The Incidence of Breast Cancer among Disabled Kansans with Medicare

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Abstract

Background. Breast cancer disparities by disability status are poorly understood. While previous studies have shown increased odds of late stage at diagnosis, it is unclear whether the incidence of breast cancer varies by disability status.

Methods. To assess cancer incidence and stage at diagnosis among disabled and nondisabled Medicare beneficiaries in Kansas, a retrospective cohort study was conducted using linked Medicare enrollment and Kansas Cancer Registry data from 2007 to 2009. Disability status was determined by the indicator for the original reason for Medicare eligibility.

Results. Among the 651,337 Medicare beneficiaries included in the cohort, there were 2,384 cases of breast cancer. The age-adjusted incidence was 313 per 100,000 among female beneficiaries with disabilities and 369 per 100,000 among nondisabled female beneficiaries. The adjusted incidence rate ratio was 0.93 (95% CI 0.73-1.18). When assessing stage at diagnosis, there was no difference in the odds of late stage at diagnosis by disability status (OR = 1.02; 95% CI 0.68-1.50).

Conclusion. No significant difference in incidence or stage at diagnosis was identified among this cohort. The use of Medicare eligibility to define disability status presented a number of limitations. Future studies should seek alternate definitions of disability to assess disparities in breast cancer incidence, including definitions using Medicare claims data.

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Introduction

While racial and ethnic health disparities in breast cancer have been the subject of much research and public health action, breast cancer disparities by disability status are poorly understood. Healthy People 2020 recognizes the significant health disparities people with disabilities face and includes several objectives to ensure people with disabilities are on track toward better health and quality of life.[1] People with disabilities are more likely to experience difficulties or delays in getting the health care they need, not have had a mammogram in the past two years, not engage in fitness activities, use tobacco, be overweight or obese, have high blood pressure, experience symptoms of psychological distress, receive less social-emotional support, and have lower employment rates.[1] It remains unclear how these factors affect the incidence of breast cancer among women with disabilities.

Literature is conflicting related to breast cancer in women with disabilities. Two studies have assessed breast cancer incidence among women with intellectual disabilities and found no significant difference in standardized incidence when compared to the general population.[2,3] There are other studies assessing disabilities and breast cancer, but the inclusion criteria and methodology varied.[4,5] Several studies on breast cancer screening have identified significant disparities among women with disabilities.[6-11]

While literature suggested disparities in cancer screening, treatment, and survival, little is known about the incidence of breast cancer associated with disability. This study assessed health disparities in people with disabilities by determining cancer incidence and stage of breast cancer diagnosis among disabled and nondisabled Medicare beneficiaries in Kansas.

Methods

The study population included female Kansans ages 65 and older who were diagnosed with breast cancer and enrolled in Medicare during 2007 to 2009. Breast cancer cases were obtained from the Kansas Cancer Registry (KCR) database using the Surveillance, Epidemiology, and End Results (SEER) Site Recode ICD-O-3/WHO 2008 value for the breast (26000).[12] Disability status was obtained from the Medicare enrollment file. Disability was defined as having an indication of disability as the original reason for Medicare eligibility. Individuals who have a medical condition that precludes their ability to work are eligible to receive Social Security disability benefits.[13] Since 1972, any individual under age 65 who has been entitled to Social Security disability benefits for 24 months is eligible for Medicare.[14]

Records from KCR were linked to those from the Centers for Medicare and Medicaid Services (CMS) by matching Social Security numbers, with further confirmation based on patient sex, race, date of birth, and zip code of residence. Cancer cases from KCR were excluded if they did not match with the Medicare enrollment file, if the cancer was identified by death certificate only, or if they were diagnosed in a year during which they did not appear in the Medicare enrollment file. Each year of the study period was treated as a separate cohort and each enrollee was counted each year they appeared in the Medicare enrollment database. This project was approved by the Institutional Review Board at the University of Kansas Medical Center.

Variables that were analyzed included date and stage at diagnosis from the KCR database, as well as age at the end of enrollment year, race, and zip code from the Medicare enrollment file. Zip and county codes were used to classify rural or urban residency and county-level poverty. The stage at diagnosis, which is classified by the SEER Summary Stage 2000 system, was categorized as localized (the tumor is confined to breast tissue), regional (the tumor has spread to nearby lymph nodes and/or adjacent tissues), and distant (the tumor has metastasized to another part of the body).[15] A Zip Code Rural-Urban Commuting Area (RUCA) approximation was applied to assign individual's residency into rural versus urban.[16] Individual-level income indicators were not available from KCR and Medicare files. The proportion of county residents below poverty using 2007 estimates from the Area Health Resources Files (AHRF) was used as a surrogate. Low or high poverty in a county was recoded using the median percent below poverty of all Kansas counties.[17]

Group comparisons were performed using Chi square or Fisher’s exact tests, when appropriate. Age-adjusted incidence was calculated using direct standardization with the 2000 US standard population. Poisson regression models were constructed to generate an adjusted incidence rate ratio (IRR) comparing the incidence of breast cancer among disabled and nondisabled beneficiaries. Logistic regression models were used to assess differences in cancer stage at diagnosis by disability status. Likelihood ratio tests were applied to assess effect modification. Deviance analysis in Poisson regression and the Hosmer-Lemeshow test in logistic regression were used to evaluate model adequacy.

Results

The study cohort included 651,337 female Medicare beneficiaries, 65 years and older, in 2007-2009. Selected characteristics are presented in Table 1. When compared to nondisabled beneficiaries, beneficiaries with disabilities tended to be younger, non-white, or residing in counties with high poverty. During the study period, 2,575 cases of breast cancer were diagnosed among Kansas women 65 and older, 2,384 of which met eligibility criteria. Crude incidence was 339 per 100,000 beneficiaries with disabilities and 370 per 100,000 nondisabled beneficiaries. When adjusting for age, breast cancer incidence was 313 per 100,000 among disabled beneficiaries and 369 per 100,000 among nondisabled beneficiaries.

Table 1. Selected characteristics of female Medicare beneficiaries, 2007-2009.

	Disabled		*Nondisabled*		
	n	*%*	*n*	*%*	*p*
Total	37,306		614,031		
Age					< 0.01
65-69	16,397	43.9%	143,386	23.3%	
70-74	9,905	26.5%	122,331	19.9%	
75-79	5,316	14.3%	115,768	18.9%	
80+	5,688	15.3%	232,546	37.9%	
Residency*					0.07
Rural	7,058	48.8%	110,601	49.4%	
Urban	7,401	51.2%	113,116	50.6%	
Race					< 0.01
White	32,284	86.7%	579,589	94.5%	
Black	3,859	10.4%	19,967	3.3%	
Other	1,096	2.9%	13,478	2.2%	
County Poverty†					< 0.01
High	24,708	67.5%	358,897	59.2%	
Low	11,918	32.5%	247,283	40.8%	

* Rural/Urban residency was based on Rural-Urban Commuting Area codes.

† County poverty was defined as the percent living in poverty being above or below the Kansas median (11.6%).

Results from Poisson regression analyses are summarized in Table 2. The incidence rate ratio of breast cancer was 0.92 (95% CI: 0.76-1.10) when comparing disabled to nondisabled beneficiaries. The incidence rate ratio associated with disability did not change significantly after controlling for potential confounders that had a p value of less than or equal to 0.20 in the univariate analysis (i.e., age, race, residency, and county level poverty; Table 1). Thus, the effect of disability on incidence did not appear to be confounded by these covariates. Interaction terms were evaluated using the likelihood ratio test and found to be not significant. The final model, adjusted for

Table 2. Breast cancer incidence rate ratios by disability status: Results from Poisson regression analysis.

Model	*Risk Factor*	*IRR*	*95% CI*	*p*
Unadjusted	**Disabled**			
	Yes	0.92	0.76-1.10	0.34
	No	1.00	-	
Adjusted	**Disabled**			
	Yes	0.93	0.73-1.18	0.54
	No	1.00	-	
	Age			
	65-69	1.00	-	
	70-74	1.21	1.03-1.41	0.02
	75-79	1.23	1.04-1.44	0.01
	80+	1.08	0.94-1.25	0.28

age, is presented in Table 2. A re-scaling was applied to correct for over-dispersion to ensure model adequacy for interpretation.

Table 3 shows the characteristics associated with late stage breast cancer at the time of diagnosis. Patients with disabilities had a higher chance of having their breast cancer diagnosed at a later stage relative to their nondisabled counterparts. However, the difference was not significant statistically ($p = 0.71$). Race was the only factor that varied significantly by stage at diagnosis. Black women were more likely to be diagnosed with late stage breast cancer than other racial groups.

Odds ratios and the corresponding 95% confidence intervals from the logistic regression are shown in Table 4. The unadjusted odds ratio associated with disability for late stage at diagnosis was 1.08 (95% CI: 0.72-1.59). When controlling for potential confounding by age, residency, race, and poverty, the odds ratio for disability remained similar (OR = 1.02, 95% CI 0.68-1.50), so the effect of disability on stage at diagnosis did not appear to be confounded by these covariates. Interaction terms were not statistically significant.

Discussion

This study utilized Kansas Cancer Registry and Medicare linked data to address the relationship between disability and incidence of breast cancer. This study did not identify a significant difference in breast cancer incidence by disability status after adjusting for age. Disability was not associated with increased odds of late stage at diagnosis. To our knowledge, this study was the first to assess cancer incidence by disability status among Medicare beneficiaries. The lack of association between disability status and stage at diagnosis is not consistent with what is reported in the literature. Several implications are worthy of a further discussion.

First, our findings suggested limitations in using Medicare eligibility to define disability and necessitate alternate methodologies for future studies of breast cancer incidence among the disabled. The primary limitation was the broad definition of disability that Medicare eligibility provides. There are published data showing a strong relationship between disability and poor health.[18] People with disabilities may be at increased risk of developing chronic

Table 3. Breast cancer stage at diagnosis by selected characteristics.

		Early Stage (n=1,586)		*Late Stage* (n=711)		
	Total	*n*	*%*	*n*	*%*	*p*
Disabled						0.71
Yes	120	81	67.5%	39	32.5%	
No	2,177	1,505	69.1%	672	30.9%	
Age						0.18
65-69	507	343	67.7%	164	32.3%	
70-74	506	349	69.0%	157	31.0%	
75-79	479	350	73.1%	129	26.9%	
80+	805	544	67.6%	261	32.4%	
Residency*						0.20
Rural	1,091	738	67.6%	353	32.4%	
Urban	1,191	835	70.1%	356	29.9%	
Race						0.01
White	2,169	1,511	69.7%	658	30.3%	
Black	73	39	53.4%	34	46.6%	
Other	52	33	63.5%	19	36.5%	
Poverty†						0.91
High	1,317	909	69.0%	408	31.0%	
Low	965	664	68.8%	301	31.2%	

* Rural/Urban residency based on Rural-Urban Commuting Area codes

† Defined as the county's percent living in poverty being above or below the Kansas median (11.6%)

Table 4. Odds ratios for late stage diagnosis: Results from logistic regression analysis.

Model	*Risk Factor*	*OR*	*95% CI*	*p*
Unadjusted				
	Disabled			
	Yes	1.08	0.72-1.59	0.71
	No	1.00	-	-
Adjusted				
	Disabled			
	Yes	1.02	0.68-1.50	0.94
	No	1.00	-	-
	Race			
	White	1.00	-	-
	Black	2.00	1.24-3.12	< 0.01
	Other	1.32	0.73-2.32	0.34

conditions, while people with certain chronic conditions also may become disabled. Disabilities can include many subtypes (e.g., intellectual/developmental, physical, and mental disabilities as well those resulting from other medical conditions). By only examining the initial reason for Medicare eligibility, the disability category or severity in this broad group of disabled individuals was unable to be assessed. Such heterogeneity of the disabled group may be contributing to our null findings. Future research may include data from other sources such as Medicaid.

Second, study criteria may be different in different studies. Roetzheim and Chiriskos reported that breast cancers in disabled women were diagnosed at a later American Joint Committee on Cancer (AJCC) stage.[5] Differences in findings between our study and the study from Roetzheim and Chiriskos may be attributed to the definitions of study factors. Our study included disabled women with breast cancer, ages 65 and older, while the latter study included disabled women with breast cancer ages 67 to 71 at the time of diagnosis. Our study used the SEER summary staging system and the prior study used the AJCC stages. Stages from both systems are similar to some extent, but they are not identical. Use of SEER summary staging allows studies of cancer burden in a statewide population (like the Kansas Cancer Registry) by including most breast cancer patients in the analysis while using AJCC staging might have excluded patients who have been treated in facilities where AJCC staging was not applied to patient's treatment plan.

Third, certain chronic conditions may lead to disability. In fact, a higher prevalence of chronic diseases (e.g., obesity, behavioral, and psychological disorders) are associated with patients with disabilities.[18] However, the interplay between a higher prevalence of chronic medical conditions, disability, and care seeking behaviors is not clear.[18-19] It is possible that breast cancer incidence in this population was affected by some of these unmeasured confounding variables.

Lastly, the study results may be affected by misclassification bias. Individuals born prior to 1908 were not eligible for Medicare before the age of 65 due to disability, given the timeline of the 1972 provision. These individuals, and those who may have been eligible but did not seek disability benefits, would be misclassified as nondisabled and would bias our findings towards the null. In addition, the individuals that are eligible for Social Security Disability Insurance (SSDI) were affected by work-limiting disabilities which likely represented a subset of all disabilities in older adults. Disabilities that did not meet the Social Security eligibility criteria and those that met eligibility criteria for Medicaid may not be represented in this study.

While no statistically significant results were identified as a result of this analysis, further epidemiological investigation with a modified definition of disability is warranted. Our null findings may be due to limitations of using Medicare eligibility to define disability status among this cohort. Future studies should use more precise definitions of disability to identify disparities in breast cancer incidence, including defining disability with the use of Medicare claims data. Such analyses may be more apt to identify disparities within the state of Kansas and better serve women with disabilities.

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Speech-Language and Cognitive Findings in Patients with HIV/AIDS

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Abstract

Background. Few adult patients with HIV/AIDS are screened regularly for speech-language and cognitive disorders even though they may manifest communication difficulties. No comprehensive studies assessing the broad range of speech-language and cognitive disorders of adults with HIV/AIDS appear in the literature. As such, clinicians may be unfamiliar with the types of communication disorders that may be manifested. This study assessed the prevalence of speech-language and cognitive disorders in adults with HIV/AIDS using a broad inventory of speech, language, and cognitive skills.

Methods. A cross-sectional design was used to investigate communication disorders in a convenience sample of patients living with HIV/AIDS. Adult patients from a general internal medicine clinic in Wichita, Kansas were recruited as they presented for medical appointments. Each participant received a speech-language and cognitive test battery consisting of 10 assessments.

Results. The primary outcomes were: (1) presence of any speech-language or cognitive disorder, and (2) degree of communication disorder, as measured by the number of positive results. Eighty-two adults with HIV/AIDS were evaluated for communication disorders. Prevalence was 95%; 78 out of 82 participants manifested abnormal findings on at least one assessment in the test battery. Test results revealed a variety of cognitive and language issues, mostly related to integrating information on the picture description task (45%), timed word generation (44%), and memory-related story retelling (35%). Two participants revealed abnormal results on all ten assessments.

Conclusions. Speech-language and cognition deficits are common in adult patients with HIV/AIDS. Every patient with HIV/AIDS should be assessed to determine the impact of these communication deficits on their daily living skills.

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Introduction

Approximately one in six Americans will experience some form of a communication disorder during his or her lifetime.[1] Although medical and non-medical treatments have advanced significantly for many individuals with communication disorders, progress has been minimal for those who have Human Immunodeficiency Virus/Acquired Immuno-deficiency Syndrome (HIV/AIDS) *and* a communication disorder. Several explanations may account for this lack of progress.

First, from our experience, few adult patients with HIV/AIDS are screened regularly for speech-language and cognitive disorders even though they may manifest communication difficulties. For example, in our general medicine clinic, referrals for

speech-language and cognitive assessments are made infrequently and only if a specific patient requests it or if a communication or cognitive deficit is an obvious problem to the attending physician. Patients with HIV/AIDS are not common in the caseloads of speech-language pathologists.

Second, no comprehensive studies of the prevalence of speech-language and cognitive disorders of adults with HIV/AIDS appear in the literature. As such, clinicians are unfamiliar with the types of communication disorders that may manifest. Researchers have focused on the prevalence of limited and specific speech-language and cognitive disorders in adults with HIV/AIDS. HIV dementia, for example, is seen in approximately 3% of patients.[2] Cognitive changes may be seen early in the course of the infection even in patients who are otherwise asymptomatic.[3-5] Common cognitive changes include problems with abstract reasoning, learning difficulties, slow information processing, and retardation of the spontaneity of speech. A large US study found 52% of HIV patients had neuropsychological impairment.[6] A Swiss study found only 16.6% of HIV subjects had completely normal neurocognitive testing.[7]

Few studies have examined speech errors (i.e., voice, fluency, or intelligibility deficits) with HIV patients. One small Indian study reported a variable pattern of voice, swallowing, and oral motor function in HIV patients.[8] Nevertheless, what remains unknown is the prevalence of a wide range of speech-language and cognitive disorders in a cohort of individuals with HIV/AIDS. Accordingly, the purpose of this study was to measure a broad inventory of speech, language, and cognitive disorders in a sample of adults with HIV/AIDS.

Methods

A cross-sectional design was used to investigate communication disorders in a convenience sample of patients living with HIV/AIDS. The study was approved by the KU School of Medicine's Institutional Review Board.

Participants. Eighty-two adult, English-speaking patients from a general medicine clinic in Wichita, Kansas, with a large population of patients with HIV/AIDS, were recruited for the study as they presented for medical appointments. These patients were diagnosed with HIV-1, but not subtyped. Each received a small stipend for their participation.

Procedures. Each participant received an evaluation of speech-language and cognitive skills. All speech-language and cognitive assessments were conducted by one of two licensed speech-language pathologists. Table 1 lists the speech, language, and cognitive assessments and the domains assessed. The battery consisted of 10 assessments and took approximately one hour. A brief description of the test battery follows:

- The speech-language-hearing interview (patient history) obtained information related to speech-language deficits from the patient's perspective and history. The format of the interview was similar to that described by Duffy.[9] Disclosure of a speech-language, hearing, or cognitive problem was recorded. Any report of a past or current speech-language, cognitive, or hearing deficit or treatment was considered a positive (i.e., abnormal) result.
- The Modified HIV Dementia Scale[10,11] consists of four subtests. The memory-registration subtest asks the subject to recall four common words in four seconds after the examiner says them. The psychomotor speed subtest asks the patient to write the alphabet in upper case letters and the time to perform the task is recorded. The memory-recall subtest asks the subject to recall the four common words presented during the memory-

Table 1. Summary of speech-language assessments for each measure in the test battery and the domain examined.

Speech-Language Protocol	**Domain Examined**			
	Speech	Language	Cognition	Structure/Function
Speech-Language-Hearing History	X	X	X	X
Modified HIV Scale			X	
Cookie Theft Description	X	X	X	
Lost Wallet Retelling			X	
Oral Mechanism Exam				X
Assessment of Intelligibility of Dysarthric Speech	X			
Grandfather Passage	X			
Boston Naming Test		X	X	
Word Generation Task		X	X	
Dysphagia Questionnaire				X

registration task. The construction subtest asks the subject to copy a line drawing of a cube and time to perform the task is recorded. A score was assigned to each subtest and a total score was used to determine severity of dementia. Any score of less than 7.5 was a positive result.

- The Cookie Theft picture description task[12] permits the subject to distinguish between relevant and irrelevant detail, to integrate information across the picture, and to draw inferences about the events depicted.[13] Picture descriptions were recorded and scored for the total number of concepts identified, as well as number of literal and interpretative concepts. The more concepts identified during the picture description, the higher the score. A score of less than 18 total concepts was a positive result.
- The Lost Wallet story[14] was read aloud to the subject, who then was asked to retell the story immediately. The number information units recalled out of 17 in the story was scored. A score less than 14 was a positive result.
- The Oral Speech Mechanism Screening Examination Revised[15] provided a standardized template for review of oral structure and function. Structural deviations were recorded. Any structural deviation of the oral mechanism important for speech production was considered a positive result.
- The Assessment of Intelligibility of Dysarthric Speech[16] quantified single-word and sentence intelligibility. The subject read ten multi-syllabic words and five five-word sentences. The percent of intelligible words was scored. Any score below 90% was considered a positive result.
- The Grandfather Passage includes all the phonemes of English.[9] The passage was read aloud by the subject and timed. A miscue analysis assessed mispronunc-iations and repeated or omitted words.[17] Reading time and the number of miscues were scored. Any score of greater than three miscues was considered a positive result.

- The Boston Naming Test[12], short version, identifies word retrieval deficits. Twenty-five line drawings were used to elicit words of varied familiarity. The number of pictures identified correctly was scored. Any score below the aged-normed mean score for normal adults was considered a positive result.
- For the word generation task, subjects named as many animals as they could in 60 seconds. The number of different animals named was scored.[18] Any score below the age-normed 50th percentile was a positive result.
- The dysphagia questionnaire determined swallowing abilities by subject report.[9] Responses were scored as either positive or negative for swallowing difficulties. The report of any difficulty chewing or swallowing was a positive result.

The primary outcomes were: (1) presence of a speech-language or cognitive disorder as signified by a positive result from any of the exams performed within the complete test battery, and (2) degree of disorder, positive results on a greater number of exams indicated a higher degree of the communication disorder.

Results

The average age of the 82 participants was 46 years (SD = 10; range 20-67). Sixty-seven percent (n = 55) were white; 74% (n = 61) were male. Forty-five percent (n = 37) completed college. The average year of HIV/AIDS diagnosis was 1999. At time of diagnosis, 18 subjects (22%) had AIDS, defined as a CD4 count less than 200/mm^3 and an AIDS indicator condition[2]; 18 (22%) were symptomatic with positive HIV serology (symptoms attributed to HIV infection), and 46 (56%) were asymptomatic or had an acute HIV infection. At the time of the study, however, 66 subjects (80%) were asymptomatic and 16 (20%) were symptomatic. None had AIDS. Nine (11%) took no HIV-related medications. Thirty-five participants (43%) took three or more HIV-related medications (combination drugs were counted as one medication).

The prevalence of communications disorders was 95%. The test battery showed 78 of 82 participants had at least one positive finding (see Figure 1). Thus, in a relatively healthy sample of subjects diagnosed with HIV, only four were evaluated within normal range on all the assessments. Sixty-two participants (75.6%) had three or more positive assessments; 33 (40%) had four or more positive assessments. Two subjects revealed abnormal results on all 10 assessments.

Table 2 reveals the percentage of participants who evaluated positive (i.e., indicating the presence of a communication disorder or deficit) on each of the speech-

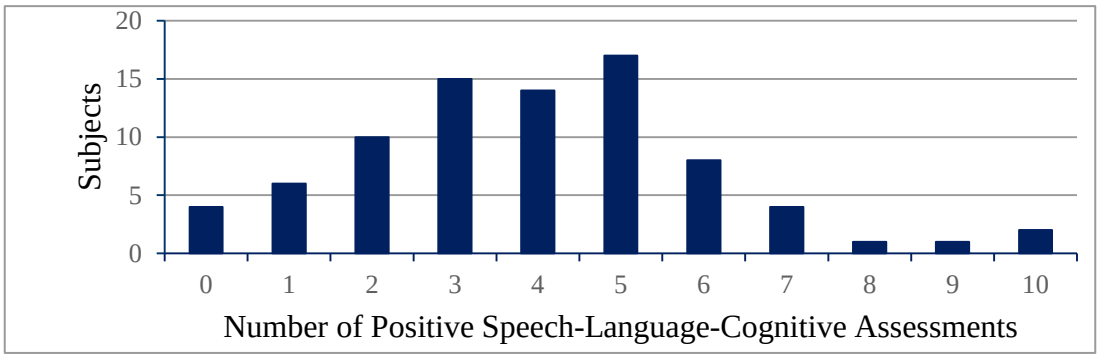

Figure 1. The number of positive speech-language-cognitive assessments by the number of subjects.

Table 2. Percentages demonstrating the presence of speech-language-cognitive deficit for each assessment in 82 subjects.

Assessment	Percentage with Deficit (n)
Speech-Language-Hearing History	78% (64)
Oral Mechanism Exam	70% (57)
Picture Description	45% (37)
Word Generation	44% (36)
Story Retelling	35% (29)
Dysphagia Questionnaire	35% (29)
Speech Intelligibility; Reading Miscues	28% (23)
Word Retrieval	28% (23)
Modified HIV Dementia Scale	24% (20)
Speech Intelligibility; Words/Sentences	9% (7)

language-cognitive assessments. The speech-language-hearing history revealed 78% of subjects had a history of a communication or cognitive disorder. Some were related to the HIV infection and its consequences (e.g., HIV dementia); some were not (e.g., work-related hearing loss). Seventy percent of subjects (n = 57) had oral mechanism deviations, most notably dental abnormalities (n = 53). Although a few subjects had notable speech distortions, none of the oral mechanism deviations caused speech to be unintelligible. A variety of cognitive and language issues were detected mostly related to integrating information on the picture description task (45%), timed word generation (44%), and memory-related story retelling (35%). The Modified HIV Dementia Scale showed the possibility of dementia for 20 (24%) participants.

Discussion

Positive speech-language-cognitive assessment findings are common in patients with HIV/AIDS. Our subjects were a broad cross-section of patients with HIV/AIDS. They were relatively healthy at the time of assessment. Yet, the proportion of subjects with all test findings in the normal range was remarkably low (5%). Conversely, the proportion with at least one identified area of concern related to communication was high (95%). This result was concerning as only 17% of the general population experience communication disorders over a lifetime.[1] The observational study design, along with co-morbidities, education levels, and socioeconomic factors, however, made it difficult to determine if HIV was the direct cause of the communication disorders. Regardless, clinicians should be aware of the high prevalence of communication disorders in patients with HIV/AIDS.

All of the test protocols administered in this battery showed the ability to identify certain aspects of speech, language, and cognitive function that may be affected in patients with HIV/AIDS. The picture description task and the verbal fluency tasks were the most useful at identifying deficits, mostly subtle language and cognitive deficits that would go unnoticed in a medical office. These deficits, however, have the potential to impact daily living and employment. An oral mechanism exam-ination is most likely to be impacted by

problems with the teeth. This issue may be related to lack of ability to afford good dental care. Poverty can limit access to health care.[19] In addition, those who cannot afford the basics in life may end up in circumstances that increase their HIV risk.

Communication disorders compromise physical health and affect the emotional, social, recreational, educational, and vocational aspects of life.[1] They affect families and social networks, including those at work and school. No direct connection between our study results and HIV/AIDS status was made; however, physicians should consider this possibility in their examination of patients. Based on our sample, issues related to speech-language and cognition should be expected, therefore, evaluated in every patient with HIV/AIDS.

Acknowledgements

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Keywords: HIV, communication disorders, cognitive disorders, hearing disorders, prevalence

Celiac Artery Dissection: A Rare Cause of Epigastric Pain

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Introduction

Isolated spontaneous celiac artery dissection is an uncommon diagnosis and is rarely considered in patients presenting with acute epigastric pain. To our knowledge, there is only one case of spontaneous celiac artery dissection associated with heavy weight lifting in an otherwise healthy individual previously reported in literature.[1,2]

Spontaneous arterial dissection is five times more common in men than in women and the average patient age is approximately 55 years.[3] Other associations previously reported are hypertension, arteriosclerosis, degeneration of arterial wall, trauma, pregnancy and arteriopathy.[2,4-7] The typical symptom of dissection is acute abdominal pain. Complications of the condition include aneurysm formation, rupture, abdominal organ ischemia or infarction, especially in the liver or spleen, and pancreatitis with elevated lipase levels.[8, 9] We report a case of celiac artery dissection presumably associated with heavy weight lifting in a construction worker.

Case Report

This case of celiac artery dissection occurred in a 44-year-old male who was a construction laborer. A week prior to presentation, he started lifting heavy weights as per his work requirement. He presented with sudden onset, sharp, epigastric pain of three-days duration. He denied any history of abdominal trauma. The pain radiated to his right upper quadrant and back. Pain was initially postprandial, however, later became constant.

The patient's past medical history was significant for smoking. He did not have any history of hypertension, diabetes, or dyslipidemia. Complete physical examination, including vital signs, were normal except for the presence of epigastric tenderness. Results of all laboratory tests, including complete blood count, basic metabolic panel, liver enzymes, lipase and lactic acid levels, were within normal range.

A sagittal contrast enhanced computed tomography (CT) scan of the abdomen showed vague eccentric filling defect in the celiac trunk with possible thrombus formation (Figure 1). A sagittal contrast enhanced CT angiogram showed a filling defect in the inferior portion of the celiac axis, worrisome for thrombosis with a dissection flap (Figure 2). The dissection flap was limited to celiac artery only. Hepatic, splenic and gastric arteries were clearly patent.

A vascular surgery consultation recommended a conservative approach with close observation. As the initial CT abdomen showed possible thrombus formation, a hypercoagulation and vasculitis work up was obtained to search for the underlying cause. All labs, including antithrombin III, protein C and S levels, Factor V Leiden, Dilute Russell Viper Venom test, antiphospholipid antibodies,

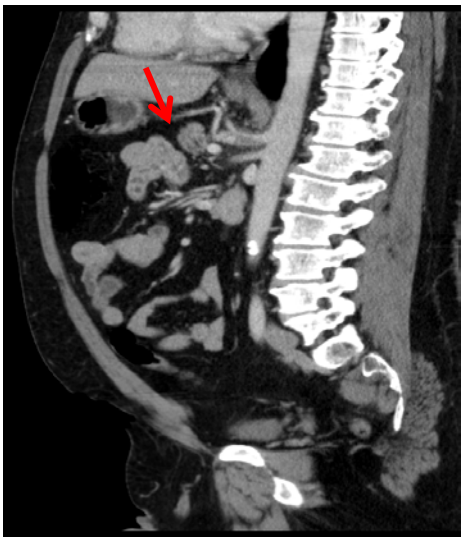

Figure 1. Vague eccentric filling defect in the celiac trunk of the abdomen with possible thrombus (red arrow).

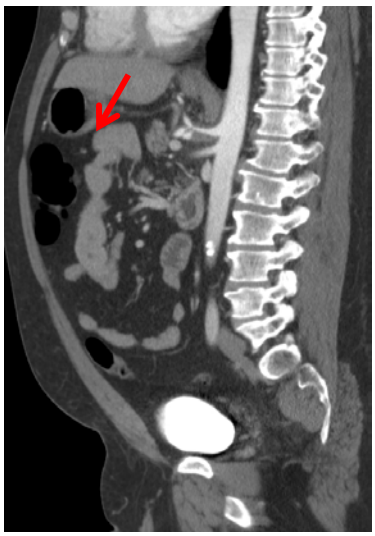

Figure 2. A filling defect in the inferior portion of the celiac axis, worrisome for thrombosis with a dissection flap (red arrow).

prothrombin gene mutation, and beta 2 microglobulin were within normal limits. Flow cytometry ruled out paroxysmal nocturnal hemoglobinuria. Janus kinase 2 mutation, antinuclear antibody, and perinuclear anti-neutrophil cytoplasmic antibodies were negative.

The patient was treated with anticoagulation therapy including heparin infusion followed by Coumadin. He was asymptomatic prior to discharge. Repeat CT angiogram of the abdomen was done at three-month intervals and showed a new finding of pseudo aneurysm formation (Figure 3). After consultation with hematology and vascular surgery, his Coumadin was discontinued and he was started on aspirin. The patient remained asymptomatic at three and six month follow-up visits.

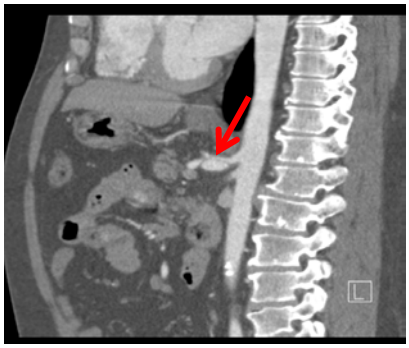

Figure 3. A new finding of pseudo aneurysm formation of the abdomen after three month intervals (red arrow).

Discussion

Cases have been reported documenting an association between arterial dissection and extreme physical exertion including heavy weight lifting, doing pushups, intense swimming, playing tennis, and racquetball.[10,11,12] In most cases, the dissection involved the ascending aorta. Some cases have been reported identifying association of aortic dissection resulting from heavy weight lifting in individuals with underlying vascular pathology such as cystic medial degeneration.[12] However, our patient was a healthy male and had no conventional risk factors for dissection besides smoking. He had never experienced any symptoms prior to this episode which seemed to be associated with his weight lifting over the period of past one week.

The proposed mechanism of dissection with weight lifting is an extreme rise in

blood pressure associated with heavy weight lifting. MacDougall et al.[13] studied arterial blood pressure of healthy males undergoing 90 minutes of heavy exercises. With each weight lift, an extreme rise in systolic and diastolic blood pressure was noticed which rose incrementally with repetitive lifts until the activity ended. One arm curl, for example, resulted in a mean rise among the participants to 225/190mm Hg. A five second Valsalva's maneuver resulted in a mean rise to 190/170 mm Hg in the group. In the first reported case of celiac artery dissection in a 45-year-old male who experienced abdominal pain while bench pressing heavy weights, a CT angiogram showed a dissection of the celiac artery extending into hepatic and splenic arteries.[1]

Conclusion

Although it cannot be stated unequivocally that weight lifting produced celiac artery dissection in our patient, weight lifting clearly leads to a great increase in blood pressure transiently, which places critical hemodynamic stress on the vessel wall. Physicians should be aware of profound cardiovascular effects of weight lifting including dangerous elevation in arterial wall tension and consider arterial dissection in the differential diagnosis of otherwise unexplained epigastric pain in any individual associated with extreme exertion and heavy weight lifting.

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Keywords: celiac artery, dissection, epigastric, pain

Pancreatic Mass is not always Adenocarcinoma

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Introduction

Ductal adenocarcinoma accounts for 85% to 90% of pancreatic tumors.[1] Non-Hodgkin's lymphoma (NHL) frequently arises in extra-nodal sites, with about 50% of patients having extra-nodal involvement. Extra-nodal involvement is seen most commonly in the gastrointestinal tract and bone marrow. Involvement of the pancreas by NHL has been reported infrequently.[2,3] Only about 0.2 - 2% of patients with NHL have pancreatic involvement at the time of presentation.[1,3,4] Histopathological examination is usually necessary to obtain a definitive diagnosis since symptoms and radiological features are similar to those of other pancreatic masses. We present a case of pancreatic lymphoma presenting as abdominal pain. The patient was diagnosed with a CT-guided biopsy and responded very well to chemotherapy.

Case Report

A 74-year-old Caucasian female patient presented with a past medical history significant for hypertension and arthritis. Her chief complaints were abdominal pain and weight loss. Pain was located in the epigastrium, sharp in nature, constant, and severe. Her symptoms began three months prior but worsened over time. She denied any fever or chills.

On examination, the patient had diffuse abdominal tenderness greatest in the epigastric region. Laboratory data showed iron deficiency anemia, sodium of 127 mEq/L, potassium of 2.9 mEq/L, and magnesium of 1.6 mEq/L. Liver function tests and lipase were normal. She underwent an abdominal ultrasound which showed a hypoechoic area in the spleen measuring 3.2 x 3.7 x 2.7 cm and was thought to be a cyst. She had worsening gastrointestinal symptoms as well as 20 pound weight loss.

A CT scan of the abdomen showed a soft tissue mass near the spleen originating from the tail of the pancreas with extension to and encasement of the superior mesenteric artery and celiac artery. At that point, unresectable adenocarcinoma of the pancreas was very high in the differential diagnosis and her options seemed to be limited. Her workup included a cancer antigen (CA) 19-9, which was 8, within the normal limit. Endoscopic ultrasound was performed with fine-needle aspiration (FNA) of the mass and sent for pathology. Her FNA was inconclusive and a CT-guided biopsy revealed a diffuse large B cell lymphoma, nongerminal center subtype with an immunoprofile positive for CD20, BCL-6, BCL-2, and negative for CD10. A positron emission tomography (PET) was negative in the neck, chest, and pelvis with no osseous or hepatic involvement of the metabolically-active neoplasm.

The patient was started on chemotherapy with R-CHOP (Rituximab, Cyclophosphamide, Doxorubicin, Vincristine and Prednisone) for six cycles. She had an excellent response to her first two cycles of chemotherapy and her repeat PET scan was

consistent with complete response. After six cycles of chemotherapy, her symptoms resolved completely.

Discussion

Diffuse large B-cell lymphoma infrequently involves the pancreas.[5,6] The presenting symptoms of pancreatic lymphoma are usually non-specific and include abdominal pain (83%), abdominal mass (58%), weight loss (50%), jaundice (37%), acute pancreatitis (12%), small bowel obstruction (12%), and diarrhea (12%).[5,6] Clues suggesting the possibility of a primary pancreatic lymphoma include a lack of jaundice, constitutional symptoms (weight loss, fever, and night sweats), an elevated serum lactate dehydrogenase (LDH) or beta-2 microglobulin level, and a normal serum CA 19-9.

Imaging is sometimes important in the diagnosis and staging of pancreatic lymphoma. CT scan is the modality commonly used for the detection of pancreatic lymphoma.[7,8] It can present either as a tumor-like, well-circumscribed hypo-echoic mass or as a diffuse enlargement infiltrating the pancreas.

Pancreatic lymphoma might be distinguished from pancreatic adeno-carcinoma by the absence of pancreatic duct involvement and the presence of surrounding lymphadenopathy.[7,8] Primary pancreatic lymphomas typically are larger than 6 cm, and surrounding lymph-adenopathy is common as with any lymphoma. Neither of these features, however, would exclude adenocarcinoma. It is unlikely to have a pancreatic adeno-carcinoma above 10 cm in size; about 60% of pancreatic lymphomas are greater than 6 cm in diameter.[9]

The location of the tumor in the pancreas does not appear to be helpful in determining whether the mass is adenocarcinoma or lymphoma. Ultrasound or CT-guided fine needle biopsy of the pancreatic mass can help in the diagnosis of pancreatic lymphoma.[7] In the absence of any pathognomonic clinical or radiological features, the diagnosis is established only on histopathological examination.

Our patient was found to have a mass on CT scan. Endoscopic ultrasound-guided (EUS) biopsy did not help in diagnosis, so CT-guided biopsy was done and pancreatic lymphoma was diagnosed. A PET scan did not show any metastasis. The patient responded very well to chemotherapy with resolution of symptoms and clearing of tumor burden on imaging.

Treatment and prognosis of pancreatic lymphoma are significantly different from those for pancreatic adenocarcinoma.[7] Anthracycline-based chemotherapy is the standard treatment for many types of NHL, and includes six to eight cycles of R-CHOP for patients of all ages.

Diffuse large B-cell lymphoma rarely presents with synchronous pancreatic and splenic localizations. On literature review, no other cases of primary lymphoma of the pancreas confounded with a splenic involvement were found. Although the splenic mass was not biopsied, it resolved after treatment with chemotherapy. Primary pancreatic lymphoma should be considered in the differential diagnosis of pancreatic tumors and an attempt to obtain a tissue diagnosis is always necessary before proceeding to radical surgery, especially on young patients.

Conclusion

Pancreatic B-cell lymphoma is a rare tumor. Most of the tumors of the pancreas are adenocarcinoma. Pathologic diagnosis is important in distinguishing both of them. It is crucial to diagnose pancreatic lymphoma because the prognosis and treatment are different. Most cases of pancreatic lym-phoma respond very well to chemotherapy.

EUS with fine needle aspiration is usually the diagnostic method used for the diagnosis of pancreatic malignancies; however, it has not been well studied in primary pancreatic lymphoma due to the rarity of the disease.

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Keywords: B-cell lymphoma, pancreatic neoplasms, adenocarcinoma

Peritoneal Tuberculosis in Dialysis: Fatal if Missed

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Introduction

The incidence of tuberculosis (TB) has been increasing worldwide.[1] Many risk factors are associated with TB, such as human immunodeficiency virus (HIV) infection, organ transplant, renal insufficiency, malignancy, and low socio-economic status.[2] Patients with end-stage renal disease (ESRD) are at increased risk of developing TB due to defective cell-mediated immunity.[3] The incidence of TB in dialysis patients is 5 to 15 times higher than the general population, increasing the morbidity and mortality in those patients.[4]

Case Report

A 60-year-old Hispanic female patient with a past medical history significant for ESRD on continuous ambulatory peritoneal dialysis (CAPD), hypertension, diabetes, and anemia presented with non-resolving abdominal pain of eight weeks duration. Pain was continuous, epigastric, and severe. She had multiple admissions for peritonitis within the prior two months without a microbiologic diagnosis. The patient had no known exposures to tuberculosis and no recent travel. She had emigrated from Mexico to the United States around 20 years prior. On physical exam, she was afebrile and tachycardic with diffuse abdominal tenderness; otherwise she had no major findings.

On admission, her complete blood count showed leukocytosis and anemia. Liver function tests were within normal limits. HIV and hepatitis panel were negative. She received broad spectrum empiric antibiotics while repeated bacterial and fungal cultures of the peritoneal fluid were negative.

Computed tomography (CT) of the abdomen and pelvis showed increased densities diffusely throughout the omental fat in the mid abdomen consistent with inflammation (Figure 1). Peritoneal fluid was cloudy in appearance and showed 735 white blood cells, 1% bands, 87% neutrophils, 1% lymphocytes, and 11% monocytes.

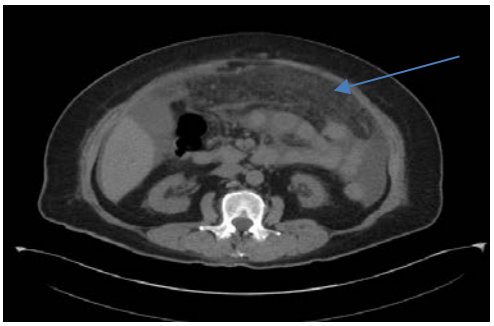

Figure 1. CT of the abdomen and pelvis showed increased density diffusely throughout the omental fat in the mid abdomen which is consistent with inflammatory changes (arrow).

Her peritoneal dialysis catheter was surgically removed and sent for cultures. A right internal jugular catheter was placed and she was switched to hemodialysis. Bacterial and fungal cultures remained negative. The acid fast bacilli (AFB) smear

was negative. AFB culture ultimately grew *Mycobacterium tuberculosis* (MTB) after four weeks of incubation on both the peritoneal catheter tip and peritoneal tissue.

She was started on isoniazid, ethambutol, pyrazinamide, rifampin and vitamin B6. Her chest-x-ray and sputum AFB cultures were negative. At two months, once susceptibilities were available, she was transitioned to isoniazid and rifampin to complete four more months. Her abdominal symptoms gradually resolved.

Discussion

Peritoneal tuberculosis is rare but remains a very important complication in CAPD patients. In 70 percent of cases, patients have symptoms for more than three months before the diagnosis is established which are usually indistinguishable from bacterial peritonitis.[5] The most common symptoms are abdominal pain (92%), cloudy peritoneal fluid (90%), and fever (78%).[6] There is a predominance of polymorphonuclear cells in the peritoneal fluid in 65% of cases, which can be misleading.

Different tests can be used to diagnose TB peritonitis in CAPD patients. AFB smear has limited sensitivity and specificity (all mycobacteria are acid fast). Culture of peritoneal tissue needs time to yield a diagnosis and has sensitivity between 38-98%.[2] Adenosine deaminase (ADA) levels have high sensitivity (100%) and specificity (97%).[7] The Quantiferon assay has a sensitivity of 93% and a specificity of 100%.[8] The polymerase chain reaction (PCR) is important for early diagnosis but has a sensitivity of 60-88 percent and specificity of 81-100 percent.[9]

The most common cause of peritonitis in dialysis patients is bacterial infection, and it should always be high in the differential diagnosis. Based upon 1108 episodes of peritonitis among 1015 CAPD patients, single gram-positive organisms, single gram-negative organisms, multiple organisms, fungi, and MTB caused 45, 15, 1, 2, and 0.1 percent of peritonitis episodes, respectively.[10] Since MTB peritonitis is very rare in CAPD, it is always important to think about it in patients with repetitive episodes of peritonitis with negative bacterial cultures and failure of antibiotic therapy. The average mortality rate is 15-30% with most significant factor being treatment delay.[6] Suspicion of TB peritonitis should be higher in patients that emigrated from countries with high prevalence of TB. Six months of anti-tuberculous therapy is effective and improves outcome.

Conclusions

A high index of suspicion is crucial to make the diagnosis of TB peritonitis in CAPD patients with recurrent peritonitis and negative bacterial or fungal cultures. Clinical findings in peritoneal TB are indistinguishable from those of bacterial peritonitis.[6] The gold-standard for diagnosis is growth of MTB from ascitic fluid or peritoneal biopsy specimen.[2] TB peritonitis could be fatal but is potentially curable if diagnosed in a timely manner.

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Keywords: peritoneal tuberculosis, dialysis, chronic kidney failure

Favorable Response to Chemotherapy in a Patient of Urachal Adenocarcinoma with Peritoneal Metastasis

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Introduction

Urachal adenocarcinoma is a rare cancer that arises from the embryological remnant of the urogenital sinus and allantois and is thought to comprise less than 1% of bladder cancers.[1,2] Mucinous adenocarcinoma is the most common subtype and presentation is similar to that of urothelial cancers, with hematuria and abdominal pain. Surgical resection is the first line treatment and better outcomes are seen with less advanced stage at diagnosis. Efficacy of chemotherapeutic regimens are being investigated; current five year survival rates are less than 50%.[1,2] We present this rare case of a gentleman diagnosed with mucinous urachal adenocarcinoma with peritoneal metastasis.

Case Report

A 70-year-old man was referred to our oncology clinic for an urachal tumor with peritoneal involvement diagnosed after inpatient workup for new onset ascites. The patient quit smoking 40 years prior and rarely drank alcohol. He had neither a family history of cancer nor any exposure to known environmental carcinogens.

The physical exam was notable for abdominal distension and ascites, causing dyspnea and hypoxia. Neither the liver nor the spleen was palpable, and there was no lymphadenopathy. Abdominal paracentesis was performed. Cytological specimen showed cellular fluid with atypical cells. Staining with mucicarmine and periodic acid-Schiff after digestion (PAS-D) showed extra-cellular mucin and small intra-cytoplasmic globules, which supported a diagnosis of mucinous adenocarcinoma (Figure 1).

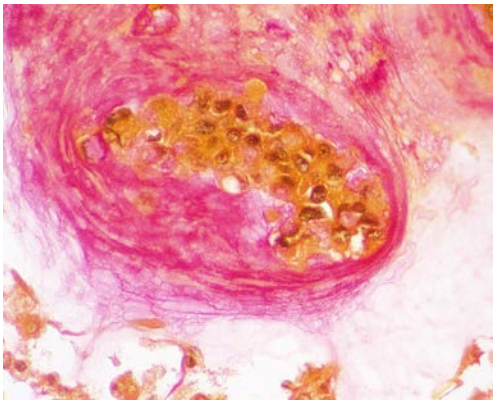

Figure 1. Cytologic specimen showed mucin-producing adenocarcinoma.

Immunohistochemistry showed tumor cells that were positive for CK-7 and CEA-m, and negative for CK20 and CD15. The absence of CK20 staining indicated that a primary GI malignancy is unlikely. Evaluation of abdomen and pelvis by computed tomography (CT) showed extensive ascites. The entire omentum had soft tissue streaking. A large, partly calcified mass extended from the dome into the urachal process measuring 7.3 cm x 4.3 cm x 8 cm (Figure 2). These histological and radiological findings confirmed the diagnosis of urachal carcinoma with peritoneal metastasis. Tumor stage was T4N0M1. A urologist recommended that surgical resection was not possible and referred the patient to oncology for

chemotherapy. Prior to chemotherapy, the patient was experiencing pain, dyspnea, and inability to carry out activities of daily living. Carcinoembryonic antigen (CEA) levels were 18.3 ng/ml. The patient was started on FOLFOX6, which consists of oxaliplatin 85 mg/m^2 intravenous (IV) infusion with leucovorin (LV) 400 mg/m^2 over two hours, followed by a 400 mg/m^2 bolus of 5-FU, followed by an IV infusion of 5-FU 2400 mg/m^2 for 46 hours. This regimen was repeated every two weeks.

He responded well as abdominal distension, pain, and dyspnea decreased, performance status improved, and CEA (Figure 3) decreased after the second cycle.

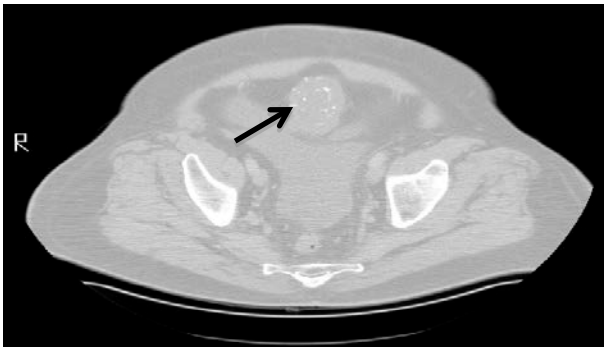

Figure 2. CT scan demonstrating calcific urachal mass.

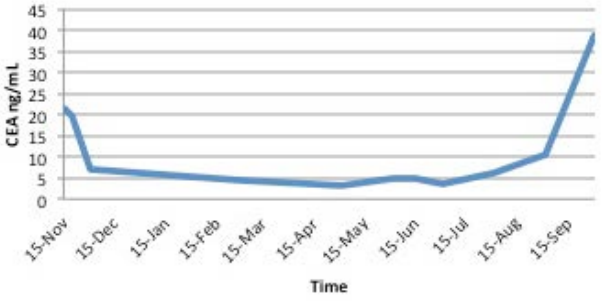

Figure 3. Carcinoembryonic antigen (CEA) response to 5-FU/FOLFOX treatment.

A CT showed decrease in ascites and no increase in tumor size after third cycle. After cycle 14, the patient developed numbness in the fingers, which was attributed to chemotherapy-induced peripheral neuropathy. In cycle 15, oxaliplatin was stopped, but leucovorin and 5-FU were continued. At this point, CEA levels plateaued at 3.45 ng/ml (Figure 3). Four more cycles with 5-FU and leucovorin were given. After cycle 18, CEA started to rise and CT showed slight increase in size of tumor. After cycle 22, CEA increased further to 10.4-ng/mL and tumor size also continued to increase. However, the patient's symptoms were well controlled. Second line chemotherapy with irinotecan was started, but the patient was unable to tolerate it due to severe vomiting. The patient opted for hospice and later died.

Discussion

The urachus is a remnant of the channel between the fetal urinary bladder and umbilicus. It is formed from the allantois between the 5th and 7th week of gestation and typically is sealed and obliterated by the 12th week. It is identified postnatally as the medial umbilical ligament.[1] Failure of the urachus to seal may result in patent urachus, cyst, fistula, diverticulum, or sinus. Urachal cancer may develop from these urachal remnants, and adenocarcinoma occurs in the vast majority of these cases, often mucin-producing.[2,3] It is a rare cancer and constitutes less than 1% of bladder tumors.[4]

Risk factors are not well defined and do not appear to be related to bladder carcinogens.[5,6] Urachal adenocarcinoma is more common in men and onset is most often during middle age. Gross or microscopic hematuria is the most common presenting symptom, though abdominal pain, fullness, and other urinary symptoms have been reported.[3,7,8]

The Sheldon criteria represent the significant clinical and pathological characteristics of urachal cancer. These include the location of the tumor in the bladder dome or anterior wall, evidence of a sharp demarcation between the tumor and surface epithelium, the absence of cystitis, and exclusion of primary adenocarcinoma located elsewhere that has spread secondarily to bladder.[9-13] If a biopsy of the bladder dome or tumor reveals adeno-

carcinoma, urachal cancer should be suspected, and diagnosis may be confirmed with immunohistochemistry.[12,14]

Urachal adenocarcinoma and colonic adenocarcinoma are histologically similar and associated with elevations in CEA and CA 19-9. No molecular markers have been discovered that have any prognostic implications.[7] Urachal adenocarcinoma commonly recurs, often with peritoneal carcinomatosis, as well as metastasis to liver and lung. Surgical resection is the only curative treatment and includes complete resection of the tumor, urachus, and umbilicus as well as pelvic lymph node dissection.[3,15-19]

The Sheldon TNM staging system or the Mayo staging system may be used for prognosis, both are about equally efficacious.[7] Negative surgical margins and no lymph node involvement are good prognostic indicators. Median survival is 48 months, with five year overall survival rate of 45%.[5] However, presence or absence of metastasis affects survival,[11] with 61% relative five-year survival in patients without metastatic disease, versus 15% relative five-year survival in patients with metastatic disease.[5] Chemotherapy may improve survival in patients with metastasis or lymph node involvement.[16]

There is no established chemotherapy regimen for metastatic urachal carcinoma, but chemotherapy regimens used for colonic adenocarcinoma have been tried.[9,16] Regimens of 5-FU and cisplatin have shown about 40% response in some studies.[16,20] Efficacy of chemotherapy regimens are being investigated.[3,19,21-23]

Conclusion

Urachal adenocarcinoma is a rare cancer that occurs most often in middle-aged men. Surgical resection may provide cure in patients with early stage disease but chemotherapeutic responses are low and most of short duration. This case represented a significant response to the FOLFOX regimen.

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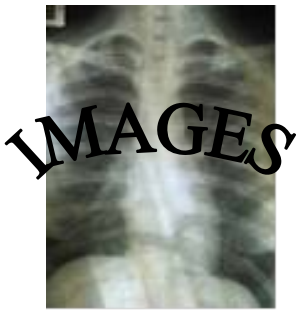
Keywords: urachal adenocarcinoma, carcinoembryonic antigen, fluorouracil, FOLFOX protocol



Organizing Pneumonia Associated with TNFα Inhibitor

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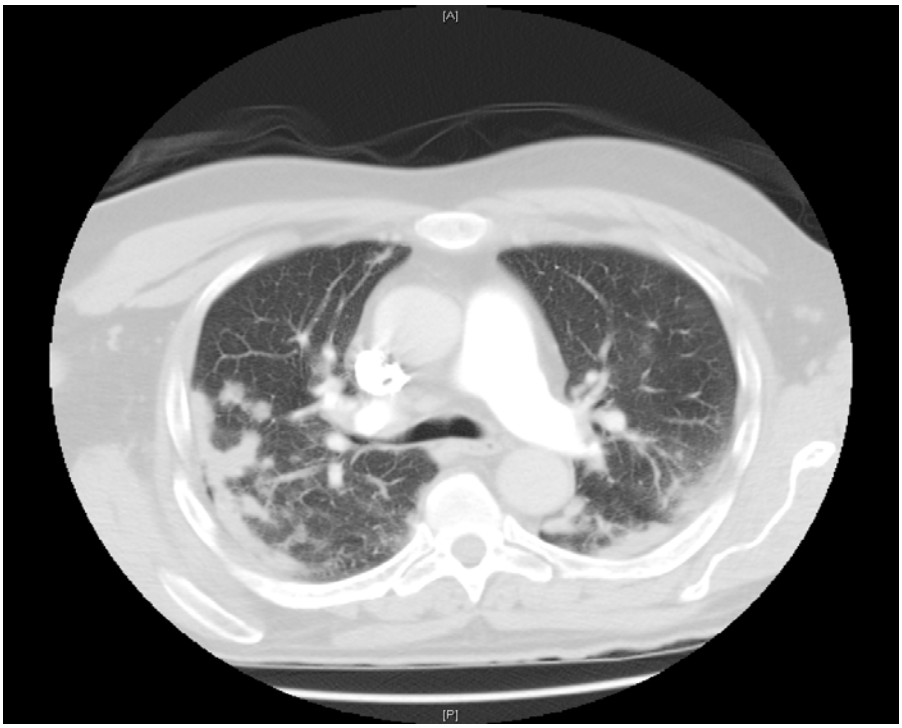


A 64-year-old man with a history of rheumatoid arthritis (RA) on treatment with methotrexate 22.5 mg weekly and adalimumab 40 mg biweekly, presented to the emergency department with complaints of cough, dyspnea, and fatigue. The patient reported cough and dyspnea over the eight weeks prior to presentation. He was treated as an outpatient for cough with a course of azithromycin, then doxycycline. However, he progressively worsened over the month prior to presentation as he began to experience dyspnea on exertion.

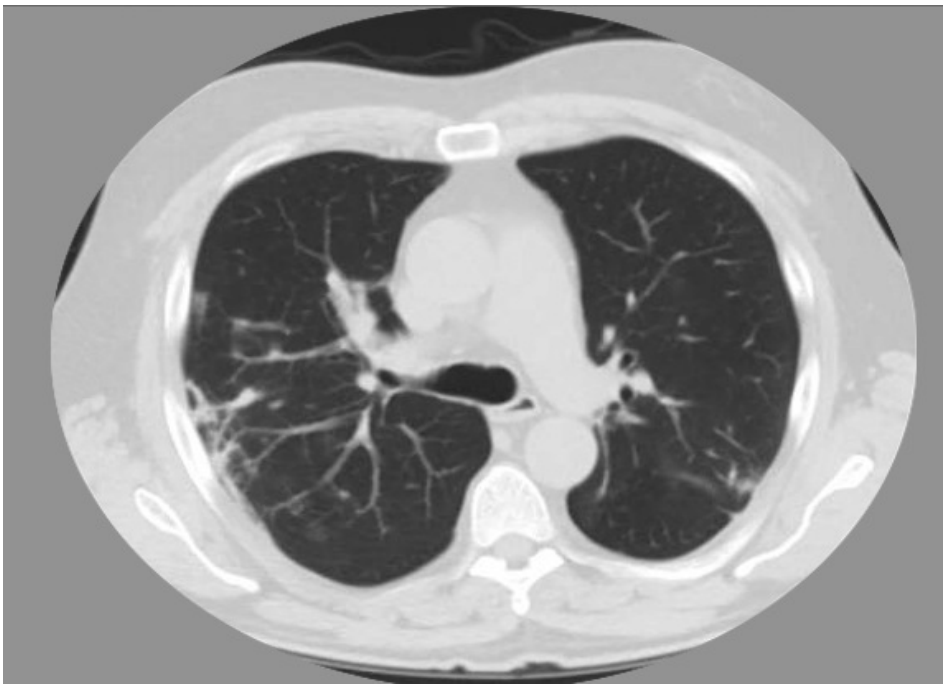
Upon presentation, the patient was afebrile with stable vital signs. He had a normal cardio-pulmonary examination. His complete blood count with differential, comprehensive metabolic panel, and brain natriuretic peptide were within normal limits. A chest x-ray revealed diffuse infiltrates. A computed tomography (CTA with/without contrast pulmonary embolism protocol) of the chest was negative for pulmonary embolism, but showed diffuse five lobe alveolar infiltrates with posterior predominance. No thoracic lymphadenopathy was noted (see image above).

The patient was admitted for further work-up of progressive dyspnea with failed outpatient treatment and a bronchoscopy was performed the next day. On bronchoscopy, the pharynx,

larynx, and trachea appeared to be normal. The right bronchial tree appeared to be normal including the right upper, middle, and lower lobes. There were no endo-bronchial lesions noted. The mucosa was normal. Next, the left bronchial tree segments were inspected including the left upper and lower lobes.

A bronchoalveolar lavage (BAL) was performed from the left upper lobe (lingula) with a total of 150 ml's of fluid instilled and 120 ml's withdrawn. The return was slightly cloudy/cellular but there were no purulent secretions. There was no evidence of diffuse alveolar hemorrhage. The BAL fluid analysis reported a white blood count of 370 cells/mcL, 6% polymorphonuclear leukocytes, and 54% lymphocytes. Gram stain of the BAL fluid revealed moderate neutrophils, acid-fast bacilli stain and culture were negative. Fungal culture showed only light growth of budding yeast. Negative results were obtained on cytology and a respiratory viral panel for cytomegalovirus, herpes simplex virus, adenovirus, and pneumocystis jiroveci pneumonia. He was initiated on prednisone 60 mg daily with a taper of 10 mg every week for a total of six weeks and was discharged from the hospital to outpatient follow-up.

Upon follow up, the patient reported his cough and dyspnea were improved from his initial presentation. Follow-up CT of the chest one month later showed significant improvement in organizing pneumonia (see figure below).



Organizing pneumonia is defined histopathologically by intra-alveolar buds of granulation tissue, consisting of intermixed myofibroblasts and connective tissue.[1] The most common pulmonary disease associated with the use of TNF inhibitor administration in systemic autoimmune disease are interstitial pneumonia and sarcoid-like disorder. Anti-TNF agents commonly are associated with interstitial lung disease (ILD) and the most are administered for RA. The most common presenting symptoms include dyspnea, cough, and fever.[2] Withdrawal of the agent and initiation of corticosteroids are treatment mainstays for organizing pneumonia. Complete resolution is reported in 40% of the cases and improvement or partial resolution in 25% of the cases with no resolution in 35%.

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Keywords: interstitial lung disease, tumor necrosis factor alpha, rheumatoid arthritis