Kansas Journal of Medicine

Volume 1, Issue 3, 2008

Table of Contents

Non-Occlusive Mesenteric Ischemia

Bassem M. Chehab, M.D.
Edgard Wehbe, M.D.
Imad I. Nassif, M.D.
University of Kansas School of Medicine–Wichita
Department of Internal Medicine

Introduction

Non-occlusive mesenteric ischemia (NOMI) is an acute mesenteric circulatory disorder that, in contrast to mesenteric arterial occlusion induced by blockage of blood flow by emboli and thrombi, is not caused by organic occlusion of blood vessels.[1] Good outcomes in NOMI are observed with early recognition and treatment.[1-2] The early symptoms and characteristics of NOMI, however, are unclear. In many cases, the disease has advanced to an irreversible stage before a definite diagnosis is made.

Case Report

A 59-year-old female presented with congestive heart failure, secondary to ischemic heart disease. She reported a two-day history of profuse watery diarrhea with mild cramping abdominal pain starting 30 minutes after eating and improving intermittently between meals. She had complained over four months of nausea and vomiting that had increased in frequency and of a 20-pound weight loss. She has been compliant with her medications and no new changes have been made within the last six months.

On physical exam, she was hypotensive with mild diffuse abdominal tenderness. The laboratory investigation showed a high white blood count of 25,000 with a bandemia of 22%.

Mesenteric ischemia was suspected and a CT scan of her abdomen showed diffuse thickened small bowel loops (Figure 1). A CT angiogram of her abdomen revealed patent mesenteric vessels (Figure 2).

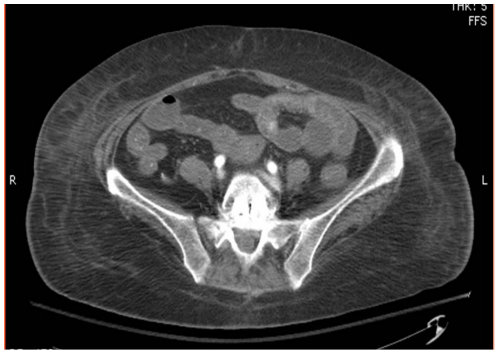

Figure 1. CT scan of the abdomen showed diffuse thickening of small bowel loops.

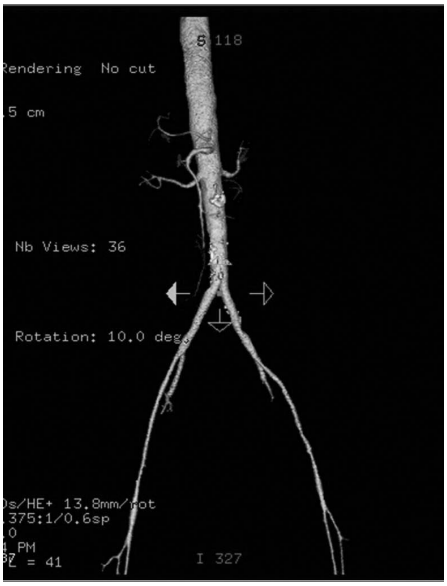

Figure 2. CT angiogram with 3-dimentional reconstruction showed patent mesenteric vessels.

A colonoscopy (Figure 3) showed necrosis from the anal margin to the left splenic margin, necrosis of the cecum and terminal ileum with preserved mucosa of the transverse and right colon consistent with a diagnosis of NOMI.

The patient went into septic shock and expired after one day.

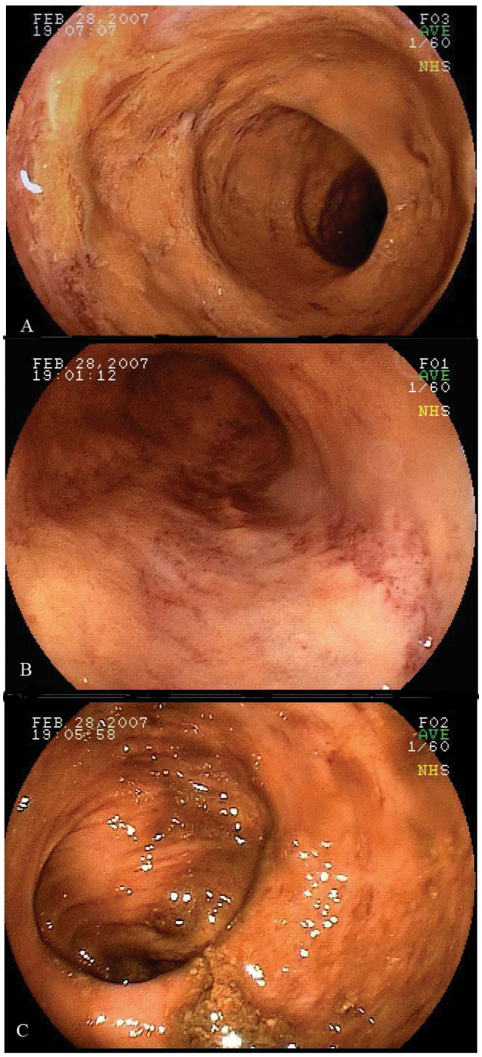

Figure 3. (A - B) Colonoscopy at the level of the sigmoid and splenic flexure showed a pale mucosal with diffuse ischemia, scattered shallow irregular ulcerations, longitudinal and irregular in form with gray-yellow exudates. (C) A diffuse ischemic mucosa of the colon is seen with overlying exudates at the hepatic flexure.

Discussion

NOMI is the result of splanchnic vasoconstriction occurring in response to a variety of systemic insults that diminish mesenteric blood flow.[1,3] The macro-vasculature is patent, but the microvascular blood flow is inadequate to meet intestinal tissue demands leading to gangrene. The consequences are disastrous and the prognosis is very poor, despite the absence of organic obstruction in the principal arteries.[1,4] NOMI accounts for more than 10% to 20% of cases of acute mesenteric circulatory disorders, mainly in elderly patients, with a mortality rate of 70% to 90%.[2-3]

The pathophysiology of NOMI involves low blood flow states such as shock, heart failure, hemodialysis, and direct splanchnic arteriolar vasoconstriction by drugs (e.g., digoxin).[2-3,5] Intestinal vasospasm due to persistent low perfusion is thought to be the inciting factor. NOMI can present with abdominal pain, nausea, vomiting, and ileus, but the characteristic early symptoms and laboratory test results are unclear. Early diagnosis is difficult and during the diagnostic process the disease slowly advances to an irreversible state with extensive intestinal necrosis.[2,4-5]

Angiography is the gold standard for diagnosis. Its invasive nature and potential for contrast nephropathy, however, makes angiography a less than optimal screening tool, thereby missing the opportunity for resolution in many cases.[1-2,5] For definite diagnosis, the absence of organic obstruction of blood vessels distributed in the necrotic intestinal region and segmented discontinuous intestinal and colonic ischemic changes with necrosis on colonoscopy or laparotomy are required.[3-4] However, the time required for definite diagnosis may compromise the chances of survival.[3]

The endoscopic feature in NOMI is segmental distribution with a clear boundary between the injured and uninvolved region. The lesions could range from marked edematous mucosa with loss of clear vascular vessel pattern to scattered shallow irregular ulcerations, longitudinal or irregular in form, with gray-yellow exudates.[8]

The role of colonoscopy is limited to the evaluation of the mucosal severities and the extent of the disease. It may be helpful in predicting clinical status and the prognosis of the patients.[6-8] It is safe and helpful in the early phase but should be performed with great care because increased pressures from insufflations could induce new ischemic lesions.[7-8]

Recently, abdominal contrast multi-detector row computed tomography upon suspicion of NOMI has emerged enabling a rapid definite diagnosis and providing vascular information comparable to that obtained in angiography. It permits subsequent early initiation of therapy and monitoring of disease resolution.[1,9]

The initial treatment is to correct predisposing or precipitating causes. Relief of acute congestive heart failure, correction of unstable or new cardiac arrhythmias, and replacement of blood volume should precede any diagnostic studies.[3-4] The main goal of current therapy for NOMI is reduction of spasm and improved perfusion of the mesenteric artery mainly with continuous administration of vasodilators into the mesenteric artery such as papaverine, prostaglandin E1, and nitroglycerine. The role of surgery is limited to diagnostic laparotomy and excision of irreversibly necrotized intestine.[3,10]

Conclusion

NOMI is increasingly more common due to the aging of the population, but the disease concept has not been established fully. Moreover, NOMI is difficult to diagnose, lacks characteristic symptoms, and is fatal in the advanced stage. Therefore, many patients may not have been diagnosed correctly and consequently may have died without receiving adequate treatment. Prognosis is related to the time of treatment initiation. Early diagnosis in suspected cases and early initiation of treatment may increase survival of NOMI patients.

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Keywords: mesenteric ischemia, non-occlusive mesenteric ischemia, ischemic colitis, endoscopy, colonoscopy.

Cough-Induced Syncope as an Unusual Manifestation of Pericardial Effusion

Hazem El-Osta, M.D.[1]
Salman Ashfaq, M.D.[1,2]
[1]University of Kansas
School of Medicine–Wichita
Department of Internal Medicine
[2]Heartland Cardiology, Wichita, KS

Introduction

Cough-induced syncope belongs to the heterogeneous group of situational syncopes. It consists of the loss of consciousness associated with a paroxysm of cough. It was first described by Charcot in 1876,[1] but the underlying mechanism remained a mystery until recently. While many clinical cases of cough syncope have been described in the literature, this report describes a unique case of cough-induced syncope in a patient with a newly-discovered pericardial effusion with near tamponade.

Case Report

A 70-year-old male presented with a five-day history of multiple episodes where he lost consciousness during or after cough. The syncopal events lasted for durations ranging from 30 seconds to about one minute. The patient also reported a two-week history of pleuritic chest pain, shortness of breath upon exercise, and cough. He initially presented with these symptoms to another facility where a computed tomography of the chest revealed a moderate to large pericardial effusion. He was transferred to our facility for further evaluation.

Upon admission, the patient was stable. His labs were unremarkable. A trans-thoracic echocardiogram showed a large pericardial effusion measuring 2.5 to 3.2 cm in maximal dimension (see Figure 1). He manifested early right atrial collapse, with no clear evidence of right ventricular diastolic collapse. The patient did not meet criteria for pericardial tamponade. He simply had a large pericardial effusion. A sizeable amount of fibrinous material was noted within the pericardial space adjacent to the visceral pericardium.

During his hospital stay, many witnessed episodes of cough-induced syncope occurred. None were associated with bradycardia, arrhythmia, or block on telemetry. During one episode, his systolic blood pressure showed a reduction from 110 mmHg to 80 mmHg. The blood pressure returned to baseline as soon as the symptoms resolved.

A biopsy of the fibrinous material was completed, without any complications, by performing a pericardial window. Approx-imately one liter of blood-tinged pericardial fluid was drained. Cytology of the fluid and biopsy of the fibrinous material were negative for malignancy. Cultures performed on the fluid also were negative for infection. No other systemic inflammatory process was diagnosed, although no diagnostic viral test was obtained. A viral pericarditis complicated by the development of a large pericardial effusion was thought to be the most likely diagnosis. Interestingly, after drainage of the fluid, the patient experienced complete relief from the cough-syncope symptoms.

He was discharged a few days later for outpatient follow-up. He has had no more syncopal episodes since his pericardial effusion was drained.

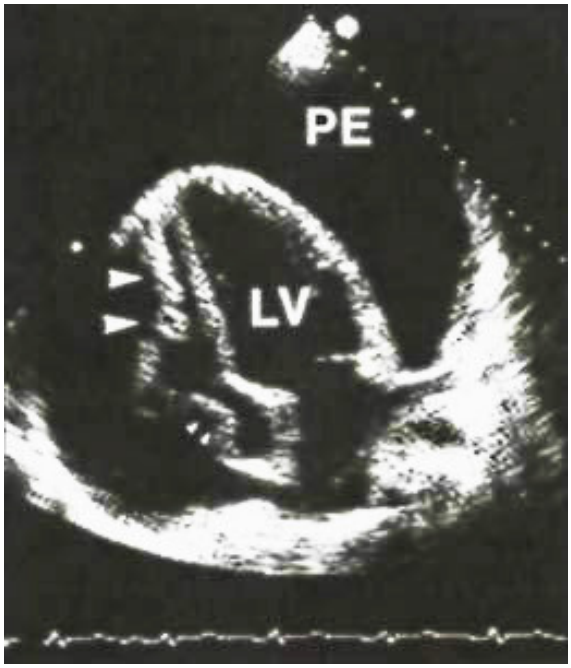

Figure 1. Pericardial effusion is evident on the two-dimension echocardiogram.

Discussion

Cough syncope is a well-recognized but uncommon phenomenon. The exact mechanism by which it occurs remains controversial and multiple theories have been put forward.[1] The most widely accepted theory is that the syncope occurs as a result of hemodynamic changes induced by the increase in the intrathoracic pressure associated with cough which can in turn limit venous return to the heart. Lowered cardiac output and blood pressure result in cerebral hypoperfusion. In some cases, impairment of the cerebral venous return precipitates cerebral hypoperfusion without necessarily a decrease in blood pressure.[2-4]

Arrhythmias also can cause cough-induced syncope. For example, atrioventricular conduction block due to hypersensitive broncho-pulmonary reflex, a neurally-mediated reflex contributing to an inappropriate chronotropic response or a premature ventricular complex, may cause cough-induced syncope.[1,5-8]

Cough-induced syncope in the patient described above was associated with hypotension. Bradycardia or arrhythmias were not documented during the episodes. Impediment of cardiac filling may have resulted from the superimposed cough causing an increase in the intrathoracic pressure. The increased intrathoracic pressure may have caused a rise in intra-pericardial pressure which likely changed the RV filling from a pre-tamponade state to a true tamponade physiology for a few seconds. The tamponade would cause a lowering in systemic blood pressure, which likely reduced cerebral perfusion causing the pre-syncope and syncope symptoms. After the coughing, the intra-pericardial pressure, the systemic blood pressure, and the cerebral perfusion return to the baseline levels and the symptoms resolve. In this patient, the syncopal episodes subsided completely after drainage of the pericardial fluid, thus supporting the correlation between the pericardial effusion and the syncopal episodes.

This case is the only known example of cough-induced syncope due to a pericardial effusion in the literature. Cough-induced syncope was described in a subject with constrictive pericarditis which has some common basic pathophysiology with pericardial effusion.[8]

Conclusion

This case presented a novel cause of cough-induced syncope. It is important to consider pericardial effusion in any case of new-onset cough-induced syncope, which consequently can provide earlier diagnosis and treatment.

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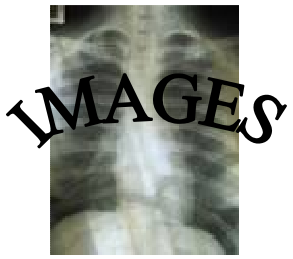
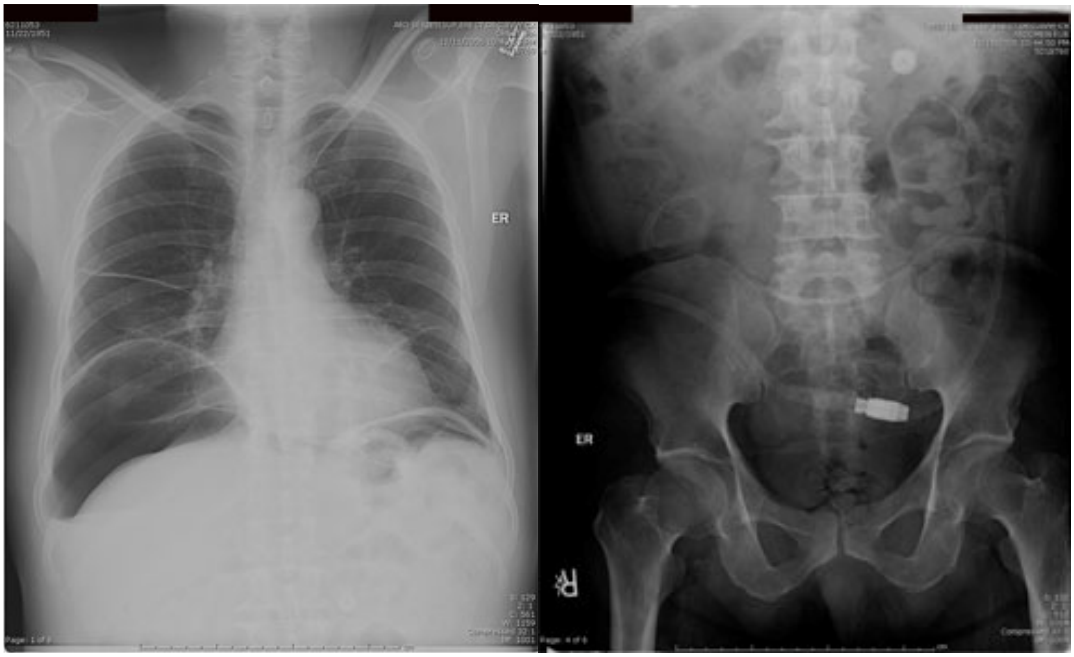
Keywords: cough, syncope, pericardial effusion, case report



Pneumoperitoneum Complicating Peritoneal Dialysis Catheter

Bassem M. Chehab, M.D.
William J. Salyers, M.D.
University of Kansas School of Medicine-Wichita
Department of Internal Medicine



A 55-year-old male presented with progressively worsening nausea over a one-year period. It began when he started peritoneal dialysis for end-stage renal disease. His nausea progressed to the point that he could not tolerate oral intake. He also had difficulty with exchanges, but the exchange fluid was clear. He denied any fever, chills, abdominal pain, change in bowel movements, hematochezia, or hematemesis.

The physical exam was unremarkable except for mild discomfort in the right lower quadrant. The peritoneal dialysis catheter was intact and appeared to be clean. The laboratory exam showed a normal white cell count and a negative peritoneal fluid culture. A KUB showed a large amount of free intra-peritoneal air. Further questioning of the patient revealed improper use of the peritoneal dialysis system as a cause of the pneumoperitoneum.

The prevalence and clinical significance of pneumoperitoneum in peritoneal dialysis (PD) patients is not defined fully in the current literature. Some reports suggested that, unlike in non-PD patients, it rarely is caused by gastrointestinal perforation.[1] Intestinal perforation can be ruled out by rapid clinical improvement with standard therapy, growth of Staphylococcus epidermidis in the peritoneal effluent culture, reduction of pneumoperitoneum after correction of the technical fault, and in contrast to the Gram-negatives and/or anaerobes usually found during bowel perforation.[2-3]

Computed tomography is more sensitive than x-ray film in detecting sub-diaphragmatic free air. No agreement exists whether the amount of sub-diaphragmatic air can differentiate bowel perforation from other causes of pneumoperitoneum. Opening the outflow tube and manual

compression of the abdominal wall might release air from the peritoneal cavity. The Trendelenburg position might facilitate air movement towards the intraperitoneal tip of the catheter and increase the effectiveness of abdominal compression.[1-2]

In conclusion, pneumoperitoneum occurs with a variable prevalence in PD patients, but rarely is related to bowel perforation. However, an aggressive examination is necessary for visceral perforation. Examination of the peritoneal effluent, the medical history, and the clinical picture help to avoid delay and unnecessary laparotomy. Once bowel perforation has been excluded, efforts must be made to find the cause of pneumoperitoneum and avoid its recurrence.

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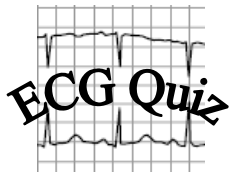
Keywords: pneumoperitoneum, cutaneous, peritoneal dialysis, case report

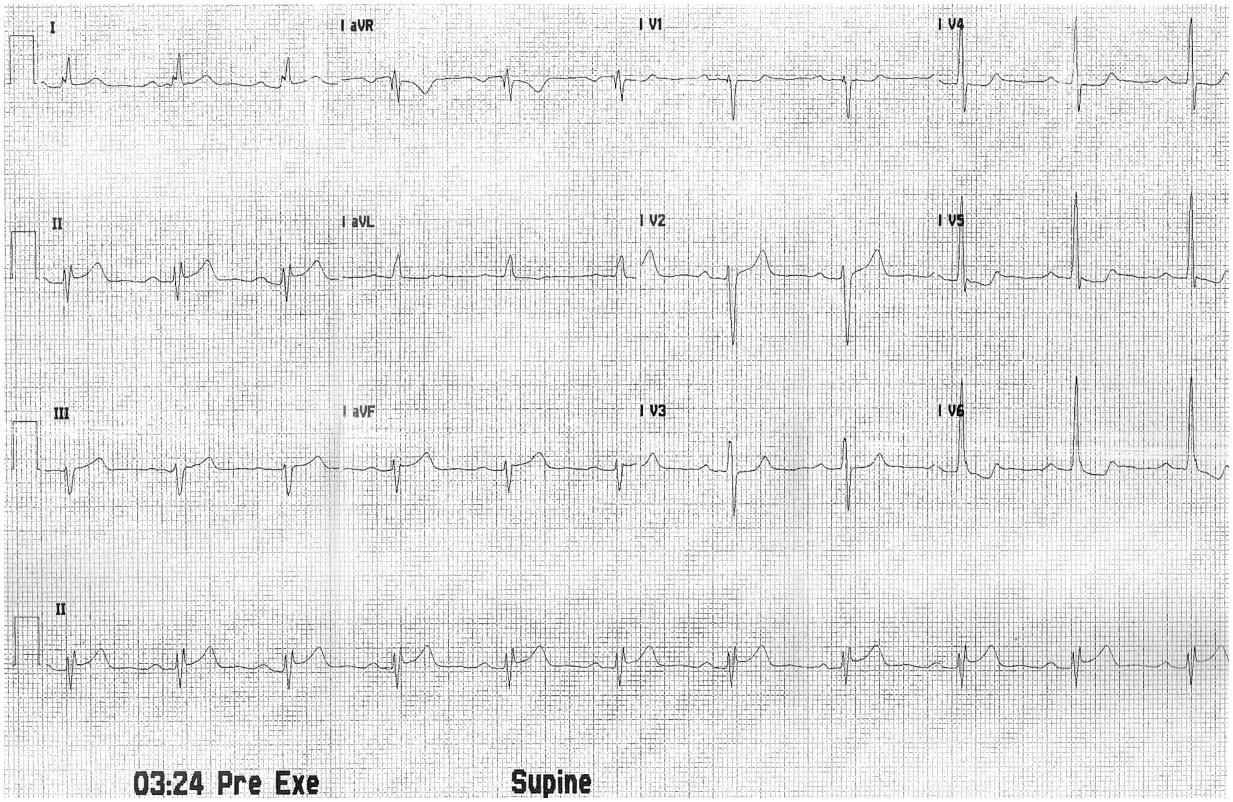


Unusual Stress ECG

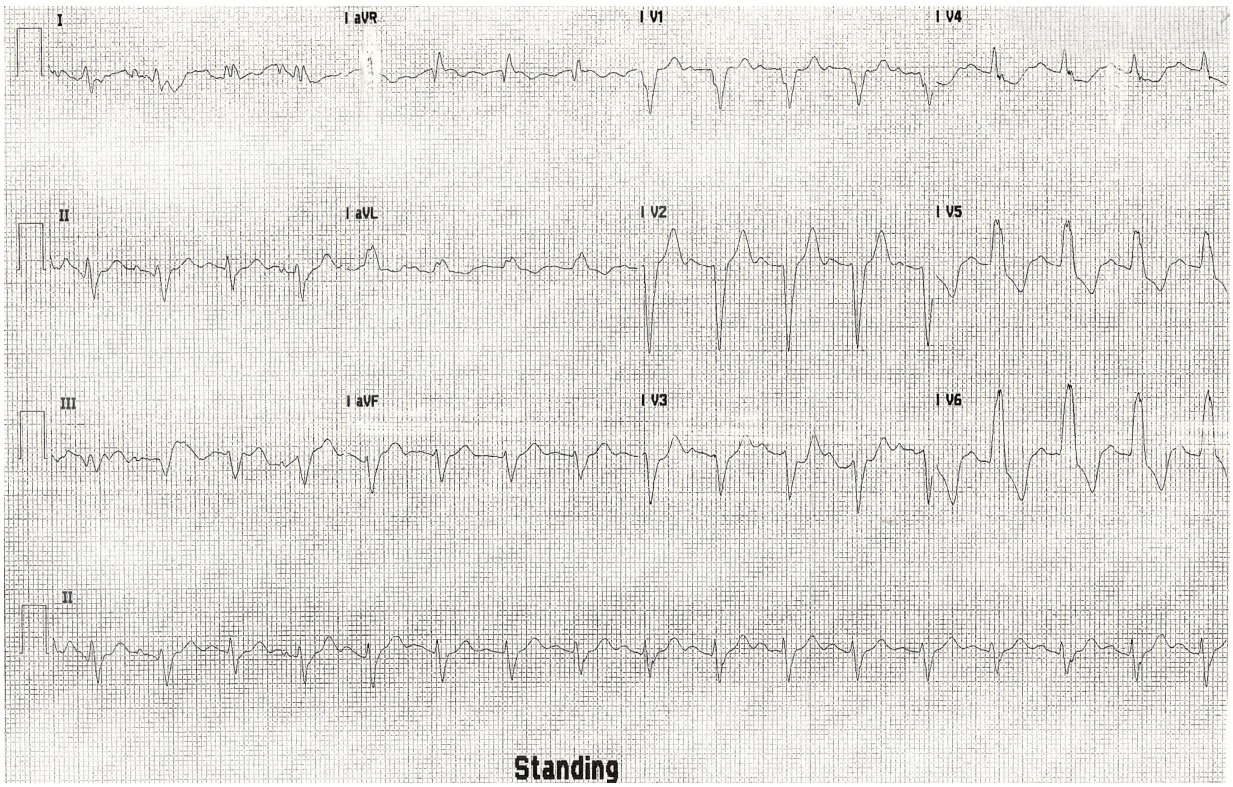
Samer Antonios, M.D.[1]
Prakash V. Raghavan, M.D.[1,2]
[1]University of Kansas
School of Medicine-Wichita
Department of Internal Medicine
[2]Raghavan Clinic, Wichita, KS

A 66-year-old white male was seen on follow-up with his cardiologist. He had a history of coronary artery disease and Percutaneous Transluminal Coronary Angioplasty with stent placement in the left anterior descending artery three years prior. His most recent echocardiogram showed a normal ventricular ejection fraction. He was hypertensive and had a history of hyperlipidemia. He also had a history of gastroesophageal reflux disease.

His ECG obtained at rest is shown below:



His ECG after 3 minutes of exercise on the treadmill is below:



What is the diagnosis?

(A) Ventricular tachycardia

(B) Multi-focal ventricular ectopy

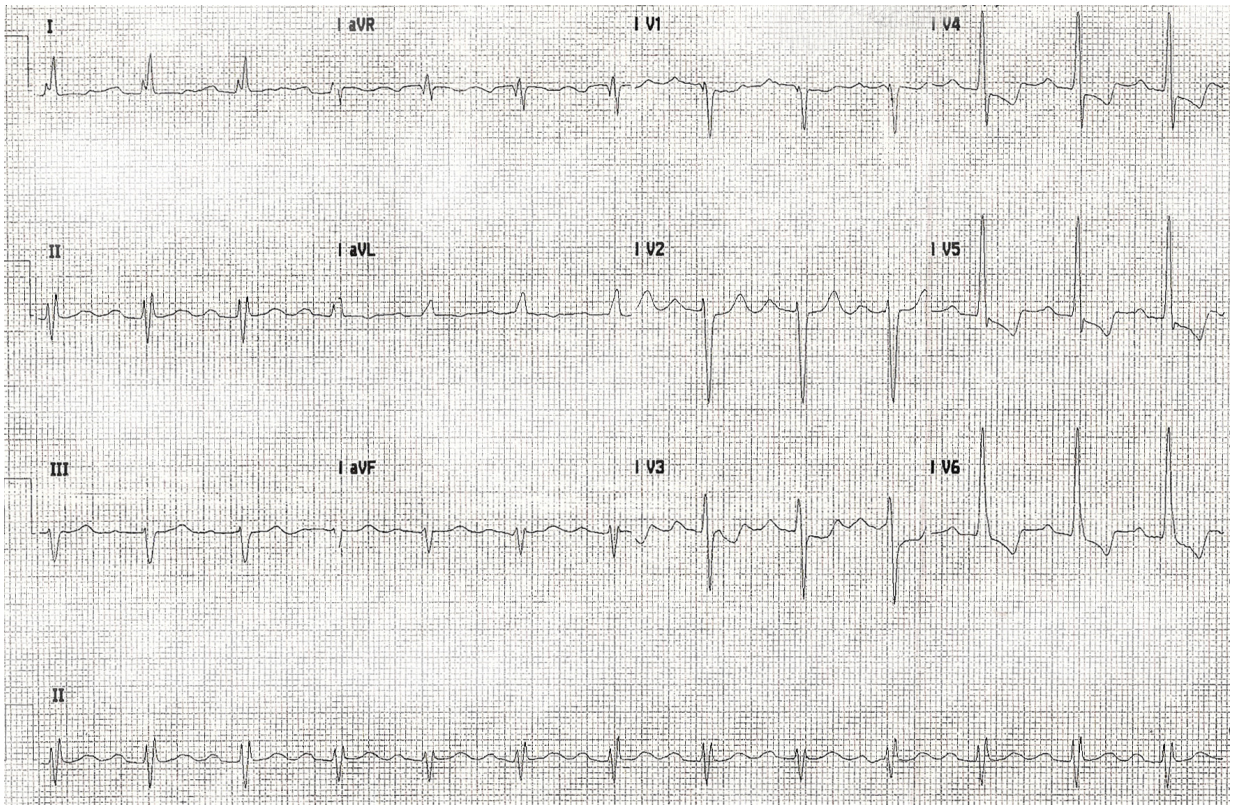
(C) Left bundle branch block

(D) Conduction through an accessory pathway.

Correct Answer: C

After the patient stopped the exercise, the left bundle branch block (LBBB) resolved and the QRS interval returned to baseline (see ECG after exercise below). Approximately 0.5% of all patients who undergo exercise testing develop a transient LBBB during exercise, but its prognostic significance is unclear.[1-3] Several published series of patients with this finding noted that exercise-induced LBBB occurs most commonly in the presence of underlying heart disease, particularly coronary artery disease (64% to 75%), but it has a variable incidence of cardiac events (36% to 50%).[3-5] Schneider and colleagues[1], based on their study with the Framingham population, noted that intermittent LBBB has the same mean age of onset and the same evolution and prognosis as those seen in subjects with fixed LBBB. A matched-control cohort study by Grady et al.[6] showed that exercise-induced LBBB independently predicted a higher risk of death and major cardiac events (adjusted relative risk, 2.78). Exercise-induced LBBB does not always denote the presence of underlying coronary artery disease. It has been described in patients with normal coronary arteries and was associated with a better prognosis.[7] It also has been described as a side effect of certain anti-arrhythmics and in the presence of other cardiac abnormalities, such as dilated cardiomyopathy, myocarditis and hypertrophic cardiomyopathy.[8-13] Although exercise-induced LBBB is rare, many new cases will be noted annually in the United States, where more than two million exercise stress tests are performed each year.[14] Therefore, exercise-induced LBBB and its prognostic significance warrants attention among physicians who request stress testing for their patients.

The ECG after exercise is below:



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Keywords: electrocardiography, bundle-branch block, coronary artery disease

Communication Disorders in Individuals with HIV/AIDS

K. James Kallail, Ph.D.[1,2], David W. Downs, Ph.D.[2], Julie W. Scherz, Ph.D.[2]
[1]University of Kansas School of Medicine-Wichita
Department of Internal Medicine
[2]Wichita State University
Department of Communication Sciences and Disorders

Introduction

The prevalence of communication disorders in individuals with Human Immunodeficiency Virus/Acquired Immunodeficiency Syndrome (HIV/AIDS) and how it compares to the general population are unknown. Treatment advances are being made both for individuals with communication disorders and those with HIV/AIDS. Unfortunately, little work has been done for those who have HIV/AIDS *and* a communication disorder. Communication disorders may have a significant impact on an individual as speech, language, and hearing often are critical to success in contemporary life. Approximately one in six Americans has some form of communication disorder.[1]

The natural history of HIV infection in a typical person without antiretroviral therapy from the time of HIV transmission to death is 10-11 years.[2] The initial event is the acute retroviral syndrome accompanied by a decline in CD4 cell counts. CD4 cell count decreases are due to HIV-induced cell death. Late stage disease is characterized by a CD4 count less than 200 cells/mm^3 and development of opportunistic infections, selected tumors, wasting, and neurologic complications. However, progress treating HIV infection has decreased mortality rates and essentially made the disease a chronic condition that can be managed long term.[3]

The prevalence rate of HIV infection among adults and adolescents was estimated at 137.0 per 100,000 at the end of 2005.[4] The prevalence of AIDS also has increased steadily since 2001. The long-term effects of HIV/AIDS and its treatment on communication abilities raise many questions that have not been answered adequately.

HIV Infection and the Central Nervous System

HIV enters the central nervous system (CNS) early in the course of infection and its effect is widespread. Up to 100% of HIV-infected adults have CNS abnormalities.[5] Prolonging life in an immunosuppressed state has resulted in increased incidence of systemic pathologies, including those of the central nervous system. If the infection progresses, various CNS pathologies appear, including opportunistic infections, primary CNS lymphoma, progressive multifocal leukoencephalopathy, peripheral or sensory neuropathy, and HIV dementia.[3] Primary infection of the central nervous system occurs when there is a direct infection by HIV. Secondary CNS complications may occur due to HIV-associated systemic disorders. Secondary infection occurs when HIV infects another system in the body through an opportunistic infection, neoplasm, or systemic disorder.

Cognitive and Speech-Language Disorders

Cognitive impairment is one manifestation of the CNS complications associated with HIV/AIDS. Cognitive changes may be seen early in the course of the infection even in patients who are otherwise asymptomatic.[3,5-6] Common cognitive changes include problems with

abstract reasoning, learning difficulties, slow information processing, and retardation of the spontaneity of speech. CNS complications in patients with HIV can reflect the consequences of medical treatment. Clinicians should distinguish to the best of their knowledge between symptoms related to the HIV disease process and the side effects of antiretroviral medications.[3]

HIV dementia is seen in approximately 3% of patients.[2] Although a decrease in incidence has been reported with antiretroviral therapy, the prevalence has increased because of patients surviving longer.[7] Slowed processing speed may underlie patient complaints related to specific cognitive deficits.[6] Psychomotor slowness is associated with severity of HIV disease and occurs in the context of other HIV-related neuropsychiatric symptoms. Speech-language symptoms are related to memory loss and cognitive slowing.[2] Language difficulties actually may be a manifestation of deficits in simple reaction time rather than language function.[8]

Primary CNS lymphomas are associated with the Epstein-Barr virus.[2] The incidence is less than 6%. Several symptoms relate to speech-language abilities including confusion, memory loss, and aphasia. Progressive multifocal leukoencephalopathy occurs in 1 to 2% of AIDS patients.[2] Cognitive impairments and speech deficits related to hemiparesis are common symptoms. Further, up to 9% of AIDS patients are referred for psychiatric evaluations.[2-3] Psychiatric illness may be associated with disorganized thinking and cognitive changes.

Auditory System Disorders

The etiologies of auditory system disorders in adults with HIV/AIDS fall into one of three broad categories: HIV/AIDS as the primary cause, opportunistic infections associated with HIV/AIDS, and iatrogenic sources. Karposi's sarcoma, for example, is the most common neoplasm in persons with HIV/AIDS.[5] It is manifested on the pinna almost exclusively in persons with HIV/AIDS and may cause conductive hearing loss when manifested in the ear canal, eardrum, or middle ear.[9] HIV infection also may damage the cochlea, eighth nerve, or both, sometimes resulting in sensorineural hearing loss; and it also may compromise neural pathways and centers in the brain resulting in central auditory disturbance.[5,10]

Opportunistic infections due to suppressed immune systems in adults with HIV/AIDS can precipitate or exacerbate auditory disorders. These opportunistic infections include otitis media or mastoiditis in the middle ear,[11] neurosyphilis,[11-13] cyto-megalovirus,[14] cryptococcal meningitis,[11,15] non-Hodgkin's lymphoma in the oral cavity,[5] pharyngeal infections associated with hemophilia-A,[16] and several other infections of the head and neck.[10,17-18] Adults with HIV/AIDS may show head, neck and otologic symptoms from opportunistic infections, including otalgia, otorrhea, tinnitus, "muffled" hearing, aural fullness, facial nerve palsy, vertigo and central vestibular, and ocular-motor disturbance.[5,10-11,17-25]

Ototoxicity from drugs used to treat adults with HIV/AIDS is the most common iatrogenic source of auditory disorders. Tseng and colleagues[26] found that 17% of HIV/AIDS patients who received azithromycin experienced drug-related hearing problems and three-fourths of these problems resolved after azithromycin was discontinued. Kohan and colleagues[20] reported on seven patients with HIV/AIDS who had persistent sensorineural hearing losses. Three patients had been exposed repeatedly to strong ototoxic drugs (i.e., aminoglycosides), while the other four had

persistent hearing losses despite standard antibiotic treatments. Simdon and colleagues[27] reported three cases of auditory dysfunction, possibly associated with nucleoside analog reverse transcriptase inhibitors. All three patients had histories of noise-induced hearing loss and tinnitus that worsened during antiretroviral therapy. Marra and colleagues[11] also reported that hearing loss was associated with antiretroviral therapy in subjects with HIV/AIDS, particularly subjects 35 years or older.

Whatever the etiology, some middle ear infections in adults with HIV/AIDS, such as chronic otitis media, supporative otitis media, and mastoiditis, are as common as those in adults without HIV/AIDS and often respond to medical treatment as well.[10,22] Conversely, pneumocystis carinii otitis media is an opportunistic infection unique to persons with HIV disease and can cause conductive or mixed hearing losses.[5,10]

Sensorineural hearing loss, which often cannot be treated medically, is more common among adults with than those without HIV/AIDS.[10,18,20,28] The likelihood of sensorineural hearing loss among adults with HIV/AIDS varies among studies depending on the characteristics of the sample and the criteria for hearing loss. In general, sensorineural hearing losses occurred in one-third to two-thirds of the subjects. They usually occurred more at high frequencies than at low frequencies and were more severe in patients with more severe HIV infections[17] or, similarly, with greater deterioration in immunologic status[28]. Furthermore, most neuro-otological disorders in HIV/AIDS patients have a central origin; both central auditory disorders and peripheral auditory disorders are more common in advanced stages of HIV/AIDS.[21,29-30] Central auditory disorders also may be more common in adults with HIV-Associated Dementia[31], though research in this area is lacking. Finally, common risk factors for hearing loss in adults in general (e.g., excessive noise exposure or aging) can impact the prevalence and severity of auditory problems in adults with HIV/AIDS.

Audiological Testing

Although HIV/AIDS can manifest problems at all levels of the auditory system, a limited variety of audiologic tests have been used to measure auditory function in the HIV/AIDS population. Clearly, the most common measure has been auditory evoked potentials, an EEG type of test in which the presence of brainwaves are recorded to predominantly non-speech sounds. Although a small number of researchers have recorded evoked responses from the upper brainstem, thalamus, and cortex,[32-33] most have used auditory brainstem response (ABR) testing which measures neural responses mainly from the eighth nerve and lower and mid brainstem.[10,25,31,34-43] The emphasis on ABR testing may arise from its ability to detect early clinical and sub-clinical pathologic changes in the eighth nerve and brainstem of adults with HIV/AIDS.[34-36,43] Larson[5] advocated that the evaluation of hearing loss in the patient with AIDS should be approached as an eighth nerve neuropathy. She suggested that audiologists complete a test battery including pure tone testing, speech audiometry, tympanometry, acoustic reflex testing, and ABR testing.

Larson's proposed auditory test battery is more extensive than most researchers have used in studies of audition in patients with HIV/AIDS.[5] Nevertheless, by focusing on measuring eighth nerve neuropathology, Larson's battery may not be comprehensive enough to track the variety of auditory problems encountered with HIV/AIDS in adults. Her battery did not include any measures of social-emotional manifestations

of hearing loss, which may be even more prominent when it accompanies HIV/AIDS. One popular valid measure of social and emotional problems that has been used with adults without HIV/AIDS is the Hearing Handicap Inventory for Adults.[44] If Larson proposed her battery today, moreover, it likely would include otoacoustic emissions testing. This is an electro-acoustic measure of the cochlea's outer hair cell function which can be compromised by ototoxic drugs.[45]

The most prominent omissions in Larson's battery are behavioral auditory tests of central auditory processing. Recent professional guidelines of the American-Speech-Language-Hearing Association[46] mandate that behavioral measures are essential for diagnosing central auditory processing disorders; conversely, they cannot be diagnosed by electrophysiological measures alone.

The most widely researched behavioral test battery of central auditory processing disorders *in adults* was developed over several decades by an audiologist, James Jerger, and his colleagues.[47-55] It employs performance-intensity functions for both monosyllabic words, performance-intensity functions for scrambled sentences presented to the same ear in noise (i.e., Synthetic Sentence Identification for ipsilateral competing messages or SSI-ICM), and scrambled sentences presented simultaneously in both ears (i.e., dichotic sentence identification or DSI). This battery can differentiate auditory disorders between the cochlea, eighth nerve, brainstem, and temporal lobe. This battery contains the essential elements in any successful central auditory test battery, efficient and effective controls over cochlear sensitivity loss, absolute speech recognition ability, and non-auditory influences.[54]

The SSI-ICM and DSI have been used to diagnose central auditory processing disorders in subjects with varying degrees of brain damage.[56] To our knowledge, however, a case study of an adult with AIDS and organic brain disease was the only study of these measures in a subject with HIV/AIDS.[30] The patient showed abnormally reduced scores on in both ears on the SSI-ICM.

As radiographic technology, such as magnetic resonance imaging, has improved over the past two decades, many audiology facilities serving adults no longer routinely use relatively expensive ABR testing for diagnosing space-occupying lesions. Conversely, nearly all audiology clinics in the United States have personnel, equipment, and materials to complete pure tone testing, speech audiometry, tympanometry, acoustic reflex testing, otoacoustic emissions testing, and the Hearing Handicap Inventory for Adults. In other words, nearly all audiologists have the capability to evaluate, in about two hours, both peripheral and central auditory function of adults with HIV/AIDS. Such a feasible, yet comprehensive, test battery may become more vital for tracking auditory problems of adults with HIV/AIDS as they live longer and grow in numbers. No researchers, however, have studied such a comprehensive test battery in HIV/AIDS patients systematically.

Summary

Speech, language, and hearing disorders are not uncommon in individuals with HIV/AIDS. Little research, however, has explored the relationship and impact between HIV/AIDS and communication abilities. Several factors related to HIV/AIDS influence communication abilities. These factors include CNS abnormalities related to the infection, opportunistic infection, and treatment effects. Clinicians should distinguish to the best of their knowledge between symptoms

related to the HIV disease process and the side effects of antiretroviral medications. Assessments of communication disorders should be obtained as necessary to provide quality care to individuals with HIV/AIDS and to maintain the quality of life that effective communication provides.

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Keywords: Human Immunodeficiency Virus, Acquired Immunodeficiency Syndrome, communication disorders, speech disorders, language disorders, hearing disorders, dementia