature	Description	No.	%
Age (mean)	60.6 years	–	–
Gender	women	14	60.8
	men	9	39.2
Etiology	trauma	5	21.8
	sepsis	1	4.3
	anticoagulants + neoplasm	1	4.3
	anticoagulants + trauma	1	4.3
	neoplasm	2	8.7
	unknown	13	56.6
Adrenal gland involved	right adrenal gland	12	52.2
	left adrenal gland	10	43.5
	bilateral	1	4.3
Clinical presentation	acute abdominal pain	5	21.7
	asymptomatic (incidentaloma)	16	69.7
	infection	1	4.3
	evidence of active endocrinopathy	1	4.3
Laboratory parameters	normal	13	56.5
	hypercortisolemia	2	8.7
	elevated metoxycatecholamines	5	21.8
	hypercortisolemia + elevated metoxycatecholamines	2	8.7
	adrenocortical insufficiency	1	4.3
Intervention	observation	14	60
	surgical treatment	9	40

Table 3. Risk factors of adrenal hemorrhage

Conditions predisposing to AH	Examples
Trauma (80% of cases)	–
Stress	• burns • hypotension • surgery (particularly orthopedic surgery)
Infectious disease	• sepsis caused by *Neisseria meningitidis*, *Pseudomonas aeruginosa*, *Escherichia coli*, *Bacteroides fragilis*, *Streptococcus pneumoniae*
Medication	• anticoagulants • antiplatelets • nonsteroidal anti-inflammatory drugs • synthetic ACTH • glucocorticosteroids
Hematologic disorders	• antiphospholipid syndrome • systemic lupus erythematosus • heparin-induced thrombocytopenia • other thrombocytopathies • thrombocytosis
Obstetric causes	• pregnancy • postpartum period • pre-eclampsia
Perinatal injury	• asphyxia • perinatal hypoxia • sepsis • fetal hematologic disorders
Adrenal gland tumor	• primary: pheochromocytoma, adrenocortical cancer, myelolipoma, lipoma, hematoma, angioma, adenoma, pseudocyst • metastatic: lung cancer, renal cancer, breast cancer, colon cancer, thyroid cancer, gallbladder cancer, melanoma
Gastrointestinal diseases	• acute pancreatitis

AH – adrenal hemorrhage; ACTH – adrenocorticotropic hormone.

the propensity to bleeding. Each adrenal gland is supplied by 3 suprarenal arteries (the superior suprarenal artery, a branch of the inferior phrenic artery; the middle suprarenal artery, a direct branch of the abdominal aorta; and the inferior suprarenal artery, a branch of the renal artery).[3,8] It should be noted that venous outflow is provided by only 1 suprarenal vein tributary to the inferior vena cava.[10] Furthermore, it has been suggested that increased capillary resistance may be a significant factor predisposing an individual to AH. Elevated ACTH and MT levels, through their vasoconstrictive effect and excessive platelet activation, may lead to reperfusion and subsequent bleeding, mainly from distal capillary vessels (Fig. 1).[6,11]

Adrenal hemorrhages can be divided into primary (spontaneous or idiopathic) and secondary AHs (of known etiology, e.g., caused by trauma, sepsis or anticoagulation therapy).[1] The risk factors of AH are summarized in Table 3.

Based on a 25-year observation, Vella et al. divided a group of 141 patients with AH into 7 categories:

1. AH detected as AI – 28 cases;
2. spontaneous AH manifesting as acute hemorrhage to the abdominal cavity – 16 cases;
3. AH with concomitant hematological disease (antiphospholipid syndrome, systemic lupus erythematosus) – 20 cases;
4. postoperative AH (patients who underwent laparoscopic procedures or laparotomy, alloplastic joint replacement and prostatectomy) – 14 cases;
5. AH in patients treated with anticoagulants – 3 cases;
6. post-traumatic AH – 4 cases;
7. AH as a complication of sepsis and/or stress – 56 cases.[8]

The cases analyzed in our study were mainly AIs with non-established etiology (70% of patients), which were categorized as group 1 according to Vella's classification. The remaining patients were classified as group 2 or 7.

The clinical presentation reflects both the intensity of bleeding and the extent of adrenal gland injury.[1,9] The clinical course in bilateral massive hemorrhage to the adrenal glands is usually dramatic and, if recognized too late, may be fatal.[12] The patients in whom AH has been incidentally diagnosed usually do not develop a typical form of AH and the course of the disease may be asymptomatic.[8,13–16] In the analyzed group, 91.4% of cases were asymptomatic; only in 1 patient was clinically overt adrenal insufficiency observed.

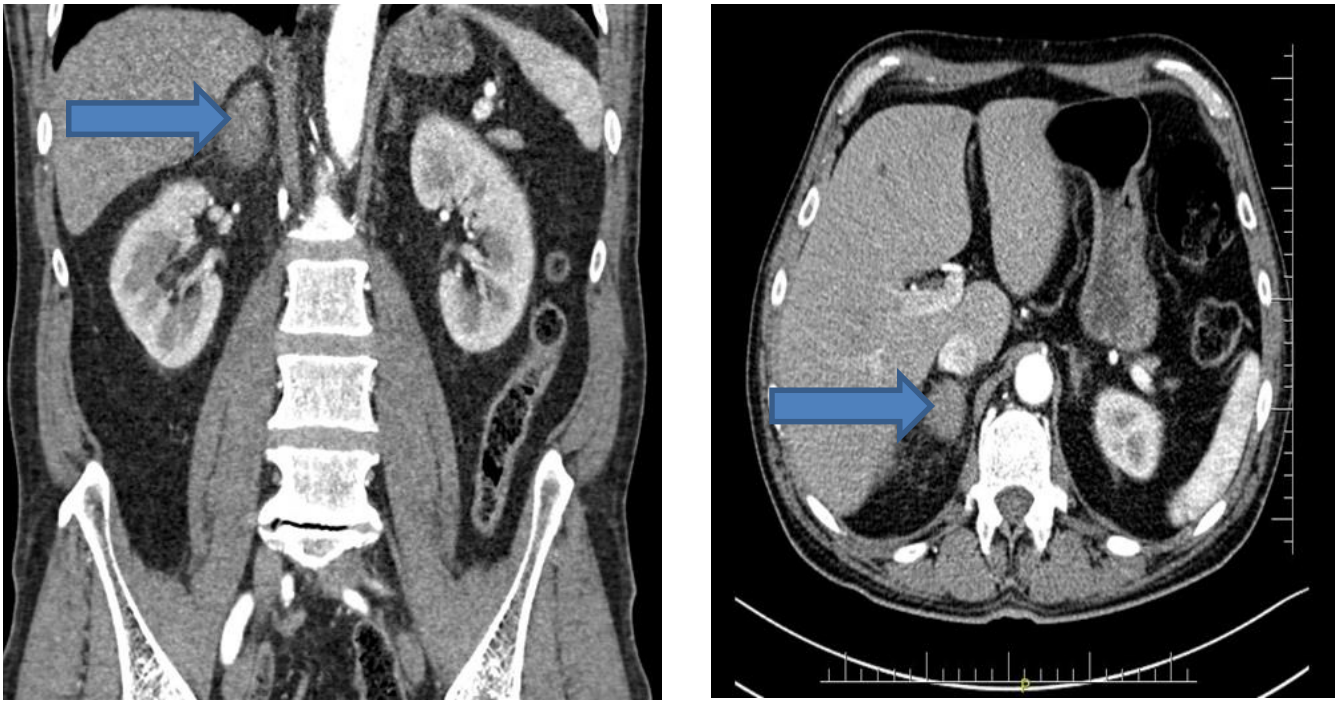

Fig. 2. Patient No. 22: right adrenal hematoma, 35 mm in diameter (arterial phase CT scan)

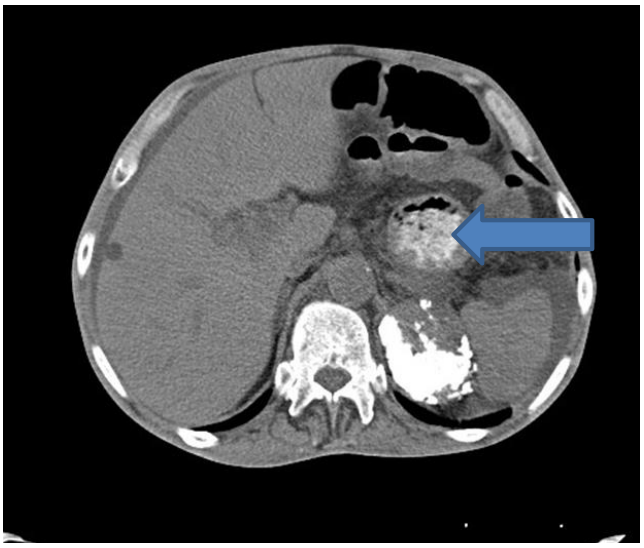

Fig. 3. Patient No. 8: left adrenal hematoma, 70 mm in diameter, with calcifications (arterial phase CT scan)

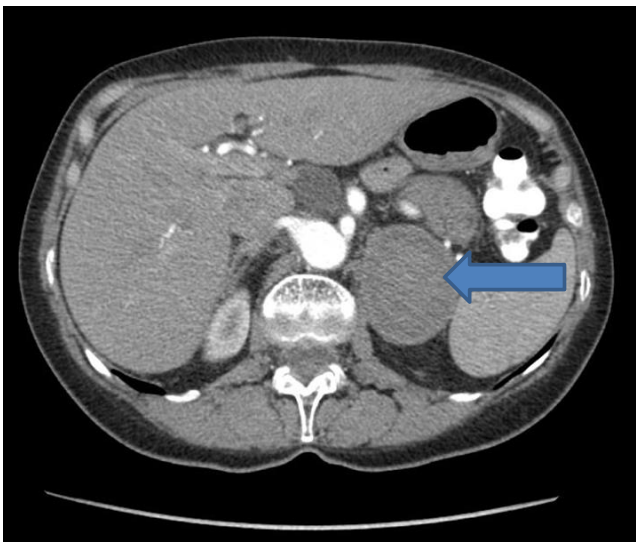

Fig. 4. Patient No. 16: left adrenal hematoma, 60 mm in diameter (arterial phase CT scan)

Abnormalities of laboratory parameters in patients with AH, such as hyponatremia, hyperkaliemia, hypercalcemia, and hypoglycemia, are biochemical markers of adrenal insufficiency. Anemia is observed in patients with massive bleeding. Thrombocytopenia and bleeding disorders occur in patients with antiphospholipid syndrome or lupus erythematosus.[8,17] Considering the risk of AH related to pheochromocytoma and adrenocortical cancer, in patients with these pathologies, the urine MT level measurement and the corticotropic axis assessment should be done.[1,18,19] In the study group, symptomatic adrenocortical insufficiency (in the patient with bilateral AH in the course of sepsis), increased secretion of MT and corticotropic axis disorders were found. Isolated hypersecretion of MT was observed in 5 patients, which could indicate an active neoplastic process (pheochromocytoma is the lesion where AH occurs most frequently);[4,5] however, MT levels only slightly exceeded normal laboratory range (50–100% above the upper limit of normal) and the patients did not present symptoms indicative of an excess of catecholamines. For this reason, the above abnormality was considered non-specific and could not be the basis for diagnosing

pheochromocytoma. Follow-up examination confirmed the spontaneous normalization of MT levels. It should be noted that 56.5% of patients had normal laboratory values. Similarly, in a group of 6 patients with AH described by Marti et al., 4 patients had normal laboratory test results.[1]

Undoubtedly, the increased availability of imaging examinations resulted in higher rates of AH diagnosis. The radiological image of AH depends on the patient's age and the duration and intensity of bleeding.[3,20] Despite the fact that ultrasonographic (USG) examination is a fast, inexpensive and widely available technique, its use in the diagnosis and monitoring of AH patients is limited to newborns and infants.[3] It has been reported that contrast-enhanced ultrasonography (CEUS) can also be used for the diagnosis of adrenal gland tumors, which might enhance the role of this technique in the detection of AH.[21,22] The method of choice for critically ill patients is CT scanning, because it allows preliminary differentiation of adrenal hemorrhagic tumors from malignant lesions (10% of pheochromocytomas, adrenocortical carcinoma and metastases).[23,24] On CT images, AH appears as a focal heterogeneous high-density (50–70 Hounsfield units) lesion (Fig. 2).[1,25,26] With the aging of the hematoma, its gradual shrinkage and even complete regression can be observed. Chronic AH can be seen as an adrenal mass with a hypodense center without calcification, known as an adrenal pseudocyst, and after a year, calcification is often found (Fig. 3). The lack of contrast enhancement allows the hemorrhagic nature of the lesion to be confirmed.[3] Moreover, Tan et al. presented 4 cases of non-traumatic AH which demonstrated features of prior adrenal congestion (adrenal gland thickening and periadrenal fat stranding) on CT scans (Fig. 4).[27] In some cases, magnetic resonance imaging (MRI) can be employed, which is particularly useful for differentiating acute bleeding from chronic bleeding.[22,25,26] The appearance of hemorrhage on MRI scans depends on the age of hemorrhage, with the signal intensity changing in relation to the progressive degradation of hemoglobin. In the acute phase (<2 days), hemorrhage is hypointense on T1-weighted images and hypointense on a T2 signal (Fig. 5). In the early subacute phase (2–7 days), AH is seen as a hyperintense T1 signal and a hypointense T2 signal. A chronic hematoma demonstrates peripheral low T1 and T2 signals due to intracellular hemosiderin, with central T2 hyperintensity and T1 isointensity.[28]

The management of patients with AH depends on their general health status. Hemodynamically unstable patients require intensive medical treatment for shock and adrenal insufficiency as well as qualification for surgical treatment. Surgery should be also considered in patients with the suspicion of pheochromocytoma and adrenocortical carcinoma, particularly if the size of the tumor in the adrenal gland exceeds 6 cm.[1,19,29] Conservative treatment should be considered in patients with bilateral AH, in patients whose AH was detected as AI, and in those with known

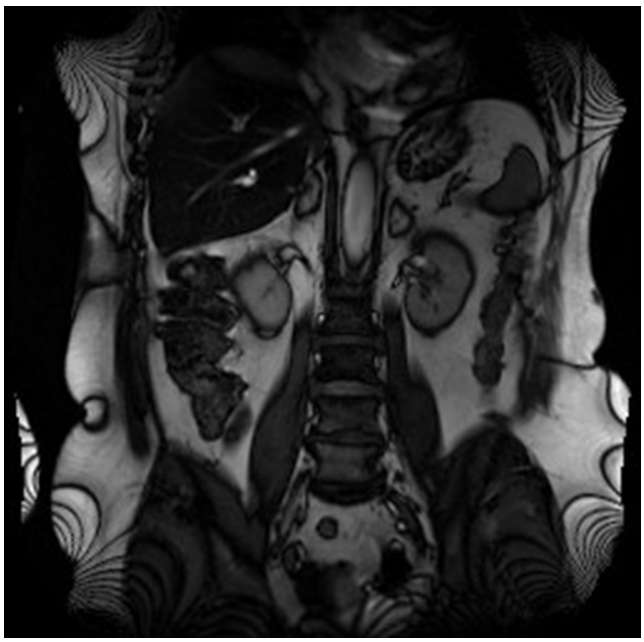

Fig. 5. Patient No. 3: bilateral adrenal hemorrhage; the lesions are 50 mm in diameter (MRI)

and reversible risk factors (e.g., AH caused by an overdose of anticoagulants).[5,30] Marti et al. described 6 cases of AH. In the group, 4 patients underwent surgical treatment, whereas the rest of the patients were re-examined.[1] One patient receiving conservative treatment was on anticoagulation therapy and, burdened with lung cancer metastasizing to the adrenal gland and adrenal hematoma, was qualified for chemotherapy. During the 6-month follow-up examination, a marked regression of the adrenal lesion was found. In another patient who had a history of septic shock complicated with neutropenia and colon perforation, and whose CT scan showed a hematoma with a pseudocyst, a complete regression of the adrenal lesion was observed at the 6-month follow-up visit. Bharucha et al. described bilateral AH with subclinical course in a patient on warfarin therapy in whom conservative treatment proved to be successful.[9] There are reports on cases of spontaneous bilateral AH manifesting with acute abdominal pain, in which the introduction of hydrocortisone replacement therapy and the withholding of surgical treatment resulted in a significant regression of the lesion confirmed by follow-up imaging.[11,24]

Among the patients included in the analysis, 9 (40%) underwent surgical treatment, and the decision was made based on the tumor size and the suspicion of active endocrinopathy. The remaining 14 patients (60%) were only examined again at follow-up. The factors that determined the choice of conservative treatment were known AH risk factors, an absence of clinical and laboratory markers of endocrinopathy, and a description of the adrenal lesion in the CT report, which unequivocally indicated AH. In 34.8% of these patients, follow-up imaging examinations showed a partial or total regression of the lesion.

Therefore, it seems that there is a need to distinguish patients with AH who do not require surgical treatment, because the natural course of AH may result in spontaneous hematoma resorption and recovery. Follow-up radiological examination is necessary in order to reassess the lesion. The patients in whom shrinkage of the tumor can be observed are likely not to require surgical treatment.

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