A Chronic Care Model Program Incorporating Group Office Visits for Obesity Treatment in Primary Care

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Abstract

Background. Obesity is a chronic disease of epidemic proportions. Primary care providers are on the front line of diagnosing and treating obesity and need better tools to deliver top-notch obesity care.

Methods. A pilot randomized trial was conducted to test a chronic care model (CCM) program for obesity compared to usual care. Primary care patients, 18 years and older, with a body mass index (BMI) between 27 and 45 were enrolled. Sixteen weekly 90-minute group office visits were structured with the first 30 minutes encompassing individualized clinical assessments and the final 60 minutes containing the group-based standardized intensive lifestyle training. The primary outcome was weight change at 16 weeks. Secondary outcomes were weight change at 24 weeks, change in diet and physical activity behaviors, self-efficacy for weight control behaviors, and physiologic markers of cardiovascular risk at 16 and 24 weeks.

Results. The participants (19 in the active arm and 10 in the control arm) were 49.8 ± 11.5 years old (mean ± SD), 97% women, 55% white, and 41% black. Weight change in the control arm at week 16 was 0.25± 2.21 kg (mean ± SD) and that for the active arm was -5.74 ± 4.50 kg (n=16). The difference between the two arms was significant (p = 0.0002). Both the intent-to-treat analysis using the last observation carried forward approach and the analysis including completers only provided similar siginificant results.

Conclusions. This study demonstrated that a CCM program incorporating group office visits was feasible and effective for obesity treatment in primary care settings.

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Introduction

According to a recent World Health Organization (WHO) report, overweight and obesity are the fifth leading cause for global deaths: at least 2.8 million adults die each year as a result of being overweight or obese.[1] In addition, 44% of the diabetes burden, 23% of the ischemic heart disease burden, and between 7% and 41% of certain cancer burdens are attributable to overweight and obesity. In the US, the obesity epidemic is particularly severe and elevating. According to the Center for Disease Control and Prevention (CDC), no state met the Healthy People 2010 obesity target of 15%.[2] Nine states have obesity rates over 30%, and the self-reported overall prevalence of obesity among US adults had increased 1.1 percentage points from 2007 to 2009. Obesity and obesity-related health issues have been a heavy burden on health

care costs. Obesity accounts for 9.1% of annual health care spending in the US. The medical care costs of obesity in the US are staggering. In 2008 dollars, these costs totaled about $147 billion.[3]

Primary care providers play a critical role in curbing the escalating obesity epidemic. There are many important reasons for mobilizing the primary care workforce around this leading public health threat, including the above-average prevalence of obesity among primary care patients compared with the general population,[4,5] the positive impact of physician advice on behavior change,[6] and routine patient-provider contact affording many opportunities for obesity care. Nonetheless, a well-known gap exists between best practices for obesity and actual care. Using a nationally representative sample, Galuska et al.[7] reported that only 42% of obese patients had been advised by their physician to lose weight in the prior 12 months. In the same study, those that received physician advice for weight loss had 3-fold higher odds of attempting weight loss than those without physician advice. Improving the recognition and treatment of obesity in primary care settings is an important national objective.[8,9]

Primary care physicians have identified a number of barriers that impact obesity care, including time, resources, insurance reimbursement, and lack of knowledge about how to incorporate obesity interventions in primary care.[4,10] A system-based, multi-disciplinary approach to obesity care may overcome some of these barriers.[11] As a previous study pointed out, basic treatment of overweight and obese patients in primary care requires a comprehensive approach involving diet and nutrition, regular physical activity, and behavioral change, with an emphasis on long-term weight management rather than short-term extreme weight reduction.[12]

The chronic care model (CCM) is an interdisciplinary team approach to chronic disease management that engages patients, providers, office staff, health system administrators, and communities.[13] Given that obesity is a chronic disease with links to several common and serious chronic diseases,[14] this clinical approach holds much promise for obesity care.[15]

Components of the CCM may be promising in obesity intervention in primary care. For example, group office visits have been supported by several prominent national medical societies as an innovation with great potential to improve chronic disease management in primary care.[16] There has been much excitement for group visits and several practice-based group case reports of improved chronic disease outcomes, more patient satisfaction, and lower costs, yet there are only a handful of studies in which group office visits have been rigorously tested.[17]

Another important component of the CCM is intensive lifestyle training. Structured behavioral interventions consistently have demonstrated adequate weight loss and weight loss maintenance.[18,19] Herein, a pilot randomized trial of a CCM program for obesity incorporating clinician group office visits and intensive lifestyle training in two academic primary care practices is described. The intensive lifestyle behavioral intervention model of the Diabetes Prevention Program (DPP) was adopted. It had been used successfully for diabetes prevention.[19] The primary goal of this study was to evaluate the feasibility of the program and to assess effect sizes for future definitive trials of the intervention.

Methods

This study was approved by the institutional review board at the University of Kansas Medical Center (KUMC).

Study design. This study was a randomized clinical trial with two arms. Eligible primary care patients were randomized to receive either: 1) standard of care consisting of patient educational materials about obesity (the control arm) or 2) a 16-week chronic care model program integrating clinician group visits with intensive lifestyle training and other components of the chronic care model program (the active arm). The randomization ratio of active to control was 2:1.

The intervention components of the active arm were derived from the general principles of the CCM,[17] including 16 weekly 90-minute group office visits. The first 30 minutes of the visit were for individualized assessments and the other 60 minutes were for intensive lifestyle training including dietary modification, physical activity, goal setting for weight control, self-monitoring, and behavioral change strategies. Weekly self-administered surveys and semi-structured individual interviews also were included to evaluate subject satisfaction with the intervention.

An interview guide was developed that consisted of a series of questions designed to elicit information about facilitators and barriers of the weight management program. To reduce potential bias, the interviewer (CG) was not involved in the trial and participants were assured that their responses would be de-identified. The interviews were not tape recorded. During the interviews, brief notes were taken and immediately following each interview, the interviewer elaborated on her notes.

At baseline, 16 weeks, and 24 weeks, all participants in the active and control arms completed a self-administered, 60-item survey and physiologic assessments querying: 1) sociodemographics, 2) comorbid obesity-related medical illnesses, 3) self-efficacy for weight control as measured by the 20-item Weight Efficacy Lifestyle Questionnaire, 4) health-related quality of life using the Short Form-12,[20] 5) depressive status using the Patient Health Questionnaire-2,[21] 6) blood pressure, fasting cholesterol profile, and fasting glucose, 7) usual dietary patterns using 24-hour dietary recalls and recorded on the Nutrition Data System Software (Univ. of MN, 2007) for daily energy, fat, fruit and vegetable, and fiber intake,[22,23] and 8) regular physical activity patterns using the International Physical Activity Questionnaire.[24] These dietary and physical activity self-report assessments have been validated and widely used for similar purposes. Health status,[25,26] and depression[27,28] are potentially important moderators of weight loss completion and maintenance. The primary outcome measure was weight (kg); all other measures were secondary outcome measures.

Participants. Eligible participants were 18 years or older with a body mass index (BMI) between 27 and 45 and weight stable in the last 3 months (i.e., within +/- 10 pounds), not taking obesity pharmacotherapy, not planning bariatric surgery in the next 12 months, not pregnant or lactating in the last 12 months, and free from terminal illness with a life expectancy greater than 12 months.

Procedure. Subjects were identified from the General Internal Medicine (GIM) and Family Medicine (FM) clinics at University of Kansas Medical Center (KUMC). The group office visits were conducted in the General Clinic Research Center (GCRC) at KUMC due to space and staff limitations in the primary care clinics. Participants were recruited during the course of clinical care during October 2007 and the protocol was conducted from November 2007 through March 2008.

In total, 78 patients were screened and 29 eligible participants agreed to participate in the study. At a 2:1 ratio, 19 participants were randomly assigned to the active arm

and 10 to the control arm and had baseline measurements. At week 16, eight participants in the control arm and 16 participants in the active arm returned for assessment. Six participants in the control arm and 17 participants returned for the assessment at week 24. Among the participants of the active arm, 14 participants returned for the assessment at week 16 and attended more than 50% of the 16 weekly sessions. These subjects were program "completers". Figure 1 describes participant enrollment and retention in greater detail.

For the intervention arm, each individualized assessments session prior to group meetings was provided by a primary care physician (author ACE), a clinic RN, or a psychologist (authors CAB or ABD). During the course of the 16-week intervention, four individualized assessments were provided by the physician, four by the nurse, and eight by one of the psychologists. For each session, all participants were assessed by the same provider. Participants were advised to set realistic, guideline-based goals for weight control including 1-2 pound weight loss per week, 150 minutes of moderate/vigorous physical activity per week, less than 25% daily calories from fat, five servings or more of fruits and vegetables per day, and an overall goal of 10% body weight loss and weight loss maintenance. At each meeting, participants reviewed goals and problem-solved to reduce barriers of weight control adherence.

Statistical analysis. Univariate analysis compared patient characteristics at baseline between the two arms. For continuous variables, such as weight, the two sample t-test was used for comparison if the normality assumptions were satisfied; otherwise, the Wilcoxon rank sum test was used. The Chi-square test was used for categorical variables such as gender and race. For the changes of outcome variables from baseline to 16 and 24 weeks, two-sided, a two-sample t-test or Wilcoxon rank sum test was used.

Three different sets of analysis were used on the changes of outcome measures. First, an analysis based on observed data only. All individuals with missing information were excluded for the corresponding analysis. Second, an intent-to-treat analysis included all individuals randomized, for which the simple last-observation-carried-forward (LOCF) strategy was used to replace missing values. The LOCF strategy is equivalent to assuming no changes since last time of measurement. Finally, given the well-known problem of recidivism in weight control studies, an analysis of "completers" included individuals in the control arm who had no missing observations and those in the active arm who had no missing measures and completed at least 50% of group office visits. As an exploratory pilot study, no control for multiple tests was considered. All analyses were conducted using STATA 10 (STATACorp LP, College Station, TX).

Analysis of the interviews was thematic in approach.[29] The participants' responses were reviewed and reduced to key themes. Discussion of the themes and topics were held between the first author (EAC) and the interviewer (CG).

Results

The 29 participants were 49.8 ± 11.5 years old (mean ± SD), 97% female, 55% white, and 41% black. Baseline body mass index (BMI) was 37.5 ± 5.4 (mean ± SD), baseline body fat percentage was 48.7% ± 4.9%, and baseline total daily energy intake as measured by one baseline 24-hour diet recall was 1738.1 ± 821.9. Additional baseline descriptions are in Table 1. Except for body fat, variables were not significantly different between the two arms.

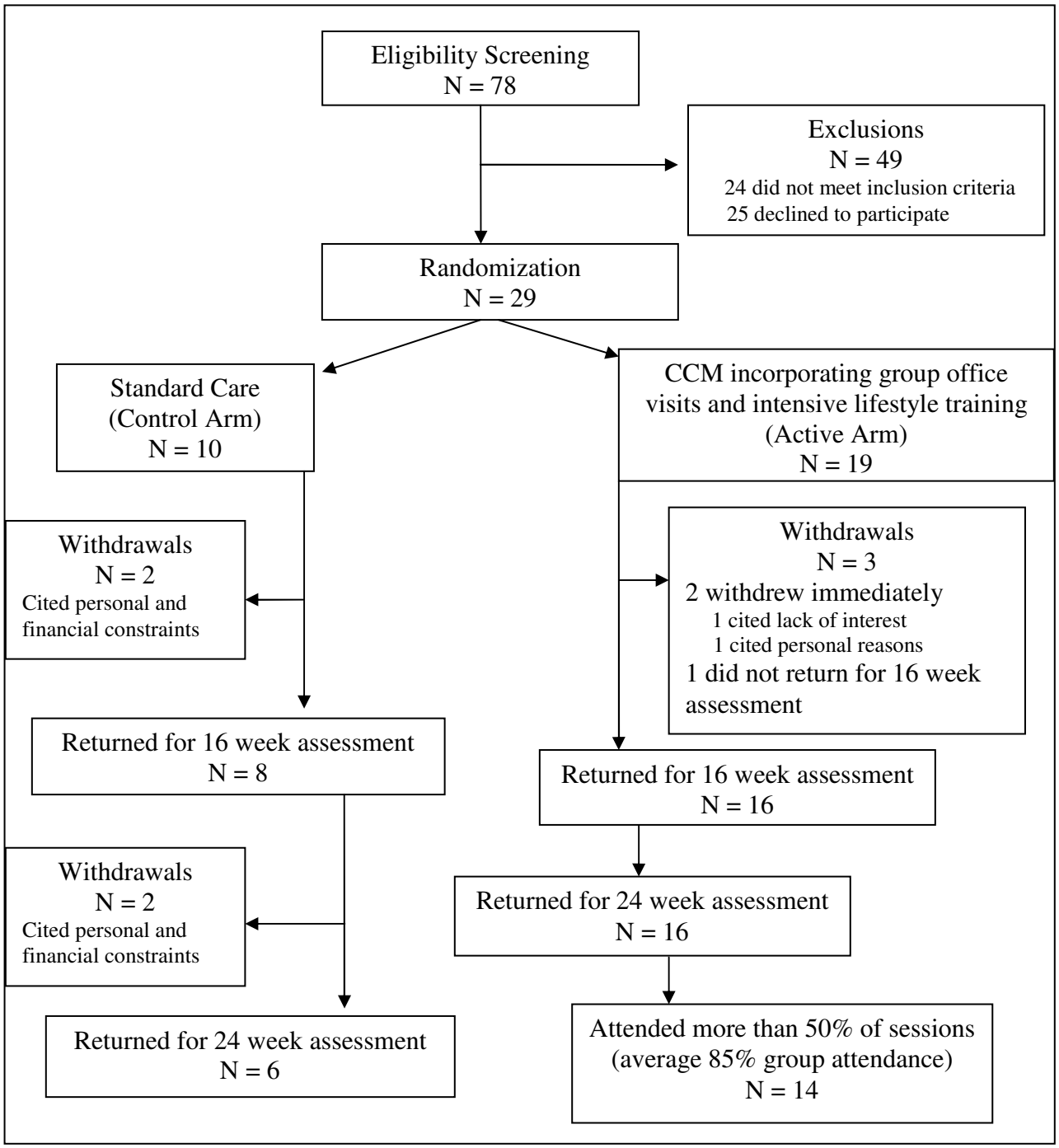

Figure 1. Participant enrollment and study completion

Based on patients with observed measures only (see Table 2), weight change in the control arm at week 16 (n = 8) was 0.25± 2.21 kg (mean ± SD) and that for the active arm was -5.74 ± 4.50 kg (n = 16). The difference between the two arms was significant (p = 0.0002) using a two-sided two-sample t-test. Figure 2 demonstrates the distributions of the observed weight change at week 16 for the two arms, showing that participants in the active arm had significant weight loss while those in

Table 1. Participant baseline characteristics by study arm.*

Participant Characteristic	Active Arm (%); N=19	Control Arm (%); N=10
Age		
Mean years ± SD	51 ± 10	48 ± 15
Gender		
Female	19 (100%)	9 (90%)
Race/ethnicity		
Black	7 (37%)	2 (20%)
White	11 (58%)	4 (40%)
Latino	1 (5%)	2 (20%)
Other	1 (5%)	
Annual Income		
<$10K	1 (5%)	1 (10%)
$10K-$20K	3 (16%)	0 (0%)
$21K-$50K	12 (63%)	6 (60%)
>$50K	3 (16%)	3 (30%)
Insurance Type		
Medicare	4 (21%)	2 (20%)
Medicaid	3 (16%)	0 (0%)
Employer Based	11 (58%)	4 (40%)
Other	2 (11%)	2 (20%)
Comorbidities		
Hypertension	9 (47%)	5 (50%)
Diabetes	5 (26%)	3 (30%)
Hyperlipidemia	10 (53%)	6 (60%)
Heart Disease	2 (11%)	2 (20%)
Arthritis	10 (53%)	4 (40%)
Sleep apnea	8 (42%)	1 (10%)
Depression	9 (47%)	3 (30%)
Anxiety	6 (32%)	4 (40%)
Regular alcohol use	2 (11%)	1 (10%)
Tobacco use	1 (5%)	2 (20%)
Body Mass Index		
Mean ± SD	38 ± 5	37 ± 6
Body Fat (DEXA assessed) **		
Mean % ± SD	50 ± 4	46 ± 6
Fasting biomarkers		
Mean ± SD		
Glucose mg/dl	103 ± 15	112 ± 39
Triglycerides mg/dl	128 ± 47	110 ± 54
HDL mg/dl	50 ± 9	48 ± 16
LDL mg/dl	115 ± 34	98 ± 37
Health Status^		
SF-12 PCS, Mean ± SD	38 ± 12	44 ± 13
SF-12 MCS, Mean ± SD	46 ± 13	51 ± 15
Depression Screening#		
PHQ 2		
Positive	3 (16%)	1 (10%)

* Cells may not sum to 100% secondary to rounding and/or missing values. Missing values are not listed here, but are available on request.

^ Short Form 12, Physical and Mental Component Summary Scores are standardized and weighted using US population estimates that summarize general health status. Scores range from 0-100 with higher scores indicating better health status (Mean ± SD PCS for US is 50 ± 10, and MCS for US is 50 ± 10).

Patient Health Questionnaire 2, ≥ 3 is positive depression screen.

** P <.05 for a two-sided test of the difference between arms.

Table 2. Weight change, dietary, and physical activity outcomes at 16 weeks.*

Outcome variable	Active Arm		Control Arm	
	Baseline N=16	16 Weeks N=16	Baseline N=8	16 Weeks N=8
Weight **				
Mean kilograms ± SD	98 ± 16	92 ± 14	102 ± 20	102 ± 21
Total daily calorie intake ***				
Mean kcal ± SD	1845 ± 1116	1210 ± 414	1746 ± 776	2091 ± 758
Total daily fat intake ***				
Mean gm ± SD	80 ± 44	35 ± 19	71 ± 29	98 ± 52
Physical Activity				
Mean minutes per week ± SD				
Vigorous Activity	116 ± 410	117 ± 237	21 ± 38	6 ± 15
Moderate Activity	65 ± 152	158 ± 256	36 ± 37	37 ± 66
Walking	256 ± 546	281 ± 621	155 ± 84	220 ± 220
WEL-Global^	88 ± 32	100 ± 23	87 ± 46	97 ± 29
Body Fat (DEXA assessed)				
Mean % ± SD	50 ± 3	49 ± 4	46 ± 6	46 ± 6
Fasting biomarkers				
Mean ± SD				
Glucose mg/dl	100 ± 9	94 ± 10	102 ± 20	107 ± 28
Triglycerides mg/dl	122 ± 36	122 ± 63	98 ± 40	106 ± 59
HDL mg/dl	50 ± 10	48 ± 10	45 ± 20	45 ± 18
LDL mg/dl	116 ± 38	112 ±38	93 ± 48	109 ± 45
Daily fruits intake Mean servings per day ± SD	1.9 ± 3.4	2.0 ± 1.8	1.4 ± 1.8	.6 ± .6
Daily vegetable intake Mean servings per day ± SD	3.3 ± 2.5	3.5 ± 2.7	3.6 ± 2.4	4.4 ± 1.1
Blood Pressure				
Mean SBP mm Hg ± SD	125 ± 16	122 ± 12	134 ± 12	134 ± 17
Mean DBP mm Hg ± SD	75 ± 9	75 ± 6	74 ± 5	76 ± 10

*****Cells may not sum to 100% due to rounding and/or missing values. Missing values are not listed here, but are available on request.

^Weight Efficacy Lifestyle Questionnaire (20 Likert items), range 20-200 (higher score=higher self-efficacy).

** P<.001 for a two-sided test of the difference between arms in terms of change from baseline to week 16.

*** P<.05 for a two-sided test of the difference between arms in terms of change from baseline to week 16.

the control arm did not. Using last-observation-carried-forward to replace missing observations (for the control arm, it was equivalent to assuming no change from baseline), the weight change in the control arm at week 16 was 0.20 ± 1.95 kg (n = 10) and that for the active arm was -4.83 ± 4.64 kg (n = 19). This difference also was significant (p = 0.004). For the active arm, weight change for "completers" (n = 14) was -6.39 ± 4.39 kg. The weight change was significant (p = 0.0001) when compared with that of the eight control participants who returned for assessment at week 16. The observed weight change from baseline to week 24 in the active arm was -5.55 kg ± 5.38 kg (n = 17) and that in the control arm was -0.61 kg ± 2.57 kg (n = 6). The weight changes at week 24 were significantly different between the two arms (p = 0.006).

Total daily calorie and fat intake decreased in the active arm by week 16 (-635 ± 702 kcal, mean ± SD, and -45 ± 25 gm, mean ± SD, respectively). Moderate physical activity increased in the active arm by week 16 (93 ± 104 minutes, mean ± SD), and walking minutes per week increased in the control arm by week 16 (65 ± 136

minutes, mean ± SD). Secondary outcomes at week 24 were not different from week 16, thus are not presented.

Attendance was 72% on average (excluding one active arm participant who dropped out before the first session). Overall, participants reported high satisfaction with the program as measured by the weekly satisfaction surveys. Results from the process evaluation via semi-structured individual interviews with participants were reviewed for common themes. The common themes are detailed in Table A1 in the Appendix. We believe this qualitative information offers important insight into the intervention process. This may be valuable in future work using a similar design to address participants' concerns and guide intervention development. Results from the interviews with providers and practice teams are available on request.

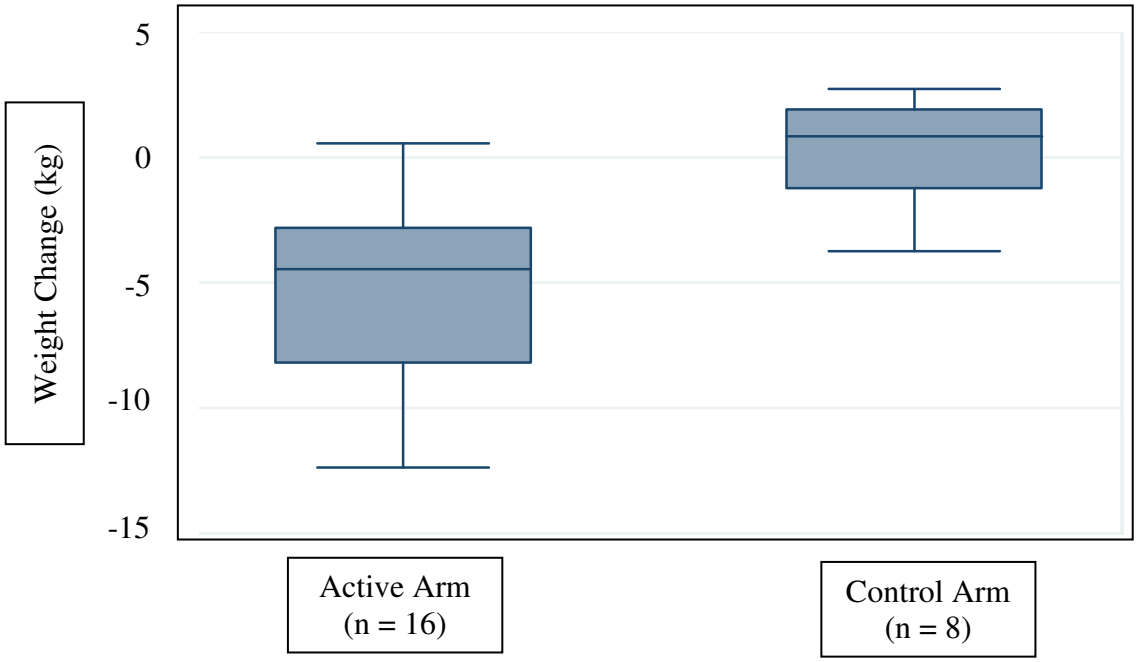

Figure 2. Weight Change at Week 16: Observed Changes Only

Discussion

This project was a pilot trial of a chronic care model for obesity incorporating group office visits and intensive lifestyle training in two academic primary care practices. Group office visits for obesity were feasible, acceptable, and effective for weight loss. Participants who completed at least 50% of sessions (74% of active arm) were successful at reaching their initial weight loss goal of 5-10% body weight loss. This goal was consistent with national guidelines for weight control in primary care settings.[8,9] This study was one of the first to translate the Diabetes Prevention Program into primary care practice, and to our knowledge was the first to use group office visits to treat obesity. Furthermore, it is one of the first studies to use the chronic care model for obesity care. These findings are promising and warrant exploration in definitive future trials.

The chronic care model for obesity was used in our past work in rural Kansas primary care settings.[30] However, patient

activation in that study was accomplished through individual telephone-based counseling rather than the group office visits used in the current study. In both the past work and the current study, provider and health system activation were more challenging than patient activation. This challenge may be related to more resource allocation, and a project focus on the patient-oriented chronic care model.

The multifaceted nature of these interventions renders it difficult to assess which aspect had the greatest impact on outcomes. Nonetheless, semi-structured process interviews with participants following the current study suggested that patient activation via the group office visits and intensive lifestyle training had the most powerful impact of any intervention piece in the current study. Given the experience of others using the chronic care model in improving outcomes in chronic disease,[31] this approach holds much promise for obesity care. Future work is needed to advance the chronic care model constructs that have not been well developed in obesity interventions to date.

In this study, group office visits were used in a novel way to manage obesity among primary care patients. Others have described successful experiences using group office visits for chronic disease management. Primary care patients with coronary artery disease were randomized to monthly group office visits including extensive nutritional education or usual care.[32] Group office visits resulted in improved physiologic outcomes, greater patient satisfaction, and better health-related quality of life. Also, group office visits improved dietary behaviors and physiologic outcomes among primary care patients with diabetes mellitus.[33]

The participants in the current study enjoyed the interface of medical management with the intensive lifestyle training. No significant changes were found in fruits and vegetables consumption, which may due to the short time frame of the study.

Limitations of this study were that the intervention was conducted in a research clinic separate from the point of care and that the medical management was directed by a research physician (ACE). This was done secondary to resource, time, and space limitations, and because it was an exploratory pilot intervention. Future work needs to test a similar model on a larger scale engaging the actual practice teams in primary care settings.

Other limitations to the current study were the small scale of the study (only 29 participants) and the disproportionate loss of the control arm participants to followup at 24 weeks (only 60% of controls returned for assessments). The loss of participants was consistent with our past work and that of others, which outlines the frustration of a usual care only arm in weight control studies. Nonetheless, clinically and statistically significant weight control outcomes were demonstrated. Also, the short time frame of the follow-up period (i.e., 24 weeks) rendered it difficult to assess the long term effect of the intervention. Weight loss maintenance as a critical component of weight control interventions in primary care settings should be examined in the future with larger studies. Other important aspects that were not covered in the pilot study, such as more effective strategies to improve compliance and cost-effective analysis, also need to be assessed in the future.

Obesity is a chronic disease of epidemic proportions in the United States. Primary care physicians have been called to optimize diagnosis and treatment of obesity. A chronic care model intervention for obesity incorporating group office visits and intensive lifestyle training may be one way of improving the quality of obesity care in primary care settings.

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Keywords: obesity, primary care, chronic disease

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Appendix

Table A1. Summary of semi-structured participant interviews by topic.

Topic Area	Summary Results from 30-Minute Interviews with 14 Participants
Visits with continuity primary care physician	About half of participants who had a clinical visit with their primary care physician during the study indicated improved obesity care
Brief structured individual visits with research physician during group office visit	Most participants indicated that these visits were helpful and encouraging. A few indicated that the visits were often rushed and disorganized.
Brief structured individual visits with study nurse during group office visit	About half indicated that the nursing visits were helpful and encouraging. The other half did not find them helpful because one of the nurses was not supportive at all, and there was not continuity of nursing care.
Brief structured individual visits with health psychologists to review health behavior goals related to weekly diet diaries	All participants found these visits very helpful in staying on track and learning new tips for weight control. Several did report that they often felt rushed and wanted more time.
Intensive lifestyle training group sessions	Most felt that the sessions were extremely helpful and they appreciated the support of others and the information learned. Several reported that sessions were rushed and they wished there was more time for in depth discussion.
Calorie and fat intake goals	Several felt that these were unattainable, but most felt that the goals were helpful in planning meals.
Food and activity logs	Several felt that the self-monitoring was cumbersome and not helpful at all. Most agreed that the idea was important, but the reality of self-monitoring was a lot of work.
Major barriers to reaching weight control goals during the study	Lack of will power, reluctance to monitor calories, lack of exercise, high stress, poor emotional state, comorbid medical conditions
General comments and feedback	Learned a lot, enjoyed the group camaraderie, would have preferred smaller group with a more comfortable room with a place to write and place materials

The Simplified Geneva Score and the Utilization of the D-Dimer and Computerized Tomography for Assessing Pulmonary Embolism

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Abstract

Background. Pulmonary embolism (PE) is clinically suspected in many patients who complain of shortness of breath or chest pain due to its nonspecific nature. The prevalence of PE, however, is low in this population. To assist physicians in diagnostic decision making, several clinical decision rules (CDR) have been developed. The appropriate use of these CDRs has been proven to decrease the need for expensive, time consuming, and invasive diagnostic imaging procedures. In this study, the appropriateness of D-dimer and CT usage was investigated to rule out pulmonary emboli based on the simplified Geneva score.

Methods. A retrospective review was performed on 74 patients with a CT scan ordered through a pulmonary embolism (PE) protocol. Using clinical data, the patients were stratified into "unlikely" and "likely" groups for the presence of PE based on the simplification of the revised Geneva score. Scores of 0-2 were graded as "unlikely" and scores of 3 or greater were "likely."

Results. There were 45/74 (60.8%) patients in the "unlikely" group. Of these, 14/45 (31.1%) received a D-dimer; eight were normal and six elevated. Only one patient in the elevated group had evidence of a PE. Of the remaining 31(39.2%) patients in the "unlikely" group that did not receive a D-dimer, only one had a PE. The "likely" group consisted of 29 (39.2%) patients of whom six received a D-dimer. Three patients had a normal D-dimer and three had an elevated level. Neither of these two groups had a PE. Of the remaining 23 (60.8%) in the "likely" group who did not receive a D-dimer, six had a PE.

Conclusions. Diagnosing pulmonary emboli using D-dimer levels and CT scans may be aided by clinical decision rules such as the simplified Geneva system. This process may lead to more effective use of medical resources.

KJM 2011; 4(4):99-104.

Introduction

Pulmonary embolism (PE) is clinically suspected in many patients who complain of shortness of breath or chest pain due to its nonspecific nature. The prevalence of PE, however, is low in those patients with these symptoms. To assist physicians in diagnostic decision making, several clinical decision rules (CDR) have been developed.[1] The appropriate use of these CDRs have been proven to decrease the need for expensive, time consuming, and invasive diagnostic imaging procedures.

Two CDRs that have been studied extensively with proven validity are the Well's rule[2] and the Geneva score[3]. The Well's rule lacks full standardization, as one

criterion includes the physician's judgment whether an alternative diagnosis is more likely than PE. The predictive value of the Well's rule may be from this subjective component.[1] The Geneva score also has weaknesses, as it has eight variables, all with different individual scores, which can make it apt to errors and less likely to be used.

A simplified Geneva score has been developed, by assigning each of the 8 variables a value of 1.[1] This simplified score was found to be equivalent to the revised Geneva score with regards to diagnosing pulmonary embolism correctly in a sample of 1049 patients. The authors concluded that the simplified Geneva scoring did not lead to a decrease in diagnostic accuracy or clinical utility.[1] Furthermore, the study also used a highly sensitive D-dimer assay to categorize people into different probability groups. The study suggested that the use of the D-dimer was important and very sensitive in ruling out PE and that its use should be limited to the group "unlikely" to have PE. It also supported the notion that the immediate use of computerized tomography (CT) should be reserved only for those in the "likely" to have PE (i.e., high probability) group.

Methods

A retrospective review was performed on CT scans (Spiral CT chest with IV contrast using 1.25 mm slices) ordered through a PE protocol from 1/5/09 to 4/7/09 at the University of Kansas Medical Center (KUMC). Patients with charts that did not contain enough data to obtain a simplified Geneva score and those who received a CT scan for any other reason besides ruling out PE were excluded. All records, laboratory, and imaging results were accessed via KUMC's electronic medical record (O_2® and CHARTMAXX®). Data collected included clinical risk factors, location of the encounter, D-dimer levels, and CT results. The D-dimer was considered as having been done only if results were obtained within 24 hours prior to the CT. Two different D-dimer tests were used and cutoffs for a normal D-dimer were 230 mg/L[4] or 1.0 g/L (manufacturer's cutoff), depending on the assay.

Patients were scored as "likely" or "unlikely" to have pulmonary embolus using the simplified Geneva scoring protocol[3] (see Table 1). Scores of 0-2 were graded as "unlikely" and scores of 3 or greater were "likely." Scoring was completed using the data closest to, but prior to the time of the CT being ordered.

Table 1. Simplified Geneva scoring to determine patients likely or unlikely to have a PE.

Variable	Score
Age > 65	1
Previous PE or DVT	1
Active cancer or cured < 1 year	1
Surgery within 1 month (under general anesthesia or lower extremity fracture)	1
Unilateral lower limb pain	1
Hemoptysis	1
Heart rate 75-94 beats per minutes	1
Heart rate > 94 beats per minute*	1
Unilateral lower limb swelling and pain with palpation	1

*Patients with a heart rate of > 95 receive one point for heart rate between 75-94 and one point for > 94, for a total of 2 points.

Results

A total of 74 patients met the eligibility criteria. The population demographics are shown in Table 2. Thirty-two (43.2%) CT scans were ordered from a non-intensive care unit (ICU) inpatient admission, 24 (32.4%) from the emergency department, 16

(21.6%) from the ICU (medical, surgical, or neurologic), and only two (2.7%) from the outpatient clinics.

Table 2. Population demographics.

Characteristic	Total (Percentage)
Over age 65	23 (31.1%
Male	33 (35.0%)
Female	41 (55.0%)
Cancer (active or cured < 1 year)	23 (31.1%)
History of DVT or PE	11 (14.9%)

The group "unlikely" to have a PE according to the simplified Geneva score consisted of 45 (60.8%) patients. Of those, 14 (31.1%) received a D-dimer; eight were normal and none had evidence of PE. Six D-dimer levels were elevated and 1 patient in this group had a PE. Out of the remaining 31(39.2%) patients in the unlikely group who did not receive a D-dimer, only one had a PE (see Figure 1).

The "likely" group consisted of 29 (39.2%) patients of which six (20.7%) received a D-dimer. Three patients had a normal D-dimer and the other three had an elevated level. None had any evidence of PE. Of the remaining 23 (60.8%) likely patients who did not receive a D-dimer, six were found to have a PE (see Figure 1).

The D-dimer ordering status was analyzed by department (see Table 3). In the emergency department 26.7% (4/15) of the "unlikely" and none (0/9) of the "likely" patients received a D-dimer. On the hospital inpatient ward, 33.3% (7/21) of the "unlikely" and 33.3% (3/9) of the "likely" patients received a D-dimer. In the Intensive Care Unit (ICU), 37.5% (3/8) of the "unlikely" and 37.5% (3/8) of the "likely" patients received a D-dimer. In the outpatient clinics, no patients in either group received a D-dimer.

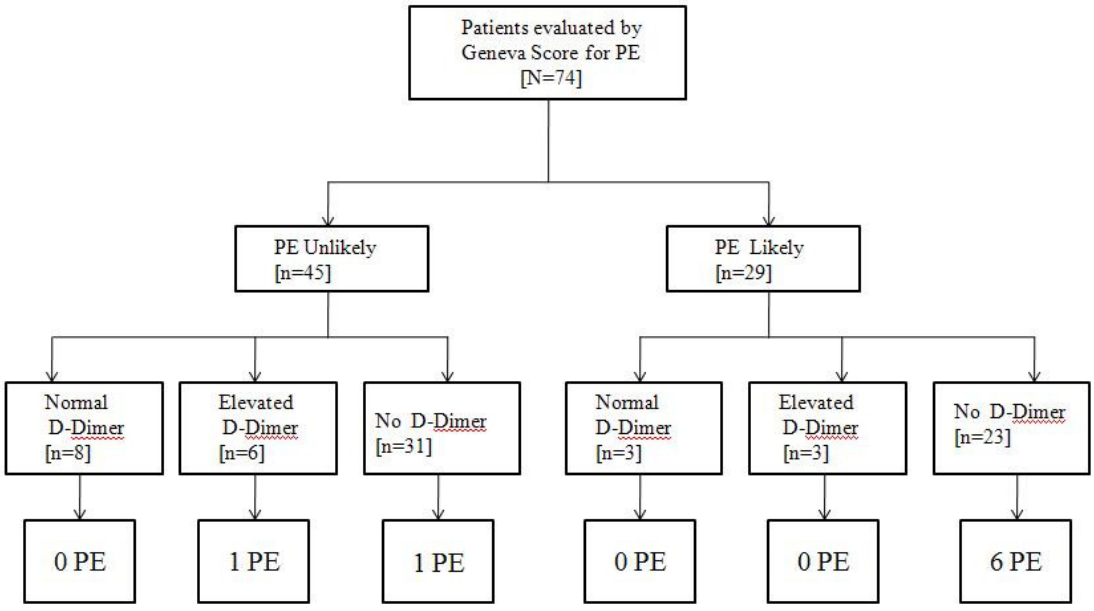

Figure 1. Stratification of patients based on the simplified Geneva score and D-dimer status. (Diagnosis was confirmed by CT scan using a PE protocol which is the gold standard investigation for PE.)

Table 3. Location of D-dimer ordering for patients who were likely or unlikely to have PE based on simplified Geneva score.

Location	"Unlikely" with D-dimer Percent (number)	"Likely" with D-dimer Percent (number)
Emergency Department	26.7% (4/15)	0% (0/9)
Hospital (Inpatient Ward)	33.3% (7/21)	27.3% (3/11)
Intensive Care Unit (Neurological, medical, surgical)	37.5% (3/8)	37/5% (3/8)
Outpatient Clinics	0% (0/1)	0% (0./1)

Statistical analysis comparing the accuracy of the simplified Geneva score against the PE protocol CT scans (the gold standard investigation) determined the sensitivity of the Geneva score to be 0.75 and specificity of 0.65. This correlated with a positive predictive value of 0.21 and negative predictive value of 0.96. The continuous receiver operating characteristic (ROC) curve was plotted (see Figure 2). The area under the curve was 0.78 +/- 0.1 [95% CI: 0.571, 0.990]. These data were comparable to those reported by Klok et al.[1] that found an area under the curve (AUC) of 0.74 [95% CI: 0.70, 0.77].

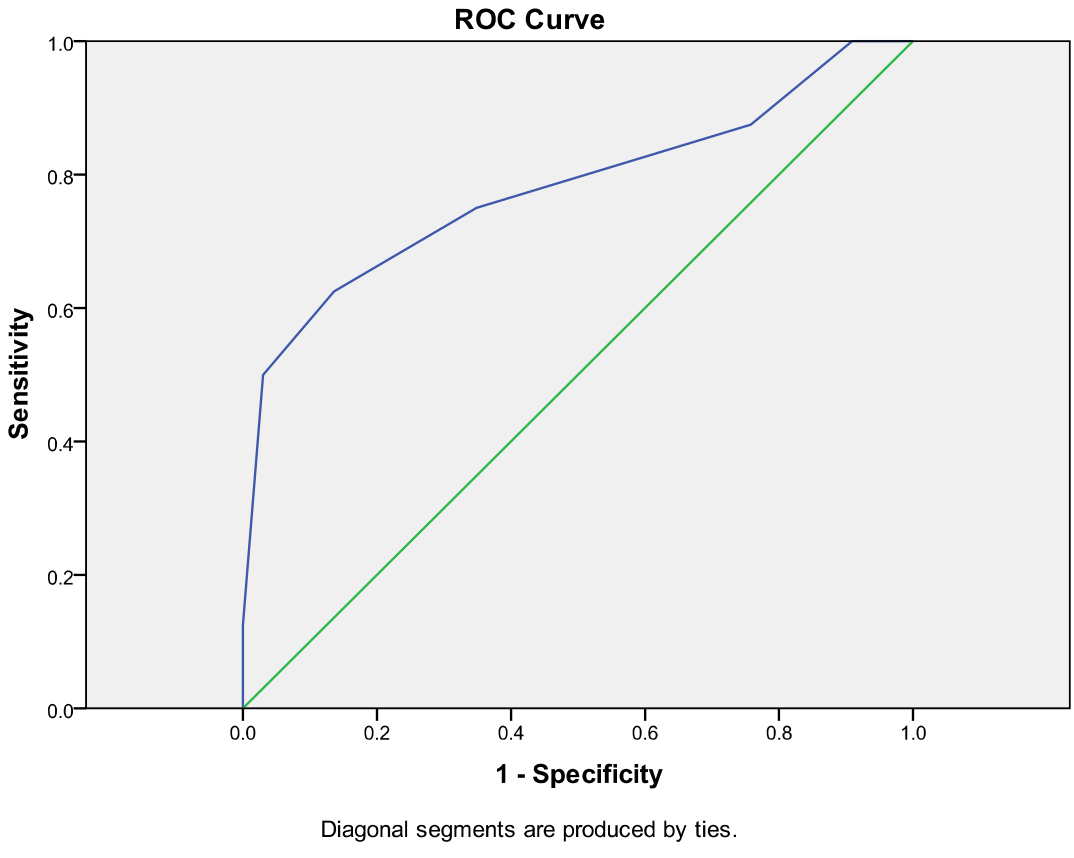

Figure 2. Continuous receiver operating characteristic curve of the Simplified Geneva Score.

Discussion

In the current healthcare climate, the allocation of limited medical resources is becoming increasingly more important. At KUMC, the charge for a CT scan ordered from a PE protocol was $2,487. In comparison, the charge for a D-dimer test was $278. In this study, the "unlikely" group may have received too many CT scans as only 2/45 (4.4%) patients had a PE. The D-dimer also seemed to be underutilized as 31/45 (68.9%) patients did not have one ordered. For the "likely" group, 6/29 (20.7%) patients had a pulmonary embolism, representing a 4.7 fold increase as compared to the "unlikely" group. In contrast to the underutilization of the D-dimer test in the "unlikely" group, there was an overutilization in the "likely" group. The D-dimer was ordered in 6/29 (20.7%) patients. These were considered unnecessary as a CT scan ordered from the PE protocol (i.e., the diagnostic gold standard) was ordered already. Of note, all 11 patients with a normal D-dimer did not have a PE. This result was consistent with the study by Klok et al.[1] who had a total of 330 patients in the "unlikely" group with normal D-dimers and had zero incidence of PE.

The utility of CDRs has been shown in multiple studies. However, if they are not used routinely, their efficiency and reliability becomes futile. When they are used judiciously, clinical judgment still must be taken before applying them to patient care. Clinical symptoms alone (as evaluated from the simplified Geneva score) did not reliably predict the presence of PE as only 20.7% (6/23) of "likely" groups had a PE.

Clinicians in a previous study[5] did not document all the elements of a CDR properly and suggested the need for paper or electronic aids in conjunction with their use. In addition, CDRs and D-dimer levels had a lower specificity in cancer patients, hinting at the need to modify established CDRs further and changing the D-dimer cut-off levels in special patient populations.[6] In our series, we had 23 cancer patients of which 3 had a PE. All 3 patients were in the "likely" group and had a negative D-dimer showing that the specificity of D-dimer in our study was not adequate, and that a different D-dimer threshold for these patients may be warranted in this population. Other strategies to reduce CT utilization are also underway, as a more sensitive D-dimer test using the Tina-quant assay reduces the number of scans by 16%[7], and a new diagnostic strategy combining clinical assessment, the D-dimer, ultrasonography, and lung perfusion scans required only 11% of patients to receive a CT scan.[8]

Our study is limited, in part, because it is a retrospective review. The clinical data were obtained through review of charts and not actual assessment of patients. The chart reviewers were not blinded to CT results. In addition, only patients who received CT scans were studied and those who were "ruled out" in other ways were excluded. Given these caveats, this study showed that stratification according to the simplified Geneva score produced a 4.7 fold (20.7% vs 4.4%) increase in the diagnosis of PE in the "likely" vs "unlikely" groups. These results provide further support for the ability of the simplified Geneva score to stratify patients according to risk. In addition, the stratification with the Geneva score and D-dimer also highlighted the fact D-dimers were underutilized in the "likely" group and given the negative predictive value of 0.96 in our study, unnecessarily ordered in the "unlikely" group.

In conclusion, diagnosing pulmonary emboli using D-dimer levels and CT scans may be aided by a CDR such as the simplified Geneva system. In those patients who are in the "unlikely" group with a

normal D-dimer, given the NPV of 0.96, a PE may be ruled out safely even without a CT scan. This process may lead to more effective usage of D-dimer levels and CT scans obviating the need for extra medical resources.

Acknowledgements
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Keywords: pulmonary embolism, D-dimer, x-ray computerized tomography, diagnosis

Endogenous Invasive Community-Acquired Methicillin-Resistant *Staphylococcus aureus* Endophthalmitis: Observations in Two Cases

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Introduction

Community-acquired methicillin-resistant *Staphylococcus aureus* (CA-MRSA) infections remain a growing problem despite the increasing armamentarium of anti-MRSA antibiotics. The majority of reported CA-MRSA infections are skin and soft tissue infections. Recently, more invasive and life-threatening infections have been recognized.[1] Ophthalmic infections have been reported less frequently and CA-MRSA endogenous endophthalmitis has not been well described.[2] The clinical presentations, treatment, and outcome of two cases of suspected invasive CA-MRSA endogenous endophthalmitis are discussed in this report.

Case Reports

Case 1. A 26-year-old man presented to a community hospital with a right arm subcutaneous abscess. Incision and drainage was performed. The wound culture grew MRSA sensitive to all antibiotics tested except oxacillin and penicillin. He was treated with trimethoprim-sulfamethoxazole. Three days later, he developed lumbago. After two weeks of symptomatic treatment, Magnetic Resonance Imaging (MRI) appeared to demonstrate L5-S1 disk herniation. Epidural corticosteroid injection was administered. Five days later, he developed syncope and supraventricular tachycardia. He was transferred to our facility for evaluation.

Laboratory results revealed leukocytosis of 17.1 $x10^9$/L, 32% bands, and erythrocyte sedimentation rate was 53 mm/hr. He was started on vancomycin 15 mg/kg IV every 12 hours. Blood cultures grew MRSA, sensitive to fluoroquinolones and resistant to oxacillin and erythromycin (VITEK Automated Microbiology System). Repeat MRI of the spine demonstrated discitis at the L5-S1 disk space with associated epidural abscess and vertebral osteomyelitis.

Over the next three days, he developed hypoxic respiratory failure. Repeat imaging revealed paravertebral abscesses, thrombosis of the adjacent inferior vena cava, and bilateral septic pulmonary emboli. He underwent surgical drainage of the paravertebral process.

On post-operative day four, examination revealed bilaterally injected conjunctiva. Ophthalmologic evaluation revealed bilateral endophthalmitis with vitreal abscesses. A vitreous sample taken from the left eye revealed no growth (on antibiotic therapy). Due to high suspicion for MRSA endophthalmitis, he received intravitreous clindamycin and vancomycin at doses of one milligram (mg) each of a 0.1 mL solution, followed by a change in systemic IV antibiotics to linezolid, 600 mg IV every 12

hours, and moxifloxacin, 400 mg IV every 24 hours. After several days, he clinically stabilized. After multiple interventions for retinal detachments over a period of months, his visual acuity improved to 20/20 in the right eye and 20/100 in the left.

Case 2. A 53-year-old female presented to an outside facility with shoulder pain. Joint aspiration was performed and cultures grew MRSA, resistant to penicillin, erythromycin, levofloxacin, and oxacillin without inducible clindamycin resistance. Blood cultures grew MRSA and intravenous antibiotics were initiated. She also had tricuspid valve endocarditis and septic emboli to bilateral lungs with positive MRSA sputum cultures. She developed decreased visual acuity and conjunctival injection. Ophthalmologic involvement was suspected. She was transferred to our facility for treatment.

On arrival, she was tachycardic, tachypneic, and mildly hypoxic. She had conjunctival erythema and edema of the right eye. Visual acuity was 20/50 and she was diagnosed with endophthalmitis per ophthalmologic examination. Laboratory studies revealed leukocytosis of 35.2 $x10^9$/L. Chest radiograph demonstrated extensive bilateral multilobar infiltrates. MRI of the right shoulder confirmed osteomyelitis of the acromion and proximal humerus.

During the hospitalization, she had repeat (negative) cultures of blood, acromioclavicular joint, and vitreous fluid from right eye (on antibiotic therapy). She was treated with intravenous vancomycin 15 mg/kg IV every 12 hours and gentamicin 80 mg IV every 8 hours, then with rifampin 600 mg IV twice daily. She also received intravitreal vancomycin 1 mg of 0.1 mL solution to the right eye. She developed a diffuse maculopapular rash on vancomycin and rifampin and daptomycin 6 mg/kg IV every 24 hours was substituted. Sepsis improved and she was discharged to complete six weeks of daptomycin therapy. Visual acuity improved to 20/20 at the time of discharge. Work-up for an underlying immunodeficiency was negative in both cases.

Discussion

Staphylococcus aureus causes a wide array of human infection from limited skin involvement to profound septic shock.[3] While invasive *S. aureus* infection was once limited to hospital-acquired strains, CA-MRSA has been emerging as a significant pathogen.[1] There are increasing reports of invasive CA-MRSA blood stream infections and death.

A clear delineation between community and health care-associated infections (HA-MRSA) is important to understanding the spectrum of CA-MRSA infections. This delineation is made possible by acceptance of more specific definitions and molecular testing. New definitions propose HA-MRSA should include patients with positive MRSA blood cultures greater than 48 hours into hospitalization, and those with home intravenous therapy, chemotherapy, home specialized nursing care, and hospital or hemodialysis clinic attendance within 30 days before *S. aureus* bacteremia as well as those hospitalized in an acute care setting for two or more days in the preceding 90 days or residence in a long-term care facility.[4,5] Genetic testing also has helped to identify specific virulent factors associated with CA-MRSA which may explain its recently increased propensity to cause invasive infection.

Exogenous endophthalmitis, including *S. aureus* endophthalmitis, has been described extensively in post-operative ophthalmologic surgery.[2,6,7] Endogenous endophthalmitis is much rarer, however, comprising only 5-10% of cases.[8] *S. aureus* represents a minority of these cases and,

until recently, differentiation between HA-MRSA and CA-MRSA was not made.

In a review of MRSA infections of the eye in an urban healthcare system, 3640 patients had a positive culture of MRSA and 70% were suspected CA-MRSA.[2] Of these, only four (8%) had endogenous endophthalmitis. Two of these patients would not have had CA-MRSA based on Friedman's definition.[5] A 2005 review of necrotizing fasciitis cases in a large US medical center identified one patient who had co-existing endopthalmitis related to MRSA bacteremia.[9] A 2006 review examined cases of MRSA culture positive patients at a US university and county hospital and found nine patients with the USA300 clone; three had endogenous endopthalmitis.[10] A retrospective study of treatment of endophthalmitis identified 14 cases of endogenous endophthalmitis over a four-year span.[11] Only one patient had *S. aureus*, however, the strain was not identified as MRSA. Another 7-year review of cases of endogenous endophthalmitis from a US university medical facility found 21 cases; five were *S. aureus* and two were MRSA. The strains were not identified further and based on information provided, only one remained as possible CA-MRSA.[8]

While *S. aureus* represents a small percentage of reported pathogens in cases of endogenous endophthalmitis, additional reports of MRSA and CA-MRSA infections are being identified and more may be missed. The true prevalence of MRSA endophthalmitis is unclear as availability of, and indications for, full ophthalmologic evaluation in MRSA bacteremia are unknown. It is unclear if specific toxic production or protein expression increases the likelihood of endophthalmitis. Colonization with MRSA also has not been found universally in subjects with endogenous endopthalmitis.[12] The aforementioned cases demonstrated need for vigilance in identifying endophthalmitis in patients with invasive MRSA infection. Reported cases, including our own, showed complications may be severe. Endogenous MRSA endophthalmitis can result in significant visual loss. In one series of 32 patients with MRSA endophthalmitis, only 36% of those with MRSA infections achieved visual acuity greater than 20/400 at three-month follow-up.[13] Retinal detachment is also common.[14] Goals should include early ophthalmologic evaluation and directed antibiotic treatment.

Standard therapy of MRSA endophthalmitis has not been defined. While the most clinical experience with MRSA endophthalmitis lies with intravitreal vancomycin, acceptable intravitreal concentrations have been demonstrated with systemic vancomycin, fluoroquinolones, daptomycin, and linezolid making these potential treatment options.[15,16] It is unclear if intravitreal antibiotics of these classes provide additional benefit to systemic therapy. Appropriate number and interval between intravitreal injections is undefined. Retinal toxicity also may limit antibiotic usage. Further studies are required.

Table 1. Reported cases of CA-MRSA endogenous endophthalmitis.

	Case	Age/ Sex	Proven CA-MRSA*	Suspected CA-MRSA**	Possible CA-MRSA***	IV Treatment	Vitreal Treatment	Visual Acuity
Our series	1	26/M		x		Vancomycin	Vancomycin/Clindamycin	20/100
	2	53/F		x		Vancomycin, then Daptomycin and Rifampin	Vancomycin	20/20
Blomquist[2]	1	40/M		x		Not Reported	Vancomycin/Ceftazidime	Not Reported
	2	43/M		x		Vancomycin /Gentamicin	Enucleation	Not Reported
Miller et al.[9]	1	45/M	x			Not Reported	Not Reported	Not Reported
Ruter et al.[10]	1	39/M	x			Vancomycin, Rifampin, Gentamicin	Vancomycin	20/40
	2	43/M	x			Vancomycin	Vancomycin	20/30
	3	39/M	x			Vancomycin	Vancomycin	20/30
	4	61/M	x			Vancomycin	Vancomycin, then Enucleation	No LP
Schiedler et al.[8]	1	75/F			x	Vancomycin	Vancomycin/Ceftazidime	20/25
Ho et al.[14]	1	66/M			x	Vancomycin	Vancomycin/Ceftazidime	20/150
	2	38/F			x	Vancomycin	Vitrectomy	HM 2 Feet
	3	74/M		x		Vancomycin	None	CF 2 Feet
	4	77/M			x	Vancomycin	Vancomycin/Ceftazidime	20/100
	5	49/F			x	Vancomycin	Vitrectomy	Enucleation
	6	18/M		x		Vancomycin	Vancomycin/Ceftazidime	Left - HM 2 Feet; Right - LP
	7	85/M			x	Vancomycin	Vancomycin/Ceftazidime	20/40
Leibovitch et al.[17]	1	37/F			x	Not Reported	Not Reported	No LP
Ness et al.[12]	1	F			x	Not Reported	Not Reported	Not Reported
	2	M			x	Not Reported	Not Reported	Not Reported

*Genetic testing performed. **No genetic testing available, however, absence of HA-MRSA risk factors, community-acquired infection known. ***Not enough provided information to exclude possibility of CA-MRSA.
LP = light perception, HM = hand motion, CF= count fingers.

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Keywords: *Staphylococcus aureus*, methicillin-resistant *Staphylococcus aureus*, community-acquired infections, endophthalmitis

The Silence of Neurosyphilis

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Introduction

Syphilis is a sexually transmitted infection. The infectious organism is a spirochete bacterium called *Treponema pallidum* subspecies *pallidum*. This organism also may transmit vertically in-utero from mother to fetus resulting in congenital syphilis. The following is a case report of tertiary syphilis in the form of tabes dorsalis. This case is of interest due to the rarity of late neurosyphilis in the immunocompetent host in the post-antibiotic era.[1]

Case Report

A 54-year-old African American male with no known medical problems presented to the emergency department during a snow storm, sitting and talking to himself in the waiting room. Dismissed by hospital security, he returned the next day and was evaluated. He denied the use of any medications except for acetaminophen. His social and family history was vague and non-contributory. He was afebrile, hemodynamically stable with laboratory results of leukocytosis and abnormal liver function tests. Computed tomography of the head without contrast (Figure 1) showed changes suggesting an acute stroke involving the left parietofrontal lobe with minimal extension into the posterolateral aspect of the temporal lobe. The patient was unable to provide a reliable history, appeared confused, and complained of dysgeusia, dizziness, blurred vision, headache, and right extremity weakness. He also complained of vague and poorly described lower extremity pain, dysuria, and orange-colored urine. He had nausea and vomiting for the previous three weeks.

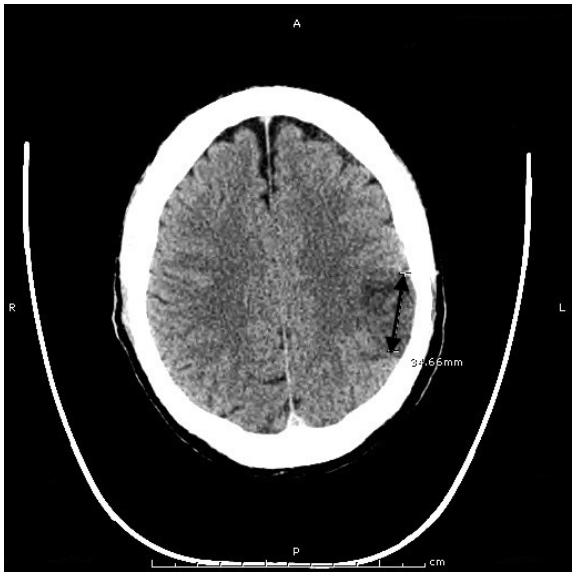

Figure 1. Abnormal geographic region of decreased attenuation in the left parietal frontal lobe. This may be reflective of an area of acute-to-subacute non-hemorrhagic infarct.

The patient was admitted to the hospital for a suspected stroke and further evaluation. The patient was alert with fluent speech but inappropriate word substitutions, neologisms, and moderate to severe

impairment of verbal comprehension. He manifested perseveration of verbal responses, anomia, dysgraphia, and he was unable to repeat sentences.

His pupils were equal, round, and reactive to light. Extraocular movements demonstrated an increase in horizontal nystagmus bilaterally and a few beats of vertical nystagmus with upward gaze. Visual fields were full and visual acuity was 20/100 O.S., 20/100 +1 O.D., without glasses using a pocket card. Facial sensation was normal with no facial weakness. Hearing for whispered numbers was normal. His tongue was at midline and no dysarthria was noted.

Strength in both upper and lower extremities and rapid alternating and fine motor movements were normal. He manifested mild postural tremor and intention tremor with finger-to-nose testing. The sensory examination was unreliable. Deep tendon reflexes were only trace in the upper extremities and at the knees but symmetrical. Plantar responses were equivocal bilaterally. His feet were swollen, firm, non-tender, and non-erythematous, with slight black discoloration of the pedal digits (Figure 2). The swelling extended above the ankles bilaterally.

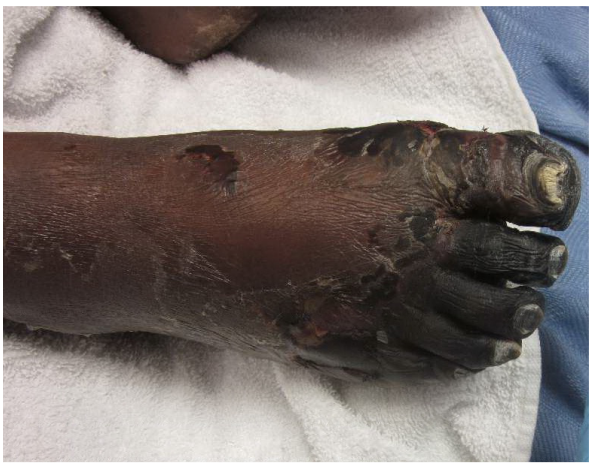

Figure 2. Gangrene of the right foot.

Laboratory results (Table 1) demonstrated an elevated white blood cell count, renal insufficiency, elevated creatine phosphokinase (CPK) consistent with acute rhabdomyolysis, and positive serology for syphilis. Emphysematous changes were noted on chest x-ray.

The patient developed a fever to 103.3^{o}F and became tachycardic and hypotensive, fulfilling Systemic Inflammatory Response Syndrome criteria.[2] The patient was transferred to the ICU and treated with IV fluids, vasopressors, and antibiotics after appropriate cultures were obtained. The CPK recovered with intravenous fluids and bicarbonate. An arterial ultrasound of the lower extremities showed absence of pedal pulses bilaterally. An orthopedic surgeon recommended pedal digit amputation secondary to wet gangrene as a complication of frost bite.

A magnetic resonance image (MRI) of the brain (Figure 3) displayed a subacute infarct in the distribution of the posterior division of the left middle cerebral artery. A magnetic resonance angiogram (MRA) showed mild distal carotid arterial atherosclerosis without other angiographic abnormality (Figure 4). Trans-esophageal echocardiogram and carotid ultrasound were unremarkable.

The patient was diagnosed with a stroke secondary to neurosyphilis-induced vasculitis and possible tabes dorsalis to explain the mostly painless nature of the wet gangrene of the toes. Therapy was initiated with penicillin G continuous infusion for three weeks with levofloxacin and metronidazole for the gangrene. The patient underwent bilateral pedal digit amputation with debridement and skin graft and was placed in a nursing home for physical rehabilitation. One-month after discharge, patient was readmitted for surgical debridement of his amputation. He had no improvement of his cognitive status.

Table 1. Laboratory results.

Complete Blood Count with Differential	
WBC	15.6 K/mcgL
Neutrophils	84%
Lymphocytes	7%
Monocytes	9%
Hemoglobin	14.3 g/dL
MCV	80.4 fL
Platelets	269,000 K/mcgL
Metabolic Panel	
Glucose	104 mg/dL
BUN	57 mg/dL
Creatinine	2.39 mg/dL
Calcium	8.6 mEq/L
Sodium	134 mEq/L
Potassium	3.8 mEq/
Chloride	98 mEq/L
CO2	22 mEq/L
Albumin	3.4 gm/dL
Total Bilirubin	1.8 mg/dL
ALK	88 U/L
ALT	182 U/L
AST	640 U/L
INR	1.3
AG	14

Specialty Tests	
Troponin	0.31 ng/mL
TSH	1.13 uIU/mL
CPK	CPK 26764U/L
Acetaminophen	<10 mcg/mL
Urine Drug Screen	Negative
ANA	Negative
Sexually Transmitted Infections	
HIV	Negative
Hepatitis Viral Panel	Negative
RPR	Reactive 1:64
Syphilis Ab IgG	Positive
Syphilis Ab IgM	Negative
Cerebrospinal Fluid	
Color	Clear
RBC	128 /mm3
WBC	2 /mm3
Neutrophils	32%
Lymphocytes	42%
Monocytes	26%
Gram stain	No organisms
Culture	No growth
VDRL	Positive 1:16
AFB	Negative
Protein	47 mg/dL
Glucose	66 mg/dL

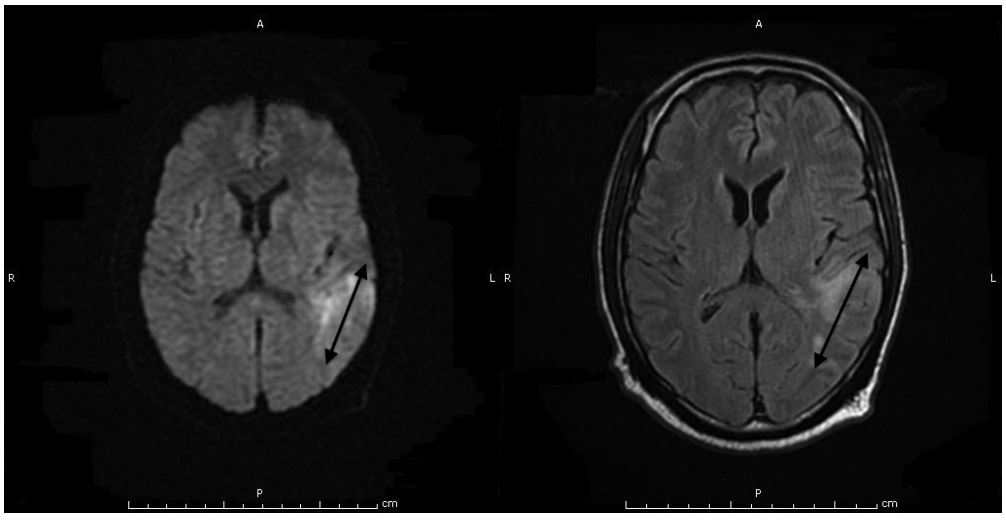

Figure 3. (Left) The T2 Flair demonstrates high signal in the left temporal parietal region and (Right) the diffusion study shows high signal on T2 Flair due to cytotoxic edema.

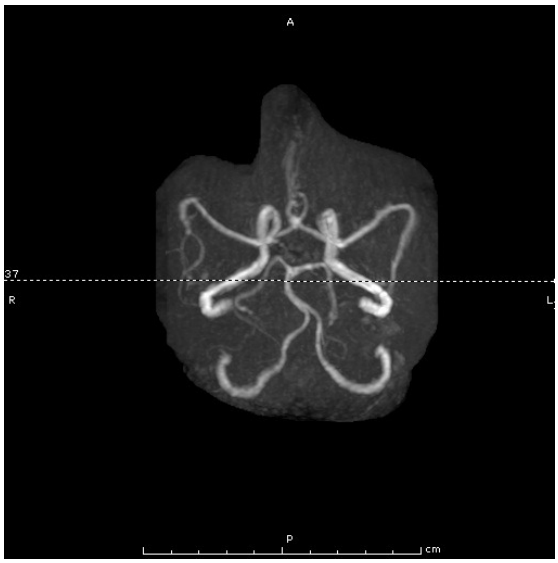

Figure 4. MRA showed no vascular occlusion in the posterior left middle cerebral artery distribution.

Discussion

Medical advancements during the 20th century have included antibiotics which have enabled physicians to provide potential cures for infections. *Treponema pallidum* subspecies *pallidum* is a bacterium under the spirochete family, taking its name from its microscopic appearance.[3,4] T. *pallidum* is a sexually transmitted infection that progresses through four stages: primary, secondary, tertiary, and latent. The former three stages usually occur within one year of inoculation. When transmitted in-utero, the infection is referred to as congenital syphilis.

Primary syphilis manifests after 2-3 weeks incubation after infection. A painless papule appears at the site which ruptures into a painless chancre ulcer with indurated margins and a non-exudative base.[3] Bilateral regional lymphadenopathy then develops. Patients tend not to address these lesions because they are painless and feel embarrassed to expose themselves. Chancres eventually heal within 3-6 weeks, while the spirochete disseminates freely.

Secondary syphilis may develop in 25% of those infected and usually is seen 3-6 months after the resolution of the chancre.[4] It most commonly presents as diffuse symmetric maculopapular lesions that occur non-sparingly over the body, including palms and soles.[5] There is also systemic constitutional symptoms and diffuse lymphadenopathy as well other skin, gastrointestinal, hepatic, musculoskeletal, renal, and ocular manifestations. It is during secondary syphilis where the cerebrospinal fluid is infiltrated.[3]

Tertiary syphilis, also known as late syphilis, includes neurosyphilis which presents with gummas, described as ulcers or an aggregation of granulomas that may occur anywhere in the body, from skin to bone.[6] Heart manifestations are usually dilatation and calcification of the aortic root and arch in addition to coronary artery narrowing with potential thrombosis.[7]

Neurosyphilis presents depending on the affected region of the nervous system.[8] Early neurosyphilis usually involves the cerebrospinal fluid (CSF), meninges, and vasculature and may either be asymptomatic or symptomatic. Late neurosyphilis usually affects the brain and spinal cord. Asymptomatic neurosyphilis may occur any time after infection but usually within

months and may be diagnosed if the CSF white blood count is more than 5 cells/μL or protein more than 45 mg/dL.

Symptomatic neurosyphilis usually occurs within the first year after infection and may present with typical meningitis-like symptoms, such as headache, nausea, vomiting, altered mental status or confusion, audiovisual impairments, and stiff neck.[8] Patients also may develop arteritis with small, medium, or large vessel disease potentially causing ischemia or infarction of brain and/or spinal parenchyma and gummas that may induce seizures.

Late neurosyphilis may present with general paresis and tabes dorsalis.[8] General paresis may have a normal neurologic examination but common abnormal findings include dysarthria, intention tremors of the face, tongue, and hands, as well as reflex abnormalities. Cerebrospinal fluid will show a reactive Venereal Disease Research Laboratory (VDRL) result of 25 to 75 cells/μL lymphocytes and 50 to 100 mg/dL protein. Atrophy is seen on neuroimaging.

Tabes dorsalis is a process that usually does not manifest until 20 years post infection and involves the dorsal columns and dorsal roots of the spinal cord.[8] Patients present with sensory abnormalities and/or lancinating pains affecting the face, back or limbs. Patients also may develop paresthesias, absent lower extremity reflexes, depressed vibratory and position sensation, attenuated touch and pain, and gastric crises which manifest as recurrent nausea, vomiting with severe epigastric pain. Argyll-Robertson pupils is one of the most common presentation in tabes dorsalis, and less so in general paresis. CSF may be normal or show 10 to 50 cells/μL lymphocytes and 45 to 75 mg/dL protein.

Our patient had a positive rapid plasma reagin with positive IgG and negative IgM syphilis antibodies representing active or recently treated syphilis. CSF findings also supported a diagnosis of late neurosyphilis. Clinically, the patient had a stroke secondary to a vascular occlusion to the left middle cerebral artery. The etiology of the vaso-occlusion is uncertain but most likely leans towards a vasculitis secondary to syphilis. The T. *pallidum* aggregate around any subarachnoid vessel, surrounding the brain or spinal cord, causes an influx of lymphocytes and plasma cells which infiltrate the arterial wall and perivascular tissue.[8] As the infectious inflammation progresses, the potential cascade of vasoconstriction complicated with vascular obliteration occurs after the release of pro-thrombotic reactants by the damaged vascular endothelium (Figure 3).

Our patient had peripheral neuropathy described as paresthesia, absent lower extremity reflexes with decreased sensation. His depressed level of sensorium did not allow him to recognize that he had suffered from frostbite and gangrene. He also complained of food tasting awful associated with nausea and vomiting. The constellation of symptoms manifested by our patient may fall under the umbrella of tabes dorsalis, amongst other possible diagnoses. A reliable history from this patient was difficult to ascertain, especially regarding how recently he had been infected with syphilis. Therefore, the possible diagnosis of tabes dorsalis cannot be ruled out.

Conclusion

Meningosyphilis is an uncommon cause of stroke in the post-antibiotic era. In untreated neurosyphilis, meningosyphilis is found in 10% of cases.[9] Its pathophysiology involves vasculitis of any vessel in the subarachnoid space with extension into the perivascular tissue resulting with ischemia and infarct. Diagnosis is made with a positive CSF-VDRL test and treatment is 18-24 million units of penicillin G infused daily for two weeks.[3]

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Keywords: neurosyphilis, tabes dorsalis, case report

Fish Oil Associated Myopathy

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Introduction

Myopathy affects 1-5% of patients prescribed statin medications[1] and less than 1% of patients prescribed fibric acid derivatives.[2] Fish oil has been used to alleviate statin-induced myopathy. No known cases of fish oil-induced myopathy have been reported.[3] Our case of fish oil-induced myopathy was confirmed by repeated symptomatology and elevation in creatine kinase with reintroduction of fish oil supplement for hypertriglyceridemia treatment.

Case Report

A 47-year-old male was referred to an endocrinology clinic for management of hypertriglyceridemia. He felt well and had no history of pancreatitis or xanthomata. His past medical history was significant for anxiety, depression, gastroesophageal reflux disease, and hypertension. Medications included alprazolam, sertraline, omeprazole, and amlodipine/benazepril.

Fasting measurement of serum lipids revealed a total cholesterol level of 195 mg/dL, triglyceride level of 722 mg/dl, and high-density lipoprotein level of 34 mg/dL. His low-density lipoprotein level was not calculated. Thyroid stimulating hormone was 1.28 uIU/mL (reference range 0.40-4.10). A two-hour 75 gram oral glucose tolerance test revealed a fasting serum glucose of 88 mg/dl and a two-hour value of 102 mg/dL. Serum electrolytes and a complete blood count were unremarkable.

The patient was placed on generic fish oil at a dose of 2 grams orally twice daily along with fenofibrate 145 mg orally daily. After two weeks, he reported muscle pain in his back and thighs bilaterally. The discomfort was not associated with increased physical activity. His serum creatinine kinase (CK) level was elevated at 340 U/L (reference range 32-237). Serum electrolytes were unremarkable. The fish oil and fenofibrate were discontinued. His muscle discomfort resolved.

Fish oil was reintroduced three weeks later at 2 grams orally twice daily. Fenofibrate was not restarted. Within two weeks, his muscle pain returned. Serum creatinine kinase level again was elevated at 268 U/L. A fasting lipid panel revealed a total cholesterol level of 259 mg/dL, triglycerides of 1180 mg/dL, and a high-density lipoprotein level of 34 mg/dL. The fish oil was discontinued, and his muscle symptoms resolved. Serum creatine kinase level one month later was normal at 159 U/L.

Discussion

The mechanism of myopathy associated with statins and fibric acid derivatives is poorly understood. Possible mechanisms for statins include reduced levels of ubiquinone,

reduced guanosine triphosphate (GTP)–binding regulatory proteins, or altered phospholipid metabolism.[1] Muscle toxicity has been associated with increased systemic or tissue statin levels, affected by gene mutations[4], drug dose, co-administration of gemfibrozil, and grapefruit[1].

The proposed mechanisms of fibrate-associated myopathy are even less clear, but also appear to involve genetic predisposition, drug interactions, and medication dose.[2] The etiologies of muscle damage with statins and fibrates are unlikely to be present with fish oil, whose proposed mechanisms of action differ significantly from either statins or fibrates.[5] The presence of diabetes, renal failure, and hypothyroidism can contribute to myopathy, but none were present in this patient.

This case demonstrated true myopathy with muscle pain and an elevation in CK associated with administration of fish oil. The initial symptoms resolved with discontinuation of fish oil and fenofibrate, but muscle pain and an elevated CK returned upon re-introduction of fish oil alone. Both resolved within one month of discontinuing the fish oil. A contributing factor may have been the type of fish oil used, as it was marketed as a nutritional supplement, not as a pharmaceutical-grade product.

Conclusion

Fish oil can be associated with myopathy either alone or in combination with fenofibrate. If signs and symptoms of myopathy do not resolve with discontinuation of other antihyperlipidemic agents, discontinuation of fish oil should be considered.

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Keywords: fish oils, myopathy, case report

Helicobacter Pylori

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Introduction

Helicobacter pylori, a gram-negative bacterium found on the luminal surface of the gastric epithelium, was first isolated by Warren and Marshall in 1983.[1] Infection with *Helicobacter pylori* has been recognized as a public health problem worldwide.[2] The prevalence of peptic ulcers in patients seropositive for *H. pylori* is seven times greater than in those who are seronegative.[3]

Before the discovery of *H. pylori*, gastroduodenal ulcer healing was achieved with the administration of H2-blockers or proton pump inhibitors (PPIs) for at least four weeks.[4] At present, *H. pylori* eradication therapy is indicated in gastroduodenal ulcer disease. Recent international consensus statements have concluded that *H. pylori* is a causal factor in peptic ulcer disease and a Group 1 carcinogen in humans and all patients with peptic ulcer associated with *H. pylori* infection should receive eradication therapy.[5]

The treatment of *Helicobacter pylori* remains a challenging clinical problem despite extensive research over the last 25 years. PPI-based triple therapy, with a proton pump inhibitor, clarithromycin (CAM), and either amoxicillin (AMPC) or metronidazole, is a widely-recommended eradication therapy.[6] Prevalence of *H. pylori* resistance to metronidazole is approximately 25%.[7] PPI-based triple therapies have shown efficacy in various clinical trials from different geographic areas.[8] Triple therapy using a PPI with clarithromycin and amoxicillin or metronidazole given twice daily remains the recommended first choice treatment.

Diagnosis

Endoscopy. Endoscopy is performed at baseline, upon completion of ulcer treatment, and one month after completion of ulcer treatment to confirm the state of the ulcer.

Histologic examination. All histologic examinations for the diagnosis of *H. pylori* infection should be carried out at baseline and one month after the completion of the ulcer treatment. Biopsies should be obtained from the two sites of the greater curvature of the antrum and the greater curvature of the upper corpus. The biopsies should be fixed in formalin and slides prepared with hematoxylin-eosin and Giemsa stains. The bacterial density should be categorized as none, mild, moderate, marked, or judgment impossible.

Gastric Ulcer

The inflammation of the gastric mucosa induced by the infection is most pronounced in the non-acid-secreting antral region of the stomach and stimulates the increased release of gastrin.[9] The increased gastrin levels in turn stimulate excess acid secretion from the more proximal acid-secreting fundic mucosa, which is relatively free of inflammation.[10] The increased duodenal acid load damages the duodenal mucosa, causing ulceration and gastric metaplasia. The metaplastic mucosa then can become

colonized by *H. pylori,* which may contribute to the ulcerative process.

Eradication of the infection provides a long-term cure of duodenal ulcers in more than 80% of patients whose ulcers are not associated with the use of nonsteroidal antiinflammatory drugs (NSAID).[11] NSAIDs are the main cause of *H. pylori*-negative ulcers. Ulceration of the gastric mucosa is believed to be due to the damage to the mucosa caused by *H. pylori*. As with duodenal ulcers, eradicating the infection usually cures the disease, provided that the gastric ulcer is not due to NSAIDs.[12]

H. pylori Treatment

Various drug regimens are used to treat H. pylori infection. Most include two antibiotics plus a proton-pump inhibitor or a bismuth preparation (or both). The most commonly used initial treatment is triple therapy consisting of a proton-pump inhibitor plus clarithromycin and amoxicillin, each given twice per day for 7 to 14 days. Metronidazole is used in place of amoxicillin in patients with a penicillin allergy.

First-line treatment. Triple eradication therapy is the most commonly used treatment protocol in *H. pylori* eradication. Eradication of *H. pylori* removes the increased risk of developing actual ulcer disease.[13] General agreement exists in that eradication of H. pylori infection with triple therapy including a PPI and two antibiotics for 7–10 days is the gold standard of treatment.[14] In the recent triple combination studies, the eradication success declines over time.[15] The preferred regimen internationally is triple therapy with a PPI, clarithromycin, and amoxicillin twice daily for 7-10 days.[16]

Second-line treatment. Second-line treatment includes bismuth, metronidazole, and tetracycline plus either a PPI or an H2 receptor antagonist (H2RA).[17] If a PPI is chosen, the regimen can be given for seven days. If an H2RA is used, however, 14 days are recommended. A recent meta-analysis of 93 studies showed a higher rate of eradication with quadruple therapy that included both clarithromycin and metronidazole than with triple therapy that included both these agents in populations with either clarithromycin or metronidazole resistance**.** [18]

Discussion

Helicobacter pylori is an important cause of duodenal and gastric ulcers. Greater than 90% of duodenal ulcers and 70% of gastric ulcers are associated with *H. pylori*.[1] Eradication of *H. pylori* is effective in healing ulcers and drastically reducing the ulcer recurrence, eliminating the need for maintenance therapy.[19] Treiber et al.[20] found that successful *H. pylori* eradication induced a better response in peptic ulcer healing, regardless of diagnosis of duodenal or gastric ulcer. Several large-scale clinical trials and meta-analyses have demonstrated that the most common first-line therapies fail in up to 20% of patients.[21]

Currently-recommended protocols include a 10-14 day treatment with: (1) a proton pump inhibitor (PPI) plus clarithromycin and amoxicillin, (2) a PPI plus clarithromycin and metronidazole, or (3) bismuth subsalicylate plus metronidazole and tetracycline.[22] The recommended duration of triple therapy is typically 10 to 14 days in the United States and 7 days in Europe.[23] Triple therapy with a proton pump inhibitor, clarithromycin, and amoxicillin or metronidazole remains an appropriate first-line therapy.

Another possible initial therapy in areas with a high prevalence of clarithromycin-resistant *H. pylori* infection (i.e., >20%) is quadruple therapy comprising the use of a proton-pump inhibitor, tetracycline, metronidazole, and a bismuth salt for 10 to 14 days.[24]

The choice of second-line treatment is influenced by the initial treatment. Treatment failure often is related to *H. pylori* resistance to clarithromycin or metronidazole (or both agents). Clarithromycin should be avoided as part of second-line therapy unless resistance testing confirms that the *H. pylori* strain is susceptible to the drug.[25] If initial therapy did not include a bismuth salt, bismuth-based quadruple therapy commonly is used as second-line therapy with eradication rates ranging from 57 to 95%.[26-29] Quadruple therapies, therefore, usually are reserved for patients who have failed one or more courses of triple therapy.[30] Some quadruple therapies are less costly and appropriate for patients in whom cost is a significant factor.

Conclusions

Helicobacter pylori is a very common disease with a broad spectrum of clinical symptoms and disorders. A PPI, clarithromycin, and amoxicillin or metronidazole remains an appropriate first-line therapy, provided that there is not a high local rate of clarithromycin resistance. A PPI used in combination with metronidazole and either amoxicillin or tetracycline is recommended in patients previously treated with a PPI, amoxicillin, and clarithromycin. Eradication of *H. pylori* infection has the potential to reduce the risk of gastric cancer development.

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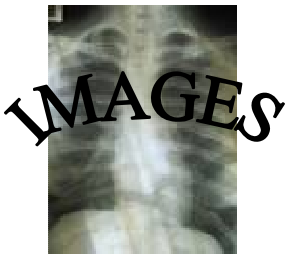
Keywords: Helicobacter pylori, gastroduodenal ulcers, review



Lung Abscess

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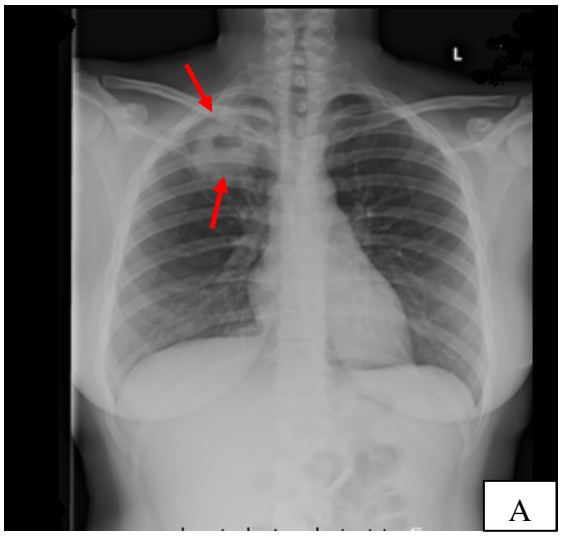
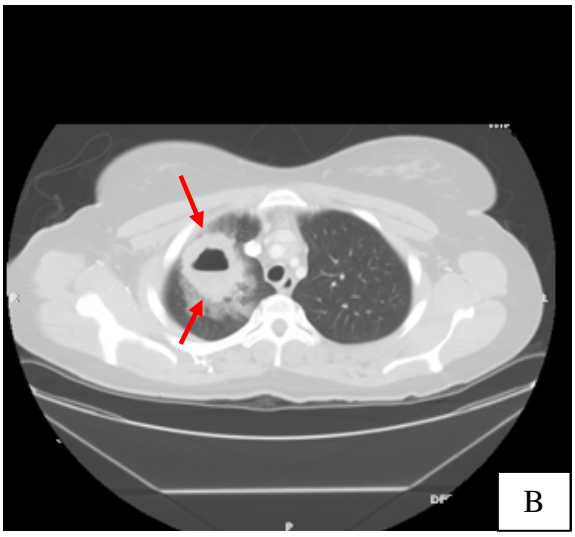


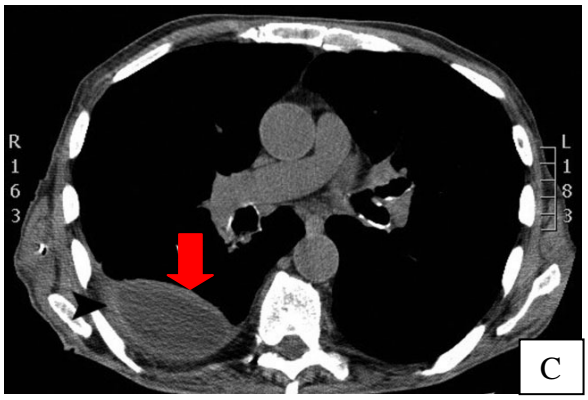


A 23-year-old female presented with a two-week history of worsening shortness of air, right-sided chest heaviness, and cough. The cough was initially dry, but later productive with foul smelling yellowish sputum. The patient denied fever, history of tuberculosis contact, or recent travel. Her physical examination showed stable vital signs and right upper lobe crackles. A complete blood count and comprehensive metabolic panel were normal. Blood and sputum cultures and a QuantiFERON®-TB Gold for tuberculosis were negative. There were no HIV antibodies. Chest radiograph showed a 6-cm cavitary lesion in right upper lobe (see arrows in Figure A). A computer tomography (CT) of the chest showed 4.9 x 5.4 cm necrotic lesion with thin and smooth walls in right upper lobe (see arrows in Figure B). A diagnosis of lung abscess was made and IV clindamycin was started. Acid-fast bacillus smear, fungal, bacterial cultures, and cytology on bronchoalveolar lavage and bronchial brushing were negative. The patient responded well to antibiotic treatment and later was switched to oral clindamycin. She was discharged receiving a total of six weeks of antibiotics.

Discussion

Lung abscess is defined as a necrotic lesion in lung parenchyma that contains pus.[1] Although the necrotizing infections are the most important cause, other important pathogenetic factors include cavitations of the necrotic center of malignant tissue or necrosis of infracted lung tissue. Differentiating lung abscess from empyema on imaging can be challenging. In empyema, the CT of the chest typically shows thickened parietal and visceral pleura separated by fluid, known as the "split pleura sign" (Figure C).[2] Fluid usually is organized in oblong fashion which

displaces surrounding lung tissue away from fluid collection. The usual CT findings of a lung abscess include a spherical cavity with a thick wall that destroys the surrounding lung with bronchograms traveling into the abscess rather than displaced like in empyema.[2-6] Color Doppler ultrasound also has shown to be a powerful tool to differentiate air fluid level in lung abscess from empyema.[7]



A CT image demonstrating thickening and enhancement of the parietal pleura and visceral pleura with fluid in between (i.e., the ''split pleura sign'').[2]

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Key Words: lung abscess, empyema

Commentary

Six Patients an Hour

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This morning I went to a doctor's office as a patient. No great problem, the nurse practitioner was instructing me to lose weight. The building was where I had worked until a stroke forced me to retire ten years ago. As I sat in the waiting room, I heard a physician being recruited to the practice say that no doctor could see six patients an hour. The receptionist had worked for me for years and she smiled as she said "You did, and did a good job at it." As I thought back, I agreed "Yes, I did."

As a fourth-year student, I had spent my precepteeship in Oakley with Jim Marchbanks, MD, the only physician in town. I spent two weeks in Oakley after I graduated so Dr. Marchbanks could have a vacation with his family. I served an internship in Wesley Hospital, now Wesley Medical Center, in Wichita. After completing that year, I moved to Oakley to join Jim in practice. There was no family practice residency at that time. Jim was an excellent physician and teacher; I was certain I could learn from him while earning a living. In 1963, he paid me $1000 a month. I was wealthy! Six months later, he told me I was seeing enough patients that he could pay me $1200 monthly.

After eight months, Jim and I were as busy as we wanted; Jim actually was busier than he cared. Then, Jim died unexpectedly. I was now the only physician in a community of about 3000. How was I to handle the practice we had built? I just worked hard!

The physicians I had tried to emulate at the University of Kansas Medical Center and Wesley Hospital worked hard. Those in Wichita made morning hospital rounds, spent the day in their offices, then made evening hospital rounds. However, there were two or three physicians sharing their practices. They shared evenings and weekends off.

I knew I could work hard, although there would be no one to share time off. Jim and I used to arrive at the hospital about 8:00 am. If we had surgery, we arrived at 7:30 or a bit earlier. By 9:00 am, we had completed rounds and had coffee with the hospital administrator, the head nurse, and anyone else who wandered in. We planned to be at the office by 9:30. Actually, we were already behind by coffee time. We usually got to our office about 10:00 am. We scheduled our last morning patients at 11:30, hoping to be finished by 12:00. Since we started late, we finished late. We often left the office at 12:30 or 1:00.

We had lunch and time for a short nap. We scheduled afternoon patients from 2:30 to 5:00 pm. We allotted fifteen minutes per visit, thirty minutes for a complete physical exam. We never refused walk-ins and rarely left the office before 5:30 or 6:00 pm, then making evening rounds at the hospital. I was happy to be home by 6:30 pm.

After Jim's death, I started my day at 7:30 am. I was not a surgeon, so that was not much different. Morning rounds and fifteen minutes for coffee at the hospital got me to my office by 9:00 and allotted fifteen minutes per routine visit. After appointments were completed, I had walk-ins and morning phone calls to return. I planned on having a half hour for lunch, a half hour nap, and returned to the office at 2:00 pm. In the afternoon, I scheduled a visit every ten minutes. By this time, I had taken care of my patients/friends for years so I rarely had to look at

an old chart to know what it contained. By the end of the afternoon, I had most of my charting left to do after the appointments were completed (my charts were quite a bit shorter then), then make phone calls and evening rounds. I usually got home by 6:30 or 7:00 pm.

Deliveries and other emergencies interrupted my office hours, evenings, and weekends, but this was to be expected. The physicians I knew in Wichita and the other doctors in Western Kansas all had similar schedules. It was my practice and I loved it. It was my office, my employees, and my schedule. And they were my patients. I knew them, took care of them, and cried as they died. I felt that I should always be willing to come when summoned. My wife was not as happy about this as I was, but she had grown up with a hardworking father and had seen other doctors' families' live as we did. She built her own life around my practice.

I often had a medical or a physician assistant student with me. That freed me for a bit of time to do my charting while a student saw a new patient. The hospital administrator hired a locums tenens physician to cover the community one weekend a month so I could be off call from Friday evening to Sunday evening. He hired a physician for a week or so to give me a summer vacation. I hired a physician assistant for six years. I had a partner for five years, then he left. I was again alone.

In 1972, we formed a Rural Healthcare Clinic (RHC). One of the requirements was to have a mid-level practitioner so from 1972 until my stroke in 2001, I worked with at least one nurse practitioner. From 1994 to 1998, another physician joined our RHC. Even after he left, two nurse practitioners worked with me.

For about ten years, I worked alone with only an occasional student in Oakley. I think I provided better than average care for my patients. Many of the students asked me if he or she had to work as hard as I did. My response was "No. I had planned on practicing with a partner. He died far too soon. I was thrown into a community where I was the only physician. I was unable to recruit a physician to remain in Oakley. While I had not planned on my solo practice, I always loved it. But you certainly don't have to live my life!"

Thus I practiced, and we lived, in Oakley for years. Our three children grew up, went to colleges, and left Oakley. I continued to practice until I fell on the ice and sustained a head injury and stroke forcing me to retire. But I would yet be practicing in Oakley if possible.