OFP
Osteopathic Family Physician

THE OFFICIAL PEER-REVIEWED PUBLICATION OF THE AMERICAN COLLEGE OF OSTEOPATHIC FAMILY PHYSICIANS

May/June, 2016
Volume 8 | Number 3
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SUPPLEMENT 1

AMERICAN DIABETES ASSOCIATION

STANDARDS OF MEDICAL CARE IN DIABETES—2016

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Osteopathic Family Physician **is the ACOFP's official peer-reviewed journal.** The bi-monthly publication features original research, clinical images and articles about preventive medicine, managed care, osteopathic principles and practices, pain management, public health, medical education and practice management.

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Articles submitted for publication must be original in nature and may not be published in any other periodical. Materials for publication should be of clinical or didactic interest to osteopathic family physicians. Any reference to statistics and/or studies must be footnoted. Material by another author must be in quotations and receive appropriate attribution.

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We are seeking clinical images from the wards that covers essential concepts or subject matter to the primary care physician. Please provide a brief synopsis of how the case presented along with 1-4 questions and approximately 1 page of education with reference to the image and questions.

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- Advances in Skin Care Diagnosis & Treatment
- Anxiety *(with OMT treatment component)*
- Current Management of the Menopausal Woman *(with OMT treatment component)*
- Direct Primary Care: Emerging Practice Alternative
- Direct Primary Care: Legal Aspects
- Vaccinations: Getting Past the Misinformation & Reaching Patients
- Pediatric GI: Chronic Abdominal Pain Eval & Treatment
- Nausea with Vomiting
- Newborn Disorders & Nutritional Guidance
- Skin and Soft Tissue Infections: It's More than Just MSRA
- Insomnia *(with OMT treatment component)*
- Movement Disorders - Parkinson's Disease, Essential Tremor, Restless Leg Syndrome *(with OMT treatment component)*
- Patient Engagement *(Help define the science of engaged research, provide tangible examples of the impact of engaged research, or answer a question or controversy related to patient engagement.)*

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Family Medicine / OMT Certification / OCC Cognitive Exam	**Electronic Testing** Regional Sites September 24, 2016	**April 1, 2016** *Late fee through June 1*
	ACOFP Annual Convention	
Family Medicine / OMT Certification / OCC Performance Evaluation Only	**Kissimmee, FL** March 16 - 19, 2017 March 14 - 17, 2017	**October 1, 2016** *Late fee through December 1*
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Family Medicine / OMT Certification Cognitive Exam	**Electronic Testing** Regional Sites April 1, 2017	**October 1, 2016** *Late fee through December 1*
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	AOA OMED Conference	
Family Medicine / OMT Certification / OCC Performance Evaluation Only	**Philadelphia, PA** October 7 - 11, 2017 October 6 - 8, 2017	**April 1, 2017** *Late fee through June 1*

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MAY/JUNE, 2016
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INSTRUCTIONS FOR AUTHORS:

Articles submitted for publication must be original in nature and may not be published in any other periodical. Materials for publication should be of clinical or didactic interest to osteopathic family physicians. Any reference to statistics and/or studies must be footnoted. Material by another author must be in quotations and receive apporpriate attribution. ACOFP reserves the right to edit all submissions. To submit a manuscript or to access additional submission guidelines visit mc04.manuscriptcentral.com/ofp.

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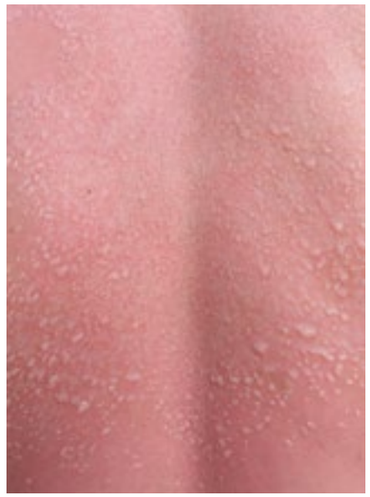

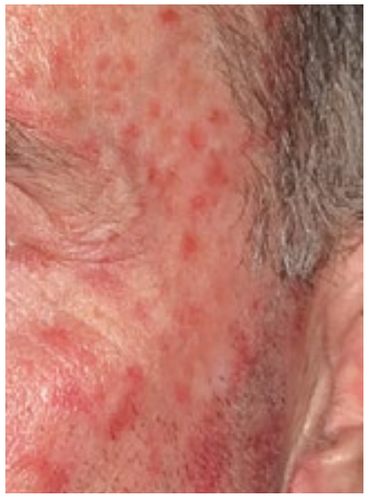

Osteopathic Family Physician

ACCEPTING SUBMISSIONS FOR THE SECTION TITLED “CLINICAL IMAGES.”

This section showcases clinical images from the wards that cover essential concepts or subject matter to the primary care physician.

Each installment of “Clinical Images” comprises 1 or 2 medical images along with a brief synopsis of how the case presented along with 1-4 questions and approximately 1 page of education with reference to the image and questions.

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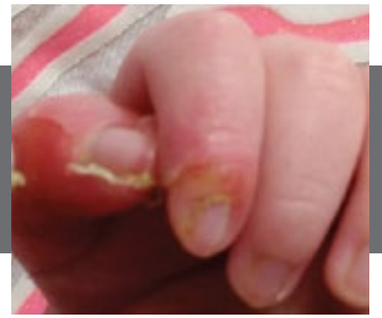

EDITOR'S MESSAGE

Sweet Summer

Amy J. Keenum, DO, PharmD, Editor, Osteopathic Family Physician

This month we serve up articles that relate to what we treat daily.

A review of the new American Diabetes Association guidelines appears this month. It brings a quick review of what we should be doing with a few changes. Setting goals for the individual is part of the new guidelines. Older patients, with shorter life expectancy may be at more risk of falling from hypoglycemia than from long-term risks of less than perfect Hba1c goals. One of the interesting points is that the eye exam can be done every 2 years if normal on 2 or more annual exams. This just makes sense, especially in well controlled, barely diabetic folks. This does not mean the insurance companies are on the same page. It has been said that it takes 9 months to birth a board question but how long does it take quality indicator monitors to get with the new guidelines?

Approaches to chest pain and COPD each have their own articles. Both discuss the underlying disease and the musculoskeletal components of the treatment with both osteopathic principles and manual treatments. COPD for example often involves coughing which leads to muscle pain and spasm. Chest pain can be from a primary musculoskeletal issue and the authors discuss evaluation and treatment.

Psoriasis diagnosis and treatment is reviewed. The article is well organized and easy to read. A patient of mine used a different treatment on each psoriatic plaque of his body to minimize exposure to steroids. Treatment can certainly be individualized to what works with the fewest side effects.

You may want to review pain management guidelines from the CDC March 2016; here primary care doctors take a big hit for controlled substance prescribing. There is no pain clinic prescribing controlled substances in my community how about yours? I would be happy to refer. It is challenge enough to treat the diabetes, hypertension and depression. Someone else can do better helping my patient with pain management? Where are you? Again, we try to do the best we can and for sure we can all try to do better.

We have had an uptick in submissions to the visual diagnosis column so after this issue we plan to run two per issue for a while.

Kids will be out of school soon and summer vacations underway.

Hope this one is your best vacation ever.

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Osteopathic Family Physician is looking for...

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CONGRATULATIONS

The journal of *Osteopathic Family Physician* applauds the following 2015 award recipients!

2015 OFP Attending Paper of the Year:

Osteopathic Considerations in the Management of Migraine in Pregnancy.

Sara Soshnick, DO; Christina Mezzone, DO; Sheldon Yao, DO; Reem Abu-Sbaih, DO

2015 OFP Resident Paper of the Year:

Dietary Supplements: Navigating the Pharmacologic Influence's of Nature's Medicine.

Andrew J. Kubinski, DO, MS

FROM THE PRESIDENT'S DESK

DO All the Good You Can DO

Larry W. Anderson, DO, FACOFP *dist*.
2016 - 2017 ACOFP President

From an early age I knew I wanted to be a physician. With the exception of my father, I'm the fifth generation of physicians who all practiced medicine in North Georgia. I'm the first DO in my family.

Becoming ACOFP's newest President is a defining moment for me and that is what I would like you to reflect on in your life. We all have them – defining moments – a point in time that determines the course of our lives.

A defining moment can be big or small. Maybe yours was when, as a child, you won a race or when you were bullied. Maybe as a student, you passed a big test or you failed a big test. Perhaps your defining moment was a failed romance.

One of my first defining moments happened when I was just 10 years old when I hurt my knee. My friend's father, who was not a physician, tended to my knee. After he looked it over for some time, he said I'd be fine.

At that moment, I thought he really cared about me. From that point on, I decided I would be a physician who touches his patients, who lets them know that he cares about them. To me, that's what it is to be an osteopathic family physician.

Many years later, after I had become an osteopathic family physician, I had another defining moment. I had been in practice about 10 years when a five-year-old boy came in as a new patient for his annual checkup. I noticed a heart murmur. His mother told me his pediatrician said not to worry and that he'd grow out of it.

I called a pediatric cardiac surgeon and presented my patient. It took three surgeons and more than three months to decide what would be the best procedure for this child because his heart condition was so rare. The surgery was successful and the child thrived.

This story is not about the child. It's about the mother. The look on her face when I said the other doctors who told he'd simply grow out of it were wrong and he needed immediate care was a look of sheer terror, as thoughts of losing her son came over her face. After the surgery, the thrill of happiness and joy of knowing that her son was going to be alright was a defining moment for me.

Serving in the military was another defining moment. It shaped my future, my respect for authority and my understanding that service often requires sacrifice – not always, but sometimes, even the sacrifice of one's own life.

That's why I asked Medal of Honor recipient, Colonel Bruce Crandall to be our Keynote Speaker at the ACOFP's Annual Convention. I wanted him to share his defining moments that were captured in the movie, We Were Soldiers, but also for us to see how that moment shaped his life for the decades that followed.

You will have defining moments in your careers when you save a life, make a cancer diagnosis that everyone else has missed, or diagnose a heart attack with minimal symptoms. When you do, you will realize that all the studying, tuition and sacrifices that you have made will be worthwhile.

My theme this year as President is "DO All the Good You Can DO." Everything starts and ends with being a DO. When you start each day, think about how you can "DO all the good you can DO."

The idea for my theme came from a partial quote from John Wesley, the leader of the Methodist movement. "Do all the good you can.

He said, "Do all the good you can. By all the means you can. In all the ways you can. In all the places you can. At all the times you can. To all the people you can. As long as ever you can."

But did you know that we all share the same, all-encompassing, defining moment?

That defining moment, common to each of us, was the moment when we first heard these inspirational words, once spoken by the founder of osteopathy, Dr. A. T. Still, who said, "Let your light so shine that the world will know you are an osteopathic physician pure and simple, and that no prouder title can follow a human name."

Similarly, Scripture tells us that Christ said "no one after lighting a lamp covers it with a jar or puts it under a bed, but puts it on a stand so that those who enter may see the light."

I challenge you to be intentional about not hiding the light of osteopathy under a bushel.

Rather, seek out someone for whom you can become a defining moment in their life – the defining moment, when – through you – they see the shining light of osteopathic medicine!

Sincerely,

Larry W. Anderson, DO, FACOFP *dist*.
ACOFP President

From the American College of Osteopathic Family Physicians.

FROM THE OUTGOING PRESIDENT'S DESK

My Year as President

Kevin V. de Regnier, DO, FACOFP *dist*.
Immediate Past President

Then it was over. It seems like I was just inaugurated and now my year as President is over. And what a year it has been.

I want to thank each of you for your support and hospitality this past year. As I have traveled the country this year, you have welcomed me to your state meetings and to our colleges and you have fed me far too well. I also want to thank all of you who have taken time to write to me about a variety of issues. While it may seem strange, I especially appreciate those who shared your concerns and frustrations. I hope that I was able to provide some answers to reassure you that the ACOFP is already addressing your concerns.

As I reflect on the accomplishments of this year, I find they are too numerous to detail here but I will mention a few.

First, I must admit that I have accomplished nothing on my own. Throughout this year, I have been supported by the finest board, committees and association staff in the profession. Your Board has been forward looking, proactive leaders. As an organization, we have tried to look 2, 3, even 5 years down the road and have taken steps to help our members be prepared for the evolving practice environment. This philosophy is reflected in our strategic plan that was recently adopted by the ACOFP Congress of Delegates.

One of the areas where the ACOFP has made great progress is in engaging in the greater profession. This is directly related to our involvement in Family Medicine for America's Health. Our participation in FMAH has opened doors in many different areas. We have become a part of "The Working Party" an organization of family medicine organizations that come together to tackle the broad issues facing family medicine. We have received invitations to join with organizations such as the Association of Departments of Family Medicine, the Association of Family Medicine Residency Directors , and the North American Primary Care Research Group. Participation with these organizations has allowed us to leverage our limited resources to provide a broader range of service to our members.

Another area where ACOFP has stepped up its game is in our Washington advocacy. This past May, Ryan McBride joined the ACOFP staff as Director of Legislative Affairs. Ryan's previous work on Capitol Hill has served us well this year and ACOFP is developing an independent voice on legislative and regulatory issues. This increased visibility has lead to invitations to participate in White House events, roundtable meetings at the Brookings Institute and other prestigious organizations. Members of Congress, Congressional staffers, CMS, and other health care organization have begun actively seeking our input on important regulatory matters.

Finally, this past year was a time of exciting developments in our Quality Markers program. The ACOFP QM program is the nation's only physician designed, association marketed patient and population data management tool. Designed to integrate directly with your EMR, it can provide the information needed to succeed in the coming payment environment. Because of our work with our data partner Symphony Performance Health, we were able to join with the Consortium for Southeast Hypertension Control in a $13 million federal grant. Through his grant, the ACOFP is now able to provide members with free access to the ACOFP QM program and practice transformation services for the next four years. You can learn more about this on our website, acofp.org.

While I have only scratched the surface on the many activities of the ACOFP, I am confident that incoming President, Larry Anderson, will continue the work we have begun and will add his own ideas to our efforts. Working together - members, leadership, and staff - we can ensure that the ACOFP continues to grow in its ability to support you in your practice.

Sincerely,

Kevin V. de Regnier, DO, FACOFP *dist*.
2015 - 2016 ACOFP President

From the American College of Osteopathic Family Physicians.

REVIEW ARTICLE

Highlights of the Updated 2016 American Diabetes Association Standards of Medical Care in Diabetes

Neil Skolnik, MD,[1] Kim Pfotenhauer, DO,[2] Eric Johnson, MD,[3] Florence Warren, DO, PGY-2,[4] & Jay H. Shubrook, DO, FACOFP[5]

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Keywords:
Diabetes
Prediabetes
Guidelines
Insulin
Obesity

Diabetes has become a national epidemic. Nearly 50% of American adults have either prediabetes or diabetes.[1] Further if trends continue, by 2050, 1 in 3 American adults will have overt diabetes.[2] The American Diabetes Association (ADA) publishes annual Standards of Medical Care in Diabetes in the January supplement of Diabetes Care.[3] This review will highlight key features of the Standards of Care and report on changes and new updates to the guidelines.

The ADA has published the Standards of Care since 1989. The Standards cover the spectrum of care, from screening and diagnosis to management and risk reduction. The ADA strives to be transparent in the development of its evidence-based guidelines, following the Institute of Medicine recommendations.

Each year, the ADA's Professional Practice Committee does a systematic MEDLINE search to find new evidence or clarify prior recommendations. This multidiscipline committee also receives feedback from the larger clinical community. The committee assigns each recommendation a rating of A, B, C, or expert opinion E, depending on the quality of evidence.

WHAT IS NEW?

A new section has been added to the Standards, "Obesity Management for the Treatment of Type 2 Diabetes." Recommendations include the comprehensive assessment of weight in diabetes and treatment of overweight/obesity with behavior modification and pharmacotherapy. This section also includes a new table of currently approved medications for the long-term treatment of obesity. Bariatric surgery as a treatment for type 2 diabetes was also added to this section.

To reflect the changing role of technology in the prevention of type 2 diabetes, a recommendation was added encouraging the use of new technology such as apps and text messaging to affect lifestyle modification to prevent diabetes.

A recommendation was made to reflect new evidence that adding ezetimibe to moderate-intensity statin provides additional ardiovascular benefits for select individuals with diabetes and should be considered.

A new recommendation was added to highlight the importance of discussing family planning and effective contraception with women with preexisting diabetes.

WHAT HAS CHANGED?

Diabetes screening recommendations have been clarified. All adults should be screened for type 2 diabetes beginning at age 45 years, regardless of weight. Testing is also recommended for asymptomatic adults of any age who are overweight or obese and who have one or more additional risk factors.

To reflect new evidence on CVD risk among women, the recommendation to consider aspirin therapy in women age >60 years has changed to include women age ≥50 years. A recommendation was also added to address antiplatelet use in patients age <50 years with multiple risk factors.

A1C recommendations for pregnant women with diabetes were changed, from a recommendation of <6.0% to a target of 6.0–6.5%.

CORRESPONDENCE:
Jay H. Shubrook, DO, FACOFP | jay.shubrook@tu.edu

HIGHLIGHTS FROM THE 2016 ADA STANDARDS OF CARE

Screening for diabetes

1. The ADA recommends that all adults age 45 or older be screened for diabetes
2. Younger adults who are overweight or obese (BMI ≥25 kg/m^2 or ≥23 kg/m^2 in Asian Americans) and who have additional risk factors should be screened
3. Screening should be repeated every 3 years if normal
4. Screening should be every year if there is evidence of prediabetes

How should you screen?

1. With fasting plasma glucose (FPG)
2. With A1C
3. With oral glucose tolerance test (OGTT)
4. With random plasma glucose (RPG)

An elevated fasting glucose or A1C should be repeated by another test separated by time to confirm the diagnosis. The addition of the A1C is to allow an additional method of screening as it may be difficult for people to get fasting labs. Point of care A1C machines allow this test to come completed with a simple finger stick.

Prevention of type 2 diabetes

People who are found to have prediabetes (Table 1) should be referred to a program that adheres to the tenants of the National Diabetes Prevention Program (NDPP). The goals of this year-long program are to use group support, problem-based learning and work toward the following goals: lose 7% of body weight, reduce dietary fat and calories, and engage in moderate intensity physical activity for 150 minutes per week. The Diabetes Prevention Program (DPP) demonstrated a 58% reduction in risk of type 2 diabetes in the intervention group, and an even greater reduction in risk of 71% in those ≥60 years.[4] Further, even 10 years after the DPP intervention the risk of developing diabetes is still reduced by 30%.[5] Find a diabetes prevention program near your practice: https://nccd.cdc.gov/DDT_DPRP/Registry.aspx.

Selected medications also have been shown to reduce the progression from prediabetes to diabetes. These include metformin, alpha-glucosidase inhibitors, orlistat and thiazolidinediones. Currently, no medication is FDA-indicated for the prevention of type 2 diabetes.

Diabetes self-management education & support

All people with diabetes should receive comprehensive diabetes self-management education and support (DSME/S).[6] This should be repeated as needed as the disease progresses or as new skills are needed to manage diabetes (such as insulin injection therapy). DSME has been shown to improve clinical outcomes and quality of life in people with diabetes and this education can result in cost savings to the patient and health care system.

Despite the benefit of receiving DSME, only 6.8% of individuals with newly diagnosed type 2 diabetes with private health insurance participated in DSME/S within 12 months of diagnosis.[7] Only 4% of Medicare participants received DSME/S and/or Medical Nutrition Therapy (MNT).[8]

Physical activity

All adults with prediabetes and diabetes should be encouraged to perform at least 150 minutes of moderate intensity physical activity each week. Children with prediabetes and diabetes should perform at least 60 minutes of physical activity per day. This activity should be of at least moderate intensity and can be broken up into smaller segments of time.

Glycemic targets

The decision of the target glucose must be individualized to the patient. Most adults should be treated to an A1C of <7.0%. Younger patients, those newly diagnosed and those without known cardiovascular disease may warrant from a more stringent glucose target. However, patients with advanced complications, long-standing diabetes, multiple comorbidities or those with limited life expectancy are better treated to a less stringent goal to balance the risks and benefits of therapy.

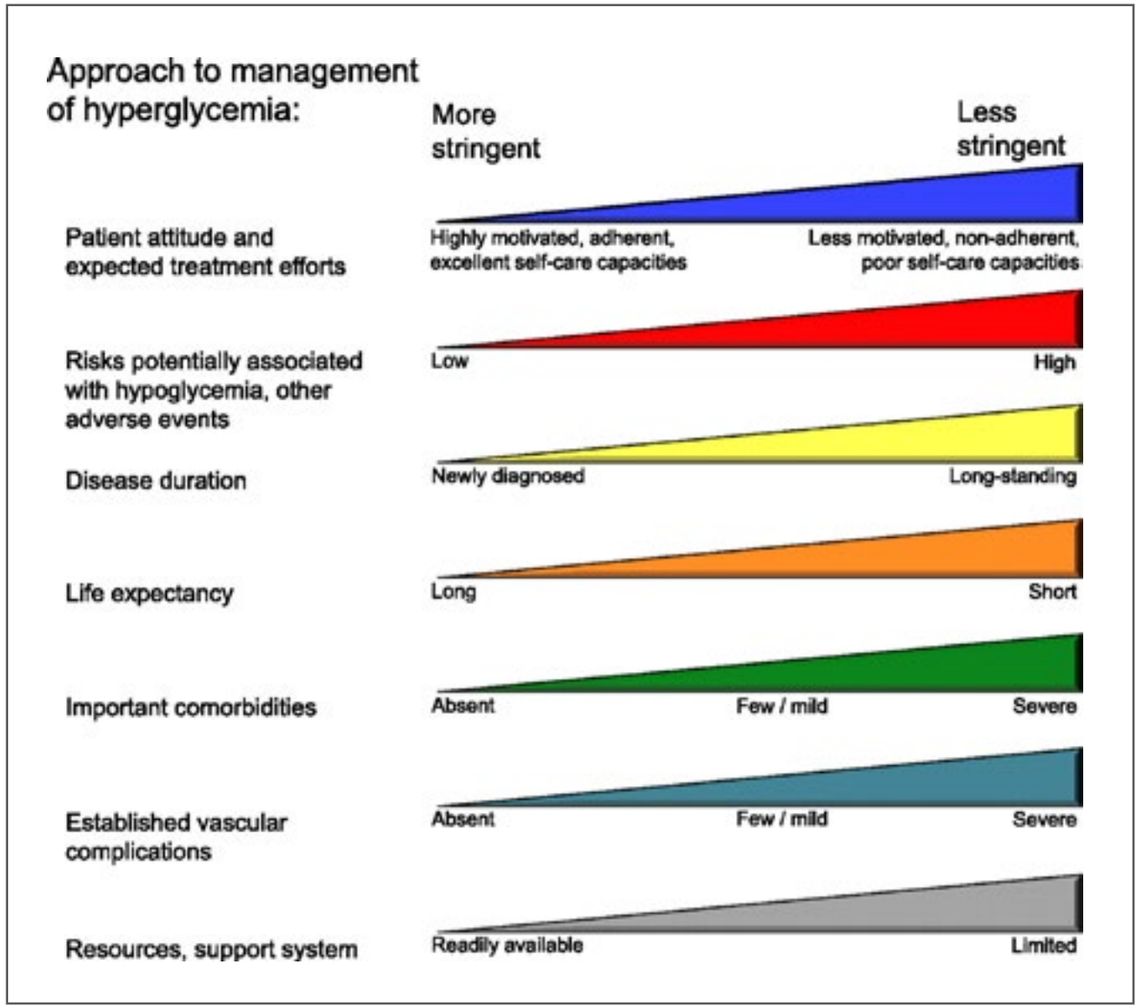
Guidance for how to individualize therapy is provided in Figure 1 *(page 14)*.[9]

TABLE 1:
Diagnostic criteria for diabetes and prediabetes

	FPG	A1C	OGTT	RPG
Normal	< 100 mg/dL	< 5.7%	< 140 mg/dL	
Prediabetes	100 - 125 mg/dL	5.7% - 6.4%	> 140 - 199 mg/dL	
Diabetes	≥ 126 mg/dL	≥ 6.5%*	≥ 200 mg/dL*	≥ 200 mg/dL**

*In the absence of unequivocal hyperglycemia, results should be confirmed by repeat testing.
**Only diagnostic in a patient with classic symptoms of hyperglycemia or hyperglycemic crisis.

FIGURE 1:
Guidance for how to individualize therapy.



Depicted are patient and disease factors used to determine optimal A1C targets. Characteristics and predicaments toward the left justify more stringent efforts to lower A1C; those toward the right suggest less stringent efforts. Adapted with permission from Inzucchi et al.9 Reprinted with permission of the American Diabetes Association, Inc. Copyright 2015.

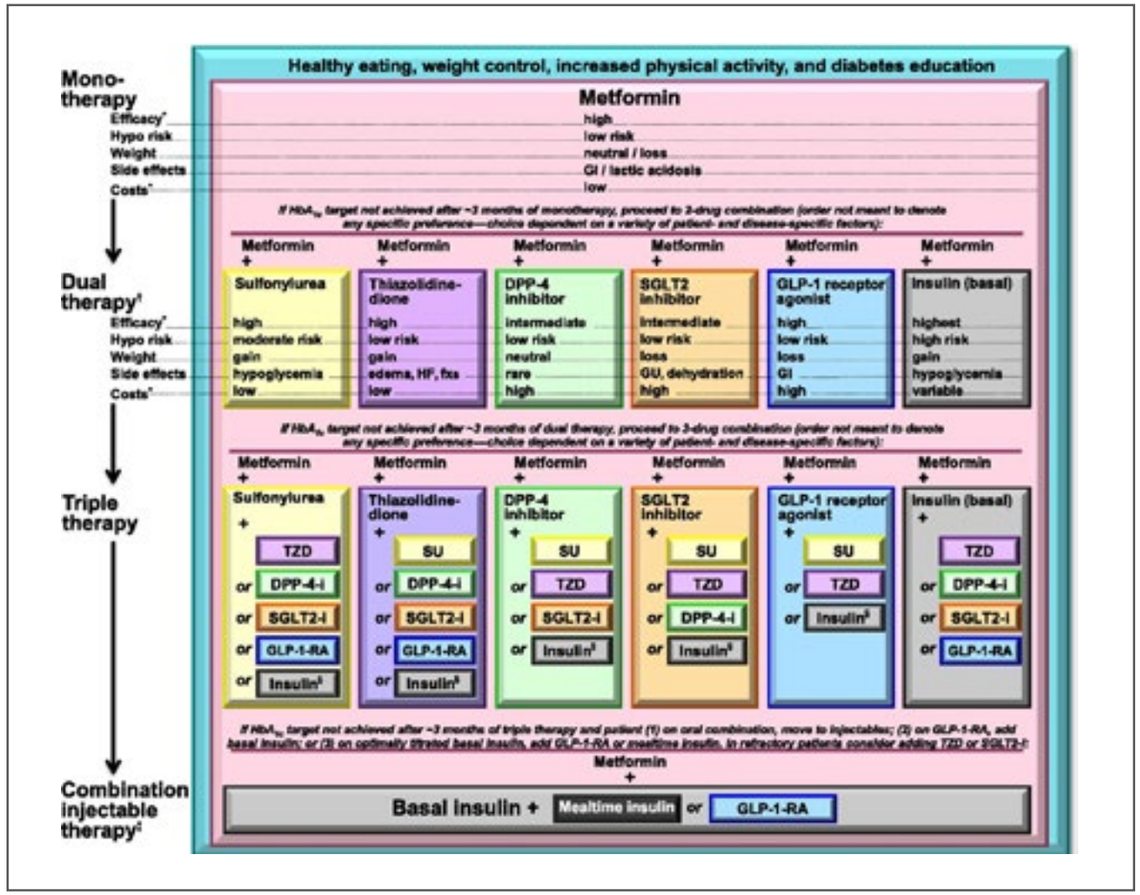
Pharmacologic treatment of type 2 diabetes

DSME/S and therapeutic lifestyle modification should be prescribed to patients at diagnosis. In addition to lifestyle changes, metformin should be started immediately for all people with type 2 diabetes, as long as it is tolerated and not contraindicated. This medication should be given at the time of diagnosis. Even a delay of 3-6 months after diagnosis can reduce the efficacy and durability of this medication (10). The patient should be evaluated at least every 3 months to see if agreed upon glucose treatment target has been achieved. If not, treatment should be intensified. Many medications are available for treatment, and guidance is available to help the clinician to decide which treatment is most appropriate for each patient.[9] See Figure 2.

Insulin therapy should be considered in patients who present with catabolic symptoms (polyuria, polydipsia and weight loss) or an A1C ≥10%, and in patients who are unable to get control with dual or triple therapy at one year after treatment has started.

Medication cost, potential side effects including hypoglycemia and weight gain, and efficacy are important factors when deciding what treatments are going to be used and avoidance of these side effects is preferred.

FIGURE 2:
Antihyperglycemic therapy in type 2 diabetes: general recommendations



The order in the chart was determined by historical availability and the route of administration, with injectables to the right; it is not meant to denote any specific preference. Potential sequences of antihyperglycemic therapy for patients with type 2 diabetes are displayed, with the usual transition moving vertically from top to bottom (although horizontal movement within therapy stages is also possible, depending on the circumstances). DPP-4-i, DPP-4 inhibitor; fxs, fractures; GI, gastrointestinal; GLP-1-RA, GLP-1 receptor agonist; GU, genitourinary; HF, heart failure; Hypo, hypoglycemia; SGLT2-i, SGLT2 inhibitor; SU, sulfonylurea; TZD, thiazolidinedione. *See ref. 9 for description of efficacy categorization. †Consider starting at this stage when A1C is ≥9% (75 mmol/mol). ‡Consider starting at this stage when blood glucose is ≥300–350 mg/dL (16.7–19.4 mmol/L) and/or A1C is ≥10–12% (86–108 mmol/mol), especially if symptomatic or catabolic features are present, in which case basal insulin + mealtime insulin is the preferred initial regimen. §Usually a basal insulin (NPH, glargine, detemir, degludec). Adapted with permission from Inzucchi et al.[9] Reprinted with permission of the American Diabetes Association, Inc. Copyright 2015.

ASSESSMENT OF HOME GLUCOSE MONITORING

Self-monitoring of blood glucose (SMBG) is a key element to help people evaluate the effectiveness of their treatments (lifestyle and medications).[11] The use of SMBG can be very helpful in medication titration, identification of hypoglycemia, and reinforcement of therapeutic lifestyle behaviors. Studies have supported a relationship between SMBG frequency and improved A1C in type 1 diabetes.

SMBG is especially important in people who are taking insulin and in those who have experienced hypoglycemia. There is not enough evidence to support the optimal frequency of SMBG on those only on oral therapy or therapeutic lifestyle changes.

SMBG requires skills and all people with diabetes should receive education on the use of a glucometer and periodic reassessment of technique. Providers should review the results of SMBG at each assessment to determine the adequacy of treatment and to identify hypoglycemia.

Cardiovascular risk reduction

Atherosclerotic cardiovascular disease (ASCVD) is the (I prefer number 1 or major) cause of death in people with diabetes. People with diabetes should have their cardiovascular risk factors evaluated and managed. Numerous studies have shown the efficacy of controlling individual factors in preventing or slowing ASCVD in people with diabetes. Large benefits are seen when multiple risk factors are addressed simultaneously. There is evidence that measures of 10-year coronary heart disease risk among U.S. adults with diabetes have improved significantly over the past decade, with a decrease in morbidity and mortality.[12, 13, 14]

Blood pressure

Blood pressure should be measured at every clinical appointment. Most people with diabetes should maintain a blood pressure below 140/90 mmHg.[15] If the blood pressure is elevated or if there is evidence of nephropathy (albuminuria or proteinuria), then an ACE inhibitor (ACEI) or angiotensin receptor blocker (ARB) should be started and titrated to the maximum tolerated dose. It is not recommended to start an ACEI or ARB in a person who is normotensive and no nephropathy, as the risks outweigh the benefits. Further, it is not recommended to use an ACEI and ARB concomitantly.

Treatment of dyslipidemia

In addition to intensive lifestyle therapy, statin use is recommended for most people with diabetes age 40 years and older. People who have diabetes age 40 years and older without additional ASCVD risk factors should consider using a moderate-intensity statin. Those people with diabetes age 40-75 years with ASCVD risk factors should consider using a high-intensity statin. Patients age 75 years and older with ASCVD risk factors should consider a moderate- or high-intensity statin. Table 2 provides guidance on statin use and intensity. The addition of ezetimibe to moderate intensity statin therapy has been shown to provide additional cardiovascular benefit compared to moderate intensity statin therapy alone, and may be considered for patients with a recent acute coronary syndrome with an LDL cholesterol ≥ 50mg/dL or in those patients who cannot tolerate high-intensity statin therapy.[16]

Antiplatelet agents

Aspirin therapy (75–162 mg/day) is recommended as a primary prevention strategy in those with type 1 and type 2 diabetes who are at increased cardiovascular risk (10-year risk >10%). However, aspirin should not be recommended for coronary disease prevention for adults with diabetes at low risk (10-year ASCVD risk <5%). Aspirin therapy is well established as a secondary prevention strategy in those with diabetes and a history of ASCVD. In patients with ASCVD and documented aspirin allergy, clopidogrel (75 mg/day) should be used. Dual antiplatelet therapy is reasonable for up to a year after an acute coronary syndrome.

Hypoglycemia

Hypoglycemia (≤70 mg/dL) is the rate-limiting step to normalizing glucose. It was previously thought to be a problem mostly for type 1 diabetes, but it is well established that many people with type 2 diabetes experience hypoglycemia. The total number of hypoglycemic episodes are greater from people with type 2 diabetes than type 1 diabetes. Episodes of severe hypoglycemia were associated with mortality in the both the ACCORD and ADVANCE trials.[17-18]

TABLE 2:
Statin intensity in the treatment of ASCVD risk in diabetes

High - intensity statin therapy	Moderate - intensity statin therapy
Lowers LDL by ≥ 50%:	**Lowers LDL by 30% to <50%:**
Atorvastatin 40–80 mg	Atorvastatin 10–20 mg
Rosuvastatin 20–40 mg	Rosuvastatin 5–10 mg
	Simvastatin 20–40 mg
	Pravastatin 40–80 mg
	Lovastatin 40 mg
	Fluvastatin XL 80 mg
	Pitavastatin 2–4 mg

Severe hypoglycemia is defined as hypoglycemia that requires assistance from another person. All patients at risk of severe hypoglycemia should be prescribed glucagon injection and their family/close contacts should be instructed on how to administer glucagon during severe hypoglycemic episodes.

Hypoglycemia may be reversed with administration of rapid acting glucose (15-20 g). Blood glucose reversal should be confirmed with SMBG after fifteen minutes; if hypoglycemia persists, the process should be repeated. Pure glucose is the preferred treatment; however, any form of carbohydrate that contains simple sugars not combined with fat or protein will raise blood glucose quickly (e.g., hard candies instead of a candy bar).

Physicians should assess at each visit if their patient is experiencing hypoglycemia. Patients should be educated on situations that increase their risk of hypoglycemia such as fasting for tests or procedures, alcohol ingestion, during or after exercise, and during sleep. Many patients who experience hypoglycemia may omit or change their treatment plans without the physician's knowledge. Hypoglycemia has substantial negative effects on a person's quality of life.

Repeated episodes of hypoglycemia can lead to hypoglycemia unawareness. Hypoglycemia unawareness is characterized by deficient counterregulatory hormone release and a diminished autonomic response, both of which are risk factors for, and caused by, hypoglycemia. Patients with hypoglycemia unawareness or an episode of severe hypoglycemia should be advised to raise their glycemic targets for at least several weeks to partially reverse hypoglycemia unawareness and reduce the risk of future episodes.

Older adults

Coordination of care and individualization of treatment plans should be considered with respect to changes in functional status and co-existing conditions, such as ASCVD and chronic kidney disease, in older patients with both type 1 and type 2 diabetes. Glycemic goals may be relaxed but hypoglycemia and hyperglycemic complications should be avoided. Lipid-lowering and aspirin therapy should be considered in the context of life expectancy. Hypertension treatment is indicated for nearly all older patients with diabetes. Older adults are a high-priority population for depression screening.[19]

Microvascular complications

Intensive blood glucose and blood pressure control can reduce the risk or slow the progression of microvascular complications.

Nephropathy: There should be annual assessment of urinary albumin (e.g., spot urinary albumin-to-creatinine ratio [UACR]) and estimated glomerular filtration rate (eGFR) in patients with type 1 diabetes with duration of ≥5 years, in all patients with type 2 diabetes, and in all patients with comorbid hypertension. For urinary albumin, two of three specimens collected within a 3 to 6 month period should be abnormal before considering a patient to have developed albuminuria.

For patients with diabetic kidney disease (DKD), dietary protein intake should be 0.8 g/kg body weight per day. ACEIs and ARBs have been shown to slow the decline in GFR in patients with elevated urinary albumin excretion (>30 mg/day). An ACEI or ARB is not recommended for the primary prevention of DKD in patients with diabetes who have normal blood pressure, normal UACR (<30 mg/g), and normal eGFR. Combined use of an ACEI and an ARB should be avoided as it provides no additional benefit for CVD or DKD and has a higher adverse event risk.

Retinopathy

Patients with type 1 diabetes should have an initial dilated and comprehensive eye examination by an ophthalmologist or optometrist within 5 years after diagnosis of diabetes. Patients with type 2 diabetes should have an initial dilated and comprehensive eye examination by an ophthalmologist or optometrist shortly after the diagnosis of diabetes. The exams should be repeated annually. If there is no evidence of retinopathy for one or more eye exams, then exams every 2 years may be considered. The presence of retinopathy is not a contraindication to aspirin therapy for cardioprotection, as aspirin does not increase the risk of retinal hemorrhage.

Neuropathy

All patients should be screened for diabetic peripheral neuropathy (DPN) starting at diagnosis of type 2 diabetes and 5 years after the diagnosis of type 1 diabetes and at least annually thereafter. Assessment should include a careful history and 10-gram (g) monofilament testing, and at least one of the following tests: pinprick, temperature, and vibration sensation. Clinicians should screen for signs and symptoms of autonomic neuropathy in patients with more advanced disease. These signs and symptoms can include: resting tachycardia, exercise intolerance, orthostatic hypotension, gastroparesis, constipation, erectile dysfunction, impaired neurovascular function, and autonomic failure in response to hypoglycemia. Control of lipids, smoking, and other lifestyle factors can reduce the progression and development of autonomic neuropathy.

The FDA has approved pregabalin, duloxetine, and tapentadol for the treatment of pain associated with DPN. Tricyclic antidepressants, gabapentin, venlafaxine, carbamazepine, tramadol, and topical capsaisin, although not approved for the treatment of painful DPN, may be effective and considered for the treatment of painful DPN.

Foot Care

An annual comprehensive foot examination to identify risk factors predictive of ulcers and amputations is recommended. The foot examination should begin with inspection and assessment of foot pulses. The exam should seek to identify loss of peripheral sensation (LOPS). The examination should include inspection of the skin, assessment of foot deformities, neurologic assessment including 10-g monofilament testing and pinprick or vibration testing or assessment of ankle reflexes, and vascular assessment including pulses in the legs and feet.

Patients who smoke or have histories of prior lower-extremity complications, a loss of protective sensation, structural abnormalities, or peripheral arterial disease (PAD) should be referred to foot care specialists for ongoing preventive care and lifelong surveillance. Patients should be screened by careful history and physical exam of pulses for PAD. Ankle-brachial index testing (ABI) should be performed in patients with symptoms or signs of PAD. ABI may be considered starting at age 50 and in patients younger than 50 years of age with risk factors.

SUMMARY

The ADA 2016 Standards of Care is a source of high-quality evidence-based recommendations for the care of people with diabetes across the lifespan. Screening for prediabetes is an important priority to identify those at risk for diabetes, as lifestyle intervention is an established preventive strategy with a new emphasis on obesity management. Individualized glycemic targets with attention to hypoglycemia can reduce the risk of diabetes complications. Studies also support evaluation and effective treatment of risk factors to reduce ASCVD in persons with diabetes. The 2016 Abridged Standards of Care can be an important resource for primary care physicians caring for those with diabetes.

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REVIEW ARTICLE

Osteopathic Considerations in the Management of Chest Pain

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Chest pain is one of the most common reasons for patients to seek medical care, accounting for about 1 to 3% of office visits to a primary care provider. The most common cause of chest pain is musculoskeletal in origin. A thorough osteopathic history and physical will help accurately diagnose musculoskeletal chest pain. Some common musculoskeletal causes of chest pain include costochondritis, lower rib pain syndrome, posterior chest wall pain syndrome, and muscle strain. Osteopathic manipulative medicine can be incorporated into the treatment of musculoskeletal chest pain.

INTRODUCTION

Chest pain is one of the most common reasons for patients to seek medical care, accounting for about about 1 to 3% of office visits to a primary care provider.[1] Of these visits, the most common cause of pain is musculoskeletal, not cardiac, in origin.[1,2]

The complaint of chest pain must be considered seriously. It can represent life-threatening medical conditions potentially involving the cardiovascular, pulmonary and gastrointestinal systems. Other causes of chest pain are less critical and can be associated with musculoskeletal dysfunctions.[3] In the hospital setting, about 20% of patients with undifferentiated chest pain are admitted for suspected acute coronary syndrome (ACS). There is an estimated cost of $8 billion for the initial care of these patients who are later discharged without a diagnosis of coronary artery disease.[4]

Since medical training teaches physicians to first rule out conditions associated with symptomatic "red flags," we must be mindful to keep other causes in our differential. Osteopathic physicians are trained to approach patients as a unit, a whole person. When history, physical examination and pertinent diagnostic tests have ruled out life-threatening causes and provide no answer for the cause of pain, it is important to remember the osteopathic principles and treatments that can help provide the necessary care for patients.

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EPIDEMIOLOGY

The number of patients with chest pain secondary to a musculoskeletal source is more common in patients presenting to their primary care clinician than an emergency department.[2] It also occurs more frequently among women than men. In Disla et al.'s study that examined the incidence of musculoskeletal chest pain, 69% of patients diagnosed with costochondritis were women.[5] In the primary care setting, frequencies of the different etiologies of chest pain are musculoskeletal 36-49%, cardiovascular 15-18%, gastrointestinal 8-19%, pulmonary 5-10% and psychiatric 8-11%.[6]

NON-MUSCULOSKELETAL CAUSES OF CHEST PAIN

The differential diagnosis of patients presenting with chest pain ranges from benign musculoskeletal etiologies to life-threatening diseases such as myocardial infarction, esophageal rupture, perforating peptic ulcer, pulmonary embolus and tension pneumothorax.[7] It is important to rule out cardiovascular, pulmonary and gastrointestinal causes of chest pain before definitively diagnosing musculoskeletal chest pain.

Coronary artery disease can lead to ischemic chest pain, which may be present in a spectrum of cardiovascular diseases including stable angina, unstable angina, non-ST elevation myocardial infarction, and ST elevation myocardial infarction.[8] Patients with myocardial infarction (MI) present with substernal chest pain, usually radiating to the shoulder, jaw or arm, which is exacerbated by exertional activity and relieved by rest or nitroglycerine. In one study (n= 94 patients), Lusiani et al. found that 32% of patients presented with atypical symptoms of MI, including abdominal pain, paroxysmal dyspnea and symptoms of pulmonary edema, with the frequency of symptoms being 33%, 17% and 13%, respectively. These atypical symptoms were most prevalent in women over the age of sixty-five years.[9] Thus, in elderly patients, risk factors for coronary artery disease should be assessed.[10]

The most common gastrointestinal cause of chest pain is gastroesophageal reflux disease (GERD), which is characterized by squeezing or burning substernal chest pain radiating to the back, neck, arms or jaw.[11] Peptic ulcer disease (PUD) can also cause chest pain and may lead to a perforation of the gastrointestinal lining which is a life-threatening emergency. Patients with perforated PUD may present with a sudden onset of severe, sharp abdominal pain.[8] Esophageal rupture, another life-threatening gastrointestinal cause of chest pain, is characterized by odynophagia, tachypnea, dyspnea, cyanosis, fever and shock.

Tension pneumothorax and pulmonary embolism are life threatening pulmonary causes of chest pain. Although the initial presentation of tension pneumothorax can vary, development of severe dyspnea, tachycardia and hypotension can occur over time. Patients may also have distended neck veins and tracheal deviation.[8] Stein et al. found that the most common symptoms of pulmonary embolism were dyspnea (73%), pleuritic chest pain (66%), cough (37%) and hemoptysis (13%).[12]

MUSCULOSKELETAL CAUSES OF CHEST PAIN

There are several key factors to consider in a patient's history when evaluating musculoskeletal causes of chest pain. Musculoskeletal chest pain includes pain related to the thoracic spine and the anterior chest wall's bony, cartilaginous and muscular structures.[13] The pain has an insidious onset and a prolonged duration that lasts for hours to days. A recent history of repetitive activity may favor the diagnosis of musculoskeletal chest pain. Deep breathing, turning, or arm movement may exacerbate the pain, which is frequently sharp and localized to a specific area near the costochondral junction.[10]

Costochondritis (also known as costosternal syndrome or anterior chest wall syndrome) is characterized by achy, sharp, pressure-like pain and tenderness of multiple joints in the costochondral junction. Pain is usually unilateral and aggravated by movements of the upper body, deep breathing or exertional activities. Signs of inflammation and swelling are usually absent. The mechanism of pain is believed to be mechanical derangement, muscular imbalance or neurogenic inflammation.[14] Diagnosis is based mainly on the ability to reproduce the pain by palpation of tender areas. Bösner et al. demonstrated that two of the following four features, localized muscle tension, stinging pain, pain reproducible by palpation and absence of cough, are associated with the diagnosis of anterior chest wall syndrome.[15]

Lower rib pain syndrome (also known as rib-tip syndrome, slipping rib syndrome, twelfth rib syndrome and clicking rib syndrome) is characterized by pain in the lower chest or upper abdomen. A tender point on the costal margin and pain that is reproduced by pressing on this area is also a characteristic of this syndrome.[16]

Posterior chest wall pain syndrome, also known as thoracic spinal pain syndrome, is relatively common in workplace settings and is associated with chest pain.[17] Thoracic disc herniation is a cause of posterior chest wall pain that should be considered in patients with dermatomal pain. Costovertebral joint dysfunction is another cause in which the patient presents with pain that is made worse with coughing or deep breathing. Palpating the costovertebral junction often reproduces the pain. There may also be areas of local hyperalgesia.[18]

Strains of the intercostal, pectoralis, internal and external oblique and serratus anterior muscles are another common cause of musculoskeletal chest pain. Acute onset of muscle strain is usually caused by trauma or overuse while gradual onset of muscle pain results from tension or anxiety. Muscle tears may present with sudden pain in the region followed by swelling and bruising.[1]

Some less common causes of chest pain are sternalis syndrome, Tietze's syndrome, xiphoidalgia and spontaneous sternoclavicular subluxation. Sternalis syndrome is localized tenderness over the body of the sternum and palpation to the area causes pain to radiate bilaterally. Tietze's syndrome is characterized by painful, localized swelling in costosternal, sternoclavicular and costochondral joints.[19] Xiphoidalgia is localized tenderness over the xiphoid process of sternum.[20] Spontaneous sternoclavicular subluxation is an anterior or cranial displacement of the clavicle that usually occurs on the dominant-hand side in women 40-60 years old. This displacement may occur due to heavy repetitive activity. Radiography can also show sclerosis of the medial clavicle in spontaneous sternoclavicular subluxation.[21]

There are systemic diseases that can cause musculoskeletal chest wall pain such as rheumatoid arthritis (RA), ankylosing spondylitis and fibromyalgia. RA is an autoimmune disease that classically arises in women of late childbearing age and it is characterized by destruction of cartilage and ankylosis or fusion of the joint. Clinical features include joint pain with morning stiffness that improves with activity. Joint-space narrowing, loss of cartilage and osteopenia are typically seen on x-ray. In a recent study of 412 subjects, RA subjects (19%) had significantly more pain and swelling in the sternoclavicular joint than healthy controls (1.9%). Also in the RA group, ultrasound abnormalities such as osteophytes (29%), synovitis (15%) and erosions (11%), were recorded in 89 sternoclavicular joints (43%) compared with 36 (17%) in the healthy control.[22]

Ankylosing spondylitis is a chronic inflammatory disease of the spine and sacroiliac joints. It is commonly seen in younger patients with a history of chronic low back pain and morning stiffness. Any deficits while examining forward flexion of lumbar spine in a younger patient may suggest ankylosing spondylitis.[23]

Fibromyalgia is characterized by chronic widespread musculoskeletal pain with sleep disturbances and fatigue. Patients with fibromyalgia can have specific bilateral tenderpoints in the upper and mid-cervical, trapezius, lateral gluteal, lateral trochanteric, medial knees and anterior costochondral regions.[1]

OSTEOPATHIC PHYSICAL EXAMINATION

A general physical examination, including an osteopathic structural exam (OSE), should be conducted to rule out cardiovascular, pulmonary, gastrointestinal and other visceral causes of chest pain. There is no "one-size-fits-all" approach to these patients, as there are many different causes.[24] Findings associated with

non-musculoskeletal causes of chest pain may include exertional pain, cough, fever, dyspnea and pain exacerbated by deep breathing.[10]

OSE findings may assist in diagnosing or ruling out visceral causes of chest pain. A viscerosomatic reflex is caused by stimulus from an internal organ that produces a reflex response in the musculoskeletal system sharing the same spinal segment innervation.[25] Chronic irritation and inflammation of the stomach lining that leads to tissue texture changes and thoracic cage somatic dysfunctions from T5-T9 is an example of a viscerosomatic reflex. Somatic dysfunction, tissue texture changes, or temperature variations may be due to viscerosomatic reflexes.[25] Chapman points may also be associated with a visceral cause of chest pain. These points are "plaque-like changes" that represent visceral dysfunction or pathology and may play an important role in narrowing down the differential diagnosis of chest pain.[25] Viscerosomatic findings are summarized in Table 1.

The initial examination for non-visceral chest pain should start at the spine and shoulders using observation, palpation and range of motion testing.[10,26] Physicians should note any tissue texture changes, asymmetry, restriction of motion and tenderness (TART) through direct palpation of the anterior and posterior chest wall. Acute changes will present with edema, tenderness, pain and tissue contraction. Chronic changes will present with tenderness, fibrosis and ropy changes.[25] Physicians should then assess the mobility of the thoracic cage with respiration and the range of motion (passive and active) of the cervical, thoracic and lumbar spine. Any areas of restriction in the spine or rib cage should be noted. Tenderness or pain in the thoracic cage that is reproduced with movement is highly suggestive of a musculoskeletal cause of chest pain.[10] Cervical spine somatic dysfunctions may contribute to postural strains and lead to pain in the chest and upper thoracic regions. Anterior structures, including the costochondral and chondrosternal joints, should also be examined.[27] Other key areas to assess may include the diaphragm, thoracic outlet and upper extremities. It is important to do a complete structural exam so as not to miss dysfunctions in other areas that may be contributing to the presenting pain.

TABLE 1:

Osteopathic Structural Findings Associated with Non-musculoskeletal Causes of Chest Pain.[24]

	OSTEOPATHIC FINDINGS	
Origin	**Viscerosomatic Reflexes**	**Chapman Points**
Cardiac	T1 - T5	Anterior: 2nd ICS Posterior: T2 lamina of TP
Pulmonary	T2 - T7	Anterior: • 3rd ICS: Upper Lung • 4th ICS: Lower Lung Posterior: • Between T3-T4 TP: Upper Lung • Between T4-T5 TP: Lower Lung
Gastrointestinal	Upper GI Tract (Stomach-Duodenum): T5-T9 Middle GI Tract (Jejunum - Proximal transverse colon): T10-T11 Lower GI Tract (Distal 1/3 of transverse colon - Rectum): T12-L2	Anterior Points: • 5th ICS: Liver, Gallbladder (right), Stomach acid (left) • 6th ICS: Gallbladder (right), Stomach peristalsis (left) • 6th or 7th ICS: Spleen (left), Pancreas (right) • 7th - 10th ICS: Small intestine • Tip of 12th Rib: Appendix Posterior Points: • Between T5 - T6 SP: Liver (right), Stomach acid (left) • Between T6 - T7 SP: Liver, Gallbladder (right), Stomach peristalsis (left) • Between T7 - T8 SP: Spleen (left), Pancreas (right) • Between T8 - T 11 SP: Small intestine • T12 TP: Appendix

Abbreviations: ICS - intercostal space; SP - spinous process; TP - transverse process.

A neurologic examination can be conducted to assess sensory and motor disturbances, evaluate peripheral reflexes and to rule out compression of cervical or thoracic nerve roots. Laboratory or radiographic studies can be conducted to rule out cardiac, pulmonary or abdominal disease, to assess for rheumatic disease and to directly assess specific anatomic regions of the chest wall.[10]

OSTEOPATHIC EVIDENCE-BASED TREATMENT

Once a musculoskeletal cause for chest pain has been determined, the osteopathic physician can treat it medically, as shown in Table 2, and with Osteopathic Manipulative Treatment (OMT) There are a limited number of high-quality studies available showing effective management of musculoskeletal thoracic pain.[28] However, manipulation can be an important aspect of treatment that should be considered in patients presenting with musculoskeletal chest pain. A recent systematic review investigated the effectiveness of non-invasive interventions for patients with chest pain and found that manipulation, as compared to acupuncture and placebo, may lead to a reduction in pain intensity. It was also noted that patients with a recent onset of pain who received multimodal management were 40% more likely to report improvement in their chest pain. This multimodal approach can include manual therapy, soft tissue therapy, exercise, heat or ice application and advice.[28]

The goals of osteopathic manipulative treatment are to relieve pain, improve circulatory and lymphatic function and to normalize autonomics or any viscerosomatic reflexes. The choice of technique to utilize depends on the patient's somatic dysfunction findings and the physician's comfort in performing the technique. In general, direct techniques, or treatment modalities that place the body into structural restrictions to treat the dysfunction, may be too painful in an acute presentation. Indirect techniques tend to be gentler and should be considered if the patient cannot tolerate direct techniques due to pain. The manipulative prescription will vary based on the patient presentation and the patient's response to treatment.[29]

TABLE 2:

Management of Musculoskeletal Chest Pain. [27]

	Considerations	Examples
Heat / Cold	Overload and overuse injuries may lead to muscle strains. Encourage patient to stop activity that may further exacerbate the injury.	Heat: Muscle spasm Cold: Reduce swelling and discomfort, acute
Topical Agents	Counsel patients on safety of application.	Capsaicin cream Salicylate-containing cream or gels Topical NSAIDs Lidocaine Patch
NSAIDs	Often used and important in patients with inflammation. Be sure that patients are aware of potential adverse effects (i.e. peptic ulcer disease, exacerbation of renal insufficiency) and that they are taking these medications appropriately.	Ibuprofen Naproxen
Muscle Relaxants	May be used, especially with acute muscle spasm. Avoid long term therapy, use in elderly patients and patients with a history of drug abuse.	Cyclobenzaprine Methocarbamol Benzodiazepines
Antidepressants	Can be used for chronic pain or pain that is neuropathic or osteoarthritic in origin.	Tricyclic antidepressants SSRIs and SNRIs
Anticonvulsants	Chronic pain	Gabapentin
Injections	Can use local glucocorticoid and/or anesthetic, often useful for arthritic pain	Hydrocortisone Methylprednisolone Triamcinolone
Narcotics	Avoid in patients with musculoskeletal chest pain. Should only be considered in isolated cases of acute exacerbations.	Short-acting, mild (i.e. codeine)
Psychiatric Evaluation	Evaluate patients for psychiatric factors that may contribute to presenting symptoms	Anxiety, depression, panic attacks

TABLE 3:
Osteopathic Manipulative Treatment

Technique	Basic Steps[24]	Indications	Cautions[24]
Myofascial Release	Direct or Indirect technique; There are many variations of myofascial release. Once an area of altered fascia has been identified, it is important to remember the mechanics involved, the anatomic relationships of the area being treated, and neural influences. A parallel or perpendicular stretch can be applied to hypertonic muscles.	Presence of somatic dysfunction in the connective tissues, i.e. fascia, muscles.[24] Helpful in patients with musculoskeletal chest pain.[4]	Open wounds, fractures, concomitant disease, internal injuries.
Facilitated Positional Release	Indirect technique; Place the patient in a neutral position while monitoring the point. A force of compression, traction, or torsion is then applied to release tissue tension and/or articular restriction.	Can be used to address superficial tissue texture change, as well as deep intrinsic muscles.[24]	Use caution in patients with osteoporosis, malignancy, rheumatologic disorders, congenital malformations, or stenosis.
Counterstrain	Indirect technique; Once most tender point is located, establish a pain scale. Passively position patient to position of greatest ease and reduced tenderness. Hold position for 90 seconds (120 seconds for ribs) while patient is relaxed.	Presence of tender points;[24] useful in patients with fibromyalgia.[30]	Fracture or ligamentous tear
Progressive Inhibition of Neuromuscular Structures	Direct technique; Locate a "primary point" (PP), the most sensitive point in the region. Locate an "end point" (EP), a point proximal or distal to the first. Determine a path between the two points. Maintain pressure on EP throughout. Initiate pressure at PP for 20-30 seconds. Compare sensitivity of PP to a secondary point (SP). If PP is less, continue with SP's until 2 cm from EP.	Hypertonicity of muscles. Hypertonicity of the pectoralis minor muscle has been associated with chest pain.[24]	Few contraindications. Avoid use with localized inflammation, abscesses, or infection.
Muscle Energy	Direct technique; Bring joint to the "feather's edge" of the restrictive barrier and direct the patient to move that part towards the direction of freedom. Physician applies an isometric counterforce to resist movement for 3-5 seconds, followed by post-isometric relaxation for 3-5 seconds. Re-engage barrier and repeat 3-5 times.	Consider treating muscles of respiration which may be restricting proper rib motion.	Patient unable to follow verbal commands. Patient with low vitality (i.e. post-surgical, post-myocardial infarction). Use caution in patients with acute injury.
Articulatory	Direct technique; Repetitive movement of a joint through its full motion until the restrictive barrier is engaged to increase range of motion.	Use when restrictive barrier is in the joint or periarticular tissues. Arthritic and frail patients tolerate this well.[24] Helpful in patients with musculoskeletal chest pain.[4]	Fracture/dislocation, neurologic entrapment, vascular compromise, local malignancy, local infection, bleeding disorder.
High Velocity Low Amplitude (HVLA)	Direct technique; Engage the barriers, while isolating the segment to be treated. A short and rapid thrust should be applied to the area during exhalation.	Somatic dysfunction with a firm barrier.[24] Has been shown to be useful in patients with musculoskeletal chest pain.[3,4] Can be used on spine and ribs to help mobilize thoracic cage.	Local metastases, osseous or ligamentous disruption. Cervical HVLA: Advanced rheumatoid arthritis, Down syndrome, advanced carotid disease.
Lymphatic Drainage	Always free restrictions at transition areas/ diaphragms first. Many different vibratory or oscillatory techniques can then be used to augment movement of lymph.	Acute somatic dysfunction, sprains/strains, inflammation, edema, tissue congestions.[24]	Deep venous thrombosis, certain stages of cancer, certain bacterial infections.

There are many different osteopathic manipulative techniques that can be utilized to address musculoskeletal chest pain. Myofascial release and soft tissue techniques can be used to reduce muscle spasm and restore symmetry, especially in patients with acute musculoskeletal chest pain.[4,24] Progressive inhibition of neuromuscular structures (PINS) and facilitated positional release (FPR) are also useful for decreasing hypertonic muscles.[24] Counterstrain is an effective technique for patients presenting with specific tender points, such as those seen in patients with fibromyalgia.[24,30] Articulatory techniques, including high-velocity low-amplitude (HVLA), can help to mobilize the thoracic cage in patients who present with decreased rib excursion, decreased range of motion, or facilitated segments with a firm endpoint.[3,4,24] Muscle energy and FPR may also be helpful with improving range of motion and decreasing muscle hypertonicity. Lymphatic techniques should be considered in patients with congestion, inflammation, or edema that may be contributing to chest pain, such as in cases of costochondritis and thoracic cage strains or sprains.[24] Gentle techniques may help balance autonomics in patients. Recent studies have shown that certain techniques have an effect on heart rate variability, increasing parasympathetic and decreasing sympathetic activity, in healthy subjects. There may be a role for these techniques in treating viscerosomatic reflexes post acute cardiac events.[31, 32] See Table 3 for a summary of possible techniques that can be used for patients with musculoskeletal chest pain, along with indications and cautions.

SUMMARY & CONCLUSION

Musculoskeletal problems are a common cause of chest pain in adults presenting to primary care physicians. The differential diagnosis of patients presenting with chest pain ranges from benign musculoskeletal etiologies to life-threatening disease. It is important to rule out cardiovascular, pulmonary and gastrointestinal causes of chest pain first. Reproducible chest wall tenderness is a major hallmark of chest pain of musculoskeletal origin. Integrating an osteopathic approach and manipulative treatment into patient care enables the physician to better diagnose and manage chest wall pain, especially when it is musculoskeletal in nature.

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REVIEW ARTICLE

Osteopathic Considerations in Obstructive Pulmonary Disease: A Systematic Review of the Evidence

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COPD

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OMM

Background: Since Dr. Still developed osteopathic philosophy many physicians have utilized osteopathic manual medicine (OMM) to treat respiratory disorders and have reported their results in case studies and research projects. With the increasing emphasis on utilizing medical interventions supported by patient-oriented outcomes, it is imperative to evaluate the current evidence regarding the use of OMM in the treatment of respiratory illnesses. The aim of this study is to review the existing evidence regarding the utilization of OMM, specifically in the treatment of patients with chronic obstructive pulmonary disease (COPD). **Method:** In order to perform a review of the existing evidence, comprehensive literature search was conducted to identify investigative studies which enrolled subjects having a diagnosis of COPD and incorporated OMM as an intervention. Articles were chosen based on those containing relevant content and were evaluated for risk of bias using a standardized tool. **Results**: Nine studies met the inclusion criteria and were reviewed for this paper. Overall, incorporating OMM into the treatment of COPD demonstrated inconsistent impact on objective pulmonary measures but when patient assessment of symptoms was included, improvement was noted. **Conclusion**: Current evidence demonstrates inconsistent findings regarding the efficacy of OMM in patients with COPD. Considering that clinical case studies and practice experience suggest this modality provides symptomatic improvement, we encourage researchers to conduct larger studies that minimize bias, incorporate patient-oriented measures, and evaluate the effect on acute exacerbations as the next steps to build the body of evidence regarding the utilization of OMM in COPD.

INTRODUCTION

Since the earliest writings of Andrew Taylor Still, MD, DO, osteopathic literature has included case reports, study proposals, and research articles assessing efficacy in treating respiratory illnesses.[1-5] Case reports have focused on somatic manifestations of respiratory disease, the role of anatomy and physiology in respiratory illness and the utilization of osteopathic manual medicine (OMM) to improve function as well as facilitate patient recovery. Investigations have evaluated the effect on recovery from infectious etiologies[3;6-9] and improvement of pulmonary function in obstructive lung diseases.[10-13]

Chronic obstructive pulmonary disease (COPD) is characterized by a chronic limitation in airflow that is progressive and not fully reversible. It is caused by a chronic inflammatory response to noxious stimuli, including but not limited to tobacco use, and resulting in parenchymal destruction and airway disease. The pathologic changes lead to air trapping and air flow limitation causing breathlessness and other classic COPD symptoms.[14] It represents the third leading cause of death in the United States[15] and fourth leading cause worldwide.[14] In addition to its role in mortality, the morbidity associated with the disease includes decreased exercise capacity and tolerance, as well as the direct and indirect costs of all medical interventions and decreased productivity in the workforce. The challenges that arise when treating the disease take a toll on the patient, the health care system, and the economy. When combined with interventions that impact the disease, such as tobacco cessation, utilization of OMM not only has the potential to slow progression, but to also improve patient functionality and healthcare costs.

When attempting to demonstrate the efficacy of OMM in treating COPD, most investigators consider evaluation and treatment of body regions highly associated with somatic manifestations of pulmonary disease, mainly in the thoracic and cervical regions as well as the ribs and diaphragm. Case reports and investigational studies have shown a correlation in somatic regions associated with viscerosomatic and somatosomatic reflex patterns related to both sympathetic and parasympathetics innervations (Figure 1).[1;2;16-17] Specifically, viscerosomatic changes related to parasympathetic innervation occur at the base of the occiput where the vagus nerve exits the cranium, somatic findings in the upper thoracic region represent changes related to the sympathetic innervation of the lungs, and the classic findings of somatic dysfunction in the region of C3-5 follows with the somatosomatic reflex pattern related to innervation of the diaphragm. Flattening of the respiratory diaphragm, rib restrictions, decreased thoracic compliance, and thoracic outlet obstruction all result from air trapping and decreasing motion of the thoracic cavity as COPD progresses.

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FIGURE 1:
Common somatic dysfunctions noted in obstructive lung disease

Cervical Region	• Suboccipital, scalene hypertonicity • OA, AA dysfunction • C3,4,5 dysfunction
Thoracic Region	• Restrictions in thoracic outlet • Hypertonicity paravertebral musculature T2-7 • Motion restrictions T2-7 • Decreased thoracic compliance, barrel chest
Rib & Other Regions	• Inaled ribs 1,2 affecting thoracic outlet • Motion restriction in ribs 5-8 • Flattened respiratory diphragm • Elevated shoulders/clavicles • Sacral extension

OA: Occipito-atlantal; AA: Atlanto-axial

The body of evidence related to OMM in COPD shows frequent discussion of regional somatic dysfunction without noting types of dysfunction present. Similarly, when case reports and studies mention OMM, some specific techniques are mentioned (thoracic pump, rib raising, doming the diaphragm), but the discussion mainly focuses on the body areas treated (Figure 2). One of the ongoing challenges associated with OMM research is determining the efficacy of an individual technique versus the impact of normalizing somatic function on the disease being evaluated.

When reviewing studies focused specifically on utilization of OMM in COPD, the predominant theory noted was utilizing OMM to decrease chest wall rigidity to improve pulmonary function tests (PFT) and in turn symptoms. Despite lacking overwhelming evidence of improved PFT results, a disease-oriented measure, a consistency is noted in subjective patient improvement. When considering the importance of patient-oriented evidence, subjective improvement in exercise tolerance and work of breathing continue to inspire investigators to explore reasons why this improvement occurs. It is the purpose of this systematic review to summarize the available evidence regarding the manifestations of COPD on the soma, and the effect of OMM on COPD.

METHODS

The objective was to perform a systematic review of the published literature on the effects of OMM in COPD.

Studies were included for review based on the following criteria: participants had a diagnosis of COPD, and use of OMM or a manipulative treatment whose description was found to be similar to OMM and would likely produce similar results. The intervention was compared to either standard care, sham manipulation, minimal touch control or patient's pretreatment baseline. The outcome measures included the effects of OMM on one or more of the following: PFTs, exercise capacity, and subjective reporting of symptoms. Ideal study design would be randomized controlled trials (RCT), however a review of the literature showed a small number of studies available and therefore other study designs were included.

A literature search was conducted using PubMed, IndexCat, OSTMED.DR, Cochrane Central Register of Controlled Trials, Google Scholar, Google Advanced, clinicaltrial.gov and TRIP database in order to identify articles for the purposes of this review. The following search terms or MeSH headings were used: manipulation, osteopathic, manipulation, spinal, lung, pulmonary disease, chronic obstructive, respiratory function test, respiratory tract disease, OMT, OMM, and COPD. The dates searched were from database inception through July 2015. Initial search results were filtered for relevance, according to our inclusion criteria, by the hospital librarian and the reviewers and the subsequent remaining articles were reviewed by two investigators. The bibliographies from relevant articles were scanned and hand searched for additional articles that met inclusion criteria.

Data was extracted using a standard table that included author, year of publication, country, study design, population inclusion criteria, participants, interventions, controls, outcomes measured, main findings, adverse effects, dropouts, comments, and limitations.

Risk of bias in individual studies

Risk of bias was accessed using the Cochrane Collaboration's tool. Individual studies were rated as having a low, high, or unclear risk of bias in the following categories: random sequence generation, allocation concealment, blinding of participants and personnel, incomplete outcome data, selective reporting and other potential sources of bias.[18]

FIGURE 2:
Techniques studied in COPD

Cervical Region	• Suboccipital release • Soft tissue • Mobilization
Thoracic Region	• Thoracic outlet release • Spinal inhibition • Increase thoracic compliance • Mobilization • Chapman's point inhibition
Rib & Other Regions	• Rib raising • Thoracic pump • Doming the diaphragm

RESULTS

Study selection

Initial search of databases was performed by the Wilkes-Barre General Hospital librarian as well as additional records identified by the researchers. Initial filter of results was performed by the hospital librarian. 282 records were reviewed by two researchers. After duplicates, records not studying COPD, records not utilizing OMM or a manipulation technique described similar to OMM, nine studies were included in the systematic review.

Characteristics of studies

The included studies originated from four different countries: four studies from the United States,[16-17;19-20] two studies from Australia,[21-22] two studies from India,[23-24] and one study from Italy.[25] Five of the studies were randomized controlled trials (RCTs), including one crossover RCT. There was one cross sectional study, two pre-test / post-test design, and one randomized cohort study. Three of the studies utilized a sham or minimal touch control.[19-20;25] Three studies had no control group.[16;13;21] Three studies included a control of a standard therapy whether it be a standard pulmonary rehabilitation program or standard medical treatment.[17;22;24] There were a variety of age ranges of included participants and severity of COPD was defined in the inclusion criteria for three of the nine studies.

Seven of the nine studies utilized an intervention that included a single or multiple OMM sessions.[16-17;19-20;23-24] Two of the studies utilized soft tissue, and spinal manipulation interventions with a descriptions that was similar to OMM treatments.[21-22] All studies measured pulmonary function tests as an outcome. Five of the studies collected subjective reports of symptoms either by phone survey or questionnaire.[17;19-22] Three studies measured exercise capacity utilizing the 6 minute walk test.[21-22;25] The specific characteristics of the included studies are presented in Table 1 *(page 32 - 35)*.

Risk of Bias Assessment

Risk of bias for included studies is summarized in Figure 3 (Review Manager version 5.3).[26]

Random sequence generation

Of the nine studies reviewed, five adequately described their method of randomization.[17;20-22;25] Noll et al 2008 stated that they utilized stratified randomization based on disease severity but did not provide a complete description of the randomization process.[19] Mascarenhas et al also did not provide a description of their randomization method.[24] Both Howell et al 16 and Bhilpawar & Arora[23] used a pre-post test design with nonrandomized sampling.

Allocation concealment

Three studies utilized sealed opaque envelopes to conceal allocation[20-22] and one study described the allocation sequence being downloaded, sealed and concealed by an investigator that did not have any clinical involvement. This investigator kept the sequence locked in a room and sequentially assigned patients based on the assignment schedule.[25] Three studies did not describe allocation concealment.[17;19;24] The remaining two studies were non-random pre-posttest design and subject to selection bias.[16;23]

Blinding of participants and personnel

Due to the nature of OMM treatments, it was not possible for the personnel providing the treatments to be blinded. Most studies did not provide a description of participant or personnel blinding therefore the risk of bias was unclear.[17;19;21-22;24] Three studies[16;20;23] did not blind participants. Zanotti et al[25] felt that their patients were adequately blinded and were not able to determine their treatment group.

Blinding of outcome assessment

Five studies provided an adequate description of blinding of personnel involved in assessing the outcome measures.[19-22;25] The remaining four studies did not provide this information.[16-17;23-24]

Incomplete outcome data

Three studies accounted for all outcome data and performed intention to treat analysis.[21-22;25] Two studies did not account for all the participants' data in their outcome analysis.[16-17] Four studies either did not report drop outs or information insufficient to determine if there was an effect on the outcomes.[19-20;23-24]

Selective reporting

Study protocols were not available so there was insufficient information to judge bias.

Other bias

Five studies declared funding sources.[16;19-22] Appropriate information regarding conflict of interest was provided for six studies.[19-23;25] Ethical approval and informed consent was described in all studies except Miller[17] and Howell et al.[16]

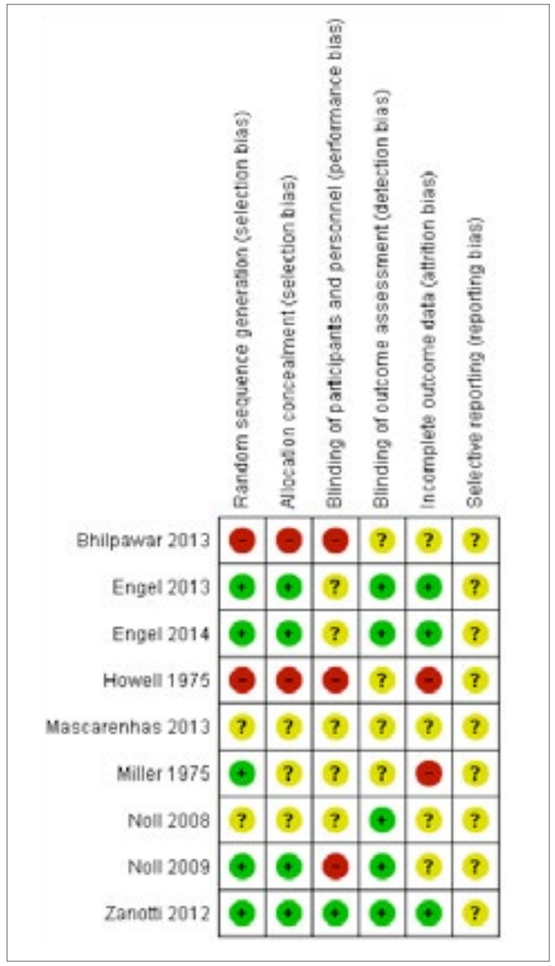
STUDY RESULTS

An overview of the main findings of each study as well as reporting of adverse effects, drop outs and other comments or limitations pertaining to each study is provided in Table 2 *(page 34 - 39)*. All studies used some form of pulmonary function tests as an outcome measure. Some studies also collected participant subjective data and/or assessment of exercise tolerance. There were varying results for PFT outcomes. Mascarenhas et al[24] did not demonstrate a significant difference in testing between the intervention group who received one five minute session of thoracic lymphatic pump (TLP) without activation plus ten minutes of salbutamol nebulization and the control group which only received the nebulization treatment. Both groups showed a significant improvement in vital capacity (VC), forced vital capacity (FVC), forced vital capacity in the first second (FEV1), and their FEV1/FVC ratio from pre to post testing. Miller[17] performed a RCT of 44 patients with COPD and found no significant difference in PFTs between the treatment and control group however some trends showing increase in residual volume (RV), Mean VC, total lung capacity (TLC) and FEV1 and decrease in partial pressure of carbon dioxide (PCO2) for the OMM group were noted. There were no description of dropouts and not all participants were accounted for in the data analysis.

Noll et al (2008)[19] studied 35 patients over age 65 with COPD and compared a single 20-minute session of seven standard OMM techniques to a sham protocol. They also received treatment of specific somatic dysfunction that was found on structural exam. The results revealed an increase in RV, TLC and the ratio of those values for the OMM group compared to the sham treatment group. The results suggested a worsening of air trapping in the OMM group when assessed 30 minutes after the treatment sessions compared to the sham group. Subsequently, Noll et al (2009)[20] studied the effect of single OMM treatments and minimal touch on PFTs of patients 50 years or older in a crossover randomized controlled trial. They hoped to demonstrate the effects seen from individual OMM techniques compared with a multi-technique protocol. The results showed that there were varying changes to PFTs for the different techniques. However in all four OMM groups there was a worsening of PFTs post-treatment (*Table 2*, *pages 34 - 39*).

FIGURE 3:

Risk of bias for included studies



+, low risk of bias, -, high risk of bias,?, unclear risk of bias

Two studies utilized a pre-posttest design. Bhilpawar & Arora[23] utilized a pre-posttest design with no control group to study 30 COPD patients selected using non-random convenience sampling and used a single 20 minute OMM session with 7 different techniques. They found that the subjects had an increase in chest expansion at the axillary and xiphisternal levels as well as a decrease in respiratory rate and improvement in peak expiratory flow rate (PEFR). The study methods and baseline characteristics of the subjects were not fully described. And there was no discussion of blinding. Howell et al 16 also used a pre-post test design. They studied 17 patients with COPD over a one year period however only analyzed the 11 patients for which they had nine months of data. They showed statistically significant decrease in PCO2, TLC and RV ($p<0.05$) and increase in O2 ($p=0.05$). A non-validated disease severity score consisting of 11 parameters from spirometry and arterial blood gases (ABGs) was the main outcome. They found an improvement in disease severity scores of 10.7%. However the non-validated nature of the score as well as the exclusion of subject data makes the interpretation of the disease severity score difficult.

Engel et al (2013)[21] performed a randomized cohort pilot study evaluating the short-term effects of these forms of manual therapy. They included 15 subjects with moderate COPD age 40-65. Participants were randomly assigned to one of three groups: soft tissue only (ST), soft tissue and spinal manipulation (ST + SM) or soft tissue, spinal manipulation and exercise (ST + SM + Ex). The pulmonary function tests they studied were FVC and FEV1. Results showed an increase in FVC in the ST + SM + Ex group compared to the ST + SM and ST only groups ($p<0.0001$). Subsequently a randomized controlled trial by Engel et al (2014)[22] studied the effects of manual therapy in conjunction with a pulmonary rehabilitation program. This study included 33 participants, mean age 65.5, with COPD who were in a pulmonary rehabilitation program (PR) and randomly assigned to PR only, ST + PR or ST + SM + PR. They performed treatments two times per week for 8 weeks between weeks 4 and 12 of PR. They assessed outcomes at week 16 and 24 of PR. Results showed that at 24 weeks the ST + SM + PR group had a significant increase in FVC compared to the PR only group ($p=0.03$).

Three studies utilized the 6-minute walk test as an outcome measure. Zanotti et al25 performed a RCT of 20 patients with severe COPD in PR. They compared four sessions of OMM tailored to the individual along with PR to PR plus a soft manipulation sham treatment. Results showed a significant decrease in RV in the OMM + PR group compared to the PR + sham therapy ($p=0.001$) and no significant difference for FEV1. The main outcome measured was results of the 6-minute walk test (6MWT). Both groups showed an increase in their 6-minute walk test, however between group analyses showed a significant increase in the OMM group (48.8m; 95% CI 17-80.6m; $p=0.04$). The two studies by Engel et al (2013 & 2014)[21-22] also utilized the 6MWT. The earlier study showed a statistically significant increase in the 6MWT for the ST + SM (120m) and ST + SM + Ex (168m) groups when compared to ST only ($p<0.0001$). Engel et al (2014)22 demonstrated a significant improvement in the 6MWT between the ST + SM + PR group compared to the ST + PR group at 16 and 24 weeks ($p=0.01$ and $p=0.02$ respectively) but there was no difference when the ST + SM + PR and ST + PR groups were compared to the PR only group.

(Continued on page 38)

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TABLE 1:
Characteristics of included studies

Author / Year /Country	Design	Population Inclusion Criteria	Participants	Intervention / Techniques used	Control	Outcomes / Measures
Bhilpawar & Arora, 2013 India	pre-posttest non-random convenience sampling	COPD with FEV1 / FVC <70%	30 patients (28 males) with COPD selected from outpt PT utilizing convenience sampling No specific baseline characteristics Ages 37-81	**Single 20 minute session utilizing 7 techniques:** Soft tissue kneading (paraspinal muscles in lower cervical and thoracic region) Rib raising Redoming the abdominal diaphragm Suboccipital decompression Thoracic inlet myofascial release Pectoral traction Thoracic lymphatic pump with activation	No control group	Chest expansion at axillary and xiphisternal level Peak expiratory flow rate Respiratory rate
Mascarenhas et al. 2013 India	Cross-sectional	Patients with stable COPD grade I-III by GOLD guidelines	50 COPD patients in pulmonary medicine dept. Recruitment not clear	**One 5 minute session** Thoracic lymphatic pump without activation plus ten minutes of Salbutamol nebulization	Control group received only ten minutes of Salbutamol nebulization	Pulmonary function tests: VC, FEV1, FVC, FEV1/FVC ratio, PEF, FEF
Howell et al. 1975 US	pre-posttest case series	COPD according to ATS criteria	17 patients with COPD over a one year period Recruitment not clear	**OMM plus routine management.** OMM "directed toward the mobilization of specific segments of the spinal column at which paravertebral tissue abnormalities were detected and at which restricted intersegmental mobility was evident."[17]	No Control group	Disease severity score derived from 11 parameters from spirometry and ABGs: pre-post testing at periodic intervals (pretreatment, 1 month and 3 months after initiation of OMM and then at 3 month intervals)
Noll et al. 2008 US	Double-blinded RCT	65 years and older with FEV1/FVC ratio <70%	35 patients OMM group: 18 pts (mean age 69.6) Sham group: 17 pts (mean age 72.2)	**Single 20 minute session of 7 standard OMM techniques:** Soft tissue to paraspinal muscles Rib raising Redoming of the abdominal diaphragm Suboccipital decompression Thoracic inlet myofascial release Pectoral traction Thoracic lymphatic pump with activation If applicable additional OMM for specific somatic dysfunctions discovered	Sham (light touch applied to the same anatomic regions for the same duration).	Baseline and post-treatment PFTs Subjective feedback on effects and blinding protocols via phone survey
Noll et al. 2009 US	Cross over RCT	50 years and older with COPD, recruited from the clinical practice, newspaper ad, local talk radio, and COPD support groups	25 subjects: mean age 68	**5 single technique treatment sessions:** 4 OMM, 1 minimal touch control 4 week wash out period **Random order:** Minimal touch control Thoracic lymphatic pump with activation Thoracic lymphatic pump without activation Rib raising Myofascial release	Minimal Touch Control	PFTs at baseline, 30 minutes post treatment Subjective report on a telephone survey
Miller 1975 US	RCT	Ages 36-65 with COPD **Height:** 145-185 cm for females 157-190 cm for males **Weight:** 41-85 kg for females 50-115 kg for males	Treatment group: n=23 Control group: n=21 Matched pairing for sex, age, gender and disease severity	**Standard Treatment plus OMM 2x per week** Methods to hyperextend the dorsal spine Techniques to increase any restrictive motion Techniques to increase lymphatic flow by applying anterior chest compression	Standard Treatment	PFTs: VC, FEV1, FEV2, FEFR, FRC, RV, TLC pH, PO2, PCO2 Diffusion studies Minute ventilation Questionnaire on Respiratory Symptoms Musculoskeletal exam

TABLE 1 (CONT.):

Characteristics of included studies

Author / Year /Country	Design	Population Inclusion Criteria	Participants	Intervention / Techniques used	Control	Outcomes / Measures
Zanotti et al. 2012 Italy	RCT: pilot study	COPD patients consecutively admitted to the pulmonary rehabilitation unit Stage III by GOLD criteria	20 stable patients with severe COPD in pulmonary rehabilitation (PR) Mean age 63, FEV1 26.9%	Pulmonary rehabilitation and 4 sessions of OMT tailored to suit the needs of the individual Treatment sessions once per week lasting 45 minutes each	Pulmonary rehabilitation plus soft manipulation sham treatments	6 minute walk test PFTs: VC, FEV1, RV, FVC
Engel et al. 2013 Australia	Randomized cohort pilot study	Age 40-65 Volunteers with moderate COPD Recruited from the general public by newspaper and radio ads	**15 subjects:** 9 male/6 female mean age 56.1 (range 49-63) moderate COPD, All white	**Subjects randomly assigned to 1 of 3 groups:** Soft Tissue (ST) ST and spinal manipulation (SM) ST, SM and exercise	No control group	FEV1, FVC Chronic respiratory questionnaire 6 minute walk test Monitoring of adverse effects
Engel et al. 2014 Australia	RCT	COPD referred by a respiratory specialist to a PR unit, ages 55-70 Non-smoker for preceding 12 months, ability to complete a 6-minute walk test	33 participants mean age 65.5 with COPD in PR	**Subjects randomly assigned to 1 of 3 groups:** Pulmonary rehabilitation ST + PR ST + SM + PR Each manual therapy session 20 minutes, before the exercise component of PR Two times per week for 8 weeks between weeks 4 to 12 of PR	PR only	BP, FEV1, FVC 6-minute walk test, St. George's respiratory questionnaire hospital anxiety and depression scale

FEV1: forced expiratory volume in 1st second; FVC: forced vital capacity; GOLD: Global Initiative for Chronic Obstructive Lung Disease; VC: vital capacity.

PEF: peak expiratory flow; FEF: forced expiratory function; ATS: American Thoracic Society; ABG: arterial blood gas; PFT: pulmonary function test; RCT: randomized controlled trial; FEFR: forced expiratory flow; FRC: functional residual capacity; RV: residual volume; TLC: total lung capacity

TABLE 2:

Summary of study results

Author / Year /Country	Main Findings	Adverse Effects / Dropout	Comments / Limitations
Bhilpawar & Arora, 2013 India	Mean increase in chest expansion at axillary level of 0.30 post treatment ($p<0.05$) Mean increase in chest expansion at xiphisternal level of 0.29 post treatment ($p<0.05$) Decrease in RR of 2.14/min ($p<0.05$) Improvement in PEFR of 11.73 ($p<0.05$)	Stated patients had no signs of discomfort	Small sample size Baseline characteristics not fully described Methods not fully described No blinding described
Mascarenhas et al. 2013 India	No significant difference in PFTs between groups Both groups showed a significant improvement in VC, FVC, FEV1, FEV1/FVC The experimental group showed an improvement in FEF 75/25	Stated technique is free from side effects	PFTs at baseline similar in both groups No description of randomization Subjects not divided based on disease severity No patient subjective data No blinding described
Howell et al. 1975 US	Improvement in disease severity scores of 10.7% Significant improvement in PCO2, O2, TLC and RV ($p<0.05$)	No description of drop outs or adverse effects	Only 11 of 17 subjects data analyzed Patients admitted at different times No description of statistical tests Missing data/patients unaccounted for Small sample size Non-validated severity score

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TABLE 2 (CONT.):
Summary of study results

Author / Year /Country	Main Findings	Adverse Effects / Dropout	Comments / Limitations
Noll et al. 2008 US	Significant improvement between OMT and control groups for 8 out of 21 pulmonary function parameters. FEF 25% (p=0.04), FEF 50% (p=0.008), FEF 25%-75% (p=0.02), and ERV (p=0.02) were significantly lower in the OMT group. RV (p=0.03) and TLC (p=0.02) were significantly increased in the OMT group. RV/TLC ratio (p=0.04) increased in the OMT group. Airway resistance decreased in the OMT group (p=0.04). Phone survey showed that both groups reported an improvement in their breathing. 53% in the OMT group and 41% in the sham group correctly guessed their group assignment.	No severe side effects One subject lost to survey follow-up Two subjects in the OMT group reported muscle soreness Four subjects in the sham group reported adverse effects including palpitations, high BP, muscle soreness and back soreness	Stratified randomization by disease severity not fully described No description of allocation concealment Small sample size Phone survey is not a validated tool
Noll et al. 2009 US	Minimal touch control: Inspiratory capacity showed a decrease from baseline post-treatment (p=0.008) Thoracic lymphatic pump with activation: Post-treatment decrease in FEFmax (p=0.001), MVV (p=0.005), ERV (p<0.0001), and SVC (p=0.04). There was a significant increase in RV (p=0.03) and RV/TLC (p=0.04) Thoracic lymphatic pump without activation: Post-treatment decrease in FVC (p=0.02), FEF25-75% (p=0.006), and MVV (p=0.02). Increase in airway resistance relative to baseline (p=0.04). Rib raising: Post-treatment decrease from baseline in FEFmax (p=0.01) and MVV (p=0.0004) Myofascial release: Post-treatment decrease in FEV1 (p=0.03), FEF25-75% (p=0.007), FEFmax (p=0.007, MVV (p=0.03), and SVC (p=0.008). No significant difference between groups from baseline to 30 minutes post-treatment. Subjects reporting perceived health benefits from the treatment: minimal touch control 41%, TLP with activation 76%, TLP without activation 67%, rib raising 68%, myofascial release 53% Subjects reporting improved breathing after treatment: minimal touch control 44%, TLP with activation 74%, TLP without activation 57%, rib raising 79%, myofasical release 50% Subjects in all group reported enjoying the treatment (71-88%) and would recommend it to others (71-95%)	Side effects were noted in 1/18 patients (6%) in the minimal touch session, 4/23 (17%) after TLP with activation, 4/21 (19%) after TLP without activation, 3/20 (15%) after rib raising, and 2/16 (13%) after myofascial release. Side effects reported were commonly muscle soreness or pain and none were severe. Missed sessions for each group described	Subjects and physicians performing OMT were not blinded Individuals collecting the data, performing the PFTs, and performing the phone survey were blinded Allocation concealment, description of randomization provided Unable to contact all patients for follow up telephone survey
Miller 1975 US	92% of treatment group reported greater walking distances, few cold/URIs, and less dyspnea than prior to treatment. Trends noted: RV: OMT group increased by 0.5L (29%), no change in control (p>0.05) Mean VC: OMT group increased 0.5 L, control group increased 0.1 L (p>0.05) TLC: OMT group increased 1.0L (17%), control group increased 0.1L (2%) FEV1: OMT group increased 2.1 L, control decreased 2.4L PCO2: OMT group decreased 5 mm Hg, control decreased 3.3 mm Hg	No description of drop outs or adverse effects	Recruitment not clear Random allocation with matched pairing No description of allocation concealment Neuromuscular exam performed by 2 physicians who were blinded to treatment group Follow up time not given / Duration of treatment not stated Not all participants/data accounted for No description of statistical analysis Small sample size
Zanotti et al. 2012 Italy	Both groups showed an increased in 6MWT PR group increased 23.7 m and PR + OMT group increased 72.5 m (p = 0.01) Between group analysis showed a significant increase in 6MWT in the OMT group compared to the PR only group (48.8 m; 95% CI 17-80.6m; p = 0.04) Significant decrease in RV in OMT + PR group compared to PR only group (-0.44L; 95% CI -0.26 to -0.62; p = 0.001) FEV1: Between group analysis showed no difference but within group analysis showed a change of FEV 1 from 0.99L to 1.13L (14%) for the OMT+PR group which is noteworthy despite not reaching statistical significance.	Reported no adverse effects or side-effects No drop-outs	Allocation concealment described Data collectors and patients blinded Statistical analysis described No patient subjective data on symptoms or quality of life
Engel et al. 2013 Australia	FVC increase in ST + SM + Ex group compared to ST + SM (1.00L) and ST only (1.01 L) groups (p<0.0001) Increase in walking distance for groups that received ST + SM (120m) and ST + SM + Ex (168m), when compared to ST only (p<0.0001) Decreased dyspnea levels reported in ST + SM (0.64) and ST + SM + Ex (0.44) groups compared to ST only group (p<0.0001)	One participant dropped out for personal reasons No major or moderate adverse effects reported Mild Adverse effects of muscle soreness after 15% of MT sessions	Random allocation described Assessor blinding to intervention ST and SM interventions administered by single clinician who was blinded to all results during the intervention phase of the study Duration 4 weeks (8 sessions at 2 sessions per week) / Small sample size Standardized duration of treatment session for each intervention group Intention to treat analysis performed

TABLE 2 (CONT.):
Summary of study results

Author / Year /Country	Main Findings	Adverse Effects / Dropout	Comments / Limitations
Engel et al. 2014 Australia	Difference between all three groups significant for FVC at 24 weeks (p=0.04) ST + SM + PR group had a significant increase in FVC at 24 weeks compared to PR only (0.40L, 98.33% CI: 0.02, 0.79; p=0.03). No difference between group for HAD or SGRQ scores. There was a difference between all three groups for the 6MWT at 16 and 24 weeks (p=0.01 and p=0.03, respectively). No difference when comparing the ST+SM+PR group or the ST+PR group to the PR only group. Significant improvement noted in the 6MWT between the ST + SM + PR group compared to the ST + PR group at 16 and 24 weeks (p=0.01 and p=0.02 respectively). No difference in blood pressure.	Two participants in the ST + PR group reported mild AE of muscle soreness Withdrawals reported	Randomization and Allocation concealment described Statistical analysis described Intention to treat analysis Baseline characteristics not all similar (gender and HAD scores) Groups not evenly distributed

FEV1: forced expiratory volume in 1st second; FVC: forced vital capacity; VC: vital capacity; PEF: peak expiratory flow; FEF: forced expiratory function; ATS: American Thoracic Society; ABG: arterial blood gas; PFT: pulmonary function test; RCT: randomized controlled trial; FEFR: forced expiratory flow; FRC: functional residual capacity; RV: residual volume; TLC: total lung capacity; MVV: maximum voluntary ventilation; ERV: expiratory reserve volume; TLP: thoracic lymphatic pump; SVC: slow vital capacity; URI: upper respiratory infection; AE: adverse effects; 6MWT: 6 minute walk test; PR: pulmonary rehabilitation; ST: soft tissue; SM: spinal manipulation; MT: manual therapy; HAD: hospital anxiety and depression score; SGRQ: St. George's Respiratory Questionnaire

Five of the studies reported some form of subjective patient data; three utilizing non-validated surveys or questionnaire[17;19-20] and two studies using a validated questionnaire.[21-22] Miller[17] utilized a questionnaire on respiratory symptoms and found that 92% of the OMM treatment group reported greater walking distance, fewer colds or upper respiratory infections, and less dyspnea than prior to treatments. The patients stated they were able to function better in their normal activities than prior to OMM treatment. No data from the questionnaire was provided for the control group, however. Two studies utilized phone surveys following treatments to collect subjective data.[19-20] Noll et al (2008)[19] found that both the OMM and sham treatment groups reported an improvement in their breathing and 53% in the OMM group and 41% in the sham group correctly guessed their group assignment. Noll et al (2009)[20] found that 71% of subjects within the minimal touch treatment group reported enjoying the treatments compared to 80-88% in the four OMM treatment groups. Most subjects would also recommend the treatment to other, ranging from 71% in the minimal touch group to ranging from 91-95% in the four OMM treatment groups. More subjects in the OMM groups reported health benefits from the treatment and improved breathing after treatment (see Table 2).

Two studies utilized validated questionnaires to collect patient subjective data. Engel et al (2013)[21] utilized the Chronic Respiratory Questionnaire (CRQ-SAS) score and found that patients in the ST + SM and ST + SM + Ex groups showed a decrease in their dyspnea levels compared to the ST only group (p<0.0001). Engel et al (2014)[22] utilized the St. George's respiratory questionnaire (SGRQ) and found no difference between the groups for SGRQ scores. They also used a hospital anxiety and depression score and found no significant difference.

One study did not mention any adverse effects.[17] There were no severe adverse effects reported in any studies and the common minor adverse effects reported were mild muscle soreness or pain which mainly resolved on their own without any treatment.[16;19-25]

DISCUSSION

The clinical case reports reviewed in preparation for this systematic review all discussed the positive impact noted when adding OMM to treatment of acute exacerbations of COPD and reinforced the positive clinical experience physicians have expressed as the basis for the studies conducted in this area. The research articles included in this review focused less on acute exacerbations and more on management of the chronic disease process. Most utilized a variation of disease-oriented markers such as PFTs, PEF, ABGs, and chest wall expansion but some also included patient-oriented outcomes such as impact on exercise capacity and frequency of symptom questionnaires. This review found that incorporating OMM into chronic disease management had the highest impact on improving patient-oriented outcomes, such as symptom improvement, while limited effect was demonstrated on disease-oriented outcomes. We also found that most of the studies had limitations associated with small study size, study design, and the potential for bias.

Previous discussions looking to explain the impact of OMM on COPD have focused on the mechanical aspect of breathing but the results of this systematic review would indicate that other means of impacting the disease should be considered as well. Techniques such as the thoracic pump and doming the diaphragm decrease congestion and improve lymphatic flow within minutes of the treatment and the evidence supports that when applied, patients report feeling better regardless of the results of lung function measurements. This may also explain the improvement noted in the case studies reviewed for this article. Improving lymphatic flow and minimizing pulmonary congestion allows the body to maximize its ability to resolve the acute disease process.

This review ran into challenges associated with limited studies that were not consistently of high quality and built from information garnered from reviewing case studies that is outside the usual spectrum of a literature review. Considering the relative infancy of osteopathic medicine and the challenges associated with performing research in OMM, case studies still serve a role in defining the impact OMM may have in treating a disease process. As knowledge and understanding of OMM study limitations increase, future investigation of OMM and COPD should minimize the challenges noted here and incorporate well-designed studies that provide evidence regarding the effects on patient-oriented outcomes. It is our hope that this review will stimulate thought regarding study design that will demonstrate the impact OMM has on treating patients with this disease process.

Considering the impact that this disease has on patients and society, continuing to explore how to best utilize OMM within the context of treating it has the potential to impact the health care system on multiple levels. Further studies might wish to focus on treatment in acute exacerbations and longer, larger studies utilizing techniques that address lymphatic flow in addition to maximizing thoracic cage function and using patient-oriented outcomes to demonstrate the value OMM can add to managing COPD.

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REVIEW ARTICLE

Treatment Options for Psoriasis

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Keywords:

Psoriasis

Psoriasis Treatment

Psoriasis Management

Psoriasis is chronic, hyperproliferative skin disorder that affects approximately 2% of the U.S. population. Treatment approaches focus on education, communication, and medications to control the disease and lessen the visible skin findings. These treatments can include moisturizers, topical steroids, vitamin D derivatives, and oral immunosuppresives. Physicians should remain aware of the common side effects, drug interactions, and toxicities of the regimens in order to prevent morbidities in these patients. Creating a therapeutic relationship with the patient will allow for optimization of the treatment plan and reduce patient anxiety and disease burden.

INTRODUCTION

Psoriasis is a multisystem disease affecting approximately 2% of the population.[1] It Is a hyperproliferative state most commonly resulting in erythematous skin papules and plaques with a silver scale.[2] Psoriasis is a chronic condition that is present throughout a patient's lifetime with periods of waxing and waning often precipitated by the initiation or cessation of treatment.[2,3]

This article will briefly describe the varying types of psoriasis, pathogenesis of the disease, how to diagnose psoriasis, and an in-depth discussion of the numerous options available to treat psoriasis. We will outline the various treatments using a stepwise approach ranging from over the counter remedies for mild psoriasis to prescription medications used for severe psoriasis. A comparison of each treatment's indication, advantages, and disadvantages will also be presented along with illustrations on how to treat patients suffering from psoriasis utilizing an osteopathic approach by focusing on the body as a unit. The goal of this review is to provide a systematic technique that one can use in order to effectively treat psoriasis patients.

BACKGROUND

Various types of psoriasis are traditionally diagnosed using morphologic descriptions. It is common for clinical findings to overlap in more than one category resulting in the implementation of numerous treatment regimens to control the patient's varying disease states.[4]

Plaque

Plaque psoriasis is the most common form of psoriasis affecting 80 to 90 percent of psoriasis patients.[1] It is defined as scaly, erythematous, patches, papules, and plaques.[2] The severity of plaque psoriasis can range from only a few plaque lesions to numerous lesions covering most of the skin surface.

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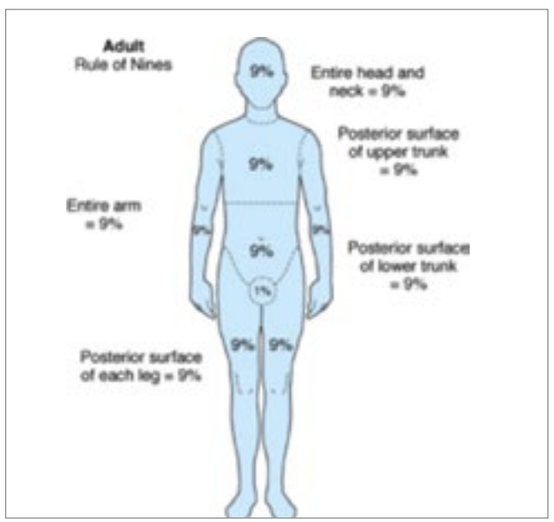
Mild to moderate disease affects approximately 80% of patients with plaque psoriasis and is defined as psoriatic lesions that cover less than 5% of the body surface area (BSA). Approximately 20% of patients are diagnosed with moderate to severe plaque psoriasis, which covers more than 5% of the body surface area or vital areas such as hands, feet, face, or genitals.[1,2,5,6] Chronic plaque psoriasis is often bilateral and symmetric.[7]

When diagnosing psoriasis, it is important to estimate the body surface area that is affected by the disease. This can easily be done using the "Rule of 9s." This divides the body into 11 equally sized areas including: head, chest, abdomen, back, buttocks, and bilaterally arm, front of leg, and back of leg each representing 9% of total body surface area.[8]

FIGURE 1:
Rule of 9's for body surface area



Erythrodermic

Erythrodermic psoriasis can result from chronic plaque psoriasis and is defined as generalized erythema that covers nearly the entire body surface area with varying degrees of scaling. As illustrated in Figure 2b, it can often appear as if the skin is severely burned. Fever, chills, and even dehydration due to fluid loss can accompany this variant of psoriasis.[4]

Guttate

Guttate psoriasis is characterized by salmon-pink drop lesions that are approximately 1-10mm in size.[1] This form of psoriasis typically has a sudden onset following a Streptococcal infection. It is seen more often in individuals younger than 30 years old *(Figure 2c)*.[4]

Inverse

Inverse or flexural psoriasis is described as lesions that develop within skin folds like axillae, groin, gluteal, and inframammary regions. Due to the moist nature of skin folds these lesions are typically erythematous plaques with minimal scaling *(Figure 2d)*.[1,4]

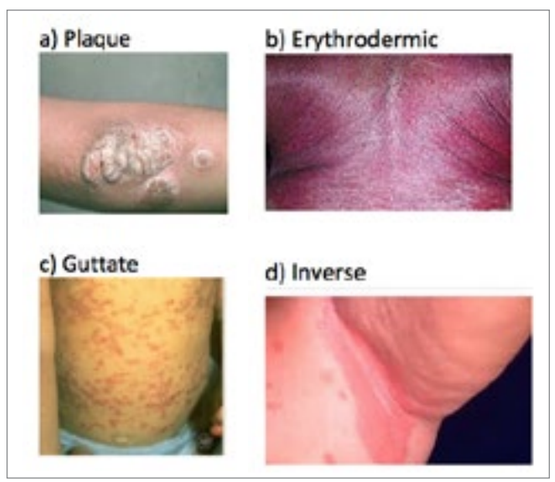
Other

There are various less common forms of psoriasis including pustular psoriasis, which can be an acute generalized condition (von Zumbusch variant) or localized to the palms and soles (palmoplantar). The von Zumbusch variant can be life threatening and is characterized by pustular lesions on an erythematous background often accompanied by fever and toxicity.[1,4] Palmoplantar psoriasis is less severe but may be functionally debilitating for the patient. These lesions can be of plaque or pustular type affecting the palms and soles.[4]

Comorbidities

Psoriasis is a complex disease of deregulated inflammation that is thought to have an immunologic pathogenesis. Due to chronic inflammation and suspected immunologic pathology there are several associated comorbidities that must be addressed when treating psoriasis.[4]

FIGURE 2:
Illustrations of differing types of psoriasis



Patients with psoriasis have an increased risk of cardiovascular disease. These patients are typically overweight or obese (BMI>25), have a higher incidence of diabetes and hypertension, and decreased high-density lipoproteins. Even after correction for risk factors in individuals unaffected by psoriasis, the probability of a psoriasis patient experiencing a myocardial infarction was significantly higher.[9] Recent studies have shown that patients with psoriasis are at an increased risk for metabolic syndrome, which is the combination of type II diabetes, hypertension, central obesity, and combined hyperlipidemia (elevated LDL, decreased HDL, elevated triglycerides).[1,10]

It is also important to note the risk of psoriatic patients developing psoriatic arthritis; which occurs in approximately 10-25% of patients and is not related to the severity of psoriasis. The most common clinical pattern is oligoarthritis accompanied by tenosynovitis of one or more hand joints.[2] According to the American Academy of Dermatology up to 90% of patients with psoriatic arthritis may also have nail changes.[4] The recently developed CASPAR (classification criteria for PsA) criteria for diagnosing psoriatic arthritic includes nail changes as a predominant feature. Therefore, if nail changes are observed the clinician's level of suspicion for arthritis should increase.[11] It is also important to note that in a small percentage of patients the presentation of arthritis will precede any skin manifestations.[11]

Apart from medical comorbidities, the prevalence of depression in patients suffering from psoriasis may be as high as 60%.[1] The psychological and emotional impact of psoriasis is difficult to assess since it may not reflect the severity of the disease. Psoriasis patients also have an increased prevalence of smoking and alcohol abuse.[1,6]

Diagnosis

The diagnosis of plaque psoriasis is based upon clinical evaluation of characteristic appearance and location of the lesions.[1,7] Most patients will complain of itchy lesions prior to diagnosis. Lesions typically present as papules and progress to form plaques. They most commonly appear on the scalp, ears, elbows, knees, umbilicus, gluteal cleft, nails, and sites of recurrent trauma.[2] The location and appearance of these lesions can significantly help distinguish psoriasis from other papulosquamous skin disorders.[3,7]

MATERIALS & METHODS

In order to research the current information pertaining to the treatment of psoriasis, a literature review was conducted using the keywords of psoriasis, psoriasis treatment, and psoriasis management. Several different search engines were used to find the current and most appropriate treatment options to treat psoriasis including PubMed, Medscape, Up to Date, and Google Scholar. To supplement these search engines The Journal of American Academy of Dermatology, American Journal of Clinical Dermatology, and learning modules published by the American Academy of Dermatology were also used.

DISCUSSION

General Approach

It is important when diagnosing a patient with psoriasis to provide education on treatment options and communicate that psoriasis is a chronic condition with no cure.[2,6,7] It may also be beneficial to refer patients to an organization such as the National Psoriasis Foundation for more information and support groups.[3,6] Realistic expectations should be explained when determining an appropriate treatment regimen with the goal of treatment being to control the disease and lessen the appearance of skin lesions.[1,7]

Topical Treatment

Topical therapy is typically first line when treating psoriasis. This option is practical for patients suffering from localized lesions or mild to moderate psoriasis affecting less than 5% of BSA.[2,3,5]

Over the Counter

There are several over the counter treatment options for plaque psoriasis. The active ingredients in treatments approved by the FDA is tar and salicylic acid.[2,3] Salicylic acid is considered a keratolytic agent that causes the outer layer of skin to shed that helps to soften psoriatic lesions and reduce the appearance of scaling.[4] Although rare, the concern of using salicylic acid is the potential for systemic absorption if it is applied to >20% BSA.[5] It can decrease the efficacy of UVB phototherapy and should be avoided prior to treatment.[2]

Tar acts to slow the hyperproliferative state of the skin and restore its appearance by suppressing DNA synthesis through lessening the mitotic labeling index of keratinocytes.[3,5] Tar also has an added benefit to reduce inflammation, itching, and scaling of psoriasis; however, it is often poorly tolerated by patients due to the foul odor, contact dermatitis, and tendency to stain clothing.[5]

Other topical treatment options available that do not contain these active ingredients can be beneficial especially if used concomitantly with other treatments. For example, heavy cream moisturizers and ointments can help skin retain moisture and reducing redness and itching. Bath solutions containing oil, oatmeal, and salts also aid in removing scales and soothing the skin.[2,4,7]

Topical Steroids

Corticosteroids are considered the main stay of topical treatments.[2,5-7] This treatment acts as an anti-inflammatory, anti-proliferative, immunosuppressant and vasoconstrictor by affecting gene transcription.[1] There are a variety of strengths and formulationsavailable that helps tailor treatment for each patient. The potency of each formulation is based on the medication's ability to produce vasoconstriction at the site of application ranging from weak to super potent preparations.

Indications

Topical corticosteroids are the first line agent for localized psoriasis (<5% BSA) and can be appropriately managed by primary care providers. When deciding on the appropriate potency for treatment the disease severity, location being treated, patient preference, and patient age are all factors that should be taken into consideration.[5,6,12] Lower potency formulations (hydrocortisone 1%) should be used on the face and intertriginous areas for a limited amount of time. The use of mid or high potency agents (betamethasone 0.05% or clobetasol propionate 0.05%) are considered appropriate for treatment of psoriasis affecting other areas of the body. The typical regimen includes two daily applications until clinical improvement occurs in which administration frequency should be reduced.[5,6]

Numerous double-blind, placebo-controlled studies have found that the use of topical corticosteroids improve psoriasis plaques; however these studies show a wide range of efficacy and only average several weeks which inhibits the assessment of long-term therapy *(See Table 1)*.[5]

Due to the variation in study design and populations make it difficult to compare each of these studies. However, a systematic review by Mason et al. has demonstrated that potent and very potent formulations are more effective at improving psoriasis plaques than mild or moderate corticosteroids.[5,13]

Disadvantages

Although topical corticosteroids are proven to have clinical benefit when treating limited plaque psoriasis, they also have side effects that must be considered. The main disadvantage to using topical treatments is lack of adherence.[5,6] This is mainly attributed to inconvenience, cost, and lack of immediate response. In order to combat the issues it is important to choose the most appropriate therapy by balancing potency to achieve a desirable outcome while also choosing a vehicle that can be tolerated by the patient.[3-6]

Topical corticosteroids are also associated with potential side effects. It is common for patients to experience local cutaneous skin atrophy, telangiectasia, striae distensae, acne, folliculitis, and purpura.[5-7] Systemic side effects are rare but can occur with long term use of potent or super potent formulations over a large BSA.[2,5] These side effects include Cushing's syndrome, osteonecrosis of the femoral head, cataracts, and glaucoma. To avoid the potential complications it is recommended that the use of class I topical steroids be limited to no more than twice daily for 2-4 weeks. Longer duration of therapy can be utilized, but appropriate monitoring including regular skin checks to assess for atrophy should be employed.[5] It is also important to note that when utilizing a potent to super potent formulation therapy should be tapered.[5] The use of topical steroid in pregnant patients is category C with unknown safety in nursing mothers.[5]

Advantages

A major advantage associated with topical corticosteroid treatment is the various strengths and formulations available. In addition to selecting the appropriate strength for your patient a variety of vehicles are also available which can significantly alter the use and penetration of the medication. Vehicle types include ointments, creams, solutions, gels, foams, tape, spray, shampoo, oils, and lotions.[3,5,6] It is important to choice a vehicle option that the patient will most likely use at the targeted site. For example, when treating the scalp shampoos, foams, or sprays are common and the patient is able to select their preference. The vehicle of choice may alter the class; for instance, flurandrenolide 0.1% as a cream is a class V, but a class I when used as a tape.[5]

TABLE 1:

Range of efficacy for each class of topical corticosteroids used in psoriasis treatment.

Class of Topical Steroid (1 - 7)	Range of Efficacy Rates	Average Duration of Therapy (weeks)
1 (Superpotent)	58% - 92%	2
2 (Potent)	68% - 74%	2.5
3, 4 (Midstrength & upper midstrength)	68% - 72%	6.7
5, 6, 7 (Least potent, midstrength, & lower midstrength)	41% - 83%	3

Non-Steroidals

In addition to topical corticosteroid treatment there is a variety of non-steroidal topical treatment options.

Vitamin D Derivatives

These formulations include calcipotriene and calcitriol, which act by binding to vitamin D receptors inhibiting keratinocyte proliferation and enhancing keratinocyte differentiation.[5] Calcipotriene has been proven effective through a systemic review of randomized control trials where only potent topical corticosteroids appeared to have a comparable outcome at 8 weeks. 70% of patients with 5-20% BSA affected by plaque psoriasis showed greater than 75% improvement in their condition compared to 19% of vehicle-treated patients.[14] Calcitriol has an additional mechanism to treat psoriasis by inhibiting T-cell proliferation and other inflammatory mediators.[6] In a systemic review, calcipotriene and calcitriol showed equal efficacy, but calcitriol appeared to be less irritating on sensitive areas of the skin compared to calcipotriene.

The greatest benefit of topical vitamin D derivatives are when used in conjunction with topical steroids.[6] Combining the use of both products has been proven to show greater benefit than with the use of either agent alone.[5]

Indications

The use of vitamin D derivatives in treatment of plaque psoriasis is considered an alternative first-line therapy.[6] For optimal therapy, a combination of topical steroids and vitamin D derivatives should be used.

Disadvantages

Side effects for vitamin D derivatives are minimal. However up to 35% of patients may experience local skin irritation including burning, pruritus, edema, peeling, dryness, and erythema.[2,15] Systemic side effects with this treatment are possible but extremely rare unless the patient is applying more than the recommended dosage of 100g/week. These side effects can include hypercalcemia and parathyroid suppression.[15] The biggest disadvantage of vitamin D derivatives is their cost compared to many generic potent corticosteroids.[6] This product is a pregnancy category C.[15]

Advantages

Vitamin D derivatives have been proven to provide improvement to plaque psoriasis, especially when used in combination with topical corticosteroids.[2,6,15] It has also been shown that with continuous use local side effects are often diminished.[7]

Retinoid

The class of drugs, specifically tazarotene, is commonly used for acne and psoriasis.[4] It works my normalizing keratinocyte differentiation, diminishing hyperproliferation, and by decreasing expression of inflammatory makers.[5] This drug has been proven safe and effective in two randomized, vehicle-controlled trails.[6] Daily administration of tazarotene gel (0.05% or 0.1%) compared favorably with the twice-daily administration of topical fluocinonide 0.05%. Furthermore, it was proven that the 0.1% cream was more effective than 0.05% cream, but had a higher incidence of local side effects.[5,6] Similarly to vitamin D derivatives, tazarotene is most beneficial when used in combination with topical corticosteroids.[2,5,6]

Indications

The use of tazarotene is an alternative first-line agent that should be used with topical corticosteroids for optimal therapy.[2,5,6]

Disadvantages

The major side effects of tazarotene are local irritation, dryness, potential photosensitizing effect, and its teratogenic properties.[2,4,5] For this reason, tazarotene is considered Pregnancy category X.[5]

Advantages

Combining this product with topical steroids or moisturizers has shown to decrease the prevalence of local irritation.[2,5,6]

Calcineurin Inhibitors

Tacrolimus and pimecrolimus, two calcineurin inhibitors used to treat psoriasis, act by blocking the synthesis of numerous inflammatory cytokines that play a role in psoriasis.[5]

Indications

The use of calcinineurin inhibitors is most effective when used on thinner skin such as the face and intertriginous regions.[5,6] Two separate eight week randomized trials found that the use of these agents show clearance of lesions or excellent improvement versus the placebo. However, a separate study of 80 patients with intertriginous psoriasis showed that the use of betamethasone valerate 0.1% was more effective than pimecrolimus.[6] It is recommended that these agents be used when topical treatment of the face or intertriginous regions are required for a prolonged period. The use of these agents has reduced side effects compared to the long-term risk of skin atrophy seen in chronic topical corticosteroid use.[5]

Disadvantages

The most common adverse effects of these medication is local burning and itching, which appears to be more significant in patients treated with tacrolimus ointment versus pimecrolimus cream.[2,5] These drugs also have a black box warning due to the lack of long-term safety data. In 2005, there was an alert placed on these medications about a potential link with cases of lymphoma and skin cancer; however, no definite causal relationship has been established.[5,6] This treatment is considered a pregnancy category C.[5]

Advantages

The efficacy of calcineurin inhibitors on sensitive areas such as the face and intertriginous regions is their biggest advantage. The ability to use this treatment without the long-term side effects of chronic topical corticosteroids is also very beneficial.[5,7]

Anthralin

The use of anthralin was formally the mainstay of treatment.[5] The exact mechanism in which this drug works is not completely understood; however, it is thought that anthralin acts by preventing T-lymphocyte activation and normalizes keratinocyte differentiation by acting directly on the mitochondria.[5]

Indications

This formulation is no longer commonly used due to its cosmetic side effects.[7]

Disadvantages

Anthralin commonly causes local skin irritation and staining of lesional and paralesional areas.[2,5] It has been demonstrated that anthralin is less efficacious than topical vitamin D or potent topical corticosteroids.[7] Anthralin is a pregnancy category C.[5]

Advantages

Physicians can use this medication as a short contact treatment in an outpatient setting. In patients with well-defined lesions, petrolatum or zinc oxide can be applied to the surrounding area prior to application of anthralin to the lessen the adverse effects.[5]

Combination Therapy

Topical therapies are most beneficial when used together with other topical Treatments.[5] There are several formulations available in order to achieve maximal therapy.

Corticosteroid & Salicylic Acid

Salicylic acid has been shown to improve the efficacy of corticosteroids by increasing penetration.[5] To ensure the risk of toxicity is not increased when adding salicylic acid to steroid treatment it is recommended that the corticosteroid should not exceed medium potency. The use of this combination is a category B recommendation and should be used when treating especially thick or scaly plaques.[5]

Corticosteroid & Vitamin D Derivatives

This combination is more efficacious than the benefit of using either as monotherapy.[2,5,6] In a four-week trial study with 1603 participants 48% of patients treated with combination calcipotriene 0.005% and betamethasone 0.064% achieved clear or almost clear results compared to 16.5% and 26.3% in patients treated with calcipotriene or betamethasone alone, respectively.[5] The use of this drug in treating plaque psoriasis in all areas of the body excluding the face is a grade A recommendation and should be considered as a first line agent when choosing an initial topical therapy option.[5]

Corticosteroid & Tazarotene

It has been demonstrated that adding topical corticosteroids to tazarotene reduces the irritating side effects of tazarotene.[4,7] Combination therapy has several potential benefits including increasing the duration of treatment benefit, increasing length of remission, and decreasing steroid induced atrophy.[5,6] This combination is a category A recommendation and could be considered first line when determining an option for optimal topical therapy.[5]

Systemic Therapy

Conventionally systemic treatment options are reserved for patients with severe psoriasis (>10%BSA); however some patients with limited psoriasis have been treated with systemically if their condition is causing debilitating symptoms such as lesions localized to palms, soles of the feet, or scalp.[2,6] Patients being treated systemically for there psoriasis should be seen regularly by a dermatologist, but it is important as a primary care physician to be aware of the potential side effects of the systemic agents, be able to recognize and monitor for adverse effects.[1,2] There are three treatment modalities that are commonly used, phototherapy, oral medications, and biologic agents.

Phototherapy

UVA and UVB wavelengths have been used to treat psoriasis. It is thought they have a direct immunosuppressive effect on Langerhans cell and an indirect immunosuppressive effect on cytokines by blocking the activation of T-helper cells.[6,16] The most commonly reported adverse effects of this therapy is erythema, itching, burning, and stinging; these typically can be managed by altering the duration of therapy. UVA and UVB therapy should be managed by a dermatologist with appropriate training and expertise in this area in order to minimize adverse effects.[4,16] Patients with a known history of lupus erythematosus or xeroderma pigmentosum should avoid phototherapy. Any patient with a positive history for melanoma, multiple risk factors for melanoma, are immunosuppressed resulting from organ transplant or taking photosensitizing mediations should be carefully screened prior to starting therapy.[6,16] As a primary care physician it is important be cautious of any changes in medication regimens that may increase a patient's susceptibility to adverse effects while receiving phototherapy. The largest concern with UVA phototherapy is the increase risk for non-melanoma and melanoma skin cancer; however, several studies have failed to show this correlation with UVB therapy.[16] It is also important to note

that pregnancy is not a contraindication to receiving UVB therapy and should be considered first line therapy in patients requiring a systemic approach with plaque or guttate psoriasis.[16]

Oral Therapy

The three most commonly prescribed oral agents for treating severe psoriasis is methotrexate, cyclosporine, and acitretin.[2,7] Each of the medications have different mechanisms of action and require various monitoring; however they are all known to cause organ toxicities. In 2014, the US Food and Drug Administration approved a new oral medication, apremilast, to treat patients with moderate to severe psoriasis.[6]

Methotrexate

Oral methotrexate competitively inhibits dihydrofolate reductase, which decreases the synthesis of folate cofactors required to make nucleic acid. When used at a therapeutic low dose methotrexate acts to suppress the immune system through inhibiting the proliferation of lymphoid tissue.[15] It is common for patients taking methotrexate to experience mild nausea, fatigue, anorexia, and stomatitis. These adverse effects can be reduced by splitting the dose, administering before bed, or by supplementing with folate.[6,15] Methotrexate is also associated with pulmonary fibrosis, hematologic abnormalities, and hepatotoxicity. Prior to the initiation of therapy patients should receive a thorough physical along with lab testing to include CBC with differential, liver function tests, and creatinine.[15] Pulmonary fibrosis should also be ruled out in any patient presenting with new pulmonary symptoms such as a cough. It is important to avoid prescribing other hepatotoxic drugs or drugs that may interfere with renal excretion of methotrexate including NSAIDs and penicillins. Methotrexate is contraindicated in pregnancy and lactation.[6,15]

Cyclosporine

This drug is a calcineurin inhibitor, which leads to decreased levels of IL-2, IL-4, and inhibits activation of T-cells.[4,15] Cyclosporine is typically reserved for treatment of significant flares that are unresponsive to other therapies, patients with severe psoriasis, or as a bridging agent when new systemic agents are being introduced.[15,17] It is associated with nephrotoxicity, hypertension, hypertriglyceridemia, and increased risk for developing cutaneous squamous cell carcinoma.[15] Due to the nephrotoxicity and hypertension associated with usage, monthly creatinine levels and yearly glomerular filtration rates are indicated.[4,15] Patients with creatinine levels greater than 25% of baseline on two separate occasions should decrease their dose 25%-50%.[15] A dose reduction should also be considered in patients with no previous history that develop hypertension. Cyclosporine is metabolized by cytochrome P450 isoenzyme CYP3A4, indicating careful evaluation of other medications prior to initiating therapy.[6,15] It is also important to keep this in mind when changing the patient's medication regimen for any other comorbid conditions. As with any systemic therapy for treating psoriasis a thorough history and physical with labs should be conducted prior to prescribing cyclosporine. Cyclosporine is contraindicated in combination therapy with PUVA or UVB due to the increase risk of squamous cell carcinoma. This drug is category C for pregnant patients.[15]

Acitretin

Oral retinoids are vitamin-A derivatives that act to treat psoriasis by modulating epidermal proliferation and differentiation by exerting an anti-inflammatory and immunomodulatory effect.[15] There are several adverse effects that have been associated with acitretin; the most severe being its teratogenicity. It is pregnancy category X due to its potential to cause cardiovascular, ocular, auditory, central nervous system, craniofacial, and skeletal abnormalities.[6,15] The half-life of acitretin is significantly increased with the ingestion of alcohol; potentially taking up to three years for the drug to be eliminated from the body and therefore should be avoided in women of childbearing age.[15] It is common for patients taking acitretin to experience mucocutaneous side effects including dry eyes, nasal and oral mucosa, hair loss, and epistaxis in varying degrees. Patients who are being maintained on acitretin should obtain a lipid profile every 2 weeks for the first 8 weeks and then every 6-12 weeks after that due to the reported effect on triglyceride levels. Adverse effects of acitretin may be exacerbated when taken concomitantly with drugs that are metabolized by cytochrome p450.[15] Studies have shown that acitretin in combination with phototherapy is more effective than either as monotherapy and decreases the risk for squamous cell carcinoma.[6] Prior to initiating therapy it is important to conduct a thorough history and physical, obtain a pregnancy test, lipid profile, and liver function tests.[6,15]

Apremilast

This newly approved treatment acts by inhibiting phosphodiesterase-4 leading to a reduced production of cytokines that are thought to be involved in the pathogenesis of psoriasis.[6] In two randomized trials, 33% and 29% of patients taking apremilast achieved a 75% improvement in their psoriasis compared to 5% and 6% of the placebo groups.[6] The reported success rate of this treatment option is lower than those achieved by cyclosporine, TNF-α inhibitors, and ustekinumab.[6] Apremilast has been reported to cause short-term diarrhea typically occurring during the onset of treatment and improving with continued use.[6] Research has demonstrated that titrating patients up to the recommended dose improves the tolerability of treatment. Other commonly reported side effects of apremilast include nausea, upper respiratory infection, headache, weight loss, and an increased risk for depression.[6] Apremilast is metabolized by cytochrome p450 and has been shown to have a reduced efficacy if given with an inducer. It is also recommended to reduce the dose of Apremilast in patients with severe renal impairment (CrCl < 30mL/min).[18] Safety and efficacy of this treatment option has not been established in patients younger than 18 years old. It is classified as pregnancy category C and has not been adequately studied in pregnant women.[18]

Biologic Agents

Biologic agents are a relatively new approach to treating psoriasis and are most commonly administered subcutaneously or intravenously.[7] The biologic therapies that are currently available in the United States include etanercept, infliximab, adalimumab, which all act to inhibit TNF-α, and ustekinumab, which is a human monoclonal antibody that targets IL-12 and IL-23. Biologic agents are routinely used when traditional systemic agents fail or are unsuitable due to comorbidities.[1,6]

TNF-α Inhibitors

Etanercept, infliximab, adalimumab all act by inhibiting the pro-inflammatory cytokine TNF-α.[1,7] Each of these drugs increases the risk of infection particularly in the upper respiratory tract.[7] Due to the subtle presentation of this adverse effect, it is important to conduct regular monitoring in these patients.

In the event the patient requires treatment with antibiotics, the TNF inhibitor should be withheld and should be avoided in any patients with chronic or recurring infections.[1] It has also been noted that TNF-α has an important role in the host response to tuberculosis (TB), putting patients taking TNF-α inhibitors at an increased risk for developing TB or experiencing a reactivation of TB. Prior to initiating therapy all patients should obtain testing for TB.[1,6,7] Additional adverse effect of this medication is the association with peripheral and central demyelinating disorders, heart disease, drug-induced lupus-like syndrome, hepatic disease, lymphoma, and skin cancer.[1] These effects warrant ongoing physical exam, TB testing, CBC, and LFT.[1,7] In general TNF-α inhibitors should be avoided in patients who have Multiple Sclerosis (MS), a first-degree relative with MS, or any active infection. Extreme caution should also be taken when prescribing TNF-α inhibitors to patients with heart failure. Due to its immunosuppressive effect, it is also important patients to avoid any live vaccinations. These drugs are considered pregnancy category B.[1]

IL-12/23 Blockers

The FDA approved use of Ustekinumab in 2009 to treat patients with moderate to severe psoriasis.[6] There has been occasional injection site reaction and rare reports of serious infection and cardiovascular events with usage of this drug. It requires similar monitoring as the other biologic agents including PPD, LFT, and CBC with ongoing physical examination. Ustekinumab is also a pregnancy category B.[6, 17]

Additional Treatment

The evidence linking psoriasis to metabolic disease is rapidly expanding and although this association does not infer causality it is vital that patient's with psoriasis be evaluated for the concomitant presence of these diseases.[10,12] By using a targeted intervention approach for patients with psoriasis, early detection of diseases that are in the spectrum of metabolic syndrome can help reduce mortality.

In addition to screening, patients should be encouraged to correct any modifiable cardiovascular risk factors including smoking cessation and lowering their BMI.[10,12] Although the predominant visual manifestation of psoriasis is cutaneous, it also affects the patients mind, body, and spirit. It can be a very aggravating disease for patients and it is vital that as a provider you spend adequate time with these individuals to address every aspect of the disease.[6] Patients suffering from psoriasis have an increased risk for psychological disorders and psychosocial disability due to the affected perception of themselves.[1,6] These symptoms can be alleviated with counseling, support groups, or psychoactive medications.[6] Due to the immunologic pathogenesis of psoriasis, it is also important to maintain the osteopathic principle that the body is capable of self-regulation, self-healing, and health maintenance. Treating the whole patient by addressing mind, body, and spirit can help improve overall quality of life.

CONCLUSION

Psoriasis is a chronic inflammatory condition affecting approximately 2% of the Population.[1] There are several approaches to treating this disease ranging from over the counter treatments to biologic injectable agents. An algorithm for approaching the treatment can be found in Figure 3. Treatment is based off the type of psoriasis and where on the body the patient is affected. When managing patients suffering from psoriasis it is important to consider the effect it has on the mind, body, and spirit paying close attention any psychological changes and monitoring for co-morbid conditions such as metabolic syndrome. The treatment of psoriasis can be complex and very frustrating for patients. With appropriate monitoring and collaboration with a dermatologist as needed, can help patients set realistic treatment goals and have an increased quality of life.

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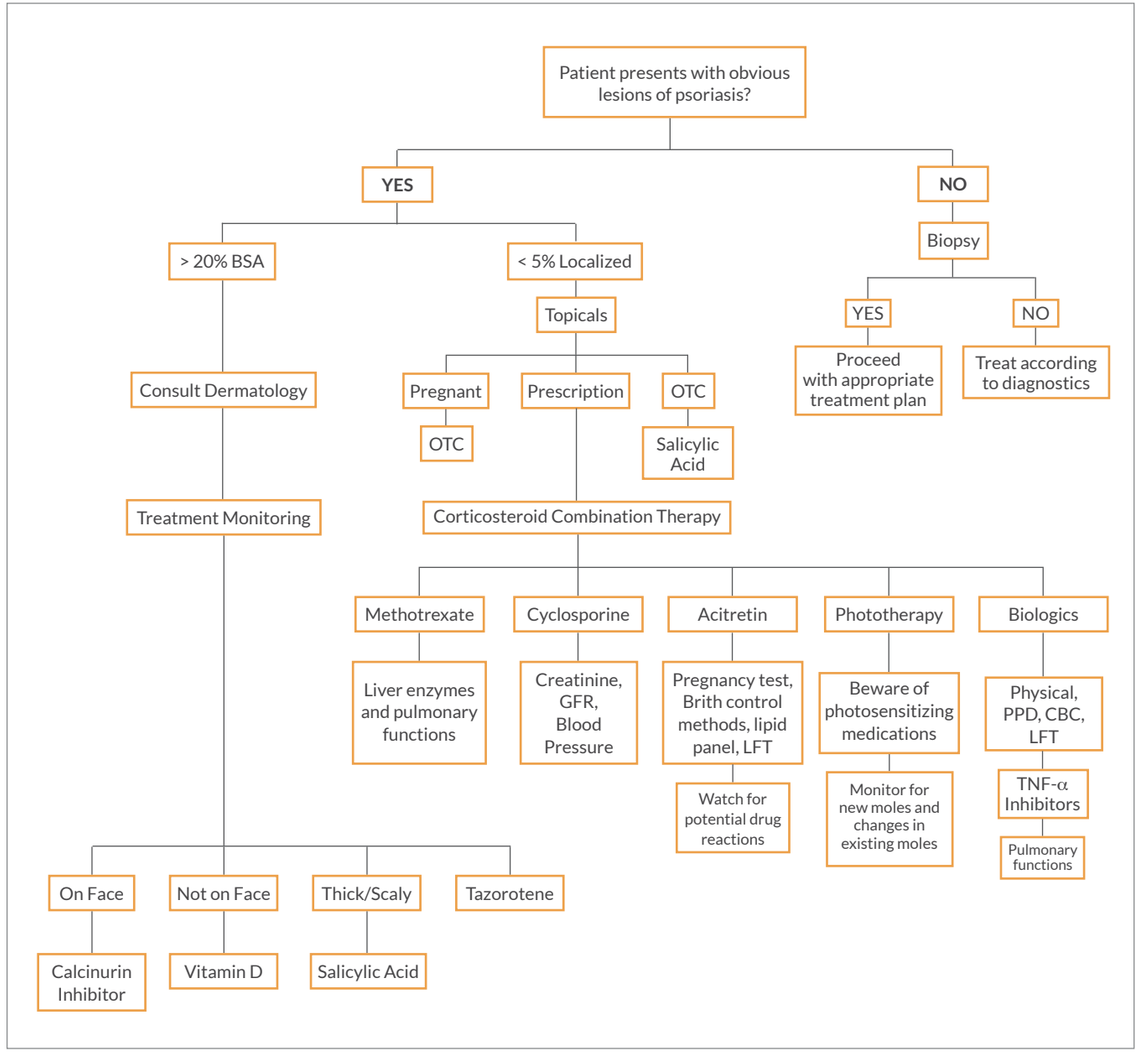
FIGURE 3:

Treatment Algorithm for Psoriasis



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Periorbital Rash

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The patient is a 47-year-old white female who presents to the clinic with right eye pain and redness of two days duration. She describes the pain as burning and constant with an intensity of 8/10. The eye problem was preceded by congestion in the ipsilateral maxillary sinus as well as pain in the ipsilateral ear and throat. She also reports a headache localized to the right periorbital region with intermittent, stabbing pains radiating to the right ear. She has tried OTC decongestants and analgesics with only temporary relief. She also tried hot/cold compresses with no relief. She has no fever and no pain or rash anywhere else on the body. Medical and family histories are noncontributory. She works as a second grade school teacher. She reports no known sick contacts but does admit to increased stress lately due to family issues.

QUESTIONS:

1. What is the diagnosis?
   a. Viral conjunctivitis
   b. Ramsay-Hunt syndrome
   c. Impetigo
   d. Rhus dermatitis
   e. Shingles with ocular involvement

2. How is this condition diagnosed?
   a. HSV titers
   b. Slit lamp exam
   c. Thorough history and physical
   d. Tzank smear
   e. All of the above aid in diagnosis

3. How is this condition treated?
   a. Aggressive pain control
   b. Antiviral/anti-inflammatory ophthalmic formulations
   c. Supportive measures
   d. Systemic antiviral therapy
   e. All of the above

FIGURE 1:

V1 dermatomal distribution of vesicular rash on erythematous base

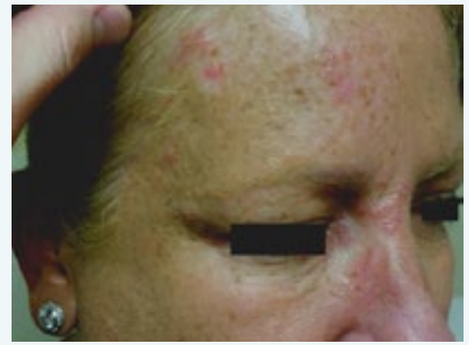
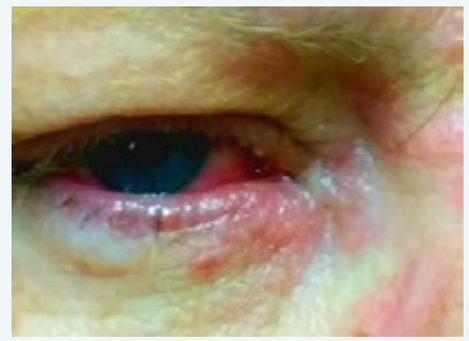


FIGURE 2:

Conjunctival injection with concomitant hyperemia and blepharitis



ANSWERS

1. What is the diagnosis?

The correct Answer is: E) Shingles with ocular involvement

The given clinical history is most suspicious for ocular shingles, also known as herpes zoster ophthalmicus (HZO). The involvement of the V1 dermatome is clearly seen (Figure 1). There is typically a flu-like prodromal phase that precedes the skin eruption. This phase can consist of fever, malaise, headache, and ocular pain.[1] Viral conjunctivitis does not usually have a significant skin rash and tends to be bilateral with significant lymphadenopathy, watery discharge, and fever.[2] Ramsay-Hunt Syndrome (RHS) typically presents with herpetic lesions involving the ear and cervical dermatomes and often is accompanied by facial hemiparesis on the affected side. Impetigo presents with pathognomonic, "honey-crusted" lesions which are usually more prominent around the mouth and nose with regional lymphadenopathy being present in 90% of cases.[4] Rhus dermatitis refers to an allergic phytodermatitis caused by exposure to the oils of plants in genus Toxicodendron. Most common in the US are poison oak, poison ivy, and poison sumac. Usually, there is a history of outdoor activities in the past 1-14 days in which the patient remembers having contact or could have had contact with these plants. The skin lesions tend to be intensely pruritic and linear in morphology. This patient had no such contact and did not complain of pruritus.[5]

2. How is this condition diagnosed?

The correct Answer is: E) All of the above

HZO is primarily a clinical diagnosis with a thorough history and physical being the key aspect. HZO is an ophthalmologic emergency, so prompt referral to an ophthalmologist for a full eye exam is recommended. A skin scraping can be performed using a 15 blade to scrape the base of the vesicle. Direct fluorescence antigen (DFA) testing can then be performed.[6] With the advent of better serological techniques, Tzank smears are not routinely performed anymore. If done, it would show the characteristic multi-nucleated giant cells upon microscopic examination.[7] HSV titers can be helpful to rule out herpes infection in equivocal cases.

3. What is this condition treated?

The correct Answer is: E) All of the above

Aggressive pain control is often necessary due to the sometimes severe pain and disability associated with post-herpetic neuralgia (PHN). In a meta-analysis of PHN pain management options by Hempenstall et al, sufficient evidence was found to support the use of strong oral opioids, TCAs, gapapentin and pregabalin. Often, a combination of these drugs is necessary to provide ample relief.[8] If ocular inflammation is present (Figure 2), then ophthalmic formulations of steroids (e.g. prednisolone), antivirals (e.g. ganciclovir), and cycloplegics (e.g. atropine) may be used. Their specific dosage intervals and combinations are decided by the ophthalmologist after thorough slit lamp exam. Supportive measures such as avoiding stress and sunlight while applying cool compresses are recommended. Oral antiviral therapy has been shown to reduce the duration of active infection.[6] If started within three days of the acute onset of rash, valacyclovir and famciclovir have also been shown to reduce the severity and incidence of PHN. An effective and accepted regimen for HZO consists of acyclovir 800 mg by mouth five times a day for one week. An additional advantage to this treatment is the low cost of acyclovir, particularly important for uninsured patients.[9]

DISCUSSION

The herpes virus and its "creeping" lesions have been described by humans since ancient times. However, it was not until the 1880s that the link between chicken pox and herpes zoster was suggested. This link was not definitively proven until the 1950s, leading to the development of a live, attenuated vaccine in 1974.[10] Approximately 20-30% of the population is affected by herpes zoster at some point in their lifetime and herpes zoster ophthalmicus (HZO) affects roughly 10-20% of those individuals.[11] In 2006, Merck received FDA approval for Zostavax® a live vaccine for patients 60 and older. A 2005 NEJM study by Oxman et al demonstrated a reduction in herpes zoster of 51.3 % in the vaccinated group.[12] Given these innovations, one would expect to see the incidence of HZO begin to fall but long-term studies and post-marketing research are needed to further evaluate the impact of Zostavax® on HZO. The risk factors for HZO appear to be consistent with the risk factors for developing shingles, namely advancing age and immune compromise. Data on why some people with shingles develop eye involvement specifically is lacking and needs further study.[1]

Herpes zoster ophthalmicus is caused by a reactivation of the varicella zoster virus (VZV) occurring in the ophthalmic branch of the trigeminal nerve (Figure 1). VZV is a member of the alpha-herpesvirus family and infects humans exclusively. Initial infection usually occurs in childhood and results in acute varicella or "chicken pox." Afterwards, the virus establishes lifelong latency and remains dormant in the cranial nerve and dorsal root ganglia.[13] Upon reactivation, the neurotropic herpes virus will travel from the trigeminal sensory ganglia up to the basal epithelium of the eye, emerging at the corneal surface where virus shedding occurs.[14] Vesicular eruptions often occur on the skin throughout the V1 dermatome (Figure 1). Of note, lesions involving the nose often portend ocular involvement. This phenomenon is coined Hutchinson's sign, and is due to the dual innervation of the nose and the globe of the eye by the nasociliary nerve.[15]

Patients usually present with a typical rash and history of a prodromal phase that preceded the rash. The prodrome typically consists of nonspecific symptoms such as malaise, fever, headache and pain in the affected eye and forehead. With the appearance of the vesicular rash, patients can exhibit conjunctival hyperemia (Figure 2), episcleritis, and drooping of the eyelid.[8,16] If untreated, keratitis and iritis occur often, and can lead to permanent loss of function.[8,13]

The potential for permanent loss of vision underscores the urgent nature of HZO and the need for early diagnosis and prompt ophthalmologic examination. The diagnosis of HZO is largely clinical and a high degree of suspicion must be maintained by the physician when evaluating periorbital skin lesions. Culture of herpes zoster is difficult due to the labile nature of the virus and thus is not routinely performed, especially with the advent of more effective laboratory techniques. Direct immunofluorescence assays can be helpful in differentiating herpes zoster infection from herpes simplex. Polymerase chain reaction (PCR) testing can be done on infected fluids and tissues to detect the herpes zoster virus.

Treatment for HZO is usually deferred to an ophthalmologist with expertise in managing corneal diseases. Systemic antiviral therapy remains the crux of therapy and is important to help reduce the progression of ocular complications. Oral acyclovir and its prodrug valcyclovir have been well studied in the setting of HZO and are the mainstays of treatment. Several randomized controlled trials (RCT) have shown that early treatment with these agents can mitigate pain and have a favorable effect on the incidence of post-herpetic neuralgia, one of the most feared complications of herpes zoster infection. Also, if taken within the first three days of the rash appearing, treatment with acyclovir has been shown to reduce the amount of viral shedding and accelerate the resolution of skin lesions.[13] Oral steroids can be used in cases of significant ocular inflammation, such as uveitis. If ocular inflammation is significant, topical ophthalmic preparations including NSAIDs, steroids, and lubricating agents may also be used.[12,13,17] the patient in this vignette was referred to an ophthalmologist and treated with oral acyclovir, ophthalmic ganciclovir and ophthalmic tobramycin/dexamethasone, to which she responded well.

HZO, while somewhat uncommon, is a very important entity to recognize. Failure to diagnose and treat this condition early can have devastating effects, including loss of sight. Although the advent of the shingles vaccine and newer antiviral medicines have decreased the occurrence of HZO, clinical suspicion must always be maintained when evaluating ocular and periorbital skin lesions. This particular patient was relatively young and otherwise healthy. Given her profession (2nd grade teacher) and somewhat subtle rash, it could have been easy to attribute the conjunctivitis to another cause, and in doing so, miss a vital diagnosis.

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CALENDAR OF EVENTS

2016

June 3 - 5, 2016
Maine Osteopathic Association
2016 Annual Oceanside Convention
Samoset Resort
Rockport, Maine
www.mainedo.org

July 27 - 31, 2016
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Champions Gate, Florida
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August 4 - 7, 2016
TOMA & Texas ACOFP Joint Annual Convention
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August 5 - 7, 2016
POFPS 41st Annual CME Symposium
Hershey Lodge
Hershey, Pennsylvania
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August 11 - 14, 2016
North Carolina ACOFP Annual Meeting
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September 20 - 24, 2016
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www.aafp.org

November 3 - 6, 2016
Inaugural Joint IOMS Annual Meeting & Scientific Seminar
Hilton Chicago
Oak Brook, Illinois
www.ioms.org

December 2 - 4, 2016
IOA Annual Winter Update
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MARCH / APRIL 2016 ANSWERS: 1. B 2. A 3. B 4. B 5. A 6. C 7. A 8. C 9. B 10. A

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DIABETES 2016 UPDATE

Peter Zajac, DO, FACOFP, Author

Amy J. Keenum, DO, PharmD, Editor • Ronald Januchowski, DO, FACOFP, Health Literacy Editor

The American Diabetes Association (ADA) recently published their updated guidelines for the care of patients with diabetes.

The standards address the importance of an individualized, patient-centered approach. It also stressed a team-based approach towards helping patients with diabetes. In addition, it provides a guide for treating different patients. It also helps create strategies for helping diabetic patients who struggle with knowledge problems, mental illness, food insecurity, and HIV.

Medical Care & Treatment Options:

If you have questions about the updated ADA standards for diabetes management, please contact your Osteopathic Family Physician. Your physician can diagnose diabetes with a thorough history and physical exam along with appropriate tests. Management includes the right treatment plan and regular visits with your doctor. Your family doctor will help you choose which current recommended treatment(s) will work best for you. In case of any emergency, you should call your doctor or 911 right away.

Updated ADA Standards for Diabetes Management Include:

- Adults without symptoms of any age, who are overweight or obese (Body Mass Index: BMI ≥ 25 or ≥ 23 in Asian Americans) and have one or more additional risk factors for type 2 diabetes should be checked for diabetes. For all patients, testing for diabetes should begin at age 45 years.
- All people with diabetes should participate in a Diabetes Self-Management Education and Support program. This program should have information that patients need to prevent the onset of diabetes and complications.
- Your doctor should encourage you to eat a healthy diet with whole grains, beans, fresh vegetables, and fruit (instead of simple sugars and carbs). If your doctor has no concerns about you doing exercise, you should perform at least 150 min/week of physical activity (such as brisk walking) over at least 3 days/week to achieve the appropriate weight loss.
- Weight loss medications may be helpful in addition to diet, physical activity, and behavioral counseling for selected patients with type 2 diabetes and a BMI ≥ 27. Bariatric surgery may be considered for adults with a BMI > 35 and type 2 diabetes, particularly if the diabetes or associated comorbidities are difficult to control with lifestyle and medication therapy.
- Do not smoke cigarettes, use other tobacco products, or e-cigarettes.
- Your physician should treat your blood pressure to a goal of < 140/90 mmHg.
- Aspirin therapy should be considered as a primary prevention strategy in most men and women with diabetes who are ≥ 50 years of age and have at least one additional major risk factor (family history of premature heart disease, high blood pressure, high cholesterol, protein in the urine, smokers) and not at risk for bleeding.
- An eye doctor should do a dilated & complete eye exam yearly for all diabetics. If you have type 1 diabetes, the first exam should be within 5 years of the onset of diabetes. Patients with type 2 diabetes should have the same eye exam performed at the time of diabetes diagnosis. Your doctor may consider exams every 2 years if you have no symptoms and previous exams were normal.
- Your physician should assess you for diabetic nerve damage at each visit. This exam should start at the time of type 2 diabetes diagnosis & about 5 years after diagnosis with type 1 diabetes and at least annually thereafter. This includes a complete foot exam.
- If you are a woman with diabetes who is of childbearing age, your physician should counsel you about the importance of near normal blood sugar control before planning pregnancy.

SOURCE(S): American Diabetes Association, Diabetes. Gov, and Up-To-Date.

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