AIDS MEDICAL TRAINING FOR NON-PHYSICIANS

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MEDICAL EVALUATION
OF PERSONS AT RISK OF
HUMAN IMMUNODEFICIENCY VIRUS
INFECTION
INCLUDING ACQUIRED IMMUNODEFICIENCY SYNDROME (AIDS)
AND RELATED CONDITIONS

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Acquired Immunodeficiency Syndrome (AIDS) and other conditions due to human immunodeficiency virus (HIV) have now established themselves as a worldwide epidemic. There has been a significant increase over the past several years in the incidence of HIV infection in smaller communities where persons at risk may be less visible.

The purpose of this booklet is to alert health professionals throughout the country to the protean manifestations of HIV infection in its earliest stages, so that proper treatment, referral and counseling may be implemented without delay.

Researchers have shown that AIDS is caused by a retrovirus which has also been referred to as "human T lymphotropic virus" (HTLV-III), "lymphadenopathy associated virus" (LAV)2, or "AIDS associated retrovirus" (ARV)3, but has been renamed "human immunodeficiency virus" (HIV)18. This retrovirus has been shown to be cytopathic and tropic for the CD4 (helper) subset of T lymphocytes, thus causing varying degrees of depletion of these lymphocytes in many individuals infected1,14,19. There are other human retroviruses that appear to cause disease but whose antibodies are not detected by current screening tests for HIV, e.g. HIV-2 21,22, HTLV-I 23. HIV has been isolated from peripheral blood lymphocytes from most patients with AIDS, AIDS related conditions including generalized lymphadenopathy, and symptomatic persons with antibody to HIV1,14.

Antibodies to HIV have been demonstrated in virtually all patients with AIDS and related conditions including persistent generalized lymphadenopathy4. Seropositivity also has been demonstrated in 49% of a random sampling of homosexual and bisexual men in San Francisco20, 56-72% of persons with hemophilia A receiving untreated factor VIII concentrate before 1985 11,12, significant numbers of women who were sexual partners of men with AIDS13, and the majority of transfusion recipients who have received blood known to be HIV contaminated24. The incidence of seropositivity in random blood donors and health care workers with no other risk factors is less than 0.2%.

A small but unknown percentage of persons may carry HIV without detectible HIV antibody; this has been demonstrated through positive HIV viral culture and/or HIV DNA polymerase chain reaction of peripheral blood lymphocytes. This HIV seronegative carrier state has been shown to last three years or more in some individuals 25. However, in most infections seroconversion occurs in less than two months after exposure 26.

The Centers for Disease Control (CDC) revised the case definition of AIDS in 1987 to include the following diseases in adults. An asterisk (*) requires a positive HIV antibody test to establish an AIDS diagnosis 17.

Opportunistic Infections:
1. Candidiasis of the esophagus, trachea, bronchi or lungs.
2. Cryptococcosis, extrapulmonary.
3. Cryptosporidiosis with diarrhea > 1 month.
4. Cytomegalovirus disease of an organ other than liver, spleen, or lymph nodes.
5. Herpes simplex infection causing a mucocutaneous ulcer that persists > 1 month; or bronchitis, pneumonitis, or esophagitis.
6. Mycobacterium avium complex or M. Kansasi disease, disseminated (at a site other than lungs, skin, cervical or hilar lymph nodes).
7. Pneumocystis carinii pneumonia.
10. Coccidioidomycosis, disseminated *
11. Histoplasmosis, disseminated *
12. Isosporiasis with diarrhea > 1 month *
13. M. Tuberculosis, extrapulmonary *
14. Salmonella septicemia, recurrent *

Neoplasms:
1. Kaposi's sarcoma < 60 years of age.
2. Kaposi's sarcoma > 60 years of age. *
3. Lymphoma of brain < 60 years of age.
4. Lymphoma of brain > 60 years of age. *
5. Non Hodgkin's lymphoma. *

Other:
1. HIV encephalopathy *
2. HIV wasting syndrome *
II. GUIDELINES FOR THE EVALUATION AND MANAGEMENT OF PERSONS AT RISK IN THE CONTEXT OF HIV INFECTION

James M. Campbell, MD

These guidelines have been devised for the medical evaluation of the patient concerned about the possibility of having been infected with HIV. This is not meant to replace the standard evaluation and care of the gay male patient (which would include routinely such tests as the RPR, hepatitis B serology, GC screen, and stool examinations for ova and parasites), IV drug user, or hemophiliac. Rather, these guidelines are designed to offer an outline for the screening of patients with regard to HIV infection.

1. HISTORY
   A. General
      Fever: Unexplained and greater than 100 degrees F.
      Night Sweats: Recurring, drenching.
      Weight loss: Unintentional, unexplained, greater than ten pounds.
      Fatigue: Interfering with work, normal activities.
      Drug allergies: Particularly to trimethoprim-sulfamethoxazole.

   B. Skin/Mucous Membranes
      Herpes Zoster (shingles)
      Herpes Simplex: increasing frequency of recurrence with unusually slow healing.
      Candidiasis: oral, perianal.
      Dermatitis: chronic or recurrent folliculitis, bullous impetigo, seborrhea, dry skin, urticaria, raised violaceous lesions.

   C. Gastrointestinal
      Unexplained diarrhea.
      Dysphagia/odynophagia.

   D. Lymphatic/Hematologic
      Lymphadenopathy: unexplained.
      Easy bruising, petechiae.
      Spontaneous bleeding.

   E. Central Nervous/Peripheral Nervous System
      Headaches: persistent or unexplained.
      Paresthesias/dysesthesias.
      Depression, mood swings.
      Incoordination.
      Visual complaints: scotomata, diplopia, unexplained blurring.
      Memory and concentration difficulties.
      Seizures.
      Weakness (local or diffuse).
      Speech (aphasia, dysarthria).

   F. Respiratory
      Cough: usually dry or minimally productive, worse with deep inspiration or exertion.
      Exertional dyspnea.

   G. Social
      Drug use: routes of administration.
      Sexually transmitted disease history: gonorrhea, syphilis, intestinal parasites, hepatitis (A, B, and NANB), non-specific urethritis, anal warts, mononucleosis, cytomegalovirus infection, scabies.
      Sexual history: sex practices, contact with persons with AIDS or at risk for developing AIDS, drug use during sex, loss of libido.

2. PHYSICAL EXAMINATION

   Complete but with particular attention to the following:
   Vital signs: weight, temperature (note time of day), respiratory rate. Temperature may normally be slightly elevated in afternoon or evening.
   General appearance.
   Skin/mucous membranes (including anoscopy): raised violaceous lesions (Kaposi's sarcoma), thrush, herpes simplex, herpes zoster, petechiae, hairy leukoplakia, severe gingivitis.
   Lymph nodes: size and location of all palpable lymph nodes; occipital, auricular, submental, anterior and posterior cervical, axillary, epitrochlear, supraclavicular, inguinal and femoral.
   Fundoscopic: "cotton wool" spots, hemorrhages.
   Abdominal: hepatomegaly or splenomegaly.

3. LABORATORY EVALUATION (See Section III. G)
REFERENCES


III. LABORATORY EVALUATION IN HIV INFECTION

Edward E. Winger, MD and James M. Campbell, MD

A. Routine

Complete blood count, differential, platelet estimation, erythrocyte sedimentation rate. Chemistry panel: particular attention should be given to low cholesterol, low albumin, elevated total globulin, elevated liver enzymes.

B. Viral and Immunologic

The introduction of serologic markers for infection by HIV has resulted in important psychologic as well as social consequences. Immunologic studies formerly used as surrogate markers for infection likewise must be used with consideration for the psychological impact of clinical staging.

VIRAL STUDIES: Several types of studies now may be undertaken. Currently, assessment of the immune response to HIV is readily available as HIV antibody through several methods which are described below. Several techniques are now available for the detection of the etiologic agent. These include HIV p24 antigen detection, direct viral detection by HIV culture, and detection of viral genomic material through amplification by the polymerase chain reaction (PCR).

HIV ANTIGEN: Detection of viral antigen provides proof of active infection thus resolving the question of current infection vs past-resolved infection with residual immune response. Regardless of technique or sensitivity of a particular antigen test, successful detection of antigen depends upon the current viral status of the patient. A negative result does not exclude current viral infection. Because of the probability of detecting antigen increases as the viral burden increases, a positive result may be useful in prognosis and/or monitoring the effectiveness of antiviral therapy. At this time, there is no FDA-licensed antigen test. It should be noted that a low incidence of positive results may be found in documented sero-negative individuals and that the probability of a false positive result increases significantly in individuals with autoimmune disorders. It is difficult to assess the true incidence of false positive results in HIV sero-positive individuals who are known to have a high incidence of auto-antibodies.

VIRAL CULTURE: The detection of infectious virus is the benchmark of HIV infection. Isolation may be made from any tissue, however, most clinically isolated virus is made from the peripheral blood. Successful isolation depends upon the sensitivity of the technique, the viral load as well as proliferative capacity of the virus. Viral culture generally is not necessary in clinical settings for either diagnosis or prognosis, and, if done by sensitive technique, is positive in almost all individuals who are HIV antibody positive.

PCR: The presence of HIV may be determined by detection of HIV-specific DNA sequences in infected cells. Current techniques are relatively insensitive to low levels of virus. The development of the polymerase chain reaction (PCR) permits preliminary amplification of a selected portion of genetic material prior to the application of the DNA detection techniques. The addition of the PCR technique permits detection of one viral genome in one hundred thousand cells. Unfortunately, the technique is expensive and is subject to a relatively high number of false positive results. Because the technique may be applied to suspected cases of HIV infection where other techniques are negative, positive results cannot be confirmed. Quantitation of antigen load is a theoretical possibility. However, it has not yet been achieved clinically.

HIV ANTIBODY: This test is the current method of choice for detection of infection by HIV. The current generation of ELISA screening assays has an extremely low level of false negative results even in high risk populations. False negative results are found early in infection before viral replication is sufficient to induce a brisk immune response and occasionally late in infection when there is severe immunologic compromise. False positive results are rare with the current generation of ELISA assays especially in high risk populations. While viral lysate assays (assays which employ antigen derived from HIV-infected cell cultures) remain the only FDA-approved assay systems, assays prepared from a combination of recombinant HIV proteins offer even greater reliability in future generations of the screening assays. In recommending that their patients take the test, physicians should encourage anonymous testing where appropriate because of possible negative impacts on employment, insurance, and privacy.
IV. A. FEVER AND HIV INFECTION

Stephen E. Follansbee, MD

Fever is one of the most common and therefore least specific signs in HIV infection. Evaluation must take into account documentation of fever, duration of fever, the daily pattern of temperature elevation, and association of fever with a careful system review. A temperature elevation of 0.5 to 0.6 degrees C (1 degree F) above baseline may be physiologic, especially in the late afternoon. A temperature greater than 38.3 degrees C is abnormal. In the context of the patient's general appearance and other factors, a fever of 3 to 5 days duration should prompt a thorough evaluation. Association of a fever with other symptoms should prompt evaluation of that system (see appropriate section). For example, fever and diarrhea should indicate evaluation for shigella, salmonella, cytomegalovirus enteritis, cryptosporidiosis, campylobacter, etc.

The extent of evaluation depends on the combination of factors listed above, whether these represent a change for the patient already identified to have AIDS, and results of previously documented fever evaluations. For example, repeating blood cultures routinely for every temperature elevation above 38.5 degrees C may be unreasonable unless there is a new associated complaint suggesting a new cause of fever. Common causes of fever to which all persons are susceptible are still common and thus do not necessitate a complete and thorough evaluation for other AIDS-related infections. The increasing tendency to use the indwelling intravenous catheters predisposes to bacterial and fungal line-associated sepsis.

The differential diagnosis of fever with generalized complaints includes:

I. Infection
   syphilis
   viruses: cytomegalovirus, adenovirus, herpes simplex, varicella-zoster, hepatitis
   protozoa: pneumocystis, toxoplasmosis, cryptosporidiosis, amebiasis
   bacteria: salmonella, shigella, legionella, pneumococcus, staphylococcus
   fungi: cryptococcus, coccidioidomycosis, histoplasmosis, invasive candidiasis
   mycobacterial: particularly M. tuberculosi and M. avium intracellulare, but also other atypical mycobacteria
   HIV: although unusual, fever may occur secondary to HIV infection of macrophages and release of endogenous pyrogens.

II. Malignancy: particularly lymphoma, which may present limited to the central nervous system, gastrointestinal tract, or other site. Although Kaposi's sarcoma usually does not produce fever, exceptions have occurred in visceral involvement, particularly of the liver and occasionally of the lung.

III. Vasculitis: not common, but possible with circulating immune complexes documented in AIDS patients.

IV. Drug-induced adverse effect: both prescription and non-prescription medication may have increased incidence of such adverse effects in AIDS patients.

A new fever pattern in the presence of changing hematologic parameters may suggest an infection. These parameters include falling hematocrit, rising erythrocyte sedimentation rate, falling or rising white blood cell count. Evaluation should include blood cultures for routine organisms, but also blood cultures for mycobacteria and fungi. Changing lymph nodes and fever may warrant a biopsy to rule out lymphoma, mycobacterial, fungal (especially cryptococcal, histoplasmal, or coccidoidal), or other infection. A chest roentgenogram may reveal an infiltrate not appreciated clinically on physical examination. A careful skin examination is important for new lesions, particularly nodular, pigmented, painful, or fluctuant sign of recent onset. A new subcutaneous "lump" may be the initial sign of staphylococcal sepsis. Biopsing or aspirating such lesions have documented unusual mycobacteria, fungi, bacteria, and even CMV. Since sinus complaints are quite commonly expressed by people with AIDS, evaluation, including sinus films and possibly needle aspiration, may be necessary to define a treatable sinusitis that may be causing fever. A gallium scan may "highlight" lymph nodes or other organs or tissue, such as the pulmonary parenchyma, not clinically abnormal by other parameters. Acute serologic testing to document infectious causes of fever, with the exception of syphilis, may be misleading. For example, IgM antibody to T. gondii in patients with AIDS and central nervous system toxoplasmosis is most often undetected. Antigen detection for cryptococcus may be an early indicator of disseminated disease before more serious, localizing symptoms. However antigen detection methods for other pathogens are still experimental.
IV.B. HIV RELATED LYMPHADENOPATHY

Donald I. Abrams, MD

Of particular interest is the syndrome of persistent generalized lymphadenopathy (PGL). This is defined by the presence of palpable lymph nodes greater than one centimeter in more than two extra-in-guinal sites persistent 6 months or longer. Identification of the human immunodeficiency virus (HIV) has allowed sero-epidemiologic studies to confirm that PGL is an AIDS related condition. Patients with PGL demonstrate nearly uniform positivity in testing for antibodies to HIV. Similarly, patients with PGL are apt be HIV positive in culture. The helper T-lymphocyte is the major target for the retroviral infection. Patients with lymphadenopathy syndrome generally have greater numbers of the T-helper lymphocytes than those who already have an AIDS diagnosis. Therefore, the virus still has viable host cells in which to replicate.

Differential diagnosis includes all those problems which cause lymphadenopathy in the general population. Currently, however, certain conditions are diagnosed with increased frequency in persons at risk of AIDS with diffuse lymphadenopathy. These include:

Kaposi's Sarcoma (KS)
Non-Hodgkins lymphoma (especially B-cell)
Hodgkins disease
Infectious etiologies:
  Cytomegalovirus infection
  Epstein-Barr infection
  Hepatitis B antigenemia
  Lues
  Typical and atypical mycobacterium infection
  Cryptococcus
  Disseminated coccidiomycosis
  Toxoplasmosis
  Persistent generalized lymphadenopathy (benign reactive hyperplasia) (PGL)(LNS)

In addition to lymphadenopathy, PGL patients often complain of associated systemic symptoms. One third of persons with this syndrome may be asymptomatic. Others may have constitutional symptoms such as night sweats, fatigue, fever and weight change (loss and gain). Both subsets will show benign reactive hyperplasia on lymph node biopsy.

Minimal work-up of the patient at risk for AIDS with lymphadenopathy should include:

1) Careful History
   Onset: ? antecedent symptoms; first node noted
   Tenderness of nodes, variation in size
   Past history of sexually transmitted disease
   Medication and recreational drug use (including intravenous use)
   Sexual history: ? exposure to HIV infected individual

2) Physical Examination
   Rule out cutaneous KS
   Rule out skin pathology possibly causing dermatopathic lymphadenopathy
   Rule out thrush, oral KS, lymphoid hyperplasia in pharynx
   Organomegaly
   Perirectal processes - herpes, warts
   Extent of adenopathy
   Patients with limited adenopathy, not diffuse (i.e. only one or two nodal groups involved) require early biopsy to rule out lymphoma.

3) Laboratory Evaluation
   CBC with differential and platelet count
   Erythrocyte sedimentation rate (ESR)
   Liver functions
   Hepatitis B serology
   RPR
   Chest X-ray (patients with lymphadenopathy syndrome should not demonstrate hilar or mediastinal adenopathy)
   Biopsy ? (vida infra)

T-lymphocyte subset analysis is indicated as part of the laboratory evaluation in patients with PGL when therapeutic interventions are being considered, since the total number of T-helper cells is often a criterion for prescription of certain drugs. Some investigators believe useful prognostic information may be gained with those patients with the most depleted helper population being the most likely to progress rapidly to AIDS. T-cell studies have a place in research protocols especially where agents are being evaluated regarding their ability to improve immune dysfunction in this subset of patients. Again, the vast majority of PGL patients have antibodies to HIV and are frequently
IV.C. PULMONARY MANIFESTATIONS OF AIDS

Jeffrey M. Davidson, MD, CM

Presentation
The clinical manifestations of pulmonary disease in AIDS are nonspecific and include fever, cough, and dyspnea. Chest x-ray will often reveal diffuse infiltrates, local densities or cavities, but also may be normal.

Interstitial Disease
The most frequent respiratory problem in AIDS is Pneumocystis carinii pneumonia (PCP). Before the advent of prophylactic therapy, it occurred in 60-70% of patients with AIDS 1. This incidence should decrease with current standards of HIV management. PCP presents with nonspecific clinical findings of variable severity, and most typically with a diffuse perihilar interstitial x-ray pattern. Focal densities and cavitary lesions are seen less frequently, and there may be no evident abnormality. If an interstitial pattern is found, sputum induction and staining for pneumocystis should be performed immediately. If the suspicion of pneumocystis carinii pneumonia is high, empiric treatment should be started at once, pending microbiological results.

Investigations
Oxygenation Studies: As part of the evaluation of pulmonary complaints in an HIV-infected person, a diffusing capacity and an oxygenation study before and after exercise should be done. These values are abnormal in more than 90% of cases of pneumocystis carinii pneumonia.

Gallium Scan: In the case of a normal or borderline chest x-ray, the gallium scan is very sensitive to the changes of pneumocystis carinii pneumonia. A Grade 3+ or 4+ result is highly suggestive, but nonspecific. The scan may be positive even before there are symptoms or x-ray findings of PCP.

LDH: The LDH concentration is usually elevated with pneumocystis carinii pneumonia but this too is very nonspecific.

Sampling for Pneumocystis
Sputum Induction: Since the cough associated with pneumocystis carinii pneumonia is nonproductive, induction of sputum production with nebulized saline is necessary to obtain an adequate sample size. Samples are then lysed with N-Acetyl Cysteine, centrifuged and stained for cytologic identification of pneumocystis trophozoites and cysts. A more sensitive procedure is an immunofluorescent smear using monoclonal antibodies. The yield using this latter technique is greater than 90%

Pneumocystis is not a commensal organism. Except for the case of convalescent PCP, the presence of any pneumocystis forms in sputum, washings or lung tissue is pathological. In a recent study of asymptomatic seropositive patients with normal chest x-rays, none of the subjects showed evidence of pneumocystis on bronchoscopy with broncho-alveolar lavage and transbronchial biopsy 2.

Other Common Pathogens in Sputum: Although fungi and acid fast bacilli can be recovered from induced sputum, it is not known whether this method is as effective as bronchoscopy in recovering these organisms.

Brocho-alveolar Lavage versus Transbronchial Biopsy: Bronchoscopy with broncho-alveolar lavage (BAL) is 90-97% sensitive in detecting pneumocystis carinii pneumonia 3. It should be performed when sputum induction is negative for PCP in individuals suspected of having PCP or other opportunistic lung infections. Transbronchial biopsy (TBB) is only slightly more sensitive at 94-97% 4, but since it carries with it the risk of pneumothorax and bleeding, bronchoscopy with BAL should be initially used alone to diagnose pneumocystis carinii pneumonia. Open lung biopsy is rarely necessary in AIDS, and should be considered only for patients who cannot tolerate bronchoscopy with BAL or TBB, or in whom there is progressive disease despite negative bronchoscopic findings.
Kaposi's Sarcoma

When this neoplasm occurs in the lung, it may be recognized by nodules, masses and/or pleural effusions on x-rays. Bronchoscopy may show lesions in the airways. Treatment with chemotherapy or radiation may be helpful.

Spontaneous Pneumothorax:

This condition is being seen more frequently in AIDS, particularly in patients with an acute or past episode of PCP. These patients should be investigated for active PCP. Treatment of a significant pneumothorax requires placement of an intercostal chest tube and pleurodesis [15].

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IV.D. AIDS - GASTROINTESTINAL SYNDROMES

Richard A. Cazen, MD

AIDS-related gastrointestinal disorders are becoming increasingly prevalent and pose a significant challenge both diagnostically and therapeutically, to the practitioner. The manifestations and pathogenetic mechanisms are quite varied in these disorders, but the symptoms, which are often intractable, present a major cause of morbidity in AIDS patients. In addition, nutritional depletion often results from several specific malabsorption syndromes, which even further impairs immunologic defenses against opportunistic infection.

The entities most often associated with AIDS can be broadly classified as follows:

NEOPLASMS:
1. Kaposi's sarcoma
2. Intestinal lymphoma
3. Anorectal carcinoma

OPPORTUNISTIC INFECTIONS
1. Cytomegalovirus
   -Pancolitis morphologically resembling ulcerative colitis, including usual complications (bleeding, fistula formation, toxic megacolon)
2. Candida albicans
   -Pharyngoesophagitis
   -Liver infection
3. Cryptococcus neoformans
   -Liver infection
4. Mycobacterium avium-intracellulare
   -Enteritis/malabsorption
   -Liver infection
5. Cryptosporidium species
   -Watery diarrhea syndrome
   -Acalculous cholecystitis
6. Isospora belli (Coccidiosis)
7. Strongyloides stercoralis
8. Herpes simplex
   -Anorectal involvement
   -Pharyngoesophagitis

SYNDROMES OF UNKNOWN ETIOLOGY
1. AIDS-related enteropathy/wasting syndrome
2. Idiopathic ulcerative esophagitis
3. Liver function abnormalities
4. Sclerosing cholangitis
5. Peliosis hepatitis
6. Idiopathic proctocolitis
7. Granulomatous hepatitis

INFECTIONS SEEN MORE COMMONLY IN BUT NOT LIMITED TO PATIENTS WITH AIDS
1. Recurrent Shigellosis
2. Recurrent Campylobacter enterocolitis
3. Salmonellosis (nontyphoidal) with dissemination

GENERAL EVALUATION

The presence of unexplained gastrointestinal symptoms in patients either previously diagnosed or considered at high risk of having AIDS should be suspect. The most common symptoms include diarrhea, flatulence, unexplained weight loss, anorexia, anal pain and discharge, and dysphagia or odynophagia. The physician must take a careful history which should include inquiries concerning the appearance of new skin lesions, fevers, chills, night sweats, jaundice, abdominal pain, and gastrointestinal bleeding. A careful sexual history including problems with prior sexually transmitted diseases, particularly hepatitis, intestinal parasites, and herpes simplex infections, is mandatory.

The physical examination should specifically include careful inspection for skin lesions suggestive of Kaposi's sarcoma and herpes simplex, icterus, lymphadenopathy, and signs of protein-calories malnutrition. The oral cavity should be inspected for suspicious lesions, and the presence of oral thrush, which is highly suggestive of immunodeficiency and may correlate with the presence of monial esophagitis in a dysphagic patient. The abdomen should be examined in the usual careful manner with particular attention paid to hepatosplenomegaly, abnormal masses, and signs of ascites. All patients should have a digital rectal and anoscopic examination with tests for occult blood and cultures as clinically indicated. Patients who present with diarrhea should routinely undergo an unprepped proctosigmoidoscopy with collection of stool specimens, especially if prior examination of the stool for enteric pathogens and parasites has been negative.

Routine laboratory examination should include a complete blood count, erythrocyte sedimentation rate, liver function tests, hepatitis and syphilis serologies, serum proteins, and a prothrombin time. A minimum of three stool specimens should be obtained for ova and parasite examination and a stool sample cultured for enteric pathogens including Campylobacter in patients with diarrhea. If the above stool tests are unrevealing and the patient's diarrhea persists, then a duodenal aspirate for Giardia examination can be performed and additional stool samples for Cryptosporidia using a sugar flotation or modified acid-fast technique can be obtained. The latter test must be specifically requested since it is not usually included in routine parasite examination.
associated with nonspecific histopathologic abnormalities involving the small intestine and colon. The D-xylose test is generally mildly diminished. One recent study suggests that small bowel bacterial overgrowth is present in most of these patients. Therefore, empiric therapy with a broad spectrum antibiotic such as tetracycline should be considered in patients with persisting gastrointestinal symptoms or signs of malabsorption in which no specific etiology has been found.

THERAPY

Therapy for gastrointestinal syndromes obviously should be directed at any organism which can be isolated. The relationship between nonpathogenic amebic parasites and AIDS and whether or not they should be treated is controversial.

DHPG (Ganciclovir, Syntex), may induce remission in as many as 75% of patients with enterocolitis secondary to cytomegalovirus infection. This drug, however, generally requires lifelong maintenance intravenous therapy. Patients with cryptosporidiosis first should be treated symptomatically with the usual antidiarrheal drugs, including codeine and paregoric. However, there have been encouraging reports concerning the efficacy of difluoromethylornithine (DFMO) in the treatment of cryptosporidiosis. Intestinal Isospora belli infection, which causes a profuse watery diarrhea syndrome hallmarked by acidosis and hypokalemia, can be successfully treated with trimethoprim-sulfamethoxazole, which should be continued prophylactically for the life of the patient. Ketoconazole given orally or intravenously may be helpful, in addition to topical therapy, in the treatment of monilial esophagitis, but amphotericin B may be required in very severe resistant cases. Acyclovir, orally or intravenously, is the drug of choice for severe herpetic esophagitis or proctitis, and may be taken to prevent painful recurrences.

Toxic complications from drug therapy involving the digestive tract are common, particularly toxic hepatitis from trimethoprim-sulfamethoxazole and acute hemorrhagic pancreatitis from pentamidine. The scope of the present discussion, however, does not permit fuller consideration of these problems.

Nutritional support is essential in all AIDS patients. In those cases where the patient's condition does not permit oral feedings, total parenteral nutrition can be of great value in selected patients. However, compared to other patients, AIDS patients in general have done poorly on chronic parenteral nutrition, and cases of recurrent bacterial sepsis and endocarditis as a complication of chronic indwelling venous catheters have been reported.
IV.E. NEUROLOGICAL COMPLICATIONS OF HIV INFECTION

Jerome Goldstein, MD

INTRODUCTION

The Human Immunodeficiency Virus (HIV) is now known to be both lymphotrophic and neurotropic. It is also known that 20-30% of HIV infected persons will present initially with a neurological complaint; that 40-50% will carry a neurological diagnosis at some time during the illness; and that 80% of autopsied cases will reveal central nervous system (CNS) involvement with HIV. Therefore, careful evaluation is mandated when the HIV infected person presents with neurological symptoms.

PRIMARY CNS DISORDERS ASSOCIATED WITH HIV
(These are presumed to be due to the direct effect of HIV.)

ASEPTIC MENINGITIS - seen during the period of seroconversion. This may be recurrent.

VASCULAR MYELOPATHY - presents as a cord lesion with a spastic paraparesis and may be associated with dementia. HIV has been cultured from the CSF of such patients.

HIV ENCEPHALOPATHY IN BOTH CHILDREN AND ADULTS marked by failure to reach normal milestones in children and the presence of a progressive dementia in adults. Presentation in adults may be subtle. Cognitive impairment is present early in the disorder, requiring psychological testing to define the problem. Later in the disease progression to obvious memory impairment is noted along with motor signs. Changes in the CT and MRI scans are those of atrophy. The CSF demonstrates possible pleocytosis but most often an elevation of the IgG index. Histopathological changes are noted throughout the brain and spinal cord structures.

OPPORTUNISTIC VIRAL INFECTIONS

PROGRESSIVE MULTIFOCAL LEUKOENCEPHALOPATHY - caused by the papovirus and usually presents with altered mental status, hemiparesis, ataxia, visual field loss and other focal deficit. This condition is best diagnosed by MRI Brain Scan which reveals multifocal areas of demyelination in the white matter of the brain. Survival is usually 6 months or less. Antiviral therapy usually has failed with cytarabine, acyclovir, and zidovudine.

HERPES SIMPLEX INFECTION - presents as encephalopathy or myelopathy.

HERPES ZOSTER INFECTION - may cause encephalomyelitis

CMV INFECTION - may cause encephalitis or retinitis - DHPG (ganciclovir) therapy has been tried and is often effective with CMV retinitis.

OPPORTUNISTIC NONVIRAL INFECTIONS

TOXOPLASMOSIS - may lead to focal and multiple granulomas presenting with seizure activity and/or focal neurological deficit. Diagnosed as ring-enhancing lesions on CT scan but better seen on MRI Brain Scan. Brain biopsy is helpful in the diagnosis. Treatment with Pyrimethamine and Sulfadiazine has been useful, even on an empiric basis.

CRYTOCOCCUS NEOFORMANS - usually presents as a meningoencephalitis and is diagnosed by lumbar puncture. The condition is treated with Amphotericin B.

CANDIDA ALBICANS - usually presents as an abscess.

ASPERGILLUS FUMIGATUS OR COCCIDIOIDES IMMITIS - can present as meningoencephalitis or multiple abscesses.

MYCOBACTERIUM AVIUM INTRACELLULARAE (MAI), LISTERIA, AND NOCARDIA - can present as meningoencephalitis or brain abscess.

PRIMARY CNS LYMPHOMA - is seen in single or multiple lesions on either CT or MR scanning. These may require brain biopsy to be distinguished from CNS opportunistic infections. Treatment with radiation therapy or chemotherapy has yielded poor results.

CEREBROVASCULAR DISORDERS - can be caused by thrombotic, embolic, or hemorrhagic events of the CNS.

HIV RELATED NEUROMUSCULAR DISORDERS

NEUROPATHIES

ACUTE GUILLAIN-BARRE SYNDROME - may occur without any other manifestations of AIDS. This has been reported in primary HIV disease.

CHRONIC INFLAMMATORY DEMYELINATING POLYNEUROPATHY - may occur without other evidence of AIDS. This presents with slowed nerve conduction velocities with conduction block, elevated CSF protein, and perivascular inflammation on nerve biopsy.
Magnetic Resonance Imaging is clearly the imaging procedure of choice in the evaluation of central nervous system symptomatology in the face of immune deficiency. This is particularly true when evaluating a focal area missed on CT scan when meningoencephalitis has been considered. It is helpful in identifying areas of demyelination in persons with progressive multifocal leukoencephalopathy.

Spinal cord involvement should be considered if there is a clinical picture and neurological examination of relevance. A careful, detailed neurological examination again is essential. MRI scanning is the diagnostic procedure of choice if cord involvement is suspected. This procedure will not only be helpful in evaluating involvement of the cord substance but also will be useful if there is unrelated pathology such as discogenic disease. The same care in the evaluation of the spinal fluid should be taken when the cord is being evaluated as in intracranial evaluation.

When there is peripheral nerve involvement, the most common complaint noted in persons with acquired immune deficiency is that of distal paresthesiae. Although there can be accompanying distal weakness, it is much less common. The presence of neuropathic symptoms can be a very early sign. The evaluation therefore should include a search for the more common metabolic causes for peripheral neuropathy, and, where applicable, electromyography with motor and sensory nerve conduction studies.

Myopathic symptoms including proximal leg weakness is best evaluated with appropriate muscle enzyme studies and muscle biopsy.

To date, there is no curative therapy for any of the neurological complications of the acquired immune deficiency syndrome. The evaluation and treatment of any of the above mentioned disorders of the central and peripheral nervous system can result in weeks to months of remission.

REFERENCES


IV.F. DERMATOLOGIC MANIFESTATIONS OF HIV INFECTION

Jim Groundwater, MD

Cutaneous disorders are common in HIV infection. These disorders may be the first to bring the patient to the attention of the medical community and they may help to fulfill the requirements for the CDC definition of AIDS. Caldiron and Bergstresser assessed 100 serial HIV positive patients in a county clinic and found that 92% had skin disorders, and there was little difference in prevalence whether the patients had AIDS, ARC or were asymptomatic. However, it appears that the greater the depletion of T-helper cells, the more likely the dermatologic manifestations. Tiana et al found that with one observation, their 20 AIDS patients had an average of 3.7 symptomatic or clinically significant cutaneous disorders per person.

Cooper et al, Lindskow, et al and others have described a transient macular eruption in association with suspected acute AIDS retrovirus infection. This usually is preceded by fever, sometimes sore throat, malaise, abdominal pain, and diarrhea. The lesions are 4-6mm erythematous papules, which are darker in the center. Involvement is primarily head, neck and trunk, but scattered lesions are seen on the extremities. It is said over one-half of HIV infected patients experience the eruption, but it may be overlooked. It generally occurs within six weeks of exposure and clears in less than two weeks.

Among the neoplastic conditions associated with HIV infection, Kaposi's sarcoma has been most common. Suffice it to say that from a cutaneous standpoint, both liquid nitrogen spray and intralesional vinblastine, usually at 0.3 mgms/cc, have been effective palliative measures. Non-Hodgkin's lymphoma, including mycosis fungoides, has been seen cutaneously. There also appears to be an increased frequency of basal cell and perhaps squamous cell carcinoma of the skin. There are reports of melanomas occurring in HIV infected patients.

Pediatric HIV infection results in similar skin manifestations with fewer neoplasms and more infections. Skin manifestations of nutritional deficiencies are seen more frequently in children than in adults.

An acneiform folliculitis is reminiscent of childhood acne. It may be difficult to treat. An unusual, often itchy eosinophilic pustular folliculitis often becomes confluent with some clearing centrally and pustular lesions at the periphery of the resultant plaques. It was first described in non-HIV infected patients in Japan and has been seen with some frequency in HIV infected patients. Some of these cases have responded to astemizole (Hismanal).

Staphylococcal lesions are extremely common in HIV infection. Persistent or recurrent low-grade staphylococcal folliculitis of the beard and other areas has been seen in HIV infections. In addition, a peculiar superficial staphylococcal bullous impetigo, usually of the axillae and groin has been noted. CDC phage typing of 10 such cultures revealed a spectrum of phage types. It may be germane that over 50% of HIV infected patients have cultured staphylococcus aureus from the groins. This is more than double the figure for the general population. Some patients present with recurrent furunculosis, ecthyma, ulcerations or peri¬rectal abscesses. All but the deeper infections respond well to chlorhexidine washes (Hibiclens) and clindamycin lotion (Cleocin T) or mupirocin ointment (Bactroban). Since the staphylococci may be resistant to penicillin, tetracycline, and/or erythromycin, dicloxacillin is preferred for the deeper lesions. Methicillin resistant organisms have been seen. Quinolines with or without rifampin may be helpful in such cases.

Persistent oral candidiasis may imply severe immuno¬deficiency whereas a positive culture alone may not. Oral carriage of candida albicans may be nearly universal. Nail and digital web involvement are also not uncommon. Although antifungals are usually helpful, ketoconazole may be required and recurrences are common. There may be esophageal involvement which implies more severe immunodeficiency.

Hairy leukoplakia of the mouth, particularly the sides of the tongue, is very commonly seen in HIV infection. Whitish corrugated plaques, which are usually asymmetric and asymptomatic, are characteristic. Histologically, hair-like keratotic projections are seen. Epstein Barr and papilloma viruses have been associated. 83% of patients with hairy leukoplakia progress to overt AIDS within 31 months. Acyclovir can be helpful.

A rapidly progressive periodontitis can be seen. This may evolve into acute necrotizing ulcerative gingivitis.
REFERENCES


V. SAFE SEX GUIDELINES
FOR PERSONS AT RISK FOR HIV INFECTION

Scientific Affairs Committee
Bay Area Physicians for Human Rights
Scientific Advisory Committee
San Francisco AIDS Foundation

PURPOSE

This discussion attempts to assign degrees of risk to various sexual practices with respect to the transmission of the human immunodeficiency virus (HIV) 1, but does not necessarily apply to transmission of other sexually transmitted diseases. It also discusses ways in which individuals at risk can help protect the integrity of their immune systems regardless of previous exposure to HIV.

As physicians, we recognize and affirm the basic need for healthy sexual expression. In the midst of this deadly epidemic, it becomes even more important to encourage touching, physical intimacy, and emotional closeness. While the following guidelines are intended to help protect us from the transmission of the HIV, we also want to emphasize that healthy sexual expression in itself nurtures physical and emotional well-being.

WHAT IS AIDS?

Acquired Immune Deficiency Syndrome (AIDS) is caused by a virus which infects and damages the immune system. Since 1983, this virus has been named by various researchers as: lymphadenopathy associated virus (LAV) 2; human T-lymphotropic virus type III (HTLV-III) 3,4; and AIDS associated retrovirus (ARV) 5. In 1986, it was renamed human immunodeficiency virus (HIV) and will be referred to as HIV in this discussion. HIV is capable of becoming incorporated into the genetic material of the cell it infects and for this reason likely will remain in the infected individual for life. It is not known whether everyone infected with HIV will develop AIDS, the most severe form of infection with this virus. If an individual is infected with HIV, the immune system usually makes antibodies to it within a few weeks to six months. A small number of infected individuals may require a longer time to form the antibodies. These antibodies apparently are not protective against the disease. Most persons testing positive for the antibody to HIV are found to have live virus and are assumed to be capable of transmitting the virus.

WHO IS AT RISK?

People who are at significant risk include: gay or bisexual men, intravenous needle users, blood and blood product transfusion recipients between 1978 and 1985, sexual partners of the above groups, and children born to HIV-infected women. All sexually active persons may unwittingly have come in contact with the virus, leading to infection.

Those, since 1978, who have been in a totally and mutually monogamous situation, have not shared needles with others, have not received blood or blood product transfusions during that time, or who were not born to infected mothers are generally not considered to be at risk.

BASIS FOR GUIDELINES AND MODES OF TRANSMISSION

As in previous versions of RISK REDUCTION GUIDELINES our knowledge of transmissibility and the risk of various activities is limited to results of completed studies of HIV and related viruses, as well as recent preliminary observations. While it was originally thought prudent to caution against contact with all body fluids which might contain HIV, additional data reported recently allow some refinement of the recommendations for risk reduction. It must be recognized that our objective epidemiologic experience is still limited to a relatively small number of people and short periods of time for observation. These recommendations may require modification based on future experience.

The evidence is overwhelming that the HIV is not transmitted through social or airborne contact, food, drink or insects. It can be stated that transmission has only been documented from infected blood, semen, vaginal secretions, and possibly breast milk. Moreover, these secretions must be introduced into the body and gain access to the bloodstream. Recognizing this and understanding the limited modes of transmission is the first and most important step in limiting the spread of infection.
Although the mode of transmission has not been precisely determined, there appears to be high risk of transmission of HIV from an infected mother to her baby before, during, or after birth 23,29,30,31. Since infection may be present in the mother without obvious outward signs, the use of the HIV antibody test for both parents is urged before conception is considered (if either partner is at risk for AIDS) and conception postponed if either partner tests positive. If HIV infection is detected in the mother after conception, some have considered therapeutic abortion because the frequency of transmission to the child is very high. Pregnancy may increase the likelihood of progressing to full-blown AIDS in the infected mother. Since virus is found in breast milk 37, breast feeding by infected mothers may be inadvisable.

PROTECTIVE BARRIERS AND LUBRICANTS:

The covering of contacting surfaces is an effective way of reducing risk by establishing a physical barrier to virus transmission. The use of condoms has been established as an effective means of preventing the spread of various sexually transmitted diseases, both by shielding the tiny abrasions of the skin from virus-contaminated surfaces and by containing semen which may bear the infectious agent 32. Disposable gloves can also protect the user from exposure to the AIDS virus. Thus use of these protective devices reduces risk whenever they are properly applied and used. It must be recognized, however, that some practice may be required to properly put on the device as to maintain its integrity. A torn condom or glove cannot be expected to decrease risk. Care must be exercised in removing the device to prevent spillage and contamination during removal. These devices must never be reused. The use of latex to cover the area has been advocated for oral/vaginal and oral/anal contact. Proper use of protective devices converts a high risk activity to a low risk. Cervical diaphragms may add some small protection to the female partner since virus may more easily enter through the single layer of the cervix than the many cell layers of the vaginal walls.

Lubricants containing nonoxynol-9 or other phenols used for easing entry of penis or hand and inside condoms for comfort, have been shown to kill the AIDS virus in the laboratory 33. Since the efficacy and time required for this action is variable and not well established, lubricants cannot be substituted for condoms or other protective devices, but may be used with them. If lubricants are to be used, they must be water-based to prevent damage to the materials used as a barrier. Petroleum or vegetable oil-based lubricants are not recommended because they may cause the condom to break.

REDUCING RISK THROUGH SAFER SEX PRACTICES:

Reducing risk for AIDS may mean making changes in sexual practices, but it does not mean denying the sexual part of one’s life. The safer sex guidelines below are recommended to all persons in groups at higher risk regardless of the results of the AIDS antibody test because of unknown or inaccurate antibody status of one’s sexual partner, and because of the risk of other sexually transmitted diseases. Sexual practices are classified into four categories: NO RISK, LOW RISK, MODERATE RISK, AND HIGH RISK.

NO RISK: Most of these activities involve only skin-to-skin contact, thereby avoiding exposure to blood, semen, and vaginal secretions. This assumes there are no breaks in the skin.

- Social kissing (dry)
- Body massage, hugging
- Body to body rubbing (frottage)
- Light S & M (without bruising or bleeding)
- Using one’s own sex toys
- Mutual Masturbation (male or external female).
  Care should be taken to avoid exposing the partners to ejaculate or vaginal secretions. Seminal, vaginal and salivary fluids should not be used as lubricants.

LOW RISK: In these activities small amounts of certain body fluids might be exchanged, or the protective barrier might break causing some risk.

- Anal or vaginal intercourse with condom
  Studies have shown that HIV does not penetrate the condom in simulated intercourse 32. Risk is incurred if the condom breaks or if semen spills into the rectum or vagina. The risk is further reduced if one withdraws before climax.
## Viral Markers in HIV Infection

<table>
<thead>
<tr>
<th></th>
<th>Normal Uninfected</th>
<th>Recent Infection (0-6mo)</th>
<th>&quot;Healthy&quot; Seropositive</th>
<th>Symptomatic Seropositive</th>
<th>AIDS Opportunistic Infection</th>
</tr>
</thead>
<tbody>
<tr>
<td>HIV Antibody (ELISA)</td>
<td>-</td>
<td>-,+</td>
<td>+++</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>HIV Antibody, Western Blot</td>
<td>-</td>
<td>-,+</td>
<td>+++</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>HIV P24 Antigen</td>
<td>-</td>
<td>-,+</td>
<td>-</td>
<td>-,+</td>
<td>-,+</td>
</tr>
<tr>
<td>HIV P24 Antibody</td>
<td>-</td>
<td>-,+</td>
<td>+++</td>
<td>+</td>
<td>++</td>
</tr>
<tr>
<td>HIV Viral Culture</td>
<td>-</td>
<td>++</td>
<td>+</td>
<td>++</td>
<td>+++</td>
</tr>
</tbody>
</table>
## Laboratory Tests Commonly Done in HIV Infection

<table>
<thead>
<tr>
<th>Test</th>
<th>Normal Seronegative</th>
<th>&quot;Healthy&quot; HIV Seropositive</th>
<th>&quot;ARC&quot; Symptomatic Seropositive</th>
<th>AIDS Opportunistic Infection (O.I)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>White Blood Count</strong> (WBC)/mm</td>
<td>4,000-11,000</td>
<td>↑,N,↓</td>
<td>N,↓</td>
<td>N,↓</td>
</tr>
<tr>
<td><strong>Lymphocyte Count, Absolute/mm</strong></td>
<td>1000-4000</td>
<td>↑,N,↓</td>
<td>N,↓</td>
<td>↓</td>
</tr>
<tr>
<td><strong>CD4 Cells (T Helper), Absolute/mm</strong></td>
<td>&gt;400 (median+850)</td>
<td>N,↓</td>
<td>↓</td>
<td>↓↓&lt;200</td>
</tr>
<tr>
<td><strong>CD8 Cells (T suppressor), Absolute/mm</strong></td>
<td>250-850</td>
<td>N,↑</td>
<td>N,↑</td>
<td>↓,N,↑</td>
</tr>
<tr>
<td><strong>CD4 Cells % of absolute lymphocyte</strong></td>
<td>30-60%</td>
<td>N,↓</td>
<td>&lt;20%</td>
<td>&lt;10%</td>
</tr>
<tr>
<td><strong>CD8 Cells % of absolute lymphocyte</strong></td>
<td>15-40%</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td><strong>CD4/CD8 ratio</strong></td>
<td>1.0-3.0 (median+1.8)</td>
<td>↓ usually&lt;1.3</td>
<td>↓ usually&lt;0.5</td>
<td>↓↓ usually&lt;0.15</td>
</tr>
<tr>
<td><strong>Hemoglobin (gm %)</strong></td>
<td>men 14-18</td>
<td>N</td>
<td>N,↓</td>
<td>↓</td>
</tr>
<tr>
<td></td>
<td>women 12-16</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Platelets/mm</strong></td>
<td>130,000-400,000</td>
<td>N,↓</td>
<td>N,↓</td>
<td>N,↓</td>
</tr>
<tr>
<td><strong>Sedimentation Rate</strong></td>
<td>&lt;15</td>
<td>N,↑</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td><strong>Beta 2 Microglobulin</strong></td>
<td>&lt;3.0</td>
<td>N</td>
<td>N,↑</td>
<td>↑</td>
</tr>
</tbody>
</table>
The Changing Natural History of HIV Infection

1. Life expectancy of those with CDC defined AIDS 1982: Median life expectancy after diagnosis of PCP: 8 months.
   1989: 19 months; a few have survived 4-5 years.
   Possible causes: survival of those with better resistance; lifestyle with fewer cofactors (i.e. STDs, drugs, etc.); prophylaxis of opportunistic infection, antiviral drugs. (AZT, ddI, interferon alpha)

2. Duration of asymptomatic HIV infection. See San Francisco Clinic study.

3. Presenting opportunistic infection - formerly pneumocystis carinii pneumonia (PCP) in about 70%, now less because of routine prophylaxis against PCP. Cytomegalovirus (CMV) and mycobacteria-avium intracellulare (MAI) infections much more common.

4. Prophylaxis against opportunistic infections.

**Routine:**

<table>
<thead>
<tr>
<th>Infection</th>
<th>Antibiotic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pneumocystis Carinii Pneumonia (PCP)</td>
<td>Aerosolized Pentamidine OR</td>
</tr>
<tr>
<td></td>
<td>Trimethoprim/Sulfa OR</td>
</tr>
<tr>
<td></td>
<td>Dapsone</td>
</tr>
<tr>
<td>Herpes Simplex (if history)</td>
<td>Acyclovir</td>
</tr>
<tr>
<td>Mycobacterium tuberculosis</td>
<td>Isoniazid</td>
</tr>
<tr>
<td>(if history positive skin test)</td>
<td></td>
</tr>
</tbody>
</table>

**Investigational**

- Toxoplasmosis
  - Clindamycin OR
  - Pyrimethamine

- Mycobacteria-avium-intracellulare (MAI)
  - Clofazamine

- Maybe in near future
  - Cytomegalovirus (CMV)
    - Ganciclovir, when oral preparation available
5. Antiviral, FDA approved
   
a. Zidovudine (AZT, Retrovir) - use is established in individuals with CD4 <200; recent studies suggest efficacy in asymptomatic individuals with CD4 <500. Side effects: anemia, neutropenia, myositis (muscle inflammation), mood alteration, gastrointestinal disturbances.
   
b. Interferon alpha - use is correlated with remission or stabilization in Kaposi's Sarcoma.
   
6. Antiviral, investigational
   
a. Dideoxycytosine (ddC) Phase II - associated with peripheral neuropathy, may be alternated with zidovudine
   
b. Dideoxyinosine (ddI) Phase II - associated with peripheral neuropathy, gout, pancreatitis
   
c. Interferon - beta, Phase II
   
d. Soluble CD4, Phase II
   
e. Compound Q - chemotherapy against HIV infected mononuclear cells, Phase I
   
7. Early intervention - increase need for medical visits, multiple drug therapy, high medical expense, visibility of HIV infection.

Disabling Conditions Associated with HIV Infection Which Do Not Meet CDC Definition of AIDS

1. Constitutional:
   
   Fever, unexplained               Common
   Diarrhea, unexplained             Common
   Fatigue                           Very common, especially with CD4 <200
   Pruritis (itch)                   Common, worse with CD4 <200
   Neuropsychiatric                  Very common, may be functional or organic

2. Musculoskeletal
   
   Myositis                          Common, may be drug induced (AZT)
   Neuropathy                        Common, may be drug induced (ddI, ddC)
3. Respiratory System -

- Sinusitis, bacterial
- Pneumonia, bacterial
- Lymphocytic interstitial pneumonia
- Pneumothorax

Common & recurrent  Common & recurrent  Less common  More common after PCP

4. Cardiovascular System

- Cardiomyopathy with congestive heart failure

Less common

5. Digestive System

- Esophagitis
- Nausea
- Diarrhea, unexplained
- Incontinence
- Cholecystitis
- Hepatitis
- Pancreatitis

Common, often herpes or candida  Common, often drug induced  Common  Usually without jaundice may be due to HIV or opportunistic infection  Less common, side effect ddI

6. Genito urinary system

- Glomerulonephritis

Less common

7. Heme and lymphatic system

- Anemia
- Neutropenia (↓ granulocytes)
- Thrombocytopenia (↓ Platelets)

Often AZT related but may be direct effect of HIV or opportunistic infection  As above. Predisposes to bacterial infection  Associated with bleeding; more severe early in HIV infection

8. Skin

- Bacterial infections (impetigo)
- Fungal infections
- Drug eruptions
- Eczema
- Psoriasis
- Itching, dryness
- Seborrhea
- Warts
- Molluscum contagiosum

Very common  Very common  Very common  Common  More common than in general population  Progresses with immunodeficiency  Very common  Common  Very common
9. Endocrine

Adrenal failure
Testicular failure
Lipid abnormalities (↑ triglycerides, ↓ cholesterol)
Diabetes Mellitus
Hypoglycemia

Usually in advanced disease
Associated with IV Pentamidine

10. Neurological

Peripheral Neuropathy
a. Guillain Barre Syndrome
b. Chronic inflammatory demyelinating polyneuropathy (CIDP) Earlier in HIV disease
c. Sensory polyneuropathy Later in HIV disease

Brain and spinal cord problems may be classified as AIDS.

Seizure disorders Common

11. Mental disorders

Difficult to distinguish mild dementia from anxiety and depression

12. Neoplasia

Lymphoma, Kaposi's Sarcoma AIDS diagnosis
Squamous cell carcinoma of anus
Squamous cell carcinoma of tongue

May be associated with increased incidence of other common tumors
AIDS REPORTED CASES
(FROM 7/81 TO 11/30/89)

Total San Francisco Cases: 7,562
Total San Francisco Deaths: 4,941
Total S.F. cases month to date: 148
Total S.F. deaths month to date: 115
Total California cases: 22,865 Cases; 14,292 Deaths
(as of 10/31/89)
Total U.S. cases: 112,241 Cases; 66,493 Deaths
(as of 10/31/89)

S.F. AIDS INCIDENCE AND MORTALITY
BY MONTH OF DIAGNOSIS OR DEATH, 1980–89

MONTH OF DIAGNOSIS OR DEATH, 11/80–11/89

*Reporting for recent months is incomplete
AIDS OFFICE
### AIDS Cases by Transmission Category and Year of Diagnosis, San Francisco, 1981 - 1989(1)

<table>
<thead>
<tr>
<th>Transmission Category(2)</th>
<th>Year of Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Adult/Adolescent</strong></td>
<td></td>
</tr>
<tr>
<td>Homosexual or bisexual male</td>
<td>26</td>
</tr>
<tr>
<td>Intravenous (IV) drug user</td>
<td>9</td>
</tr>
<tr>
<td>Homosexual/bisexual IV drug user</td>
<td>0</td>
</tr>
<tr>
<td>Hemophilic/coagulation disorder</td>
<td>0</td>
</tr>
<tr>
<td>Heterosexual contact(3)</td>
<td>0</td>
</tr>
<tr>
<td>Transfusion recipient</td>
<td>0</td>
</tr>
<tr>
<td>None of the above/Other(4)</td>
<td>1</td>
</tr>
<tr>
<td><strong>Children (0-12 years)</strong></td>
<td></td>
</tr>
<tr>
<td>Hemophilic/coagulation disorder</td>
<td>0</td>
</tr>
<tr>
<td>Transfusion recipient</td>
<td>0</td>
</tr>
<tr>
<td>Child of high risk/AIDS parent(5)</td>
<td>1</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>31</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Transmission Category(2)</th>
<th>Year of Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1988</td>
</tr>
<tr>
<td><strong>Adult/Adolescent</strong></td>
<td></td>
</tr>
<tr>
<td>Homosexual or bisexual male</td>
<td>1314</td>
</tr>
<tr>
<td>Intravenous (IV) drug user</td>
<td>49</td>
</tr>
<tr>
<td>Homosexual/bisexual IV drug user</td>
<td>130</td>
</tr>
<tr>
<td>Hemophilic/coagulation disorder</td>
<td>1</td>
</tr>
<tr>
<td>Heterosexual contact(3)</td>
<td>12</td>
</tr>
<tr>
<td>Transfusion recipient</td>
<td>5</td>
</tr>
<tr>
<td>None of the above/Other(4)</td>
<td>1</td>
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<tr>
<td><strong>Children (0-12 years)</strong></td>
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<tr>
<td>Hemophilic/coagulation disorder</td>
<td>0</td>
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<tr>
<td>Transfusion recipient</td>
<td>0</td>
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<tr>
<td>Child of high risk/AIDS parent(5)</td>
<td>1</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>1536</td>
</tr>
</tbody>
</table>

(1) Cases reported through 11/30/89.
(2) Cases with more than one risk factor (other than the combinations listed in the tables) are tabulated only in the most likely transmission category.
(3) Includes persons who have had heterosexual contact with a person with AIDS or at risk for AIDS and persons without other identified risks who were born in countries in which heterosexual transmission is believed to play a major role although precise means of transmission have not yet been fully defined.
(4) Includes patients on whom risk information is incomplete (due to death, refusal to be interviewed or loss to follow-up), patients still under investigation, men reported only to have had heterosexual contact with a prostitute, and interviewed patients for whom no specific risk was identified.
(5) Epidemiologic data suggest transmission from an infected mother to her fetus or infant during the perinatal period.
# Acquired Immunodeficiency Syndrome (AIDS) Monthly Surveillance Report

## Summary of Cases Meeting the CDC Surveillance Definition in San Francisco

### Cases Reported through - 11/30/89

**Aids cases by year of diagnosis and race - 1981-1989.** *(1)(2)*

<table>
<thead>
<tr>
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</thead>
<tbody>
<tr>
<td></td>
<td>N %</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>White</td>
<td></td>
<td>27</td>
<td>87.1</td>
<td>89</td>
<td>86.4</td>
<td>259</td>
<td>87.8</td>
<td>513</td>
<td>83.4</td>
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<tr>
<td>Black</td>
<td></td>
<td>5</td>
<td>12.9</td>
<td>9</td>
<td>5.6</td>
<td>15</td>
<td>5.2</td>
<td>59</td>
<td>9.7</td>
</tr>
<tr>
<td>Latino</td>
<td></td>
<td>13</td>
<td>58.8</td>
<td>9</td>
<td>4.7</td>
<td>0</td>
<td>0.0</td>
<td>0</td>
<td>0.0</td>
</tr>
<tr>
<td>Asian/Pac I.</td>
<td></td>
<td>0</td>
<td>0.0</td>
<td>0</td>
<td>0.0</td>
<td>0</td>
<td>0.0</td>
<td>0</td>
<td>0.0</td>
</tr>
<tr>
<td>Native Amer.</td>
<td></td>
<td>0</td>
<td>0.0</td>
<td>0</td>
<td>0.0</td>
<td>0</td>
<td>0.0</td>
<td>0</td>
<td>0.0</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>31</td>
<td>103</td>
<td>295</td>
<td>581</td>
<td>886</td>
<td>1317</td>
<td>1554</td>
<td>1536</td>
</tr>
</tbody>
</table>

### AIDS Cases by Race/Ethnic Group, Sex, and Age Group, San Francisco, 1981 - 1989 *(1)*

<table>
<thead>
<tr>
<th>AGE</th>
<th>White Male</th>
<th>White Female</th>
<th>Black Male</th>
<th>Black Female</th>
<th>Latino Male</th>
<th>Latino Female</th>
<th>Asian/Pac I. Male</th>
<th>Asian/Pac I. Female</th>
<th>Native Amer. Male</th>
<th>Native Amer. Female</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 - 4</td>
<td>3</td>
<td>3</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>5 - 14</td>
<td>4</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>15 - 49</td>
<td>732</td>
<td>97</td>
<td>72</td>
<td>9</td>
<td>191</td>
<td>18</td>
<td>142</td>
<td>4</td>
<td>30</td>
<td>9</td>
</tr>
<tr>
<td>50 - 69</td>
<td>1789</td>
<td>201</td>
<td>180</td>
<td>14</td>
<td>136</td>
<td>12</td>
<td>145</td>
<td>4</td>
<td>33</td>
<td>9</td>
</tr>
<tr>
<td>60 +</td>
<td>507</td>
<td>7</td>
<td>19</td>
<td>1</td>
<td>7</td>
<td>0</td>
<td>10</td>
<td>0</td>
<td>9</td>
<td>0</td>
</tr>
</tbody>
</table>

### Notes:

1. Cases reported through 11/30/89.
2. This table cumulates cases by DATE OF DIAGNOSIS rather than DATE OF REPORT. Because of this difference, totals may differ from those in other tables and will change with late reports and new data or information.
3. Age and sex of Native American cases are not disclosed because it would compromise the confidentiality of those cases.
Summary of Cases Meeting the CDC Surveillance Definition in San Francisco
Cases Reported through - 11/30/89

AIDS Cases by initial diagnosis, San Francisco, 1981 - 1989(1). Cases meeting the old case definition for AIDS

<table>
<thead>
<tr>
<th>Initial Diagnosis (old definition)</th>
<th>N</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pneumocystis carinii pneumonia; definitive</td>
<td>3823</td>
<td>50.6</td>
</tr>
<tr>
<td>Kaposi's sarcoma, &lt; 60 yrs.; definitive</td>
<td>1749</td>
<td>23.1</td>
</tr>
<tr>
<td>Non-Hodgkins lymphoma, HIV+; definitive</td>
<td>259</td>
<td>3.4</td>
</tr>
<tr>
<td>Cryptococcosis, extrapulmonary; definitive</td>
<td>229</td>
<td>3.0</td>
</tr>
<tr>
<td>Candidiasis of the esophagus, trachea, bronchi, or lungs; definitive</td>
<td>162</td>
<td>2.1</td>
</tr>
<tr>
<td>Mycobacterium avium complex or M. Kansasi disease, disseminated; definitive</td>
<td>143</td>
<td>1.9</td>
</tr>
<tr>
<td>Cryptosporidiosis, chronic intestinal; definitive</td>
<td>116</td>
<td>1.5</td>
</tr>
<tr>
<td>Cytomegalovirus disease; definitive</td>
<td>102</td>
<td>1.3</td>
</tr>
<tr>
<td>Toxoplasmosis of the brain; definitive</td>
<td>48</td>
<td>0.6</td>
</tr>
<tr>
<td>Herpes simplex virus infection; definitive</td>
<td>37</td>
<td>0.5</td>
</tr>
<tr>
<td>Progressive multifocal leukoencephalopathy; definitive</td>
<td>36</td>
<td>0.5</td>
</tr>
<tr>
<td>Primary lymphoma of the brain, &lt; 60 yrs.; definitive</td>
<td>26</td>
<td>0.3</td>
</tr>
<tr>
<td>Histoplasmosis; disseminated; HIV+; definitive</td>
<td>16</td>
<td>0.2</td>
</tr>
<tr>
<td>Isosporiasis, chronic intestinal, HIV+; definitive</td>
<td>12</td>
<td>0.2</td>
</tr>
<tr>
<td>Lymphoid interstitial pneumonia/pulmonary lymphoid hyperplasia, &lt; 13 yrs.; definitive</td>
<td>2</td>
<td>0.0</td>
</tr>
<tr>
<td><strong>Subtotal (old definition)</strong></td>
<td><strong>6760</strong></td>
<td><strong>89.4</strong></td>
</tr>
</tbody>
</table>

AIDS Cases by initial diagnosis, San Francisco, 1981 - 1989(1). Cases meeting the new case definition for AIDS

<table>
<thead>
<tr>
<th>Initial Diagnosis (new definition)</th>
<th>N</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>HIV wasting syndrome; definitive</td>
<td>184</td>
<td>2.4</td>
</tr>
<tr>
<td>HIV encephalopathy (&quot;AIDS dementia&quot;); definitive</td>
<td>170</td>
<td>2.2</td>
</tr>
<tr>
<td>Pneumocystis carinii pneumonia; presumptive</td>
<td>153</td>
<td>2.0</td>
</tr>
<tr>
<td>Toxoplasmosis of the brain; presumptive</td>
<td>98</td>
<td>1.3</td>
</tr>
<tr>
<td><em>M.</em> tuberculosis, extrapulmonary; definitive</td>
<td>52</td>
<td>0.7</td>
</tr>
<tr>
<td>Kaposi's sarcoma; presumptive</td>
<td>49</td>
<td>0.6</td>
</tr>
<tr>
<td>Cytomegalovirus retinitis with loss of vision; presumptive</td>
<td>39</td>
<td>0.5</td>
</tr>
<tr>
<td>Cryptosporidiosis of the esophagus; presumptive</td>
<td>33</td>
<td>0.4</td>
</tr>
<tr>
<td>Mycobacterial disease (not <em>M.</em> tuberculosis), disseminated; definitive</td>
<td>9</td>
<td>0.1</td>
</tr>
<tr>
<td>Coccidioidomycosis, disseminated; definitive</td>
<td>4</td>
<td>0.1</td>
</tr>
<tr>
<td>Mycobacterial disease (unspecified species), disseminated; presumptive</td>
<td>4</td>
<td>0.1</td>
</tr>
<tr>
<td>Salmonella septicemia, recurrent; definitive</td>
<td>3</td>
<td>0.0</td>
</tr>
<tr>
<td>Pneumocystis carinii pneumonia, HIV-</td>
<td>2</td>
<td>0.0</td>
</tr>
<tr>
<td>Bacterial infections, recurrent, &lt; 13 yrs.; definitive</td>
<td>1</td>
<td>0.0</td>
</tr>
<tr>
<td>Primary lymphoma of the brain, any age; definitive</td>
<td>1</td>
<td>0.0</td>
</tr>
<tr>
<td><strong>Subtotal (new definition)</strong></td>
<td><strong>802</strong></td>
<td><strong>10.6</strong></td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>7562</strong></td>
<td><strong>100.0</strong></td>
</tr>
</tbody>
</table>

(1) Cases reported through 11/30/89.
## Survey of Aerosolized Pentamidine Use in San Francisco County

(As of September 30, 1989)

<table>
<thead>
<tr>
<th>Facility</th>
<th>Total Patients Receiving</th>
<th>Cost Per Treatment</th>
<th>Patients Enrolled in Federal Subsidy Program</th>
</tr>
</thead>
<tbody>
<tr>
<td>San Francisco General Hosp.</td>
<td>140</td>
<td>$251.28</td>
<td>51</td>
</tr>
<tr>
<td>Pacific Presbyterian Medical Center</td>
<td>533</td>
<td>$280.10</td>
<td>42</td>
</tr>
<tr>
<td>Mt. Zion Hosp.</td>
<td>256</td>
<td>$261.46</td>
<td>46</td>
</tr>
<tr>
<td>Davies Med. Ctr.</td>
<td>900</td>
<td>$267.10</td>
<td>0</td>
</tr>
<tr>
<td>Children's Hosp.</td>
<td>130</td>
<td>$207.96</td>
<td>1</td>
</tr>
<tr>
<td>St. Luke's Hosp.</td>
<td>67</td>
<td>$182.00</td>
<td>0</td>
</tr>
<tr>
<td>St. Francis Memorial Hosp.</td>
<td>115</td>
<td>$269.94</td>
<td>0</td>
</tr>
<tr>
<td>St. Mary’s Hosp.</td>
<td>35</td>
<td>$290.00</td>
<td>0</td>
</tr>
<tr>
<td>U of C SF Medical Ctr.</td>
<td>400</td>
<td>$175.00</td>
<td>2</td>
</tr>
<tr>
<td>Kaiser Permanente</td>
<td>558</td>
<td>**</td>
<td>0</td>
</tr>
<tr>
<td>VA</td>
<td>120</td>
<td>**</td>
<td>0</td>
</tr>
<tr>
<td>Alan Levin, M.D.</td>
<td>60</td>
<td>$200.00</td>
<td>0</td>
</tr>
<tr>
<td>Sutter Street Surgery Ctr.</td>
<td>10</td>
<td>$265.00</td>
<td>0</td>
</tr>
<tr>
<td>M. Conant, M.D.</td>
<td>150</td>
<td>$215.00</td>
<td>0</td>
</tr>
<tr>
<td>Stonewall Med. Group</td>
<td>10</td>
<td>$245.00</td>
<td>0</td>
</tr>
<tr>
<td>Caremark Connection</td>
<td>40</td>
<td>$250.00</td>
<td>0</td>
</tr>
<tr>
<td>Virx</td>
<td>10</td>
<td>$185.00</td>
<td>0</td>
</tr>
<tr>
<td>T. Schiller, M.D.</td>
<td>51</td>
<td>$212.00</td>
<td>0</td>
</tr>
</tbody>
</table>

**Total Patients Enrolled in Federal Subsidy Program**: 142

**Total Patients Receiving Aerosolized Pentamidine (A-P) per month including those enrolled in health benefits or clinical trials**: 3595

---

**Key**

1. Total number of patients receiving Aerosolized Pentamidine (A-P) per month including those enrolled in health benefits or clinical trials.
2. Cost per treatment refers to 300 mg. of pentamidine bid in solution.
3. Patients enrolled in federal subsidy program do not pay for drug or surcharges, but are responsible for facility or physician charges, if any.

---

**Source:** Patient Advocates for Necessary Treatment

---

*From Bay Area Reporter*  
November 1989
CUMULATIVE PERCENT WITH AIDS

Figure 1. Kaplan-Meier progression time to AIDS, San Francisco City Clinic Cohort Study

San Francisco Epidemiologic Bulletin, November 1989
### Neurological Complications in AIDS Spectrum Patients

<table>
<thead>
<tr>
<th>CNS Disease</th>
<th>%</th>
<th>Known Medical Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>A. Viral Syndromes</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Atypical Aseptic Meningitis</td>
<td>7</td>
<td>None</td>
</tr>
<tr>
<td>Herpes Simplex Encephalitis</td>
<td>3</td>
<td>ARA-A or Acyclovir</td>
</tr>
<tr>
<td>Progressive Multifocal Leukoencephalopathy</td>
<td>2</td>
<td>Possibly ARA-A</td>
</tr>
<tr>
<td>AIDS Dementia</td>
<td>7</td>
<td>None</td>
</tr>
<tr>
<td><strong>B. Non Viral Syndromes</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Toxoplasma Gondii</td>
<td>32</td>
<td>Pyrimethamine &amp; Sulfa Diazine</td>
</tr>
<tr>
<td>Cryptococcus Neoformans</td>
<td>13</td>
<td>Amphotericin B &amp; 5-Flurocytosine</td>
</tr>
<tr>
<td>Candida Albicans</td>
<td>2</td>
<td>Amphotericin B &amp; 5-Flurocytosine</td>
</tr>
<tr>
<td>Atypical Mycobacteria</td>
<td>2</td>
<td>INH, Ethambutol &amp; Rifampin</td>
</tr>
<tr>
<td>Other</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td><strong>C. Neoplasms</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Primary CNS Lymphoma</td>
<td>5</td>
<td>Radiation Therapy</td>
</tr>
<tr>
<td>CNS &amp; Systemic Lymphoma</td>
<td>4</td>
<td>Radiation Therapy &amp; Chemotherapy</td>
</tr>
<tr>
<td>Kaposi's Sarcoma</td>
<td>.1</td>
<td>Radiation Therapy &amp; Chemotherapy</td>
</tr>
<tr>
<td><strong>D. Cerebrovascular Accidents</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Endocarditis</td>
<td>3</td>
<td>Treat Etiology</td>
</tr>
<tr>
<td>Herpes Zoster Arteritis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Idiopathic Thrombocytopenia</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>E. Neuropathies, Myopathy</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Distal Symmetrical Neuropathy</td>
<td></td>
<td>Amitriptyline</td>
</tr>
<tr>
<td>Chronic Inflammatory Polyneuropathy</td>
<td></td>
<td>Consider Plasma Pheresis</td>
</tr>
<tr>
<td>Vacuolar Myelopathy</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>F. Metabolic &amp; Systemic Changes</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypoxia secondary to PCP</td>
<td></td>
<td>Treat Etiology</td>
</tr>
<tr>
<td>Electrolyte Imbalance</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nutritional Deficiencies</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


Prepared by A. Roccellari, Ph.D.
Department of Psychiatry - SFGH
Table 1
Environmental Safety & Props Check List
Suggestions

1. Reality Orientation Cues
   a. Calendars, clocks
   b. blackboard, day-date
   c. daily activities

2. Lock doors.
   Install new locks for wanderers, bells on door knobs as an alarm system.

3. Nails in window sills to prevent being opened more than 6-3”.

4. Check hot water temperature. Turn water heater down.

5. Open stairwells: install infant gate

6. Remove area/throw rugs.


8. Do not rearrange furniture.


10. Medications/toxic household goods safely stored.

11. Install bright night lights.


13. Medi... with "brain damage", or "memory loss" bracelet/necklace

14. Bars for bathtub

15. Chair for shower.


17. Legal documents stored.

18. Canes, walkers, trapezes for decreased motor function.

19. Tight or constricting clothing (2° to edema).

20. Storage of sharps (knives, scissors, razors, saws, etc).

21. Storage of power tools.

22. Label rooms and objects with large signs, especially bathroom.
Table 2

Early S/SXs / ADC

Symptoms

A. Cognitive
Memory loss (names, historical details, appointments), long term memory usually preserved.

B. Behavioral
Apathy, social withdrawal, "depression", agitation, confusion, hallucinations.

C. Motor
Ataxic, leg weakness, tremor, decreased coordination, impaired handwriting.

Signs

A. Mental Status Examination
Psychomotor slowing, impaired serial 7s or recent memory tasks, "normal" bedside exam, organic psychosis.

B. Neurological Exam
Impaired rapid movements, gait ataxia (impaired tandem gait, rapid turns), leg weakness, hyperreflexia, tremor ("physiological"), dysarthria, impaired smooth pursuit eye movements

Table 3
The Late Manifestations of AIDS Dementia

Symptoms

A. Cognitive
Global dementia, confusion, distractability, delayed verbal responses.

B. Behavioral
Vacant stare, restlessness, disinhibition, organic psychosis.

C. Motor
Slowling, truncal ataxia
Weakness: legs > arms
Pyramidal track signs: apasticity, hyperreflexia, tremor.

Additional Neuro Signs

Myoclonus
Seizures
Incontinence


Prepared by A. Boccellari, Ph.D., Dept of Psychiatry - SFGH.
Table 5
Agitated/Angry Patient

A. Distraction

Primary Distraction

1. Change the subject
2. Refuse to respond to the content.
3. TV/Music often helps.
4. Folding clothes or other simple tasks with no expectation of success (anything with hands).
5. Give simple finger foods.

B. 6. Leave the room for a few minutes, they may forget why they are angry.

C. 7. Remove the patient from the agitating situation.

D. 8. Decrease stimulus.


F. 10. Say NO gently, but firmly.

G. 11. Have the patient evaluated for low dose neuroleptics, i.e. Haldol.
Table 36-4
Guide to Differentiating Dementia from Depression

<table>
<thead>
<tr>
<th>Dementia</th>
<th>Depresssion</th>
<th>Dementia</th>
<th>Depression</th>
</tr>
</thead>
<tbody>
<tr>
<td>Affect</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Observe the client's overall mood and self-presentation throughout the interview. Enlist interpretations from family and caregivers. “How has your mood been lately?”</td>
<td>Depressed, feelings of despair that are pervasive, persistent. May be anxious or hypomanic. Not influenced by suggestions. May be flat, withdrawn, sad, tearful.</td>
<td>Elicit level of orientation regarding person, time, place date, etc. May vary with different times of the day.</td>
<td></td>
</tr>
<tr>
<td>Labile, fluctuating from tears to laughter, not consistent or sustained: may show apathy, depression, irritability, euphoria or inappropriate affect. Normal control impaired, susceptible to content of interview.</td>
<td></td>
<td>Fluctuating with varying levels of awareness. Disoriented for time, place.</td>
<td></td>
</tr>
<tr>
<td>Memory</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ask specific questions geared toward assessing recent and remote memory. Ask client to recall recent events.</td>
<td></td>
<td>May have some confusion, not as profound as in dementia.</td>
<td></td>
</tr>
<tr>
<td>Decreased attention. Decreased for recent events. Confabulation. covers up memory loss. Shows irritability when memory tested. Perseveration, dwells on certain topics inappropriately.</td>
<td>Difficulty in concentration. Impaired learning of new knowledge. Decreased attention with secondary decrease in recent memory. May not respond when tested or will admit can’t remember.</td>
<td>Fatigue, failing health complaints with vague complaints of pain in head, neck, back. Typical complaints as: decreases in sleep, appetite, weight, libido, energy, and constipation.</td>
<td></td>
</tr>
<tr>
<td>Intellect</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Assess the client’s cognitive functioning, taking into account cultural and educational factors, which will influence results. Ask the client to perform simple math equations and solve simple problems.</td>
<td>Impaired, decreased as tested by serial 7s, similarities, recent events. Impaired but can perform serial 7s and usually remember recent events.</td>
<td>Psychotic Symptoms</td>
<td></td>
</tr>
<tr>
<td>Mainly visual. May occur in psychotic depressions. Mainly auditory; hallucinations and delusions of a morbid quality.</td>
<td></td>
<td>Enlist interpretations from family and caregivers. Often (but not always) psychotic material will become evident during interview. (Are you being plotted against? Do you ever see hear smell things others do not?)</td>
<td></td>
</tr>
</tbody>
</table>

Box 18-3

MAJOR DEPRESSIVE EPISODE

Note: A "major depressive syndrome" is defined as criterion A below.

A. At least five of the following symptoms have been present during the same two-week period and represent a change from previous functioning: at least one of the symptoms is either (1) depressed mood, or (2) loss of interest or pleasure. (Do not include symptoms that are clearly due to a physical condition, mood-incongruent delusions or hallucinations, incoherence, or marked loosening of associations.)

1. Depressed mood (or can be irritable mood in children and adolescents) most of the day, nearly every day, as indicated either by subjective account or observation by others.
2. Markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day (as indicated either by subjective account or observation by others of apathy most of the time).
3. Significant weight loss or weight gain when not dieting (e.g., more than 5 percent of body weight in a month), or decrease or increase in appetite nearly every day (in children, consider failure to make expected weight gains).
4. Insomnia or hypersomnia nearly every day.
5. Psychomotor agitation or retardation nearly every day (observable by others, not merely subjective feelings of restlessness or being slowed down).
6. Fatigue or loss of energy nearly every day.
7. Feelings of worthlessness or excessive or inappropriate guilt (which may be delusional) nearly every day (not merely self-reproach or guilt about being sick).
8. Diminished ability to think or concentrate, or indecisiveness, nearly every day (either by subjective account or as observed by others).
9. Recurrent thoughts of death (not just fear of dying), recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan for committing suicide.

B. 1. It cannot be established that an organic factor initiated and maintained the disturbance.
2. The disturbance is not a normal reaction to the death of a loved one (uncomplicated bereavement).

Note: Morbid preoccupation with worthlessness, suicidal ideation, marked functional impairment or psychomotor retardation, or prolonged duration suggest bereavement complicated by major depression.

C. At no time during the disturbance have there been delusions or hallucinations for as long as two weeks in the absence of prominent mood symptoms (i.e., before the mood symptoms developed or after they have remitted).

D. Not superimposed on schizophrenia, schizotypal disorder, delusional disorder, or psychotic disorder NOS.

Treatment & Management of AIDS Dementia

1. Orientation
   - Use clocks, calendars, orientation blackboard, night light, current event groups.

2. Structured Environment
   - The environment should be kept consistent, with routine schedule.
     Sudden changes can cause worsening of confusion.
   - Use sign posts to help pt find way around.
   - Familiarity - keep familiar objects around pt (photos, familiar objects).
   - Speak of familiar names, places, interests pt has had in the past.
   - If possible, have the same staff members assigned to pt.

3. Communication
   - Give pt only 1 thing to do at a time.
   - Info. should be presented slowly and one step at a time.
   - Don't assume pt is following what is being said to them.
   - Repeat instructions and have pt repeat instructions to make sure they understand.
   - If pt can still read, encourage pt to keep track of things by writing them down.

4. Assess Level of Stimulation Needed
   - Too much or too little stimulation can lead to confusion, agitation, fearfulness.
   - Have a few friends/family visit regularly - not a large group at one time.
   - Give pt objects to touch that are of different textures (flannel, silk, etc.).
   - Alternate between use of radio and T.V.
   - If bedridden, consider physical therapy (e.g., passive range of motion)
     or massage.

5. Environmental Support
   - Provide explanation to the pt and significant others about pts condition.
   - Educate family members/friends about what to expect from pt.
   - Provide brief psychotherapy sessions for pt.
   - Evaluate frequently for suicidal ideation, and impulsivity.

6. Medication Issues in treating AIDS organic psychosis
   - Minimize all meds which depress CNS (e.g., analgesics, sedative hypnotics).
   - If needed, treat with phenothiazines.
   - Use high potency, low anti-cholinergic medications & use lower dose than usual.
   - Avoid high anti-cholinergic drugs because:
     a) They can worsen OBS
     b) They also decrease oral secretions that help inhibit the growth of opportunistic infections such as oral thrush

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The Role of Psychiatry: Evaluation and Treatment of the Altered Mental Status in Persons with AIDS

SAMUEL TUCKER

The psychiatric management of AIDS-related psychosis and organic deterioration as an emergent discipline has been only gradually recognized. Although published data about AIDS-related psychiatric problems has been minimal, I fear that a great deal more psychiatric problems will emerge as the epidemic continues. The majority of my information comes from anecdotal case reports in the psychiatric literature, from other colleagues, and from personal experience (Holland, Tross 1985, Hoffman 1984, Kermani, Drab, Alpert 1984, Loewenstein, Scharfstein 1983–84).

AIDS is frequently complicated by central nervous system dysfunctions. (See preceding chapter.) Dementia in the absence of opportunistic infections is also being recognized as a primary presentation of AIDS. It is now strongly asserted that HIV is neurotropic—that is, it attacks the neurons—and it is the probable cause for these progressive dementing processes (Shaw et al. 1985). One report showed that only 19 of the 110 patients were without neurological changes at autopsy (Jordan et al. 1985). I also have anecdotal reports from colleagues that up to 50 to 70 percent of the patients that they have seen in consultation demonstrated certain aspects of dementia.

When a psychiatric consultation is requested on AIDS patients because of mental status changes, one is confronted with the need to assess rapidly whether the cause of the process is potentially treatable. Delineating an etiology is imperative in order to expedite appropriate medical treatment. Opportunistic infections and their concomitant high fevers, polypharmacy, or specific agents such as amphotericin B, which is used for cryptococcosis and other fungal infections, may all foster altered mental status. The psychiatric consultant must be prepared to assess and differentiate deliriums from depression or dementia, which have overlapping symptoms.
Delirious patients may be differentiated by several specific features. A delirium is usually of rapid onset, occurring over some hours to days. Delirious patients tend to show increased psychomotor activity, such as agitated pacing or rocking. Their sleep-wakefulness pattern may also be altered, with more activity at night. Psychotic sensory-perceptual disturbances, including visual, auditory, and somatic hallucinations and persecutory delusions are not uncommon in these people. A clouded sensorium, making any cognitive testing impossible, is also a hallmark of a delirious state. In other words, the patients are grossly disoriented, not knowing where they are or what is happening to them.

Many AIDS patients are presenting with some of the aspects of these symptoms. Deliriums, as a rule, tend to have a traceable and often a treatable etiology. Examples of this are, perhaps, the severe bitemporal headaches of incipient cryptococcal meningitis, or drug toxicity from one or several combined medications that may foster a delirium state.

If a treatable delirium or focal lesion is ruled out, the psychiatric consultant must next differentiate between a functional depression and dementia as the reason for the mental status changes. One-third to one-fifth of all hospitalized medically ill patients manifest some form of depressive symptomatology (Derogatis et al. 1983). The diagnosis of major depression in medically ill patients is clouded because many of the symptoms of depression may also result from their medical illness. Fatigue, weight loss, difficulty in sleeping, and anorexia are all symptoms held in common by AIDS and depression.

To differentiate between depression and a medical etiology, one should emphasize cognitive and affective symptoms while minimizing their somatic concerns (Cavanaugh 1983). Certain aspects of the Beck Depression and Hopelessness Scale are helpful in making this diagnosis. These include presence of low self-esteem, feeling like a failure, loss of interest in other people and relationships, feeling as if being punished for something, recurrent suicidal ideation, difficulty carrying through in making decisions and, also, frequent crying spells. A history of major depression, previous psychiatric treatment, particularly with medication, or suicide attempts in the past also give clues as to whether or not this is a functional depression as opposed to dementia.

Nevertheless, an unexplained onset of depression in AIDS patients who had formerly been coping adequately with their disease may herald the onset of a central nervous system process other than just a functional depression. Case reports in the psychiatric and medical literature describe a dementing process attributable to AIDS. Dementing ill-
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...nesses are usually of an insidious onset; they occur over a long period of time. And they are marked by a clear sensorium in the presence of measurable cognitive deficits. Such deficits include the inability to retain new information, confusion, disorientation, and short- and long-term memory deficits. These parameters are also true for the organicity that is beginning to be seen in AIDS patients.

The case reports describe patients who are confused, with a loss of interest in personal appearance and hygiene, and displaying inappropriate behaviors in public. The spectrum of presentation ranges from a pathetic, passive cooperation—an almost lobotomized attitude—and total unconcern about the situation to temper tantrums, inappropriate disrobing, decreased frustration tolerance, affective lability, anxiety, paranoia, suspiciousness, hostility, and delusional and frankly psychotic thought processes. On examination, most show what are called "frontal release signs" in the absence of any focal neurological findings. Serial EEGs may demonstrate diffuse slowing, and CT scans may show cortical atrophy with a ventricular enlargement. Many of these patients are appearing in psychiatric hospitals without a previous AIDS diagnosis. Only serial cognitive assessments, abnormal EEGs, and CT scans permit the eventual diagnosis of a dementing process resulting from AIDS.

What possible treatments and interventions do we have? They do not raise great expectations. Treatment of delirium primarily entails addressing the underlying etiology. As was mentioned, illnesses such as infectious agents may be treated, which allows the patient to return to the usual level of adaptive functioning. Polypharmacy, causing altered mental status, or the presence of street drugs or much alcohol must also be eliminated in order for the client to clear his sensorium. In the functionally psychotic, severely agitated, or deliriously demented patient, neuroleptic medication may be necessary. High-potency, low-cholinergic medications such as haloperidol or thiothixene at minimal doses are favored to clear these states for several reasons. They reduce agitation and eliminate psychotic ideation without too much sedation or without problems causing orthostatic hypotension, and they do not significantly lower the seizure threshold. Avoiding medication with anticholinergic activity—chlorpromazine and thioridazine are just two—is preferred because the patient has a central nervous system dysfunction and may be taking other medications. Anticholinergic activity can exacerbate the disorientation, causing memory difficulties and even producing a toxic psychosis. Moreover, anticholinergic activity dries the secretions of the oral and pharyngeal membranes, which are an inhibiting mechanism in the growth of opportunistic in-
fections like thrush or Candida. Benzodiazepines, another class of medications, which includes diazepam, should be avoided for a delirious patient, as they tend to increase confusion and agitation by depressing higher cortical function.

The treatment of depression also entails the use of the least anticholinergic medications. The drugs most commonly used are alprazolam and trazedone. Reports indicate that trazedone causes an often reversible process called priapism, and therefore alprazolam is usually the drug of choice. If the patient is severely ill and extremely depressed, when rapid mobilization is needed, you can use a stimulant such as methylphenipate, being aware that insomnia, agitation, and tachycardia are troublesome side effects of such stimulants and also of other stimulants, such as amphetamines.

This discussion about medication should not overshadow the basic importance of various therapy techniques, such as supportive, insight, cognitive, and behavioral models, in order to help patients cope with the extremely difficult process they are undergoing, and also to help dementing patients who still have sufficient cognitive capabilities to understand what is happening to them.

Successful treatment of AIDS dementia entails much more than just medications for all involved. One needs to turn to a parallel literature to find a successful model for the management of the dementing patient—both in the hospital and in the community. The Alzheimer's patient, suffering from another progressive dementing illness, provides one successful model (Powell and Courtice 1985, Reisberg 1983). On an inpatient basis, a staff educated to recognize and care for people who are undergoing a dementing process should be able to contain patients' behavior. In-service programs are imperative to educate nursing staffs in management strategies for dementing patients. This is particularly important in medical wards where nurses may not be accustomed to working with patients with behavioral problems like those seen in organic diseases—especially with AIDS patients, who so often are quite young.

Management on medical wards can be enhanced by placing patients on an involuntary status if the staff feels they are gravely disabled, which means unable to provide housing, food, or shelter for themselves, or by accepting them on an inpatient basis. Involuntary status allows some hospitals to provide round-the-clock sitters to assist nurses in controlling and monitoring difficult behaviors. I am not quite sure if staffs on medical wards know what that means. In psychiatry we often have to place patients on an involuntary basis if they are suicidal, homicidal, or unable to care for themselves. If you place
someone on an involuntary basis, many insurance carriers including MedCal and Medicare will then allow for the payment of continuous sitters, whose presence can greatly enhance nursing care in monitoring difficult behaviors.

On an outpatient basis, the overall strategy is simply to keep patients as functionally autonomous as is medically responsible and feasible. Early on in the AIDS dementia, depending on the level of cognitive impairment and physical stamina, patients may be allowed to go home and may function adequately with minimal assistance. Later, as the disease and the dementing process continue, patients may require closer supervision, eventually even round-the-clock care-givers. This supervision can be an overwhelming burden to lovers, family, and friends, who usually are the ones able and willing to provide this needed service. For Alzheimer's patients and, we hope, for AIDS patients, we are going to have to develop "respite programs." These must be created in order to alleviate periodically the tremendous responsibilities of the care-givers, who can place dementing or sick patients in a hospital or convalescent home for a brief circumscribed period of time.

Educational programs for care-givers about the disease, the responsibilities involved, and how to use community services are crucial. Support groups for care-givers have to be developed; and day-care programs may also have to be developed in the future. Public health and home visiting nurses will have to make regular assessments during the course of the disease. Therefore, these professionals, too, will need extensive in-service programs to learn evaluatory and management skills.

Many factors have to be considered about the personal safety of patients in the home. Considerations include: Can an individual patient dress himself, take his medication, or medications, especially those that may be toxic in combination? Can he take care of laundry, buy food, cook meals, or simply feed himself? Is he able to count money in order to be able to shop? Able to drive a car? To take a cab? Or a bus? Is he able to follow through on any given task? These questions must be assessed in an ongoing manner as the disease progresses.

Reality orientation devices must be placed in the home for the patient's environmental safety. Such devices include very large calendars and signs that define and distinguish rooms and their functions (you would be surprised how many people cannot even remember what is in a room when the door is closed). Signs over the stove that say, for example, "Turn it off," or "Is the gas turned off?" are valuable if these patients forget from moment to moment what to do and where and how to do it. A current picture of patients should be available to iden-
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tify them to the police in case they wander away from home. Firearms and other weapons must be removed. Demented people are frequently paranoid and may also be suicidal. They may accidentally or purposefully hurt themselves or others. These are just a few of the practical tips that help keep someone safely at home as long as is medically feasible and responsible.

In conclusion, the goals of the interventions described are to maintain patients at their highest level of adaptive functioning and to maintain their greatest degree of autonomy, personal integrity, and independence as well as to address and treat those processes that can be treated until the time that it is possible to cure them.

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