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
**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 intravitaly 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
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). It should be noted that venous outflow is provided by only 1 suprarenal vein tributary to the inferior vena cava. Furthermore, it has been suggested that increased capillary resistance may be a significant factor predisposing to AH. Elevated ACTH and MT levels, as a result of 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.

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. The clinical course in bilateral massive hemorrhage to the adrenal glands is usually dramatic and, if recognized too late, may be fatal. 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. In the analyzed group, 91.4% of cases were asymptomatic; only in 1 patient was clinically overt adrenal insufficiency observed.

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### 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 metoxy catecholamines  | 5   | 21.8 |
|                             | hypercortisolemia + elevated metoxy catecholamines | 2 | 8.7 |
| 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 thrombocytopenies                                                 |
|                               | • thrombocytosis                                                          |
| Obstetric causes              | • pregnancy                                                               |
|                               | • postpartum period                                                       |
|                               | • pre-eclampsima                                                          |
| 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.
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

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)
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 de-
dcribed by Marti et al., 4 patients had normal laboratory
test results.1

Undoubtedly, the increased availability of imaging exa-
ninations resulted in higher rates of AH diagnosis. The ra-
diological 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, inexpen-
sive 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 ul-
trasonography (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 pheochromocytos-
as, 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 com-
plete regression can be observed. Chronic AH can be seen
as an adrenal mass with a hypodense center without calci-
fication, 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 periadre-
nal 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),
m 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 dem-
onstrates peripheral low T1 and T2 signals due to intracel-
larular 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 sus-
picion of pheochromocytoma and adrenocortical carci-
noma, 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
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 antico-
agulation therapy in whom conservative treatment proved
to be successful.9 There are reports on cases of spontane-
ous 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 fol-
low-up imaging.11,24

Among the patients included in the analysis, 9 (40%) underw ent surgical treatment, and the decision was made
based on the tumor size and the suspicion of active endo-
crinopathy. 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
docrinopathy, and a description of the adrenal le-
sion in the CT report, which unequivocally indicated AH.
In 34.8% of these patients, follow-up imaging exa-
ninations showed a partial or total regression of the lesion.

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