West Virginia Medical JOURNAL

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West Virginia State Medical Association

The Voice of Medicine in West Virginia

WVU Healthcare welcomes Multiple Sclerosis specialist to our neurology team

WVU Healthcare is proud to introduce our newest neurology specialist, Javier Gonzalez, MD. In addition to his interests in brain and spinal tumors, Dr. Gonzalez specializes in the treatment of auto-immune disorders such as Multiple sclerosis.

WVU Healthcare offers a multi-disciplinary team of experts including specialists in neuro-ophthalmology, neuro-psychology, and neuro-oncology.

For your patient's convenience, our facility offers many on-site services that can be scheduled during one location visit, including advanced imaging, infusion, and rehabilitation. We welcome the opportunity to partner with you in the treatment of your patients with auto-immune disorders.

Javier Gonzalez, MD, earned his medical degree at Our Lady of the Rosary University School of Medicine. He completed a neuro-oncology fellowship at the University of Texas MD Anderson Cancer Center and a neurology residency at the University of Texas Medical Branch.

Dr. Gonzalez is board certified in neurology and is a member of the American Academy of Neurology, the Society for Neuro-oncology, and the American Society of Clinical Oncology.

WVU Healthcare

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West Virginia
e-Directive Registry

Call with questions: 877-209-8086, or visit www.wvendoflife.org.

2013 Healthcare Summit

The Greenbrier Resort
White Sulphur Springs, WV

August 23-25

You are invited to attend the 2013 WVSMA Healthcare Summit, August 23-25 at The Greenbrier. For your convenience, you can register for the Healthcare Summit through our safe, secure website at www.wvsma.org. A receipt will be e-mailed to you immediately following payment. If you wish to register by fax, fill-in the registration form on page 37 and fax it to 304-925-0345.

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Friday Evening Inaugural Celebration and Gala Dinner Dance

Join us Friday evening for dinner and dancing along with the installation of 2013-2014 WVSMA President Reginald McClung, MD and WVSMA Officers. The event is black-tie encouraged and reservation required. Be sure to sign-up for this special event when you register for the 2013 WVSMA Healthcare Summit. Individual tickets are $150 and couples tickets are $250. The event will begin at 7:00 pm immediately following the Friday evening Reception hosted by West Virginia University School of Medicine and Joan C. Edwards School of Medicine at Marshall University.

Lodging Reservations

Make your lodging reservations today! Call The Greenbrier directly at 1-877-394-4137. Make sure to tell them you are attending the WVSMA/Foundation Healthcare Summit to receive our special discounted room rate of $269 per night for a standard or intermediate room. Rooms do fill-up quickly, so we encourage you to make your reservations now.

For additional details on this year's program, please visit our we bsite at www.wvsma.org. If you have any questions please feel free to contact Karie Sharp, WVSMA Conference Coordinator at (304) 925-0342 ext. 12 or karie@wvsma.org

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About the cover: *Field of Red—Tundra-like windswept open meadows in the Bear Rocks Preserve of Dolly Sods Wilderness include subalpine heath barrens of blueberry, cranberry, huckleberry, rose azalea and rosebay rhododendron. Fall weather brightens these regions to glowing red fields of color scattered between unique formations of white sandstone and quartz rocks.*

Scientific Articles

Upcoming Events

August 23-25, 2013
Annual Healthcare Summit
The Greenbrier
White Sulphur Springs, WV

September 20-21, 2013
Appalachian Addiction & Prescription Drug Abuse Conference: A Paradigm for the Epidemic
Embassy Suites, Charleston, WV

FEATURES

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President's Message

2013 WVSMA Legislative Wrap-up

by Hoyt J. Burdick, MD
WVSMA President
2012-2013

The 2013 Regular Session of the West Virginia Legislature concluded Thursday, April 18. Over two hundred bills passed both the House of Delegates and State Senate and are now in the hands of Governor Tomblin for his review and signature. Health care is always among the list of most considered legislation and I am proud to report that your WVSMA was at the Capitol each and every day throughout the 60 day session advocating on your behalf!

This year, like every year, about two thousand bills are actually introduced and each must be carefully reviewed and analyzed. History reminds us that good bills do not just pass on their own and bad bills are not automatically rejected. Every bill, good or bad, has a constituency pushing for its passage. What happens under the dome in Charleston matters and impacts almost every aspect of our profession. Be assured, your membership investment in the WVSMA is working for you.

The WVSMA was well represented at the Capitol this year. Brett Tubbs and Sue Baek, Esq., were an effective team representing the interests of physicians and the patients we serve throughout the legislative session. From bill introductions, committee action to final votes on the House and Senate floor, Brett and Sue ensured the voice of medicine was heard.

The WVSMA also worked in close collaboration with other 'like-minded' advocates such as the Academy of Family Physicians, Academy of Ophthalmology and the Hospital Association to build strong coalitions in support of pro-medicine measures and to oppose bills that would harm the doctor patient relationship and jeopardize access to quality health care in our State.

The WVSMA legislative committee under the leadership of past-president John Schmidt, MD, met every two weeks throughout the legislative session to provide our advocacy team with direct physician input on the bills being discussed. We also significantly increased our efforts to communicate with you, our members, by emailing out each week a comprehensive report on the happenings at the Capitol.

Here is a brief summary of several bills we were particularly engaged in this year.

Bills that passed

SB 21 – Creating Health Care Provider Transparency Act – promotes the use of identification badges by health care workers when providing direct patient care. The WVSMA advocated for this bill because of the need to increase the clarity and transparency of the qualifications of all health care providers who are caring for patients. The legislation directs the Secretary of the Department of Health and Human Resources to promulgate rules on the design and content of the ID badges. The effective date of when the new badges will be required is July 1, 2015.

SB 101 - Clarifying MPLA applies to nursing homes and their health care providers – a decade ago, physicians and hospitals led the charge to reform our State's broken judicial system by successfully pushing for the enactment of strong legal reforms in the legislature. While nursing homes were specifically mentioned in those important tort reform measures, recent lawsuits have attacked the reforms' application to the nursing home environment. SB 101 was supported by the WVSMA and helps ensure, moving forward, that nursing homes receive the full benefit of our hard fought legal reforms.

SB 265 – Authorizing DHHR promulgate legislative rules – outlines the definition and framework of the licensed pain clinic facilities act, a major section of last year's comprehensive controlled substance bill approved by the legislature. Most of the attention of the bill this year was on defining the threshold at which a medical practice would meet the 'pain clinic' definition and be subject to stringent licensure and regulatory requirements. SB 265 also contained the policies to implement the new mandatory 3 hour CME requirement on best practice opioid prescribing.

SB 108 – Creating Unintentional Pharmaceutical Drug Overdose Fatality Review Team – current state law provides for three fatality

review teams--child fatality, domestic violence and maternal mortality. SB 108 restructures the current teams under the bureau for public health and adds a new, additional focus on unintentional pharmaceutical drug overdoses. All teams will be multidisciplinary in their member composition and directed to oversee and coordinate the examination, review and assessment of fatalities in these target populations.

SB 464 – Regulating tanning facilities – imposes new regulation and requirements on commercial tanning facilities and sets age limits on people that are allowed to use a tanning devise at these facilities. No person under the age of 14 will be allowed to use a tanning device while those between the ages of 14 and prior to turning 18 can only tan with written parental consent. SB 464 does provide a specific exemption to the new law for any health care provider performing any action within the scope of his or her practice. The law grants local health departments the authority to enter and inspect a tanning facility in order to determine compliance.

HB 2108 – Making the offense of failure to wear safety belts a primary offense – moves from 'secondary offense' to 'primary offense' the failure to wear a seat belt when driving. The current 'secondary offense' law only permits law enforcement to cite a driver for failure to wear a seat belt if the driver has been stopped for some other reason. Making seat belt use a 'primary offense' allows law enforcement to stop a driver solely for failing to wear a seat belt. Studies show our State's seat belt use currently stands at about eighty present. Seat belt laws in most states already make it a 'primary offense' and statistics show our new 'primary offense' requirement will likely increase seat belt usage by another five to ten percent and save lives.

SB 336 – Relating to interscholastic athletics concussions and head injuries – establishes in state law a nationally supported evaluation and return-to-play protocol for all athletes suspected of suffering a concussion during an athletic contest or practice. The original bill also included provisions to clarify the important liability protections under current code for volunteer team physicians. The liability reforms, unfortunately were dropped in the final bill.

SB 22 – Requiring maternity services coverage for all health insurance plan dependents in certain circumstances – mandates that PEIA, the state's health insurance program, must include maternity coverage for the dependents of PEIA covered workers. This brings PEIA on par with the current coverage requirements on commercial insurance carriers.

HB 2729 – Allowing schools to voluntarily maintain and use epinephrine auto-injectors – enables properly trained school personnel to have readily available Epipens for use in a medical emergency.

HB 2513 – Improving enforcement of drugged driving – adds to current state law that when operating a motor vehicle, the driver is giving implied consent to being tested for drugs just as suspected impaired drivers can have their blood alcohol content tested. The bill requires new training for law enforcement officers and limits testing to a specific list of controlled substances.

Bills that did not pass

HB – 2689 Authorizing miscellaneous Boards and Agencies to promulgate legislative rules – contained the rules submitted by the West Virginia Board of Optometry to activate their broader scope of practice approved by the legislature two years ago. The rules were amended early in the legislative process to make it clear that any future scope of practice expansion efforts would first have to go through an independent analysis and the rule making review process. Optometrists opposed these provisions and the Board of Optometry, unsuccessful in getting the amendments dropped, decided to withdraw the rule in its entirety.

SB 27 – Relating to the administration of opioid antidote in emergency situations – would have given first responders access to opioid antagonist such as Naloxone in an effort to reduce deaths due to opioid overdose. The WVSMA supported this legislation because studies clearly show increased availability and use of this opioid antagonist by properly trained first responders saves lives. The bill also specified that data on opioid overdoses be collected and reported to the Legislative Oversight Commission on Health and Human Resources Accountability.

HB 2457 – Relating to health care records – was an effort by the trial lawyers to reduce or eliminate what health care providers are allowed to charge by law for copying medical records. The WVSMA and Hospital Association worked together in opposition to this bill and were successful in seeing that it did not pass.

SB 10 – Permitting independent initiation of disciplinary proceedings by certain licensing boards and SB 11 – Relating to WV schedules of controlled substances – both bills were part of the continuing effort to address our abuse, misuse and diversion of controlled substances. SB 10 would have streamlined the process of initiating an investigation by professional licensing boards based on information compiled by the newly established review committee under the West Virginia Board of Pharmacy. SB 11 would place dosage and refill restrictions on Schedule III Hydrocodone controlled substances.

SB 201 – Permitting expedited partner therapy – a priority legislative initiative of the WV Perinatal Partnership and supported by the WVSMA, this bill would have allowed a physician to give a prescription to a patient with a diagnosed STD to be given to the patient's partner without having to have a direct physician/patient relationship with the partner. The bill also had strong liability protections for the prescriber.

Visit www.wvsma.org for a complete legislative wrap-up report detailing the health care bills of the 2013 Legislative Session.

West Virginia Physicians at the Boston Marathon
Their experience...


Dr. Shamma


Dr. Deer


Dr. Cucuzzella


Dr. Kim


Dr. Dundervill

Courtesy of the WV Daily News & Register-Herald

Dr. Zainab Shamma-Othman, a pulmonologist in Lewisburg, was among the runners in Monday's Boston Marathon and was nearing the finish line when the explosions occurred.

Shamma-Othman was not among the injured, according to her husband, Beckley neurologist Dr. Joe O. Othman.

"She said she was just five minutes from the finishing line (when the explosions occurred)," Othman said. "She had to stop. She didn't know what was going on."

Othman said he only had one brief telephone conversation with his wife in the hours following the race's tragic conclusion.

"She got to the hotel two hours after the incident," Othman said, noting he had been unable to contact Shamma-Othman in the immediate aftermath of the explosions, but their daughter had made a connection with her mother via text message.

WVVA spoke with Boston Marathon runner Dr. Zainab Shamma.

Dr. Shamma said she could see the police presence as she approached the finish line, which was blocked off when she approached the area where the bombings took place.

Rumors circulated, and she described the confusion she and the other runners were feeling as they tried to make sense of what had happened.

She also mentioned difficulty getting back to her hotel because of heightened security in the area.

Courtesy of WCHS Radio

Charleston medical doctor Tim Deer says he went to Massachusetts General Hospital Monday to see if he could help in the minutes after the two explosions at the Boston Marathon.

Deer, who had earlier completed the race, says he and about nine other doctors showed up to lend a helping hand but weren't needed.

"At Mass General they were very well-staffed and I was impressed by their ability," Deer, whose primary training is as an anesthesiologist, said. "They were able to get people to Mass General pretty quickly, it's only about a mile away."

Deer told his story on Charleston radio station V100 Tuesday morning.

"It's a great event and it was a wonderful day and then this happens. I really just encourage everybody to pray for the victims and their families," he said.

Deer and two friends, Chris Kim and Bob Dunderville, were taking pictures near the finish line when the explosions took place Monday afternoon.

"It was chaotic," Dr. Deer said. "There were people running. A lot of emotions. No one really knew what happened, terrorism, a gas line, or what it was."

Deer says he was evacuated from the area and then a short time later went to the hospital. He says surgeries were already taking place and some of the injured were in ICU.

Dr. Deer has completed more than a half-dozen Boston Marathons and plans on going back next year.

"The people of Boston are great people. They do a wonderful job and I don't think we can let something like this change who we are as Americans," Deer said.

Courtesy of the Charleston Gazette

Forty-six runners from West Virginia entered the race, according to the marathon's website, http://www.baa.org.

Runners from Charleston were Timothy Deer, Robert F. Dundervill III, Christopher Kim and Nathaniel Orders. Munsey said they were not injured.

Dundervill, a Charleston ophthalmologist, was walking to his hotel room after finishing the race, according to his wife, Lisa.

"When he heard the explosion, he called me to see if I was near a computer to look up and see what was going on," she said. "I heard it from him so I knew immediately he was OK."

Courtesy of West Virginia Broadcasting

Jefferson County physician Dr. Mark Cucuzzella, an avid runner and the founder of Freedom's Run, an annual marathon that takes place in the Eastern Panhandle, was among 46 West Virginians entered in the Boston Marathon when bombs exploded at the finish line Monday afternoon.

This is Cucuzzella's twentieth Boston Marathon. He said about 4 ½ hours into the race two bombs went off.

"It became pretty chaotic; people were trying to find their way, find their families, going in and out of hotels," Cucuzzella said. "Hundreds of police and ambulances emerged on the scene, SWAT teams, dogs, Humvees, more stuff than you could imagine showed up within 15 minutes to the finish line area."

Cucuzzella completed the race in 2 hours 43 minutes and had returned to his hotel about two blocks away from the finish line when the bombs exploded. He ran the race with about 100 members of the American Medical Athletic Association.

Cucuzzella said the race and all mass transportation was stopped leaving a lot of participants out on the 26 mile course and forced to find their way back to the city.

"And people could be three, four, five miles out from the finish wondering what's going on," Cucuzzella said. "And a lot of kindness of strangers, just a few people I've spoken to here in the last 20 minutes were taken in by homes, taken in by businesses along the route because they're out there running."

"It was about 45 degrees and kind of cold and you stop running and are instructed to just find your way back to your hotel," Cucuzzella said. "One gentleman walked about six miles back to the hotel with his t-shirt and got back and he was looking pretty beat up and cold and shivering and I'm sure that was harder than the marathon itself."

Cucuzzella said it's sad that something like this happened at a healthy, outdoor event like the Boston Marathon that brings people from around the world to visit America.

"Words just can't really describe, it's not just myself, pretty much everyone here in the city right now, it's a sense of shock," he said.

Many thanks to our physicians who kindly sent their photos and comments.

This is our group taking photos about 1 minute before the bombing. From left to right. (Bob Dunderville 3:37. Chris Kim. 3:32. Tim Deer 3:08). We were about 100 yards away **on Boylston street. The first bomb went off and we saw the** plume of smoke and then the second one which shook the ground. We were standing and hundreds of people started running towards us. We were surrounded by the marathon busses and Initially they made us clear the area. We did not realize the degree of horror until we got to our hotel 4 blocks away. I went to Massachusetts General hospital to try to help, but the trauma team was in full swing and fully staffed. I walked back to the hotel and the streets were empty. It was a surreal and numbing moment. The city remained on high alert and everyone was asked not to leave their hotel.--*Dr. Tim Deer*

Dr. Zainab Shamma-Othman before the race.

Drs. Deer & Kim at the **finish line.**

Risk Factors Predicting Fractures in Early Postmenopausal Women

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Abstract

Few studies exist evaluating fracture prediction in women aged 50-59. Clinical risk factors are important determinants for fracture prediction in younger postmenopausal women since most fractures occur outside the range of an osteoporotic bone mineral density. Although fracture incidence rates in this age group are about one-half of those aged 60-69, considerable costs and loss of quality-adjusted life years are still incurred in this age group. We sought to determine what clinical risk factors would predict subsequent fractures. Questionnaires were mailed out to 546 rural women who underwent osteoporosis screening 8.3 years previously by bone densitometry and a 24-item clinical risk factor assessment. Our survey had a 55% response rate and found that 11.9% of respondents had subsequent fractures. A prior fracture history, self-reported rheumatoid arthritis, and menopause age ≤40 were significantly associated with subsequent fractures. A logistic regression analyses showed only a prior fracture history and menopause age ≤40 were predictive variables. Although we were unable to associate increased fracture risk in this age group other reported risk factors of an osteoporotic bone density, FRAX formula, smoking, parity, chronic illnesses, and no hormone replacement during a normal postmenopausal status, these should not be ignored.

Introduction

Declining bone mineral density (BMD) along with increasing clinical risk factors (CRFs) are associated with a sharp increase in osteoporotic fractures in women after age 60.[1] Women aged 50-59 have about 40% fewer fractures and a 45% lower prevalence of osteoporosis than those aged 60-69.[2] Fractures in this younger postmenopausal age group, however, still incur considerable costs and loss of quality-adjusted life years.[2,3]

The use of CRFs for fracture prediction in women aged 50-59 appears practical since most fractures occur with a normal or osteopenic BMD rather than osteoporosis.[4] The online availability of the FRAX formula developed by the World Health Organization uses 9 CRFs (height, weight, age, prior fracture history, parental history of hip fracture, smoking, 3 or more units of alcohol daily, 3 or more months of glucocorticoid therapy, rheumatoid arthritis, and secondary causes of osteoporosis) with or without incorporating the femoral neck BMD.[5-7] A FRAX CRF threshold of ≥20% absolute probability of a major osteoporotic fracture (hip, spine, wrist, or shoulder) or ≥3% at the hip over a 10-year period is considered as high risk. Others, however, have reported success by using simpler CRF assessments.[8-11]

Although routine BMD screening has been recommended to identify fracture risk in women aged 65 or older, a recent update of the US Preventive Services Task Force (USPSTF) guidelines recommended that Caucasian women under age 65 have BMD screening if CRFs by the FRAX tool are at a 9.3% or greater risk of a major osteoporotic fracture (hip, vertebral spine, wrists, or humerus) occurring over the next 10 years.[12] The use of this lower ≥9.3% CRF threshold combined with a low BMD will identify more women at high fracture risk since the majority who fracture in this age group do not have an osteoporotic BMD.[13-14] The purpose of this study is to report risk factors associated with a subsequent fracture occurrence in a sample of rural women aged 50-59 who were evaluated 8.3 years previously.

Materials and methods

Questionnaires were mailed out in 2008 to 546 women (97.7% Caucasian) aged 50-59 who had previously participated in osteoporosis screening by a traveling van throughout various rural areas in West Virginia from 1998-2001. Height and weight in light clothing and a self-administered questionnaire containing 22 different items were obtained at the time of the examination. All received a peripheral forearm (pDXA) BMD at the one-third (33%) radius of the non-dominant forearm. The pDXA device (GE Lunar) employed had a 2.09% short-term coefficient of variation with a mean BMD (SD) of 0.779 (0.016) gm/cm^2 and a 1.91% long-term coefficient of variation, determined by measuring every 40th or 50th woman on the same day with a mean BMD (SD) of 0.778 (0.015) gm/cm^2. Women were referred to a physician if two or more risk factors were present or a BMD reading of osteoporosis.

Table 1. ***Baseline characteristics of women aged 50-59 from initial survey for determining fracture risk***

	311 responders	255 non-responders	p-value
Age, mean (SD), yrs	53.93 (2.80)	53.76 (2.93)	0.485
Body-mass index, mean (SD)	27.50 (5.53)	29.03 (6.56)	0.003
BMD T-score, mean (SD)	-0.579 (1.34)	-0.702 (1.45)	0.294
Follow-up duration, mean (SD), yrs	8.34 (0.55)	8.33 (0.55)	0.882
	Prevalence	**Prevalence**	
Exercise (<15 minutes daily)	64 (45.5)	124 (48.6)	0.456
Dairy products (< 1 cup daily)	123 (39.7)	103 (40.4)	0.863
Seizure disorder	2 (0.65)	4 (1.57)	0.417
Rheumatoid arthritis	18 (5.81)	27 (10.6)	0.037
Liver disease	4 (1.29)	2 (0.78)	0.695
Juvenile diabetes	1 (0.32)	3 (1.18)	0.332
Thyroid treatment	58 (18.7)	57 (22.4)	0.285
Postmenopausal	198 (63.6)	162 (63.5)	0.996
Hormone replacement therapy	167 (53.7)	119 (46.7)	0.09
Current smoker	39 (12.6)	70 (27.5)	<.001
Alcohol-3 or more units daily	2(0.65)	0(0.00)	0.504
Prednisone (6 or more months)	6 (1.94)	11 (4.31)	0.10
Menopause ≤ age 40	20 (6.45)	18 (7.06)	0.774
Previous fracture	29(9.32)	24(9.41)	0.54
Family history osteoporosis	73 (23.6)	49 (19.2)	0.21

Abbreviations: BMD=bone mineral density.

Our questionnaire sought to determine whether a subsequent fracture had occurred and, if so, at what site. We included a check-off list to determine whether various brand name agents which have anti-fracture efficacy had been taken for at least one year after the initial evaluation.

Our risk factor assessment for subsequent fracture prediction used baseline demographic variables, forearm BMDs, the FRAX formula, other CRFs not included in the FRAX (menopause ≤age 40, dairy consumption, exercise, and seizure disorder), and a low BMD added to FRAX CRFs.[12,14] We also evaluated women without subsequent fractures who were at high risk to determine whether any intervention had been undertaken. In this group, we considered high risk by prior fractures, chronic glucocorticoid therapy, menopause age ≤40, BMD T-scores of ≤-2.5, or T-scores between -2.0 and -2.5 with FRAX CRFs ≥10.[12-16]

Analyses of variables in contingency tables were assessed by X^2 test or Fisher's exact test. The mean (SD) was presented for continuous variables. *P*-values of <0.05 were considered significant and underwent a regression analysis to determine which independent factors best predicted fracture with odds ratios and 95% confidence intervals. This study was approved by the Charleston Area Medical Center-West Virginia University Institutional Review Board.

Results

Our mail survey had a 55% (311/486) response rate (80 women could not be contacted because of inadequate postal addresses). Responders did not differ from non-responders in age or BMD T-scores but appeared healthier with significantly less self-reported rheumatoid arthritis, less smoking, and a lower body mass index **(Table 1)**.

Table 2. ***Baseline variables in women with a subsequent fracture or no fracture after a follow-up period of 8.34 years.***

	Fractures (n=37)	No fractures (n=274)	p-value
Age at baseline, mean (SD), yrs	53.92 ±2.99	53.93 ±2.77	0.98
Age at follow up, mean (SD), yrs	62.76 ±3.07	62.68 ±2.86	0.88
Body-mass index, mean (SD)	27.87 ±6.22	27.45 ±5.44	0.66
BMD T-score, mean (SD)	-0.469(1.62)	-0.574 (1.30)	0.66
FRAX CRFs, mean (SD)[a]	8.47% (4.68)	7.43% (2.84)	0.18
	Prevalence	**Prevalence**	
Exercise (<15 minutes daily)	16/37 (43.2)	127/274 (45.6)	0.79
Dairy products (< 1 cup daily)	13/37 (35.1)	110/274 (40.1)	0.56
Seizure disorder	0/37 (0.00)	2/274 (0.73)	1.00
Rheumatoid arthritis	5/37 (13.5)	13/274 (4.7)	0.03
Liver disease	0/37 (0.00)	4/274 (1.5)	1.00
Juvenile diabetes	0/37 (0.00)	1/274 (0.36)	1.00
Thyroid treatment	8/37 (21.6)	50/274 (18.3)	0.62
Postmenopausal	27/37 (73)	200/274 (73)	1.00
Family history osteoporosis	9/37 (24.3)	36/274 (23.4)	0.60
Current smoker	3/37 (8.1)	36/274 (13.1)	0.60
Alcohol-3 or more units daily	0/37 (0)	2/274 (0.7)	1.00
Prednisone (6 or more months)	2/37 (5.4)	4/274 (1.5)	0.15
Prior fracture history	7/37 (18.9)	20/274 (6.9)	0.02
BMD T-score ≥-1.0	24/37 (64.9)	177/274 (64)	0.97
BMD T-score ≤-1.0 to ≥-2.5	7/37 (18.9)	77/274 (28.1)	0.24
BMD T-score ≤-2.5	6/37 (16.2)	21/274 (7.7)	0.08
Baseline HRT users	24/37 (64.9)	156/274 (56.9)	0.36
HRT use during follow-up[b]	7/37 (18.9)	55/274 (20.1)	0.87
HRT use discontinued during follow-up period	19/24 (79.2)	108/156 (69.2)	0.32
Never HRT use	11/37 (29.7)	111/274 (40.5)	0.21
Menopause age ≤40	6/37 (16.2)	14/274 (5.1)	0.01
FRAX CRFs ≥9.3% with osteopenia or osteoporosis[c]	7/37 (18.9)	26/274 (9.49)	0.08

Abbreviations: BMD=bone mineral density; CRFs =clinical risk factors; HRT=hormone replacement therapy.
a Percent values represent 10-year absolute probability of major fracture.
b Includes those who maintained baseline HRT or were prescribed HRT during follow-up period .
c Forearm bone density added but not incorporated into FRAX formula.

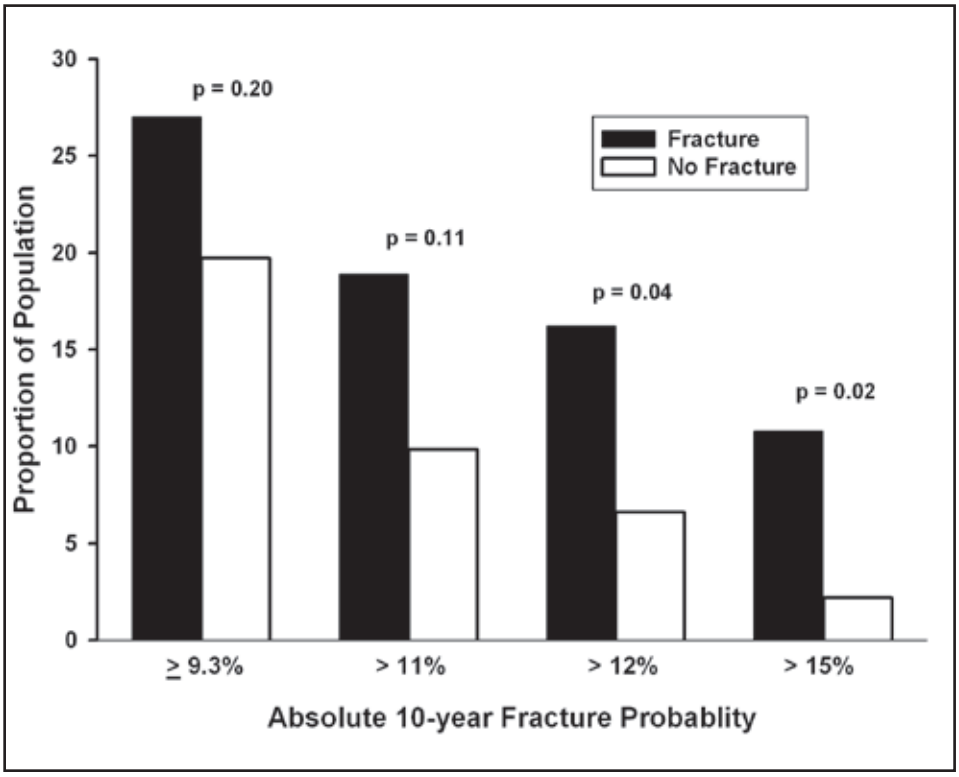
A mean (SD) follow-up period of 8.34 (0.55) years revealed that 37 of 311 (11.9%) women subsequently sustained major fractures at the wrist (18.9%), spine (5.4%), hip (2.7%), not listed (2.7%) and minor fractures (70.2%), e.g., clavicle, ribs, fingers, pelvis, ankle, etc. A univariate analysis found those with subsequent fractures had significantly higher self-reported rheumatoid arthritis, menopause age ≤40, and a prior fracture history **(Table 2)**. After a logistic regression analysis, the odds ratio (95% CI) was 2.72 (1.04-7.15)

(*P*=0.045) for a prior fracture history and 3.40 (1.19-9.95) (*P*=0.026) for menopause ≤age 40. Menopause age ≤40 had significantly lower mean (SD) BMD T-scores than the residual population [-1.67 (1.70) vs -0.483 (1.72), respectively, *P*= 0.003].

Neither the FRAX formula at a ≥9.3% CRF threshold, osteopenia, nor osteoporosis alone were predictive of fractures. Furthermore, adding a low BMD (osteopenia or osteoporosis) independently to the FRAX CRFs ≥9.3% threshold did not add to the predictive value. Increasing the FRAX CRFs threshold to ≥12%, however, significantly improved fracture prediction but had a low sensitivity **(Figure 1).**

Baseline HRT users trended toward significance with a mean (SD) BMD T-score of -0.417 (1.02) when compared to non-users -0.692 (1.45), respectively, *P*=0.052. Subsequent fractures, however,

Figure 1.
FRAX clinical risk factors without incorporated bone density on subsequent fractures.



were not significantly different in women with baseline HRT use, baseline HRT use not maintained, HRT use during the follow-up period, and never use of HRT.

Anti-osteoporosis agents, including HRT, were prescribed for at least one year in 89.2% (33/37) with subsequent fractures. After evaluating women who did not subsequently fracture, 19.7% (54/274) were considered at high risk. Of these, 75.9% (41/54) received at least one or more bone remedial agents.

Discussion

An evaluation of 311 early postmenopausal women with a baseline mean age of 53.9 found that the variables of a prior fracture, menopause under age ≤40, and self-reported rheumatoid arthritis were significantly associated with a subsequent fracture after 8.3 years. After a regression analysis, only the independent variables of a prior fracture history and menopause age ≤40 remained significant.[15]

A prior fracture history has been emphasized as a powerful predictor of future fractures independent of other CRFs.[15-21] A meta-analysis of early postmenopausal women with a prior wrist fracture have a relative risk (RR) of 2.0 times for subsequent fractures.[16] Vertebral fractures have an incidence of 40% lower than wrist fractures in this age group; however, about two-thirds of these remain asymptomatic and are detected incidentally. Prior vertebral fractures display a RR 4.4 times for subsequent vertebral fractures and 2.3 times for subsequent hip fractures, whereas women who experience a prior hip fracture have a RR of 2.5 times of subsequent hip fractures and 2.3 times risk of subsequent vertebral fractures.

We noted an 8.1% prevalence of osteoporosis in women with a baseline prior fracture which is consistent with other reports in this age group.[14,22] The rate of bone loss closest to the time of fracture appears to account for this increased fracture risk at all sites and is independent of the BMD.[23] In this regard, initiating treatment in those with a prior fracture without performing a BMD is recommended in the United Kingdom from a cost perspective.[24] Decisions to in initiate treatment in this situation could be made on an individual basis related to underlying risk factors.

Using CRFs alone at a ≥9.3% risk threshold from the FRAX formula or adding low forearm BMD T-scores, not incorporated into the FRAX formula, did not improve future fracture prediction. The reason for this may be related to the high baseline HRT use since no adjustments exist for the protective effect of HRT use in the FRAX formula. Additionally, the mean age in our sample was considerably younger than the mean age of the FRAX formula. This likely had an impact since CRFs increase and BMD declines considerably after age 60.[1] One study found that the FRAX did not predict fractures in younger postmenopausal women any better than parity, age, fracture history, and BMD.[15] We found, however, applying a FRAX CRF threshold of ≥12% significantly improved fracture prediction, but only about 15% of women who subsequently fractured were identified and about one-half of these occurred at minor sites. Although the FRAX estimates the 10-year absolute probability of fracture at a major site, no prediction values exist for minor fracture sites.[5-7] The optimum threshold using FRAX CRFs in perimenopausal and younger postmenopausal women requires more studies.

Our proportion of subsequent fractures at the wrist, vertebral spine, and hip of women in this age group was similar to predominantly Caucasian women reported in Olmstead County, MN.[14] The proportion of minor fractures we noted was approximately identical to Olmstead County, MN, but our fracture incidence was higher. Wrist fracture incidence rates increase during the early postmenopausal years and than plateau after age 60 throughout the remaining years.[25] On the other hand, incidence rates sharply increase for hip, vertebral, and humeral fractures after this age. Identifying and treating women during the early post-menopausal years may have an impact on future fracture reduction, especially after age 60. This becomes especially important in West Virginia which has a predominantly Caucasian population and is second nationally in women aged 65 or older.[26]

The common prescribing of HRT a decade ago was shown to reduce fractures.[15,21] The marked decline in continuing HRT we noted during the follow-up period was most likely influenced by the risks reported from the Women's Health Initiative Study.[27] Although higher BMD T-scores were observed in women with baseline HRT use, no protective effect on fracture reduction was observed once HRT use was discontinued. Discontinuing HRT results in a loss of fracture prevention due to a rapid loss of bone over a one and one-half year period with BMD returning to baseline values.[28,29] The prescribing of various other bone anti-resorptive agents during the 8.3 year follow-up period confounds our interpretation.

Menopause age ≤40 had a profound effect on increasing the future fracture risk and has been verified by others.[18,30] Significantly

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lower baseline BMD T-scores when compared to the residual population may explain this. No information was available on what proportion of women had oophorectomies since this results not only in less estrogen aromatization in bone and fat, but also less circulating androgenic hormones to maintain bone density.[31] Furthermore, 40% in this group received HRT at baseline, and only 15% maintained this during the follow-up period. Prescribing HRT in this instance, regardless of reported risks, still should be considered as a primary intervention. A cost-effective review in younger postmenopausal women showed HRT benefits through a substantial increase in quality-adjusted life years gained, reduction in coronary artery disease, fracture, and colon cancer.[32] These benefits outweighed the risks of death from pulmonary embolism, stroke, and breast cancer.

Our study has several limitations. First, the self-reported rheumatoid arthritis in our survey is higher than the true 1-2% prevalence in the general population. This observation should require verification by a physician prior to FRAX inclusion. Second, forearm BMDs likely under-estimate the true prevalence of osteoporosis when compared to central densitometry. The pDXA, however, is recommended as the alternative site for a BMD when central densitometry is unavailable.[33] Only about one-fifth of counties in West Virginia have central densitometry technology. Third, we did not have information on what proportion of fractures resulted from a low-trauma event (e.g. falling from a standing position); however, high-trauma fractures (e.g., falling from stairs, or a ladder, or a motor vehicle accident) in women also are associated with a low BMD.[34,35] Additionally, since our rural population in central Appalachia has nearly the lowest per capita and household incomes in the U.S. and a higher prevalence of osteoporosis, our data may not reflect other regions.[36,37] The strength of our study is an 8.3 year follow-up period.

In conclusion, we were able to identify only a prior history of fracture and menopause age ≤40 without HRT as independent predictors of future fracture risk in early postmenopausal women. The FRAX formula cutoff ≥9.3% threshold did not significantly identify high fracture risk until a ≥12% threshold was achieved; however, only a small proportion of the population of those who subsequently fractures was found. Risk factors of an osteoporotic BMD, chronic illnesses, smoking, HRT nonuse, and parity defined by other studies in younger postmenopausal women should also raise awareness of increased fracture risk.

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Ocular Demodicosis

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Abstract

We present a case of blepharitis with symptoms lasting two years in duration and refractory to a host of prior medical treatments, including antibiotics, corticosteroids, cyclosporine, and baby shampoo. We recognized the clinical presentation as pathogomonic for demodicosis caused by the parasitic mite, demodex folliculorum, confirmed with light microscopy, and treated appropriately with tea tree oil and hygiene measures--achieving full resolution of symptoms. We highlight the presentation, treatment, and underscore demodicosis as an important, under recognized cause of blepharitis.

Introduction

Blepharitis is a very common condition of the eye, responsible for about 5% of all eye conditions reported to general practitioners, with increased prevalence in the >50 age group.[1] It can be divided into the posterior and anterior subtypes, the former being more common and associated with meibomian gland dysfunction. The anterior subtype, however, is most commonly caused by stapylococcus, seborrheic dermatitis and rosacea. Demodicosis is a condition which refers to infestation with the parasitic mite *demodex* species, *folliculorum or brevis* (see Figure 1),and can lead to both types of blepharitis: anterior (*d. folliculorum)* and posterior (*d. brevis)*. Recognizing demodicosis as the cause of blepharitis is often simple, due to pathognomonic cylindrical dandruff (Figure 2), and important, due to its recalcitrance to all standard medical therapies, except tea tree oil.[2] Nonetheless, demodicosis often goes unrecognized due to lack of awareness and is mistreated as a typical blepharitis. In this paper, we present a case of anterior blepharitis caused by the demodex folliculorum species.

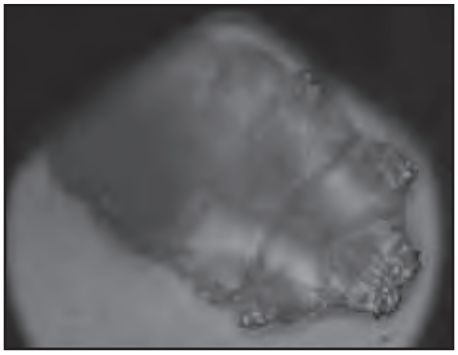
Case Presentation

A 60 year old female, wife of a physician, presented to the clinic with eye irritation, burning, and itching that has been present over the past two years. She has seen multiple medical practitioners for this problem, including her primary care provider, two optometrists, and one ophthalmologist. She wears contact lenses but denies any decrease in visual acuity or peripheral vision. She denies scotomas, headaches, allergies, and other systemic symptoms, including musculoskeletal, gastrointestinal, or genitourinary symptoms. Her past medical history is significant only for a history of early cataracts. Her past medical trials with these providers include antibiotic drops, corticosteroid drops, cyclosporin (for dry eyes), and baby shampoo. Her social history reveals that her dogs sleep next to her.

On physical examination, she is found to have erythematous eyelid margins, with cylindrical dandruff at the base of most of the eyelashes (Figure 1). Her conjuctiva are also slightly red, and spontaneous tearing is noted. Her visual acuity is 20/20 bilaterally, and she does not have any seborrheic dermatitis or rosacea on any other visible portion of her body.

Based on her presentation--blepharitis with cylindrical dandruff, refractory to medical trials with standard agents, and a history of sleeping next to her dogs, a presumptive diagnosis of blepharitis due to ocular demodicosis (infestation with the mite demodex follicularum) was made. For confirmation, we epilated 4-6 lashes bearing the cylindrical dandruff and visualized the parasitic demodex mite under simple light microscopy. The patient was then started on a regimen of tea tree oil shampoo, whereby she would massage the 5% tea tree oil shampoo into her eyelid margins for 5 minutes, twice a day after washing her face with baby shampoo. She continued this practice for 4-6 weeks, along with hygiene measures (change bedding, keep pets out of bedroom, discard used makeup containers, treat spouse with same regimen), and achieved a full resolution of symptoms. After several months of being symptom free, her blepharitis returned for a period of 7 days but was promptly relieved again with another course of tea tree oil.

Figure 1.
Picture of demodex folliculorum *species. Demodex brevis looks very similar, except that the body is shorter in length.*

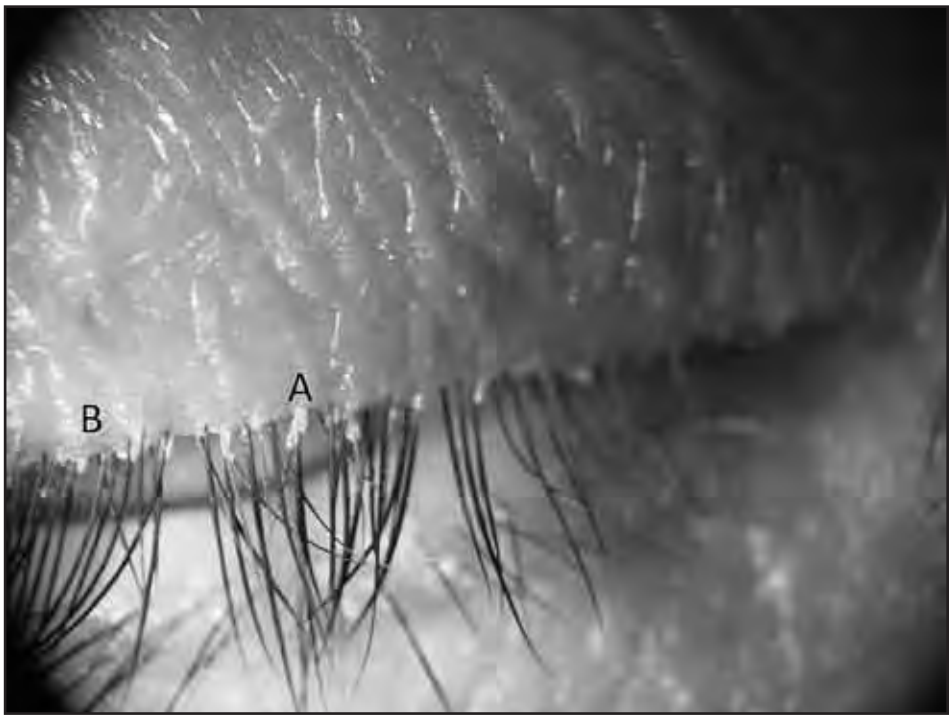


Discussion

Demodicosis is an infestation with the parasitic mite, *demodex* species (Figure 1), of which there are

Figure 2.
Picture of Eyelid with cylindrical dandruff due to ocular demodicosis/ blepharitis.

Figure 3.
Picture of Eyelid with both cylindrical dandruff (A) due to ocular demodicosis, and (B) typical dandruff due to concomitant bacterial infection.



two types: *demodex folliculorum and demodex brevis*. Demodex occurs in the general population on the eyelids and nose in 4% of people less than 19yo, 30% of 20-80yo, and 47% of those greater than 80yo, but often it occurs as a commensal organism and does not cause symptoms.[6] For instance, demodex was found to be present in 100% of patients with cylindrical dandruff on their eyelashes--a pathognomonic sign of demodex infestation (Figure 2)--but also in 22% of those with clean lashes.[2,8]

Demodex folliculorum tunnels its way down the hair shaft towards the follicle. The abrasive action of its claws is believed to result in epithelial hyperplasia and increased keratinization. For nutrients, demodex pierces epithelial cells and consumes cytoplasm and debris.[3] During its life cycle, waste may accumulate and harbor bacteria, viruses, and rickettsia.[3] *D. folliculorum* may also serve as a vector for *Staphylococcus aureus*, and has been associated with a perifollicular lymphocytic infiltration.[4] The perifollicular inflammation, epithelial hyperplasia, and follicular plugging cause the clinical blepharitis. These follicular changes make the eyelash more brittle and can lead to madarosis (lash loss), misalignment, or trichiasis (lash abrasion of cornea). Left untreated, serious sequelae ranging from conjunctivits to corneal superficial opacities, corneal neovascularization, and marginal corneal infiltration can result when the inflammation spreads from the eyelid.[2]

Demodex brevis is a similar organism except that it infests the meibomian and sebaceous glands. It can affect the lipid layer of the tear film, leading to dry eyes, and occasional cylindrical dandruff formation. *D. brevis* infection predisposes to meibomian gland dysfunction and chalazion formation, including in the pediatric population.[5]

Demodex infestation is also the cause of two other conditions: a dermatologic condition known as Pitryiasis folliculorum (rosacea-like skin rash in humans) and mange in dogs. Mange is a disease in dogs which causes dogs to lose patches of their fur, and is most frequently associated with demodex canis, which is species specific, though demodex folliculorum has been documented to infest dogs and their owners.[7] For this reason, one of the hygiene measures used to eradicate *demodex* is keeping pets away from sleeping surfaces.

Testing can be performed for confirmation or in cases without cylindrical dandruff but a high index of suspicion. The simplest method is to epilate some lashes with cylindrical dandruff and visualize under a light microscope. Normal saline is generally sufficient to see the moving demodex mites, but flurosceine staining has been shown to enhance the detection.[9] In vivo laser scanning confocal microscopy has also been shown to be able to diagnose, predict the number of mites, and to follow the course of treatment noninvasively (without epilation of the lashes).[10]

Tea Tree Oil (TTO) is the gold standard therapy, as all standard medical therapies fail to kill *demodex,* even *in vitro*.[2] When combined with eyelid hygiene,

TTO is able to eradicate demodex infestations in 77-100% of patients.[2] The simplest treatment regimen is 5% TTO shampoo massages twice a day for 4-6 weeks.[7]

Conclusion

The common presentation for demodicosis includes ocular irritation, blepharoconjunctivitis, cylindrical dandruff, and symptoms refractory to usual medical therapies in an immunocompetent person aged greater than 50. Cylindrical dandruff alone suggests demodicosis. Consider demodicosis in refractory cases of blepharitis, even in children. Treat with tea tree oil shampoo and eyelid hygiene to achieve complete symptom resolution and to prevent serious vision threatening sequelae.

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Introduction

In 2010, 17.4 million Americans were estimated to be using marijuana, and 4.6 million were using the drug on a daily basis.[1] Marijuana has long been thought of as an anti-emetic, leading many groups to advocate for the legalization of the drug for medical use. Sixteen states have already legalized the use of medical marijuana. In California alone, there is estimated to be over 750,000 medical marijuana users.[2] However, with the increasing prevalence, there have been reports over the last decade of a cyclic vomiting syndrome which is actually associated with excessive marijuana use.

Cyclic Vomiting Syndrome is defined as recurrent episodes of vomiting with normal periods of health in between episodes.[3] There appears to be a subset of patients with cyclic vomiting associated with marijuana use. The disease has been coined Cannabinoid Hyperemesis Syndrome (CHS). It was first described in 2004 by an Australian group of investigators who described 10 patients with the syndrome. The clinical features are (1) long term, excessive use of marijuana; (2) cyclical vomiting pattern that often begins years after initiation of the drug; (3) compulsive hot showering with symptom relief; and (4) resolution of symptoms with abstinence from marijuana.[4]

We present a case series of four patients that we have encountered over the span of 1 year at our tertiary care institution in the hopes of educating other physicians of this underrecognized syndrome. Two of the four patients had extensive and potentially unnecessary diagnostic evaluations and interventions, including surgery, which could have been avoided if the diagnosis was made earlier in their clinical course.

Case Series

Patient #1

Patient #1 was a 28 year old white male who presented to our general medicine clinic for a second opinion in regards to his cyclic abdominal pain and vomiting. His symptoms had been ongoing for the last four years. He had accompanied nausea and vomiting. The episodes occurred every few months and lasted for several weeks at a time. The patient had undergone a Nissen Fundoplication for what was presumed to be intractable GERD. He went on to have a cholecystectomy which again did not relieve his symptoms.

During the patients evaluation he was found to have normal labs. Porphyria panel was also negative. The patient had multiple CT scans and abdominal films as well as EGD with small bowel biopsy and colonoscopy. All results were negative.

Further history revealed that the patient had been smoking marijuana since the age of 17. He smoked on average three joints daily. The only thing that relieved his symptoms were hot showers. The patient was counseled that he should abstain from marijuana. However, he has continued his chronic use of the drug. He continues to have cyclic episodes of abdominal pain and vomiting.

Patient #2

Patient #2 is a 32 year old African American male who has been hospitalized at our facility twenty four times in the last two years. He presented with cyclic episodes of vomiting and abdominal pain. He was also observed to take multiple hot showers which subjectively relieved his symptoms.

Diagnostic workup included imaging studies (CT scans, plain films, and abdominal ultrasounds) which were always negative. Laboratory testing on multiple occasions were always normal except for minor elevations in creatinine due to volume depletion, which responded quickly to fluid resuscitation each time. Urine drug screens were persistently positive for marijuana.

In regards to his marijuana use, the patient had been smoking marijuana since age 19. He smoked up to 2-3 times daily. He struggled considerably with abstaining. He continues to use cannabis and continues to present with the same symptoms despite frequent counseling.

Patient #3

Patient #3 is a 23 year old white female with ten days of epigastric pain and vomiting. She had three previous episodes in the past. The patient's symptoms were improved with hot showers. She was admitted for IV hydration.

Diagnostic work up included radiographic imaging (CT scans and

Table 1. Characteristics of each patient in case series.

Patient	Age	Sex	Race	Duration of Illness	Relief with Hot Showers	Hospitalizations In the last 1 yr	**Age at first** Marijuana Use	Average daily Marijuana Use	Abstinence from Marijuana	Resolution of Symptoms
#1	28	Male	White	4 yrs	Yes	10	17	1-3	No	No
#2	32	Male	African American	2yrs	Yes	12	19	2-3	No	No
#3	23	Female	White	1yr	Yes	1	15	3	No	No
#4	22	Male	White	6 mo	Yes	2	13	3	Yes	Yes

plain films) which was negative. Basic labs were normal. She did have mild elevation of amylase to 144 and lipase to 92, both of which normalized the following day. This was likely due to her persistent vomiting.

The patient had a history of chronic marijuana use since the age of 15. She was smoking three times daily on average. The patient was educated on the syndrome and planned to quit upon discharge. However, she was readmitted for the same symptoms two months later and it was found that she had resumed her heavy use of marijuana.

Patient #4

Patient #4 is a 21 year old male who presented with two days of abdominal pain and vomiting. He had two similar episodes which had occurred in the past six months. This was his second hospitalization. During his stay, nursing found him to be in the shower each time they went to assess him. He stated that hot showers relieved his symptoms but they quickly returned when he came out.

Diagnostic imaging studies were negative. Basic labs were also normal. He improved after 2 days of IV fluids.

The patient had a history of marijuana use since the age of 13. He smoked at least three times daily, often more. Both the patient and his parents were educated about CHS and the importance of abstaining from marijuana. The patient has abstained since that time and has had no further episodes.

Please refer to Table 1 and Table 2 for a summary of the patient characteristics and diagnostic tests.

Discussion

These four patients likely represent a larger group with CHS which has gone undiagnosed. Patient #1 has had significant morbidity from his symptoms including multiple hospitalizations and poor quality of life. He has had multiple unnecessary diagnostic tests performed and has undergone major surgical intervention for the symptoms with no relief. Although, there was a significant delay in diagnosis of CHS, the patient also has not stopped his marijuana use and still has recurrent symptoms. We also see in patient #2, significant morbidity and repeated hospitalizations due to CHS. The patient has acknowledged the connection of his disease to chronic marijuana use, but still has been unable to stop.

CHS was first described in 2004 by an Australian group of physicians who noticed similar symptoms in 10 patients with chronic, long term marijuana use. They described the patients as having cyclic vomiting and abdominal pain. They also found that most of the patients had the peculiar finding of compulsive hot showering which relieved their symptoms. They were able to follow the 10 patients and found that only those who abstained from marijuana had relief of symptoms.[4] There have been additional case reports which have demonstrated the same symptomatology. These case reports have documented 45 patients with a mean age of 31years. Of the patients that were followed, 40 abstained from marijuana for an extended period of time and all had symptom resolution. Seven of those patients relapsed

Table 2. Diagnostic testing results and procedures performed on patients from case series. Lab values are from the patients' first encounters at our institution. Radiographic testing and procedures were performed at any point in the patients' illness.

Patient	Labs	Imaging		Procedures
		Study	Result	
#1	WBC 8.1; Hg 15; Platelets 268 Na 136; K 3.5; Cl 102; Bicarb 25; BUN 17; Creatinine 0.8; glucose 102 Bili 0.2; AST 28; ALT 44; Alkp 56; GGT 29; amylase 54; lipase 21 Celiac Panel: Negative Porphyria Panel: Negative U/A[A]: negative UDS[B]: positive for cannabinoids	CT abdomen and pelvis (performed between 5-10 times)	Normal	Nissen Fundoplication Cholecystectomy EGD with small bowel biopsy with normal findings Colonoscopy with normal findings
		RUQ U/S[C]	Normal	
		Plain abdominal xray	Normal	
#2	WBC 5.6; Hg 12.8; Platelets 365 Na 144; K 3.3; Cl 105; Bicarb 28; BUN 5; Creatinine 0.9; Glucose 85 Bili 0.6; AST 20; ALT 14; Alkp 82; GGT 20; amylase 126; lipase 126 U/A negative UDS: positive for cannabinioids	CT abdomen and pelvis	Normal	
		RUQ U/S	Normal	
		Plain abdominal xray	Normal	
#3	WBC 8.2; Hg 14; Platelets 266 Na 139; K 3.9; Cl 110; Bicarb 23; BUN 8; Creatinine 0.58; Glucose 101 Bili 1; AST 28; ALT 26; Alkp 48; GGT 17; LDH 187; amylase 144; lipase 92 Urine Pregnancy: negative U/A: negative UDS: positive for cannabiniods	CT abdomen and pelvis	Normal	EGD with normal findings
		RUQ U/S	Normal	
		Plain abdominal xray	Normal	
#4	WBC 16.7; Hg 15; Platelets 277 Na 142; K 3.9; Cl 106; Bicarb 23; BUN 17; Creatinine 1.16; Glucose 134 Bili 1.6; AST 39; ALT 28; Alkp 82; amylase 33: lipase 27 U/A: Negative UDS: Positive for cannabinoids	CT abdomen and pelvis	Normal	
		RUQ U/S	Normal	
		Plain abdominal xrays	Normal	
		Fluroesophagram	Normal	

[A]U/A: urinalysis
[B]UDS: urine drug screen
[C] RUQ U/S: right upper quadrant ultrasound

and had return of their symptoms with recurrent marijuana use.[5-13]

Many questions remain about CHS in regards to the pathology and mechanism of the disease. There has been little more than hypothesized answers to these questions. The first question is why would cannabinoids, which have previously been shown to have anti-emetic effects,[14] cause a pro-emetic disease? The answer may lie with the lipophilic nature and long half life of the chemical.[4] All the patients had excessive use which began at a young age. This may result in accumulation to the point of toxicity in genetically susceptible patients. In addition, the CB-1 receptors which mediate the anti-emetic effect of cannabinoids centrally are also present in the enteric plexus. However, activation of the enteric receptors causes decreased peristalsis and intestinal secretions which may lead to the pain and nausea.[4]

The other main question is in regards to the bizarre behavior of compulsive hot showering. It has previously been demonstrated that cannabinoids affect the limbic system of the brain. Cannabinoid toxicity may disrupt the balance of satiety, digestion, and thermoregulation and the disruption may settle with hot showers.[4] Another hypothesis is that hot showers redistribute blood from the splanchnic circulation to the skin. This temporarily

relieves the chronic stimulation occurring in the CB-1 receptors of the gut resulting in symptom relief.[13] Obviously more research is needed at the biochemical level to elucidate the molecular pathways.

The purpose of this case series is to alert other physicians to the disease. It is important to recognize the symptoms of CHS in order to prevent unnecessary diagnostic testing and procedures as well as to hopefully "cure" the disease by educating the patient to stop smoking marijuana. Although important to avoid repeated diagnostic testing, it is still essential to rule out other acute causes of the symptoms. In patients presenting with recurrent episodes of vomiting, it is appropriate to ask if hot showers relieve the symptoms. Previous authors have recommended asking "have you ever tried marijuana to relieve the nausea?" which may uncover a history of chronic marijuana use.[15] This disease also raises the question of whether a urine drug screen should be added to the initial evaluation of patients with Cyclic Vomiting Syndrome. We recommend continued counseling to abstain from marijuana when the diagnosis is made. This may be the most difficult aspect of treating the disease since these patients are often physically and psychologically addicted to the drug.[16] We emphasize the importance of assisting cessation and educating the patients about CHS in order to avoid continued hospitalizations and unnecessary testing.

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Cetacaine Induced Methemoglobinemia: Overview of Analysis and Treatment Strategies

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Abstract

Methemoglobin is formed upon iron oxidation of the heme molecule from ferrous (Fe^{2+}) to its ferric (Fe^{3+}) state. Normal methemoglobin levels in the body vary between 1-2% of the total hemoglobin. Cause of methemoglobinemia can be inherited or acquired. Inherited causes include an enzymatic deficiency in the enzyme cytochrome b5 reductase where as acquired causes are most commonly from routinely used medications. Herein, we present to you a case of methemoglobinemia after Cetacaine (a benzocaine based topical anesthetic) utilization during a transesophageal echocardiography. Some of the other common potential inciting agents are also discussed here along with an overview of treatment strategies.

Introduction

Methemaglobinemia is a commonly occurring clinical condition with its etiology related to either inherited or acquired causes. Congenital or inherited causes of methemoglobinemia result due to decreased activity of the enzyme cytochrome b5 reductase resulting in diminished enzymatic reduction of the hemoglobin molecule. Acquired causes are more prevalent and familiarity with known precipitants is necessary to address any acute presenting symptomology.[1] Conversion of heme iron to its ferric state makes the heme particle incapable of binding oxygen, resulting in a firmer binding of the oxygen molecule to the heme particle. This situation results in inadequate oxygen delivery to the tissues leading to hypoxia and other life-threatening conditions.

Cetacaine spray is a benzocaine based topical anesthetic which is commonly used in endoscopic procedures. Although, methemaglobinemia is a known side effect of benzocaine based anesthetics, the acuity and the variability in presentation requires quick understanding of the complaints and a specific solution. Presenting complaints mask a myriad of other differentials and although uncommon, immediate recognition is necessary for this potentially fatal condition.

Case Presentation

A 70 year old patient with streptococcus bacteremia was scheduled for a transesophageal echocardiogram (TEE). This patient had earlier denied any history of, IV drug abuse or any other cardiac interventions in the past. Physical examination findings were negative for any noticeable murmurs, dermatological findings or any other vascular phenomenon. Topical cetacaine was utilized as part of the operative protocol with a decline noted in the oxygen saturations within 20 minutes of administration. The patient at this point denied any significant complaints such as chest pain or shortness of breath. Patient also did not demonstrate any neurological or mental status changes during this episode. An ABG was then obtained which confirmed adequate oxygenation with PaO2 levels of 148. A methemoglobin level was then collected which returned with elevated levels of 33%. Decision was then made to give methlene blue at 1mg/kg dosing with improvements noted within 1 hour of administration. Repeat ABG's demonstrated methemoglobin levels of 16.8%. Patient was transferred to the ICU and was kept on continuous pulse oximetry for the next 36 hours. No further interventions with methlene blue were made given the patients asymptomatic status. Other differentials for this patient's hypoxia/hypoxemia could include hypoventilation, right to left shunt, atrioseptal defects or a ventilation perfusion mismatch. These were excluded by the patient's normal echocardiographic findings, chest X-ray and arterial blood gas results.

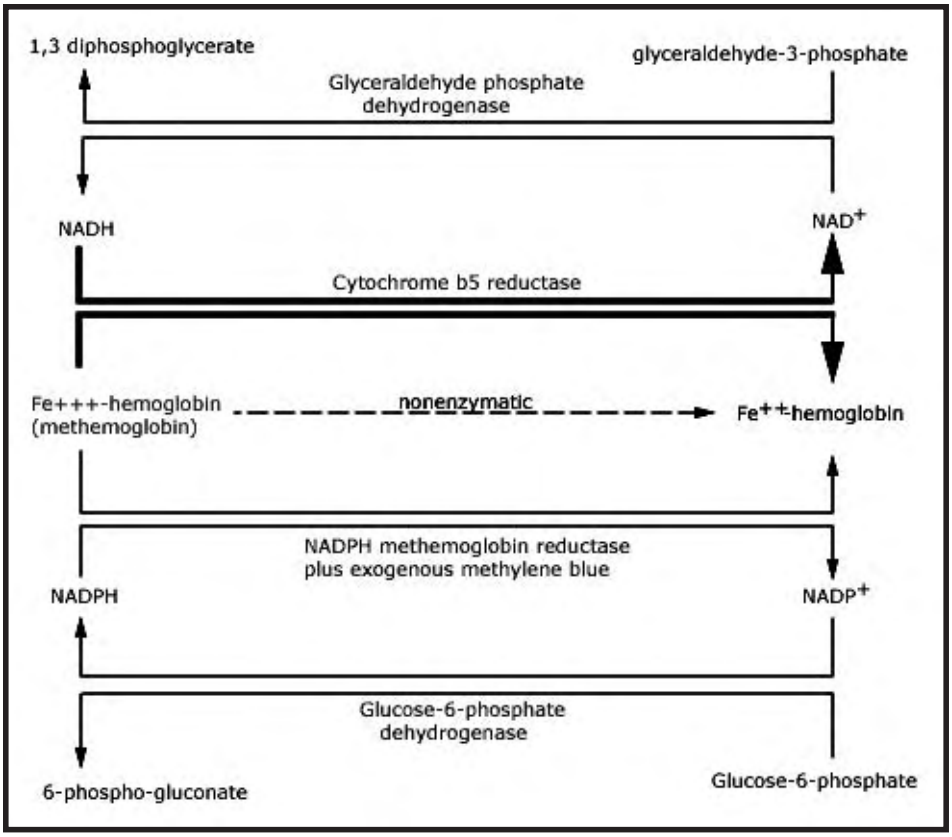
Discussion

Methemoglobinemia leads to a diminished oxygen supply resulting in a "saturation gap" between the oxygen saturations on a pulse oximetry and the arterial blood gas.[5] Presenting complaints could include fatigue, malaise, dyspnea, headaches, dysrhythmias, coma and or death.[7] This condition could be secondary to a genetic defect involving the cytochrome b5 reductase pathway (Figure 1). Reduction of methemaglobin to hemaglobin involves two pathways: 1) Cytochrome b5 reducatase pathway 2) NADPH glucose 6 phosphate dehyrogenase in the hexose monophosphate shunt pathway (Figure 1).[8] Other more commonly

Table 1: Substances that can cause Methemoglobinemia[4]

Inorganic Agents	Nitrates, fertilizers, chlorates, copper sulfates – fungicides
Organic Agents	Amyl Nitrate, Isobutyl Nitrite, Sodium Nitrite, Nitroglycerin, Nitroprusside, Nitric Oxide, Nitrogen Dioxide, Trinitrotoluene, Combustion products
Drugs	Local Anesthetics: Benzocaine, Lidocaine, Prilocaine Pyridium, Anti-malarials – Primaquine, Chloroquine, Rasburicase, Cyclophosphamide, Ifosfamide, flutamide, Acetaminophen, Acetanilid, Phenacetic, celecoxib, Zopiclone, Methlene Blue (high doses in G6PD-Deficient patients) Antibiotics: Sulfonamides, Nitrofurantoins, P-aminosalicylic acid, Dapsone
Industrial/Household Agents	Aniline Dyes, Nitrobenzene, naphthalene (moth balls), aminophenol, nitroethane (nail polish remover)

Figure 1.
The major pathway for methemoglobin reduction is via cytochrome b5 reductase (thick arrows). An alternative pathway, which requires an exogenous electron acceptor such as methylene blue, is via NADPH methemoglobin reductase. Only a small amount of methemoglobin is reduced via nonenzymatic pathways (dashed arrow).



seen causes could be from exposure to a potential inciting agent (Table 1).

Symptoms usually occur between 15 to 60 minutes of product administration with delayed reaction typically seen in products involved with slow absorptive mechanisms, such as with powder application. Sites of systemic absorption include broken skin tissue, inflamed gastric sites, eczematous skin and respiratory mucosa with the risk of a reaction increasing with number and duration of sprays administered.[2] Package instructions typically recommend a 1-2 second spray but given human performance limitations, no reliable estimate fractions can be predicted. Symptoms are typically noticed at methemoglobin concentrations of greater than 15%.[5] List of potential inciting agents are listed below.

Clues at the bedside include: persistent cyanosis, tachypnea, low pulse oximetry with normal PaO_2 levels on an ABG and a classic chocolate brown appearance of the arterial blood.[9]

Confirmation of the diagnosis requires CO-oximetry testing, which utilizes multiple wavelengths of light to detect serum methemoglobin levels. Other diagnostic modalities include a positive Kronenberg test and the presence of an oxygen saturation gap.[2] Confirmation with the Evelyn-Malloy assay is required as follow up since the co-oximeter can falsely read methylene blue as if it was methemoglobin.[5] Recent advances in technology have led to the creation of a device (The Rainbow Rad 57) which possess the ability to measure methemoglobin and carboxyhemoglobin in a noninvasive manner allowing a significant improvement in the ability to quickly diagnose and address these medical emergencies.[3]

Treatment strategies employed are different for an acquired cause as compared to a congenital cause. The current mainstay of treatment for an acute presenting condition involves the utilization of methylene blue. Methylene blue directly reduces the quantity of methemoglobin in the blood and is administered intravenously in a dose of 1 to 2 mg/kg given as a 1% solution over 5 minutes.[9] The dose maybe repeated if no resolution of symptoms is achieved within 1 hour. It should also be noted that doses of methylene blue in excess of 7mg/kg can precipitate and or worsen Methemoglobinemia.[5] Other treatment strategies include: Exchange transfusion, hyperbaric oxygen and ascorbic acid. Careful monitoring in the ICU is typically required for 24 to 36 hours given the possibility of rebound methemoglobinemia after exposure.[3]

Conclusion

Benzocaine related methemoglobinemia is an important clinical problem and requires physicians to be extra vigilant when utilized. Topical anesthetics have been reported to cause methemaglobinemia, but this adverse event is extremely rare and is not usually listed as one of the possible complications of procedures involving topical anesthetic use. Majority of patients are able to tolerate benzocaine based anesthetics but some patients will unfortunately develop methemoglobinemia upon exposure. Predicting the population at risk is not possible but given the severity of this condition, prompt recognition and treatment is needed.

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Spinal Cord Intramedullary Cavernoma: A Case Report

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Abstract

Background: Spinal Cord Intramedullary Cavernoma is a rare disease. It is a vascular disorder composed of capillary-liked vessels without intervening neurons within a spinal lesion. It may only be discovered incidentally or may be diagnosed after a neurologic deficit. Patients may present with weakness which could mimic other neurologic pathology.

Case: A case of a 65 year old with history of hypertension and diabetes mellitus. He had previous microdissectomy of the lumbar L4-L5 disc. He presented with progressive lower leg paresis, urinary retention and obstipation. An MRI revealed a cavernous angioma at the T5 level .

Conclusion: A multitude of neurologic deficits could lead to a patient presenting with a Spinal Cord Cavernoma. Prompt imaging is warranted in cases presenting with the symptoms to allow appropriate diagnoses and treatment. The clinician must be aware of this rare, but debilitating disease complex.

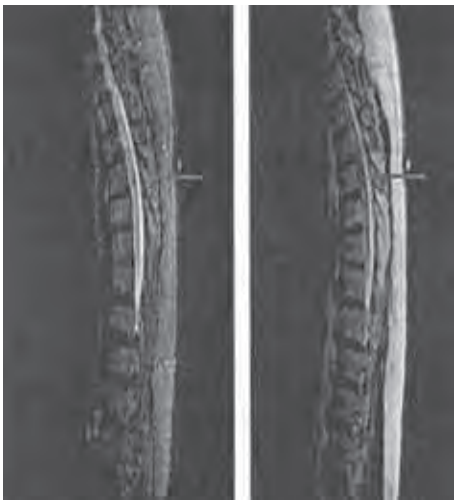
Introduction

Spinal cord intramedullary cavernoma is a rare disease and is a challenge to diagnose. It may present asymptomatically but may also be a potential source of significant morbidity. It was first observed in 1903 when an autopsy of a 35 y/o woman revealed a lesion that had bled at the level of L1. In 1912, the first successful surgery to remove an intramedullary cavernous malformation was performed.[1] This report demonstrates a case of a 65 year old white male who presented with acute onset and progressive decline in neurologic function was found, on exploratory surgery, to have cavernoma in the thoracic area.

Case Report

A 65 year-old, right handed white male with a history of hypertension and diabetes mellitus type 2 presented to our institution due to bilateral lower leg paresis, urinary retention, and obstipation having trials of several laxatives at home. Past surgical history is significant for lumbar microdissectomy at the L4-L5 level, he did state that symptoms began about 1 week prior to admission with left leg paresis and some urinary retention. On examination, He had good strength in his upper extremities and good hand grips. Strength testing of the lower extremities revealed diminished strength to the bilateral hip flexion (1-2/5), bilateral knee extension (1/5), bilateral ankle dorsiflexion and plantar flexion (0/5), as well as bilateral first toe extension (1/5). Passive range of motion was unremarkable. There was no noted fasciculation or Babinski reflex. He was hyporeflexic (1/4) to knee-jerk and ankle-jerk testing of the lower extremities. The patient had vibratory sensation and light and deep proprioceptive sensation around the umbilicus. There was decreased vibratory sense and pinprick to the lower extremities. There was no proprioception sensation to the feet and rectal tone was noted to be decreased. MRI of the cervical, thoracic and lumbosacral spine with and without contrast was done. Neurosurgical consultation was also obtained. MRI revealed areas of blood products involving a segment of the cervical cord (C4-C5 through C7-T1) and a possible cavernous angioma was noted at T5. An abnormal signal intensity in the mid to lower thoracic region consisting of increased T2 prolongation was said to represent acute infarct **(Fig.1)**. Patient then was transferred to a tertiary care center. Patient was confirmed to have a cavernous angioma at T5 with acute infarct. The patient did not require surgical intervention. He underwent intensive rehabilitation at a rehabilitation hospital. One year after this insult, the patient had made significant progress with the majority of muscle groups recovering 3-4/5 strength. He was also able to ambulate with assistive devices.

Figure 1. *MRI sagittal image of thoracic spine showing increased signal intensity throughout the thoracic cord. At T5 level, there is a focal area of dephasing which is heterogeneous more likely a cavernous angioma (STIR left, T2W).*



Discussion

Cavernous malformation (CM), also known as cavernous angioma, cavernous hemangioma or cavernoma, is a highly vascular lesion that can be found anywhere

Figure 2. *An image showing a spinal intramedullary cavernoma*

in the body.[2] It is a vascular lesion, composed of enlarged mass of sinusoidal type vessels with a single layer of endothelium and an absence of neuronal tissue within the lesions (**Fig.2)**.[3,8,10] Cavernous malformations have an estimated prevalence of 0.4-0.5%.[3] Only about 3-5% of all cavernous malformation involve the spine cord, mostly involving the intramedullary compartment. The exact cause of CMs are not known. Most cases are sporadic (50-80%) but, 10-30% of cases with multiple lesions in the spinal cord are familial and are associated with an autosomal-dominant defect chromosome (3, 7 q), Individuals with a CM and this chromosomal defect carry a higher risk of hemorrhage.[2,10] The peak incidence of presentation is usually in the 3rd – 4th decade of life.[2,3] Spinal intramedullary cavernous malformations in adults occur more frequently in women than in men with a female/male ratio of approximately 2:1. It is suggested that a hormonal effect may play a role in the presentation of clinical symptoms. In adults, cavernomatous malformations occur mostly in thoracic spinal cord while in children they occur with even distribution in the thoracic and cervical spinal cord.[8]

CMs may be discovered only incidentally at autopsy and never cause any symptoms or may cause a variety of neurologic deficits. Clinical presentation varies with level of involvement and whether there is infarct noted at presentation. The typical presentation of a bleeding spinal cavernoma is an episodic sensorimotor deficit. There is occasional recovery of symptoms due to slow seepage of blood from the cavernous malformation followed by gliosis or thrombosis within the cavernoma. This can lead to altered blood flow in the nearby neural tissues, thus leading to progressive myelopathy.[2,4,5,6] Several factors may contribute to the spinal cord injury following hemorrhage. Ischemic damage may arise from compression and decreased blood flow of adjacent cord structures. Vasospasm could occur as a result of exposure of spinal cord to red blood cells and inflammatory mediators and vasogenic edema could result from the breakdown of blood-spinal cord barrier.[7]

MRI is the imaging modality of choice in the evaluation of patients presenting with myelopathy. A cavernous malformations typically appears as a well-defined "popcorn-like" lesion with a heterogenous signal intensity on both T1- and T2-weighted images due to blood products in various stages of evolution. Areas of increased signal intensity with or around the lesion and a rim of low-signal intensity due to the hemosiderin pigment may surround the lesion **(Fig.3).** Recent hemorrhage in the cord may present with cord edema.[2,4,10] The appearance of a cavernous malformation with edema may be unclear and mimic other lesions such as neoplasm, including ependymoma, astrocytoma, hemangioblastoma, metastasis, multiple sclerosis plaque, and arteriovenous malformation. Cavernous malformation cannot be differentiated from neoplasm on a single study but can be suggested if the lesion contains a hemosiderin rim and is stable on serial studies.

Management for asymptomatic intramedullary cavernous malformation still remains controversial. Location of the lesion and the symptoms at presentation usually dictate the course of management. It is suggested that asymptomatic lesions which are exophytic should be excised, as well as symptomatic lesions that are exophytic. Deeper, asymptomatic lesions are more likely to be managed conservatively with regular clinical-radiological follow-up. **Figure 4**, provides an algorithm for management of spinal intramedullary CMs.[1]

There are factors associated with outcome after surgery. These include pre-operative health status, duration of symptoms and location of the

Figure 3. *These sagittal MR images of the spine (T1W left, T2W right) show a rounded spinal lesion with surrounding low signal on both sequences. The low signal appears to expand the central canal both proximally and distally. The low signal is due to old blood products (eg hemosiderin) which are typically found surrounding cavernomas.*

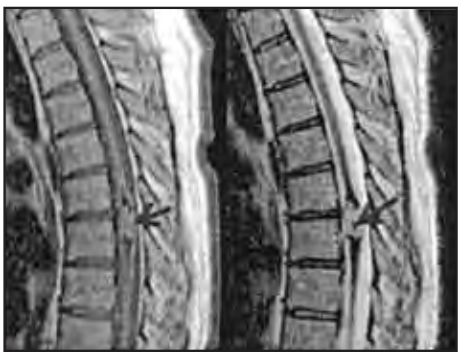
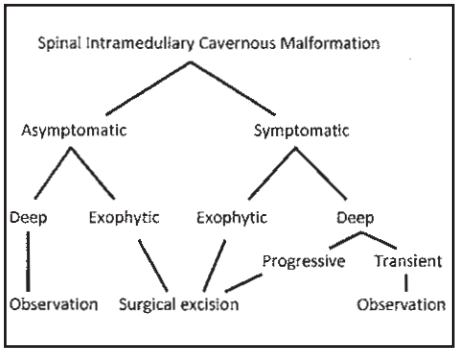


Figure 4. *Management of spinal intramedullary CMs.*



lesion. Age, sex and lesion size does not correlate with postoperative outcome.[8] In a study by Hans-Jakob Steiger et al., the most important prognostic factor for the functional outcome was the preoperative functional grade and duration of symptoms. Patients with a history of symptoms lasting more than 3 years fared worse than patients with a shorter duration of symptoms.[9]

The natural course for spinal cord cavernoma is not known. It is estimated that the annual risk of hemorrhage from intramedullary spinal cord cavernoma is about 1.6% per lesion per year. This is higher when compared with cerebral lesions in which the risk is 0.2-0.7% per year.[4]

Patients who undergo surgical excision of a CM need close follow-up. MRI 6 weeks to 6 months after surgery is required to evaluate the extent of resection and serve as a new baseline for comparison for future studies. Cavernous malformation can recur if not excised completely. Lesions that are radiation induced tend to recur. Familial cases are at risk to develop new lesions throughout their lifetime.[3] It is estimated that about 45% of patients with spinal cavernoma will also have an intracranial cavernoma, while only about 5% of patients with a intracranial cavernoma will have spinal cavernoma. This leads to a management rational that, if a spinal cavernoma is found, MRI of the head should also be done.[6]

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Pepto Bismuth Associated Neurotoxicity: A Rare Side Effect of a Commonly Used Medication

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Abstract

A 56 years old female with medical history significant for collagenous colitis and GERD for which she was taking Pepto Bismuth for months. She presented with progressive confusion for two weeks, followed by myoclonus, tremors, gait instability and visual hallucinations. Patient was admitted and comprehensive work up was done over a ten day course. This included a CBC, CCP, CT head, MRI brain, EEG, Lumbar puncture, and various antibody and serology testing which were all essentially unremarkable. It was noted that patient had been taking OTC Pepto Bismuth chronically for GI symptoms. Based upon the unrevealing work up, serum and urine samples for Bismuth levels were sent and returned markedly positive in both samples. Bismuth was held on admission and over the ten day hospitalization, patient showed gradual improvement of her cognitive function. She also showed resolution of her abnormal movements, myoclonus and visual hallucinations. Her gait continued to improve and required extended period of physical therapy post discharge. Her subsequent follow up visits showed resolution to baseline at four months post discharge.

Introduction

Bismuth preparations are commonly used over-the-counter medications for various gastrointestinal symptoms. Generally speaking, they are considered to be safe. However, multiple literature sources have reported significant neurological side effects. These include mental status changes (i.e., memory loss, confusion, delirium, psychosis, depression) and abnormal movements (i.e., ataxia, tremors, myoclonus, seizures).[1]The first cases of Bismuth encephalopathy were reported in 1974 by Burns et. al. Many more cases have been reported, especially in France where the condition has almost been recognized as an epidemic.[2] We describe a case of encephalopathy associated with prolonged use of Pepto Bismuth.

Case Report

A 56 year old female with no known past neurologic history presented with two weeks of progressive confusion and difficulty concentrating. This was followed by myoclonic jerks, hand tremors, gait instability, and visual hallucinations which were all noted upon presentation. Her past medical history includes collagenous colitis, irritable bowel syndrome, hypothyroidism, hypoparathyroidism, hypertension, GERD and depression/anxiety. The patient reported taking Pepto Bismuth 45ml three times a day for several weeks for chronic GI symptoms. Two days prior, Eszopiclone was changed to Quetiapine to aid her sleep. She was also taking Remifenim (Over the counter medication containing Black Cohosh) for post-menopausal symptoms. This was held for concerns of serotonin syndrome; however no changes were noted. No other recent changes in her medications were made.

On clinical examination, the patient was awake but disoriented with poor attention span. Vital signs were: temperature 37.0 C, blood pressure 165/94 mmHg, pulse 117 per minute, respirations 16 per minute and saturation of 98% on room air. She had frequent myoclonic jerks and tremors on intention. She had increased tone in all muscle groups. Deep tendon reflexes were increased and her planters were up going. The patient's speech was slow, but not dysarthric and cranial nerves were intact. Significant gait instability with ataxia was noted as well.

During admission patient had comprehensive work up for various possible etiologies for her encephalopathy. A complete blood count, complete comprehensive metabolic panel, thyroid function test and ammonia level were essentially unremarkable. A CT scan and subsequent MRI brain scan were negative as well. Neurology was consulted for further recommendations. Electroencephalography was done and showed moderate encephalopathy of non-specific etiology with no signs of seizures or spikes to suggest Creutzfeld-Jakob disease. A lumbar puncture (LP) performed was essentially negative for infection, only mildly elevated protein. The LP analysis showed glucose 68mg/dl, protein 56 mg/dl, WBC 5 cells, and RBC 3 cells. This also included negative cultures (including AFB, bacterial and fungal), negative HSV PCR, CSF RPR, VDRL, and cryptococcal antigen. Other lab data included serum B12 769 pg/ml (Normal range 246-911 pg/ml), TSH 1.074 mIU/ml (normal range 0.370- 4.420), free T4 1.41ng/dl (normal range 0.75- 2.00ng/dl), and ESR 21mm/hr (normal range 0-20 mm/hr). Further antibody and

serology testing of anti microsomal antibodies, thyroglobulin, Lyme serology, and perineoplastic antibodies were negative.

Duloxetine and Escitalopram were held upon admission for concerns about serotonin syndrome. In light of the unrevealing work up thus far, a toxicology consult was obtained. They suggested toxic etiology possibly related to Bismuth. Therefore, a blood sample to check for Bismuth level was sent and Bismuth blood level was 397 µ/L (normal range 0-9 µ/L).

While holding Bismuth over the 10 day hospital course, the patient became more alert, less somnolent and showed improvement in her cognitive function. Although she did not return to her baseline by this point, her myoclonus, rigidity and visual hallucinations had resolved. She was discharged on day 10, with significant improvement. She was discharged to an inpatient rehab facility as her gait was not entirely back to baseline. Patient continued to show improvement in her cognitive and motor functioning on subsequent office follow up visits. She continued to have home physical therapy for an additional two months after the short, skilled nursing stay. Four months post discharge patient showed complete resolution of her symptoms with the exception of fine residual tremors.

Discussion

Bismuth toxicity is known to cause subacute progressive encephalopathy associated with abnormal movements such as myoclonus and ataxia as seen in this patient. The mechanism of this toxicity is not known. Bismuth salts are absorbed in the GI tract in small quantities. Bismuth has a half-life of 20-30 days in the blood. Its long half life is due to the storage in multiple organs, such as kidneys, lung, spleen, liver, brain, muscle and enterohepatic circulation. KrügerG et.al. suggested that Bismuth can cross the blood brain barrier. This subsequently causes reduction of the oxidative decarboxylation of pyruvate resulting in decreased utilization of oxygen and glucose.[3] The patients suffering from encephalopathy had Bismuth daily during a period varying from 3 months and twenty years.[4]

There are two phases of encephalopathy associated with Pepto Bismuth use:[2,4]

1. Prodromal phase: This is a slowly progressive phase over 2-8 weeks and characterized by various neuro-psychological symptoms. These include asthenia, depression, headaches, gait disturbances, lack of concentration and memory impairment.
2. Acute phase: A more rapidly progressive phase characterized by

severe confusion, hallucinations, ataxia, dysarthria, myoclonic jerks and rarely seizures.

Upon discontinuation of Bismuth, complete resolution of symptoms typically takes six to twelve weeks. Abnormal movements, agitation and hallucination disappear first, while confusion and ataxia disappear later.[4] In severe cases, chelation with BAL (Dimercaprol or British anti-lewisite) caused more rapid recovery.[5] There are few case reports of Bismuth toxicity resulting in fatal outcomes. J.L. Liessens et.al. reported a fatal case of toxic encephalopathy due Bismuth toxicity with autopsy findings of non-specific axonal lesions including a widespread loss of Purkinje cells in the cerebellum.

Bismuth neurotoxicity can cause memory impairment and can be misdiagnosed as Alzheimer's dementia. The possibility of Bismuth encephalopathy needs to be considered in the differential diagnosis of possible Alzheimer dementia.[6] Rapid decline in memory associated with myoclonus and ataxia points more toward the diagnosis of Bismuth encephalopathy rather than Alzheimer's dementia.

Our patient progressed in a typical way with slow decline in her cognitive function characterized by lack of concentration and confusion over a few weeks. She was then severely confused and was hospitalized. She had hallucinations and abnormal movements as described above in the acute stage of her disease. Upon discontinuation of Bismuth she showed classical resolution of symptoms. This goes in line with previously published reports about Bismuth toxicity.

We believe that this presentation helps to increase physicians' and patients' awareness about this widely used medication, and the potential serious side effects associated with misuse of Pepto Bismuth.

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Electronic Environmental Reporting Project

Information previously unavailable to the public and to the medical community will soon enhance the connection between public health and clinicians.

The Centers for Disease Control awarded the West Virginia Bureau for Public Health (WVBHP) a grant in 2011 to strengthen the infrastructure of the 49 local health departments (LHD) in West Virginia. One component of the grant was funding for an electronic environmental health reporting system that would provide the LHDs the capability to perform and record inspections and investigations of their regulated facilities as well as document animal bite encounters.

Following research of environmental health electronic reporting systems that would allow for on-site inspections, activity tracking, and billing/invoicing, for all state-mandated environmental health programs and completion of the procurement process, HealthSpace, USA, Inc. was awarded the project in December 2011. The deployment of the software was divided into three phases. Phase I of the project began in January 2012. Twenty-six (26) local health departments are currently using the software that is powered by a tablet computer. The implementation schedule calls for all forty-nine (49) local health departments to be using the software by January 2014.

Before the implementation of this software program, there was no standard method of storing and filing environmental health data. Some environmental health records were in paper form, other LHDs were using various software programs to track permits and inspections. With the new software, facility information is stored consistently, creating an additional foodborne illness outbreak investigation tool. This tool will include the tracking and monitoring of foodborne illness complaints reported to environmental health at the LHD. This software will provide the ability to monitor major foodborne illness risk factors. It will also provide an improved ability to identify trends as related to outbreaks and more readily connect outbreaks that cross jurisdictional boundaries, thus, allowing for more timely notification to the medical community. Additionally, the data available will include animal bite encounters. The access to more timely and patient specific information may be helpful to the clinician in assessing the need for post-exposure prophylaxis.

Any clinician with an internet connection will have the ability to access relevant public health information. A physician would benefit from collaboration from public health by obtaining information, such as foodborne illnesses, in a timely and more pertinent fashion.

Fred R. Barley, R.S.
Electronic Environmental Reporting Program Manager

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HHS finalizes the rule guaranteeing 100 percent funding for new Medicaid beneficiaries

Health and Human Services (HHS) Secretary Kathleen Sebelius announced March 29, 2013, a final rule with a request for comments that provides, effective January 1, 2014, that the federal government will pay 100 percent of the cost of certain newly eligible adult Medicaid beneficiaries. These payments will be in effect through 2016, phasing down to a permanent 90 percent matching rate by 2020. The Affordable Care Act authorizes states to expand Medicaid to adult Americans under age 65 with income of up to 133 percent of the federal poverty level (approximately $15,000 for a single adult in 2012) and provides unprecedented federal funding for these states.

"This is a great deal for states and great news for Americans," HHS Secretary Kathleen Sebelius said. "Thanks to the Affordable Care Act, more Americans will have access to health coverage and the federal government will cover a vast majority of the cost. Treating people who don't have insurance coverage raises health care costs for hospitals, people with insurance, and state budgets."

Today's final rule provides important information to states that expand Medicaid. It describes the simple and accurate method states will use to claim the matching rate that is available for Medicaid expenditures of individuals with incomes up to 133 percent of poverty and who are defined as "newly eligible" and are enrolled in the new eligibility group. The system is set up to make eligibility determinations as simple and accurate as possible for state programs.

Under the Affordable Care Act, states that cover the new adult group in Medicaid will have 100 percent of the costs of newly eligible Americans paid for by the federal government in 2014, 2015, and 2016. The federal government's contribution is then phased-down gradually to 90 percent by 2020, and remains there permanently. For states that had coverage expansions in effect prior to enactment of the Affordable Care Act, the rule also provides information about the availability of an increased FMAP for certain adults who are not newly eligible.

The rule builds on several years of work that HHS has done to support and provide flexibility to states' Medicaid programs ahead of the 2014 expansion, including:

- 90 percent matching rate for states to improve eligibility and enrollment systems;
- More resources and flexibility for states to test innovative ways of delivering care through Medicaid;
- More collaboration with states on audits that track down fraud; and
- Specifically outlining ways states can make Medicaid improvements without going through a waiver process.

For more information on the improvements made to Medicaid, please visit: http://www.medicaid.gov/State-Resource-Center/Events-and-Announcements/Downloads/MMF_Jan-Dec-2012_FINAL.PDF

For the full text of the final rule, please go to http://www.ofr.gov/inspection.aspx.

CMS Is Now Accepting 2014 ePrescribing Hardship Requests to Avoid Penalties

Physicians will receive a 2 percent penalty in 2014 if they do not meet the requirements of the Medicare ePrescribing (eRx) program this year, meet one of the limited exemption categories, or obtain approval for a hardship exemption.

From March 1 through June 30, 2013, CMS has re-opened the Quality Reporting Communication Support Page to allow physicians to request a hardship exemption for 2014.

Physicians who do not meet one of the exemption or hardship categories must: (1) report the eRx measure via claims (10 eRx events for individual physicians and larger numbers for groups using GPRO); or (2) register for the meaningful use (MU) of electronic health records (EHR) incentive program by June 30, 2013; or (3) achieve MU under the EHR Incentive Program during one of the reporting periods needed to avoid an eRx penalty in 2014.

For additional information on the 2014 eRx penalties and how to avoid them, review CMS' 2014 eRx Payment Adjustment Fact Sheet on the cms website, www.cms.gov.

Congratulations 2013 Certified Medical Office Manager (CMOM) Class!

by Barbara Good, CMC, CMOM, CMCO
Physician Practice Advocate, WVSMA

Mark Blake	***Huntington, WV***	**Wanda Lowers**	***Parkersburg, WV***
Amy Callihan	***Charleston, WV***	**Joyce McClung**	***Summersville, WV***
Sandra Calvert	***Kingwood, WV***	**Lynn McCormick**	***Parkersburg, WV***
Amanda Clark	***Martinsburg, WV***	**Floyd Metzger**	***Huntington, WV***
Terri Frye	***Charleston, WV***	**Mark Nelson**	***Charleston, WV***
Susan George	***Charleston, WV***	**David O'Dell**	***Huntington, WV***
Robin Hoblizell	***Charleston, WV***	**Michelle O'Dell**	***Charleston, WV***
Patricia Kelly	***Parkersburg, WV***	**Diana Withrow**	***Charleston, WV***

Sixteen office administrators and managers from across West Virginia recently completed the 2013 Certified Medical Office Manager (CMOM) class. The class, taught by Practice Management Institute's faculty instructor Rose Moore, was facilitated by WVSMA Physician Practice Advocate, Barbara Good.

Attendees gave high praise for both the course material and instructor. Class members now await the results of their certification exam before receiving the prestigious Certified Medical Office Manager credential.

In addition to the excellent instruction, the four day course afforded everyone a terrific opportunity to network with fellow management professionals.

Class members acknowledged that they learned much more information from the CMOM course than they could have anticipated. Said one attendee, "CMOM is essential to both experienced and less experienced practice managers. The class content contains an extensive overview of regulations and guidelines, as well as best practices for all medical offices". Another attendee wrote "Even if you have been in healthcare management all your life; this class is a must in order to keep abreast of ongoing changes in healthcare!"

One attendee waited three years to take the course and was glad to have taken it. She is now preparing to take other PMI certification courses.

To sum it all up, one attendee stated "After 20 years in the medical field, I realized how much I still didn't know. This class is a wonderful learning experience, and I highly recommend it!"

If you are interested in future certification courses for yourself or your practice manager, please contact Barbara Good at 304-925-0342, ext. 11 or barbara@wvsma.org.

WVU Health Sciences faculty, students go global

Providing care in exotic, remote locales

During the month of April, 17 WVU Health Sciences students are travelling to locations around the world to gain knowledge about other cultures and what those cultures can teach them about people, healthcare and problem solving.

WVU faculty preceptors are accompanying students rotating in Barbuda, China, Fiji and Ghana, while Amizade Global Service Learning is facilitating the rotation in Brazil – a first for the WVU Global Health Program.

"Global health rotations are a great opportunity for inter-professional education," Global Health Program Director Melanie A. Fisher, M.D., M.Sc., said. "Medical students get to work side by side with dental students, public health students, nursing students and pharmacy students. That's a theme of the future – healthcare is a team effort."

Based in the Amazon city of Santarém, the Brazil rotation provides inter-professional experiences for four medical students and one dental student. WVU School of Medicine Professor Christopher J. Martin, M.D., M.Sc., explained that in addition to providing care at hospitals and clinics, students work aboard a river boat.

"They are serving native people on a boat," Dr. Martin said. "For many people, this is the only care they get. Students will live and work on the boat. What better way to promote inter-professional education than to be in close quarters?"

WVU School of Medicine fourth-year student Sunjay Mannan, who is rotating in China for the month of April, was eager for the opportunity to sharpen his problem-solving skills.

"Most eastern cultures respect the condition of the group over that of the individual," Mannan said. "So I am looking forward to seeing how individuals in China deal with their problems, while they try to preserve the integrity of their respective groups."

Students expect to encounter challenges such as language barriers, bumper-to-bumper traffic and lacking healthcare resources. They also are anticipating stark differences, given the excess we often experience in American culture.

Medical student Alison Spiker, who is serving in Fiji with her husband Grant Morris, said they have gotten "hooked" on international medical work, an occurrence that Fisher said is common among students in the Global Health Program. Spiker and Morris plan to continue in the global health field throughout their lives.

"Professionally, I can see this experience as enhancing my understanding of the logistics required to plan and execute such a grand endeavor," Spiker noted. "I will, undoubtedly, grow on a personal level from my experience in Fiji. Self-reflection will definitely occur while abroad, and my husband and I will certainly come home with refined goals for our life in medical mission work."

Follow WVU Global Health on Twitter to keep up with the global health rotations in Barbuda, Brazil, China, Fiji and Ghana.

WVU School of Medicine students and husband and wife team, Grant Morris and Alison Spiker, pack medical supplies in preparation for their rotation in Fiji.

Funding for collaborative medical research announced at Marshall University

Marshall University Joan C. Edwards School of Medicine officials announced this spring $150,000 in funding for six research grants associated with the school's translational medicine research program.

The Marshall Health Translational Pilot Grant program, created in 2012, encourages collaborative research between basic scientists and clinical physicians in an effort to speed up the process of laboratory discovery to clinical application for patients. The grants are funded by Marshall Health, which is the faculty practice plan for the School of Medicine and supports the clinical, educational, research and services missions of the school.

Beth Hammers, executive director of Marshall Health, says the pilot grant program provides one year of support at $25,000 for each grantee, with additional funding based on progress of the research.

"Medical research is essential to the development of new medical treatments and cures for patients," Hammers said. "We are thrilled to help stimulate a robust, viable grant program which pairs basic scientists from Marshall University with School of Medicine physicians to work on projects which will lead to the betterment of our community."

The investigators and their projects are listed below:

- Dr. Pier Paolo Claudio, Department of Biochemistry and Microbiology, and Dr. Anthony Alberico, Department of Neuroscience – "Chemotherapy resistance and sensitivity testing in tumors of the central nervous system"
- Dr. Elaine Hardman, Department of Biochemistry and Microbiology, and Dr. James Jensen, Department of Surgery – "Feasibility and Safety of Nutritional Supplementation with Omega-3 Fatty Acids to Reduce Prostate Specific Antigen Rise in Men with Biochemical Failure after Prostatectomy or External-Beam Radiotherapy"
- Dr. Nalini Santanam, Department of Pharmacology, Physiology and Toxicology, and Dr. Paulette Wehner, Department of Cardiology – "Perivascular Fat Relation to Hypertension—Appalachian Heart Study"
- Dr. Nalini Santanam, Department of Pharmacology, Physiology and Toxicology, and Dr. Abid Yaqub, Department of Medicine, Endocrinology Section – "Impact of Technology-based Behavioral Intervention on Molecular and Clinical Parameters in Patients with Type 2 Diabetes"
- Dr. Monica Valentovic, Department of Pharmacology, Physiology and Toxicology, and Dr. Brenda Dawley, Department of Obstetrics and Gynecology – "Prenatal Exposure to Heavy Metals and Polycyclic Aromatic Hydrocarbons (PAH) Alter Umbilical Cord Blood Levels of Thyroid Hormone and Vitamin D"
- Dr. Hongwei Yu, Department of Biochemistry and Microbiology, and Dr. Yoram Elitser, Department of Pediatrics – "Investigate the distribution of segmented filamentous bacteria (SFB) in American children and the presence of SFB with childhood diseases"

Other current translational research under way at the School of Medicine includes a partnership with the University of Kentucky as part of the National Institutes of Health's Clinical and Translational Science Awards program, which also is aimed at speeding the time for laboratory discoveries to benefit patients.

Match 2013

Marshall University's 73 graduating medical students learned their residency placements on "Match Day" in March.

Just over 55 percent of graduating seniors will enter fields defined as primary care in West Virginia – family medicine, internal medicine, obstetrics/gynecology, internal medicine/pediatrics and pediatrics – continuing Marshall's mission of educating physicians for the nation's rural areas. Additionally, about 40 percent of the class will remain in West Virginia, with 25 new doctors training at Marshall.

Dr. Marie Veitia, assistant dean of student affairs, said this year's match is a strong one for Marshall students.

"We are delighted that our students matched into highly competitive fields of medicine such as anesthesiology, radiology, orthopaedics and ophthalmology at programs across the country," Veitia said. "Marshall students are heading to programs at Yale, Wake Forest and the University of California-Davis."

WVSOM recognized as top medical school for 15th year

The West Virginia School of Osteopathic Medicine (WVSOM) continues to be recognized as one of the nation's top medical schools for rural medicine.

The institution is ranked No. 9 in rural medicine by the U.S.News & World Report "America's Best Graduate Schools" 2014 annual publication. WVSOM is also ranked No. 13 in family medicine. The rankings recognize institutions that offer top programs in business, law, medicine, engineering and education, among other specialties. This is the 15th consecutive year WVSOM's medical programs have received recognition.

"A commitment to educating primary care physicians who will serve in rural areas is at the heart of WVSOM's mission," said President Michael Adelman, D.O., J.D. "WVSOM's recognition as a top medical school in rural and primary care speaks to the dedication of the school's faculty and staff to delivering an education which will enable our students to provide holistic, compassionate and capable care to their future patients."

Among all medical schools in the nation, WVSOM is No. 3 in the percentage of graduates entering primary care residences from 2010 to 2012.

Medical school deans and senior faculty from across the country rate the educational programs and determine specialty rankings. Results were calculated from a survey of 149 accredited M.D. and D.O. medical schools across the country.

Governor proclaims April 3 WVSOM Day

Recognizes institution's contributions to the state

Governor Earl Ray Tomblin recognized the West Virginia School of Osteopathic Medicine's efforts in educating lifelong learners and providing excellence in medical education. The governor presented a proclamation formally declaring April 3, 2013 as "West Virginia School of Osteopathic Medicine Day" in the state because of the school's mission to serve West Virginia and provide quality health care for its residents.

"West Virginia is home to hundreds of osteopathic doctors and we're blessed with an outstanding training facility — the West Virginia School of Osteopathic Medicine," the governor said to an intimate group of WVSOM representatives. "The impact of the school and its doctors truly stretches across the state."

WVSOM has produced more than 2,500 osteopathic physicians, many of whom practice as primary care and family medicine physicians. Tomblin said he is especially thankful to the physicians who provide service in rural communities.

"I want to recognize President Dr. Michael Adelman and Dr. Rodney Fink, chairman of WVSOM's board, and the many faculty members and administrators who have worked hard to provide our doctors with the knowledge and skills needed to bring patient-centered, evidence-based medicine to West Virginia," he said.

Governor Tomblin delivered the framed proclamation to Adelman and Fink, with applause from guests.

"WVSOM is proud of the physicians we have trained during the past 40 years of our existence and we continue to be committed to training physicians who provide service to West Virginia," Adelman told the governor. "We thank you for this honor. It is deeply appreciated."

The proclamation wasn't the only recognition WVSOM received at the state capitol on Wednesday. Senate Resolution No. 55 recognizes the West Virginia School of Osteopathic Medicine for excellence in medical education and its many contributions to the state of West Virginia. The Senate adopted the resolution on April 3 with support from 14 senators.

There Is Power in Numbers

The West Virginia State Medical Association (WVSMA) appreciates the confidence and support from the following **Group Practices** who have already established their 2013 WVSMA membership. It is our privilege to serve you! We look forward to being your advocate in 2013 and beyond!

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Obituaries

The WVSMA remembers our esteemed colleagues...

Clyde A. Burgess, MD

Clyde A. Burgess, MD, 82, of Green Bank, W.Va., went home to the Heavenly Father on February 14, 2013.

Born May 3, 1930, in Oak Hill, W.Va., he was the son of the late Clyde Austin and Marshie Honaker Burgess.

He graduated from Oak Hill High School in 1948 and attended the University of Dayton and graduated from the University of Cincinnati School of Medicine in 1961.

He served in the US Air Force during the Korean Conflict rising to the rank of Staff Sergeant and retired from the US Army as a Colonel in 1996.

Dr. Burgess practiced medicine in Berkley Springs, Philippi and Clarksburg, W.Va. and Ashland, Ky. as well as serving with the US Army in Alaska and Kentucky. He retired to the beautiful mountains of Pocahontas County and attended Hebron Baptist Church in Green Bank.

Dr. Burgess is survived by his wife of 59 years, Christine Haga Burgess. Other survivors include a son, Andrew (Linda Gale) Burgess of Fairbanks, Alaska; a daughter, Kimberly Burgess, MD of Charleston, W.Va.; and a sister, Kathryn Burgess Terwilleger of Willow Street, PA.

In lieu of flowers, the family requests donations to the Gideons or a charity of your choice.

William E. Gilmore, MD

Dr. William Edmund Gilmore, 94, of Whispering Pines, died Tuesday, Feb. 12, 2013, in Pinehurst, surrounded by his loving family.

Dr. Gilmore was born Sept. 22, 1918, in Wheeling, W.Va., son of the late Dr. and Mrs. John W. Gilmore. He graduated from West Virginia University and the University of Wisconsin-Madison School of Medicine and Public Health. He completed his internship at Philadelphia General Hospital prior to being commissioned as a Lieutenant Junior Grade in the U.S. Navy and serving in both the Atlantic and Pacific theaters during World War II. He then completed his residency at the University of Wisconsin-Madison before establishing his practice in general and thoracic surgery in Parkersburg, W.Va., from 1950-1987.

Dr. Gilmore's distinguished career included leadership roles in several professional organizations, such as governor of the West Virginia Chapter of the American College of Surgeons; president of the West Virginia State Medical Association; president of the medical staffs at Camden-Clark Memorial and St. Joseph's hospitals in Parkersburg; president of the Parkersburg Academy of Medicine; and clinical professor of surgery, West Virginia University School of Medicine. The University of Wisconsin Medical Alumni Association honored Dr. Gilmore in 1993 with the Ralph Hawley Distinguished Service Award.

Dr. Gilmore is survived by his wife of 69 years, Maxine Merrill Gilmore, of Whispering Pines; their five children and spouses, including Susan G. Mouyal (and Pierre), of Atlanta, William E. Gilmore Jr. (and Mary Lee), of Southern Pines, Dr. John W. Gilmore II, of Palm Springs, Calif., Betsy G. Balassone (and James), of Los Altos, Calif., and Scott M. Gilmore (and Kimberly), of Charlotte; and five grandchildren, Merrill W. Balassone, of Los Angeles, Elizabeth G. Balassone, of San Francisco, James M. Balassone II, of Los Altos, Mackenzie S. Gilmore, of Charlotte, and Katherine A. Gilmore, of Charlotte.

In lieu of flowers, memorial donations may be sent to the charity of the donor's choice in honor of Dr. William E. Gilmore.

Rene Octaviano Sullesta, MD

Rene Octaviano Sullesta, M.D., of Charleston, went into the grace of Heaven on March 11, 2013, after a battle against cancer.

He received a doctor of medicine degree from the University of Santo Tomas, Manila, Philippines. He did post-graduate work at Frankford Hospital, Philadelphia, Pa. He practiced urology for 30 years and was on staff at Charleston Area Medical Center and St. Francis Hospital. Professional affiliations included fellowship in the American College of Surgeons, American Urological Association, West Virginia State Medical Association and Tri-State Filipino-American Association.

In his spare time Rene enjoyed tending his garden, fishing and singing in the church choir. A true believer in God, he did not fear death, instead looking forward to eternal salvation in Christ.

Surviving are his wife, Joan; son, Michael (Ashley) of Wheeling; daughter, Rebecca (Brian) of Washington, D.C.; sister, Enriqueta Sullesta-Sason (Jan); sister, Alicia Velasco (Nono); niece, Ann Velasco; and nephew, John Mark Velasco, all of the Philippines.

In lieu of flowers, the family suggests donations may be made in memory of Rene to a charity of your choice.

2013 WESPAC Contributors

The WVSMA would like to thank the following physicians, residents, medical students and Alliance members for their contributions to WESPAC. These contributions were received as of April 15, 2013:

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WESPAC is the West Virginia State Medical Association's bipartisan political action committee. We work throughout the year with elected officials to make sure they understand the many facets of our healthcare system.

WESPAC's goal is to organize the physician community into a powerful voice for quality healthcare in the West Virginia Legislature. We seek to preserve the vital relationship between you and your patients by educating our legislators about issues important to our physicians.

WESPAC contributions provide critical support for our endorsed candidates. Your contribution can make the difference between a pro-physician/patient candidate winning or losing.

To make a contribution to WESPAC, please call (304) 925-0342, ext. 12

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Please direct all membership inquiries to: **Mona Thevenin, WVSMA Membership Director at 304.925.0342, ext. 16 or mona@wvsma.org.**

The Advantages of Purchasing Disability Insurance from the West Virginia Medical Insurance Agency

WEST VIRGINIA MEDICAL INSURANCE AGENCY
"Meeting the insurance needs of physicians"

by: Steve Brown, Agency Manager

In 2011, the West Virginia Medical Insurance Agency entered into an arrangement with Ameritas Life Insurance Corporation (then Union Central Life Insurance Company) to provide a 15% premium discount for physicians who are members of the West Virginia State Medical Association and purchase their disability insurance from Ameritas through the West Virginia Medical Insurance Agency.

In the last year the availability of the Ameritas discount has been expanded to members of the West Virginia Academy of Family Physicians (WVAFP), the West Virginia Medical Group Managers Association (WVMGMA) and the Office Managers Association of Health Care Providers (OMA).

This article was designed to pinpoint the areas where the West Virginia Medical Insurance Agency's Ameritas disability insurance product is most beneficial to individual physicians and how it can be expanded, if necessary, by the addition of group disability insurance benefits from Sun Life Financial to make our offering a superior disability insurance program.

Note the following points of emphasis for individual physician purchases of disability insurance:

1. SPECIALTY OWN OCC DEFINITION OF DISABILITY: As a physician, if you have limited your duties to the performance of the usual and customary functions of a specific, professionally recognized medical specialty, that will be considered your occupation.

2. BEST RESIDUAL/RECOVERY BENEFIT IN THE INDUSTRY: Critical for physicians. Upon recovery and return to work in your occupation on a FULL TIME BASIS, a residual benefit will be paid if you maintain a loss of at least 15% of your net earned income and that loss is a direct result of your previous disability. This benefit may be paid for the REMAINDER OF THE INSUREDS BENEFIT PERIOD as long as the insured maintains at least a 15% loss in their pre-disability net income.

3. INDUSTRY EXCLUSIVE FEATURES AT NO ADDITIONAL COST:

a. Non-disabling Injury Benefit: Pays the insured a benefit for expenses caused by injuries or damage to natural teeth - up to 50% of your basic benefit, not to exceed $3000 PER INJURY. This benefit does NOT coordinate with, nor is reduced by, payments you may have received from medical insurance.

b. Good Health Benefit: Reduces the elimination period two days for each year you do not receive a monthly disability benefit under the policy. The Non-disabling Injury Benefit does NOT affect the Good Health Benefit. EX: If you did not receive benefits under the policy for 15 years, your elimination period would be 30 days shorter: i.e. a 90- day EP would be reduced to a 60 day EP.

c. COBRA Premium Benefit: This benefit reimburses the insured for COBRA health insurance premiums up to $1000 per month for a maximum of 18 months.

4. BUSINESS OVERHEAD EXPENSE POLICIES: this policy is designed to help pay office operating expenses, including staff salaries, when the insured is off on a claim. The Salary Substitute Rider provides ADDITIONAL cash for the first six months of the claim for purposes of hiring another medical professional to continue to see your patients in your office.

Note the following features which allow us to expand our individual disability product to a superior disability insurance program by adding group benefits through Sun Life Financial:

1. GUARANTEE ISSUE WITH NO MEDICAL QUESTIONS. For groups of 5 or more employees guarantee issue coverage of 5,000 to 20,000 a month.

2. NO OFFSET FOR INDIVIDUAL DISABILITY INSURANCE POLICIES.

3. SPECIALTY OWN OCCUPATION PROTECTION. Board certification is not required

4. BUSINESS PROTECTION COVERAGE. Pays a benefit to the practice if a physician or key employee is disabled, with no expense verification required.

5. COVERAGE CANNOT TERMINATE FOR THE FOLLOWING:

a. Physician moves or resides outside of the USA while on claim

b. Physician can work part-time but refuses to do so.

6. INCOME LOSS TEST. No Income loss is required during the elimination period and income from procedures performed before the disability are not included.

Disability insurance is essential for physicians and we are able to design a plan specifically to meet your needs. To have a no-cost, no-obligation evaluation of your disability insurance needs call Steve Brown, agency manager, at 1-800-257-4747 Ext 22 (304-925-0342 Ext 22) or 304-542-0257 (cell).

Disability Insurance

Do I Need It?

"The Social Security Administration estimates that 3 out of 10 Americans will become disabled before they retire." *

+

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*Source: CDA 2010 Consumer Disability Awareness Study

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ORIGINALITY: Articles submitted for publication become the sole property of the West Virginia Medical Journal. Prior publication is unacceptable. The Publications Committee reserves the right to edit any material submitted. Scientific articles are to be prepared in accordance with the "Uniform Requirements for Submission of Manuscripts to Biomedical Journals." Please go to www.icmje.org for complete details.

AUTHORS: A cover letter from the corresponding author, complete with physical mailing address and email address, should be submitted with the manuscript. Persons listed as authors should have participated sufficiently in the work to take public responsibility for the concept. No more than six authors will be listed. Other contributors may be recognized in an acknowledgement.

FORMAT: Submit articles by email or on CD. Microsoft Word is preferred, but other programs are acceptable. All tables or figures should be created separately from the body of the manuscript as .tif, .jpg or .pdf files in a high resolution format with the corresponding file names such as, Table 1, Figure 1, etc. Legends should be included for all tables and figures.

STYLE: Manuscripts are to be limited to 2500 words and approximate the style adopted by the American Medical Association as illustrated in JAMA and detailed in the AMA's Manual of Style. An abstract of 150 words or less should accompany each manuscript, stating the exact question considered, the key points of methodology, key findings, and the conclusion directly supported by the findings.

REFERENCES: References should be prepared in accordance to the "American Medical Association Manual of Style." These instructions for authors are available online at www.jama.com. If a manuscript contains more than 20 references, the additional references may be abridged due to space constraints. In this case, a notation to contact the authors for the complete list is published at the end of the article.

PHOTOGRAPHS: Photos are printed in black and white. Please submit digital files either from a digital camera or scan at 300 dpi at 100%. Use arrows to point to areas of interest.

NOTE TO AUTHORS: The WV Medical Journal inside pages traditionally print in black and white. If authors wish to have photos and figures printed in color, there is a $1,100 charge per article to help defray the printing costs to the Association. Please indicate your preference when submitting an article. If your article is accepted for publication, you will be invoiced for the charges in advance of publication.

Please send articles to the managing editor via email: angie@wvsma.org. For additional information, contact Angela L. Lanham, Managing Editor, at (304) 925-0342, Ext. 20.

INSTRUCTIONS SPECIFIC TO CASE REPORTS

1. The WVMJ will consider case reports that will remind readers of important clinical lessons, shed light on the possible pathogenesis of a disease, prevent errors, describe unusual presentations, do away with misconceptions, present a rare disease or problem in context, describe a novel procedure or treatment, describe unusual associations of symptoms or diseases, describe unexpected outcomes, or present information that make a clear point useful to the readership.

2. A cover letter to the editor must accompany the manuscript, listing
   a. How this report will advance the understanding of a disease, drug or medical problem in general.
   b. Is this of interest to a particular specialty or to a broader clinical audience?

3. Case reports must be designed as follows:
   a. Abstract: 100-120 words listing what is being reported, the outcome and the lesson(s) learned.
   b. Introduction (180-220 words): a brief background leading to a statement of the paper's purpose. All the elaboration regarding the disease or clinical situation must not be presented in this section, and should instead be part of the discussion.
   c. Case presentation (400 words): orderly narrative (symptoms, signs, relevant exam, diagnosis, etc) with stated and clearly presented rationale for the course(s) of action taken.
   d. Discussion (350-600 words): relevant information about the disease or problem being presented, putting the case in context. A comparison with similar cases in the literature must be included, with such information presented—if possible—in table form.
   e. Conclusion (50 words): clearly state the main conclusions derived from this experience.
   f. References: Up to 20 references will be published, but if space is limited, additional references will be abridged. WVMJ will print a notation to the reader to contact the author for additional references.

4. Figures must depict valid information and have markers pointing to the area of interest. Submit only high quality photos and tables, which are large enough to fill a 2-3/8 inch space at 100%.

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Authors are required to submit a "Response to Reviewers" in a separate document, along with their revised manuscript.

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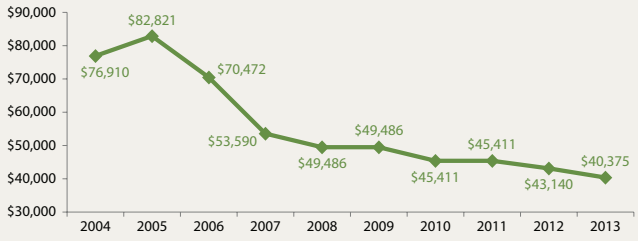

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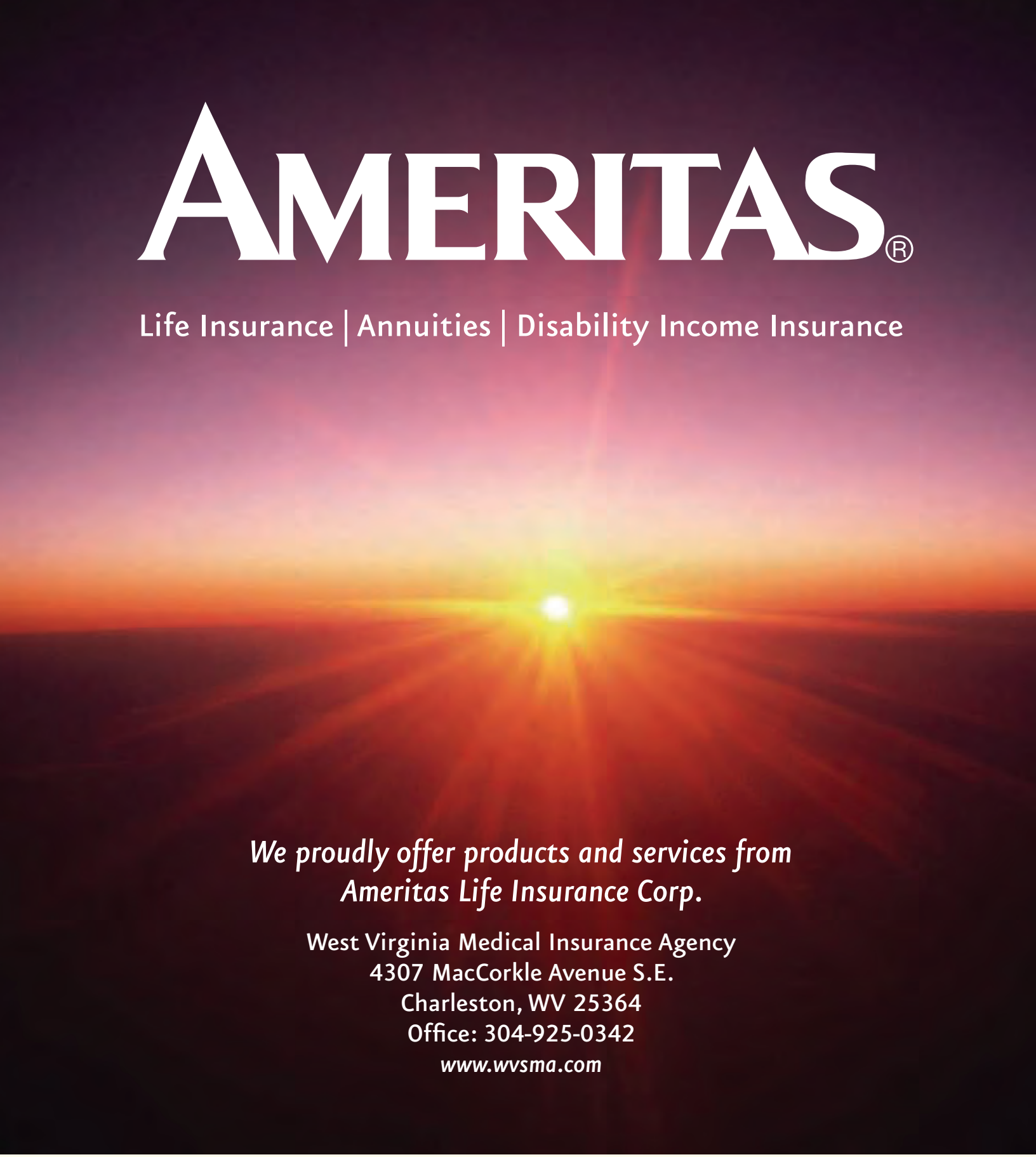

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REDI

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West Virginia Responder Emergency Deployment Information system

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Who can register?

- Registration is open to West Virginia's health and medical professionals, and others who live or work in West Virginia

How can I help?

- You can help by being willing to assist during a health related emergency or event and by registering in WV REDI

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- Please remember that "volunteer" truly means volunteer. You can choose, at any time, to decline any request that you receive for deployment

How do I register?

- To register go to **www.wvredi.org** and click on "register now"

Where do I get more information?

- For more information, call **304-558-6900 ext. 2009**

Register today to be prepared for tomorrow!

Visit the www.wvredi.org homepage and click on "register now."

WEST VIRGINIA Department of Health & Human Resources

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