BACTERIAL INFECTIONS IN ENDOCRINOLOGY

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ABSTRACT

Bacteria are microscopic organisms that are ubiquitous in the environment and human body. Some bacteria exhibit symbiotic relationship with the human body, while other bacteria are harmful and cause various diseases. Bacteria may infect the endocrine glands either by direct invasion or local or hematogenous spread. Suppurative bacterial infections can involve the pituitary, thyroid, adrenals, and gonads. In the majority of cases, specific risk factors predispose the endocrine glands to such infections. This in turn may lead to temporary or permanent endocrine dysfunction. There may also be states of hormone excess following bacterial infections. This is particularly noted in cases of bacterial thyroiditis. Permanent endocrine dysfunction following bacterial infections will warrant life-long hormone replacement therapy. In acute stages of infection, intravenous or oral antibiotics are the cornerstone of management. The choice of antibiotic is guided by culture and sensitivity report. Sometimes, however, empirical antibiotic therapy may need to be continued as no organism may be isolated on culture. Empirical therapy should provide coverage for gram positive, gram negative, and anaerobic bacteria. If there is abscess formation in any endocrine gland, it may require aspiration and drainage. In this chapter, we have discussed the risk factors, bacteriology, clinical presentation, diagnosis, and management of common bacterial infections involving endocrine glands.

INTRODUCTION

The incidence of bacterial infections of endocrine glands is low when compared to that in other organs of the body. The endocrine glands that may be affected by bacterial infections are: pituitary, thyroid, adrenals and gonads. Bacterial infection of parathyroid glands is extremely rare. Certain risk factors may predispose the glands for infection.

In general, bacteria may be classified as gram positive, gram negative, and miscellaneous categories. The classification of medically important bacteria is highlighted in another chapter of the Endotext (1). Among all the bacteria, *Mycobacterium tuberculosis* remains the most common agent involving the endocrine glands (2). *Mycobacterium tuberculosis* is a weakly gram positive highly aerobic bacterium that can cause tuberculosis in any organ of the body. This organism can affect the adrenal glands and lead to primary adrenal insufficiency. In developing countries, tuberculosis remains the most common cause of primary adrenal insufficiency.

Tuberculosis can also affect pituitary, thyroid, and gonads. In this chapter, we are discussing only adrenal tuberculosis, since tuberculosis of the Endocrine system has been covered in great details in another chapter (3). Apart from *Mycobacterium tuberculosis,* the other common bacteria that may affect the endocrine system are *Staphylococcus aureus, Streptococcus pneumoniae, Neisseria meningitides, Escherichia coli, Chlamydia trachomatis, Pseudomonas aeruginosa, Klebsiella pneumoniae, Treponema pallidum,* and *Yersinia enterocolitica* among others. We have tried to present a spectrum of bacterial infections of various endocrine glands including their clinical presentation, investigations, management, long-term prognosis, and follow up.

BACTERIAL INFECTIONS OF PITUITARY

Infections of the pituitary gland are rare but may cause clinical problems because of the non-specific nature of the presentation (4). Among the various infectious agents, bacterial infections including Mycobacterial infections seem to be the most common. The various bacterial agents causing infection of the pituitary gland are summarized in the table 1. The common bacterial infections of the pituitary gland are described below.

Table 1. Bacterial Agents Causing Infection of Pituitary-Hypothalamus

Bacterial class	Organism
Gram positive bacteria	*Staphylococcus aureus, Streptococcus pneumoniae*
Gram negative bacteria	*E coli, Pseudomonas aeruginosa, Neisseria meningitides*
Spirochaete	*Treponema pallidum*
Mycobacterium	*Mycobacterium tuberculosis*

Pituitary Abscess

EPIDEMIOLOGY AND RISK FACTORS

Pituitary abscesses are a very rare clinical entity and account for less than 1% of pituitary lesions (4). The first case of pituitary surgery involving an abscess was described in 1848. Since then, there have been around 300 such cases reported in the literature (4, 5). Risk factors include underlying pituitary diseases such as a pituitary adenoma, Rathke's cyst, craniopharyngioma, lymphocytic hypophysitis, immunocompromised states (uncontrolled diabetes mellitus, tuberculosis, HIV infection, after solid organ transplantation, chemotherapy, radiotherapy), history of surgical exploration in pituitary hypothalamic region, and spread of local infection from meninges and paranasal sinuses (5-7). Rarely, abscess may develop in a normal pituitary gland (6, 8).

BACTERIOLOGY

In the majority of cases, culture is negative in pituitary abscess, with only 19.7% cases showing growth of bacteria (9). The most common organisms isolated are *Streptococci* and *Staphylococci.* Other bacterial organisms are *Escherichia coli, Mycobacteria, Neisseria,* and anaerobes (6, 10). As culture is negative in most of the cases, it is important for empirical antibiotic therapy to cover gram positive, gram negative and anaerobic bacteria. Rarely, a fungal etiology is seen.

CLINICAL PRESENTATION

Clinical presentation can be classified with respect to chronicity as: acute (within days to weeks), subacute (less than a month) and chronic (more than a month). Acute and subacute abscesses have fulminant presentation while chronic abscess has a more indolent course (5). In the initial stages, patients present with headache (67%), fever, meningismus, and malaise. With progression of the disease, neurological symptoms like altered sensorium, seizures, and coma can occur.

Extension of infection in nearby areas can lead to visual dysfunction (45%), extra ocular movement defects, and other cranial nerve palsies (4, 8, 9, 11).

Both anterior and posterior pituitary hormonal hypofunction can be seen with a pituitary abscess. In the largest series of pituitary abscesses with 60 cases over 23 years, anterior pituitary hormone deficiencies were reported in 81.8% patients whereas diabetes insipidus was reported in 47.9% of the patients. In the same study, 9.3% had isolated hypogonadism, 3.7% had isolated ACTH deficiency, 1.8% had isolated hypothyroidism, and 1.8% hypothyroidism and ACTH deficiency (9).

DIAGNOSIS

The investigation of choice for the diagnosis of pituitary abscess is MRI (Magnetic Resonance Imaging) with proper sellar cuts. On T1 weighted images, pituitary abscess appears iso-intense to hypo-intense while on T2 weighted images, it is iso-intense to hyper-intense. There is a characteristic rim of enhancement after gadolinium injection around the abscess site (9, 11). Diffusion-weighted imaging (DWI) shows high signal intensity with a decrease in the apparent diffusion coefficient in the region of pus collection (9, 11).

MANAGEMENT

Trans-nasal trans-sphenoidal surgery and drainage of the abscess is the treatment of choice. The sphenoid sinus may require exploration if extrasellar invasion is suspected. Along with surgical exploration, the patient should be started on intravenous antibiotics empirically with ceftriaxone (alternatives are cefotaxime and cefepime) along with metronidazole for anaerobic coverage. In case of suspicion for *Staphylococcus aureus*, vancomycin should be added (9, 12). Further intensification or alteration of antibiotics is subjected to clinical improvement and culture and sensitivity reports. Microbiological etiology may not be identified in the majority of cases. Hence, it is imperative to give proper broad-spectrum coverage empirically.

PROGNOSIS AND FOLLOW-UP

With current standard of care, mortality rate is 10 % and chance of recurrence is <13%. In about 25% of cases hormonal recovery occurs. After recovering from a pituitary abscess, these patients should be followed up by serial MRI at 3, 6 and 12 months (12). Monitoring for anterior and posterior pituitary hormone deficiency should be done in any patient with a pituitary abscess. Replacement with corticosteroid, thyroid, gonadal, and growth hormone therapy may be required if the patient develops deficiency of any of these hormones. Replacement with vasopressin therapy may be required if patient develops central diabetes insipidus following a pituitary abscess.

Hypopituitarism Caused by Treponema Pallidum Infection

Syphilis caused by *Treponema pallidum* (a spirochete) may involve the pituitary- hypothalamic region causing syphilitic gumma with non-caseating granulomas (13, 14). It is more common in patients with underlying human immune deficiency virus (HIV) infection. Diagnosis can be made by demonstration of the spirochete in the samples of sellar tissues following

trans sphenoidal surgery. Immunological diagnosis can be made by measuring titers of anti-Treponemal antibody in the serum. Treatment consists of intravenous followed by oral antibiotics (13-15). Penicillin is the drug of choice for syphilis. In patients who are allergic to penicillin, doxycycline is a good alternative.

BACTERIAL INFECTIONS OF THE THYROID

It is rare for bacteria to invade the normal thyroid gland because of the rich vascular supply, good lymphatic drainage, separation of thyroid gland from other structures by fascial planes, high iodine content, and production of hydrogen peroxide inside the gland (16). Both iodine and hydrogen peroxide have bactericidal properties.

Acute Suppurative Thyroiditis

EPIDEMIOLOGY AND RISK FACTORS

Acute suppurative thyroiditis is rare and is usually due to bacterial infection of the thyroid gland. In severe cases, it can lead to abscess formation and spread to surrounding structures leading to acute obstruction of the respiratory tract. More than 90% of the patients are less than 40 years of age, with females being more commonly affected than males (17, 18). The incidence of acute suppurative thyroiditis lies between 0.1% and 0.7% of all thyroidal illnesses (19). In children acute suppurative thyroiditis is usually due to persistent pyriform sinus and almost always affects the left lobe and is often recurrent (20-22). Risk factors for acute suppurative thyroiditis are summarized in table 2 (23).

Table 2. Risk Factors for Acute Suppurative Thyroiditis
Common risk factors
Pyriform sinus fistula – more common in children and young adults and associated with recurrent disease
Immunocompromised status – AIDS, blood malignancies, uncontrolled diabetes (more common risk factor overall)
Other risk factors
Thyroglossal cyst
Patent foramen cecum
Congenital brachial fistula
Spread of adjacent suppurative infection into thyroid
Anterior esophageal perforation
Underlying thyroid disorders like chronic autoimmune thyroiditis, goiter, and thyroid malignancy
Fine need aspirations/biopsy of thyroid
Dental abscess/ treatment
Systemic autoimmune disorders

BACTERIOLOGY

Although bacterial agents account for the majority of cases, acute suppurative thyroiditis can also be caused by fungal (immunosuppressive status), parasitic, and tubercular etiology. Common bacterial organisms include *Staphylococcus aureus, Streptococcus pyogenes, Staphylococcus epidermidis,* and *Streptococcus pneumoniae.* Rarely other causative bacteria include *Klebsiella* species, *Hemophilus influenzae, Streptococcus viridans, Arcanobacterium haemolyticum, Eikenella corrodens*, *Salmonella* species, and *Enterobacteriaceae*. In the context of immunosuppressed states like HIV-AIDS, acute suppurative thyroiditis can be caused by *Mycobacterium tuberculosis,* atypical mycobacteria*, Salmonella* species*, Nocardia* species and *Treponema pallidum (19, 24).*

CLINICAL PRESENTATION

Acute suppurative thyroiditis due to bacterial etiology has a very rapid onset and progression of symptoms if not addressed. The common manifestations are fever, neck pain, and dysphagia. Thyroid gland may be tender on palpation and sometimes there may be swelling with fluctuation suggestive of localized pus collection (25). Very rarely infection can spread to nearby anatomical structures resulting in a more dramatic presentation with stridor due to laryngeal involvement requiring urgent tracheostomy (26). It is important to differentiate this condition from subacute thyroiditis which also presents with systemic symptoms and neck pain (Table 3) (see below).

DIAGNOSIS AND MANAGEMENT

Laboratory investigations are consistent with acute inflammation characterized by leukocytosis with shift to left, elevated erythrocyte sedimentation rate, raised C- reactive protein (CRP), and other acute inflammatory markers (23). In cases of severe disease, blood cultures may be positive. Ultrasonography of the thyroid may reveal an abscess. The latter requires aspiration and pus should be sent for microbiological diagnosis. Typical findings of acute suppurative thyroiditis on ultrasound are perithyroidal hypoechoic space, effacement of the plane between the thyroid and surrounding tissues, and unilateral presentation [Fig 1] (27). Computed Tomography (CT) offers better spatial resolution and can be used in cases where ultrasound is not showing characteristic findings or when there is involvement of nearby soft tissue structures. Barium swallow studies may be required to diagnose a pyriform sinus, especially in children when there are recurrent episodes of suppurative thyroiditis (28).

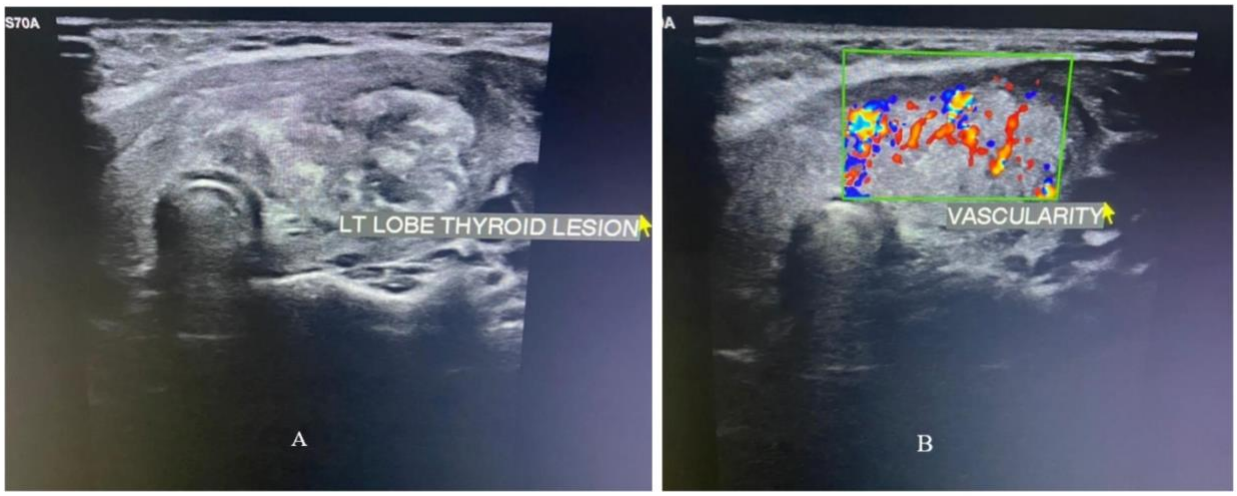

Fig 1. A. Ultrasound of the thyroid showing enlargement of the left lobe of the thyroid with heterogenous echotexture, suggestive of thyroiditis B. Ultrasound Doppler showing increased vascularity of the left lobe of the thyroid

Aspiration or surgical drainage of pus with intravenous empirical broad-spectrum antibiotics (especially in sick patients) is the cornerstone of management for acute suppurative thyroiditis. If the patient is immunocompromised, antifungal therapy should be added to initial therapy. In case of extensive involvement of nearby structures, surgical debridement of involved areas may be needed. With respect to culture sensitivity, antibiotic therapy can be modified and once clinical improvement occurs, patients can be switched to oral antibiotics. If there is presence of pyriform fistula, it should be treated either surgically (removal of entire tract with thyroidectomy) or by ablation (21, 29).

Subacute Thyroiditis

Subacute thyroiditis (also termed as granulomatous, giant cell, or deQuervain's thyroiditis), is usually due to a viral illness following respiratory illness. Rarely, bacterial infections like *Mycobacterium tuberculosis, Treponema pallidum,* or *Yersinia enterocolitica* may cause subacute thyroiditis. Tuberculous thyroiditis is discussed in another chapter (2). Differentiating features of subacute thyroiditis and suppurative thyroiditis are presented in table 3 (19, 30, 31).

Table 3. Differentiating Acute Suppurative Thyroiditis and Subacute Thyroiditis		
Features	**Acute suppurative thyroiditis**	**Sub-acute thyroiditis**
Etiology	Usually bacterial in origin	Usually follows viral upper respiratory tract infection
Presentation	Rapidly evolving, patient can be very toxic with extensive involvement	Presents with systemic symptoms over days to week
Age	Children, 20 to 40 years	20 to 60 years
Sex	Slight female preponderance	More common in females
Fever	72%	54%
Neck pain	70%	77%
Neck tenderness	Usually, unilateral (Left sided involvement due to persistent pyriform sinus)	Bilateral and migratory
Redness over skin	Common	Not present
Swelling with fluctuation suggestive of abscess formation	Common	Not present
History of sore throat	Absent	Present
Clinical features of thyrotoxicosis	Not common	Common in the initial phase
Laboratory		
Leukocytosis	82%	25 to 50 %
Raised ESR	90%	85%
Abnormal thyroid function test	44%	60%
FNAC	Pus	Giant cells, granulomas
Ultrasound Thyroid	Hypoechoic areas with abscess formation, usually unilateral	Ill-defined hypoechoic areas, usually in bilateral lobes
RAIU study	Normal	Decreased in the initial thyrotoxic phase
^{18}F FDG PET	Increased uptake	Increased uptake
CT scan	Useful when ultrasound is doubtful and when infection extends into peri thyroid tissue	Not useful
Treatment	Antibiotics & drainage of pus	NSAIDS, glucocorticoids in severe cases and sequential follow up of thyroid function tests.

FNAC- fine needle aspiration cytology; RAIU- radioactive iodine uptake; NSAIDS-Non steroidal anti-inflammatory drugs

BACTERIAL INFECTIONS OF ADRENALS

Tuberculosis of Adrenals

Tuberculosis of the adrenal glands is the most common cause of primary adrenal insufficiency in developing countries. An autoimmune etiology remains common in developed countries. Tuberculous infection of the adrenal gland occurs from hematogenous spread from pulmonary or genitourinary sites (32). Adrenals are the most common endocrine gland involved in tuberculosis (2). The symptoms are usually non-specific with generalized weakness, easy fatiguability, loss of weight, loss of appetite, pain in abdomen, and gradually progressive darkening of complexion (Fig 2). These symptoms and signs of adrenal insufficiency do not occur until more than 90% of the glands are destroyed (33). Patient can have low grade fever if the tuberculosis is active and cough and hemoptysis if associated pulmonary involvement is present. In the majority of the cases, the tuberculosis infection may not be active with only a past history of pulmonary tuberculosis (33). Untreated patients may present with adrenal crisis during times of stress. Laboratory investigations reveal low serum cortisol and high plasma adrenocorticotrophic hormone (ACTH). Sometimes, ACTH stimulation test (short synacthen test) may be needed. Adrenal insufficiency is ruled out if serum cortisol level one hour post synacthen (ACTH) stimulation is more than 500-550 nmol/L (14-20 ug/dL depending on the assay). Electrolyte abnormalities noted in adrenal insufficiency are hyponatremia and hyperkalemia. Computed tomography shows bilateral enlarged adrenal masses with areas of necrosis and caseation. In long standing cases, there may be evidence of calcifications (33). Diagnosis is confirmed by adrenal biopsy showing caseating granulomas with acid fast bacilli. Other methods like culture and molecular techniques can be used for diagnosing tuberculosis in biopsy samples. Anti-tubercular treatment (ATT) along with both glucocorticoid and mineralocorticoids remain the treatment of choice. ATT consists of isoniazid - INH (5 mg/kg /d), rifampicin (10 mg/kg /d), pyrazinamide (30 mg/kg /d), and ethambutol (20 mg/kg/d) for 3 to 6 months, subsequently isoniazid and rifampicin for 6 to 12 months (34). In case of multi drug resistant tuberculosis, ATT may be altered with respect to the pattern of resistance. It may require second line medications and longer duration of therapy. Patients usually require lifelong replacement therapy with glucocorticoids and mineralocorticoids.

Apart from *Mycobacterium tuberculosis,* in the context of HIV-AIDS and other immunocompromised states, *Mycobacterium avium intracellular* and *Mycobacterium chelonae* may also cause primary adrenal insufficiency.

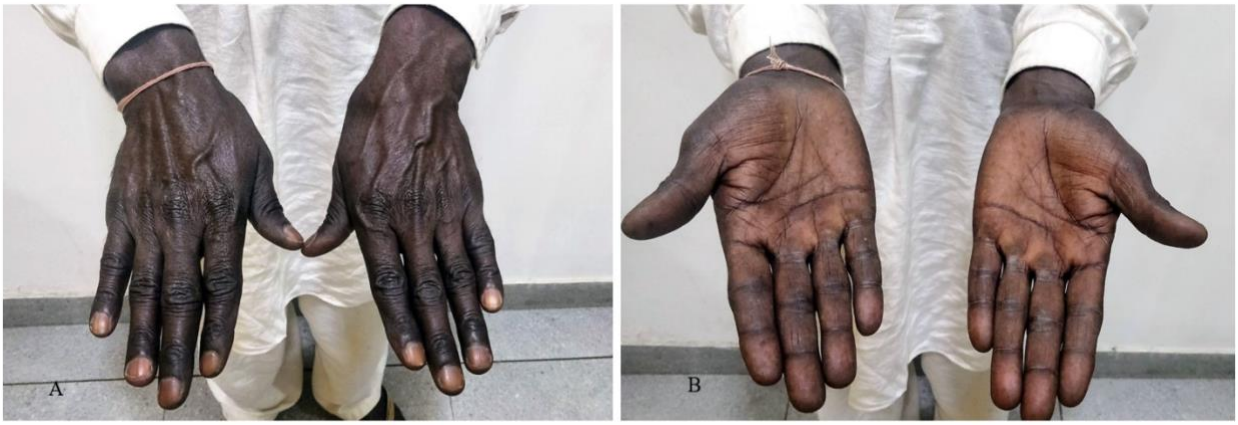

Fig 2. A. Darkening of the skin in the dorsum aspect of hands in a patient with primary adrenal insufficiency due to adrenal tuberculosis B. Darkening of the palmar aspect including palmar creases of the same patient

Adrenal Abscess

An adrenal abscess is a rare clinical condition with very few cases reported in the literature. Organisms that are implicated are *Mycobacterium*, anaerobes, *Salmonella, Nocardia,* and *E coli*. Treatment includes drainage of abscess and antibiotic therapy (35-40). The choice of antibiotic is guided by culture and sensitivity report. In culture negative cases, broad spectrum antibiotics with coverage for gram positive, gram negative, and anaerobic organisms should be considered.

Waterhouse-Friderichsen Syndrome

Waterhouse-Friderichsen syndrome (WFS) or purpura fulminans is an uncommon clinical entity associated with bilateral adrenal hemorrhage in the setting of severe bacterial sepsis, which was first reported by Rupert Waterhouse and Carl Friderichsen (41). The initial version of this syndrome was classically described with *Neisseria meningitidis* sepsis. But later it was found that a similar clinical picture was seen with other bacterial infections such as *Streptococcus pneumoniae*, *Hemophilus influenzae*, *Escherichia coli*, *Staphylococcus aureus*, Group A beta-hemolytic *Streptococcus, Capnocytophaga canimorsus*, *Enterobacter cloacae*, *Pasteurella multocida*, *Plesiomonas shigelloides*, *Neisseria gonorrhoeae*, *Moraxella duplex*, *Rickettsia rickettsia*, *Bacillus anthracis*, *Treponema pallidum,* and *Legionella pneumophila* (42-44).

Adrenal glands are predisposed to hemorrhage because around 50-60 small adrenal branches from 3 main adrenal arteries form a subcapsular plexus that drains into the medullary sinusoids through only a few venules (43). Therefore, an increase in adrenal venous pressure due to any cause may lead to hemorrhage. These bacteria may invade the adrenals directly or may produce endotoxins to cause adrenal necrosis and hemorrhage. There is also evidence of microthrombi within the adrenals along with disseminated intravascular coagulation (DIC). Pathologically, organisms are hardly demonstrated in the adrenal specimens (45). The patients are usually sick and present with profound adrenal crisis and shock. A petechial rash is usually present on the trunk, lower limbs, and mucous membrane and its severity correlates with the degree of thrombocytopenia (44). Treatment involves admission to an intensive care unit and resuscitation with intravenous fluids, intravenous glucocorticoids, and appropriate antibiotics.

BACTERIAL INFECTIONS OF GONADS

Bacterial Infections of Testes

EPIDEMIOLOGY

Infection of the epididymis can occur in both children and adults. In severe cases, the inflammation can spread further into testis and present as epididymo-orchitis. If the duration of illness is less than 6 weeks, it is termed as acute epididymo-orchitis, whereas duration more than 6 weeks is termed as chronic. In children, it usually occurs between two and thirteen years of age, whereas in adults, it is common between twenty and thirty years of age (46).

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Causative organisms in younger males less than 35 years of age are Neisseria gonorrhoeae and *Chlamydia trachomatis.* In older men, causative organisms include *Escherichia coli*, other coliforms, and *Pseudomonas*. Rare bacterial causes include *Ureaplasma* species, *Mycoplasma genitalium*, *Mycobacterium tuberculosis*, and *Brucella* species (47-49). Risk factors for epididymitis include urinary tract infections, sexually transmitted diseases, bladder outlet obstruction, prostate enlargement, and urinary tract surgeries or urogenital procedures. In homosexual men, an enteric bacterial etiology is common (46, 50).

CLINICAL PRESENTATION

Acute epididymitis presents as localized testicular pain. On palpation, there may be swelling in the posterior part of the testis that represents an enlarged testis and inflamed epididymis. More advanced cases present with secondary testicular pain and swelling (epididymo-orchitis). There could be redness of scrotum and hydrocele (reactive fluid collection secondary to infection) (Fig 3). A positive Prehn sign (manual elevation of the scrotum relieves pain) is more often seen with epididymitis than testicular torsion (46).

Fig 3. Swelling of bilateral testes with reddening of the skin overlying the scrotum, suggestive of epidymo-orchitis

DIAGNOSIS AND MANAGEMENT

In all cases of acute epididymo-orchits, it is important to rule out acute surgical conditions like testicular torsion and Fournier's gangrene. All patients should undergo routine urine microscopy, urine for culture and sensitivity, and a urine nucleic acid amplification test (NAAT) for *N. gonorrhoeae* and *C. trachomatis*. NAAT is helpful in diagnosing infections where urine cultures are negative (51). Management depends on the severity of illness, history suggestive of sexually transmitted diseases, and reports of NAAT (summarized in table 4) (46, 52).

Table 4. Management of Acute Epididymo-Orchitis

Clinical scenario	**Likely organisms**	**Choice of empirical antibiotic therapy ***
Children < 14 years	Various possibilities – secondary to anatomical issues	Treatment based on urine culture results and referral to urologist.
Individuals at risk of sexually transmitted diseases but do not practice anal intercourse	*N. gonorrhoeae* and *C. trachomatis*	Single injection of ceftriaxone 500mg intramuscular and oral Doxycycline 100mg twice daily for 10 days. Alternative for doxycycline – Azithromycin Alternative for ceftriaxone- Gentamycin
Individuals at risk of sexually transmitted diseases but do practice anal intercourse	*N. gonorrhoeae, C. trachomatis and enteric pathogens*	Single injection of ceftriaxone 500mg intramuscular and oral Doxycycline 100mg twice daily for 10 days plus oral levofloxacin 500 mg once daily for 10 days
Individuals at lower risk of sexually transmitted diseases Recent urinary tract surgery or instrumentation	Enteric pathogens	Oral levofloxacin 500 mg once daily for 10 days

*Further treatment should be adjusted based culture and NAAT results; severe cases may require hospitalization and intravenous antibiotics.

Bacterial Infections of Ovaries

Isolated infection of ovaries is not common. It is usually part of pelvic inflammatory disease. In severe cases, it may present as tubo-ovarian abscess. Tubo-ovarian abscesses are often polymicrobial and typically contain a predominance of anaerobic bacteria. Common organisms include *Escherichia coli, Bacteroides fragilis*, other *Bacteroides* species, *Pepto-streptococci,* and anaerobic streptococci (53). Diagnosis is based on history, physical examination, ultrasound suggesting tubo -ovarian mass or abscess, and microbiological diagnosis. Treatment consists of admission, intravenous antibiotic therapy, and aspiration of abscess if needed. Patients who do not respond, will need surgical intervention (54).

OTHER LINKS BETWEEN BACTERIA AND ENDOCRINE DISORDERS

Yersinia enterocolitica has been implicated in the pathogenesis of autoimmune thyroid disease (55). Immunoglobulins from patients with *Yersinia* infection inhibit binding of TSH to thyrocytes (56). This could be explained by structural similarity between *Yersinia* outer membrane proteins (YOP) and epitopes of the TSH receptor (55, 56).

Role of gut microbiome has recently implicated in the metabolic syndrome, obesity, and diabetes (57). Many metabolites produced by gut microbes get absorbed into the circulation. They may act on specific receptors to regulate metabolism (58, 59). Also, some bacterial components can act as endocrine factors controlling metabolism (58).

CONCLUSION

Bacterial infections of the endocrine glands are rare. Pituitary abscesses usually occur in the setting of underlying pathology of the pituitary gland. It is commonly caused by *Streptococci* and *Staphylococci.* MRI of the sella demonstrates a characteristic rim of enhancement after gadolinium injection. Treatment of pituitary abscess is trans-sphenoidal surgery and intravenous antibiotics. Culture is positive in only 19.7% of cases. Acute suppurative thyroiditis is commonly caused by *Streptococci* and *Staphylococci.* Important risk factor for acute suppurative thyroiditis in children is pyriform fistula, whereas in adults, it is more common in immunocompromised states. Acute suppurative thyroiditis appear as hypoechoic area on ultrasound. It is treated by ultrasound guided drainage of the abscess and antibiotic therapy. Acute suppurative thyroiditis should be differentiated from subacute thyroiditis. Primary adrenal bacterial infections other than tuberculosis are rare. Waterhouse-Friderichsen syndrome (WFS) is an uncommon clinical entity associated with bilateral adrenal hemorrhage in the setting of severe bacterial sepsis. It is classically described with *Neisseria meningitides*, but may be associated with other bacteria as well. Toxins produced by bacteria can cause necrosis, hemorrhage, and microthrombi within the adrenal gland leading to WFS. Infection of the epididymis can occur in both children and adults. Sometimes, the inflammation spreads further into testis and presents as epididymo-orchitis. Common bacterial agents causing epididymo-orchitis are *N. gonorrhoeae and* C. *trachomatis. Enteric pathogens should be suspected if there is history of homosexual practice.* Management depends on the severity of illness, history of suggestive sexually transmitted diseases, and reports of NAAT (urine nucleic acid amplification test).

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