Adrenal disease: a clinical update and overview of imaging. A review

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Aim. The aim of this article is to provide an overview of the most frequent clinically significant adrenal diseases and to describe the latest trends in their diagnoses, particularly by means of imaging techniques.

Methods. The authors reviewed standard textbooks and subsequently conducted a search using the PubMed (Public/Publisher MEDLINE) electronic database by the year 2013 with the following search terms: adrenal masses, adrenal adenoma, phaeochromocytoma, adrenocortical carcinoma, metastases, incidentalomas, hypercortisolism, hyperaldosteronism.

Results. If adrenal disease is clinically suspected, hormone tests are performed to detect adrenal hyperfunction and imaging studies are used to assess the nature of adrenal lesion. The most frequent syndromes include hypercortisolism, primary hyperaldosteronism, and phaeochromocytoma. The clinically most significant pathologies of the adrenal glands are adenomas and adrenal hyperplasia, adrenocortical carcinomas, phaeochromocytomas, and metastases. Given the availability and improved quality of imaging techniques, adrenal incidentalomas are detected increasingly often. In these cases, it is necessary to rule out hormonal activity and malignancy. Incidentalomas can be associated with clinical syndromes of adrenal hormone overproduction. In most cases, they are clinically silent. In some cases, the definitive diagnosis can be determined as early as during the initial examination with an imaging technique (most frequently, a CT scan). If the finding is inconsistent, other imaging techniques can be used: CT contrast washout analysis, MRI, SPECT or PET/CT.

Conclusion. In the case of adrenal gland disorders, correct interpretation of the results of laboratory tests and imaging studies is essential for further management of these patients.

Key words: adrenal masses, adrenal adenoma, phaeochromocytoma, adrenocortical carcinoma, metastases, incidentalomas, computed tomography, PET/CT

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INTRODUCTION

The management of adrenal diseases has recently undergone major development as a result of rapid advances in laboratory and, in particular, imaging techniques. The available therapeutic options are also improving. If adrenal disease is suspected clinically, the diagnostic algorithm initially involves hormone tests to detect adrenal hyperfunction. These are then followed by imaging studies addressing the morphological presentation of adrenal pathology, including evaluation of adjacent structures (Fig. 1).

Given the ever improving availability of imaging techniques, the issue of adrenal incidentalomas is increasingly relevant. In contrast to cases presenting with clinical symptoms, the incidental finding on imaging studies is the first detection of pathology, only then followed by a hormone test and a thorough clinical examination focused on the adrenal glands. Incidentalomas can be associated with some of the most common clinical syndromes of adrenal hormone overproduction (hypercortisolism, hyperaldosteronism, or phaeochromocytoma); however, the majority of them are clinically silent.

Fig. 1. Normal appearance of the adrenal glands. Normal CT appearance of the adrenal glands in the coronal plane (yellow arrow).
The most frequent pathologies of the adrenal glands are adenomas and adrenal hyperplasia, most often detected incidentally. Other pathological findings of the adrenal glands include carcinomas, phaeochromocytomas, and metastases. It is essential to distinguish adrenal adenomas from these entities since they usually require a different therapeutic approach.

The aim of this article is to provide an overview of the most frequent clinically significant adrenal diseases and to describe the role of imaging techniques in diagnosing these conditions.

ADENOMA

Adenoma is the most frequent adrenal tumour; it is of a benign nature. In the majority of cases, it is found incidentally on imaging and exhibits no hormonal activity. When hormonally active, it may produce cortisol or aldosterone. Adenomas can be accompanied by the following clinical syndromes: hypercortisolism and hyperaldosteronism.

Hypercortisolism (Cushing’s syndrome)

Cortisol overproduction due to a primary tumour of the adrenal cortex results in the peripheral type of Cushing’s syndrome. It is mostly caused by adrenal adenoma, less often by carcinoma. Other types of Cushing’s syndrome include the central type (overproduction of ACTH in the pituitary gland) and ectopic ACTH secretion (in some malignant tumours, e.g. small-cell lung carcinoma).

Clinical presentation is nonspecific. Central-type obesity, moon face, and hirsutism can be seen. Purple striae are characteristic. Impaired gonadal function, arterial hypertension, and osteoporosis also occur.

Laboratory screening for Cushing’s syndrome utilizes tests for increased 24-h urinary excretion of cortisol and the absence of the normal circadian rhythm of plasma cortisol on the cortisol curve. Dexamethasone suppression tests and adrenocorticotropic hormone (ACTH) tests are also performed. These tests are used not just to confirm the diagnosis of Cushing’s syndrome, they also help classify it by its type.

Hyperaldosteronism

Primary hyperaldosteronism is a condition caused by oversecretion of aldosterone in the adrenal cortex. It is among the most frequent causes of secondary hypertension. It is generally estimated to be present in 5-10% of patients with arterial hypertension, with some sources reporting as much as 14.4% of patients. In specialized centres in selected groups of patients with moderate and severe hypertension, the proportion of patients with primary hyperaldosteronism is higher: 5-20%; according to some authors even as much as 37% (ref.3,4).

In primary hyperaldosteronism, aldosterone oversecretion is independent of the renin-angiotensin system. Conversely, increased aldosterone secretion in secondary hyperaldosteronism is a result of increased renin production (such as in diuretic treatment, renal artery stenosis, congestive heart disease, liver cirrhosis, and others). In primary hyperaldosteronism, the following two forms are predominant: one caused by aldosterone-producing adenomas of the adrenal cortex (Conn’s syndrome) and another called bilateral adrenal hyperplasia. The distinction between these two most common causes of primary hyperaldosteronism is absolutely essential for correct therapeutic approach. Adrenal adenoma is indicated for surgical treatment, i.e. adrenalectomy. In contrast, bilateral hyperplasia is treated medically. Laboratory distinction between the two types of primary hyperaldosteronism is not possible and, unfortunately, even the results of imaging studies (adrenal CT or MRI) are often equivocal5,6.

The gold standard for distinguishing these two types of primary hyperaldosteronism is adrenal vein catheterization with sequential blood sampling to determine aldosterone and cortisol levels. This determines whether hormone overproduction is unilateral or bilateral.

The clinical presentation of primary hyperaldosteronism is nonspecific. Symptoms caused by hypokalaemia may occur: fatigue, paraesthesia, and cardiac rhythm disorders. Moderate to severe arterial hypertension is almost invariably present. Laboratory findings typically include hypokalaemia, elevated potassium excretion, and metabolic alkalosis.

Laboratory tests to measure plasma renin and aldosterone levels are essential. The ratio of plasma aldosterone concentration to plasma renin activity is the most sensitive screening test. Dynamic suppression tests assessing inhibition of aldosterone secretion may also be used to confirm the diagnosis.

However, the individual types of primary hyperaldosteronism cannot be distinguished based solely on laboratory methods. Each patient with primary hyperaldosteronism should undergo imaging studies (CT or MRI) in order to rule out adrenocortical carcinoma. However, the finding of imaging studies can still be misleading5,6. As a result, most authors tend to recommend that adrenal vein catheterization be performed prior to contemplated adrenalectomy in all patients with primary hyperaldosteronism regardless of the results of imaging studies5,6,10.

Finding on imaging studies

An adenoma is typically of a smaller size. It is usually detected on imaging studies completely incidentally. It is mostly of a homogeneous structure (Fig. 2 a,b).

In general, adenomas can be divided into two groups: lipid-rich adenomas and lipid-poor adenomas. This fact determines the appearance of adenomas on CT and MRI scans6,11.

The former group accounts for more than 70% of adenomas. Owing to a high intracytoplasmic lipid content, it presents with a density lower than 10 Hounsfield units (HU) on an unenhanced CT scan. Unfortunately, approximately 30% of adenomas contain too little lipids and consequently have a higher density on unenhanced CT scans6,11,12.

Adenomas with high content of intracellular lipids can also be detected using chemical shift MRI. Chemical shift
The origin of the right suprarenal vein is mistaken for a tumor. According to Matsuura, the origin of the right suprarenal vein is most frequently apparent ventrolaterally, and this is where it can be mistaken for a small adenoma expanding from the adrenal contour (authors' own observation).

On a PET/CT scan, most commonly using 18F-fluorodeoxyglucose (18F FDG PET/CT), the vast majority of adenomas do not accumulate the radiopharmaceutical. According to meta-analyses, 18F FDG PET/CT scanning generally has a high sensitivity (97%) and specificity (91%) in distinguishing between a malignant and benign adrenal lesion.

Unilateral or bilateral adrenal hyperplasia may also have a similar appearance to adrenal adenoma(s) on imaging studies. A definitive distinction of adenoma from hyperplasia is not possible by means of imaging techniques. The finding may not be unequivocal even on histopathological examination since the hyperplastic nodule can be encapsulated. The evaluation must be comprehensive and includes the presence/absence of a continuous capsule, signs of expansive growth with compression atrophy of the surrounding tissue, and the number of foci, with a solitary nodule being rather indicative of adenoma and multiple ones of hyperplasia. Clinical decision making is significantly aided by finding of unilateral hormone overproduction.

**MYELOLIPOMA**

Myelolipomas are benign adrenal tumours. They consist of two parts: fatty and haematopoietic tissues. They are hormonally silent and clinically asymptomatic, with only large myelolipomas manifesting by pain or by retroperitoneal haemorrhage. Myelolipomas grow very slowly. They require no treatment; only larger symptomatic myelolipomas may require surgical removal.

**Finding on imaging studies**

Owing to their lipid content, myelolipomas typically have low density on CT scans, lower than 0 HU, or often less than -50 HU. Because of the presence of haematopoietic tissue, their density is slightly higher than that of the surrounding retroperitoneal fat. Their MRI appearance corresponds to that on CT scans. Due to its typical appearance, the diagnosis of myelolipoma can be definitive based only on CT or MRI finding. It does not accumulate the radiopharmaceutical on 18F FDG PET/CT.

**PHAEOCROMOCYTOMA**

Phaeochromocytomas are tumours of chromaffin cells of the sympathoadrenal system with sustained or paroxysmal catecholamine hypersecretion causing arterial hypertension. They most commonly arise in the adrenal medulla, but can also occur extra-adrenally (these tumours are referred to as paragangliomas). The majority of phaeochromocytomas are benign.
The prevalence of phaeochromocytomas is estimated to range from 1:4,500 to 1:1,700 with an annual incidence of 3-8 cases per 1 million population. Seventy to seventy-five percent of phaeochromocytoma cases arise in the adrenal gland and 25-30% occur extra-adrenally. Phaeochromocytomas account for 0.05%-0.1% of cases of sustained hypertension. Approximately 30-35% of phaeochromocytomas and paragangliomas are hereditary.

These can be a part of hereditary multiple endocrine neoplasia type 2 (MEN 2) syndrome, occur in neurofibromatosis type 1 (NF-1) and type 2 von Hippel–Lindau (VHL 2) syndrome, or be associated with hereditary paragangliomatosis with succinate dehydrogenase (SDH) gene mutations. In SDHB gene mutations, recurrent, aggressive, and metastatic paragangliomas develop.

Distinguishing the biological nature of phaeochromocytoma by means of imaging techniques alone is impossible in most cases. If distant metastases are present, a malignant form can be expected. The distinction between benign and malignant phaeochromocytomas is very difficult even for a histopathologist. It is necessary that the evaluation be done by a pathologist experienced in assessment of adrenal tissue. When evaluating the specimen, it is not sufficient to use only the principles of general oncology. Histological evaluation must be comprehensive and include immunohistochemical examination of proliferation activity. Only then the possibility of recurrence or metastases can be addressed with a certain degree of likelihood. However, conclusive histopathological signs of malignant phaeochromocytoma generally remain controversial. Long-term surveillance of patients following surgery for phaeochromocytoma is therefore warranted. Inadequate surveillance is still the most common error in postoperative follow-up care of phaeochromocytoma patients. When no long-term regular check-ups of clinical status, biochemical markers and imaging techniques are carried out, patients with unrecognized malignant phaeochromocytoma may present after several years with already generalized disease.

The clinical signs of phaeochromocytoma tend to be varied. The presentation of a phaeochromocytoma typically includes sustained or paroxysmal hypertension, often resistant to standard treatment. Headaches, sweating, chest pain, shortness of breath, cold and wet skin, pallor, tremor, anxiety, nervousness, and dyspeptic complaints can all occur during a paroxysm. In untreated phaeochromocytomas, neuroretinopathy, cardiomyopathy, and stroke may develop. Determination of free plasma normetanephrine and metanephrine is the gold standard in diagnosing catecholamine hypersecretion. When available, levels of free methoxytyramine can be used. If the values of these markers are not convincing, the clonidine test can be used to confirm the diagnosis. This test is also capable of identifying patients with falsely elevated catecholamines. Caffeine, an amine-rich diet, and some drugs, particularly tricyclic antidepressants are significant sources of false-positive results of catecholamines and metanephrine.

Once the diagnosis of phaeochromocytoma has been established, proper preparation is vital prior to its surgi-
Fig. 5. Phaeochromocytoma. 5a. CT appearance of left adrenal phaeochromocytoma in the coronal plane (yellow arrow). 5b. MIBG/SPECT appearance of extra-adrenal phaeochromocytoma in the coronal, axial, and sagittal planes. 5c. Photograph of a dissected specimen of adrenal phaeochromocytoma. 5d. Benign phaeochromocytoma. Detail of tumour cells of compact tumour nests (yellow arrow) delimited by a dense sinusoidal capillary network (blue arrow). The cell nuclei are uniform (however, nuclear polymorphism in benign phaeochromocytoma is frequent and is not a marker of malignancy. The evaluation of malignancy is comprehensive). (HE staining, 100x). 5e. Malignant phaeochromocytoma. The tumour is composed of irregular solid nests (blue arrow) of cells with nuclear polymorphism; tumour necroses (yellow arrow) and vascular invasions were present in the central part of the tumour. Ki-67 proliferative activity reached 20%. (HE staining, 40x).

Finding on imaging studies

Like carcinomas, phaeochromocytomas tend to be larger in size. They are soft-tissue expansions that often contain necrotic parts. Following the administration of a contrast medium, there is often very intensive heterogeneous enhancement on CT or MRI with appearance of necrotic areas (Fig. 5a). An unequivocal distinction from a carcinoma or a metastasis based only on imaging techniques is not possible. Likewise, it is not possible to clearly distinguish benign from malignant variants of phaeochromocytoma. Only in patients in whom distant metastases are detected, a malignant phaeochromocytoma-
Fig. 6. Adrenal carcinoma. 6a. CT appearance of left adrenal carcinoma in the coronal plane (yellow arrow). 6b. Adrenocortical carcinoma. The tumour is composed of solid trabecular and solid alveolar parts. Nuclear polymorphism may or may not be detected and does not represent a conclusive sign of malignancy. Large necroses as well as capsular and vascular invasions were found to be present in the tumour. Ki-67 proliferative activity reached 30%. (HE staining, 40x). 6c. Adrenocortical carcinoma. Detail of tumour cells with eosinophilic cytoplasm and nuclear polymorphism (yellow arrow). (HE staining, 200x).

Fig. 7. Adrenal metastasis. 7a. PET/CT appearance of metastases in both adrenal glands in the coronal plane (blue arrow). 7b. Photograph of a dissected specimen of metastasis in the adrenal gland. 7c. Metastasis of adenocarcinoma (yellow arrow). (HE staining, 100x).
The administration of contrast medium has been refuted. A possibility of inducing hypertensive crisis following the intravenous administration of a contrast medium has been refuted. A biopsy, on the other hand, significantly increases the risk of catecholamine release into circulation. Therefore, a biopsy is contraindicated when the primary tumour does not accumulate increased radiopharmaceutical accumulation is seen in malignant phaeochromocytoma associated with von Hippel–Lindau syndrome. In these cases, it is possible to use PET with 18F-fluorodihydroxyphenylalanine with higher sensitivity compared to CT. Scintigraphy with radioactive iodine-labelled metaiodobenzylguanidine can also be utilized for detection. 123I-MIBG may fail to detect adrenal phaeochromocytoma associated with von Hippel–Lindau syndrome. In these cases, it is possible to use PET with 18F-fluorodihydroxyphenylalanine with higher sensitivity than scintigraphy with MIBG. Routine use, however, is still hindered by low availability and high costs. Unfortunately, some phaeochromocytomas have lost their membrane noradrenergic transport system and fail to uptake these markers.

**ADRENOCORTICAL CARCINOMA**

Adrenocortical carcinoma (ACC) is a less frequent, but clinically very significant adrenal tumour with a high malignancy potential. The incidence of adrenocortical carcinoma is reported to be 1 to 2 cases per 1 million population; the prevalence is estimated to be 4-12 cases per 1 million population. It occurs slightly more in women (1.5:1). In 50-60% of patients, adrenocortical carcinomas manifest by adrenal hyperfunction. Most commonly, it is Cushing’s syndrome which occurs in 30-45% of hormonally active ACCs. Unlike benign adrenocortical tumours, carcinomas may produce several groups of steroids. Histopathological diagnosis is not simple and it is advisable that it be done by a pathologist experienced in this field. When evaluating the malignant nature of an adrenocortical tumour, both clinical manifestations and macroscopic as well as microscopic features of the tumour need to be taken into consideration. Histological signs of malignancy include diffuse pattern of growth, vascular and capsular invasion, tumour necroses, increased mitotic activity, cellular polymorphism, and high proliferation activity detected by immunohistochemical analysis. In order to classify the tumour as malignant, the presence of several of the above-mentioned signs is usually required. For example, nuclear polymorphism can be quite minimal and vascular invasion may not be apparent in a given sample (Fig. 6b). Despite complete resection in stage I-III tumours, approximately 40% of patients develop metastasis within two years.

**Finding on imaging studies**

Adrenocortical carcinomas have a significantly larger size in comparison with adenomas. They frequently have a strikingly heterogeneous structure on imaging studies. They can invade the surrounding structures and propagate to the adrenal vein, renal vein, or even the inferior vena cava. Substantial progression in size is characteristic. With the use of 18F FDG PET/CT, increased radiopharmaceutical accumulation is seen in adrenal carcinomas.

**ADRENAL METASTASES**

The adrenal gland is a very well vascularized organ; therefore, it is a frequent site of metastases. On autopsy, metastases into the adrenal glands are found in up to 27% of patients with malignant epithelial tumours. Metastases intensively accumulates the radiopharmaceutical on 18FDG PET/CT scans. Scintigraphy with radioactive iodine-labelled metaiodobenzylguanidine can also be utilized for detection. However, it is still hindered by low availability and high costs. Unfortunately, some phaeochromocytomas have lost their membrane noradrenergic transport system and fail to uptake these markers.

**Finding on imaging studies**

Progression of size is typical for metastases into the adrenal glands. With a larger size, heterogeneous structures are present, which becomes evident particularly following intravenous administration of a contrast medium. Metastases intensively accumulates the radiopharmaceutical on PET/CT scans with 18F-fluorodeoxyglucose. A significant exception are metastases of malignancies in which the primary tumour does not accumulate this radiopharmaceutical.

**GANGLIONEUROMA**

Ganglioneuroma is a benign tumour composed of ganglion cells, occurring predominantly at a younger age. A CT scan typically shows a spherical solid tumour with sharp demarcation. It can contain calcifications.
NON-HODGKIN LYMPHOMA (NHL)

Adrenal lymphoma is one of the less frequent findings. The appearance of adrenal lymphoma on imaging studies is nonspecific. The diagnosis of lymphoma is relevant for the management since the mainstay of treatment is systemic chemotherapy; surgery is not indicated.

CYSTS

Cysts are a rare finding in adrenal glands. When found, they are often detected incidentally (Fig. 8a). They can be endothelial or, less frequently, epithelial or parasitic. Pseudocysts occurring as a result of bleeding are more frequent (Fig. 8b) (ref.53). If the cyst wall exhibits nodular enhancement on imaging studies, the differential diagnosis must take into consideration a cystic adrenal tumour, in particular phaeochromocytoma and carcinoma53.

BLEEDING/HAEMATOMA

Bleeding into the adrenal glands most commonly occurs in the neonatal period and is rarer in adulthood. It can sometimes be encountered in severe abdominal injury, particularly in polytrauma in which it more frequently occurs on the right side (Fig. 9a) (ref.44). Furthermore, it can be a complication of adrenal venous sampling55. If the involvement is bilateral, it is most commonly associated with coagulation disorders (Fig. 9b) (ref.22). An incidentally found old adrenal haematoma that can be partially or completely calcified is more typically encountered in adulthood.

TUBERCULOSIS (TB)

In adults, adrenal involvement occurs as a result of postprimary TB. In the elderly population, adrenal calcifications due to a previous disease may be encountered that can be apparent on a plain radiograph.

NEUROBLASTOMA

Neuroblastoma is a malignant invasive tumor from the adrenal medulla. It occurs typically in early childhood. Diagnosis is usually done by ultrasound. CT shows large mass exceeding midline. A heterogeneous structure is predominant, with calcifications, necroses and bleeding. The growth of this tumor can occlude large vessels of the retroperitoneum and dislocate surrounding structures. It can spread to the spinal canal or to the inferior vena cava.

ADRENAL INCIDENTALOMAS

With the ever-increasing availability of imaging techniques, incidental detection of expansions in the adrenal glands becomes increasingly frequent56. Adrenal incidentalomas are masses in the adrenal gland incidentally detected by imaging techniques in patients in whom no pathological finding in the adrenal gland was clinically suspected. Incidentalomas are found on abdominal CT scans in approximately 5-10% of patients (Fig. 10) (ref. 57).

If an adrenal incidentaloma is found, it is important to rule out malignancy and secretory activity. Even at the time of initial detection using an imaging technique (most frequently, a CT scan) the finding should be evaluated by a radiologist regarding its nature. The classic division of incidentalomas into benign and malignant based only on their size (smaller ones - more likely to be benign, larger ones - more likely to be malignant) (ref.28) is merely
of an indicative value. The primary reason is an overlap in the size of the individual groups. Another reason is related to the increasing general availability of imaging techniques. Given the high examination rates with imaging techniques in the population, incidentalomas tend to be detected much earlier, in comparison with the two previous decades, and thus tend to be of a smaller size. Accordingly, malignant lesions and phaeochromocytomas can also be of a smaller size due to their earlier detection. According to some authors, an adenoma is of a stationary size on imaging studies. However, if observed on CT for several years, many adenomas exhibit clear, even though minimal progression in size.

The value of unenhanced density is absolutely crucial in distinguishing adenomas from other adrenal diseases. A low unenhanced density occurring in 70% of adenomas is caused by a high cytoplasmic lipid content. It rarely occurs in metastases, adrenocortical carcinomas, and phaeochromocytomas. Based on the value of unenhanced density, incidentalomas can be divided into two groups: adenomas and non-adenomas. The unenhanced density value of 10 HU is, by most authors, considered essential for distinguishing between adenomas and non-adenomas.

However, approximately 30% of adenomas contain small amounts of lipids and thus have a density higher than 10 HU on unenhanced scans. This fact poses a serious differential diagnostic challenge with respect to distinguishing other clinically significant pathological findings with a higher unenhanced density. Thus, an unenhanced CT scan alone cannot be used to distinguish adenoma with a low lipid content from carcinoma, phaeochromocytoma, metastasis, and/or other less frequent adrenal findings (old haematoma, ganglioneuroma, lymphoma, etc.). Since all these pathological findings may have a similar presentation following intravenous administration of a contrast medium, an enhanced scan alone is of no major use in distinguishing these groups. It is useful only if delayed scans with washout characteristics are performed. Adenomas show a rapid washout of contrast medium in comparison with primary carcinomas, metastases, and phaeochromocytomas in which washout of contrast medium is slower. An absolute washout of more than 60% and a relative washout of more than 40% is indicative of adenoma; conversely, values of absolute washout below 60% and those of relative washout below 40% correspond...
to non-adenomas (carcinoma, metastases, phaeochromocytoma) (ref. 69-71). The sensitivity, specificity, and accuracy of washout characteristics in distinguishing adenoma from malignancy are 89-98%, 92-95%, and 91-96%, respectively (ref. 60, 68, 72-77). Calculation of absolute or relative reduction in density can thus be used to distinguish adenomas from non-adenomas. By contrast, delayed scans failed to show differences among these three groups of non-adenomas 67. Delayed scans are very useful in approximately 30% of adenomas that do not contain larger amounts of lipids and remain difficult to distinguish by means of unenhanced CT or chemical shift MR imaging 67, 71.

In terms of hormonal activity, adrenal incidentalomas can be divided into two main groups: tumours with no hormonal activity and endocrine-active tumours. The majority are found to be non-functional lesions (up to 85%) (ref. 69). Lesions with hormonal activity most typically present by subclinical Cushing’s syndrome 69. Histopathologically, the most commonly detected adrenal incidentalomas are adenomas; other pathological findings (myelolipomas, phaeochromocytomas, carcinomas, metastases, and other rare causes) are less frequent, with the percentages being different in different authors 59, 60, 61.

Based on imaging techniques and hormone tests, either surgical management or surveillance of the patient are indicated. Biopsy of the adrenal expansion is associated with general risks and may be of no benefit. Therefore, in the vast majority of cases, further management is determined only based on the morphological presentation on imaging studies and biochemical analysis of secretory activity. Differential diagnosis and correct interpretation of the incidentaloma finding are therefore essential, and the decision-making process particularly important when the patient has already been diagnosed with an extra-adrenal malignancy (e.g., lung carcinoma). In these cases, the distinction of the nature of the lesion is absolutely crucial for further management: metastases require an active approach while observation alone is sufficient in non-functional adenoma.

Follow-up examinations with imaging techniques are a widely discussed issue in incidentalomas. No general consensus on the need for further follow-up CT examinations and their frequency has been reached so far 62, 63.

In summary, if an incidentaloma has a density of less than 10 HU on CT, it is an adenoma and the diagnosis is definitive (an exception is myelolipoma, with its appearance with negative densities causing no diagnostic confusion). If an incidentaloma has a density of more than 10 HU and washout characteristics are not unequivocal, there are two strategies for follow-up management. Firstly, the examination can be repeated within a certain time period in order to ascertain whether or not the incidentaloma has distinctly enlarged. Secondly, a PET/CT scan can be performed, in which case benign nature can be expected in the absence of radiopharmaceutical accumulation.

CONCLUSION

The issue of adrenal disease is increasingly current due to major advances in laboratory diagnosis, genetics, options of imaging techniques, and, not least, major progress in surgical techniques for these conditions. Clinically most significant pathological findings in the adrenal gland include adenoma, phaeochromocytoma, carcinoma, and metastases.

These findings may be non-functional, but may also be associated with clinical syndromes. Also very relevant are adrenal incidentalomas due to the increasing frequency of their detection because of the increasing availability and development of imaging techniques.

AUTHORSHIP CONTRIBUTIONS


CONFLICT OF INTEREST STATEMENT

None declared.

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