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Infectious Disease Problems in Adolescents

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Adolescents usually enjoy good health. As a measure of this, they have the lowest incidence of physician contact of any age group (1). Nevertheless, infections common to adolescents create significant morbidity, accounting for 80–85% of days lost from school (1). Infections are three of the ten leading reasons that adolescents seek medical care (2).

The infectious diseases of special importance in adolescents include respiratory infections, mononucleosis syndrome, hepatitis, sexually transmitted genitourinary infections, and the exanthem illnesses, measles and rubella. Also, certain illnesses have more specific manifestations in adolescents than in patients of any other age group. For example, although the Epstein–Barr virus (EBV) usually results in asymptomatic infection and, less frequently, is associated with a number of clinical syndromes in children (3,4) (i.e., Burkitt's lymphoma and nasopharyngeal carcinoma), it regularly causes mononucleosis in adolescents and young adults (5). Similarly, Mycoplasma pneumoniae appears to cause pneumonia more commonly in adolescents than in other age groups (6). Also, complications of some illnesses, such as sinusitis, are also more frequent and serious in adolescents (7). Many serious childhood infections and their complications, such as Reye's syndrome, remain prominent in adolescents (8).

Although it is impossible to avoid all infections in this age group, physicians can help and advise adolescents to adopt health practices that reduce the risk of infections and their complications.

**Respiratory Infections**

The incidences of nonspecific upper respiratory infections (URIs) and some lower respiratory infections in adolescents approximate those in adults (9). In all age groups, the majority of these infections occur between mid-September and early May (10). Respiratory infections are responsible for nearly 60% of the days that adolescents lose from school (9). Symptom complexes include the common cold, pharyngitis, otitis media, sinusitis, and pneumonia.

**The Common Cold**

The common cold is the most frequent respiratory infection. The usual pathogens, listed in descending order of frequency, include rhinovirus (25–30%); coronavirus (approximately 10%); respiratory syncytial virus, parainfluenza, and adenovirus (approximately 15%); other viruses, either undiscovered or undiagnosed (45%); and other infectious agents, such as group A, G, or C streptococcus, mycoplasma, etc. (5–10%). The clinical characteristics of the common cold include nasal discharge, nasal obstruction, sneezing, mild sore throat, and cough. Although the median duration of symptoms is 7–10 days, illness lasts 14 days or longer in 25% of adolescents.

The manifestations of the common cold are so typical that special diagnostic tests are not needed. Symptomatic treatment is appropriate. Decongestants are usually more appropriate than antihistamines (11). In adolescents, complications of the common cold are rare, but can include sinusitis, otitis media, and pneumonia.

**Pharyngitis**

Pharyngitis has a variety of causes in adolescents. These causes include group A streptococcus, which
accounts for approximately 21% of the cases, and EBV infection, which accounts for 1–5% of the cases (12). The rest of the cases are presumed to be viral, with the exception of a small percentage caused by Neisseria gonorrhoeae (13). It is difficult to determine the etiology of pharyngitis and to propose a course of therapy on the basis of clinical findings (14). Although illness is usually self-limited, its duration is influenced by the etiologic agent and the prescribed therapy. For example, the clinical course of a streptococcal infection is shortened significantly when appropriate antibiotics are prescribed early (15).

Throat cultures are advisable for patients whose primary symptoms are throat pain, fever, and cervical lymphadenopathy or exudative pharyngitis. The optimal culture site is the tonsillar surface (16). Direct-swab rapid diagnostic tests for group A streptococcus, based on enzyme-linked immunosorbent assays (ELISA technique), have been well evaluated for office use and allow the practitioner to initiate antibiotic therapy, when necessary, early in the course of the illness (17).

Therapy for pharyngitis is determined by the nature of the etiologic agent. The best example of a cause amenable to therapy is streptococcal infection, which ideally is treated with penicillin, either orally (250 mg of phenoxy methyl penicillin qid) or by a single intramuscular injection of benzathine penicillin (1.2 million units). Gonococcal pharyngitis is best treated with 4.8 million units of intramuscular procaine penicillin G administered 30 minutes after a 1.0-g dose of oral probenecid.

The most significant complication of pharyngitis is peritonsillar abscess (18). Primarily a disease of adolescents and young adults, peritonsillar abscess is characterized by dysarthria, trismus, and discomfort in swallowing. In addition to high doses of antibiotics (aqueous penicillin G, 2–4 million units q 4 hr), patients require incision and drainage, often followed by a tonsillectomy.

**Otitis Media**

Otitis media is the second leading reason that patients under age 15 seek medical care, although it is far less common in older adolescents (19). Bacterial etiologic agents of otitis media include *Streptococcus pneumoniae*, *Hemophilus influenzae*, *Streptococcus pyogenes* group A, *Branhamella catarrhalis*, and *Staphylococcus aureus*. A number of respiratory viruses have likewise been implicated as etiologic agents in this illness. As with many disorders, the median duration of symptoms depends upon the promptness of diagnosis and the method of treatment (20). Antibiotic therapy with amoxicillin (500 mg tid), trimethoprim-sulfamethoxazole (one double-strength tablet bid), or cefaclor (250 mg tid) is appropriate in the adolescent. With treatment, complications are rare.

**Sinusitis**

The sinuses are affected when the normal drainage of mucus and other secretions into the nasal passages is hindered. The usual pathogens include *S. pneumoniae*, *H. influenzae*, anaerobic organisms, mixed bacteria, *S. aureus*, *S. pyogenes*, *B. catarrhalis*, gram-negative bacteria, influenza, and parainfluenza. The incidence of these pathogens has varied from study to study.

Sinusitis can develop in the course of any upper respiratory infection. It is often associated with facial pain (periorbital or supraorbital) and purulent nasal discharge. Headache and nasal obstruction, which alters the patient’s sense of taste and smell, may also be present. Only about half of patients have an elevated temperature. Eyelid edema and tearing are indicative of ethmoid involvement. Cough, particularly daytime cough, is an important sign (21).

The clinical diagnosis is based on the presence of sinus tenderness, purulent nasal discharge, and daytime cough of at least ten days’ duration. Although opacity of the sinus on transillumination is a helpful finding, transillumination is not a consistently reliable diagnostic technique (22). Radiography is the most specific test for sinusitis. Mucosal thickening is usually visible, and the presence of air-fluid levels in the sinus cavity confirms the diagnosis.

Once again, the duration of illness is determined by the speed of diagnosis and therapy. Appropriate therapy includes antibiotics and local as well as systemic decongestants. With topical decongestants care must be taken to avoid the rebound phenomenon (rhinitis medicamentosa). Amoxicillin (500 mg tid) is the most frequently prescribed antibiotic for initial oral therapy in outpatients. Trimethoprim-sulfamethoxazole and cefaclor serve as second-line agents. Symptomatic relief of pain is best provided by acetaminophen.

Teenagers are at considerable risk of developing complications of sinusitis because their frontal sinuses are rather recently developed. Complications include meningitis, brain abscess, orbital cellulitis, osteomyelitis, and cavernous sinus thrombosis. For this reason, if a prompt response to outpatient oral therapy does not occur, hospitalization to enable in-
travenous administration of antibiotics (i.e., chloramphenicol and oxacillin or cefuroxime) should be considered.

**Pneumonia**

The usual pathogens causing pneumonia include *M. pneumoniae* (which accounts for 50% of the cases), *S. pneumoniae*, and viruses such as influenza, parainfluenza, and adenovirus. *Pneumocystis carinii*, although rare, must be considered in patients at high risk for human T-cell lymphotropic virus (HTLV)-III infection.

Cough and fever are the most common clinical characteristics of pneumonia. The nature of the fever can sometimes suggest etiology. For example, fever associated with multiple chills suggests *M. pneumoniae*, whereas a single, shaking chill is more characteristic of *S. pneumoniae*. Leukocytosis can also suggest etiology; it is less common with viruses and mycoplasmal infection than with bacterial pneumonia.

The median duration of pneumonia is 14 days, although adolescents may recover much sooner. Teenagers can generally resume normal activities after being afebrile for 48 hours.

Diagnostic tests for pneumonia include a chest x-ray and sputum examination and culture. In adolescents, however, the routine sputum culture will not provide a diagnosis for 70-80% of community-acquired pneumonias. Similarly, a Gram stain is often nondiagnostic and may be even less helpful than a routine culture (23). Even chest x-rays are less useful than clinical diagnosis based on an appropriate history and physical examination (24).

Treatment of pneumonia is based on the most probable etiologic organism. Most adolescents are candidates for an outpatient antibiotic regimen. Erythromycin, 500 mg q 6 hr, is appropriate. Patients with an underlying disease (i.e., sickle cell disease, malignancy) or concomitant bronchoconstriction, as well as those who fail to respond to an outpatient regimen, may require hospitalization. Because complications from pneumonia are rare in adolescents, the presence of complications (i.e., empyema, lung abscess, or bacteremia) may be indicative of an underlying health problem.

**Respiratory Disease with Particular Relevance for Adolescents**

**Influenza**

Influenza is an acute, usually self-limiting, febrile illness that occurs in outbreaks of varying severity almost every winter. Adolescents play a major role in the spread of influenza and frequently account for the first cases (25). For example, the first few cases of swine flu in New Jersey in 1976 occurred in teenage army recruits at Fort Dix (26). These cases gave rise to a national vaccination program.

Although two distinct strains of the influenza virus, A and B, can infect humans, they cannot be distinguished on the basis of clinical signs and symptoms. Patients with either type of influenza usually have systemic symptoms, including fever, a chilly feeling or frank shaking chills, headache, myalgia, malaise, and anorexia. These symptoms progress rapidly to include dry cough, nasal discharge, and stuffiness. The median duration of illness is 10-14 days, depending on prior immunity and whether the patient receives the antiviral agent amantadine hydrochloride. Diagnosis is based almost entirely on clinical findings as cultures are expensive and the results are not immediately available.

Preventing infection via strain-specific vaccine or prophylactic amantadine provides the most efficacious approach to influenza outbreaks (27). Adolescents with chronic cardiac or respiratory illness (including severe asthma), severe anemia, immunocompromise, or severe renal or metabolic illness should be given the vaccine. When prevention is impossible or unsuccessful, treatment with amantadine can shorten the duration of fever and systemic and respiratory symptoms by about 50% if the illness is caused by type A virus and the drug therapy is initiated within the first 48 hours of infection.

Although complications in adolescents are rare, influenza can cause croup, chronic obstructive pulmonary disease, bacterial pneumonia, myositis, and myocarditis. Many of the 21 million deaths in the 1918-1919 outbreak were due to viral pneumonia in otherwise healthy young adults (28). The most dramatic and dangerous complication in adolescents is Reye's syndrome, which has been reported in the convalescent phase of the flu since the early 1970s (29). There is apparently a strong epidemiologic association between salicylate intake and Reye's syndrome (30), with the majority of recently reported cases occurring in adolescents (8).

**Mycoplasma pneumoniae**

*Mycoplasma pneumoniae* is responsible for 50% of cases of pneumonia in adolescents and young adults. This organism also causes many other clinical syndromes, including tracheobronchitis, myringitis, pharyngitis, gastroenteritis, hepatitis, meningitis,
and myocarditis (31). Headache and multiple chills are the most important symptoms of pneumonia caused by mycoplasmal infection. The course of the illness is often protracted, and x-rays may not clear for two to six weeks (32).

Diagnostic tests for pneumonia rarely reveal the etiology of the illness. X-rays may show a unilateral, lower-lobe, segmental bronchopneumonia. The peripheral leukocyte count is equivocal; it may be normal, depressed, or elevated. Cold agglutinins are usually present, but in varying titer. Cold agglutinins are usually present, but in varying titer. Cold agglutinins are usually present, but in varying titer. Cold agglutinins are usually present, but in varying titer. Cold agglutinins are usually present, but in varying titer. Cold agglutinins are usually present, but in varying titer. Cold agglutinins are usually present, but in varying titer. Cold agglutinins are usually present, but in varying titer.

Definitive diagnosis of mycoplasmal pneumonia is based on the combination of an antibody rise and a sputum culture showing \textit{M. pneumoniae}. However, growth on agar may take two to three weeks, rendering results useless for initiating drug therapy.

The treatment options for pneumonia caused by mycoplasmal infection are straightforward as the organism is sensitive to both tetracycline and erythromycin (500 mg of either medication qid). Erythromycin is a particularly attractive therapeutic agent because it is equally effective against other common pneumonia pathogens. Pleural effusions and spread of infection within the lung are the most common pulmonary complications of mycoplasmal pneumonia, but they are uncommon in the adolescent population. Death from pneumonia in adolescents is extraordinarily rare.

**Anticipatory Guidance for Respiratory Infections**

Several points about anticipatory guidance must be emphasized. First, supportive therapy is the most important part of treatment for the adolescent with undifferentiated respiratory symptoms. Most adolescents with respiratory infections will not require antibiotic therapy. Second, adolescents with chronic health problems must be appropriately immunized. At this time, the most useful immunizing agent is for influenza. Finally, because the salicylates are associated with an increased incidence of Reye's syndrome (8,29,30), adolescents with respiratory infections and/or fever should be treated with acetaminophen rather than aspirin.

**Mononucleosis Syndrome**

Mononucleosis is an acute infection most commonly caused by the Epstein–Barr virus, a member of the herpesvirus group. The EBV was first isolated in tissue culture cell lines derived from Burkitt's lymphoma. The observation of EBV antibody seroconversion in a laboratory technician with infectious mononucleosis led to the etiologic connection between EBV and the mononucleosis syndrome (33). The incidence and prevalence of infectious mononucleosis are greater in closed groups of adolescents and young adults, i.e., at boarding schools, detention centers, colleges, and military reservations. The illness causes significant morbidity and even mortality in some individuals.

**Symptom Complex**

The mononucleosis syndrome presents with a classic triad of sore throat, fever, and cervical lymphadenopathy, all of which can have severe clinical manifestations. Other characteristic symptoms include malaise, headache, fatigue, and anorexia. Splenomegaly occurs in approximately 50% of patients and is greatest during the second and third weeks of illness. Illness usually lasts for two to three weeks, although fatigue and exercise-induced exhaustion can remain for several months.

**Epidemiology**

Epstein–Barr virus infections are a widespread phenomenon, particularly among people in lower socioeconomic groups and third-world countries (34–36). In general, the incidence of mononucleosis is greatest in individuals who have not encountered the virus prior to adolescence, although EBV can cause illness in younger children (3). Individuals who enter a high-risk setting without previous exposure to the virus have a seroconversion rate of 12% per annum (37,38). By adulthood, 90–95% of all persons have antibodies to EBV (39).

Initial viral replication takes place in the pharyngeal epithelial cells (40), and the virus can be recovered in oropharyngeal secretions for prolonged periods (41). Exchange of infected saliva during intimate oral contact or from inanimate objects, such as eating and drinking utensils, is the usual mode of transmission (42,43). The EBV appears to be transmitted only minimally in standard living situations.

Like other members of the herpesvirus group, EBV can be maintained in a host in a latent form until it is activated or transformed, which often occurs under conditions of immunosuppression (44,45). This latency period accounts for the occurrence of illness or viral replication in renal transplant patients and may explain the recently described virologic findings in chronic mononucleosis syndrome (46,47).

In adolescents and young adults, other infectious
agents can cause illnesses that mimic EBV-induced mononucleosis. Among these agents are cytomegalovirus, *Toxoplasma gondii*, *Treponema pallidum* (secondary stage syphilis), rubella virus, adenovirus, the hepatitis viruses, and *S. pyogenes*. Noninfectious diseases that mimic EBV-induced mononucleosis include serum sickness, systemic lupus erythematosus, sarcoidosis, and lymphoma.

**Diagnosis**

Mononucleosis is diagnosed by clinical manifestations and laboratory findings. Diagnostic abnormalities in routine laboratory procedures include 1) relative or absolute lymphocytosis, 2) atypical lymphocytes (more than 10% of total lymphocytes), 3) relative or absolute neutropenia (48), 4) mild thrombocytopenia, 5) mild transaminitis, with or without overt hepatitis (49), and 6) mild elevation of cryoglobulins.

There are also specific tests for EBV antibody. Heterophil antibodies, mostly of the IgM class, develop transiently during the course of infectious mononucleosis and are seen in 90–95% of the cases associated with EBV. These antibodies, first described in 1932 by Paul and Bunnell, are antibodies to antigens on cell surfaces of nonhuman species (50). The monospot test, a single-dilution slide test that uses sensitized horse red blood cells, is almost as sensitive as the heterophil test (51) and is very useful in screening for these antibodies. Although this test gives quick results and is generally specific for infectious mononucleosis, false-negative results can occur when heterophil antibody titers are low; false-positive results also occasionally occur. The clinical finding most predictive of a positive heterophil test is the presence of axillary adenopathy (52).

The EBV-specific serologic tests may be diagnostic in the small percentage of patients who are heterophil negative (53). These tests consist of measuring antibodies to the viral capsid antigen (VCA), the viral early antigens (EA), and the viral nuclear antigens (EBNA). The VCA is the most common EBV-specific assay, and evidence of IgM and IgG antibodies is of value in making the serodiagnosis of primary EBV infection. The EA test measures two separate antigens and their respective antibodies—anti-D and anti-R—which are observed in 70–85% of patients in the acute phase of infectious mononucleosis (33). The viral nuclear antigens appear late in the course of infectious mononucleosis, and positive EBNA can document EBV infection when early serum samples are not available.

**Therapy**

Supportive therapy with appropriate guidance is the best approach to treat infectious mononucleosis. Sore throat and fever should be treated with antipyretics, but because of the occasional presence of relative thrombocytopenia, aspirin should be avoided. Steroids should be reserved for patients with severe, life-threatening complications, such as airway obstruction, neurologic complications, thrombocytopenia, hemolytic anemia, or myocarditis. Antiviral agents have not yet been shown to have a therapeutic role, although intravenous acyclovir may eventually prove useful for serious infections (54,55).

**Complications**

Most complications of the mononucleosis syndrome resolve without significant sequelae. Possible complications can best be grouped by organ system.

Hematologic complications, namely, autoimmune hemolytic anemia and severe thrombocytopenia, occur in up to 5% of patients (56,57). These disorders are usually self-limited but may respond to corticosteroids (58). Splenic rupture, a rare but serious complication, is most often associated with trauma (59), although it can occur spontaneously (60). Cardiorespiratory complications, including myocarditis, pericarditis, and pneumonia, have been reported in adolescents but occur more commonly in children (61,62).

Neurologic complications occur in less than 1% of cases of infectious mononucleosis (63). When these complications do occur, they are frequently the primary manifestation of the illness. Complications of the central nervous system include Guillain–Barre syndrome, Bell’s palsy, transverse myelitis, and meningoencephalitis myelitis. Fortunately, most of these complications will remit without lasting neurologic deficit.

Death from mononucleosis is rare. It is most frequently associated with x-linked immunodeficiency (64).

Viral-related malignancies such as African Burkitt’s syndrome (65,66) and nasopharyngeal carcinoma (67) may also occur. The role of EBV as a cause of lymphomas and Hodgkin’s disease is under investigation (68,69).

**Anticipatory Guidance**

Advising adolescents of ways to avoid contracting infectious mononucleosis is difficult. Health practitioners can share certain information with patients
and parents that will help them to cope with the disease in themselves and others. For example, supportive care is the hallmark of rehabilitative services. Patients with infectious mononucleosis do not require isolation as the disease is poorly transmitted from person to person (70). Platelet abnormalities occur in a small percentage of patients, and therefore acetaminophen is preferred for symptomatic relief of fever and myalgia. Finally, because of the risk of subclinical splenic enlargement, patients should avoid contact sports and strenuous activities for at least four weeks after signs and symptoms of infection remit.

**Hepatitis**

**Epidemiology**

Viral hepatitis is most frequently caused by one of three types of agent: hepatitis virus A, hepatitis virus B, and the so-called non-A, non-B agents, of which there appear to be at least two. Individuals between the ages of 10 and 20 comprise 17.4% of hepatitis A cases, 9.3% of hepatitis B cases, and 11.1% of non-A, non-B hepatitis cases (70). In 1983 alone, 1,360 cases of hepatitis A in teenagers were reported, 829 cases of hepatitis B, and 301 cases of non-A, non-B hepatitis (71).

All of these hepatitis forms have worldwide distribution. Acquisition of the antibody to the hepatitis virus is closely linked to socioeconomic status and geographic location (72,73). As with mononucleosis, individuals acquire antibodies for these viruses during childhood, usually via asymptomatic infection.

Personal contact with someone who has hepatitis A is the most important risk factor for acquisition of symptomatic illness (71). For hepatitis B, the risk factors are personal contact with an infected individual, intravenous drug abuse, homosexual activity in males, recent dental work, and recent transfusion of blood or blood products. For non-A, non-B hepatitis, the risk factors are surgery, hospitalization, dental work, and drug abuse.

The classic mode of transmission of hepatitis A is by fecal–oral contact. Attendance at day-care centers is currently the most important cause of the spread of this disease in both children and adults in North America, accounting for approximately 10% of cases (74). Young children acquire the infection (usually asymptotically) and spread it to staff, siblings, and parents. Hepatitis B and non-A, non-B hepatitis are usually spread through the blood or body secretions. Hepatitis A is a more frequent cause of epidemics in adolescents than is hepatitis B (75,76). Spread of hepatitis B among classroom contacts (77) and teenage wrestlers has been reported (78).

**Symptom Complex**

The type of hepatitis is difficult to distinguish by the clinical symptoms alone. All forms usually have a prodromal phase, characterized by fever, malaise, fatigue, headache, anorexia, nausea, vomiting, myalgias, and/or right upper quadrant tenderness. In general, the younger the patient, the less severe the manifestations of the illness (79). Nearly 80% of the patients with viral hepatitis develop jaundice (71). In fact, because hepatitis is most specifically suggested by scleral and skin jaundice, many cases without jaundice may go undiagnosed or unreported. The clinical course of the illness varies but usually lasts for one to three weeks (80).

Although clinical manifestations alone will not reveal the etiology of the hepatitis virus, certain characteristics are specific to each form of the illness. The incubation period for hepatitis A is 14–50 days, with a mean of 28 days. Diarrhea is more frequent in type A than in any other type, occurring in approximately 20% of patients. Upper respiratory symptoms, encephalitis, and aplastic anemia, although rare, occur more often with hepatitis A.

Hepatitis B usually has an incubation period of 60–110 days (81). More than the other types, it is associated with extrahepatic manifestations, including urticaria and arthritis, and, much less commonly, glomerulonephritis and vasculitis. In about 10% of patients with symptomatic hepatitis B, serologic tests continue to reveal markers of acute infection. Most of these patients continue to have mildly elevated liver-associated enzymes, which are indicative of chronic persistent hepatitis (81). This generally benign, nonprogressive disease is characterized by chronic portal inflammation, with little or no fibrosis. Some patients develop cirrhosis.

Diagnosis of the non-A, non-B forms of hepatitis is essentially done by exclusion. The incubation period varies from two weeks to six months, with a mean of about eight weeks. Half of the cases of non-A, non-B hepatitis are anicteric. Although there are rare instances of chronicity, the course is usually benign (82).

**Diagnosis**

Hepatitis is usually diagnosed by clinical signs and symptoms—especially jaundice—and by laboratory evidence of elevated liver-associated enzymes.

Specific serologic tests for etiology of viral hepati-
tis are now available, with certain limitations. The presence of serologic markers precedes the clinical illness itself. Type A markers are antibodies directed at specific viral proteins. These antibodies are of IgM and IgG classes. The presence of the former suggests acute infection. The markers for hepatitis B are more complicated; they include the hepatitis B surface antigen (HBsAg) and its antibody, anti-HBs, as well as core antigen antibody (anti-HBc), e antigen, and its antibody (anti-e). In the classic pattern both HBsAg and anti-HBc are acutely positive, but this pattern may not occur in all cases. Anti-HBc is almost always positive, but a single negative test for HBsAg does not definitely exclude acute hepatitis B (83). The persistence of antigen markers without concomitant antibody suggests chronic infection (84). Specific serologic markers are available for non-A, non-B hepatitis.

**Therapy**

In the absence of specific treatment for acute viral hepatitis, the major emphasis is placed on support, symptomatic care, and prevention of transmission. There is no convincing evidence to justify the use of corticosteroids in acute hepatitis, regardless of its severity (85).

Prophylaxis for hepatitis A involves passive immunization with gamma globulin (86) for all close contacts of the infected patient. Patients exposed to hepatitis B can be given a safe, effective vaccine (87), which consists of highly purified and triple-inactivated HBsAg obtained from the serum of chronic carriers (88). In some exposure situations, the hepatitis B vaccine is used in combination with a special high-titer immune globulin—hepatitis B immune globulin (HBIG)—to allow for immediate as well as continuing protection (89).

**Complications**

The overall incidence of complications from viral hepatitis is low. Complications are limited to those problems already discussed in the review of the specific clinical presentations.

Mortality rates vary from 0.2–2.0%. Fatalities are less frequent with type A than with type B or non-A, non-B hepatitis. The overall mortality rate for viral hepatitis is about 1%. Death can occur rapidly in patients with fulminant hepatitis (90).

**Anticipatory Guidance**

To prevent hepatitis A: 1) limit the amount of raw shell fish eaten; 2) administer appropriate immune serum globulin (ISG) when possible exposure has occurred; and 3) give prophylactic ISG to patients traveling to third-world countries. To prevent hepatitis B: 1) promptly administer HBIG and the hepatitis B vaccine to individuals with known exposure; 2) check the serologic status of adolescents with high-risk lifestyles (i.e., male homosexuals and intravenous drug users) and vaccinate those who are not protected; and 3) properly screen all blood products before use.

**Genitourinary/Sexually Transmitted Infections in Adolescents**

At puberty, preadolescents undergo physiological changes that transform them into sexually mature individuals. The accompanying psychological and social changes frequently result in sexual activity. Eighty percent of males and 70% of females have had intercourse during their teenage years (91), and the average age of first sexual intercourse is 16.

During these years of bodily change and emerging sexuality, symptoms attributed to the genital organs become a special concern of adolescents. Some of the most common presenting complaints include urinary tract infections, vaginal discharge, urethral discharge, and genital lesions. In sexually active teenagers, these complaints are especially important because they are frequently caused by sexually transmitted diseases (STDs). An awareness of the most likely etiologies of each of these conditions will facilitate differential diagnoses and medical management of urogenital disease.

**Urinary Tract Infections in Adolescent Females**

Urinary tract infections (UTI) are among the major reasons for physician visits by adolescent females. In women, the usual etiological organism is *Escherichia coli*, but in adolescent and young adult females, *Staphylococcus saprophyticus* is also common. *Chlamydia trachomatis* is a frequent cause of dysuria and acute urethral syndrome (92). The risk factors for urinary tract infections include sexual intercourse, pregnancy, behavioral factors (i.e., diaphragm use (93) and voluntary urinary retention (94)), anatomical abnormalities, and underlying disease.

The diagnosis of UTI is based on both clinical symptoms and laboratory criteria. The clinical symptoms of lower tract infection (cystitis and urethritis) include frequent urination, dysuria, burning pain during urination, suprapubic discomfort, and passage of cloudy and occasionally blood-tinged urine. When fever, flank or back pain, and rigors occur,
upper tract involvement must be assumed to be present. Because the symptoms of vaginitis often resemble those of UTI (95), it is important to determine quickly the site of infection. A bacteria count of over 100,000 organisms per milliliter of unspun urine is the old standard for making the diagnosis of a UTI. New data suggest that this criterion is too restrictive and will not correctly identify women with overt infections (96). If women are experiencing such symptoms, lower counts, 100–1,000 of organisms per milliliter of unspun urine, should be considered diagnostic of infection.

Treatment of UTIs depends upon the location of the infection within the urinary tract. Lower-tract infections can usually be treated effectively with oral, single-dose therapy of trimethoprim-sulfamethoxazole, two to four double-strength tablets. Because single-dose therapy will not eradicate upper-tract infections (kidney), failure to respond to short-course therapy will establish a diagnosis of upper-tract infection (97). Upper-tract infections require a 7–14-day or longer course of therapy with trimethoprim-sulfamethoxazole (one double-strength tablet bid for 14 days) or amoxicillin (500 mg po tid) for 14 days. If significant systemic signs and symptoms are present, the patient should receive intravenous antibiotics (gentamicin, 80 mg iv q 8 hr, or ampicillin, 1 g iv q 6 hr). Prophylaxis is used to prevent recurrent symptomatic infection (98).

A lower-tract infection that has not been adequately treated can lead to pyelonephritis (99). Stones can result from recurrent infections. But sepsis is fortunately rare in adolescents.

Anticipatory guidance for UTIs includes the following: do not retain urine voluntarily, urinate after sexual intercourse, do not leave diaphragm in longer than necessary, drink plenty of fluids, and maintain good personal hygiene.

Vaginitis and Cervicitis

Vaginitis and cervicitis are also common in adolescent and young adult females. The rising incidence of vaginal discharge results from the physiological and behavioral changes that occur at puberty. Hormonal changes and ovulation make the vagina and cervix more vulnerable to infection by a number of pathogenic organisms; sexual activity, which results from behavioral changes, makes the female even more susceptible to infection. A careful history of sexual activity and use of contraception must be obtained, and a complete pelvic exam should be performed.

In adolescents the infections that cause vaginal discharge, in order of frequency, include the following: fungal vaginitis, usually caused by Candida albicans; trichomoniasis; Gardnerella vaginitis; gonococcal vaginitis, caused by Neisseria gonorrhoeae; and nonspecific urethritis, caused by C. trachomatis. Diagnostic features and therapeutic regimens for these infections are shown in Table 1. The risk factors for vaginitis and cervicitis include sexual activity and multiple sexual partners, tight clothing, oral contraceptives, underlying illness (diabetes), poor hygiene, and pregnancy (100).

The chlamydial and gonorrheal sexually transmitted infections can cause pregnancy loss (101,102), pelvic inflammatory disease (PID), and a variety of nonlocalized infections. The incidence of complications from vaginitis and cervicitis that are not sexually transmitted, however, is very small.

Pelvic Infections in Adolescent Females

Vaginitis or cervicitis caused by either N. gonorrhoeae or C. trachomatis can ascend, causing endometritis, salpingitis, and peritonitis. Any combination of these disease entities is commonly known as pelvic inflammatory disease (PID). The incidence of PID in adolescents is great; 20–24 year olds are the only age group with a higher rate than 15–19 year olds (103).

Adolescents face an increased risk of acquiring PID for several reasons. They tend to have a larger number of sexual partners (104), and their immature anatomy is easily infected by a variety of sexually transmitted pathogens (105). Adolescents’ frequent use of barrier methods of contraception (106) contributes to the problem, as does poor follow-up of STDs.

Diagnosis of PID is based on physical and laboratory findings. The major criteria for clinical diagnosis are a history of abdominal pain, lower abdominal tenderness (especially rebound), cervical motion tenderness, and adnexal tenderness. Minor diagnostic criteria include fever, leukocytosis, elevated sedimentation rate, abnormal sonogram, culdocentesis revealing bacteria, and vaginal discharge. Three or more major clinical features are necessary for diagnosis. If only three major criteria are identified, two or more minor criteria may be used as supplements (107).

Adolescents with PID usually respond well to therapy. The decision to treat the individual as an inpatient or as an outpatient depends upon the severity of illness, a prior history of PID, coincidental pregnancy, the presence of an IUD, inability to com-
Table 1. Vaginal Discharge in the Adolescent Female

| Organism              | History                                                                 | Physical exam                                                                 | Lab findings                                                                 | Treatment                                      |
|-----------------------|--------------------------------------------------------------------------|-------------------------------------------------------------------------------|------------------------------------------------------------------------------|------------------------------------------------|
| *Candida albicans*    | Intense vulvar and vaginal pruritus; external dysuria; predisposing factors include diabetes mellitus, pregnancy, obesity, poor hygiene, antibiotic and birth control use | Erythema and inflammation of the vulva and vagina with thrushlike patches; thin watery to thick discharge | pH of discharge is 4.5; vaginal smear with 10% KOH shows hyphae and buds; confirmation by culture on Nicker- son's or Sabouraud's media | Clotrimazole 100 mg intravaginally at bedtime for 7 days, or 200 mg for 3 days, or 500 mg once; miconazole 100 mg intravaginally at bedtime for 7 days, or 200 mg for 3 days |
| *Trichomonas vaginalis* | Profuse vaginal discharge; dysuria; pruritus; dyspareunia                 | Profuse frothy, thin, foul-smelling greenish-yellow discharge; friable cervix with sub-epithelial hemorrhages; erythematous vaginal mucosa with punctate vaginal eruptions | pH of discharge is 5–7; motile trichomonads on wet mount | Oral metronidazole 2 g in one dose or 250 mg tid for 7 days for the patient and partner |
| *Gardnerella vaginalis* | Foul-smelling discharge; pruritus; dysuria; dyspareunia; patient may be asymptomatic | Scant to profuse foul-smelling homogeneous creamy white-gray discharge; very little vaginal irritation | pH of discharge is 5–6; wet prep shows clue cells (agglutination of the organism to the epithelial cells); positive whiff test; culture on blood agar | Oral metronidazole 500 mg bid for 7 days for patient and partner |
| *Neisseria gonorrhoeae* | Dysuria; dysmenorrhea; dyspareunia; patient may be asymptomatic          | Mucopurulent discharge from cervical os; erythema of cervix; there may be associated urethritis, bartholinitis, salpingitis, or disseminated infection | Growth on Thayer-Martin medium | Oral amoxicillin 3 g with probenecid 1 g; alternative procaine penicillin 4.8 million U IM with oral probenecid 1 g, spectinomycin 2 g IM or cefoxitin 2 g IM, either regime followed by oral tetracycline 500 mg qid for 7 days or doxycline 100 mg bid for 7 days; treatment for patient and partner |
| *Chlamydia trachomatis* | Dysuria, dysmenorrhea; dyspareunia; may be asymptomatic                  | Copious mucopurulent discharge from the cervical os; erythema of the cervix; may be associated with urethritis, salpingitis perihepatitis, Reiter's syndrome | Growth of *C. trachomatis* in intracellular cultures greater than 10 polymorphonuclear leukocytes on endocervical smear | Oral tetracycline 500 mg qid for 7 days or doxycline 100 mg bid for 7 days |

*Source:* Adapted from Kastrinakis, Wilson, and D'Angelo (128).

Inpatient and outpatient regimens for the treatment of PID are shown in Table 2. Outpatients should be scheduled for follow-up visits on days 2, 7, and 14 to assess the progress of therapy. Complications of PID include tubo-ovarian abscess (108), recurrent infections (109), ectopic pregnancy (110), and infertility (111).

**Urethritis in Adolescent Males**

Most of the same infectious etiologies of vaginal discharge in adolescent females can cause urethral discharge and dysuria in adolescent males. As with the
Table 2. Treatment of Pelvic Inflammatory Disease

| INPATIENT TREATMENT OF PID | OUTPATIENT TREATMENT OF PID |
|---------------------------|----------------------------|
| A. 1. Doxycycline 100 mg IV bid plus cefoxitin 2 g IV qid, or | A. Cefoxitin 2.0 g IM plus 1.0 g probenecid po, or |
| 2. Gentamicin or tobramycin 2.0 mg/kg IV followed by 1.5 mg/kg q 8 hr | Amoxicillin 3.0 g po plus 1.0 g probenecid po, or |
| Continue IV drugs for at least 4 days and at least 48 hr after defervescence; then complete 10–14 days of therapy with: | Procaine penicillin 4.8 million U, IM, at two sites plus 1.0 g probenecid po, or |
| B. 1. Doxycycline 100 mg po bid (For regimen A.1.) | Ceftriaxone 250 mg IM, plus |
| 2. Clindamycin 600 mg po bid (For regimen A.2.) | B. Doxycycline 100 mg po bid for 10–14 days, |

Source: 1985 STD Treatment Guidelines (130).

adolescent female, physicians must obtain a thorough history of sexual activity, preferences, and use of contraception in adolescent males. A complete genital exam must be performed, with particular attention to the testes and epididymis.

Urethritis is usually classified as gonococcal or nongonococcal urethritis (NGU). Gonococcal urethritis classically presents with dysuria and a copious yellow-green discharge two to seven days after exposure. Nongonococcal urethritis has a longer incubation period (one to three weeks) and, frequently, a more indolent onset with scantier discharge and less pain (112). The symptoms of these two types of urethritis are similar regardless of microbiologic cause. The duration of symptomatic illness varies, depending upon when appropriate therapy has been administered. Diagnostic features and therapeutic regimens for these infections are shown in Table 3.

Complications of urethritis are rare in adolescents, although urethritis has been known to cause prostatitis (113) and epididymitis. Also, in young males, chlamydia infection has been associated with Reiter’s syndrome in those genetically predisposed to this form of arthritis (114).

Genital Ulceration

Most genital lesions encountered in adolescents and young adults are due to STDs. The most common lesions are vaginal warts and herpes simplex infection, usually with type II virus. Other causes include syphilis, chancroid, lymphogranuloma venereum, and granuloma inguinale. The clinical characteristics of genital ulceration varies, depending upon the etiologic organism, and diagnosis usually requires laboratory confirmation. Diagnostic features and therapeutic regimens are outlined in Table 4.

Toxic Shock Syndrome (TSS)

Adolescent females account for a disproportionate number of the reported cases of Toxic Shock Syndrome (TSS), despite the fact that most teens do not use intravaginal catamenial products (115,116). Those that do use these products, however, are at a higher risk for developing TSS than women who are older and use the same feminine hygiene products, probably because of the immature anatomy of the adolescent genital tract. Therefore, clinicians should provide anticipatory guidance aimed directly at preteen patients and their parents.

Because the adolescent’s genital tract is not completely developed until ovulation occurs regularly, teenagers should be cautioned not to use intravaginal catamenial products until two years after the onset of menarche, except under special circumstances or for brief periods of time (117). These products also should not be used for overnight protection. If intravaginal catamenial products are used, they should be changed at least every four hours regardless of the degree of menstrual flow, and patients who have experienced symptoms suggestive of TSS should not use them for at least four years after the illness and only then after appropriate cultures have demonstrated that the S. aureus is no longer present.

Acquired Immunodeficiency Syndrome (AIDS)

AIDS is relatively uncommon in adolescents. As of June 1985, a total of 59 cases had been reported for individuals between the ages of 10 and 19 (118). Risk factors include homosexual or bisexual practices, intravenous drug abuse, hemophilia, history of prior blood transfusion, and Haitian heritage (119).

The clinical characteristics of AIDS are related to a deficiency in T-lymphocytes caused by infection with human T-cell lymphotropic virus (HTLV-III). This deficiency in the immune system makes the host susceptible to a number of opportunistic infec-
Table 3. Urethral Discharge in the Adolescent Male

| Organism                        | History                                                                 | Physical exam                                      | Lab findings                                      | Treatment                                                                 |
|---------------------------------|--------------------------------------------------------------------------|----------------------------------------------------|---------------------------------------------------|---------------------------------------------------------------------------|
| Trichomonas                     | Dysuria; most often asymptomatic                                        | Scant thin, yellow discharge, if any               | Wet prep showing motile trichomonads (this is rare finding in males)    | Oral metronidazole 2 g in one dose or 250 mg tid for 7 days for the patient and partner |
| Nongonococcal urethritis:        | Symptoms are mild and not acute; scant urethral discharge; may be asymptomatic | Thin white or clear mucoid penile discharge may be associated with epididymitis/orchitis | Polymorphonuclear leukocytes on Gram stain without organisms; growth of C. trachomatis in intracellular culture | Oral tetracycline 500 mg qid for 7 days or doxycycline 100 mg bid for 7 days for the patient and partner |
| Chlamydia trachomatis or Ureaplasma urealyticum (T-mycoplasma) | Acute onset of symptoms after an incubation period of 2–6 days; dysuria, frequency of urination; may be asymptomatic | Copious mucopurulent discharge may be associated with periurethral abscess or epididymitis | Gram-negative diploci intracellularly on Gram stain; growth on Thayer–Martin media | Oral amoxicillin 3 g with probenecid 1 g; alternatives: procaine penicillin 4.8 million U IM or cefoxitin 2 g IM; with oral probenecid 1 g; spectinomycin 2 g IM, either regimen followed by oral tetracycline 500 mg qid for 7 days or doxycycline 100 mg bid for 7 days for patient and partner |

Source: Data from Kastrinakis, Wilson, and D’Angelo (128).

...if some of the most prominent being *P. carinii* pneumonia, disseminated infections with *Mycobacterium avium* and *intracellularis*, cryptococcal meningitis, *C. albicans*, cryptosporidium diarrhea, and disseminated herpes infections. Malignancies such as Kaposi’s sarcoma and carcinoma of the rectum are seen in older persons but are rare in adolescents.

The diagnosis of AIDS is based on appropriate laboratory findings, particularly the antibody to HTLV-III. There is no known treatment for the underlying immune defects in AIDS, and there have been no reports of spontaneous reversal of this condition.

Sexual preferences and orientation must be addressed honestly with teenage males because of the steady increase in the prevalence of AIDS in the adult male homosexual population. Adolescent males need to be advised of the sexual practices that put them at high risk for this fatal disease, such as oral–anal or genital–anal contact.

Males who have had homosexual experiences should be screened for hepatitis B antibody. When results are negative, patients should be offered the hepatitis B vaccine. Using barrier contraception (condom) will prevent many sexually transmitted infections in homosexual, as well as heterosexual, individuals.

### Anticipatory Guidance

Adolescents, like adults, need to be firmly warned about the dangers of sexually transmitted urogenital infections. In an attempt to limit the incidence and complications of STDs, particular attention must be paid to a history of sexual activity and preferences, the menstrual cycle in the female, use of contraception in both males and females, and the coexistence of STD syndromes. The importance of careful genital and pelvic examinations in the evaluation of adolescents cannot be overemphasized. Finally, treatment of STDs is incomplete without appropriate patient follow-up, therapy of partners, and counseling about prevention.

### Measles and Rubella

The widespread use of vaccines to prevent measles and rubella has resulted in dramatic decreases in the incidence of both these illnesses in the United States (120). The very success of the vaccination program...
### Table 4. Genital Ulcers

| Condition | History | Physical Exam | Lab Findings | Treatment |
|-----------|---------|---------------|--------------|-----------|
| Venereal warts (condylomata acuminata) | Recurrent raised lesions; usually asymptomatic; women may have vaginal itching, dyspareunia, vaginal bleeding after intercourse | Raised multiple pink verrucous papules around the genitalia and rectum | Changes consistent with human papilloma virus on Pap smear | Topical podophyllin, trichloroacetic acid, cryotherapy, laser therapy |
| Herpes progenitalis | Usually painful ulcers, systemic symptoms including fever, myalgias, arthralgia during primary infection, may be recurrent | Small, usually multiple transient ulcers and vesiculopustules; inguinal adenopathy | Multinucleated giant cells by cytology, growth of herpes simplex virus in viral cultures; positive herpes immunofluorescence | Oral acyclovir 200 mg 5 times daily or topical acyclovir qid for two weeks for primary infection; treatment for recurrent infection is controversial but oral acyclovir as prescribed above may help |
| Syphilis | Painless genital ulcer | Single clean, indurated, non-tender ulcer with raised border; inguinal adenopathy | Darkfield exam positive for *T. pallidum*; positive serology | Syphilis less than 1 yr: penicillin G ben- zathine 2.4 million U IM once |
| Lymphogranuloma venereum | Mildly painful ulcers, systemic symptoms | Very transient ulcer followed by development of fluctuant, tender unilateral inguinal adenopathy | Lymphogranuloma venereum complement fixation titers; isolation of the organism from the bubo (lymph node), urethra, or cervix | Oral tetracycline 500 mg qid for 14-21 days; alternative; oral erythromycin 500 mg qid for 14-21 days |
| Granuloma inguinale | Chronic painless ulcers indolent course; endemic in the tropics | Begins as a papule; becomes multiple, spreading, contiguous lesions, may be extragenital involvement; "pseudobubo" or subcutaneous swelling may occur | Demonstration of the Donovan body on an impression smear from a biopsy | Oral tetracycline 2 g daily for 4 wk |
| Chancroid | Very painful ulcer; rare in the United States | Purulent, soft indurated, superficial lesion; ragged edge; tender, suppurative adenopathy | Gram-negative coccobacillus on smear; growth of *H. ducreyi* on special media | Oral trimethoprim-sulfamethoxazole 2 tabs bid for 14 days; alternative; oral erythromycin 500 mg qid for 14 days or tetracycline 500 mg qid for 14 days |
| Pediculosis pubis | Intense pruritus | Excoriated papules | Mites on microscopic exam | One application of 1% Lindane lotion or shampoo; hygiene |
| Moluscum contagiosum | Usually asymptomatic | Whitish nonumbilicated papules | None | Resolve spontaneously; excision or cryotherapy |

*Source: Data from Kastrinakis, Wilson, and D'Angelo (128).*
has resulted, paradoxically, in a change in the epidemiology of these diseases. Those who care for adolescents need to be aware of these illnesses, their manifestations, and approaches to their prevention.

**Epidemiology**

Both measles and rubella were widely prevalent infections until the appearance of vaccines in 1963 and 1969, respectively. The vaccines abruptly reduced the incidence of these infections and changed their epidemiology. Caused by RNA viruses, measles and rubella are spread when droplet nuclei from infected patients are inhaled by susceptible hosts. Once the virus is introduced into a susceptible population, the cases multiply quickly, as dramatically demonstrated in recent outbreaks of measles on a number of college campuses (121).

With minimal changes in production technique, the rubella vaccine has remained a highly efficacious, live attenuated vaccine (122). The measles vaccine, on the other hand, has undergone a number of changes. The original measles vaccine, a killed vaccine, was available from 1963 until 1967. At that time, a live attenuated vaccine was introduced, which took the place of the killed vaccine. Individuals who received the original killed vaccine appear to be still susceptible to measles infection and need to be revaccinated with the contemporary vaccine. Also, the schedule for administering the vaccine has been altered with the realization that infants vaccinated before 15 months of age have a less reliable response to the vaccine. Anyone given the vaccine as an infant needs revaccination to ensure protective antibody titer (123).

**Symptom Complexes**

Once exposed to the measles virus, a susceptible individual will develop illness in 10–14 days. The prodrome consists of fever, cough, coryza, and conjunctivitis. Koplik spots—raised blue-gray specks on the buccal mucosa—appear 24 hr before the rash and are characteristic of measles. The rash is seen first on the neck and face; it proceeds downward to involve the whole body. The rash is maculopapular and erythematous. After three to five days, it fades slowly and eventually disappears.

In individuals who have received killed measles vaccine, a syndrome characterized by a rash that begins on the extremities, high fever, and pneumonia can occur after exposure to measles (124). This illness, known as “atypical measles,” is probably mediated by a hypersensitivity reaction to the virus in a partially immune host. In rare cases, atypical measles have been documented despite revaccination with live attenuated vaccine in persons previously given the killed vaccine (125).

Infection with the rubella virus produces a milder illness than that caused by the measles virus. The prodrome, consisting of fever and malaise, is similar to that of measles; mild coryza and conjunctivitis might also occur. The associated rash is less dramatic and, as with measles, it starts on the neck and/or face. The rash usually lasts three to five days. Lymphadenopathy, a hallmark of rubella infection, occurs before the onset of the rash and frequently persists after the rash has subsided. The posterior auricular and suboccipital nodes are most likely to increase in size and are often the last to resolve.

**Diagnosis**

Diagnosis of both of these rash illnesses rests primarily on recognition of the clinical symptoms. Serologic confirmation of the presence of type-specific antibodies serves little function in terms of immediate diagnosis but is extremely important from a public health standpoint.

**Prevention and Treatment**

Both infections can be prevented by appropriate vaccination. Vaccine efficacy ranges from 90–95% for both present vaccines (126). Prior to 1972, the recommendation of the Advisory Council on Immunization Practices of the U.S. Public Health Service was to vaccinate at 12 months. Therefore, persons born before 1972 should be considered candidates for revaccination against measles. The measles and rubella vaccines can be administered singly, in combination with each other (MR), or with each other and mumps (MMR). Systemic reactions, including fever and myalgia, are common but rarely severe.

No specific treatment is indicated for clinical infections. Although rubella remains a major public health problem because of the threat of congenital rubella syndrome in infants born to mothers who contract rubella in the first trimester of pregnancy, it rarely causes severe illness in adolescents. On the other hand, measles complications, including encephalitis, pneumonia, and subacute sclerosing panencephalitis, can be severe and even fatal in adolescents and young adults (127).
Anticipatory Guidance

All adolescents born prior to 1972 should be considered candidates for revaccination with measles vaccine. Those born before 1969 are candidates for rubella vaccine. Health care providers should help ensure that students be excluded from schools unless they can prove that they are appropriately vaccinated or they have proof of prior infection.

If measles or rubella is suspected in an adolescent, reassessment of vaccine status is necessary for all close contacts, particularly schoolmates. Ill adolescents must be quarantined away from other potentially susceptible adolescents.

Summary

Although adolescents are a generally healthy group, infection can create significant morbidity and occasional mortality. As in all patients, early diagnosis and treatment can shorten the duration of infection and limit complications. Physicians should provide teenage patients with guidance necessary to prevent transmission and recurrence.

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