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Adrenal Imaging

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1.1 Introduction

Most adrenal adenomas are initially detected incidentally by computed tomography (CT), in patients who undergo the examination for other indications. But CT and magnetic resonance imaging (MRI) are also used in the investigation of adrenal hyperfunction.

Adrenal adenoma is the most common adrenal mass that is seen on cross-sectional imaging, usually CT and MRI. The majority of these lesions contain abundant lipid and can be seen on unenhanced CT as low density masses measuring less than 10 Hounsfield units [HU], and exhibit loss of signal intensity on out-of-phase (opposed phase) gradient-echo MR images. Adenomas also exhibit rapid intravenous iodinated contrast enhancement washout and therefore can be distinguished from malignant lesions which do not exhibit this feature.

CT and MRI can be used to stage adrenal cortical carcinomas and detect pheochromocytomas. FDG PET scans can help differentiate adrenal metastases from adenomas by their strong avidity for FDG, but some adenomas show mild tracer uptake.

There are several masses such as uncomplicated adrenal cysts, adrenal myelolipomas and acute adrenal hemorrhage which can be readily characterized on CT and MRI.

Utility of various imaging modalities in diagnosis of adrenal gland masses:

- Ultrasound: Ultrasound is sensitive but not specific for diagnosis adrenal masses
- Computed Tomography (CT):
 - Most commonly used modality for detection and characterization of adrenal masses
 - Measuring the unenhanced attenuation value of adrenal mass is important for diagnosing lipid rich adenoma

- Use of contrast enhancement washout values are also useful in distinguishing between adenomas and malignant lesions
- The absolute per cent enhancement washout can be calculated by measuring the enhanced attenuation, the delayed enhanced, the unenhanced values and using the following formula:

Absolute enhancement Washout

$$= \frac{\text{Enhanced attenuation value} - \text{Delayed attenuation value}}{\text{Enhanced attenuation value} - \text{Unenhanced attenuation value}}$$

- When non-contrast scans have not been obtained, and only contrast enhanced scans have been obtained, delayed images of the adrenal mass can be performed at 15 min following initial injection of intravenous contrast, and relative enhancement washout calculated as follows:

Relative enhancement Washout

$$= \frac{\text{Enhanced attenuation value} - \text{Delayed attenuation value}}{\text{Enhanced attenuation value}}$$

- Threshold values of greater 60% for absolute and 40% for relative enhancement washout have been found to be over 90% specific for adenoma diagnosis.
- Magnetic Resonance Imaging (MRI):
 - Qualitative analysis: The most important sequence of the adrenal MR imaging protocol is chemical shift imaging sequence. Chemical shift imaging is performed with in-phase and out-of-phase sequences. Loss of signal intensity of the adrenal mass using the spleen as reference organ, on out-of-phase, compared with in-phase pulse sequence is diagnostic for the presence of intracellular lipid
 - Quantitative Analysis:

$$\text{Percentage loss of signal} = \frac{\text{*SI on in-phase} - \text{SI on opposed-phase}}{\text{SI on in-phase}} \times 100$$

*SI: Signal Intensity

>16.5% loss of SI on out-of-phase images as compared with in-phase images has
>90% specificity for adenoma diagnosis.

1.2 Cushing's syndrome

The most common cause of adrenocortical steroid hormone excess is Cushing's disease due to pituitary hypersecretion of ACTH. However, primary adrenal causes are an important part of the differential diagnosis, and diagnostic plan.

Clinical features

- Facial plethora, dorsocervical fat pad, supraclavicular fat pad, truncal obesity, easy bruisability, purple striae, hirsutism, impotence or amenorrhea, muscle weakness, and psychosis.

- Hypertension.
- Hyperglycemia.
- Includes Cushing's disease (excess adrenocorticotrophic hormone [ACTH] produced by pituitary adenomas) and Cushing's syndrome (ectopic ACTH syndrome or primary adrenal disease resulting in glucocorticoid secretion independent of ACTH stimulation).
- Symptoms and Signs
 - Truncal obesity, hirsutism, moon facies, acne, buffalo hump, purple striae
 - Hypertension
 - Hyperglycemia
 - Weakness
 - Depression
 - Growth retardation or arrest in children.
- Laboratory Findings
 - Overnight, low-dose dexamethasone suppression test and measurement of urinary free cortisol establishes diagnosis
 - No suppression and elevated urinary cortisol suggest Cushing's syndrome.
 - Detection of elevated midnight cortisol level suggests Cushing's syndrome (midnight plasma level or late-night salivary cortisol sampling).
 - Once Cushing's syndrome established, measure plasma ACTH level
 - A normal or elevated ACTH level suggests pituitary adenoma or ectopic ACTH secretion.
 - Suppressed ACTH is diagnostic of hyperadrenocorticism due to primary adrenal disease.
 - If ACTH-dependent Cushing's disease and no clear pituitary lesion on MRI, may proceed to petrosal sinus sampling with corticotropin-releasing hormone (CRH) stimulation: a central to peripheral ACTH gradient suggests Cushing's disease, while no gradient suggests ectopic ACTH secretion.

Pathophysiology

- Rare forms of ACTH-independent Cushing's syndrome include macronodular hyperplasia.
- Pigmented micronodular hyperplasia is associated with the syndrome of Carney complex (also includes cardiac myxomas and lentigines).
- Rarely, ectopic adrenal tissue can be the source for excess cortisol secretion; most common location is along the abdominal aorta.
- Ectopic ACTH syndrome usually caused by small-cell lung cancers or carcinoids but can result from tumors of the pancreas, thyroid, thymus, prostate, esophagus, colon, ovaries, pheochromocytoma, and malignant melanoma.

Treatment

- Resection is best treatment for cortisol-producing adrenal tumors or ACTH-producing tumors.
- Pituitary irradiation may be necessary if pituitary surgery fails.
- Medical treatment may be indicated to control hypercortisolism, or when patients not cured by resection or when complete resection is impossible.

Imaging findings

Adrenal hyperplasia

- Often seen in patients with Cushing's syndrome and less commonly in Conn's syndrome.
- May be diffuse or nodular and is typically bilateral (Figs 1.1 and 1.2).

Adrenal adenoma

- Most are less than 3 cm in size.
- Can be of varying density on CT and MRI.
- Lipid-rich adenoma. Attenuation value of 10 HU or less at unenhanced CT (Fig. 1.3).
- Adenomas usually have absolute enhancement washout of >60% (Fig. 1.4) and relative enhancement washout of >40%.
- Greater than 16.5% loss of signal intensity on out-of-phase, compared with in-phase MRI pulse sequences (Fig. 1.5).



Figure 1.1 Bilateral adrenal cortical hyperplasia. Axial contrast-enhanced CT image shows nodular thickening of adrenal glands bilaterally in patient with Cushing's syndrome.



Figure 1.2 Bilateral adrenal cortical hyperplasia. Axial out-of-phase MR image shows nodular thickening of adrenal glands bilaterally in patient with Cushing's syndrome.



Figure 1.3 Lipid-rich adenoma. Axial unenhanced CT shows a right adrenal mass measuring 8 HU.

- Functioning and non-functioning adenomas, appear similar based on imaging as do Cushing's and Conn's adenomas.

Adrenal cortical carcinomas can also cause Cushing's syndrome (see below for imaging appearances of adrenal cortical carcinoma).

1.3 Primary hyperaldosteronism

Introduction

Primary hyperaldosteronism is a relatively common and underdiagnosed condition that contributes to hypertension in about 1% of hypertensive people. The condition is very effectively treated, and so screening programs have become routine in some places.

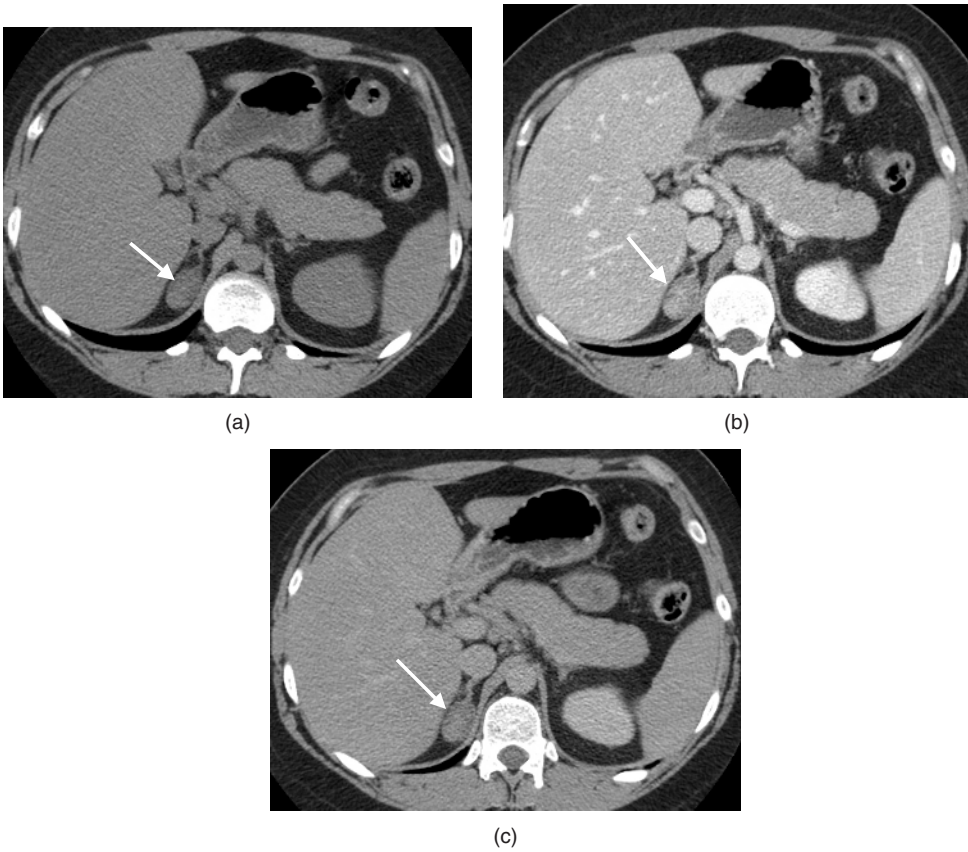


Figure 1.4 Lipid-poor adenoma. Axial unenhanced CT shows a right adrenal mass measuring 27 HU (arrow) (a). Following intravenous contrast enhancement the mass measures 96 HU (arrow) (b) and 50 HU (arrow) on delayed images (c), respectively. This mass had an absolute enhancement washout of 67%. Absolute Washout = $96 - 50 / 96 - 27 \times 100 = 67\%$.

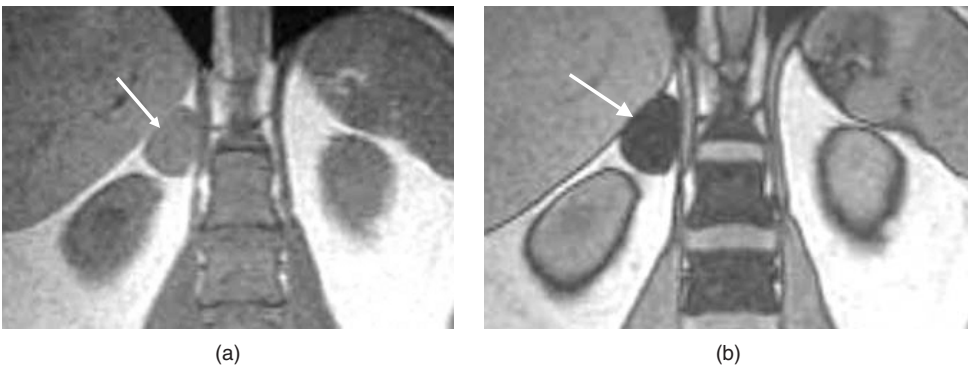


Figure 1.5 Adrenal adenoma. Coronal in-phase (a) and out-of-phase (b) MR images show an adrenal mass (arrow) which exhibits a typical decrease in signal intensity on the out-of-phase image.

Clinical features

- Hypertension with or without hypokalemia.
- Elevated aldosterone secretion and suppressed plasma renin activity.
- Metabolic alkalosis, relative hypernatremia.
- Weakness, polyuria, paresthesias, tetany, cramps due to hypokalemia.
- Common subtypes of primary hyperaldosteronism: aldosteronoma (75%) and bilateral adrenal hyperplasia (25%).
- Rare subtypes of primary hyperaldosteronism: unilateral primary adrenal hyperplasia, aldosterone-producing adrenocortical carcinoma, glucocorticoid-remediable hyperaldosteronism (familial hyperaldosteronism type 1).
- Symptoms and signs
 - Hypertension
 - Headaches
 - Malaise
 - Muscle weakness
 - Polyuria
 - Polydipsia
 - Cramps
 - Paresthesias
 - Hypokalemic paralysis (rare).
- Laboratory findings
 - Hypokalemia
 - Hypernatremia
 - Metabolic alkalosis
 - Elevated plasma aldosterone to renin ratio ($\gg$ 20)
 - Elevated plasma aldosterone concentration ($\gg$ 15 ng/dL)
 - Elevated urine/serum aldosterone level with PO or IV sodium challenge.

Treatment

- Surgical therapy for patients with aldosteronoma and unilateral primary adrenal hyperplasia.
- Medical therapy for bilateral adrenal hyperplasia, or poor surgical candidates.
- Surgery
 - Nearly always laparoscopic approach.
 - Unilaterality best defined by adrenal vein sampling for aldosterone and cortisol

- Indications

- Unilateral aldosteronoma
- Unilateral primary adrenal hyperplasia.

- Contraindications

- Bilateral adrenal hyperplasia.
- Removal of aldosteronoma normalizes potassium, but hypertension is not always cured.
- 33% of patients have persistent, mild hypertension (easier to control than before operation).
- Medications
 - Spironolactone: competitive aldosterone antagonist.
 - Amiloride: potassium-sparing diuretic.
 - Other antihypertensive agents such as ACE inhibitors and calcium channel blockers.

Imaging findings

Adrenal hyperplasia

- May be diffuse or nodular and is typically bilateral (Figs 1.1 and 1.2).

Adrenal adenoma

- Most are small and less than 2 cm in size.
- Usually much smaller than Cushing's adenoma.
- Can have varying appearances of CT and MRI.
- Lipid-rich adenoma- Attenuation value of 10 HU or less at unenhanced CT (Fig. 1.3).
- Absolute enhancement washout >60% (Fig. 1.4) and relative enhancement washout >40%.
- Greater than 16.5% loss of signal intensity on out-of-phase, compared with in-phase MRI pulse sequences (Fig. 1.5).
- Functioning and non-functioning adenomas, appear similar based on imaging as do Cushing's and Conn's adenomas.

Adrenal cortical carcinomas rarely cause Conn's syndrome.

1.4 Pheochromocytoma

Introduction

Pheochromocytomas are tumors that develop from the adrenal medulla. The hormonal function typically includes production of catecholamines, and the characteristic syndrome that follows. These tumors can be benign or malignant.

Clinical features

- Episodic headache, excessive sweating, palpitations, and visual blurring.
- Hypertension, frequently sustained, with or without paroxysms.
- Postural tachycardia and hypotension.
- Elevated urinary catecholamines or their metabolites, hypermetabolism, hyperglycemia.
- Early recognition during pregnancy is key because if left untreated, half of fetuses and nearly half of the mothers will die.
- Epidemiology.
 - Found in <0.1% of patients with hypertension.
 - 5% of tumors discovered incidentally on CT scan.
 - Most occur sporadically.
 - Associated with familial syndromes, such as:
 - Multiple endocrine neoplasia type 2A (MEN 2A)
 - MEN 2B
 - Recklinghausen disease
 - von Hippel-Lindau disease.
 - Pheochromocytomas are present in 40% of patients with MEN 2
 - 90% of patients with pheochromocytoma are hypertensive.
 - Rule of 10s:
 - 10% malignant
 - 10% familial
 - 10% bilateral
 - 10% multiple tumors
 - 10% extra-adrenal.
 - Hypertension less common in children.
 - In children, 50% of patients have multiple or extra-adrenal tumors.
- Symptoms and signs
 - Episodic or sustained hypertension.
 - Triad of palpitation, headache, and diaphoresis.
 - Anxiety, tremors.
 - Weight loss.
 - Dizziness, nausea, and vomiting.
 - Abdominal discomfort, constipation, diarrhea.
 - Visual blurring.
 - Tachycardia, postural hypotension.
 - Hypertensive retinopathy.

- Laboratory findings
 - Hyperglycemia.
 - Elevated plasma metanephrines.
 - Elevated 24-h urine metanephrines and free catecholamines.
 - Elevated urinary vanillylmandelic acid (VMA).
 - Elevated plasma catecholamines.
- Avoid arteriography or fine-needle aspiration as they can precipitate a hypertensive crisis.

Treatment

α -Adrenergic blocking agents should be started as soon as the biochemical diagnosis is established to restore blood volume, to prevent a severe crisis, and to allow recovery from the cardiomyopathy.

- β -blockade should generally be established after α -blockade, and prior to operation
- surgery
 - Indications
 - All resectable pheochromocytomas should be excised.
 - Contraindications
 - Unresectable systemic disease.
 - Inadequate medical preparation (α -blockade).
- Medications
 - α -Adrenergic blocking agents, such as phenoxybenzamine.
 - Other agents include metyrosine, prazosin, and calcium channel blockers.
 - β -Adrenergic blocking agents can be used only after full α -blockade has been achieved.
 - Avoid opioids as they stimulate histamine release.
- Prognosis
 - Operative mortality is 1–2%.
 - Mild to moderate essential hypertension may persist after surgery.
 - Treatment with ^{131}I -MIBG may help patients with metastatic or recurrent malignant pheochromocytomas.

Imaging findings

- May be homogeneous or heterogeneous, solid or cystic complex masses.
- May show calcification.
- Smaller tumors tend to have a more uniform attenuation.

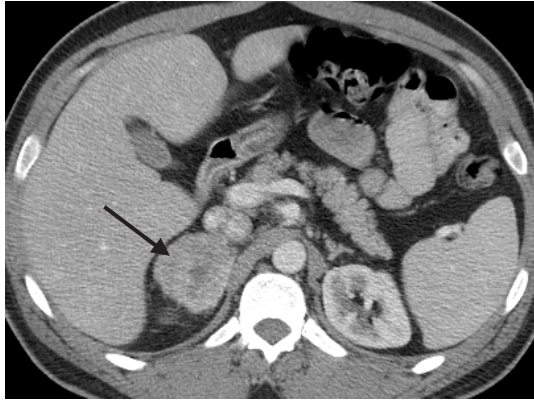


Figure 1.6 Adrenal pheochromocytoma. Axial contrast enhanced CT shows a heterogeneously enhancing right adrenal mass (arrow).

- Typically enhance avidly with intravenous contrast administration but can be heterogeneous (Fig. 1.6).
- Most have an absolute enhancement washout of less than 60%, and a relative enhancement of less than 40%, but washout features are variable.
- Show less than 16.5% loss of signal intensity on out-of-phase, compared with in-phase MRI pulse sequences.
- The classic ‘light bulb bright’ signal on T2-weighted images is infrequently seen.
- Pheochromocytomas occur in association with various syndromes such as; multiple endocrine neoplasias (MEN2), Von Recklinghausen neurofibromatosis (NF1), and Von Hippel-Lindau disease (VHL) (Fig. 1.7).

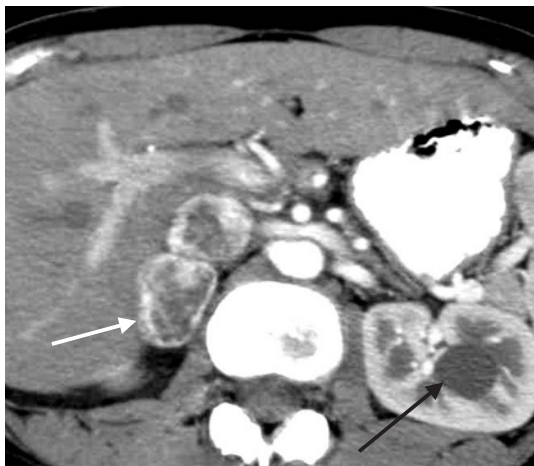


Figure 1.7 Adrenal pheochromocytoma. Axial contrast enhanced CT shows a heterogeneously enhancing right adrenal mass (arrow). Also note visualization of a left renal cyst (arrow) in this patient with Von Hippel Lindau disease.

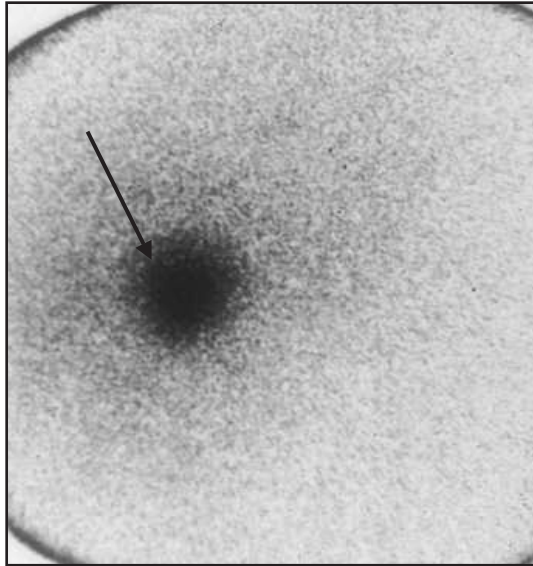


Figure 1.8 Adrenal pheochromocytoma. Radionuclide MIBG scan shows increased tracer uptake in the region of a right adrenal mass.

- Pheochromocytomas usually exhibit high uptake on MIBG scintigraphic examination (Fig. 1.8).

1.5 Adrenal cortical carcinoma

Introduction

Adrenocortical carcinoma is typically an aggressive malignancy with a poor prognosis, though less aggressive forms do occur. The tumors can present either due to hormone production, or due to mass effect from the primary or metastatic lesions.

Clinical features

- Variety of clinical symptoms through excess production of adrenal hormones.
- Complete surgical removal of the primary lesion and symptomatic metastatic sites, if possible, has been the mainstay of treatment.
- Epidemiology
 - These tumors are rare; 1–2 cases per million persons in the USA.
 - Less than 0.05% of newly diagnosed cancers per year.
 - Bimodal occurrence, with tumors developing in children less than 5 years of age and in adults in their fifth through seventh decade of life.
 - Male:female ratio is 2:1, with functional tumors being more common in women.

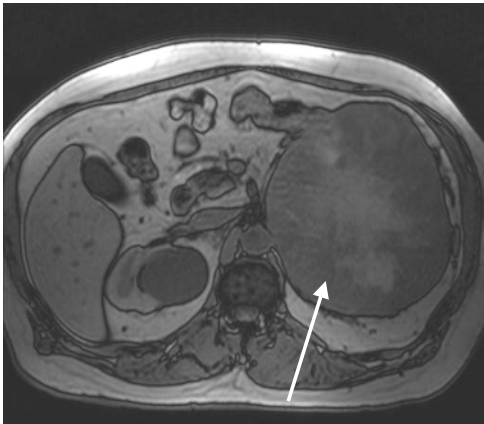
- Left adrenal involved slightly more often than the right (53% vs. 47%); bilateral tumors are rare (2%).
- 50–60% of patients have symptoms related to hypersecretion of hormones (most commonly Cushing's syndrome and virilization).
- Feminizing and purely aldosterone-secreting carcinomas are more rare.
- 50% of patients have metastases at the time of diagnosis.
- Symptoms and signs
 - Symptoms of specific hormone excess (cortisol excess, virilization, feminization).
 - Palpable abdominal mass.
 - Abdominal pain.
 - Fatigue, weight loss, fever, hematuria.
- Laboratory findings
 - All laboratory abnormalities depend on hormonal status of tumor.
 - Elevated urinary free cortisol or steroid precursors.
 - Loss of normal circadian rhythm for serum cortisol.
 - Low serum adrenocorticotrophic hormone (ACTH).
 - Abnormal dexamethasone suppression test.
 - Elevated serum testosterone, estradiol, or aldosterone levels.

Treatment

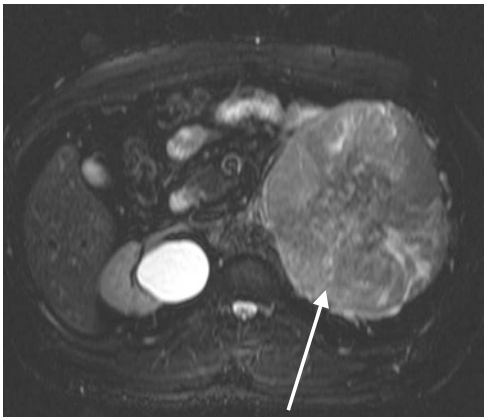
- Surgery is the only treatment that can cure or prolong survival.
- Laparoscopic surgery not recommended because of spread of tumor, fragility of tumor, and the possible need to resect adjacent involved organs.
- For local recurrent disease, reoperation is the only effective therapy and may prolong life.
- Surgery
 - Indications
 - Disease localized to the adrenal, or local spread.
 - Contraindications
 - Widely metastatic disease.
- Medications
 - Mitotane (an adrenolytic agent) can be used as adjuvant therapy; controls endocrine symptoms in 50% of patients but does not generally affect survival.
- Prognosis
 - Tumor stage at the initial operation predicts survival.
 - Median survival is 25 months.
 - 5-year actuarial survival is 25%.
 - 5-year survival with grossly complete surgical resection is 50%.



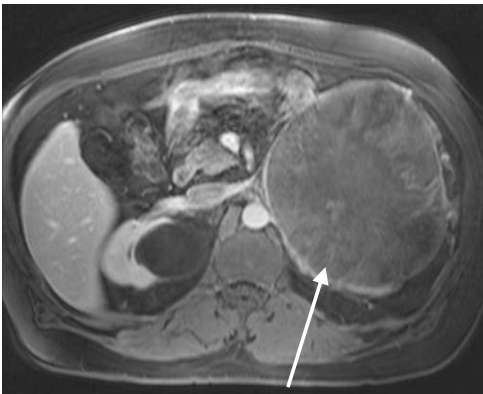
(a)



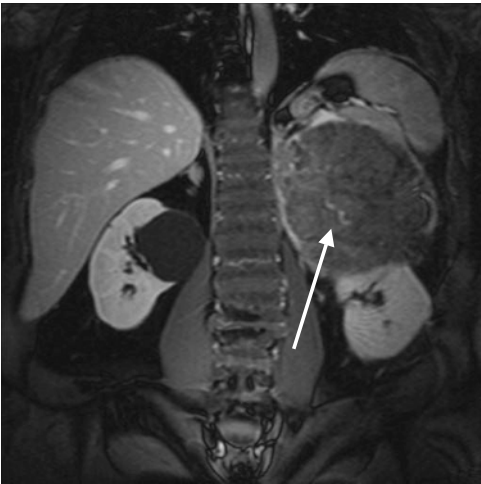
(b)



(c)



(d)



(e)

Imaging findings

- Usually large tumors at diagnosis, most larger than 6 cm.
- Heterogeneous on CT and MRI, owing to the presence of internal hemorrhage, calcification and necrosis (Fig. 1.9).
- Large tumors tend to invade the adrenal vein and inferior vena cava.

1.6 Adrenal incidentaloma

Introduction

Incidentally identified adrenal tumors found on studies performed for other indications than symptomatic adrenal disease are common. Evaluation of patients who have these tumors can depend on the age of the patient and the size of the tumor. Although adrenal cortical adenomas are the most common lesions that present as incidentalomas, a wide variety of benign and malignant masses as well as non-hyperfunctioning and subclinical hormonally active masses can present in this manner.

Clinical features

- Incidence of diagnosis has increased with use of ultrasonography, CT, and MRI for various, non-related conditions of the abdomen.
- Diagnosis includes such conditions as non-functioning adrenocortical adenoma, functioning adenoma, pheochromocytomas with subclinical secretion of hormones, and adrenocortical carcinomas.
- Major issues are to determine whether the tumor is hormonally active, or a carcinoma, or neither.
- Most simple adrenal cysts, myelolipomas, and adrenal hemorrhages can be identified by the imaging characteristics alone.
- Adrenal cysts can be very large.
- Since most tumors are non-functioning adenomas, the work-up should avoid unnecessary procedures and expense.
- Non-functioning adrenal tumors that are greater than 5 cm have a higher risk of cancer.
- An adrenal mass ≥ 3 cm in a patient with a previously treated malignancy is very likely a metastasis.

Figure 1.9 Adrenal cortical carcinoma. (a) Axial contrast enhanced CT shows a large heterogeneously enhancing left adrenal mass containing calcification (black arrow) and low attenuation area of necrosis (white arrow). (b-e) MR images in a different case of adrenal carcinoma shows heterogeneous regions (arrows) prior to and following intravenous gadolinium administration due to degeneration and necrosis.

- Primary tumors that metastasize to the adrenal gland include: lung, breast, colon, renal cell carcinoma, malignant melanoma, uterine, and prostate.
- Epidemiology
 - Found in 1–4% of CT scans.
 - Found in 6% of random autopsies.
 - Incidence increases with age.
 - Over 80% are non-functioning cortical adenomas.
 - 5% each are preclinical Cushing's syndrome, pheochromocytoma, and adrenocortical carcinoma.
 - 2% are metastatic carcinoma.
 - 1% are aldosteronoma.
 - 25% of pheochromocytomas are found incidentally.

Workup and treatment

- Complete history and physical exam, with specific reference to previous malignancies, symptoms of Cushing's syndrome, hypertension, virilization, or feminization.
- All patients, even those without hypertension, should have plasma metanephrines and 24-h urinary fractionated catecholamines determined to evaluate for pheochromocytoma.
- All patients should have a serum cortisol, 24-h urine collection for cortisol, and an overnight dexamethasone suppression test.
- Patients who are hypertensive should have serum potassium, and plasma aldosterone and renin activity measured.
- Consider obtaining a dehydroepiandrosterone (DHEA) level (potential marker for adrenocortical carcinoma).
- If above studies show the tumor to be non-functional, the size of the tumor and the patient's overall medical condition determine management.
- If metastasis is suspected and pheochromocytoma is ruled out, then CT-guided biopsy is useful.

Imaging findings

Most commonly these are 'non-functioning' adrenal cortical adenomas, but functioning tumors such as adrenal cortical carcinoma and pheochromocytoma may also present as 'incidental' adrenal masses.

Adrenal adenoma

- Most are less than 3 cm in size.
- Can have varying appearances on CT.

- Lipid-rich adenoma. Attenuation value of 10 HU or less on unenhanced CT.
- Absolute enhancement washout $>60\%$ and relative enhancement washout $>40\%$.
- Greater than 16.5% loss of signal intensity on out-of-phase, compared with in-phase MRI pulse sequences.

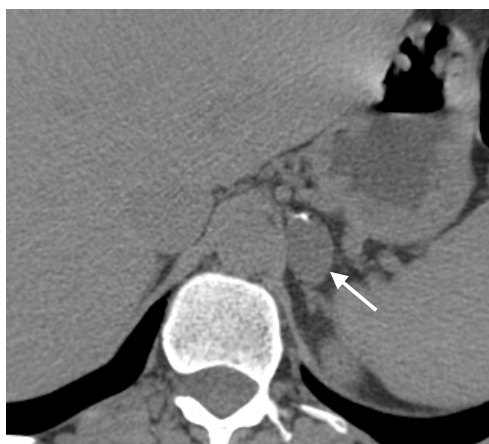
Other masses include the following:

Adrenal cyst

- Can have features of simple cyst such as attenuation of less than 20 HU on unenhanced image and no enhancement following intravenous contrast administration (Fig. 1.10).
- Hypointense on T1-weighted images and hyperintense on T2-weighted images, with no soft-tissue component and no internal enhancement (Fig. 1.11).
- Pseudocysts typically arise after an episode of adrenal hemorrhage or trauma.
- Adrenal pseudocysts may have a complicated appearance on CT and MR images, with septations, blood products, soft-tissue component secondary to hemorrhage or hyalinized thrombus, and curvilinear calcification (Fig. 1.12). Pseudocysts can be indistinguishable from malignant tumors.

Adrenal myelolipoma

- Benign tumor composed of marrow elements such as mature adipose tissue (fat), hematopoietic tissue and calcification/ossification.
- Fat can be diagnosed by the presence of areas of negative attenuation value by CT (Fig. 1.13) and on MR, by suppression of signal intensity on fat suppressed images, when compared with non-fat suppressed images.



(a)



(b)

Figure 1.10 Adrenal cyst. Axial unenhanced CT shows a left adrenal mass measuring 18 HU (arrow) (a). Following intravenous contrast enhancement the mass did not show significant enhancement (arrow) (b).

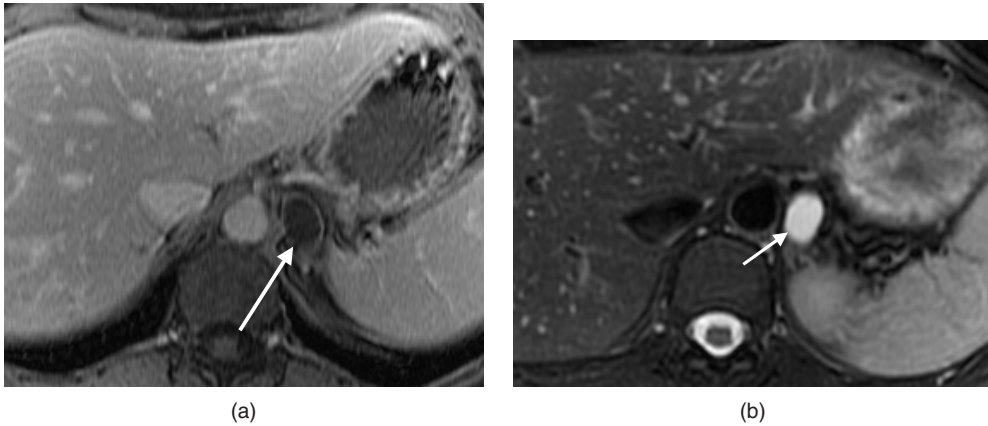


Figure 1.11 Adrenal cyst. Axial contrast enhanced T1 weighted (a), and axial fat suppressed T2 weighted (b) MR show a left adrenal mass exhibiting a hypointense signal on T1-weighted images (arrow) and hyperintense signal on T2-weighted images (arrow) with no soft-tissue component and no internal enhancement.



Figure 1.12 Adrenal pseudocyst. Axial contrast enhanced CT shows a right calcified adrenal mass measuring 19 HU.

Adrenal hemorrhage

- Usually bilateral.
- Can be seen in post-operative states, trauma, sepsis and in patients with blood dyscrasias or receiving anticoagulant therapy.
- If unilateral usually due to trauma or following liver transplantation (right sided)
- High density on unenhanced images (Fig. 1.14).
- Appearance overlaps that of other lesions following contrast enhancement.



Figure 1.13 Adrenal myelolipoma. Axial contrast enhanced CT shows a left adrenal mass with an intralesional area of fat indicated by a negative attenuation value of -28 HU (arrow). Note that qualitatively this area is of similar attenuation to the adjacent intra-abdominal fat.

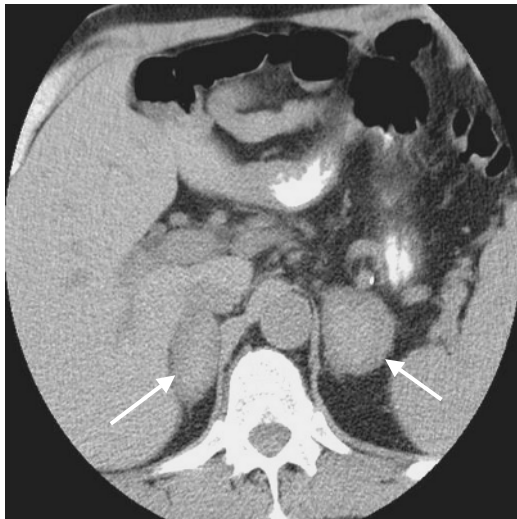


Figure 1.14 Bilateral adrenal hemorrhage. Unenhanced CT shows bilateral adrenal masses (arrows) with high attenuation.

Metastases (Fig. 1.15)

- Common primary tumors include the lung, breast, GI tract and pancreas.
- Can be of varying size.
- Attenuation higher than 10 HU on unenhanced CT, as they do not typically contain intracellular lipid.



Figure 1.15 Adrenal metastases. Contrast-enhanced CT shows diffuse hepatic and bilateral adrenal metastases (arrows) in patient with advanced melanoma.

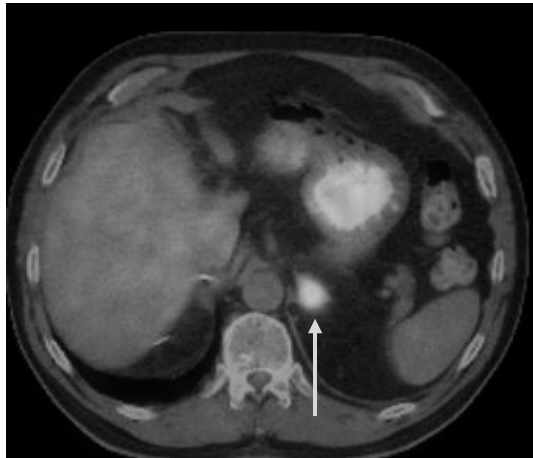


Figure 1.16 Adrenal metastasis. Fused PET/CT scan shows marked tracer uptake in a small left adrenal mass (arrow).

- Absolute enhancement washout of less than 60%, and a relative enhancement of less than 40%.
- Usually heterogeneous.
- Less than 16.5% loss of signal intensity on out-of-phase, compared with in-phase MRI pulse sequences, except in metastatic clear cell renal carcinoma which may contain foci of lipid.
- High tracer activity on FDG PET scans usually differentiates malignancy (metastasis or adrenal cortical carcinoma) from adenoma. Comparison to liver uptake is often useful for this assessment (Fig. 1.16).