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57 CHAPTER AIDS AND THE GASTROINTESTINAL TRACT

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1. What is the role of barium esophagram for patients with AIDS (acquired immunodeficiency syndrome) and esophageal symptoms?

Barium esophagram has a limited role in patients with AIDS. Infections are the most common cause of esophageal disease in patients with AIDS. Although many of these infections have a characteristic appearance on barium radiography, overlap is frequent, thus mandating a definitive diagnosis by other means before prescribing antimicrobial therapy. In addition, some therapies for these disorders (e.g., corticosteroids) may be associated with significant toxicity, further emphasizing the importance of a histologic diagnosis. Last, in patients with severe odynophagia, the barium study may be inadequate because severe pain on swallowing will limit the amount of barium that can be swallowed, thus limiting the quality of the study. For these reasons, endoscopy with biopsy is the preferred diagnostic modality in this group of patients as this will yield a definitive diagnosis in 75% of cases.

2. What is the role of empiric therapy for new-onset esophageal symptoms in patients with AIDS?

Candida esophagitis is the most common cause of esophageal disease in patients with AIDS presenting with dysphagia or odynophagia (Fig. 57-1). Because of this high prevalence, an empiric approach to new-onset esophageal symptoms with potent antifungal therapy is commonly undertaken. A randomized study using a loading dose of 200 mg of fluconazole followed by 100 mg/day for 10-14 days showed both efficacy and cost-effectiveness, Because Candida esophagitis responds very rapidly to fluconazole, in the patient who does not symptomatically improve within the first few days of treatment, endoscopic evaluation to exclude other causes of disease (viral esophagitis) should be performed.

With improving HIV/AIDS therapies, patient commonly have CD4 counts higher than 200 cells/mL. In these patients, an empiric trial of a proton pump inhibitor is reasonable for symptoms consistent with gastroesophageal reflux disease (GERD). If symptoms do not improve, endoscopic evaluation to exclude other causes of disease is indicated.

3. What are the most common causes of esophageal ulceration in AIDS?

The most common causes are cytomegalovirus (CMV) and idiopathic esophageal ulcer (IEU). On endoscopy, CMV and IEU appear most often as large, well-circumscribed solitary ulcerations, with normal-appearing surrounding mucosa. Multiple ulcers may also be observed. Antiretroviral medications such as didanosine (ddI) and zidovudine (AZT) have also been associated with pill-induced esophagitis. Herpes simplex virus (HSV) is usually associated with multiple small, shallow esophageal ulcerations, often raised with a volcano crater appearance. GERD can also present with ulcerations of the distal esophagus generally involving the gastroesophageal junction; these lesions are generally linear and superficial. Neoplasms (e.g., lymphoma), parasites (e.g., leishmania), and fungal infections (e.g., histoplasmosis and *Candida* spp.) are rare causes of esophageal ulcers (Table 57-1).

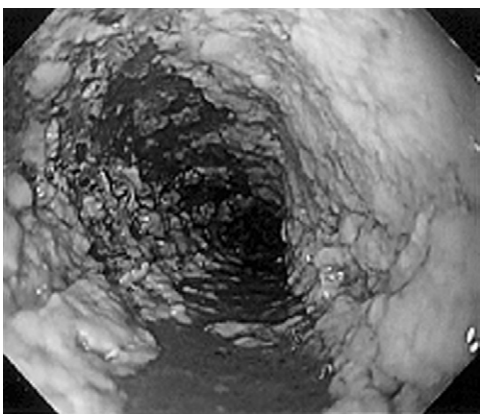

Figure 57-1. Candidal esophagitis. Yellow plaques coating the esophageal wall are typical for *Candida.* Note that on one portion of the wall, the material has been removed and the underlying mucosa is normal.

4. What biopsy technique should be used to sample an esophageal ulcer?

The exact number of biopsies required for maximal sensitivity is not clearly established, but several studies suggest the range of 8 to 10. It is important to obtain biopsy samples from the ulcer margin and from the ulcer base. This is because biopsy of the ulcer edge reveals a cytopathic effect that is present in squamous epithelium associated with HSV; conversely, CMV resides in granulation tissue in the ulcer base. The role of culture and cytology for esophageal ulcers is not settled. If all biopsies are negative for viral, bacterial, fungal, and parasitic infections, a diagnosis of IEU can be made.

Table 57-1. Reported Causes of Esophageal Ulcers in AIDS

Viruses	Cytomegalovirus, herpes simplex virus type II, Epstein-Barr virus, papovavirus, human herpes virus-6
Fungi	*Candida* spp., *Histoplasma capsulatum, Cryptococcus neoformans,* mucormycosis, aspergillosis, *Penicillium chrysogenum, Exophiala jeanselmei*
Bacteria	*Mycobacterium avium*-complex, *Mycobacterium tuberculosis, Bartonella henselae, Nocardia asteroides, Actinomyces israelii*
Protozoa	Cryptosporidia, *Leishmania donovani, Pneumocystis carinii*
Tumors	Non-Hodgkin lymphoma, Kaposi sarcoma, cancer (squamous cell and adenocarcinoma), lymphoma
Pill-induced	Zalcitabine, zidovudine, other
Gastroesophageal disease, idiopathic	Idiopathic esophageal ulcer

5. What is AIDS-cholangiopathy? How do patients present?

AIDS-cholangiopathy is a spectrum of biliary tract abnormalities resembling sclerosing cholangitis that can be caused by a wide array of microorganisms and neoplasms, usually in patients with advanced immunodeficiency. Almost all patients have a CD4 count less than 200 cells/mL and most have a CD4 count less than 50 cells/mL. Patients generally present with epigastric or right upper quadrant pain, fever, and malaise. Although
AIDS-cholangiopathy is a cholestatic disease, jaundice and pruritus are uncommon. The most common laboratory finding in this syndrome is a markedly elevated alkaline phosphatase, usually more than three times the upper limits of normal. Typically bilirubin is not elevated and rarely exceeds 3 mg/dL, and transaminases are only mildly elevated. Generally, these patients have a dilated bile duct that is identifiable on abdominal ultrasonography. The diagnosis is best established by endoscopic retrograde cholangiopancreatography (ERCP). Several cholangiographic patterns have been described, including papillary stenosis, sclerosing cholangitis, combined papillary stenosis and sclerosing cholangitis, isolated intrahepatic disease, and long extrahepatic bile duct strictures. The most common pattern is papillary stenosis with intrahepatic sclerosing cholangitis. Endoscopic sphincterotomy is appropriate only for the relief of pain in patients with papillary stenosis. Unfortunately, the disease is progressive and antimicrobial therapy has no influence on its outcome. Treatment with highly active antiretroviral therapy (HAART) is associated with decreased mortality.

6. What are the most common causes of AIDS-cholangiopathy? How are they diagnosed?

1. *Cryptosporidium parvum*
2. Microsporidia
   Enterocytozoon bieneusi
   Encephalitozoon intestinalis
   Encephalocytozoon cuniculi
3. CMV
4. *Mycobacterium avium*-complex (MAC)
5. *Cyclospora cayetanensis*
6. Non-Hodgkin lymphoma
7. Kaposi sarcoma

The diagnosis is usually established by obtaining biopsy specimens of the ampulla or duodenal mucosa, bile duct biopsy, aspirated bile specimens, or biliary epithelial brush cytology. Despite its infectious origin, medical therapies aiming at the eradication of these organisms have not produced marked improvement in AIDS-cholangiopathy. Treatment with HAART is associated with decreased mortality.

7. What are the most common causes of pancreatitis in HIV-infected patients?

Several studies have documented chronic and/or recurrent elevations of serum amylase and lipase in up to 50% of patients with AIDS. Pancreatograms at the time of ERCP have shown abnormalities of the pancreatic ducts consistent with chronic pancreatitis. These findings have led many investigators to hypothesize that pancreatic insufficiency from chronic pancreatitis is an important cause of chronic diarrhea in AIDS; however, most cases of chronic pancreatitis are attributable to conditions, such as alcohol abuse. The most common medications associated with pancreatitis in AIDS are pentamidine, ddI, and zalcitabine (ddC). Protease inhibitors frequently cause hyperlipidemia. Ritonavir is associated with the most dramatic increases in triglycerides with 10% of patients developing severe hypertriglyceridemia. Pancreatitis is well described in patients with elevations in triglycerides from protease inhibitors. Reported infectious causes of pancreatitis include CMV, HSV, MAC, and tuberculosis. An infectious cause of pancreatitis is difficult to establish and would require pancreatic biopsy.

8. How has HAART affected the incidence of opportunistic gastrointestinal (GI) disorders?

Since the introduction of protease inhibitors and HAART in 1995, there has been a constant and dramatic decline of GI opportunistic disorders in AIDS patients. It is postulated that improvement in the immune status, as reflected by an increase in CD4 cells, prevents the development of opportunistic disorders. In several reports, symptoms resolved even before any changes in the total CD4 cell count were apparent, suggesting that the antiretroviral medications promote elimination of the offending GI infection, probably secondary to immune-boosting mechanisms independent of the CD4 cell count and intrinsic antimicrobial activity.

9. What is the recommended workup for diarrhea in AIDS?

When evaluating an AIDS patient with diarrhea, careful attention should be directed to the history and physical examination. Enteritis (small bowel diarrhea) is associated with voluminous, watery bowel movements, abdominal bloating, cramping, borborygmi, and nausea. Abdominal pain, if present, tends to be periumbilical or diffuse. Abdominal examination reveals an increase in number and frequency of bowel sounds, which may be high-pitched. Conversely, colitis (large bowel diarrhea) is characterized by frequent, small bowel movements, with the presence of mucus, pus, and/or blood (dysentery). Patients with prominent involvement of the distal colon also have proctitis symptoms, such as tenesmus, dyschezia (pain on defecation), and proctalgia (rectal pain) (Fig. 57-2).

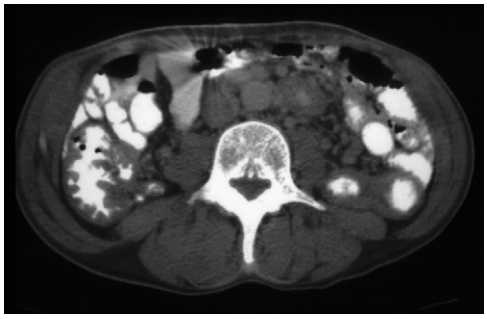

Figure 57-2. Cytomegalovirus colitis. Abdominal computed tomography scan shows colonic wall thickening most pronounced in the right colon.

It is also important to consider patient exposures. A history of new medications or an alteration in a current regimen, such as antiretrovirals or antibacterials, is important because many protease inhibitors are associated with diarrhea and antibacterials are associated with *Clostridium difficile* colitis. In febrile patients, blood cultures should be obtained for common bacteria. If the CD4 count is below 50 cells/mL, blood cultures for MAC should be obtained. If stool and blood culture studies are negative, the next step is endoscopic evaluation with biopsy. In the presence of colitis symptoms, flexible sigmoidoscopy or colonoscopy is recommended. Table 57-2 summarizes the studies and laboratory tests used in the evaluation of diarrhea in AIDS. Table 57-3 lists the most common infectious causes of diarrhea in AIDS. Table 57-4 lists common associations between exposures and infections.

10. Describe the clinical features of HSV proctitis in AIDS.

HSV proctitis is the most common cause of nongonococcal proctitis in sexually active homosexual men. HSV proctitis classically presents with tenesmus, purulent rectal discharge, severe proctalgia, fever, constipation, and anorectal bleeding. Painful inguinal lymphadenopathy is an almost universal finding. The pain tends to distribute in the region of the sacral roots (i.e., buttocks, perineal region, and posterior thigh). Because of the neural involvement by HSV and the presence of severe pain, patients may complain of impotence and difficulty in initiating micturition.

Table 57-2. Studies and Laboratory Tests Used in the Evaluation of Diarrhea in AIDS

Stool	Cultures (*Salmonella, Shigella, Campylobacter* spp.) Toxin (*Clostridium difficile*) Ova and parasites (*Giardia lamblia, Entamoeba histolytica, Cryptosporidium* spp.) Modified Kinyoun acid-fast (*Cryptosporidium* spp., *Isospora* belli) Concentrated stool (zinc sulfate, Sheather sucrose flotation) (microsporidia)
Blood	Cultures (*Mycobacterium avium*-complex, *Salmonella, Campylobacter* spp.) Antibodies (*Entamoeba histolytica*, cytomegalovirus [CMV])
Gastrointestinal fluids	Duodenal aspirate (*Giardia lamblia*, microsporidia) Electron microscopy (*Cryptosporidium* spp., adenovirus)
Biopsy stains	Hematoxylin-eosin Giemsa or methenamine silver (fungi) Methylene blue–azure II–basic fuchsin (microsporidia) Fite (mycobacteria)
Immunohistochemical stains (CMV), immunologic methods	In situ hybridization (CMV) DNA amplification (CMV) Culture of tissue CMV Herpes simplex virus Mycobacteria

Table 57-3. Infectious Causes of Diarrhea in AIDS

VIRUSES	BACTERIA	PARASITES	FUNGI
Cytomegalovirus	*Salmonella* spp.	*Giardia lamblia*	*Histoplasma capsulatum*
Astrovirus	*Shigella* spp.	*Entamoeba histolytica*	*Candida albicans*
Picornavirus	*Campylobacter jejuni*	Microsporidia	
Coronavirus	*Clostridium difficile*	*Enterocytozoon bieneusi*	
Rotavirus	*Mycobacterium avium*-complex	*Encephalitozoon intestinalis (formerly Septata)*	
Herpesvirus	*Treponema pallidum*	*Cyclospora cayetanensis*	
Adenovirus	*Spirochetes*	*Cryptosporidium* spp.	
Small round virus	*Neisseria gonorrhoeae*	*Isospora belli*	
HIV	*Vibrio cholerae*	*Blastocystis hominis* (?)	
	Aeromonas spp.		
	Pseudomonas spp. (?)		
	Staphylococcus aureus		

Table 57-4. Sources of Infectious Diarrhea

INFECTIOUS AGENT	ASSOCIATION
C. *difficile*	Recent antibiotics, nursing home or hospital exposures
Cryptosporidiosis Microsporidiosis	Recent visit to a farm, contact with farm animals, use of a public swimming pool
Giardia	Camping, stream water
Mycobacterium avium	CD4 count less than 50
Cyclospora cayetanesis	Common cause of diarrhea in South America
Microsporidiosis	Uncommon in the southern United States
Rotavirus	Common cause of diarrhea in Australia

Visual inspection and anoscopy commonly reveal the following lesions: vesicles, pustular rectal lesions, or diffuse ulcerations. HSV is a pathogen of the squamous mucosa; therefore, diffuse proctitis involving the entire rectum is rare. In severe cases, the columnar rectal and sigmoid mucosa has been involved. The differential diagnoses of HSV proctitis include lymphogranuloma venereum (*Chlamydia trachomatis*), *Entamoeba histolytica, Salmonella* spp., and *Campylobacter jejuni.*

11. What is the preferred endoscopic procedure for the evaluation of diarrhea in AIDS?

The advantage of endoscopy is that it permits direct visualization of the mucosa and retrieval of tissue for histologic examination. The diagnostic yield of colonoscopy in HIV-infected patients with chronic diarrhea and negative stool studies ranges from 27% to 37%; CMV is the most common etiology identified. Because CMV colitis is usually present in the distal colon, sigmoidoscopy with biopsy may be a sufficient workup, but in 13% to 39% of cases of CMV enterocolitis, the virus can be detected in the right colon only. Therefore, if CMV is suspected as the cause of diarrhea, a full colonoscopy is warranted, especially if sigmoidoscopy is negative. However, it is still not clear whether colonoscopy has a higher yield than flexible sigmoidoscopy for the detection of organisms other than CMV. Evaluation with colonoscopy would be prudent if sigmoidoscopy is negative and right-sided abdominal complaints are reported. The value of upper endoscopy and small bowel biopsy in the evaluation of chronic diarrhea has also been demonstrated, although specific treatment options for most small bowel pathogens are limited. Some would obtain ileal biopsy at the time of colonoscopy rather than proceed with upper endoscopy and biopsy. The most commonly detected organisms involving the small bowel are cryptosporidia and microsporidia.

12. What is the most common cause of viral diarrhea in AIDS?

CMV is one of the most common opportunistic infections in patients with AIDS, occurring late in the course of HIV infection when immunodeficiency is severe (CD4 lymphocyte count less than 100/mm^3). CMV has been identified in mucosal biopsy samples in as many as 45% of patients with AIDS and diarrhea, especially in those patients with negative stool studies. CMV causes both enteritis and colitis. A number of other viral pathogens have been reported to involve the GI tract in patients with AIDS, but their clinical importance remains to be determined. Examples include adenovirus, rotavirus, astrovirus, picobirnavirus, and coronavirus. There are also reports that HIV itself can be isolated from enterocytes and colonic cells, but its role in causing disease is uncertain. HSV can cause proctitis that mimics diarrhea because of the rectal mucous discharge. However, HSV does not cause enterocolitis because it invades the squamous mucosa, not the columnar epithelium, such as the one lining the colonic and small bowel mucosa.

13. What are the treatment options for CMV enterocolitis?

The natural history of CMV colitis is variable. In untreated patients, it usually has a chronic course characterized by progressive diarrhea and weight loss, although occasionally symptoms and histologic abnormalities remit spontaneously. Unlike CMV retinitis, for which strong evidence supports induction therapy followed by lifelong maintenance therapy, the optimal duration of therapy and the need for maintenance therapy in CMV colitis are undefined. Consensus guidelines recommend 3 to 6 weeks of induction therapy followed by maintenance therapy if there is a history of relapses. Two antivirals (foscarnet and ganciclovir) have been studied extensively in the therapy of CMV colitis and/or enteritis. Cidofovir, the newest intravenous agent, has been used primarily in patients with retinal disease, but in our experience it is effective for GI disease as well. The newest agent is valganciclovir. This drug can be given orally and achieves serum levels similar to intravenous ganciclovir. Studies for GI disease are limited. Funduscopic examination at the time of diagnosis of CMV enterocolitis is mandatory, because duration of therapy is considerably longer for disseminated diseases than for disease limited to the GI tract.

A number of open-label trials of ganciclovir for HIV-infected patients with CMV GI disease have demonstrated clinical improvement in approximately 75% of patients. Open-label trials of foscarnet have yielded comparable results. The only placebo-controlled trial of ganciclovir in AIDS-associated CMV colitis found no clinically significant differences, probably because the treatment period was only 2 weeks. A randomized trial comparing ganciclovir with foscarnet in 48 AIDS patients with CMV GI disease found similar clinical efficacy (73%), regardless of the location of disease (esophagus vs. colon). Endoscopic improvement was documented in over 80% of patients. For all patients, institution of HAART is important and, if there is an immunologic response, long-term maintenance therapy can be discontinued.

14. Name the parasites that cause diarrhea in AIDS.

Among the protozoa, *C. parvum* is the most common parasite causing diarrhea in AIDS and has been identified in up to 11% of symptomatic patients. Although a cause of acute diarrhea, cryptosporidiosis is found most commonly in HIV-infected patients with chronic diarrhea. In some studies of HIV-infected patients with chronic diarrhea, microsporidia (*E. bieneusi* and *E. intestinalis*) are the most commonly identified pathogens. Giardia is also a consideration in patients with diarrhea, especially when chronic and associated with the upper gastrointestinal symptoms of nausea and bloating. *Isospora belli* is a rare GI pathogen in HIV-infected patients in North America, whereas it is endemic in many developing countries, such as Haiti.

15. Compare the clinical features and therapies for cryptosporidiosis and microsporidiosis.

Gastrointestinal microsporidial infection is generally attributed to two species: *E. bieneusi* and *E. intestinalis*. In general, intestinal disease is relatively mild in contrast to the severe diarrhea typical for cryptosporidiosis. Loose stools and mild weight loss are common with colonic symptoms typically absent. Gastrointestinal bleeding suggests another diagnosis as this infection does not cause mucosal ulceration. Although stool studies can establish the diagnosis, small bowel biopsies, of either the duodenum or ileum, with special stains are more sensitive. Although there is no effective antimicrobial therapy for *E. bieneusi,* albendazole is highly effective for *E. intestinalis.* As with all opportunistic infections in AIDS, HAART may result in clinical remission.

Cryptosporidia are a common cause of chronic diarrhea in HIV-infected patients with severe immunodeficiency. There are at least 40 species of Cryptosporidia, but the most common cause of human disease is *Cryptosporidium muri*. Cryptosporidia infect and then reproduce within the columnar small intestinal cells. Infection can occur from person-to-person or animal-to-person or from waterborne transmission (e.g., swimming pools, lakes). Therefore, a severely immunodeficient patient with AIDS not taking HAART should be advised to avoid contact with farm animals, public pools, and lakes. The life cycle is completed in a single host. Autoinfectious cycles follow ingestion of a few oocysts, leading to severe disease and persistent infection in severely immunodeficient hosts. The diarrhea is generally voluminous and watery. Dehydration and weight loss are common in patients with advanced immunodeficiency. Disease severity correlates with immune function. The stool may contain mucus but rarely contains blood or leukocytes. The disease may wax and wane, but persistent and/or progressive disease may be manifested by dehydration and electrolyte imbalances. Constitutional symptoms are prominent, including low-grade fever, malaise, anorexia, nausea, and vomiting. Both of these infections improve with reconstitution of the immune system following successful HAART.

16. Which bacteria most commonly cause diarrhea in AIDS?

Campylobacter, Salmonella, and *Shigella* spp. and *C. difficile*. *Yersinia enterocolitica, Staphylococcus aureus*, and *Aeromonas hydrophila* have also been associated with severe enterocolitis in HIV-infected patients. *C. difficile* colitis has become the most frequent bacterial cause of diarrhea in HIV-infected patients, perhaps because of frequent exposure to antimicrobials and requirement for hospitalization. MAC is a common pathogen in patients with advanced immunosuppression (i.e., CD4 count less than 50 cells/mm^3). An incidence of 39% has been described when the CD4 count remains less than 10/mm^3. Tuberculosis is most frequent in developing countries and is less likely to present with diarrhea alone.

17. What is bacillary peliosis hepatis (BPH)?

BPH produces multiple cystic blood-filled spaces in the liver. BPH is caused by an infection with the bacteria *Bartonella henselae* (formerly *Rochalimae*) and occurs in patients with advanced AIDS. Patients present with generalized and nonspecific symptoms, such as fever, weight loss, and malaise. Abdominal pain, nausea, vomiting, and diarrhea may be prominent. Skin manifestations include reddish vascular papules that can be confused with Kaposi sarcoma. On abdominal examination, hepatosplenomegaly and lymphadenopathy are the most prominent features. Histopathology of the liver lesions shows multiple cystic blood-filled spaces within fibromyxoid areas. The treatment of choice is erythromycin for at least 4 to 6 weeks, but doxycycline is a safe alternative.

18. Describe the management of HIV wasting syndrome.

AIDS wasting is defined as an involuntary weight loss of 10% from baseline over 12 months or 5% over 6 months. Approximately 20% of patients with HIV will develop wasting. With the advent of HAART, the incidence of AIDS wasting has decreased. However, the prevalence of wasting at the time of AIDS diagnosis has increased. The weight loss is typically lean muscle mass, which has a dramatic impact on quality of life. This loss of muscle mass is associated with increased mortality, accelerated disease progression, and impaired functioning. Many strategies have been developed to treat HIV wasting. Nutritional counseling and dietary supplements are effective in increasing fat free mass. Additionally, specific supplementation of L-glutamine, beta-hydroxy-beta-methylbutyrate, and L-arginine have been shown to increase lean muscle mass. Growth hormone and testosterone increase lean body mass, but these treatments have significant side effects. Some evidence shows anti–tumor necrosis factor therapies increase weight, but these agents present safety concerns in immunocompromised individuals. Resistance exercise provides significant increases in lean body mass as well as strength. Regimens vary, but typically they consist of a three-times-a-week regimen with a clearly defined number of repetitions and percent of maximal output for each repetition. Exercise is inexpensive and without reported side effects and thus is an ideal first-line therapy.

19. When do you initiate hepatitis B virus (HBV) therapy in the setting of HIV?

HBV/HIV coinfection represents a significant problem in HIV care. As HAART has improved the prognosis in HIV/AIDS, significant increases in morbidity and mortality due to liver disease have been observed. HBV and HIV are acquired by similar mechanisms and thus coinfection is common. Patients with coinfection of HIV and HBV have higher HBV DNA levels and are less likely to convert from HBeAg+ to HBeAb+, indicating a poorer response to HBV therapy. Patients with a HBV DNA greater than 2000 and F2 or greater fibrosis on biopsy should have HBV treatment. If a patient has cirrhosis, he or she should be treated if HBV DNA is greater than 200. For patients with a high CD4 count, HBV monotherapy that is not active against HIV should be first-line therapy. When initiating HAART, HBV also should be treated with two antiviral agents active against HBV. If CD4 counts are between 350 and 500 cells/mL, one can elect to treat both HIV and HBV. HAART with two agents active against HBV should be used instead of HBV monotherapy in these individuals.

20. Why is it important to know the HBV treatments that are also active in treating HIV?

Initiating HBV monotherapy that is also active in treating HIV can result in HIV resistance, potentially limiting HAART options. Furthermore, if HAART is initiated without concurrent HBV treatment, immune reconstitution can result in a potentially life-threatening flare of untreated HBV. Table 57-5 shows treatments active against HBV and HIV or HBV alone.

Table 57-5. Hepatitis B Treatments and HIV Activity

TREATS HIV AND HBV	TREATS HBV WITHOUT HIV RESISTANCE
Lamivudine	Interferon/PEG-IFN
Tenofovir	Adefovir (at 10 mg dosing)
Emtricitabine	Telbivudine (in vitro)
Entecavir (in vivo)	

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