Urogenital Imaging

A Problem-Oriented Approach

Editors

Sameh K. Morcos

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and

Henrik S. Thomsen

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To the memory of my late parents Kamel and Monira who gave me so much unconditionally.

To my beloved wife and daughters Sandra, Sarah, Hannah and Rebecca whose tremendous love and support I will always cherish.

Sameh K. Morcos, Sheffield, UK

Contents

Foreword

This book has the unique aim to provide the greatest utility to the modern radiologist practicing diagnostic imaging of the genitourinary system. In this time of great pressure within the medical environment, where time is always of the essence, one can even question the usefulness of any book. Many practitioners quickly go the internet for a "google search". Unfortunately, although the internet can provide extremely valuable information quickly, it also can provide an enormous amount of misinformation quickly. Unless one knows and trusts the source, the internet although readily available may lead to uncertainty. In addition, the internet often provides "factoids" bereft of context.

This book is designed for the busy modern world, by providing key information representing a consensus of current practice in a very accessible fashion. The nineteen chapters cover, in moderate depth, all the most important topics relating to the male and female genital and urinary tracts. The forty three authors are truly an international group of experts in the field. Each chapter provides information on usual presentation of disorders, clinical, laboratory and pathologic features and the best current information on imaging features critical for diagnosis. A brief presentation on current therapy is also included.

A critical decision was made in the preparation of this book as to the style of presentation of the information. Elegant phraseology has been bypassed in favor of a "bullet point" approach, along with heavy use of tables and extensive illustration of typical features of common lesions. This results in an "information dense" text. Despite the relative brevity of the chapters, each is packed with useful information. That information can be accessed by the reader extremely quickly – whether searching for a single key point, such as how to calculate relative washout of an adrenal mass and the best diagnostic cutoffs, or whether desirous of a quick overview of a topic. This book in fact would be an excellent choice for someone reviewing for a radiology exam such as ABR MOC. The authors have concentrated in each chapter on accepted key diagnostic features; the character of the authors makes this truly an international consensus.

What one will not find in the book is extensive discussion of the history of imaging of any area. It does not include the initial description of some sign, or the early attempts for criteria that failed. There is not an extensive list of references, nor a discussion of controversial areas. Rare entities or extremely unusual presentations of common lesions are not included in general. Rather the focus is on well accepted key points on clinical aspects of the more common diseases and the accepted findings that allow one to make an imaging diagnosis. Some presentation, although limited, is given of imaging techniques. This is not meant to teach in detail how to perform exams correctly. The heavy emphasis in most chapters is on Computed Tomography (CT) and Magnetic Resonance Imaging (MRI), as those methods currently are most useful for final diagnosis in most areas. However, in appropriate areas,

such as kidney transplant, prostate disease, female pelvis and male scrotum, ultrasound is well covered. As it has been largely superseded, little attention is paid to intravenous urography or to most radiologic methods. This is not a text that extensively reviews nuclear medicine techniques.

I am proud to have been a contributor to this text. I expect many radiologists will find it a most useful guide to the best of current practice of imaging diagnosis of the genitourinary tract. The size of the book is designed such that it can be kept close at hand for easy and frequent use.

Professor Philip J. Kenney

Preface

This book is designed to offer both radiologists and clinicians focused information about different important aspects of the urogenital system. Each chapter provides concise information about clinical features, pathology and imaging findings of the aspect under consideration.

The book does not follow the traditional style of scientific textbooks. The text is presented mainly in bullet format which the reader will find easy to follow and remember. Many images demonstrating important diagnostic features of different diseases are provided in each chapter. A list of key references is provided at the end of each chapter. A diagnostic algorithm is also provided whenever appropriate.

The book is aimed mainly at urologists, nephrologists, gynecologists, general radiologists and trainee radiologists. Specialized urogenital radiologists will also find the book a quick reminder of important features of different conditions in their field of interest.

We are most grateful to all the authors who covered their topics expertly and clearly. We are fortunate to have so many eminent colleagues contributing to this book.

We hope you will find the book informative and a good reference to refresh your knowledge in the field of urogenital imaging.

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1
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

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- 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:

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

- 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:

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

- 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 bruisabilility, purple striae, hirsutism, impotence or amenorrhea, muscle weakness, and psychosis.

- Hypertension.
- Hyperglycemia.
- Includes Cushing's disease (excess adrenocorticotropic 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).

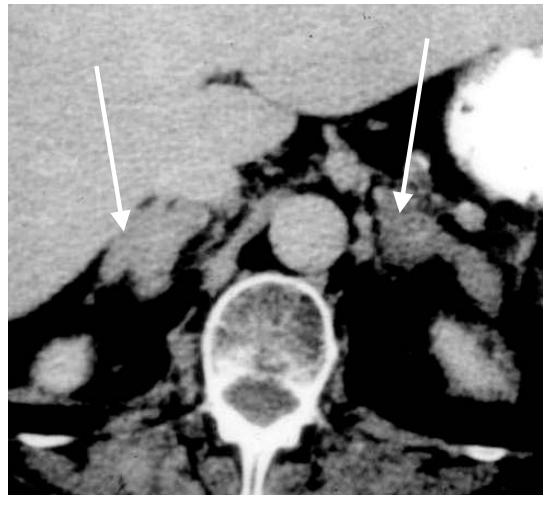


Figure 1.1 Bilateral adrenal cortical hyperplasia. Axial contrast-enhanced CT image shows nodular thickening of adrenal glands bilaterally in patient with Cushing's syndrome.

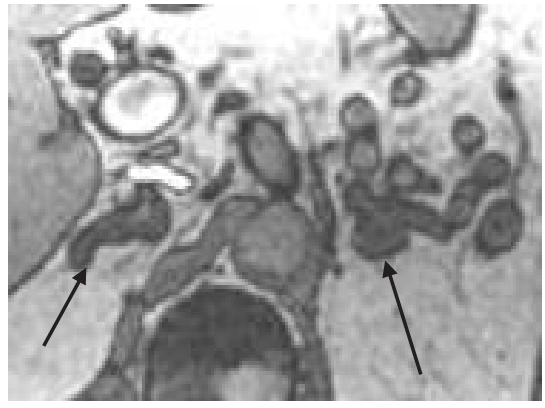


Figure 1.2 Bilateral adrenal cortical hyperplasia. Axial out-of-phase MR image shows nodular thickening of adrenal glands bilaterally in patient with Cushing's syndrome.

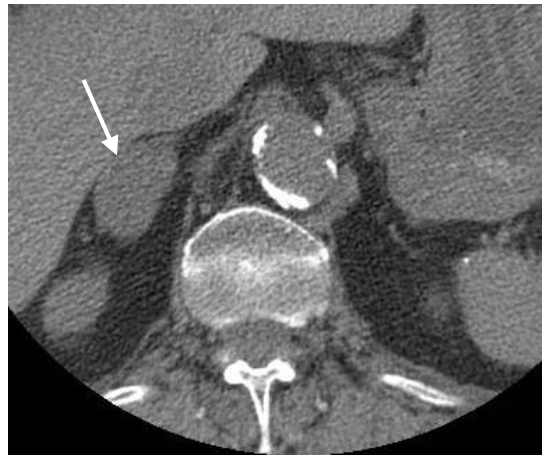


Figure 1.3 Lipid-rich adenoma. Axial unenhanced CT shows a right adrenal mass measuring 8 HU.

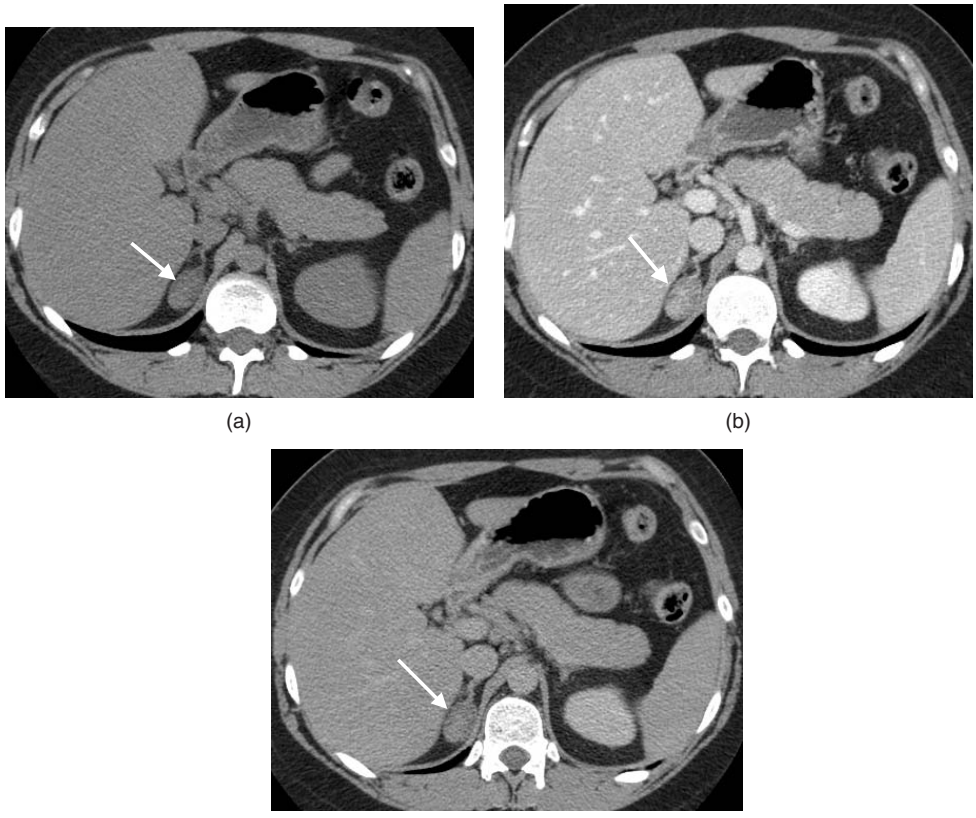
- Functioning and non-functioning adenomas, appear similar based on imaging as do Cushing's and Conn's adenomas.

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

1.3 Primary hyperaldosteronism

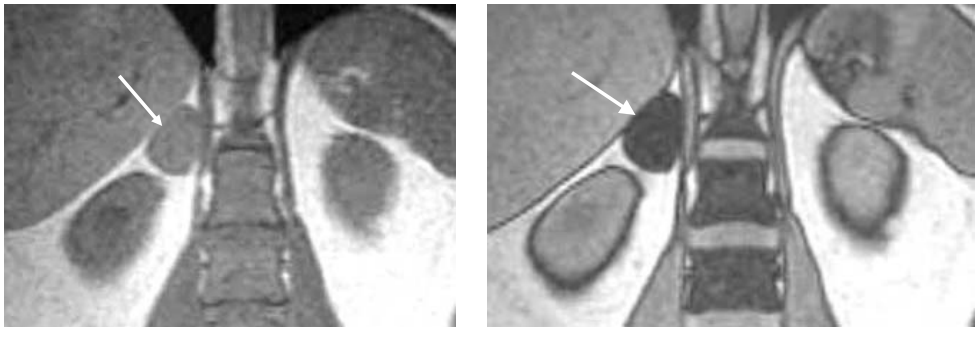
Introduction

Primary hyperaldostronism 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.



(a) (b) (c)

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 $= 96 - 50/96 - 27 \times 100 = 67\%$.



(a) (b)

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.