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Secondhand Tobacco Smoke Exposure Among Hospitalized Nonsmokers With Coronary Heart Disease

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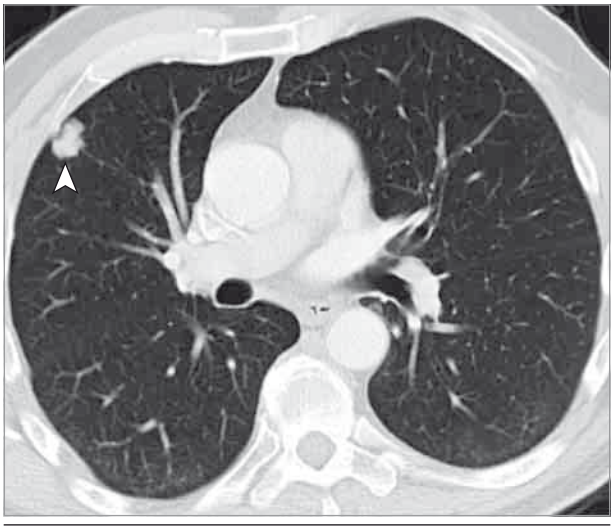
Figure. Computed Tomographic Scan of Chest (Case 3)



Non–small cell adenocarcinoma of the lung (arrowhead) that caused symptoms attributed to chronic Lyme disease.

Shortly afterward, an expanding 18 × 15-cm erythematous rash appeared below his left shoulder. The rash resolved but malaise and fatigue recurred. Two additional doxycycline courses provided only transient improvement. Five months after his initial diagnosis, the patient was referred to an infectious disease specialist for presumed chronic Lyme disease.

The results of the physical examination and laboratory evaluation were normal except for a slightly elevated white blood cell count. Results of serologic testing for Lyme disease were consistent with previous infection (Table). The patient had a remote 18 pack-year history of smoking. The chest radiograph revealed a 1.1-cm nodular mass in the right upper lobe confirmed by computed tomographic scan (**Figure**). Further evaluation demonstrated stage I non-small cell adenocarcinoma, which was successfully resected.

Discussion | Patients 1 and 2 had no evidence of ever having Lyme disease. Patient 3 likely had true *Borrelia burgdorferi* infection for which antibiotic therapy was appropriate; however, subsequent symptoms were incorrectly attributed to persistent infection.

Chronic Lyme disease is a misleading term that should be avoided.[2] *Posttreatment Lyme disease syndrome* is the proper term for patients with a verified previous *B burgdorferi* infection who experience fatigue, arthralgias, or other symptoms 6 months or more after antibiotic treatment when all other conditions have been ruled out.[1,2,5]

We are not suggesting that every patient with nonspecific symptoms, such as fatigue, joint pain, or abdominal pain, should be aggressively evaluated for cancer. Rather, we present these cases to demonstrate delays in diagnosis that come from assuming that patients have chronic Lyme disease.

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Study concept and design: Nelson, Elmendorf.
Acquisition, analysis, or interpretation of data: All authors.
Drafting of the manuscript: Nelson, Elmendorf.
Critical revision of the manuscript for important intellectual content: All authors.
Administrative, technical, or material support: Nelson, Elmendorf.
Study supervision: Mead.

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Secondhand Tobacco Smoke Exposure Among Hospitalized Nonsmokers With Coronary Heart Disease

Exposure to secondhand tobacco smoke (SHS) increases adult nonsmokers' risk of cardiovascular disease by 25% to 30%.[1] Among nonsmokers hospitalized with acute coronary syndrome, SHS exposure is associated with a higher likelihood of subsequent cardiovascular and all-cause mortality as well as reinfarction.[2,3] Hospitalized nonsmokers with coronary heart disease (CHD) should avoid SHS exposure after discharge, but little is known about the frequency of SHS exposure in this population or whether clinicians (including nurses, nurse practitioners, physician's assistants, and physicians) address it. The present study assessed self-report and biochemical measures of SHS exposure among hospitalized nonsmokers with CHD and explored patients' beliefs and the clinicians' actions about SHS.

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Methods | The study was approved by the Massachusetts General Hospital/Partners Health Care System Institutional Review Board. Participants provided oral consent; they re-

Table 1. Characteristics of the Sample and Measures of Secondhand Tobacco Smoke Exposure in 214 Patients

Characteristic	No. (%)
Age, mean (SD), y	68 (11)
Male sex	160 (74.8)
Race/ethnicity	
Non-Hispanic white	173 (80.8)
Non-Hispanic black	2 (0.9)
Hispanic	11 (5.1)
Asian	4 (1.9)
Other	24 (11.2)
Educational level	
High school diploma or less	85 (39.7)
Some college or vocational school	27 (12.6)
4-y College graduate	102 (47.7)
Primary discharge diagnosis	
Coronary arteriosclerosis (*ICD-9*, code 414)	113 (52.8)
Acute myocardial infarction (*ICD-9*, code 410)	47 (22.0)
Chest pain (*ICD-9*, code 780-786)	22 (10.3)
Aortic valve disorder (*ICD-9*, code 424-427)	12 (5.6)
Congestive heart failure (*ICD-9*, code 428)	10 (4.7)
Esophageal disorder (*ICD-9*, code 530)	2 (0.9)
Other	8 (3.7)
Any household member smokes	29 (13.6)
Relationship of household smoker to patient[a]	
Spouse	12 (5.6)
Child ≤18 y	0
Child >18 y	15 (7.0)
Other adult	7 (3.3)
Smoking ban	
Home[b]	145 (67.8)
Car[c]	138 (72.3)
Home and car[d]	116 (54.2)
Exposure to secondhand tobacco smoke	
Self-report (30 d before hospital admission)	
Any site (home, car, or work)	47 (22.0)
Home	27 (12.6)
Car	34 (15.9)
Work	15 (7.1)
Home or car	37 (17.3)
Self-report (7 d before hospital admission)	
Any site (home, car, or work)	33 (15.4)
Home	17 (7.9)
Car	12 (5.6)
Work	16 (7.4)
Home or car	22 (10.3)
Saliva cotinine level	
≥0.20 ng/mL	15 (8.2)
Median	<0.20, Nondetectable[e]
Range	<0.20-5.77
≥0.05 ng/mL	29 (40.3)
Median	<0.05, Nondetectable[f]
Range	<0.05-2.29

Abbreviation: *ICD-9*, *International Classification of Diseases, Ninth Revision*.

SI conversion factor: To convert cotinine to nanomoles per liter, multiply by 5.675.

[a] Patients could have more than 1 smoker in their household.

[b] No one was allowed to smoke vs all other response options.

[c] No one was allowed to smoke vs all other response options among 191 patients with a car.

[d] No one was allowed to smoke in the home and, if the patient owned a car, in the car.

[e] Limited to the 184 analyzable samples of the 214 samples collected because some samples were not of sufficient volume for analysis. The limit of quantification was 0.20 ng/mL for all samples.

[f] Limited to 72 analyzable samples with an assay limit of quantification of 0.05 ng/mL.

ceived no financial compensation. The study was conducted in the inpatient cardiac service of Massachusetts General Hospital, Boston. Eligible patients were aged 18 years or older, reported no tobacco or nicotine replacement use, had ischemic CHD as an admission diagnosis, spoke English, were medically stable, had no significant cognitive impairment, and were hospitalized for 48 hours or less. Consenting patients had a bedside interview regarding their demographics; SHS exposure in their home, car, and work; home and car rules about smoking; beliefs about the risk of SHS exposure; and interventions regarding SHS exposure by "a doctor, a nurse, or other health care professional."[4] A saliva sample was collected for an assay of cotinine, a nicotine metabolite with a 16-hour half-life.[5,6] The limit of quantitation of the assay was 0.20 ng/mL for the first 112 samples and 0.05 ng/mL for the last 72 samples (to convert cotinine to nanomoles per liter, multiply by 5.675). The discharge diagnosis was obtained from the medical records.

Results | Between May 25, 2010, and January 27, 2011, a total of 3152 nonsmokers were admitted to the cardiac service; of these, 2192 individuals (69.5%) had a CHD diagnosis, 230 (7.3%) met the eligibility criteria, and 214 (6.8%) enrolled in the study. The primary reasons for ineligibility were more than 48 hours since admission (41.7%) and discharge before the research staff could visit (34.5%). **Table 1** reports characteristics of the sample.

Secondhand tobacco smoke exposure was reported by 47 patients (22.0%) in the 30 days before hospital admission and by 33 patients (15.4%) in the 7 days before admission (Table 1). Twenty-nine patients (13.6%) lived with a smoker, who was most likely an adult child or a spouse. Two-thirds of the patients (67.8%) reported having a household smoking ban, and 72.3% of the patients with a car reported having a car smoking ban.

Among the 184 individuals with sufficient samples for analysis, 15 (8.2%) had detectible cotinine (≥0.20 ng/mL). Among the 72 saliva samples analyzed with the more sensitive assay, 29 (40.3%) had detectable cotinine (≥0.05 ng/mL) (Table 1).

Table 2. Awareness of the Health Risks of SHS in 214 Patients

Characteristic	No. (%)
Beliefs about SHS	
Harmful to nonsmokers' health	
Very/somewhat	192 (89.7)
Not very/not at all	15 (7.0)
Do not know	7 (3.3)
Increases nonsmokers' risk of "heart attack"	
A lot/somewhat	121 (56.5)
A little/not at all	47 (22.0)
Do not know	46 (21.5)
Increases your own risk of "heart attack"	
A lot/somewhat	121 (56.5)
A little/not at all	56 (26.2)
Do not know	37 (17.3)
Worry about current SHS exposure	
Very worried	31 (14.5)
Somewhat/a little worried	73 (34.1)
Not at all worried	108 (50.5)
Do not know	2 (0.9)

Abbreviation: SHS, secondhand tobacco smoke.

Most patients (89.7%) believed that SHS was harmful to nonsmokers' health (**Table 2**). Although 56.5% of the respondents believed that SHS exposure increased nonsmokers' risk of "heart attack," 22.0% disagreed and 21.5% did not know. Similar results were found when patients were asked if SHS exposure increased their own risk of "heart attack" (Table 2). Half of the patients were "not at all" worried about their SHS exposure.

Only 37 patients (17.3%) recalled that a hospital physician or nurse had asked about their SHS exposure since admission. Only 21 (9.8%) had been asked if they lived with a smoker, and only 3 (1.4%) individuals were advised in the hospital to keep their home or car smoke free.

Discussion | The findings of this study make a strong case for the need to address SHS exposure more effectively in inpatient cardiology practice. Nonsmokers who were hospitalized with CHD were rarely screened for SHS exposure or advised to avoid it, even though 15.4% reported recent SHS exposure and 40.3% had detectable levels of a biomarker of SHS exposure. It is likely that SHS exposure is similarly overlooked in outpatient cardiology practice. Hospitals and health care systems are missing an opportunity to identify and intervene in this major modifiable cardiovascular risk factor.

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Study concept and design: Japuntich, Shenhav, Park, Winickoff, Rigotti.
Acquisition, analysis, or interpretation of data: Japuntich, Eilers, Shenhav, Winickoff, Benowitz, Rigotti.
Drafting of the manuscript: Japuntich, Winickoff, Rigotti.
Critical revision of the manuscript for important intellectual content: Eilers, Shenhav, Park, Winickoff, Benowitz, Rigotti.
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Study supervision: Japuntich, Park, Rigotti.

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Invited Commentary

Learning to Act on Secondhand Tobacco Smoke Exposure to Limit Risk for Coronary Heart Disease

Secondhand tobacco smoke (SHS) consists of a combination of mainstream smoke that is exhaled from a smoker and sidestream smoke that is given off by a burning cigarette. There is no known safe level of SHS exposure, and it is associated with multiple health risks including sinorespiratory disease, cancer, and cardiovascular disease. In children, SHS increases the risk of pneumonia, bronchitis, severe asthma, and sudden infant death syndrome, and in adults SHS increases the risk of chronic obstructive pulmonary disease and sinus disease. Multiple national and international agencies have classified SHS as a human carcinogen because it increases the likelihood for individuals to develop cancers of the lung, breast, sinus, head, and neck. Secondhand tobacco smoke also increases the incidence of acute coronary events, a finding that is underscored by the observation that implementation of smoking bans in public places has been associated with decreased hospitalization for acute coronary events. Nonsmokers hospitalized with an acute coronary syndrome appear to be particularly vulnerable to SHS exposure because they have higher rates for 30-day mortality, reinfarction, and hospital readmission.[1,2] This is not just a matter of academic interest because reports suggest that as many as 29% of the patients seen by the cardiology service during an inpatient admission[3] and 40% of the general hospitalized population[4] have detectable evidence of SHS exposure.

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It is in this context that the study by Japuntich and colleagues[5] is particularly relevant. The authors performed a cross-sectional survey of 214 nonsmokers admitted to an inpatient cardiology service with a diagnosis of coronary heart disease between 2010 and 2011. They measured exposure to SHS, assessed patients' risk awareness related to their exposure, and examined the degree to which clinical staff addressed patients' SHS risk through counseling. The authors explored SHS exposure by patient self-report and confirmed it by measuring the nicotine metabolite cotinine in saliva using high-performance liquid chromatography and atmospheric pressure ionization tandem mass spectrometry with detection limits of 0.2 ng/mL for the first 112 samples and 0.05 ng/mL for the last 72 samples (to convert cotinine to nanomoles per liter, multiply by 5.675). Results showed that 15.4% of the participants reported SHS in their home, car, or workplace in the week before their admission. Only 8.2% of the patients had a detectable salivary cotinine level using the higher detection limit of 0.2 ng/mL or more, whereas 40.3% had a detectable cotinine level using the lower detection limit of 0.05 ng/mL or more. Almost all participants (89.7%) knew that SHS exposure was harmful, but only 56.5% were aware that SHS exposure increased a nonsmoker's risk of myocardial infarction or specifically increased their own risk of myocardial infarction. Most disconcerting is the finding that only 17.3% of the patients remembered being asked about their SHS exposure by a health care worker after their admission, and only 1.4% reported receiving any counseling related to this exposure. The study was limited because it was conducted in a single institution, and only a portion of the patients had salivary cotinine measured using the more sensitive detection limit of 0.05 ng/mL or more.

This study is important because it clearly demonstrates where the health care community has failed to translate research into action. In this case, the deleterious effects of SHS exposure on patients admitted with acute coronary syndrome are clear. Despite this knowledge, very few health care professionals inquire about SHS exposure and virtually none follow through with counseling, which could be lifesaving. It is not easy to translate important therapies into clinical practice, as is evidenced by the low delivery of guideline-based care for many chronic diseases.[6,7] This is a case in which the electronic health record could make a big difference in both the inpatient and outpatient settings through the development of "prompts" to inquire about SHS exposure and "hard stops" to encourage counseling during what may be a prime teachable moment. Regardless of the method used to stimulate counseling by health care providers, the present study emphasizes the need to allocate energy and resources to uncover the effects of SHS exposure and learn how to maximally implement these findings in patients to improve their health.

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