



Fig. 17.14 The systemic features of Cushing's syndrome.

Secondary hyperaldosteronism. When renal glomerular function is reduced, for example through a fall in blood volume, the renin-angiotensin system (Fig. 17.12) stimulates aldosterone secretion from the zona glomerulosa in an attempt to correct this. This physiological response is known as secondary hyperaldosteronism, which is by far the commonest form of hyperaldosteronism.

Diagnosis. The diagnosis of primary hyperaldosteronism rests on two criteria: plasma aldosterone must be raised while renin is low. This is to distinguish it from secondary hyperaldosteronism, in which aldosterone levels are raised but are an appropriate response to high renin levels.

Hypersecretion of sex steroids

Some adrenal cortical adenomas secrete sex steroids, most commonly androgens. In Cushing's syndrome, quantities of androgens are occasionally secreted along with the glucocorticoids, causing virilisation of females, especially those with adrenocortical carcinomas. Early congenital enzyme defects of the pathways of steroid synthesis may result in excess production of sex steroids. The most rare example is 'congenital adrenal hyperplasia' due to deficiency of the enzyme 21-hydroxylase, needed for the synthesis of both cortisol and aldosterone (Fig. 17.15).

Failure of cortisol production leads to increased ACTH secretion, resulting in hyperplasia of the adrenal cortex. The production of androgens occurs before the metabolic block caused by the enzyme deficiency, and their excessive secretion results in masculinisation of females and precocious puberty in males. 21-Hydroxylase deficiency is serious because deficiency of mineralocorticoids causes life-threatening salt loss unless replacement therapy is given.

Tumours

Adenoma. In addition to those 'functioning' adrenal cortical adenomas that present by causing Cushing's or Conn's syndromes, a clinically unsuspected 'non-functioning' adenoma occurs in about 2% of adults. The adenoma is a pale yellow circumscribed nodule, perhaps 20-30 mm in diameter (Fig. 17.16). The cells have clear cytoplasm owing to their high lipid content (Fig. 17.17).

Carcinoma. Adrenal cortical carcinoma is rare; these tumours are usually hormone-secreting, with a tendency to produce androgens. They are commonly large (over 100 g) and exhibit invasive growth. Examination of the adjacent adrenal cortex and that of the opposite gland may give a clue as to the function of the neoplasm; glucocorticoid-secreting tumours will suppress ACTH, resulting in atrophy of the non-neoplastic adrenal cortex.

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Table of Contents General And Systemic Pathology 5th Edition

1. Understanding the eBook General And Systemic Pathology 5th Edition
 - The Rise of Digital Reading General And Systemic Pathology 5th Edition
 - Advantages of eBooks Over Traditional Books
2. Identifying General And Systemic Pathology 5th Edition
 - Exploring Different Genres
 - Considering Fiction vs. Non-Fiction
 - Determining Your Reading Goals
3. Choosing the Right eBook Platform
 - Popular eBook Platforms
 - Features to Look for in an General And Systemic Pathology 5th Edition
 - User-Friendly Interface
4. Exploring eBook Recommendations from General And Systemic Pathology 5th Edition
 - Personalized Recommendations
 - General And Systemic Pathology 5th Edition User Reviews and Ratings
 - General And Systemic Pathology 5th Edition and Bestseller Lists

5. Accessing General And Systemic Pathology 5th Edition Free and Paid eBooks
 - General And Systemic Pathology 5th Edition Public Domain eBooks
 - General And Systemic Pathology 5th Edition eBook Subscription Services
 - General And Systemic Pathology 5th Edition Budget-Friendly Options
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 - ePub, PDF, MOBI, and More
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 - General And Systemic Pathology 5th Edition Enhanced eBook Features
7. Enhancing Your Reading Experience
 - Adjustable Fonts and Text Sizes of General And Systemic Pathology 5th Edition
 - Highlighting and Note-Taking General And Systemic Pathology 5th Edition
 - Interactive Elements General And Systemic Pathology 5th Edition
8. Staying Engaged with General And Systemic Pathology 5th Edition
 - Joining Online Reading Communities
 - Participating in Virtual Book Clubs
 - Following Authors and Publishers General And Systemic Pathology 5th Edition
9. Balancing eBooks and Physical Books General And Systemic Pathology 5th Edition
 - Benefits of a Digital Library
 - Creating a Diverse Reading Collection General And Systemic Pathology 5th Edition
10. Overcoming Reading Challenges
 - Dealing with Digital Eye Strain
 - Minimizing Distractions
 - Managing Screen Time
11. Cultivating a Reading Routine General And Systemic Pathology 5th Edition
 - Setting Reading Goals General And Systemic Pathology 5th Edition
 - Carving Out Dedicated Reading Time
12. Sourcing Reliable Information of General And Systemic Pathology 5th Edition
 - Fact-Checking eBook Content of General And Systemic Pathology 5th Edition
 - Distinguishing Credible Sources
13. Promoting Lifelong Learning

- Utilizing eBooks for Skill Development
 - Exploring Educational eBooks
14. Embracing eBook Trends
- Integration of Multimedia Elements
 - Interactive and Gamified eBooks

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