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**Case 1****Adrenal Hemorrhage**

A 30-year-old patient who suffered a traffic accident presented to the emergency room with right upper quadrant pain.

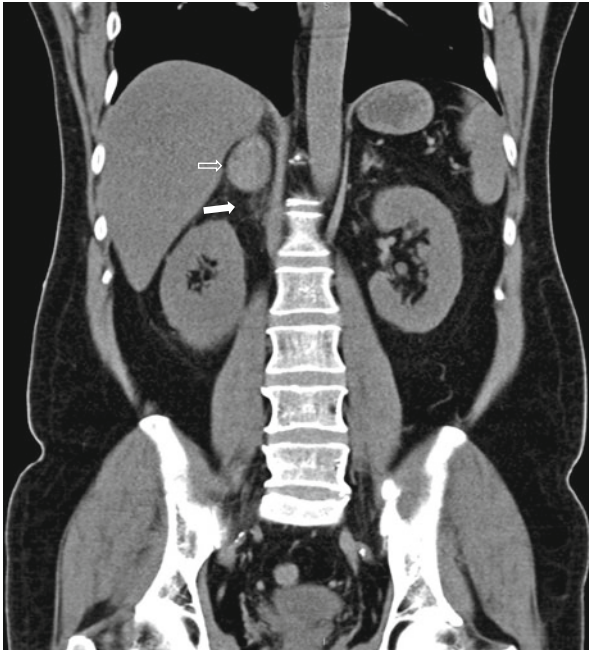


Fig. 2.1

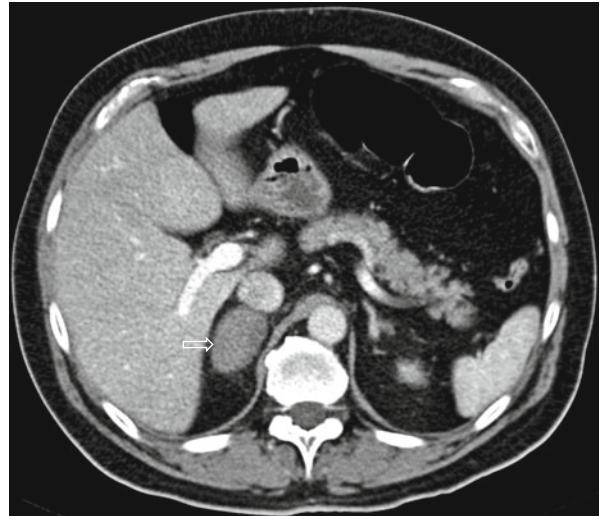


Fig. 2.2



Fig. 2.3

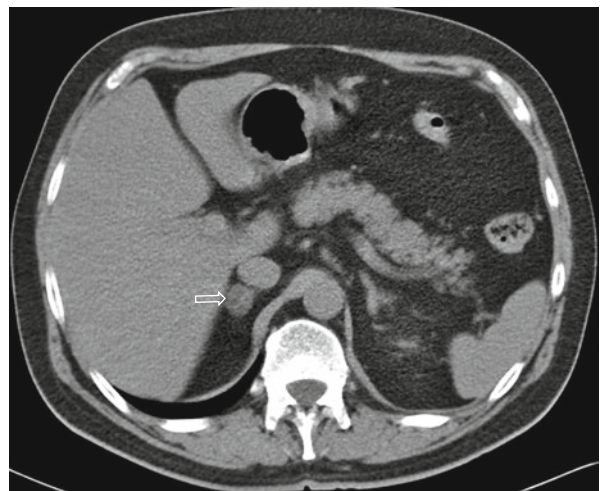


Fig. 2.4

The causes of adrenal hemorrhage can be classified as traumatic and nontraumatic. Direct trauma is the main cause of unilateral adrenal bleeding, more frequently right-sided. Adrenal hemorrhage is commonly associated with injuries of high severity. Right-sided hemorrhage is associated with liver, spleen, bilateral renal injuries, and pneumothorax; when hemorrhage involves the left adrenal, splenic, left kidney, and pneumothoracic injuries are the most common accompanying injuries. Periadrenal stranding is identified in most cases.

On the other hand, direct trauma is seldom the origin of bilateral adrenal hemorrhage. Nontraumatic hemorrhage of the gland in adults is uncommon and can be classified into four categories:

Stress (surgery, burns, sepsis, hypotension, etc.).

Hemorrhagic diathesis or coagulopathy (heparin-associated thrombocytopenia, antiphospholipid antibody syndrome, anticoagulation, use of steroids, etc.).

Underlying adrenal tumors (myelolipoma, pheochromocytoma, adrenocortical carcinoma). Idiopathic/spontaneous.

Potential causes of unilateral or bilateral hemorrhage are listed in Table 2.1

**Table 2.1** Potential causes of unilateral or bilateral hemorrhage

| Unilateral                   | Bilateral                             |
|------------------------------|---------------------------------------|
| Trauma/biopsy                | Stress                                |
| Spontaneous (rule out tumor) | Hemorrhagic diathesis or coagulopathy |
| Tumor                        | Spontaneous                           |

CT is the imaging modality of choice in patients with clinical suspicion of adrenal hemorrhage. Acute adrenal hemorrhage appears as enlarged adrenal gland with irregular margins but commonly retaining adreniform shape. However, sometimes the adrenal hemorrhage may present as an adrenal mass. At unenhanced CT, acute bleeding shows high attenuation (50–90 HU). No enhancement following IV contrast is observed. In doubtful cases, findings of decreased density and size at follow-up as well as the presence of calcifications may help confirm the diagnosis.

MR imaging is the most sensitive and specific modality for diagnosing adrenal hemorrhage. The appearance of adrenal hemorrhage on MRI depends on the progression of the bleeding. In the subacute phase, the hemorrhage shows high signal on T1-weighted sequences, secondary to the presence of methemoglobin. No postcontrast enhancement is observed in acute phase. During the chronic phase there is a ring of low signal on T2-weighted sequences, secondary to the presence of hemosiderin.

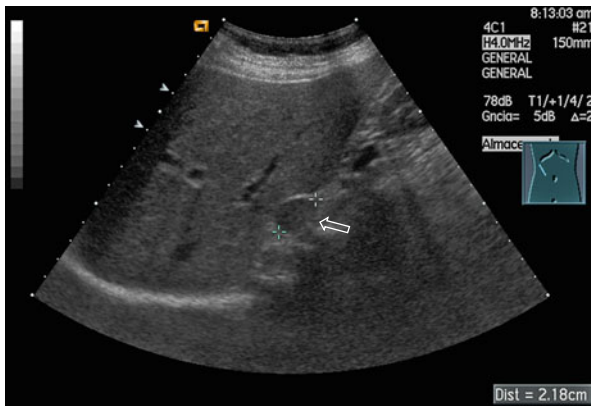
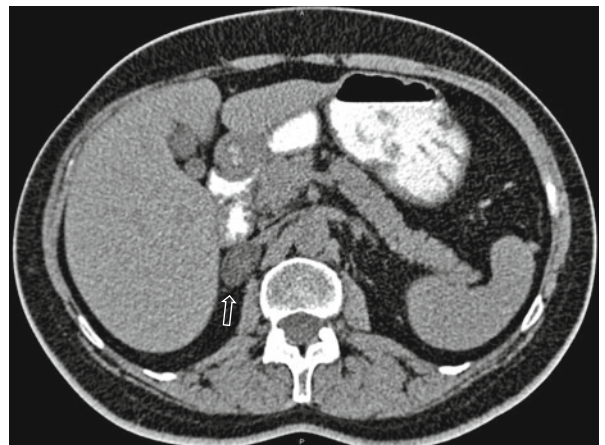
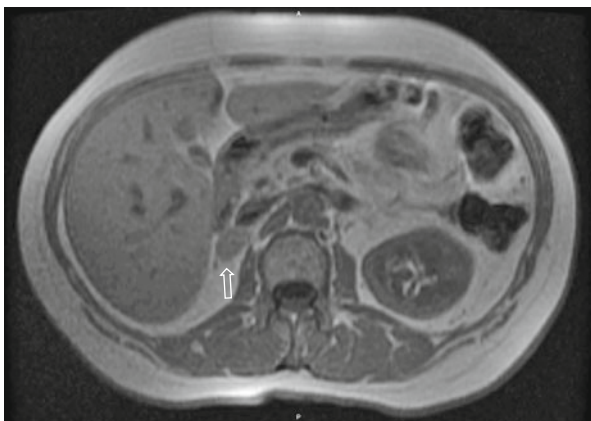
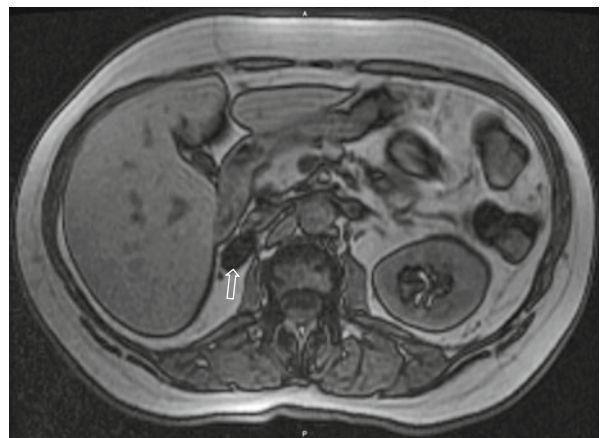
Coronal/axial unenhanced CT (Figs. 2.1 and 2.2) shows right adrenal lesion (arrow) of 4 cm in size. This lesion is hyperdense (49 HU) at unenhanced CT. Slight stranding of adjacent fat is observed (arrow). Axial enhanced CT at portal phase (Figs. 2.2 and 2.3) shows no significant enhancement of the right adrenal lesion following iv contrast (52 HU) (arrow). It is important to keep in mind that adrenal hemorrhage is commonly associated with other injuries, as in this case where it appears associated with a laceration of the liver dome (segment VII) (arrow). Axial unenhanced CT (Fig. 2.4) performed 6 months later shows that the lesion has decreased in size and is hypodense (arrow). Stranding is not observed in the adjacent fat.

## Comments

## Imaging Findings

**Case 2****Lipid-Rich Adenoma**

A 56-year-old female with a history of episodes of renal colic. An US was performed to detect urolithiasis, which showed an incidental finding of a right adrenal nodule.

**Fig. 2.5****Fig. 2.6****Fig. 2.7****Fig. 2.8**

The adrenal adenomas are benign tumors of the adrenal cortex, commonly unilateral. The estimated prevalence in general population is approximately 6%, without significant differences between men and women. The prevalence of adrenal adenomas increases with increasing age. Approximately 85% are nonfunctioning and clinically inapparent, and 15% are functioning and may develop clinically apparent or subclinical hormone hyperproduction (frequently due to autonomous cortisol or aldosterone secretion, and less often due to sex hormone secretion). Therefore, all patients with an adrenal lesion should undergo a hormonal evaluation. On CT or MRI may present as small-sized homogeneous lesions (usually <4 cm in diameter) with smooth borders. Nevertheless, these are nonspecific characteristics, given that other malignant and benign adrenal lesions can present similar features.

In the presence of an incidentally discovered adrenal mass in patients not known to have cancer, the initial diagnostic hypothesis may be that of an adenoma. However, metastasis, lymphoma, carcinoma, pheochromocytoma, hemorrhage, adrenal hyperplasia, infectious diseases (tuberculosis, histoplasmosis, blastomycosis), neuroblastoma, ganglioneuroma, hemangioma, and hemangiosarcoma should be included in the differential diagnosis.

Seventy percent of adrenal adenomas have a high intracellular fat content (lipid-rich adenomas), detection of which is the cornerstone of establishing diagnosis with a high confidence. Nonenhanced CT is the mainstay of image diagnosis of lipid-rich adrenal adenomas due to its high sensitivity and specificity, low cost, and easy interpretation. An attenuation value  $\leq 10$  HU (Hounsfield units) is diagnostic of adrenal adenoma. If an incidental adrenal mass is detected on routine contrast-enhanced CT acquired in the dual-energy mode, it is possible to suppress the iodine signal, thus allowing density measurements of adrenal lesions on virtual nonenhanced imaging.

Chemical shift MRI is another technique that has a high diagnostic accuracy in the characterization of lipid-rich adenomas. It consists in acquiring T1-weighted gradient-echo in-phase and opposed-phase sequences. The signal intensity loss in the lesion on opposed-phase images relative to the spleen (visual comparison) is indicative of the presence of intracytoplasmatic lipids.

US (Fig. 2.5) demonstrates the presence of a well-delineated nodule in the right adrenal area (open arrow).

A nonenhanced CT (Fig. 2.6) is performed and shows a homogeneous low attenuation (3 HU) right adrenal nodule of 2.7 cm with smooth borders (open arrow). Based on these features, especially the low attenuation values, a diagnosis of lipid-rich adenoma can be established.

A MRI is also performed with T1-weighted gradient-echo in-phase (Fig. 2.7) and opposed-phase (Fig. 2.8) sequences. There is an evident signal intensity loss in the adrenal nodule (open arrows) on opposed-phase with regard to the in-phase images, which is indicative of the presence of intracytoplasmatic lipids and therefore is diagnostic of adenoma.

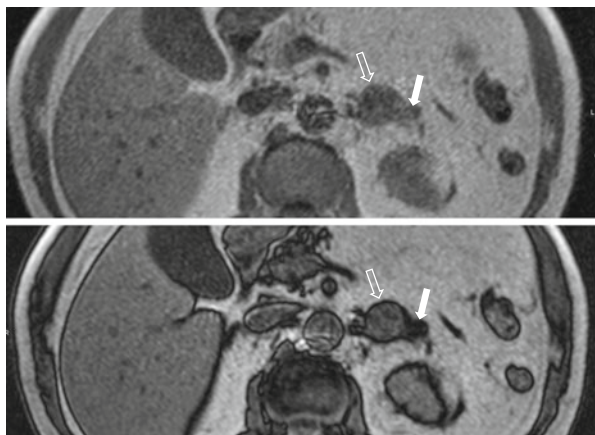
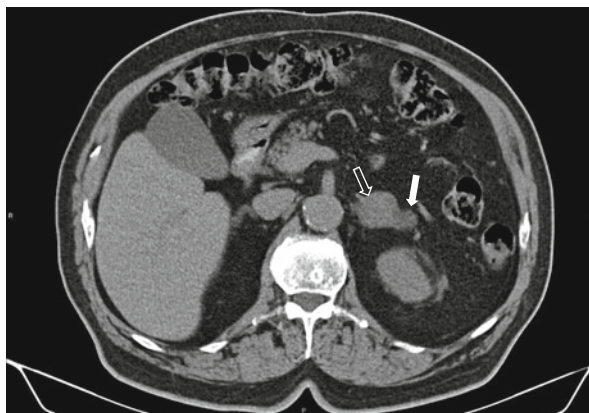
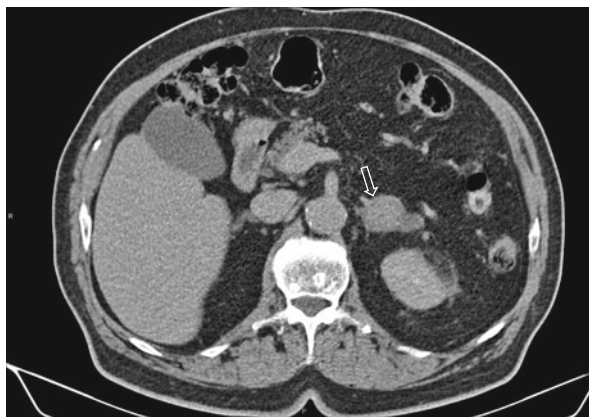
## Comments

## Imaging Findings

**Case 3****Lipid-Poor  
Adenoma****Comments**

A 67-year-old female who undergoes a lumbar spine MRI due to history of chronic low back pain, which showed an incidental finding of a left adrenal nodule. An abdominal MRI was performed, followed by a three-phase adrenal CT, as part of the diagnostic workup.

Approximately 30% of adrenal adenomas have a low intracellular fat content (lipid-poor adenomas). They have an unenhanced CT attenuation greater than 10 UH and therefore cannot be reliably differentiated from other adrenal lesions with this technique. Although some

**Fig. 2.9****Fig. 2.10****Fig. 2.11****Fig. 2.12**

morphological features (small size, homogeneity, smooth contour) are suggestive of its benignity, other benign and malignant adrenal lesions can have similar features. The differential diagnosis must be established mainly with metastasis, carcinoma, pheochromocytoma, and lymphoma, which are lesions that could have an important impact on prognosis of patients. The clinical and biochemical parameters, as well as the previous history play an important role in the characterization of adrenal lesions with CT attenuation values greater than 10 HU at nonenhanced CT. Diagnosis of lipid-poor adenomas is a challenging task for radiologists. Chemical shift MRI technique is a useful technique for study of adrenal lesions with nonenhanced attenuation of 10–30 HU. It can be explained by the fact that, despite their high unenhanced CT attenuation, they have sufficient lipid content to allow their characterization. The signal intensity loss in the lesion on opposed-phase images relative to the spleen is indicative of the presence of intracytoplasmatic lipids and therefore is diagnostic of adenoma.

The dedicated adrenal CT protocol consists of an initial nonenhanced CT followed by enhanced CT at 60 s and 15 min after initiating the intravenous injection of the contrast material. It will permit calculation of absolute washout (or relative washout if nonenhanced CT images were not acquired initially):

$$\text{Absolute Washout} = (\text{AC 60 sec} - \text{AC 15 min}) / (\text{AC 60 sec} - \text{AC nonenhanced}) \times 100$$

$$\text{Relative Washout} = (\text{AC 60 sec} - \text{AC 15 min}) / (\text{AC 60 sec}) \times 100$$

\*AC = attenuation coefficient (HU)

Absolute washout values  $\geq 60\%$  (or relative washout values  $\geq 40\%$ ) has demonstrated high accuracy in diagnosis of adrenal adenoma, independently of its lipid content.

$^{18}\text{F}$ -FDG PET/CT has high sensitivity but lower specificity to differentiate between benign and malignant adrenal lesions, with a 5% false-positive rate.

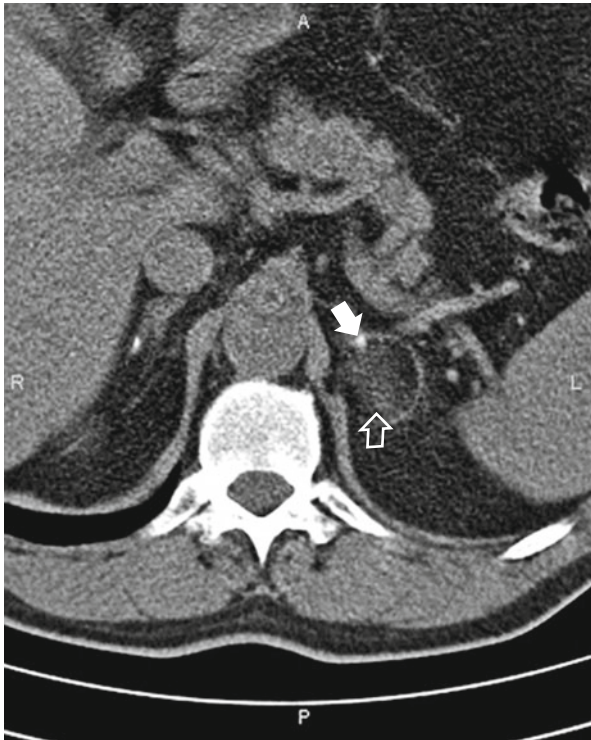
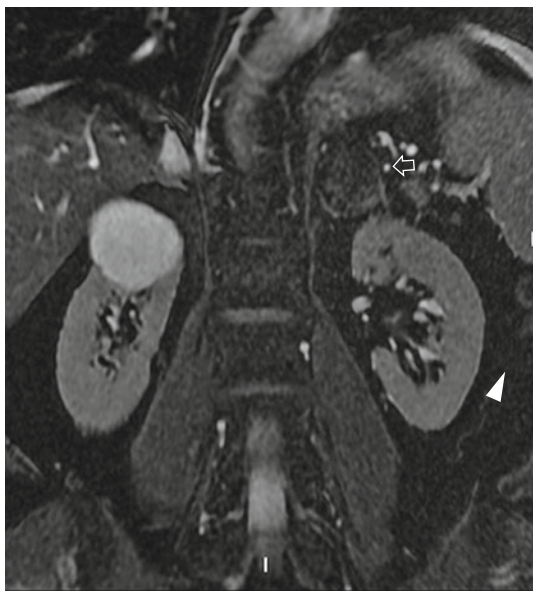
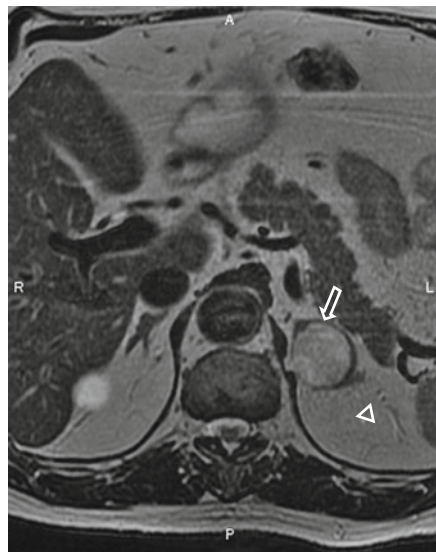
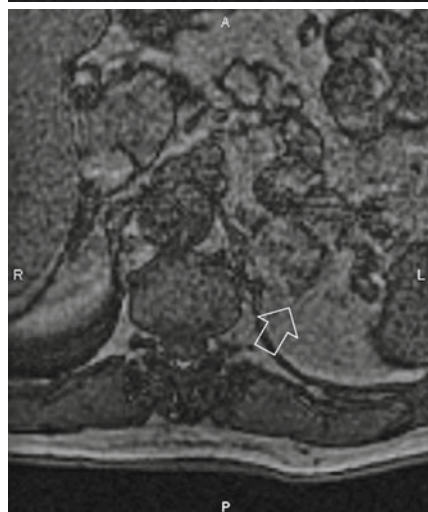
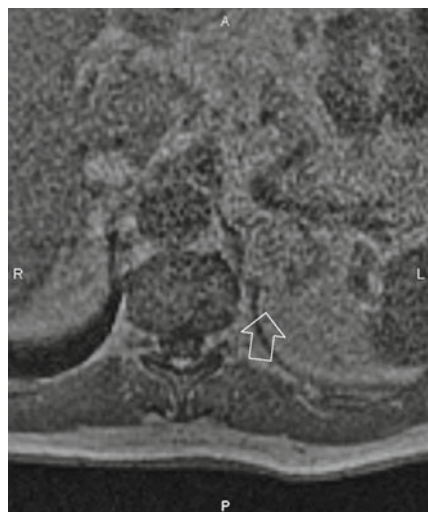
A small percentage of these lesions will remain indeterminate despite the use of all available noninvasive diagnostic imaging techniques. Depending on patient's characteristics, imaging features as well as the level of suspicion, adrenalectomy, fine needle aspiration, or expectant management with serial biochemical and imaging follow-up should be recommended.

An MRI with T1-weighted gradient-echo in-phase (Fig. 2.9a) and opposed-phase (Fig. 2.9b) sequences is performed. There is a 2.8 cm left adrenal nodule (open arrows) which does not show significant signal intensity loss on opposed-phase with regard to the in-phase images.

A three-phase abdominal CT with specific adrenal protocol is then performed, and demonstrates a well-delimited homogeneous left adrenal nodule (open arrows) with non-enhanced (Fig. 2.10) attenuation values of 24 HU. The enhanced images acquired at 60 s (Fig. 2.11) and 15 min (Fig. 2.12) show attenuation values of 86 and 44 HU, respectively. The absolute washout value is  $\geq 60\%$  and therefore is diagnostic of lipid-poor adenoma.

A small left adrenal nodule can also be seen on CT and MRI (arrows). It has low attenuation values (–6 HU) on nonenhanced CT (Fig. 2.10) and shows signal intensity loss on opposed phase (Fig. 2.9), compatible with a lipid-rich adenoma.

## Imaging Findings

**Case 4****Myelolipoma****Fig. 2.13****Fig. 2.15****Fig. 2.14****Fig. 2.16**

A 61-year-old man, a heavy smoker, with an incidental adrenal finding on a chest CT scan. Follow-up of the adrenal lesion was performed with MR.

Myelolipomas are rare benign nonfunctioning adrenal tumors present in 0.08–0.2% of autopsies. They are composed of a variable proportion of mature fat and bone marrow elements. There is no sexual predominance and, although typically unilateral, they can be multiple and bilateral. Punctate calcifications can be seen in 20% of cases. Myelolipoma is generally asymptomatic and usually detected as an incidental finding in cross-sectional imaging performed for other purposes. Rarely, patients may present with acute flank pain or hypovolemic shock secondary to spontaneous bleeding.

As with renal angiomyolipomas, confident diagnosis is achieved detecting the presence of fat in an adrenal mass. The suprarenal localization determines the differentiation with angiomyolipomas, and this is where multiplanar images of reconstructed multidetector CT or sagittal and coronal MRI sequences are most helpful.

On CT, fatty areas have attenuation values below  $-30$  HU. Areas of bone marrow elements present as soft tissue densities within the lesion. The margins are well defined except when bleeding extends the lesion into the adjacent fat.

The fatty elements of myelolipomas behave as the retroperitoneal fat in the different MRI sequences, typically hyperintense in T1WI. Fat-suppressed images show signal intensity loss in fatty areas. No chemical-shift should be expected because fat in these lesions in the form of adipose tissue is not related to intracellular lipids as opposed to adrenal adenomas. Bone marrow elements have no characteristic signal intensity and may appear variable depending on its composition. Areas of hemorrhage vary its signal intensity depending on the time passed since bleeding.

Myelolipomas have no malignant potential. The clinical relevance of these lesions lies in the increased spontaneous bleeding risk with size, especially when larger than 10 cm.

Nonenhanced axial CT (Fig. 2.13) demonstrates the presence of a predominantly fat nodular lesion in the left adrenal gland (open arrow). The lesion has a punctate peripheral calcification (arrow). Figure 2.14: T2-weighted axial image at the same level as Fig. 2.13 shows internal signal intensity similar to the surrounding fat (open head arrow). T2-weighted coronal fat-suppressed MR image (Fig. 2.15) shows signal intensity drop of the fatty tissue from the lesion (open arrow), similar to the surrounding retroperitoneal fat (head arrow) compared to Fig. 2.12. In-phase (a) and out of phase (b) axial T1-weighted MR images (Fig. 2.16) depict no drop of signal in the fatty tissue of the angiomyolipoma as opposed to adenomas.

## Comments

## Imaging Findings

## Case 5

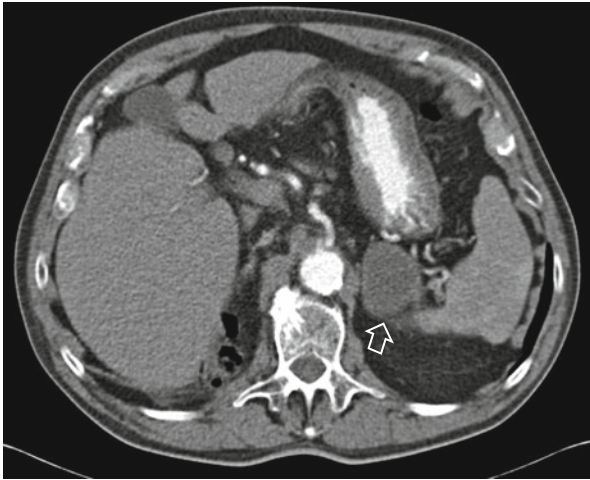
■  
Adrenal Ganglioneuroma

Fig. 2.17

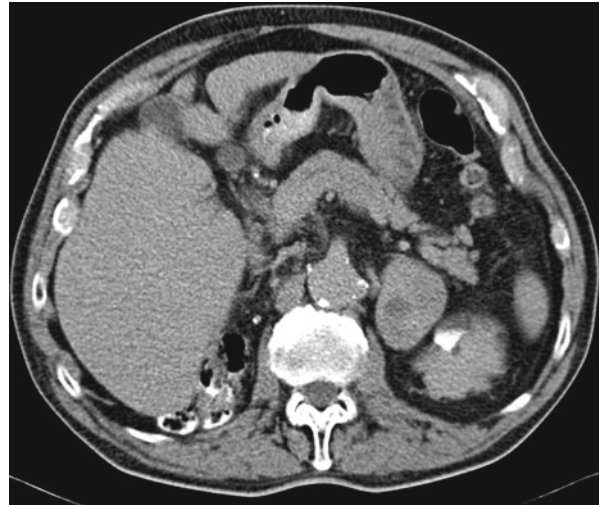


Fig. 2.18

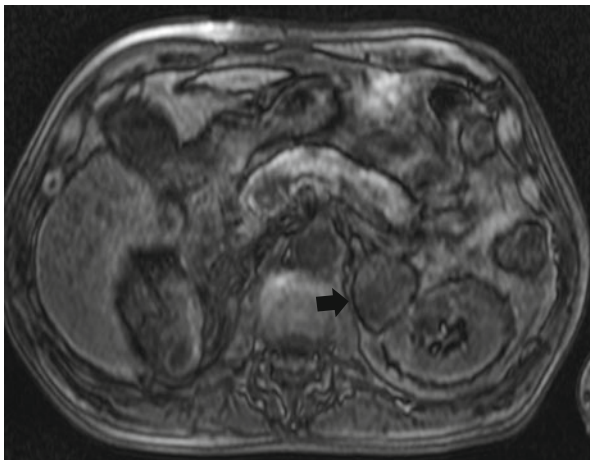


Fig. 2.19

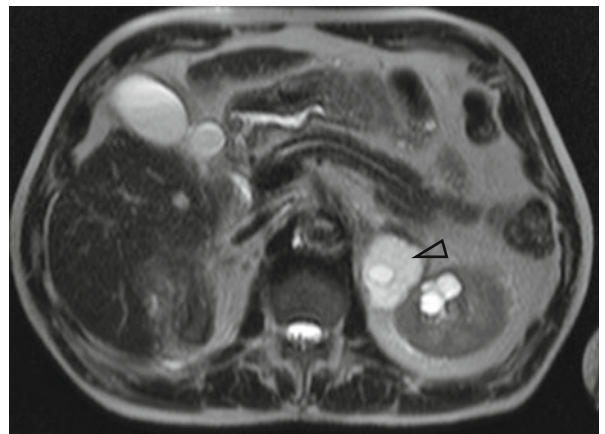


Fig. 2.20

Imaging workup of a 71-year-old male with recurrent bladder tumor. No previous abdominal scan had been performed.

Ganglioneuromas are uncommon neurogenic slow-growing tumors that may arise from any of the paravertebral sympathetic ganglia, occasionally from the adrenal medulla. Unlike ganglioblastomas and neuroblastomas – also neurogenic tumors derived from the sympathetic neurons of the neural crest – ganglioneuromas lack malignant potential and are therefore considered benign.

They are usually found in the posterior mediastinum or in the retroperitoneum, and 15–30% are adrenal.

A low percentage of cases are symptomatic due to catecholamine secretion or present as unspecific abdominal pain or palpable mass. Asymptomatic ganglioneuromas account for about half of the cases and are discovered incidentally in young adults with no sex predilection.

Adrenal ganglioneuromas appear on CT as a well-defined solid homogeneous mass. Size at diagnosis is generally over 5 cm. On nonenhanced CT they usually have low attenuation values and demonstrate a low or moderate enhancement after contrast administration. Calcifications can be present in approximately half of the cases.

On MR, they have low signal intensity on T1-weighted sequences and heterogeneous high signal intensity on T2-weighted sequences. Slow and late progressive enhancement can be seen with contrast, especially on delayed images.

A typical whorled appearance has been described for ganglioneuromas.

Ganglioneuroblastomas are similar tumors that unlike ganglioneuromas have immature tissue that is malignant or has malignant potential. They are less frequent and appear in childhood. The adrenal gland is the most common site of origin. They may present with abdominal pain and distension. As aggressive tumors, they may have irregular contours and invasion of adjacent organs but there are no typical imaging features on CT. Signal intensity on MR of ganglioneuroblastomas and neuroblastomas is similar to that of ganglioneuromas. Dynamic MR helps in the differential diagnosis as the formers have increased early enhancement unlike ganglioneuromas.

Final diagnosis is histological after surgical resection that is generally indicated because they are usually nonfunctional adrenal masses with size greater than 5 cm. Prognosis of completely resected mature adrenal ganglioneuromas is excellent.

Arterial phase axial CECT (contrast enhanced) (Fig. 2.17) shows a homogeneous hypodense well-circumscribed left adrenal mass (open arrow). There is no enhancement in arterial phase. No calcifications are present in this case. Axial CECT on delayed phase (Fig. 2.18) demonstrates delayed enhancement, as compared to Fig. 2.17. Diffuse low signal intensity can be seen on precontrast axial T1-weighted image with fat saturation (Fig. 2.19, solid arrow). Delayed diffuse enhancement similar to CT was seen on postcontrast images (not shown). Axial T2-weighted image (Fig. 2.20) shows hyperintense signal intensity of the lesion (open head arrow).

## Comments

## Imaging Findings

Case 6

Pheochromocytoma



Fig. 2.21

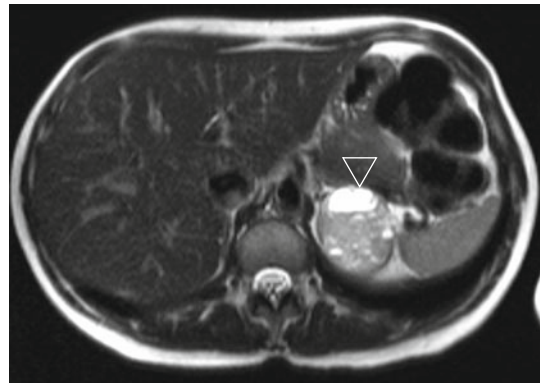


Fig. 2.22

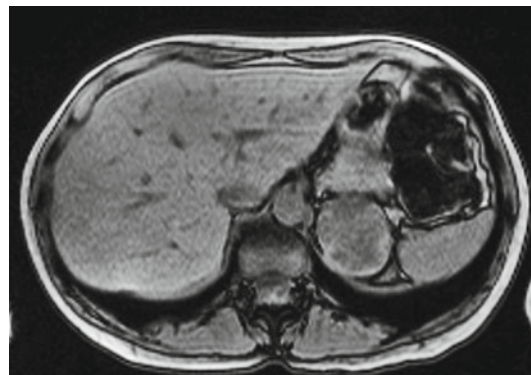


Fig. 2.23

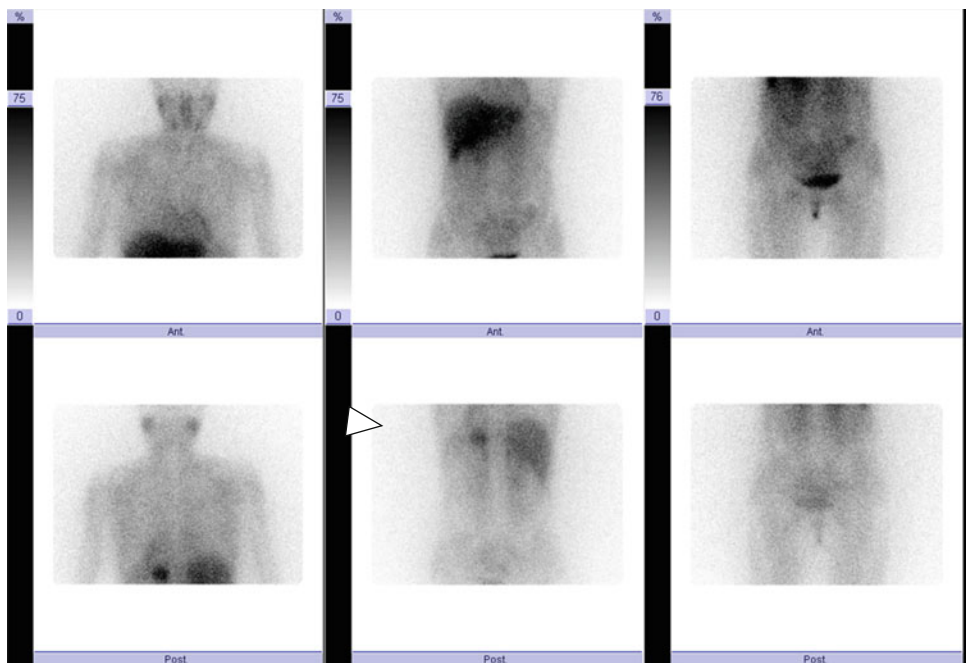


Fig. 2.24

A 38-year-old patient presented with a 3-year history of palpitations, sweating, hand tremor, and fatigue.

Pheochromocytoma is a neuroendocrine tumor of the adrenal medulla that secretes catecholamines. These tumors are typically unilateral and benign, but may be bilateral and malignant in 10% of cases. Approximately 10% of pheochromocytomas are extraadrenal and are then called paragangliomas. The clinical diagnosis is suspected in a young patient with hypertension; however, it is important to know that pheochromocytomas are the cause of hypertension in less than 1% of hypertensive patients. Laboratory tests for the evaluation of suspected pheochromocytoma include plasma catecholamine levels, 24-h urine vanilmandelic acid, and metanephrine levels (the sensitivity of these tests is >99% and specificity is 89%).

CT is the imaging modality of choice to confirm the diagnosis when clinical and laboratory tests suggest pheochromocytoma. Small pheochromocytomas usually appear as oval and well-defined lesions. Large lesions (>4–5 cm) are more likely to contain areas of necrosis or hemorrhage, as they are hypervascular tumors. Generally, pheochromocytomas contain no fat. However, in a small percentage of cases pheochromocytomas may have enough intracellular lipid content to present attenuation values <10 UH, which would make them indistinguishable from adenomas. Values of absolute washout and relative washout are similar to those of metastasis (<60% and <40%, respectively). Moreover, attenuation values in the portal phase tend to be higher than those of adenomas (up to 110–120 HU), although there is overlap. Heterogeneous enhancement of larger lesions is often observed making these pheochromocytomas indistinguishable from other adrenal lesions; therefore, biochemical correlation is required for diagnosis. In the clinical practice contrast administration is often avoided in case of high suspicion of pheochromocytoma as it might cause hypertensive crisis.

T2-weighted MRI sequences are more specific than CT because of the hyperintensity resulting from the presence of a cystic component. These tumors also manifest with moderate signal intensity on T2-weighted images. Pheochromocytomas rarely lose signal in out-of-phase (except for the rare cases of microscopic fat). After gadolinium administration, pheochromocytoma shows a marked and persistent enhancement.

Coronal contrast enhanced CT arterial phase (Fig. 2.21) shows a left solid hypervascular adrenal mass with well-defined margins (open arrow) and small hypodense areas suggestive of areas of cystic change (arrow). The lesion does not contain fat or calcification. An MRI was also performed. Axial T2-weighted MRI sequences (Fig. 2.22) show a heterogeneous mass with areas of hyperintensity (open arrowhead) resulting from cystic component. No signal loss in out-of-phase sequences was seen (Fig. 2.23). I-131 MIBG scintigraphy (Fig. 2.24) demonstrates focal deposition of radiotracer in left suprarenal location (posterior) (open arrow), with no activity in other areas studied.

## Comments

## Imaging Findings

**Case 7****Adrenal Metastases**

A 77-year-old patient presented with a 5-year history of anorexia and weight loss. An abdominal CT study was performed.



**Fig. 2.25**



**Fig. 2.26**



**Fig. 2.27**



**Fig. 2.28**

Metastases are the most common malignant lesion involving the adrenal gland. Primary tumors that most often metastasize to the adrenal glands are the lung (40%), breast (20%), gastrointestinal tract, renal cell carcinoma, thyroid, and melanoma. Although adrenal metastases are frequent, we need to take into consideration that 50% of adrenal masses in oncologic patients are benign (mostly nonfunctioning adenomas). However, adrenal metastases in the absence of a known primary tumor (true incidentalomas) are rare. A rapid growth in 4–6 months, reduction in size following chemotherapy, or the appearance of new lesions is very suggestive of metastases. Metastases are usually bilateral but may also be unilateral.

It is important to emphasize that adrenal metastases have no characteristic imaging features. Larger lesions at CT imaging are accompanied by data that suggest malignancy such as ill-defined and nodular margins, heterogeneous attenuation, and a ring of enhancement or local invasion. They can also develop central necrosis. Calcifications are rare. The presence of concomitant metastases in other organs is unusual. Small metastases may resemble adenomas. Metastases show an absolute percentage enhancement washout of less than 60% and a relative percentage enhancement washout of less than 40%.

At MR imaging, adrenal metastases exhibit low signal intensity on T1-weighted images and high signal intensity on T2-weighted images, with progressive enhancement after administration of contrast material. The most important diagnostic feature is the lack of signal loss on out-of-phase images (in comparison with adrenal adenomas).

<sup>18</sup>F-FDG PET/CT has a sensitivity of 97% and a specificity of 91% for differentiating between benign and malignant lesions, and the qualitative analysis (visual comparison between the adrenal mass uptake and liver uptake) has better accuracy compared to the quantitative analysis. PET is a highly accurate technique to differentiate between malignant and benign adrenal disease for lesions >1 cm, especially in the setting of cancer patients, other imaging studies being usually unnecessary in this subgroup of patients. However, this technique has limitations: 9% rate of false positive, due to a variety of benign causes, and a 3% rate of false negatives due to hemorrhagic or necrotic metastases, small size (<10 mm), or derived from tumors of low metabolism (bronchioloalveolar carcinoma and carcinoid tumors). PET/CT is also a high-cost technique and of limited availability.

In those lesions that grow gradually, fine needle aspiration (FNA) can be used before deciding for surgery. In cancer patients with a high suspicion of metastatic disease, FNA is particularly indicated when the definite diagnosis may involve a change in the therapeutic management.

Coronal enhanced CT scans at nephrographic and excretory phase (Figs. 2.25 and 2.26) show left adrenal mass (open arrows) of 9 cm in size, with heterogeneous enhancement, irregular margins, and central necrosis (arrows). In addition, a small satellite lesion of 1 cm cranial to the mass is observed (open arrowheads). Moreover, a lytic lesion with soft tissue component in the right sacroiliac region is observed (arrowheads). Axial enhanced CT at nephrographic phase (Fig. 2.27) shows in the middle third of the right kidney an enhancing lesion measuring 3 cm suggestive of renal cell carcinoma (star). Axial contrast-enhanced CT image (Fig. 2.28), obtained with lung window settings, shows four round and small nodular lesions of different sizes in lower lobes, suggestive of metastasis (small arrows).

## Comments

## Imaging Findings

## Case 8

### Adrenocortical Carcinoma

A 67-year-old male presented with an 18-month history of progressive right-sided flank pain/discomfort with weight loss, asthenia, and anorexia. A large mass was palpable in the right upper quadrant of the abdomen on physical examination. Biochemical and endocrine evaluation showed normal adrenal function.

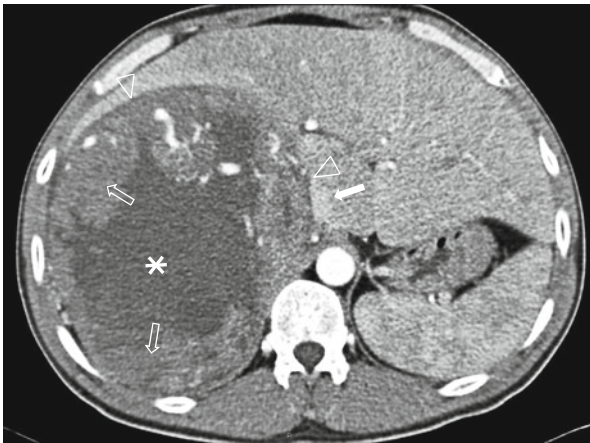


Fig. 2.29



Fig. 2.30

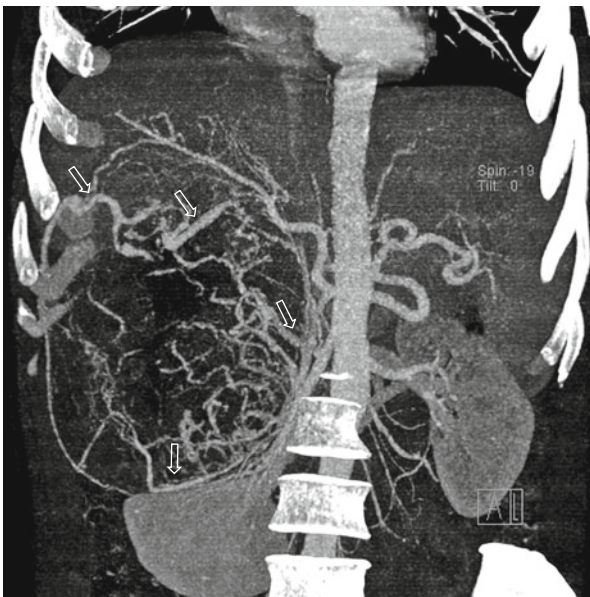


Fig. 2.31



Fig. 2.32

Adrenocortical carcinomas (ACC) are rare malignant tumors arising from the cortex of adrenal gland with an annual incidence of 0.5–2 patients/million population. Approximately 50% are functional, and Cushing's syndrome is the most frequent presentation. Up to 5% of all incidentally found adrenal masses are ACC, and its prevalence is related to the size (2% of tumors  $\leq 4$  cm, 6% of tumors  $>4$  cm and  $\leq 6$  cm, and 25% of tumors larger than 6 cm). Large-sized lesions have also worse prognosis, with a 5-year survival rate of less than 50%. At the time of diagnosis, 20–50% of patients have metastasis.

ACC presents characteristically as large heterogeneous masses in the suprarenal region, frequently displacing adjacent organs, with irregular, nodular, and poorly defined margins. Heterogeneous enhancement after intravenous administration of contrast material, mainly peripheral, is characteristic. On MRI, there may be an inhomogeneous predominantly low signal intensity on T1WI and high signal intensity on T2WI; and on CT, may show low attenuation areas with foci of high attenuation that correlate with necrosis and hemorrhage. Calcifications may be found in 20–30% of cases. The presence of these features as well as local invasion, distant metastasis, and lymphadenopathies are suggestive of ACC. In some cases, there may be tumor extension into the inferior vena cava via the adrenal veins, the identification of which is crucial for surgical planning.

Nevertheless, lesions  $<5$  cm may be homogeneous, with smooth and well-delineated borders, without invasion of adjacent structures. Therefore, adenoma and small-sized metastases must be included in the differential diagnosis.

On chemical shift MRI, ACC do not exhibit signal loss on opposed-phase images because they generally do not contain intracellular fat.

ACC are fast-growing tumors and it is unusual that untreated malignant lesions do not increase in size over a period of 6 months. Therefore, the stability of tumor size on imaging follow-up may exclude the diagnosis of ACC.

The definitive diagnosis is usually established by histology and immunohistology (core needle biopsy or excisional surgery).

A multiphasic abdominal MDCT is performed. Late arterial phase axial CT through the upper abdomen (Fig. 2.29) and oblique coronal MPR image from portal venous phase CT (Fig. 2.30) show a large hypervascularized right flank/upper quadrant abdominal mass which presents heterogeneous enhancement after intravenous administration of contrast material, mainly peripheral (open arrows), as well as a central area of necrosis (asterisks). The mass displaces the right kidney inferiorly and compresses the liver and inferior cava vein (arrow). There is a thin hypodense cleavage plane (open arrowheads) between the tumor and the liver, inferior cava vein and kidney, which excludes direct tumor invasion. Calcifications can be seen inside the tumor (arrowhead).

Oblique coronal thick-slice MIP image (Fig. 2.31) shows marked hypervascularization with thick neoformed arterial vessels (open arrows).

Coronal thin-slice MIP image (Fig. 2.32) shows compression of the inferior vena cava which is permeable (open arrows).

## Comments

## Imaging Findings

## Case 2.9

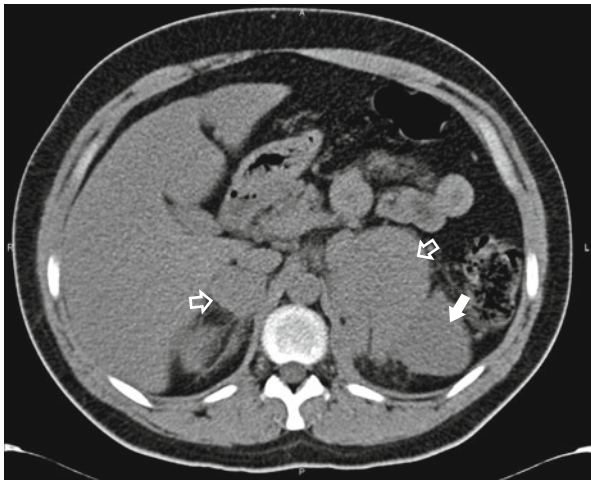
■  
Lymphoma

Fig. 2.33

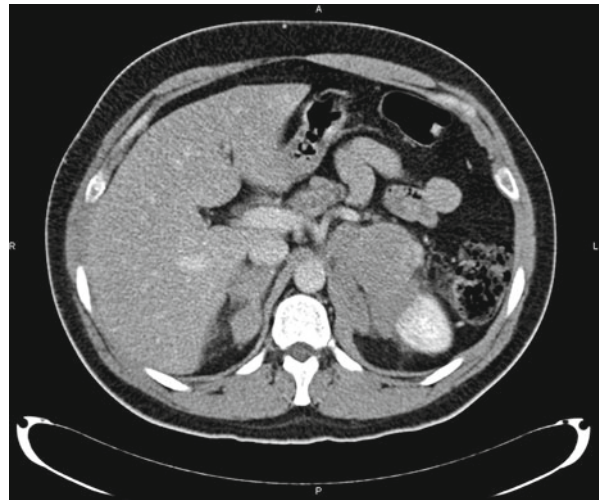


Fig. 2.34

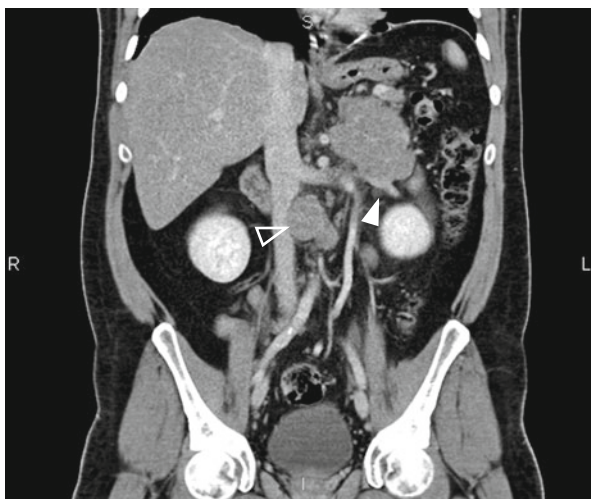


Fig. 2.35

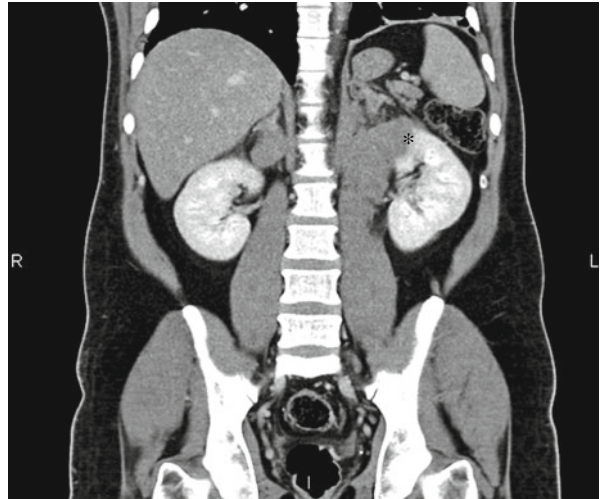


Fig. 2.36

A 31-year-old man with AIDS is evaluated for abdominal pain. A renal mass is seen on ultrasound and further characterization is performed with abdominal CT.

Lymphomas are lymphoproliferative malignancies that are classified into Hodgkin and non-Hodgkin; the latter includes more than 30 types. Extranodal lymphoma refers to involvement of organs and tissues other than lymph nodes, thymus or, in non-Hodgkin lymphoma, also the spleen. Extranodal lymphoma is more common in non-Hodgkin lymphomas and in patients with recurrent disease or immunodeficiency-related diseases such as AIDS.

Although lymphoma of the adrenal gland is uncommon, in 4% of patients with non-Hodgkin lymphoma there is adrenal involvement. Primary adrenal lymphoma is extremely rare and most of adrenal lymphomas are secondary to extraadrenal non-Hodgkin lymphomas usually associated with retroperitoneal adenopathy. Diffuse B-cell type is the most common type of primary adrenal lymphoma.

They may replace normal gland and although they are usually clinically silent, they may present with adrenal insufficiency symptoms (Addison disease), especially when bilateral. Up to 50% of cases are bilateral, a percentage that rises to 70% in primary adrenal lymphomas.

On NECT, lymphomas normally appear as a big soft tissue mass, mainly hypodense with preserved adrenal shape. When large, they may appear heterogeneous or as a complex cystic mass due to central necrosis. Smaller lesions tend to be homogeneous and are difficult to differentiate from lipid-poor adenomas. Contrast enhancement is moderate being less than the renal cortex. Calcification is rare in the absence of treatment.

On MR, adrenal lymphomas have low signal intensity on T1-weighted images and a typical heterogeneous signal on T2 WI.

On US lymphomas appear as homogeneously hypoechoic lesions with echogenic areas within the mass.

The presence of concomitant multiple big retroperitoneal lymphadenopathy or ipsilateral renal involvement suggests diagnosis of adrenal lymphoma.

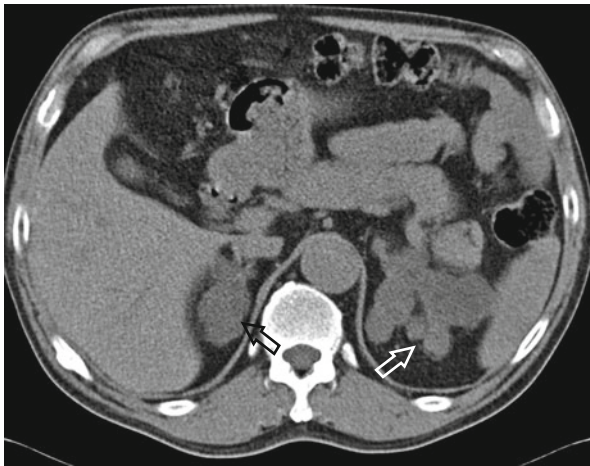
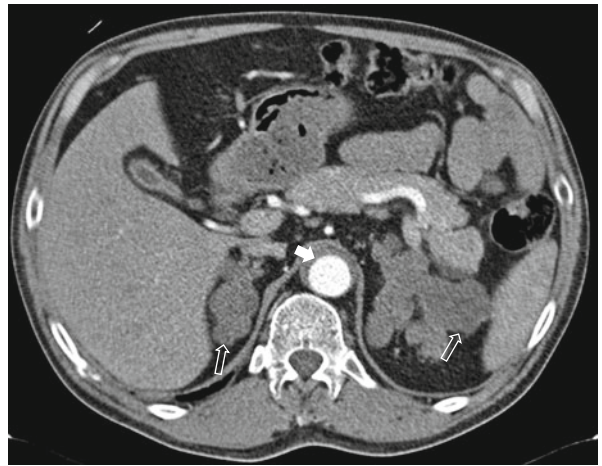
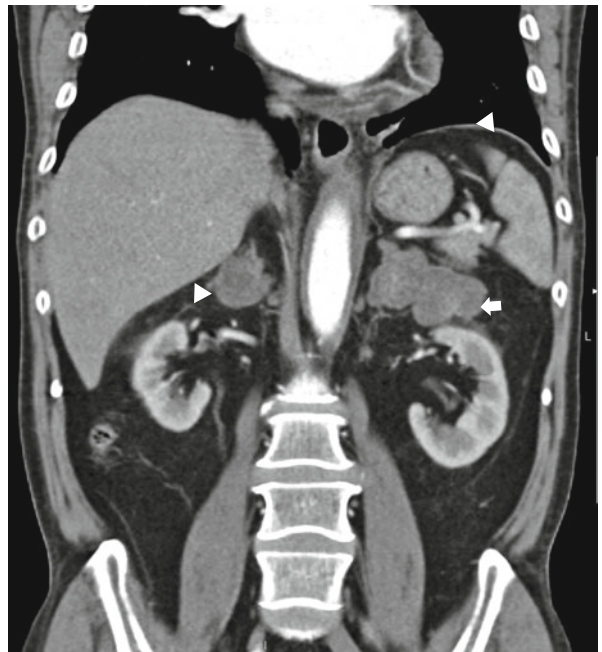
Axial nonenhanced CT (Fig. 2.33) shows bilateral adrenal masses (open arrows). The left adrenal contacts widely with the upper pole of the kidney (solid arrow). On CECT (Fig. 2.34), mild homogeneous enhancement is seen in the adrenal masses, lower than kidney enhancement. Coronal CECT (Fig. 2.35) depicts associated retroperitoneal interaortocaval lymphadenopathy (open head arrow). The adrenal mass reached and encased the renal artery without infiltration (solid head arrow). On CECT (Fig. 2.36), the retroperitoneal adrenal mass infiltrates the left upper pole of the kidney (asterisk).

## Comments

## Imaging Findings

**Case 10****Adrenocorticotropin-Independent  
Macronodular Adrenal Hyperplasia**

A 53-year-old male was admitted to the emergency room and was diagnosed of a type B thoracic aorta dissection on the CT examination with bilateral adrenal nodules. Subsequent hormonal study showed increased cortisol in urine and low adrenocorticotropin (ACTH).

**Fig. 2.37****Fig. 2.38****Fig. 2.39****Fig. 2.40**

The most common cause of Cushing's syndrome is iatrogenic. Endogenous Cushing's syndrome is divided into ACTH-dependent and ACTH-independent forms. The latter is less common (around 20%) and always caused by primary adrenal disease.

ACTH-independent macronodular adrenal hyperplasia (AIMAH) is one of the causes of ACTH-independent Cushing's syndrome. Other causes include adrenal neoplasms (functional adenoma or carcinoma) that account for 95% of the cases, and primary pigmented nodular adrenocortical disease (PPNAD).

The pathogenesis of this disease still remains unclear and several hypotheses are under debate, including autonomous hypersecretion after chronic ACTH stimulation or secretion in response to other stimulating hormones other than ACTH.

AIMAH occurs in patients with a mean age older than those with ACTH-independent functional adenomas – around the fourth decade of life – and has a slight male predilection. Although a few familial cases have been reported, AIMAH is generally sporadic. Symptoms associated with this condition are hypertension, weight gain, and glucose intolerance or diabetes mellitus.

Normal adrenal glands have an inverted V or Y shape on axial CT imaging and normal limb thickness is <5 mm. Adrenal hyperplasia is defined by histopathological examination; however, large lesions may be characterized by imaging techniques. ACTH-dependent Cushing's syndrome adrenal hyperplasia appears as diffuse bilateral enlargement or nodular hyperplasia (micro or macronodular) with nodules generally <2 cm. PPNAD is usually seen as micronodular hyperplasia.

CT appearance of AIMAH is quite unique, showing massive bilateral adrenal hyperplasia with multiple nodules in the limbs that range from 1 to 5.5 cm, being larger than those of other diseases. Nodules, as in lipid-rich adenomas, are hypodense on unenhanced CT images. As CT imaging is characteristic, MR is not mandatory but can still be useful. On T1-weighted MRI sequences, nodules are seen as hypointense in relation to liver, slightly hyperintense on T2-weighted images and with a drop of signal on out-of-phase images. I-131-labeled adrenal scintigraphy shows increased bilateral uptake.

The nodules in PPNAD are hypointense on T2-weighted images, and nodules of hyperplasia on ACTH-dependent Cushing's syndrome tend to be isointense.

The treatment of choice is bilateral adrenalectomy that may be performed by laparoscopy.

Nonenhanced axial CT image (Fig. 2.37) shows bilateral adrenal hyperplasia (open arrows) seen as multiple nodules, some >2 mm, along the adrenal limbs. Lipid-rich nodules are seen as hypodense nodules. Contrast-enhanced CT scan in arterial phase (Fig. 2.38) shows the aortic dissection (solid arrow). Mild enhancement, more evident at the periphery of the nodules is depicted (open arrow). Contrast-enhanced CT scan in venous phase (Fig. 2.39) depicts better the peripheral enhancement of the nodules. Coronal image (Fig. 2.40) demonstrates the characteristic macronodular hyperplasia of AIMAH (solid arrowhead).

## Comments

## Imaging Findings

## Further Reading

### Books

- Federle MP (2008) *Expertddx: abdomen*. Lippincott Williams & Wilkins, Salt Lake City
- Federle MP, Jeffrey JRB, Woodward PJ (2010) *Diagnostic imaging: abdomen*. Amirsys, Salt Lake City
- Lee JKT (2006) *Computed body tomography with MRI correlation*. Lippincott Williams & Wilkins, Philadelphia
- Ros PR, Morteale KJ (2007) CT and MRI of the abdomen and pelvis: a teaching file. Wolters Kluwer/Lippincott Williams & Wilkins, Philadelphia
- Zagoria RJ (2006) *Genitourinary radiology: the requisites*. Mosby, Philadelphia

### Webpages

- <http://www.annals.org>
- <http://edips-download.myesr.org/>
- [http://poster.ng.netkey.at/esr/online\\_viewing/index.php?module=view\\_search&task=search&searchFulltext=adrenal](http://poster.ng.netkey.at/esr/online_viewing/index.php?module=view_search&task=search&searchFulltext=adrenal)
- <http://www.rsna.org/Education/>
- [www.uroweb.org](http://www.uroweb.org)

### Articles

- Blake MA, Cronin CG, Boland GW (2010a) Adrenal imaging. *AJR Am J Roentgenol* 194(6):1450–1460
- Blake MA, Prakash P, Cronin CG (2010b) PET/CT for adrenal assessment. *AJR Am J Roentgenol* 195(2):W91–W95
- Boland GW (2010) Adrenal imaging: why, when, what, and how? Part 1. Why and when to image? *AJR Am J Roentgenol* 195(6):W377–W381
- Boland GW, Blake MA, Hahn PF, Mayo-Smith WW (2008) Incidental adrenal lesions: principles, techniques, and algorithms for imaging characterization. *Radiology* 249(3):756–775
- Boland GW (2011a) Adrenal imaging: from Addison to algorithms. *Radiol Clin North Am* 49(3):511–528
- Boland GW (2011b) Adrenal imaging: why, when, what, and how? Part 2. What technique? *AJR Am J Roentgenol* 196(1):W1–W5
- Boland GW (2011c) Adrenal imaging: why, when, what, and how? Part 3. The algorithmic approach to definitive characterization of the adrenal incidentaloma. *AJR Am J Roentgenol* 196(2):W109–W111
- Boland GW, Dwamena BA, Jagtiani Sangwaiya M, Goehler AG, Blake MA, Hahn PF et al (2011) Characterization of adrenal masses by using FDG PET: a systematic review and meta-analysis of diagnostic test performance. *Radiology* 259(1):117–126
- Caoili EM, Korobkin M, Francis IR, Cohan RH, Platt JF, Dunnick NR et al (2002) Adrenal masses: characterization with combined unenhanced and delayed enhanced CT. *Radiology* 222(3):629–633
- Elsayes KM, Mukundan G, Narra VR, Lewis JS Jr, Shirkhoda A, Farooki A et al (2004) Adrenal masses: mr imaging features with pathologic correlation. *Radiographics* 24(Suppl 1):S73–S86
- Guo YK, Yang ZG, Li Y, Deng YP, Ma ES, Min PQ et al (2007) Uncommon adrenal masses: CT and MRI features with histopathologic correlation. *Eur J Radiol* 62(3):359–370
- Johnson PT, Horton KM, Fishman EK (2009) Adrenal imaging with MDCT: nonneoplastic disease. *AJR Am J Roentgenol* 193(4):1128–1135
- Kenney PJ, Wagner BJ, Rao P, Heffess CS (1998) Myelolipoma: CT and pathologic features. *Radiology* 208(1):87–95
- Leite NP, Kased N, Hanna RF, Brown MA, Pereira JM, Cunha R et al (2007) Cross-sectional imaging of extranodal involvement in abdominopelvic lymphoproliferative malignancies. *Radiographics* 27(6):1613–1634
- Miller JC, Blake MA, Boland GW, Copeland PM, Thrall JH, Lee SI (2009a) Adrenal masses. *J Am Coll Radiol* 6(3):206–211
- Miller JC, Blake MA, Boland GW (2009b) Definitive characterization of adrenal lesions. *BMJ* 338:a3092
- Miller FH, Wang Y, McCarthy RJ, Yaghamai V, Merrick L, Larson A et al (2010) Utility of diffusion-weighted MRI in characterization of adrenal lesions. *AJR Am J Roentgenol* 194(2):W179–W185
- NIH State-of-the-Science Statement on management of the clinically inapparent adrenal mass (“incidentaloma”). (2002) NIH Consens State Sci Statements. 19(2) 1–23.
- Pena CS, Boland GW, Hahn PF, Lee MJ, Mueller PR (2000) Characterization of indeterminate (lipid-poor) adrenal masses: use of washout characteristics at contrast-enhanced CT. *Radiology* 217(3):798–802
- Rockall AG, Babar SA, Sohaib SA, Isidori AM, Diaz-Cano S, Monson JP et al (2004) CT and MR imaging of the adrenal glands in ACTH-independent Cushing syndrome. *Radiographics* 24(2):435–452
- Sahdev A, Reznek RH, Evanson J, Grossman AB (2007) Imaging in Cushing’s syndrome. *Arq Bras Endocrinol Metabol* 51(8):1319–1328
- Song JH, Mayo-Smith WW (2011) Incidentally discovered adrenal mass. *Radiol Clin North Am* 49(2):361–368
- Terzolo M, Stigliano A, Chiodini I, Loli P, Furlani L, Arnaldi G et al (2011) AME position statement on adrenal incidentaloma. *Eur J Endocrinol* 164(6):851–870
- Verma A, Mohan S, Gupta A (2008) ACTH-independent macronodular adrenal hyperplasia: imaging findings of a rare condition: a case report. *Abdom Imaging* 33(2):225–229
- Wong KK, Arabi M, Bou-Assaly W, Marzola MC, Rubello D, Gross MD (2011) Evaluation of incidentally discovered adrenal masses with PET and PET/CT. *Eur J Radiol* Feb 3. [Epub ahead of print]

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