

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

September/October, 2017

Volume 9 | Number 5

# EDITOR'S MESSAGE

Time Invested Now Helps the Patient Later

#### **REVIEW ARTICLES**

Lifestyle Management for People with Diabetes - What You Need to Know from the ADA Standards of Care

A Guide to Treating the Symptoms of Menopause

Parsonage Turner Syndrome

Evaluation & Management of Functional Abdominal Pain in Children

#### **CLINICAL IMAGES**

Subcutaneous Inguinal Mass







# 2018 CALL FOR PAPERS

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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- Approach to Polyarthritis for the Primary Care Physician
- · Chronic Abdominal Pain: Tips for the Primary Care Provider
- · CPPD: Common and Under Recognized
- · Direct Primary Care: Emerging Practice Alternative
- · The Food Allergy Revolution
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- · Newborn Disorders & Nutritional Guidance
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   (Help define the science of engaged research, provide tangible examples
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The content should include the following:

Abstract Discussion
Introduction Conclusions

Methods Acknowledgments

Results



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# EDITOR'S MESSAGE

# Time Invested Now Helps the Patient Later

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

A review article related to the everyday non-pharmacologic management of diabetes is included this month. This is a common challenge to most osteopathic family physicians. When patients with diabetes are diagnosed, present for their annual visit, or have evidence that further changes are required, counseling is a part of that care. Treatment of diabetes is a four-legged table. The first leg is pharmacotherapy that supports the patient and has been reviewed in other articles in this journal. But this is a condition best not treated with drugs alone.

The table's second leg is exercise. Aerobic exercises, like walking, running, or swimming; resistance training that increases muscle mass; balance improvement exercise like yoga or tai chi, and flexibility, like stretching, needs to be a regular part of the life of a diabetic patient. Patients need to organize their time to include exercise.

The third leg, psychological support is required to help a patient manage their diabetes. If a patient is depressed it can be very hard to do the self-care required to manage diabetes.

The fourth and final leg is nutrition counseling that describes the multi-factorial dietary issues related to diabetes care. Many patients have access to a dietician and referral may be made to improve their dietary choices. Some physicians work in health care systems where there are teams of professional diabetes educators or there may be group visits to aide diabetic patients. Some osteopathic family physicians work in settings where the osteopathic physician provides much of this counseling. We can do all of this counseling but it takes time. Time invested now helps the patient later.

In A Guide to Treating the Symptoms of Menopause, we are asked, "What if instead of waiting for women to bring up these issues, we asked our patients about them during comprehensive exams?" By OFP's proactively addressing the health problems of menopausal women, the author delves into the possibility of being able to improve their overall quality of life.

OFP primarily focuses on the common diagnoses we see every day, the exception this issue is a review article about one of the less common causes of shoulder pain, Parsonage-Turner Syndrome. Sounds very painful and might require narcotics.

Our visual case this month describes a 27-year-old female presenting to the E.R. with a subcutaneous inguinal mass. Not to be a spoiler, because many of you like to guess the answer - let me add we have a thorough review with questions for that section.

A condition often seen in primary care is functional abdominal pain in children. The article in this issue reviews the evaluation and management of the diagnosis of exclusion.

Hope everyone had a great summer.

# FROM THE PRESIDENT'S DESK



### The Patient-Centric Practice - Small Changes Keep Patients Coming Back

Rodney M. Wiseman, DO, FACOFP *dist*. 2017 - 2018 ACOFP President

Family Medicine is the "gateway" to health care in America, with the goal to ensure that all consumers have access to primary care services and understand the components of the "medical neighborhood." Our patients should think of Family Medicine first, the guardians of well-care. The place where our patients first think of going for the flu, a sinus infection, minor injuries. Those with chronic conditions come to us for monitoring, testing, coaching on how to improve their health, and medication refills. How can you as ACOFP members ensure that your patients continue coming back?

Regular communication is key. Right now, we lead a 21st century doctor-patient relationship, where the patient becomes the captain of his or her ship, and we are navigators. Your clinic becomes a team; therefore, the onus is on you, the doctor and your staff to contact the patient, not the other way around. Reach out to patients to keep the relationships warm - ensure they are coming in for well-care visits, monitor pre-chronic or chronic conditions, check on a new symptom(s), refill prescriptions, or keep them up-to-date on vaccines. The quality of the visit is vital to whether the patient feels like your office, your staff, and you are part of a patient-centric environment that cares. Having them feel that you are rooting for them and you're interested in them can make a tremendous amount of difference. Patients do not put their trust in machines or devices, they put their trust in you!

Embracing the patient-centered approach is an important part of the transition to value-based medicine. Key is the frequency of contact. Kaiser reported in August that they have the highest patient retention rate, 94 percent, of any hospital in the country and they know their patients better than anyone. Kaiser credits this successful approach to the frequency of communication with its patients and "knowing" its patients better than anyone else.<sup>1</sup>

Using technology can have a positive impact on the physicianpatient relationship. Telemedicine is an important element of Medicare's Chronic Care Management (CCM)<sup>2</sup> program. Medicare patients who have two or more chronic conditions and who sign a letter of consent can be treated under CCM. Medicare pays \$42 per month per qualified CCM patient, separate from other Medicare reimbursement, to coordinate the care of this high-risk group.

This patient segment drives up costs with hospital stays and emergency room visits. The additional touch point to monitor their condition or check on their medication regimen is well worth the investment. You, as the physician leader of the team, can use tele-

medicine for follow-up care, medication management, or care for minor urgent conditions. There are some restrictions on the type of telemedicine software you can use. It needs to be HIPPA compliant and needs to record date and time of the two-way interaction, (in case of a CMS audit).

Patient portals are another way to increase communication of important and timely medical information to the patient. Test results are often posted in hours or one day, instead of the patient waiting several days for a call from the office. Patients appreciate being able to see their last visit, and all their test results over time on one screen. Once the patient's account is set up, it is as easy as logging in to any e-mail account.

Patients appreciate having the additional convenience. It builds loyalty. Your EMR vendor may offer the software for a patient portal. An important tip is to have the e-mail address of the patient portal printed on the appointment reminder cards as patients may not remember it if you simply tell them. Having a patient portal also gains you credit in the CMS Quality Payment Program (QPP) towards Practice Improvement Activities for 2017.<sup>3</sup>

For patients who urgently need to be seen, keep several 15-minute appointments open each day. Patients will appreciate this, and it will keep them coming to your office instead of going to the Urgent Care down the street, or the Emergency Department. This is another way to show you are a patient-centered family practice.

Patients come to your office to see you, to talk to you about their problems, they want to be heard and acknowledged. Imagine if you were the patient and the doctor you have been seeing for a decade now comes into the room, sits down, and puts a computer between the two of you, not even looking at you, the patient. This is unfortunately the reality for many physicians and patients today. Think about how this makes the patient feel – left out, not important, distanced. It is not a favorable scenario if you are going to keep your patients coming back.

Entering the information into the EMR takes hours a day, while trying to see the same number of patients per day. But, what is more important than the relationship with that patient? I have heard some creative solutions. Have your nurse or physician extender see the patient first and record the symptoms and conduct the medication reconciliation while entering the data in the EMR. When you come in to see the patient, you can talk to the patient face-to-face, sitting or standing, and then conduct the physical

exam. Then you will have time to review the notes in the EMR, and enter the diagnosis and treatment. The patient feels cared for and connected, and you have entered most of the information into the EMR. Everyone wins.

Flex your hours. Most of today's workforce are on the clock from 8 a.m. to 5 p.m. Most Family Medicine offices are open 8 a.m. to 5 p.m. Some are closed every day for lunch from noon to 1 p.m. If you are a full-time working patient or caregiver, that does not leave flexibility to go to the doctor outside of the work day, or on lunch break. Employees today are under pressure to do more, in less time, with less pay. For some, even mentioning the words, "doctor's appointment" to an employer is not well received.

Consider one of these options to be more flexible and patient-centric: consider changing the office lunch hour to 11 a.m. to noon so you can see patients during their lunch hour; stay open one night per week until 7 p.m. starting at 9 a.m. or 10 a.m. that day; and for those who really want to be patient-centric, identify one Saturday per month to work from 8 a.m. to early afternoon (taking Friday afternoon off, in exchange). These small changes will tell your patients that you understand and are willing to meet their needs.

Patients notice these changes and will thank you by being loyal to your practice, and may even send a new patient your way.

Rodney M. Wiseman, DO, FACOFP dist.

Rodray M. Wisonan DO FAWAP, dist.

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#### REVIEW ARTICLE

# Lifestyle Management for People with Diabetes - What You Need to Know From the ADA Standards of Care

Kim Pfotenhauer, DO,<sup>1</sup> Joy A. Dugan, MPH, PA-C,<sup>2</sup> Sarah Bradley,<sup>3</sup> Jay H. Shubrook, DO, FACOFP, FAAFP,<sup>4</sup> & Andrew S. Rhinehart, MD, FACP, FACE, CDE, BC-ADM, CDTC<sup>5</sup>

#### **Keywords:**

**Diabetes Mellitus** 

Lifestyle

Nutrition

Exercise

Family physicians are on the front line of diabetes care. This year, the American Diabetes Association's (ADA) Standards of Medical Care features a more robust section on lifestyle management, which is always a critical component of diabetes management. In addition, the ADA recently published an updated position statement on physical activity/exercise and diabetes.

This article highlights the most important lifestyle recommendations that are useful to busy family physicians that will help achieve good outcomes for patients with diabetes, with particular attention to the new exercise position statement. The complete ADA Standards and position statement can be found at professional.diabetes.org/ofp.

#### INTRODUCTION

Lifestyle management is a fundamental aspect of diabetes care and includes physical activity, nutrition therapy, smoking cessation counseling, psychosocial care, diabetes self-management education (DSME), and diabetes self-management support (DSMS).¹ Patients and physicians should work together to individualize treatment plans that set goals for lifestyle modification and establish a strong framework for follow-up education and support.

#### PHYSICAL ACTIVITY & EXERCISE

ADA recommends people with diabetes participate in four types of exercise: aerobic, resistance, flexibility, and balance exercises.

#### **Aerobic**

Cycling, swimming and walking constitute aerobic exercise as they rely on aerobic metabolism and involve the continuous movement of large muscle groups.<sup>2</sup> Performing regular aerobic exercise is associated with lower cardiovascular and overall mortality among all individuals including those with diabetes.<sup>3</sup> Aerobic exercises improve cardiorespiratory function and insulin sensitivity. Added benefits of aerobic exercise for individuals with diabetes include

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decreased A1C, blood pressure, insulin resistance, and lipid profiles.<sup>4,5</sup> High intensity interval training (HIIT) can be safely performed by individuals with diabetes without deterioration of control in type 1<sup>6,7</sup> and improved control in type 2 diabetes.<sup>8,9</sup>

#### **Resistance Training**

Resistance or strength training includes both body weight and weighted exercises. Adults with diabetes should engage in 2–3 sessions/week of resistance exercise on nonconsecutive days. All individuals benefit from resistance training through increased muscle mass, strength, bone mineral density, insulin sensitivity, and cardiovascular health. Given diabetes is a risk factor for both low muscle mass and decrease strength, resistance training is an important aspect of overall fitness. Resistance exercises decrease exercise-induced hypoglycemia associated with type 1 diabetes. Each resistance training session should consist of at least one set (group of consecutive repetitive exercise motions) of five or more different resistance exercises involving large muscle groups. Resistance training can improve A1C by as much as 0.57% and may have additional benefits.

#### **Flexibility Exercises**

Flexibility exercises take joints into full ranges of motion. <sup>16</sup> Flexibility training is recommended 2–3 times/week for older adults with diabetes. Since glycation end products are accelerated by hyperglycemia, <sup>17</sup> flexibility and balance exercises are especially important for individuals with diabetes. While stretching does not affect glycemic control, it will increase range of motion around joints. <sup>18</sup>

<sup>&</sup>lt;sup>1</sup>Touro University College of Osteopathic Medicine

<sup>&</sup>lt;sup>2</sup>Touro University College of Osteopathic Medicine

<sup>&</sup>lt;sup>3</sup>American Diabetes Association

<sup>&</sup>lt;sup>4</sup>Touro University College of Osteopathic Medicine

<sup>&</sup>lt;sup>5</sup>Glytec

#### **Balance Exercises**

Balance exercises are recommended 2–3 times/week for older adults with diabetes. Even with peripheral neuropathy, balance exercises are associated with a decreased fall risk.<sup>18</sup> Yoga and tai chi may be included based on individual preferences to increase flexibility, muscular strength, and balance; it may also promote glycemic control.<sup>19,20</sup>

Further, all individuals, especially those with type 2 diabetes, should reduce sedentary behavior. This includes prolonged sitting and screen time. Light activity every 30 minutes can have blood glucose benefits in type 2 diabetes. In type 2 diabetes, postprandial walking for 15 minutes improves glycemic control. Highly sedentary individuals are at increased risk of mortality and morbidity. Fig. 25-29

#### PHYSICAL ACTIVITY & TYPE 1 DIABETES

Youth with type 1 diabetes experience many health benefits from physical activity participation and doing both aerobic and resistance exercise may be beneficial.<sup>30</sup> In adults with type 1 diabetes, regular physical activity has been associated with decreased mortality.<sup>31</sup> Individuals using continuous subcutaneous insulin infusion (CSII) or multiple daily injections (MDI) as a basal bolus regimen can exercise with few restrictions.

In general, aerobic exercise decreases blood glucose levels if performed during postprandial periods  $^{32}$  and prolonged activity done then may cause exaggerated decreases  $^{33\cdot35}$  but this can be highly variable.  $^{36}$  Mixed activities, such as interval training or team/individual field sports, are associated with better glucose stability than those that are predominantly aerobic.  $^{37\cdot41}$ 

Variable glycemic responses to physical activity  $^{42}$  make uniform recommendations for management of food intake and insulin dosing difficult. To prevent hypoglycemia during predominantly aerobic exercise lasting  $\geq 30$  minutes additional carbohydrate intake of 10-15 grams and/or reductions in insulin are typically required. For activities performed with relative hyperinsulinemia (after bolus insulin), 30-60 g of carbohydrate per hour of exercise may be needed.  $^{43}$ 

Blood glucose concentrations should always be checked before exercise undertaken by individuals with type 1 diabetes. The target range for blood glucose prior to exercise should ideally be 90-250 mg/dL. As an alternative or a complement to carbohydrate intake, reductions in basal and/or bolus insulin dose should be considered for exercise induced hypoglycemia prevention; lowering insulin levels adequately during activity may reduce or eliminate the need for carbohydrate intake. For example, a 20% reduction in basal insulin for individuals on MDI can be made for doses both before and after exercise. 44 CSII users can reduce 45 or suspend 46 insulin delivery at the start of exercise, but this strategy does not always prevent hypoglycemia. 46,47 Frequent blood glucose checks are required when implementing insulin and carbohydrate adjustments.

Continuous glucose monitoring (CGM) may decrease the fear of exercise-induced hypoglycemia in type 1 diabetes by providing blood glucose trends that allow users to prevent and treat hypoglycemia sooner. 48 CGM coverage is becoming more common and this can be a very useful tool for you and your patients.

#### PHYSICAL ACTIVITY & TYPE 2 DIABETES

Achieving regular aerobic exercise increases muscle insulin sensitivity in type 2 diabetes<sup>49</sup> and prediabetes.<sup>50</sup> Intensive lifestyle interventions with at least 175 minutes per week of exercise can sustain weight loss, cardiorespiratory fitness, glycemic control, and blood pressure and lipids with fewer medications and fewer associated comorbidities associated with diabetes (e.g., diabetic kidney disease, retinopathy).<sup>51,52</sup>

Women with preexisting diabetes, particularly type 2 diabetes, and those at risk for or presenting with gestational diabetes mellitus should be advised to engage in regular moderate physical activity prior to and during their pregnancies as tolerated.

While the research is still inconclusive, a minimum 60 min/day of moderate-to-vigorous physical activity, including strength-related exercise at least 3 days/week is recommended for youth with type 2 diabetes.<sup>53</sup>

Physical activity and lifestyle changes can prevent or delay type 2 diabetes. Structured lifestyle interventions that include physical activity (> 150 min/week) can reduce the risk of developing type 2 diabetes by 40-70% in at-risk individuals with impaired glucose tolerance. 54-57

# MINIMIZING EXERCISE-RELATED ADVERSE EVENTS IN PEOPLE WITH DIABETES

#### Hypoglycemia

Exercise-induced nocturnal hypoglycemia is a major concern. Hypoglycemic events occur typically within 6-15 h post exercise, 9 although risk can extend out to 48 h. 60 The risk of nocturnal hypoglycemia may be minimized through 20% reductions of daily basal insulin dose with reduced prandial bolus insulin and low glycemic index carbohydrate feeding following evening exercise for those on MDI. 61 For CSII users, basal rate reductions of 20% at bedtime for 6 h after afternoon exercise may limit nocturnal hypoglycemia. 62 Inclusion of a bedtime snack, glucose checks overnight, and/or use of CGM with alarms and automatic pump suspension may also be warranted. 63-64

#### Hyperglycemia

Exercise-induced hyperglycemia is more common in type 1 diabetes. This may be purposeful in terms of defensive maneuvers to prevent hypoglycemia. Purposeful insulin omission before exercise can promote a rise in glycemia, as can malfunctioning infusion sets during exercise.<sup>65</sup> Individuals with type 2 diabetes may also experience increases in blood glucose after aerobic or resistance exercise, particularly if they are insulin users and administer too little insulin for meals before activity.<sup>66</sup> Overconsumption of carbohydrates before or during exercise, along with aggressive insulin reduction, can promote hyperglycemia during any exercise.<sup>61</sup>

To correct post exercise hyperglycemia, a conservative (50% of usual) correction can be administered.<sup>67</sup> Exercise should be post-poned or suspended if blood ketone levels are elevated (≥1.5 mmol/L), as blood glucose levels and ketones may rise further with even mild activity.

# HEAT-RELATED ILLNESS DURING PHYSICAL ACTIVITY

Physical activity increases body heat production and core temperature, leading to greater skin blood flow and sweating. With increasing age, poor blood glucose control, and neuropathy, skin blood flow and sweating may be impaired in adults with type 1  $^{68,69}$  and type  $2^{70}$  diabetes, increasing the risk of heat-related illness. Older adults with diabetes or anyone with autonomic neuropathy, cardiovascular complications, or pulmonary disease should avoid exercising outdoors on very hot and/or humid days.

#### **Medication Effects**

Insulin secretagogues can contribute to exercise-induced hypoglycemia. If exercise-induced hypoglycemia has occurred, a decreased dose on exercise days may help reduce further hypoglycemia risk. Metformin, dipeptidyl peptidase 4 inhibitors, glucagon-like peptide 1 receptor agonists, sodium-glucose co-transporter 2 inhibitors, and thiazolidinediones are generally safe and no dose adjustment for exercise is necessary.

#### Managing Physical Activity with Health Complications

Macrovascular and microvascular diabetes-related complications could develop and worsen with inadequate blood glucose control. 71.72 Vascular and neural complications of diabetes often cause physical limitation and varying levels of disability requiring precautions during exercise. However, for most patients with diabetes-related complications, physical activity is beneficial.

#### Cardiovascular Disease

After a stroke or myocardial infarction, exercise should be started in a supervised cardiac rehabilitation program. Starting at a low intensity and progressing as able to activities that are more moderate is recommended. Patients with coronary artery disease should also consider exercising in a supervised cardiac rehabilitation program, at least initially. Exertional angina may be silent in some with diabetes and during exercise heart rate should be kept ≥10 bpm below that heart rate in which exercise-related angina in induced.

Both aerobic and resistance training may lower resting blood pressure and should be encouraged. Blood pressure medications can cause exercise-related hypotension, making monitoring important. Patients should be instructed to stop exercise immediately if symptoms of myocardial infarction (such as chest pain, radiating pain, shortness of breath, and others) or stroke (occurring suddenly and often affecting only one side of the body) occur during physical activity and seek medical attention.

Patients with heart failure should be advised to avoid activities that cause an excessive rise in heart rate and instead consider doing low- or moderate-intensity activities.

#### Peripheral Artery Disease

Those with peripheral artery disease should be taught that lower-extremity resistance training improves functional performance<sup>73</sup> and the preferred aerobic activities include low- or moderate-intensity walking, arm ergometer, and leg ergometer.<sup>74</sup>

#### Retinopathy

If proliferative diabetic retinopathy or severe non-proliferative diabetic retinopathy is present, vigorous-intensity aerobic or resistance exercise may be contraindicated because of the risk of triggering vitreous hemorrhage or retinal detachment. Consultation with an ophthalmologist before engaging in an intense exercise regimen may be appropriate.

#### Peripheral Neuropathy

Decreased pain sensation and a higher pain threshold in the extremities result in an increased risk of skin breakdown, infection, and Charcot joint destruction with some forms of exercise. Therefore, a thorough assessment should be done to ensure that neuropathy does not alter kinesthetic or proprioceptive sensation during physical activity, particularly in those with more severe neuropathy. Studies have shown that moderate-intensity walking may not lead to an increased risk of foot ulcers or reulceration in those with peripheral neuropathy who use proper footwear.<sup>75</sup>

In addition, 150 min/week of moderate exercise was reported to improve outcomes in patients with prediabetic neuropathy. All individuals with peripheral neuropathy should wear proper footwear and examine their feet daily to detect lesions early. Anyone with a foot injury or open sore should be restricted to non-weight-bearing activities.

#### Autonomic Neuropathy

Autonomic neuropathy can increase the risk of exercise-induced injury or adverse events through decreased cardiac responsiveness to exercise, postural hypotension, impaired thermoregulation, impaired night vision due to impaired papillary reaction, and greater susceptibility to hypoglycemia.<sup>77</sup> Cardiovascular autonomic neuropathy is also an independent risk factor for cardiovascular death and silent myocardial ischemia.<sup>78</sup> Therefore, individuals with diabetic autonomic neuropathy should undergo cardiac investigation before beginning physical activity more intense than that to which they are accustomed.

#### Diabetic Kidney Disease

Physical activity can acutely increase urinary albumin excretion. However, there is no evidence that vigorous-intensity exercise increases the rate of progression of diabetic kidney disease, and there appears to be no need for specific exercise restrictions for people with diabetic kidney disease.<sup>79</sup>

#### Orthopedic Limitations

Individuals with diabetes are more prone to structural changes to joints that can limit movement.<sup>80</sup> Regular flexibility training will help maintain greater joint range of motion.<sup>81</sup> Strategies include stretching within warm-ups or after an activity to increase joint range of motion, strengthening muscles around affected joints with resistance training, and avoiding activities that increase plantar pressures with Charcot foot changes.

Arthritis is common in lower-extremity joints, particularly in older adults who are overweight or obese. Participation in regular physical activity is possible and should be encouraged, as moderate activity may improve joint symptoms and alleviate pain. Most low- and moderate-intensity activities are acceptable, but more

non-weight-bearing or low-impact exercise may be undertaken to reduce stress on joints. Patients should perform range-of-motion activities and light resistance exercise to increase strength of muscles surrounding affected joints and avoid activities with high risk of joint trauma, such as contact sports.

#### **Technology**

Wearable activity trackers such as pedometers are a helpful behavior-change tool for increasing total steps but not improving A1C.<sup>82</sup> Technology-based strategies using Internet delivered physical activity recommendations have been successful among individuals with type 2 diabetes at increasing physical activity compared to usual care.<sup>83</sup>

#### **NUTRITION THERAPY**

For many, the most challenging part of a treatment plan is determining what to eat and following a food plan. There is not a one-size-fits-all eating plan. All individuals with diabetes should receive individualized medical nutrition therapy (MNT), preferably provided by a knowledgeable registered dietitian. MNT delivered by a registered dietitian is associated with A1C decreases of 0.3-1% for people with type 1 diabetes<sup>84-86</sup> and 0.5-2% for people with type 2 diabetes.<sup>87-90</sup> Emphasis should be on healthy eating patterns containing nutrient-dense, high-quality foods with less focus on specific nutrients. The Mediterranean, <sup>91</sup> Dietary Approaches to Stop Hypertension (DASH), <sup>92,93</sup> and plant-based diets<sup>94</sup> are all examples of healthful eating patterns.

#### Weight Management

There is strong consistent evidence that modest persistent weight loss can delay the progression from prediabetes to type 2 diabetes. 95,96 Also beneficial in type 2 diabetes management, a sustained reduction of 5% of initial body weight has been shown to improve glycemic control and reduce the need for glucose-lowering medications. 97-99 A sustained weight loss of ≥7% is optimal for many obese individuals in order to improve glycemic control, lipids and blood pressure. 100 Intervention programs can facilitate weight loss by the combination of reduction of calorie intake and lifestyle modification.

#### Carbohydrates

Carbohydrate intake from whole grains, vegetables, fruits, legumes, and dairy products, with an emphasis on foods higher in fiber and lower in glycemic load, should be advised over other sources, especially those containing sugars. People with diabetes and those at risk should avoid sugar-sweetened beverages in order to control weight and reduce their risk for CVD and fatty liver and should minimize the consumption of foods with added sugar that have the capacity to displace healthier, more nutrient-dense food choices.

#### **Protein**

Research is inconclusive regarding the ideal amount of dietary protein need to optimize either glycemic control or CVD risk.<sup>101</sup> Some research has utilized meal plans with slightly higher levels of protein (20-30%), thought to contribute to increased satiety, to successfully manage type 2 diabetes.<sup>93</sup> Dietary protein should be maintained at the recommended daily allowance of 0.8 g/kg body

#### GOALS OF NUTRITION THERAPY

- To promote and support healthful eating pattern, emphasizing a variety of nutrient-dense foods in appropriate portion sizes, in order to improve overall health and specifically to:
  - a. Achieve and maintain body weight goals
  - b. Attain individualized glycemic, blood pressure, and lipid goals
  - c. Prevent or delay the complications of diabetes
- To address individual nutrition needs based on personal and cultural preferences, health literacy and numeracy, access to healthful foods, willingness and ability to make behavioral changes, and barrier to change
- To maintain the pleasure of eating by providing nonjudgmental messages about food choices
- To provide an individual with diabetes the practical tools for developing healthy eating patterns rather than focusing on individual macronutrients, micronutrients or single foods

weight/day for those with diabetic kidney disease. In individuals with type 2 diabetes, ingested protein appears to increase insulin response without increasing plasma glucose concentrations. Therefore, carbohydrate sources high in protein should not be used to treat or prevent hypoglycemia.

#### Dietary Fat

Diets emphasizing monounsaturated fats, such as a Mediterranean-style diet, may improve glucose metabolism and lower CVD risk. <sup>102</sup> Eating foods rich in long-chain omega-3 fatty acids, such as fatty fish and nuts and seeds, is recommended to prevent or treat CVD; however, evidence does not support a beneficial role for omega-3 dietary supplements.

#### Sodium

Patients with diabetes should follow the guidelines for the general population and limit sodium consumption to <2,300 mg/day. There is some evidence that lowering sodium to 1,500mg/day may be indicated for those with hypertension and diabetes.<sup>103</sup>

#### **Micronutrients & Supplements**

In people with diabetes, there is no clear evidence of benefit from herbal or other supplementation without underlying deficiencies. <sup>104</sup> However, in a recent report from the Diabetes Prevention Program Outcomes Study, vitamin B12 deficiency may be associated with metformin use. <sup>105</sup> Periodic testing of vitamin B12 should be considered in patients treated with metformin particularly in those with anemia or peripheral neuropathy.

#### Alcohol

Moderate alcohol use (one drink per day for women and two drinks per day for men) does not have major detrimental effects on long-term blood glucose control in people with diabetes. Hypoglycemia, weight gain and hyperglycemia are risks associated with alcohol consumption in those with diabetes. 106

#### **Nonnutritive Sweeteners**

The use of nonnutritive sweeteners has the potential to reduce overall calorie and carbohydrate intake if substituted for caloric sweeteners without compensation by intake of additional calories from other food sources. Nonnutritive sweeteners are generally safe to use within the defined acceptable daily intake levels.<sup>107</sup>

#### SMOKING CESSATION: TOBACCO & E-CIGARETTES

Physicians should advise all patients not to use cigarettes and other tobacco products or e-cigarettes. One study in smokers with newly diagnosed type 2 diabetes found that smoking cessation was associated with amelioration of metabolic parameters and reduced blood pressure and albuminuria at 1 year. 108

Although some patients may gain weight in the period shortly after smoking cessation, recent research has demonstrated that this weight gain does not diminish the substantial CVD benefit realized from smoking cessation. <sup>109</sup>

There are no rigorous studies that have demonstrated that e-cigarettes are a healthier alternative to smoking or that e-cigarettes can facilitate smoking cessation. More extensive research of their short- and long-term effects is needed to determine their safety and their cardiopulmonary effects in comparison with smoking and standard approaches to smoking cessation. 110-112

#### **PSYCHOSOCIAL CARE**

Motivating patients with diabetes to make lifestyle changes can be very challenging. However, it is also important to recognize when patients are overwhelmed from the demands of managing this complex, chronic disease. Physicians should routinely assess psychosocial status, including diabetes distress.

Diabetes distress (DD) is very common and is distinct from other psychological disorders. \$^{113-115}\$ DD refers to significant negative psychological reactions related to emotional burdens and worries specific to constant behavioral demands including physical activity and food intake. The prevalence of DD is reported to be 18–45% with an incidence of 38–48% over 18 months. \$^{116}\$ DSME has been shown to reduce DD. \$^{117}\$ It may be helpful to provide counseling regarding expected diabetes-related versus generalized psychological distress at diagnosis and when disease state or treatment changes. \$^{118}\$

# DIABETES SELF-MANAGEMENT EDUCATION & SUPPORT

In accordance with the national standards for diabetes self-management education and support, all people with diabetes should participate in DSME to facilitate the knowledge, skills, and ability necessary for diabetes self-care and in DSMS to assist with implementing and sustaining skills and behaviors needed for ongoing self-management, both at diagnosis and as needed thereafter. Health care teams can collaborate to improve outcomes. After physicians work with patients to create a care plan, educators, dieticians, and other members of the care team can provide reinforcement, encouragement and knowledge to adhere to the plan.

Four critical time points have been defined when the need for DSME and DSMS should be evaluated by the medical care provider and/or multidisciplinary team, with referrals made as needed:<sup>119</sup>

- 1. At diagnosis
- Annually for assessment of education, nutrition, and emotional needs
- When new complicating factors (health conditions, physical limitations, emotional factors, or basic living needs) arise that influence self-management
- When transitions in care occur DSME focuses on supporting patient empowerment by providing people with diabetes the tools to make informed self-management decisions.<sup>120</sup>

Studies have found that DSME, in an individual or group setting, is associated with improved diabetes knowledge and self-care behaviors, lower A1C, lower self-reported weight, improved quality of life, healthy coping, and reduced health care costs. 121-123 Better outcomes were reported for DSME interventions that were over 10 h in total duration, included follow-up with DSMS, were culturally and age appropriate, were tailored to individual needs and preferences, and addressed psychosocial issues and incorporated behavioral strategies. 124

DSME is associated with an increased use of primary care and preventive services and less frequent use of acute care and inpatient hospital services. Patients who participate in DSME are more likely to follow best practice treatment recommendations, particularly among the Medicare population, and have lower Medicare and insurance claim costs. Phase a finder on its website to access its recognized diabetes education programs: professional. diabetes.org/erp.

#### **SUMMARY**

Lifestyle management is central diabetes care. Lifestyle changes can reduce a person's risk from developing type 2 diabetes, can reduce risk of complications in people with diabetes and can improve glucose control. While physical activity and MNT recommendations will vary for each patient, physician engagement in therapeutic lifestyle change can help all people with diabetes. The American Diabetes Association has resources for the busy physician to help patient engage in healthy lifestyle change. A team-based approach that utilizes mental health professionals, registered dietitians, and diabetes educators also can benefit the patient and reduce the education burden on the physician.

#### DISCLOSURES

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#### **REVIEW ARTICLE**

# A Guide to Treating the Symptoms of Menopause

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#### **Keywords:**

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**Vasomotor Symptoms** 

Menopausal-related symptoms, which include hot flushes, night sweats, vaginal dryness, difficulty sleeping, sexual dysfunction, and depression/anxiety, among others, are experienced by roughly 80% of women and are therefore a common complaint in the primary care setting. Systemic high-dose hormone replacement therapy (HRT) was the mainstay of treatment until a large systematic review determined that the risk of HRT greatly outweighed the benefits to the patient. Since that time, women and their physicians have turned to a variety of therapies; behavioral, alternative, and off-label use of mainstream medications to treat the life-altering symptoms of menopause. Systemic HRT is still the most effective therapy on the market, although it is now prescribed in the lowest effective dose for the shortest possible duration. This article will review available mainstream, alternative and osteopathic treatments for the myriad of symptoms experienced by women during menopause and the menopausal transition.

#### INTRODUCTION

Menopause is defined retrospectively as absence of menses for 12 months and occurs at the median age of 51 years.<sup>1</sup> Perimenopause, or 'menopause transition' occurs within years of menopausal onset when the body experiences decreased levels of progesterone and estradiol with increasing levels of follicle stimulating hormone (FSH). Menses may become irregular during this period with many women experiencing menorrhagia when menses does occur. The final menstrual period occurs after the ovaries cease to produce estradiol or progestin.<sup>2</sup> The primary complaints during this period include vasomotor symptoms (VMS) or "hot flushes" and night sweats, and vaginal symptoms, typically related to vaginal atrophy.3 VMS are experienced by up to 75% of women and can severely impede sleep and quality of life. Genitourinary symptoms are related to vaginal atrophy and dryness in addition to urinary symptoms of urgency, dysuria and recurrent urinary tract infections. These symptoms can interfere with relationships, sexual function, and overall quality of life.

As osteopathic primary care physicians we focus on the health of the whole individual. Menopause is a time of life when many things are changing a woman's body that affect all aspects of her life; both mental and physical. Symptoms related to vaginal atrophy are experienced by up to 60% of midlife women by the fourth year of menopause, while an estimated 7% of these women seek treatment for their symptoms.<sup>4</sup> What if instead of waiting for women

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to bring up these issues, we asked our patients about them during comprehensive exams? By proactively addressing the health problems of menopausal women we can improve their quality of life thereby improving their overall health and sense of well-being.

#### **METHODS:**

Included in this article are major society guidelines compiled from the North American Menopause Society and the American College of Obstetrics and Gynecology. Also included are review articles of randomized controlled clinical trials. The PubMed database and Cochrane Library were searched for randomized controlled trials (RCTs) and meta-analyses of RCTs related to the study topic, with an emphasis on data published over the last 15 years. Alternative treatment studies were included for completeness sake and often included small study populations.

# SYMPTOMS OF MENOPAUSE & THEIR PATHOPHYSIOLOGY

#### **Primary Symptoms**

Vasomotor symptoms typically manifest themselves as "hot flushes", aptly named for the sudden sensation of extreme heat accompanied by redness, warmth and/or sweating of the face, neck, shoulders, or chest. Each instance may last from 1-5 minutes and may be accompanied by diaphoresis, flushing, chills, clamminess, anxiety, and occasionally, by palpitations. VMS may be so severe that they interfere with sleep causing chronic fatigue. Women with hot flushes typically experience episodes for five to seven years, although some women may be afflicted with symptoms for over 15 years.¹ 87% of those affected by VMS, experience them daily, with 33% of women experiencing >10 episodes per day.²

Sexual symptoms, such as dyspareunia and decreased vaginal lubrication, are commonly related to vaginal atrophy. Atrophy of the vaginal mucosa is directly related to the decrease in systemic estrogen, which causes thinning of vaginal tissue, loss of vaginal rugae and elasticity and loss of subcutaneous fat in the labia majora.<sup>2</sup> Up to 40% of North American women will experience symptoms of vaginal dryness, discharge, itching and or dyspareunia during menopause.5 The Women's Health Initiative (WHI) showed that women who reported sexual satisfaction in their relationships tended to have lower BMIs, be more physically active, and have fewer hot flushes and night sweats.<sup>6</sup> Dyspareunia is a common problem in post-menopausal women, affecting 8-22% of this population.<sup>7</sup> Dyspareunia often negatively affects a women's interest in sexual intercourse, and a subsequent decline in coitus frequency exacerbates vaginal dryness. Unlike vasomotor symptoms, the genitourinary symptoms of menopause typically worsen without treatment.8

Estrogen withdrawal is responsible for atrophy and decreased lubrication of the vaginal tissue, predisposing the now fragile epithelium to trauma during intercourse. The sex steroid precursor hormone dehydroepiandrosterone (DHEA) produced by the adrenal glands also drops throughout a woman's life beginning around age 30. An important step in the maturation of vaginal cells responsible for vaginal elasticity is intracellular conversion of DHEA into estrogen. DHEA can only be used by tissues in possession of receptors capable of converting the sex steroid precursor into its active form; a capacity that is lost by endometrial cells after menopause. When compared to placebo, intravaginal DHEA (Prasterone) decreased all subjective vaginal symptoms of menopause when compared to placebo without increasing endometrial thickness.

Important to note are pathological causes of vulvovaginal symptoms. Vulvar dystrophies such as lichen sclerosis, lichen planus, and squamous cell hyperplasia should be ruled out in any individual complaining of vaginal pain. A careful pelvic exam should be performed including biopsies of any areas of thickening, lesions or discoloration to rule out any pre-malignant/malignant conditions.<sup>2</sup>

Urinary incontinence is experienced by nearly 50% of women in the menopause transition and increases with age. Weak pelvic floor muscles and poor urethral support lead to stress incontinence, which is not directly related to menopause, although it is the most common cause of incontinence in middle age females.

#### **Secondary Symptoms**

Additional symptoms related to menopause and aging include weight gain, heart disease, insomnia, mood changes, cognitive decline and osteoporosis. Vaginal pH, which is typically acidic to prevent overgrowth of fungi and bacteria and subsequent inflammation and infection tends to increase during the menopause transition resulting in recurrent inflammatory vulvovaginitis.<sup>25</sup>

Weight gain during the menopause transition is typically around 5 lbs. and can be attributed to lifestyle and activity changes, as opposed to hormonal causes. Weight gain is associated with cardiovascular disease and worsening of vasomotor symptoms. First line treatment for weight gain should be adherence to low fat diet high in fruits and vegetables and increased physical activity. Medical treatments include phentermine, diethylpropion, orlistat, lorcaserin and phenterine/topiramate. Bariatric surgery generally results

in better long-term weight loss in morbidly obese patients (BMI >40 kg/m $^2$  or >35 kg/m $^2$  with comorbid conditions) with resolution of comorbid conditions than lifestyle changes or pharmacologic therapy and should be considered in patients that have failed these therapies. $^2$ 

Women in menopause and the menopause transition are often affected by insomnia related to disrupted sleep triggered by hot flushes and night sweats. Poor sleep is also related to decreased sexual activity and sexual satisfaction; in the WHI trial women with self-reported insomnia and sleep duration under 6 hours nightly tended to have fewer sexual encounters and experience decreased sexual satisfaction.<sup>6</sup> Effective options to treat insomnia include hormonal and non-hormonal therapy in addition to cognitive behavioral therapy specially geared toward insomnia treatment.<sup>10</sup>

Depression is a common complaint experienced by women both post menopause and during the menopause transition. Women with depression self report decreased quality of life independent of the number of hot flushes experienced. It is therefore important to screen women in this stage of life for psychological comorbidities, as treating depression may improve overall sense of wellbeing.

Many women feel a subjective decline in cognitive function throughout the menopausal transition and into menopause, which can be related to a variety of factors prevalent during this period including insomnia, depression, and medication use.<sup>2</sup> The Study of Women's Health Across the Nation (SWAN) revealed that women actually experience a linear decline in cognitive function throughout the menopausal transition and into menopause. Over the ten year study period, processing speed was found to be the harbinger of cognitive decline, although not all women experienced cognitive loss in other areas and there was great variability between individuals when it came to decline in cognitive function with some improving over time.<sup>12</sup>

# TREATMENT OPTIONS FOR VASOMOTOR SYMPTOMS

#### Hormone Replacement Therapy (HRT)

Oral HRT was the initial treatment of choice for women experiencing menopause-related symptoms as it is the most effective means of controlling vasomotor symptoms related to menopause. Systemic HRT fell out of favor when the WHI, a double blind placebo controlled trial, was terminated early after data showed an increased risk of MI, CVA, DVT/PE, and breast cancer in women taking combined estrogen/progesterone therapy for vasomotor symptoms.<sup>1</sup> Since that time, treatment has been targeted towards specific menopausal symptoms, using the lowest effective dose of HRT when required, for the shortest possible duration (see Table 1, page 22).

General adverse effects of systemic HRT during treatment include breast tenderness, vaginal bleeding, bloating and headaches. These effects tend to be experienced less frequently in women taking low dose or ultra-low dose therapy, although the effectiveness of treatment of VMS may be diminished.

Other formulations of systemic HRT with FDA approval for treatment of VMS include a combination conjugated estrogen plus the

**TABLE 1:**Available Formulations of Estrogen

	Ultra-Low Dose*	Low Dose	Standard Dose
Conjugated Estrogen	N/A	0.3-0.45 mg/d	0.625 mg/d
Micronized Estradiol	0.25 mg/d	0.5 mg/d	1 mg/d
Transdermal Estradiol	0.014 mg/d	0.025 mg/d	0.0375-0.05

<sup>\*</sup>Mixed evidence as to effectiveness in reducing VMS,<sup>13,14</sup> currently not FDA approved for this use

selective estrogen receptor modulator bazedoxifene (brand name Duavee), and progestin. The combination of estrogen and bazedoxifene, has a black box warning that includes increased risk of dementia in patients over 65 years, endometrial cancer, stroke and DVT, although it was not found to cause endometrial hyperplasia and has lower risk of vaginal bleeding when compared to conjugated estrogen/medroxyprogesterone. Progestin is generally prescribed in combination with Estrogen preparations in women with a uterus to prevent endometrial hyperplasia and endometrial cancer. Studies have shown that progestin is an effective treatment for VMS, however; progesterone is generally not prescribed alone. This is due to the WHI's finding of increased risk of breast cancer in those patients on estrogen/progesterone combination compared to those on estrogen alone; leading to the belief that the progesterone component was the culprit in these situations.

#### **Non-Hormonal Medical Treatments**

The selective serotonin reuptake inhibitor (SSRI), paroxetine, is the only SSRI with FDA approval for the treatment of VMS in menopause. Although less effective than systemic HRT, it effectively reduced the frequency of VMS within four weeks of initiating treatment across study populations. <sup>17</sup> The most common adverse effects experienced during treatment were nausea and dizziness, which generally resolved with time and dose adjustment. About one third of women will experience a relapse in VMS symptoms within three weeks of discontinuation of SSRI therapy. <sup>18</sup> The starting dose of paroxetine is 7.5mg daily and should be considered in women complaining of concomitant depression.

Clonidine is a centrally-active  $\alpha 2$  agonist and anti-hypertensive used off-label at a dose of  $0.1\,\text{mg/day}$  for the treatment of VMS. It is less effective than HRT, SSRIs and gabapentin and only modestly more effective than placebo in decreasing VMS.³ Due to its multitude of adverse effects including hypotension, headache, light-headedness, xerostomia, sedation and constipation and the risk of developing rebound hypertension upon cessation of therapy, clonidine is not commonly prescribed for VMS.³ Clonidine may be considered in hypertensive women complaining of symptoms related to VMS.

Gabapentin used at 900 mg daily (300mg TID) resulted in a 51% decrease in hot flush frequency and improved the overall quality of life of study participants. Side effects included dizziness, unsteadiness and drowsiness, although these symptoms improved over the course of treatment. Gabapentin does not currently hold FDA approval for this indication, although practitioners intending to treat peripheral neuropathy may monitor patients for a concomitant decrease in hot flush frequency.

Stellate ganglion block is performed by locating the anterior tubercle of C6 while the patient is lying in the supine position. A needle is inserted into the pre-vertebral fascial plane, contrast dye is injected, and fluoroscopy is used to confirm proper needle positioning. The area is then injected with 0.5% bupivacaine and the needle is removed. Patients are evaluated for a positive Horner's sign (which includes miosis, ptosis and anhydrosis) to determine the success of the ganglion block. Stellate ganglion block decreases frequency and intensity of moderate to severe hot flushes over a six month period, but not in a statistically significant manner when compared to sham injections.<sup>20</sup> The role of the stellate ganglion in VMS is not well understood.

#### **Behavioral Changes**

Behavioral techniques are generally aimed at self-cooling or avoidance of foods that are thought to trigger VMS. 50-80% of women use non-hormonal therapies for VMS associated with menopause. Self-cooling techniques include wearing layers, using a fan between sheets at night, or application of cold packs during hot flush. Triggers include alcohol, caffeine, spicy food, or hot liquids/foods. Clinical evidence does not support the effectiveness of these measures.<sup>2</sup>

Exercise has not been shown to decrease frequency or severity of VMS, although one study showed improved quality of life in menopausal women who participated in aerobic exercise three times weekly when compared to those who did not.<sup>21</sup> When comparing yoga to aerobic exercise and placebo in a factorial design randomized controlled trial, yoga was found to decrease interference of hot flushes on quality of life, although it did not affect overall frequency of VMS, while exercise was found to increase physical functionality without having any bearing on VMS.<sup>22</sup> Studies have also not proven yoga or aerobic exercise to be of benefit in the treatment of insomnia.<sup>23</sup>

The Women's Health Initiative Dietary Modification trial studied over 17,000 post-menopausal women who were randomized to either a low-fat, high fruits, vegetables and whole grain diet or the study control group. VMS were measured at baseline and at one year from a 34 item survey. Women with mild symptoms at baseline who lost weight were more likely to eliminate VMS at one year. Participants who lost  $\geq$  10% of body weight were 56% more likely to no longer report hot flushes and night sweats after one year, although women who followed the low fat diet were more likely to eliminate VMS irrelevant of weight loss. <sup>24</sup>

#### **Alternative Medicine**

Many women pursue acupuncture as an alternative to systemic medical treatments, due to their myriad of side effects, for the life-disrupting symptoms of menopause. While modest benefit in hot flush frequency and severity has been noted in randomized controlled trials, acupuncture is not more effective than sham needle treatments across study populations.<sup>25</sup> The North American Meno-

pause Society therefore recommends against acupuncture for the treatment of menopause-related symptoms.

Although Black Cohosh is the most commonly prescribed over the counter medication for the vasomotor symptoms of menopause, very little is known about its mechanism of action.<sup>3</sup> A Cochrane review concluded that black cohosh does not reduce either frequency or intensity of VMS and had insufficient evidence to support improvement in vaginal symptoms or overall quality of life in menopausal women.<sup>26</sup> The average dose used in studies was 40mg daily. Although there were initially concerns over hepatotoxicity caused by the supplement,<sup>27</sup> those claims have since been determined to be unfounded.<sup>28</sup>

Other commonly prescribed supplements in menopause include evening primrose and ginseng. While evening primrose is a good source of prostaglandin E and  $\gamma$ -linolenic acid, it has not been found to have any effect on menopausal VMS. Ginseng has been used for centuries in Asia to treat a variety of conditions. While commonly prescribed for treatment of menopausal symptoms, no randomized controlled trials have shown it to be an effective treatment. There is also concern over the potential side effect of postmenopausal bleeding found in case studies due to its estrogen-like effect on the vaginal mucosa.

Pycnogenol is a pine bark extract available on the market in a variety of dosages. When compared to placebo in a trial of 156 perimenopausal participants, 30mg of pycnogenol was found to significantly improve vasomotor symptoms after both four and 12 weeks of treatment. Interestingly, it was additionally found to improve insomnia in study participants over the 12 week study period.<sup>31</sup> It is unknown whether other pine bark extracts are effective in the treatment of menopausal symptoms.

Pollen extract has a high level of the antioxidant superoxide dismutase. It does not contain pollen shells which are the allergenic component of pollen, so it is safe to give in patients with a reported pollen allergy. 64 women were followed for one month before initiation of the trial to determine frequency and intensity of hot flushes and then for three months after starting daily pollen extract supplementation in the form of Femal pills. Femal was found to decrease frequency of hot flushes by 22%. Study participants did not experience any change in vaginal dryness or bleeding or fluctuation in levels of FSH, estrogen, testosterone or sex-hormone binding globulin.<sup>32</sup> The North American Menopause Society concludes that while pollen extract does not have estrogen activity and was effective in reducing VMS and increasing quality of life in one study, more studies are needed to determine safety and efficacy.

# TREATMENT OPTIONS FOR GENITOURINARY SYNDROME OF MENOPAUSE

#### **Hormone Replacement Therapy**

Systemic estrogen treatment is actually considered less effective than topical estrogen for vaginal symptoms of menopause. Topical estrogen need not be combined with progesterone to prevent endometrial hyperplasia or cancer.<sup>14</sup> Topical estrogen (available in cream, ring, and vaginal tablets) leads to plumping of the vaginal mucosa and increased lubrication and sensation during intercourse, which may improve patient sexual satisfaction.<sup>33</sup> Intravag-

inal estrogen can improve urinary urgency and dysuria in women with over-active detrusor muscles leading to urge incontinence. It is indicated for treatment of vaginal symptoms unresponsive to conservative measures.

Intravaginal DHEA, brand name Prosterone or Intrarosa, was shown in two double blind placebo controlled trials to improve the symptoms of vulvovaginal atrophy and improve dyspareunia. Its use was approved by the FDA in November 2016.<sup>34</sup>

#### **Medical Treatment**

Ospemifene, a non-steroidal estrogen receptor agonist/antagonist (ie selective estrogen receptor modulator) with greatest effect on vaginal epithelium, is the first non-hormonal treatment with FDA approval for moderate to severe dyspareunia. After 52 weeks of therapy, Ospemifene 60mg daily was well tolerated by patients and did not show adverse effect on endometrial or breast tissue. Adverse events include increase hot flushes, although in clinical studies, increased VMS were not severe enough to lead to discontinuation of therapy.<sup>25</sup>

Pharmacologic treatment for urge incontinence focuses on anticholinergic therapy (ie solifenacin, oxybutynin, flavoxate, hyoscyamine, tolterodine), which antagonizes acetylcholine at muscarinic receptors to relax bladder smooth muscle and inhibit involuntary detrusor contractions.<sup>35</sup>

#### **Non-Medical Treatment**

Vaginal moisturizers and lubricants are a readily available over the counter solution. Vaginal lubricants are shorter-acting than moisturizers, which can be applied every few days. Lubricants are intended for application before or during intercourse and are either water or silicone based; silicone-based treatments are longer-lasting in smaller amounts, although they may interfere with male erection.<sup>36</sup> Often, they must be applied multiple times during one encounter.

Urinary incontinence related to poor urethral support and weak pelvic floor muscles could be effectively treated with weight loss, physical therapy and pessaries. Behavioral therapies for urge incontinence include caffeine and fluid restriction and bladder retraining. Surgical treatment, most commonly mid-urethral sling, has a success rate of 85% but lacks good long-term data.<sup>2</sup>

# INTEGRATION OF OSTEOPATHIC MANIPULATIVE THERAPY

#### **Anatomy**

The ovaries are innervated by spinal cord levels T10-11, while the uterus derives its innervation from T12-L1 and the sacral splanchnic nerve. Pain from the cervix and upper vagina is sensed by sacral levels 2-4. The pudendal nerve provides somatic efferent innervation to the skeletal muscles of the perineum and general somatic afferents from the external genitalia (see Table 2, page 24).<sup>37</sup>

#### Osteopathic Manipulative Treatment (OMT)

Very few studies have been carried out to determine the effect of OMT on menopausal symptoms. One unblinded study evaluated 30 women who underwent 'Fox's low force technique', a procedure

#### TABLE 2:

Structure	Levels	Parasympathetic Role	Sympathetic Role
Sweat Glands	Various spinal levels contribute to sympathetic and parasympathetic tone	Sweating on palms of hands	All other sweat glands through cholinergic function
Ovaries	Parasympathetic: CN X Sympathetic: T10-11	Exact effect unknown	Exact effect unknown
Uterine Fundus	Parasympathetic: pelvic splanchnic (S2-4) Sympathetic: T10-L2	Relaxation	Constriction
Cervix	Parasympathetic: pelvic splanchnic (S2-4) Sympathetic	Constriction	Relaxation

invented by the study's author and applied to specific spinal segments that were felt to be out of alignment, and determined it be an effective treatment for VMS in menopause. 38,39 There are, however; no randomized controlled trials evaluating the effect of OMT on menopausal symptoms despite the wide array of techniques available to treat menopausal women. 40,41

#### Conclusion

Menopausal symptoms are a common complaint encountered in the primary care setting. While there are a variety of pharmacologic, behavioral and alternative treatments available today, none are as effective in reducing the vasomotor and vaginal symptoms as hormone replacement therapy (systemic or topical). As is the case with most processes that effect quality of life, the physician must chose from the available treatment modalities to find the best fit for the individual patient. None are as prepared as the osteopathic physician, who can take a full-body approach to the patient and chose the treatment(s) that best fit the patient's symptoms, risk factors, medical comorbidities and lifestyle.

#### **AUTHOR DISCLOSURE**

No relevant financial affiliations.

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#### REVIEW ARTICLE

## Parsonage Turner Syndrome

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#### **Keywords:**

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**Neuralgic Amyotrophy** 

Shoulder Pain

**Brachial Plexus** 

Suprascapular Nerve

The potential causes of shoulder pain are numerous, ranging from trauma, to disease, to hereditary issues. Here we report on a rare, but emerging potential culprit. Parsonage-Turner Syndrome (PTS) is a neurologic disorder affecting peripheral nerves, usually the upper trunk of the brachial plexus. It is generally characterized by the rapid onset of severe shoulder pain and eventual muscle wasting and weakness in the affected area. PTS is of unknown etiology, but postulated causative factors include disease, heredity, medicines, trauma, and surgery. A definitive diagnosis of PTS is difficult as symptoms overlap with other shoulder ailments such as rotator cuff pathologies, impingements, adhesive capsulitis, cervical spondylosis, and suprascapular neuropathy. Ultimately patient history, MRI, and EMG are necessary to confirm PTS. Conservative treatment, including pain management and physical therapy, is key. Most patients progressively recover within a period of 3 years. The present case involves a 22 year-old white male and former collegiate athlete. The patient underwent a surgical procedure in order to cosmetically correct an injury sustained previously in an automobile accident, and presented with severe left shoulder pain two days post-op. Atrophy, and weakness of infraspinatus and supraspinatus muscles were observed three months after surgery, and EMG was positive for suprascapular nerve dysfunction. The patient was treated with oral steroids and hydrocodone, and underwent physical therapy for a year. Most of the strength and bulk in both muscles was regained three years after initial presentation, but he failed to have a full recovery.

#### INTRODUCTION

Because many work and recreation activities involve repetitive movements, shoulder pain is not uncommon, even in young otherwise healthy adults. In many cases, the cause of such pain is readily identified, and appropriate treatment is prescribed. In other cases, the source of shoulder pain may be more difficult to diagnose, particularly in a primary care setting. Parsonage-Turner Syndrome (PTS) is a rare neurologic disorder, but with a classic presentation. In 95% of cases, the syndrome is characterized by a sudden bout of acute, severe shoulder pain, followed by progressive neurologic deficits, notably muscle wasting and weakness in the affected area, and sensory abnormalities. 1,2,3 However, due to its presumed rarity, an initial diagnosis of PTS is not likely. Instead, patient discomfort is often attributed to more common neck, shoulder, and upper limb conditions.<sup>4,5</sup> Treatment for PTS is generally conservative, and resolution is often spontaneous. Therefore, it is important to avoid misdiagnoses, which can result in costly, unnecessary, or counterproductive procedures, and prolonged patient suffering.<sup>5,6,7</sup> Fortunately, understanding the classic signs of PTS can reliably lead to a diagnosis in the vast majority of cases.<sup>3</sup>

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#### CASE PRESENTATION

A healthy, 22 year-old white male presented with atrophy and weakness of the left supraspinatus and infraspinatus muscles. Three months prior, the patient underwent a fat grafting operation under general anesthetic. The procedure involved removal of adipose tissue in the abdominal area and subsequent transplant to the subcutaneous tissue of the left forehead. Two days after the procedure, the patient awoke to excruciating pain in his left shoulder. The pain was described as 10/10, and mainly localized to the posterior aspect of the shoulder, with no radiation. The patient experienced similar pain for the next two days, during which time he was limited to sleeping in a recliner. Pain medication was administered, which lessened pain severity to about 2/10. The pain resolved spontaneously after three days, and pain medication was no longer necessary. However, while lifting weights three months later, the patient noticed weakness in his left arm. Two months after the onset of weakness, he began to notice atrophy of his left supraspinatus and infraspinatus muscles. Physical exam was remarkable for 3/5 muscle strength in abduction and a positive empty can test in the left arm. Vitals were within normal limits and review of systems was unremarkable for fever, chills, night sweats, weight changes, neck pain, headache, nausea, and vomiting. The patient was prescribed prednisolone, and an MRI, nerve conduction study, and physical therapy were ordered.

#### **INVESTIGATIONS**

No labs were performed, and the MRI came back negative showing no signs of inflammation, muscle tears, cysts, or abscesses. A nerve conduction study of the left upper extremity was performed eight months after onset of initial symptoms, and it showed abnormalities in the infraspinatus and supraspinatus muscle.

#### **TREATMENT**

The patient was treated with Prednisolone for four weeks. The medication was discontinued after two weeks due to side effects of the medication. He did not notice any changes after taking the medication. The patient was then referred to physical therapy for six months, after which the patient's muscle mass increased by about 50% and strength improved. Another six months of physical therapy was performed and muscle mass and strength continued to improve to about 80-85% of baseline.

#### **OUTCOME & FOLLOW-UP**

The patient responded well to physical therapy. However, full muscle mass and strength were not restored. Infraspinatus was more atrophic than supraspinatus, and there was more atrophy on the medial scapular border. Muscle strength was not 100% but 5/5. Physical therapy was discontinued after 12 months due to lack of insurance coverage. Activities of daily living are not affected and he is able to perform weight lifting activities again with only slight limitation in certain exercises, specifically those involving abduction.

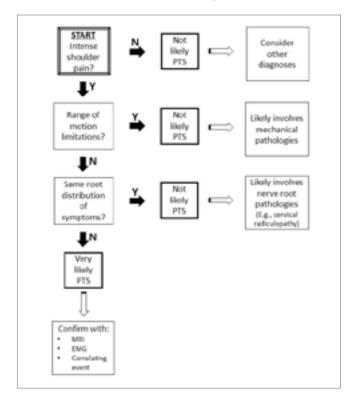
#### **DISCUSSION**

PTS, which is now also known as brachial plexitis, neuralgic amyotrophy, or shoulder girdle neuritis, among others, was likely first identified in the late nineteenth century. 1,2 However, it was not until the 1940s, with two articles appearing in the Lancet, that this syndrome was recognized as such.8,9 In the latter piece, appearing in 1948, Parsonage and Turner described their findings in 136 cases as "shoulder girdle syndrome", later to be known as Parsonage-Turner syndrome. 9 The etiology of PTS is unknown, but a wide range of causes have been postulated, including infection, trauma, surgery, anesthetics, antibiotic therapy, immunization, pregnancy, heavy exercise, and heredity. 1,10,11 The syndrome has been considered rare, historically occurring in 2-3 out of 100,000 people.1 However, findings that are more recent have questioned the classification of PTS as a rare disease, since it may be 30-50 times more common than previously thought.<sup>12</sup> Indeed, such figures suggest that 1 in 33 patients with new onset neck, shoulder, or arm complaints have PTS. Patients aged 3 months to 74 years have been afflicted, with a peak incidence in the third and seventh decades.<sup>5</sup> PTS occurs more frequently in males, but recent findings indicate that neither sex, nor age of onset is helpful in distinguishing PTS from other shoulder ailments.<sup>12,13</sup> PTS is primarily a disorder of the upper trunk of the brachial plexus and its branches, but it has been documented in other peripheral nerves, including those associated with breathing and phonation, and the lumbosacral plexus. $^{2,6,14,15,16,17}$  Finally, in 97% of cases the disease is asymmetric. $^{13}$  Though PTS is a well-recognized clinical syndrome, with a very characteristic presentation, a clear diagnosis may be difficult for a few reasons. Foremost, it is a rare, possibly under-diagnosed syndrome, which may not immediately enter consideration in a primary care setting. Consequently, PTS is likely to be mistaken for other, more common conditions such as rotator cuff pathologies, suprascapular neuropathy, acute calcific tendinitis, adhesive capsulitis, cervical spondylosis, tumors, and cervical radiculopathy. Compounding the problem of PTS's relative obscurity is the fact that no single test exists to confirm the syndrome. However, when equipped with the proper knowledge, the practitioner can reliably provide an initial diagnosis of PTS simply from patient history. Subsequent confirmation can generally be obtained from MRI, and EMG results.

According to van Alfen,<sup>14</sup> a solid preliminary diagnosis of PTS can be made by posing these three questions (*Figure 1*).

#### FIGURE 1:

Severe shoulder pain is the single, best indicator of PTS. Therefore, the presence of such pain is likely to result in a diagnosis of this syndrome. However, progression downward, from the "start" box, should further confirm the diagnosis. Moving rightward from any of the first three boxes on the left reduces the likelihood of a PTS diagnosis.



#### Question 1:

Was there severe pain, unlike any previously experienced by the patient? If yes, PTS is likely. If no, PTS is still possible, but alternative diagnoses should be considered. Our patient described 10/10 pain; 9/10 and above are hallmarks of PTS. The intense pain often occurs upon awakening from sleep, is present in over 95% of cases, and manifests in one or more episodes over a few days to a week. 1.12.14

#### Question 2:

Were there limitations to passive arm abduction and external rotation? If no, PTS is likely. If yes, structural pathologies, such as bursitis, calcifying tendonitis, adhesive capsulitis, rotator cuff tears, impingements, are likely. Our patient had full movement of his shoulder joint despite the pain, which most certainly ruled out the aforementioned conditions.

#### Question 3:

Did neuralgic deficits occur in the same root distribution? If no, PTS is likely. If yes, cervical radiculopathy is more likely. In our patient, two observations supported a diagnosis of PTS. One, the patient did not experience any radiating pain, which would imply same root distribution. Second, subsequent testing determined the patient's only neurological deficit involved the suprascapular nerve.

Before the episode of intense pain, our patient's history was generally unremarkable, in particular regarding activities or conditions typically associated with more common shoulder pathologies. The patient was a former college football player, but denied events that may have led to mechanical trauma of the shoulder in the period leading up to the development of pain. Moreover, the patient was right-handed, and the afflicted shoulder was the left one, a situation that further diminished the likelihood of mechanical injury to the shoulder area.

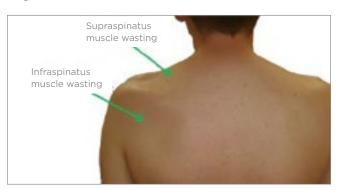
The patient's MRI was unremarkable, showing no signs of inflammation, muscle tears, cysts, or abscesses. This finding was valuable in excluding more common causes of shoulder pain such as rotator cuff pathologies, impingement syndromes, labral tears, or adhesive capsulitis. Though an MRI can exclude many conditions, it also represents a key tool in diagnosing PTS, since it is sensitive to signal abnormalities related to denervated muscle. However, the MRI for this patient was taken several months after the onset of the bout of severe shoulder pain, which may explain, at least in part, the negative result.

EMG abnormalities in PTS are generally evident within 3 weeks of the onset of symptoms.<sup>20</sup> Since PTS is believed to involve disruption of nerve conduction, EMG testing is considered the most important diagnostic test available.<sup>1,5,20</sup> Moreover, EMG results permit the localization of the pathology to specific nerves and the muscles they supply. Our patient demonstrated an abnormal response to nerve stimulation in both infraspinatus and supraspinatus muscles, but normal function in all other nerves and muscles of the shoulder area. As well, our patient experienced atrophy of both supraspinatus, and infraspinatus muscles (*Figure 2*). In the majority of cases, deterioration of the affected muscle begins immediately, or within days of the remission of pain, but again in other cases, muscle wasting may not be noticed for months after the period of

#### FIGURE 2:

In this patient, notice the shallow depressions above, and below the scapular spine. These are indicative of supraspinatus and infraspinatus muscle wasting, similar to what we observed in our patient.

Image source: www.shoulderdoc.co.uk



intense pain. <sup>1,21,22</sup> While these results are very typical of PTS<sup>1,2,19,23</sup> they are also suggestive of suprascapular neuropathy (SSN). <sup>24</sup> However, a diagnosis of SSN was not favored, for two major reasons. First, the patient was afflicted on his non-dominant side. SSN is typically, though not exclusively associated with traction of the nerve. <sup>24,25</sup> Traction may result from repetitive activities associated with manual labor, or sports that feature prominent overhead activities, such as throwing. <sup>24</sup> Traction of the suprascapular nerve may occur due to space-occupying lesions at the suprascapular, or spinoglenoid notches. <sup>24</sup> However, the negative MRI would seemingly rule out these possibilities. Second, SSN is most commonly associated with insidious onset of dull, aching pain. <sup>25</sup> In contrast, PTS is most commonly, though not always, associated with the sudden onset of constant, excruciating pain. <sup>1,2,21,23</sup>

In the absence of a definitive test for PTS, the steps described thus far can reliably diagnose the syndrome. Nonetheless, the diagnosis of PTS can be additionally confirmed by identifying an event in the patient's history that may correlate with the onset of symptoms. As mentioned previously, dozens of causative factors for PTS have been postulated in the literature. 1,2 In the present case, many of these factors were ruled out by patient history. For instance, antecedent disease, vaccination, or administration of medicine were all possibilities, but the patient denied any of these. As explained above, mechanical trauma was also excluded, based on patient history. Stressful exercise has been suggested as a cause for PTS, and the patient, a former college athlete, had a history of participation in strenuous exercise. However, such exertion was habitual for him and thus seems less likely as a precipitating factor. PTS may be caused by an autosomal dominant variant in the SEPT9 gene, but testing for this was not performed.<sup>14</sup> In any case, reports in the literature suggest that the hereditary form of PTS is tenfold less likely than the idiopathic form, and it generally occurs in the second decade of life, with recurrent attacks in almost 75% of patients.<sup>2,14</sup>

In the present case, a singular event stands out. Approximately three days before the onset of pain, the patient underwent fat graft surgery as detailed above. The association of PTS with surgery has been well-documented in the literature, and of all known precipitating factors, appears to be most relevant to this case. It is unclear what specific aspect of surgery may lead to PTS, but iatro-

genic injury to the brachial plexus, stress-induced suppression of the immune system, inflammation, and anesthetics, have all been proposed. 9.26.27.28.29.30.31.32.33.34.35.36.37.38

Treatment for PTS is based on both symptoms and etiology.<sup>20,37</sup> Accordingly, no single or specific treatment modality for PTS has been validated.<sup>39</sup> In general, the treatment for PTS is conservative, and may be divided into two phases, which reflect the typical course of symptoms.<sup>6,5,20</sup>

#### Phase One

During this phase, the period of intense pain, NSAIDs, antiinflammatories, opiates, and steroids may be prescribed with varying degrees of success. In the present case, a combination of NSAIDs and opiates were very effective in lowering the patient's perceived pain from 10/10 to about 2/10. Such success in relieving patients' pain has been reported elsewhere, and it has further been suggested that administration of these drugs may accelerate recovery.<sup>1,4,40,41</sup> However, resistance of PTS-associated pain to such medicines has also been noted.<sup>12</sup> Moreover, dos Santos<sup>20</sup> has suggested that absent a clear etiology for PTS (viral vs. autoimmune, or other), administration of specific classes of drugs should be guarded.

#### Phase Two

During this phase, which commences when pain has subsided, the goal is to reestablish range of motion, and restore muscle strength.<sup>20</sup> At first, muscle strength could be sufficiently compromised to warrant use of a sling.<sup>42</sup> However, with consistent physical therapy over a course of 8 to 12 weeks, and encouragement that the condition will improve, positive results are generally observed.<sup>42</sup> During rehabilitation, TENS (transcutaneous electrical nerve stimulation), and other similar modalities can help alleviate pain.<sup>1,20</sup> Acupuncture may also be helpful, but reports of its success are mainly anecdotal.<sup>1,10</sup> Surgery is indicated to remove any pathology (e.g., cyst, lesion), or if sufficient recovery has not occurred after two years.<sup>4,6</sup>

Overall prognosis is good with long-term pain or weakness being a possible complication. Most patients recover by three years after onset of symptoms, however, about 10% of patients experience continued deficits. <sup>26</sup> Patients with the hereditary form of PTS tend to have more severe symptoms and a higher recurrence rate. In the present case, pain subsided within a few days after onset of symptoms but after three years, minor weakness in abduction remained.

#### CONCLUSION

PTS it is a rare condition of unknown etiology and pathophysiology, most commonly affecting the upper trunk, and branches, of the brachial plexus. It has a characteristic presentation of sudden, acute, and intense pain followed by predictable neuromuscular deficits. Several factors or conditions have been postulated to cause PTS, among them, heredity, disease, and multiple forms of medical treatment, including surgery. In the present case, a young, healthy male developed PTS following a surgical procedure unrelated to the brachial plexus, or shoulder region. A positive diagnosis is generally made from patient history, with confirmation by EMG, and

MRI. Treatment is conservative, generally beginning with medicine to alleviate the initial pain, and continuing with physical therapy and related protocols, which restore muscular strength and range of motion. Increased incidences of PTS suggest this has been an historically under-diagnosed condition. As such, it is valuable to educate primary care providers on this syndrome to avoid costly, or destructive misdiagnoses.

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No relevant financial affiliations.

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#### **REVIEW ARTICLE**

# Evaluation & Management of Functional Abdominal Pain in Children

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#### **Keywords:**

**Chronic Abdominal Pain** 

Organic Abdominal Pain

Non-Organic Abdominal Pain

**Pediatrics** 

Non-organic or functional, abdominal pain is pain that exists free of a pathologic state. This is the most common type of chronic abdominal pain in children. It interrupts a child's normal daily activities and causes stress and anxiety amongst families. In the past, it has been poorly treated due to misconceptions and inadequate knowledge of evaluation and application of treatment. A proper history and physical evaluation of the abdominal pain is required for accurate diagnosis. ROME IV criteria is the diagnostic criteria used for children once organic chronic abdominal pain is ruled out. Treatments that are available for use in non-organic chronic abdominal pain in children consist of behavioral therapies, diet changes, and recently approved pharmaceutical medications.

#### INTRODUCTION

Chronic abdominal pain is diagnosed by having at least three episodes of pain that interfere with function over at least three months. This is however, skewed in clinical practice. Chronic abdominal pain is clinically diagnosed when pain exceeds one month. Once this diagnosis is made, chronic abdominal pain can then be separated into organic and nonorganic pain. Organic pain is pathologic pain due to a disease process (Table 1, page 34 and 35). Non-organic pain, otherwise known as functional pain, is without evidence of anatomic, neoplastic, inflammatory, or metabolic abnormalities. Non-organic abdominal pain is the most common cause of chronic abdominal pain among children in the US. Once organic causes are eliminated, the recently updated ROME IV criteria is used to differentiate types of nonorganic abdominal pain (Table 2, page 35). Adequate evaluation and treatment of this pain will prevent excessive testing, relieve stress of family, and help a child to remain active in daily living.

# EVALUATION OF CHRONIC ABDOMINAL PAIN

A comprehensive history and physical with a quick follow up is critical in the evaluation of chronic abdominal pain in children. Since extensive lab and radiology testing is not warranted in most cases of chronic abdominal pain, the history and physical is critical to gain the parent and child's trust. Gained trust prevents families from having continued anxiety over the condition, finding repeated second opinions, having unwarranted tests done on the

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child, and causing the child to have a stigma placed on him or her. A complete history and physical is also unmatched in the process of forming a differential. The beginning of the history can immediately set the course for deciding between organic and non-organic causes of abdominal pain. Symptoms documented during the history have a strong predictive value for differentiating organic and non-organic abdominal pain in children. The most common chief complaints of children diagnosed with organic abdominal pain in one study was fever, pain not located in periumbilical area, nocturnal pain, weight loss, growth disorder, and general abdominal pain.<sup>3</sup> Some types of symptoms have been shown to be more commonly consistent with organic disorders. "Alarm findings" are symptoms which have been shown to suggest pain caused by an organic state. The most common "alarm findings" are gastrointestinal bleeding, involuntary weight loss, deceleration of linear growth, significant vomiting, chronic severe diarrhea, persistent right upper or right lower quadrant pain, family history of inflammatory bowel disease, unexplained fever, and unexplained physical exam findings.

Due to the inherent chronic nature of the pain, the guardian and child may be tired of speaking about pain during the history. This can make the subjective portion of the exam more challenging. For this reason, it is important to express empathy during the history and physical, as well as address concerns as they present. The physician can also ask family what they think is causing the pain. This can help the physician to understand what the concerns are of the family in order to better address them. After the family's concerns are met, the child should be questioned and examined alone. This situation helps the child to speak freely and may help to elucidate biopsychosocial characteristics. These characteristics will not help in the differentiation of organic and non-organic pain, however they will add to the story and potentially elucidate an exacerbating cause of the pain. A biopsychosocial model of pain includes factors like mood and behavior as potential causes of pain. Relationship roles are included in this model and play a role in chronic abdominal

pain in children. Parental attention to a child's abdominal pain has been shown to worsen and cause pain. Pain can also be caused by a psychological response to traumatic events. A common screening tool for psychosocial issues is the HEEADSSS Questionnaire.<sup>4</sup> Once the social history is clear from this extensive survey, it can be compared to the onset of abdominal pain to elucidate correlation and causation.

Regarding the physical exam, there are some important rules to follow for best practice outcomes. Distraction techniques can help to objectify the physical exam. Talking to the patient while palpating the abdomen is one method elicited. It is also important to auscultate the abdomen prior to palpating. It is important to examine the perianal, however rectal exams in children are debated. Some sources state that this is traumatizing to a child, and some state that it is required for a proper stool sample, checking for fistulas, and checking for constipation. It is however, suggested to check for occult blood on perianal examination. Patients with "alarm findings" need to be further evaluated by specific testing. Additional evaluation and testing is not warranted in patients who do not have "alarm findings." Other physical exam findings that should be documented are general appearance, growth parameters, vital signs, sexual maturity rating, and a full abdominal exam. Guarding has more often shown to be absent with deeper sources of pain. Carnett sign can help differentiate abdominal wall pain from visceral pain. Abdominal wall pain will be more consistent with the continued presence of pain upon contraction of the abdominal wall muscles.

The history and physical in children does have limitations. For instance, children may not be aware of anatomical terms, and have yet to understand forms of pain. Children often have difficulty describing the feeling and location of pain. However, the description and location of pain may not play a significant role in diagnosis. Location of abdominal pain in children with chronic abdominal pain was found to be not significantly different from children without chronic abdominal pain who occasionally have abdominal pain.5 Aside from location of pain and its description, children can have difficulty describing the timeline of the chief complaint. Diaries and journaling are recommended for children with chronic pain with the help of adult supervision. Important factors to journal are time and duration of the pain, possible triggers for the pain, remedies that were tried for the pain, and whether or not the pain prevented activities. Prospective pain and stooling diaries, and parent report of symptoms were predictors of pain maintenance, while child report of symptoms were not.6 Children also tend to report less episodes of pain compared to their pain diaries. 6 Regarding associated symptoms, they have not been found to be helpful when differentiating functional and organic pain. Increased stress and lifestyle events have also shown to not play a role in differentiating organic or functional abdominal pain. Table 2 (page 35) summarizes symptoms and physical exam findings that best differentiate functional from organic pain in children.

#### DIAGNOSING CHRONIC ABDOMINAL PAIN

Table 1 (page 34 and 35) shows the diagnostic technique for the most common presentations of chronic organic abdominal pain. A diagnosis of functional abdominal pain is more elusive, and interestingly more common. The two approaches to take are diagnosis by criteria of lack of alarm findings with a normal physical exam, or

by using the ROME IV criteria. The ROME IV criteria are a diagnostic classification system established by expert consensus and has developed adequate validity over years in the diagnosis of chronic abdominal pain.<sup>7</sup>

The most recent update to ROME IV was in early 2016, which suggested a change in terminology. Recurrent abdominal pain should not be used as a diagnosis. It has recently been suggested that this terminology now be used as a symptom due to the potential for etiology to be organic or non-organic. Terminology is also changing based on current stigmas associated with abdominal pain. Prior to 2016, ROME III criteria referred to nonorganic abdominal pain disorders to be interchangeable with functional gastrointestinal disorders (FGID). In May of 2016, ROME IV criteria suggested moving away from the term FGID, and instead using "Disorders of gut-brain interaction" (DGBI) to break away from current stigmas.

DGBI is based on a diagnosis of exclusion using the 2016 ROME IV criteria. The ROME IV diagnostic criteria for DGBI is defined as episodic or continuous pain occurring at least once a week for at least 2 months with insufficient criteria for other chronic disorders. The definition goes on to state that there must be no evidence of inflammatory anatomic, metabolic, or neoplastic process accounting for the patient's symptoms to be diagnosed with functional abdominal pain. Although not related to the ROME IV criteria, there are some common features associated with DGBI. It is unrelated to meals or activities, self-limiting, vaguely localized or isolated, last less than 1 hour in 55% of cases and less than 3 hours in 90% of cases. BDGBI is defined as having only a symptom of pain, without other associated symptoms or related issues. This is what differentiates it from other types of chronic abdominal pain. Other types of non-organic abdominal pain are presented in Table 2.

#### TREATMENT OF CHRONIC ABDOMINAL PAIN

Best treatment outcomes for non-organic abdominal pain are general behavioral strategies, education, and a therapeutic relationship between the doctor and family. General behavioral strategies consist of parental understanding and continuing the child's normal activities as much as possible. Distraction techniques and muscle relaxation techniques have also been shown to have positive outcomes. Having a child create images of the problem, and then focusing on resolving the problem has been shown to be an effective treatment. Similar distraction techniques have been shown in relationships between children and parents. When parents distract children instead of giving attention to the pain, children reported less pain.<sup>5,9</sup> Abdominal pain in children has also been relieved by fortifying the social support system of children with parent stemmed issues.<sup>10</sup> Children have been shown to create passive coping skills which may consist of complaining of abdominal pain. In these cases, Cognitive Behavioral Therapy for both the parent and the child have been effective strategies for relieving chronic abdominal pain.<sup>11</sup> Hypnotherapy has also shown some benefit.<sup>12,13</sup> Besides behavioral therapy, education can have a great impact on outcomes. Education should first consist of addressing the family's concerns. Living with a child with chronic abdominal pain can create many stressors. Addressing the issues the family is facing first will relieve a family's stress, by solving a problem that has been present chronically. Taking concerns seriously will also build respect for the therapeutic relationship and decrease the stress between the parent and child relationship (continued on page 36).

 $\label{eq:total common chronic organic abdominal pain diagnoses in children. \cite{Common chronic org$ 

Pathology	Common Symptoms	Diagnostic Test
GASTROINTESTINAL TRACT		
Chronic constipation	Hx of stool retention, evidence of constipation on examination	Hx and PE; plain x-ray of abdomen
Lactose intolerance	Symptoms may be associated with lactose ingestion; bloating, gas, cramps, and diarrhea	Trial of lactose-free diet; lactose breath hydrogen test
Parasite infection (especially Giardia)	Bloating, gas, cramps, and diarrhea	Stool evaluation for O&P specific immunoassays for <i>Giardia</i>
Excess fructose or sorbitol ingestion	Nonspecific abdominal pain, bloating, gas, and diarrhea	Large intake of apples, fruit juice, or candy or chewing gum sweetened with sorbitol
Crohn disease	abdominal pain, bloody diarrhea, fever, weight loss	biopsy of bowel wall, imaging
Peptic ulcer	Burning or gnawing epigastric pain; worse on awakening or before meals; relieved with antacids	Esophagogastroduodenoscopy, upper GI contrast x-rays, or MRI enteroscopy
Esophagitis	Epigastric pain with substernal burning	Esophagogastroduodenoscopy
Meckel diverticulum	Periumbilical or lower abdominal pain; may have blood in stool (usually painless)	Meckel scan or enteroclysis
Recurrent intussusception	Paroxysmal severe cramping abdominal pain; blood may be present in stool with episode	Identify intussusception during episode or lead point in intestine between episodes with contrast studies of GI tract
Internal, inguinal, or abdominal wall hernia	Dull abdomen or abdominal wall pain	PE, CT of abdominal wall
Chronic appendicitis or appendiceal mucocele	Recurrent RLQ pain; often incorrectly diagnosed, may be rare cause of abdominal pain	Barium enema, CT
GALLBLADDER & PANCREAS		
Cholelithiasis	RUQ pain, might worsen with meals	Ultrasound of gallbladder
Choledochal cyst	RUQ pain, mass ± elevated bilirubin	Ultrasound or CT of RUQ
Recurrent pancreatitis	Persistent boring pain, might radiate to back, vomiting	Serum amylase and lipase ± serum trypsinogen; ultrasound, CT, or MRI-ERCP of pancreas

Pathology	Common Symptoms	Diagnostic Test
GENITOURINARY TRACT		
Urinary tract infection	Dull suprapubic pain, flank pain	Urinalysis and urine culture; renal scan
Hydronephrosis	Unilateral abdominal or flank pain	Ultrasound of kidneys
Urolithiasis	Progressive, severe pain; flank to inguinal region to testicle	Urinalysis, ultrasound, IVP, CT
Other genitourinary disorders	Suprapubic or lower abdominal pain; genitourinary symptoms	Ultrasound of kidneys and pelvis; gynecologic evaluation
MISCELLANEOUS CAUSES		
Abdominal migraine	See text; nausea, family Hx migraine	Нх
Abdominal epilepsy	Might have seizure prodrome	EEG (can require > 1 study, including sleep-deprived EEG)
Gilbert syndrome	Mild abdominal pain (causal or coincidental?); slightly elevated unconjugated bilirubin	Serum bilirubin
Familial Mediterranean fever	Paroxysmal episodes of fever, severe abdominal pain, and tenderness with other evidence of polyserositis	Hx and PE during an episode, DNA diagnosis
Sickle cell crisis	Anemia	Hematologic evaluation
Lead poisoning	Vague abdominal pain ± constipation	Serum lead level
Henoch-Schönlein purpura	Recurrent, severe crampy abdominal pain, occult blood in stool, characteristic rash, arthritis	Hx, PE, urinalysis
Angioneurotic edema	Swelling of face or airway, crampy pain	Hx, PE, upper GI contrast x-rays, serum C1 esterase inhibitor
Acute intermittent porphyria	Severe pain precipitated by drugs, fasting, or infections	Spot urine for porphyrins

# **TABLE 2:**Most common chronic non-organic abdominal pain diagnoses in children.<sup>2</sup>

Vomiting and aerophagia	Abdominal pain-related functional	Childhood functional abdominal pain
Adolescent rumination syndrome	gastrointestinal disorders	Childhood functional abdominal pain syndrome
Cyclic vomiting syndrome	Functional dyspepsia  Irritable bowel syndrome	Constipation and incontinence
Aerophagia	Abdominal migraine	Functional constipation
	/ Buommar migranic	Nonretentive fecal incontinence

This is also a form of treatment for the child because the parent child relationship has been known to exacerbate or initiate pain. Another form of education is reassurance when warranted. Reassurance is the ability to induce relief in that a condition is not worrisome. Family members may require repeated reassurance with non-functional abdominal pain, simply due to the chronic nature of the disease. Highlighting the basis of diagnosis being symptomology, rather than a positive finding on a diagnostic test is recommended, as well as pointing out the overall repeatedly normal physical exam findings. This can help reassure the family that this is not a serious condition. Besides behavioral treatments and education, pharmaceutical treatments are also occasionally warranted. Placebo high fiber diets have shown benefit as treatment. In some cases, a probiotic<sup>5,14</sup> laxative<sup>15</sup> or peppermint oil may be warranted.15 While mechanism of action of these treatments is unclear, the low cost and ability to improve gastric motility is a benefit to the patient, without causing harm. Severely symptomatic patients will have already tried these. Generally, prescription medications are reserved for severe chronic pain. Some of the current options for this include flunarizine, 16 drotaverine hydrochloride, 17 off-label indication for famotidine, and cyproheptadine as an appetite stimulant.

#### **AUTHOR DISCLOSURE**

No relevant financial affiliations.

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CLINICAL IMAGES

# **Subcutaneous Inguinal Mass**

Carli Cooper, DO, Amanda Ford, DO, Suma Thomas, DO & Thomas Meek, DO

Carolinas Health Care Blue Ridge - Family Medicine Residency

A 21-year-old white female presented to the emergency department with intermittent right lower quadrant pain associated with a palpable nodularity that has waxed and waned in size and tenderness over the past year. The lesion seems to be more prominent around the time of her menses. Her symptoms significantly worsened over the past 3 days with associated nausea and vomiting. She denies any radiation of the pain. Further denies dysuria, constipation, diarrhea, or significant dysmenorrhea otherwise. The patient's past medical history is significant only for an uncomplicated Caesarian section delivery 6 year's prior (G1, P1). Prior to her presentation to the emergency department, she did not use any type of intervention.

Physical exam revealed the patient to be afebrile, with stable vital signs, and in no acute distress. Abdominal evaluation revealed a small palpable subcutaneous mass with associated tenderness, approximately 1cm x 1cm in her right inguinal region with remainder of exam otherwise unremarkable. Laboratory evaluation included a urine pregnancy test, a complete metabolic panel, complete blood count, and screening for Sexually Transmitted Infections – all of which were unremarkable. An ultrasound revealed a heterogeneous 1.3 cm x 0.6cm x 0.8 cm mass deep within subcutaneous tissue near proximal right femur (Figure 1). Radiology recommended that she undergo repeat ultrasound in two weeks to evaluate for interval change and if persistent, consider fine needle aspiration (FNA) versus excision biopsy. She was given naproxen and ondansetron with recommended follow-up with her primary care physician.

Repeat ultrasound found the mass to be persistent with no appreciable interval change. An abdominal pelvic CT scan was performed which revealed a 3.8 x 3.9 cm right adnexal mass (Figure 2) and incidental note of multiple bilateral basilar 4-6 mm pulmonary nodules were noted with recommended 6 month follow up for surveillance (Figure 3). She underwent excision of mass from right lower abdominal wall under general anesthesia without any complications (Figure 4). The pathology report revealed yellow to pink fibro-fatty tissue with cystic spaces, some filled with blood (Figure 5).

#### QUESTION:

#### What is the most likely diagnosis?

- A. Endometrioma
- B. Epidermal Inclusion Cyst
- C. Lipoma
- D. Lymphadenitis
- E. Seroma

#### FIGURE 1:

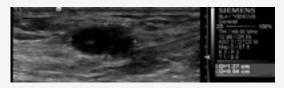


FIGURE 2:

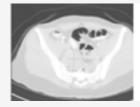


FIGURE 3:

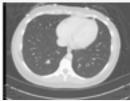


FIGURE 4:



FIGURE 5:



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1877-5773X/\$ - see front matter. © 2017 ACOFP. All rights reserved.

#### **ANSWER**

#### What is the most likely diagnosis?

The correct answer is:

A) Endometrioma

#### DISCUSSION

An Endometrioma is a well circumscribed mass of uterine mucosal tissue outside of the uterus and is a form of endometriosis. Endometriosis is a broad term that describes extra-uterine growth of mucosal uterine tissue. Endometriomas have been described in several organ systems including lungs, gallbladder, central nervous system, small and large bowel, kidney, bladder, extremities, abdominal wall and peritoneum. 1,2,3,4 One of the most common presentations (as in our case) is abdominal wall endometriosis (AWE), which typically presents as a palpable abdominal mass that is more painful around menses. Typically these occur subsequent to surgeries such as Cesarean sections, though there have been cases of abdominal wall endometriomas in women who have had no surgeries.<sup>2,5</sup> The most common presentation is in the rectus abdominis muscle following a cesarean section.<sup>2,6</sup> 96% of cases occur within the pelvic cavity.<sup>7</sup> For this reason, there was a high degree of suspicion for the larger intra-pelvic mass found on CT, though this was later determined to be an ovarian cyst which completely resolved upon follow up imaging. Our diagnostic approach began with ultrasound to help better characterize the lesion (solid or cystic) and to better help guide further work up. Although fine needle aspiration was recommended for tissue analysis, the performing surgeon elected to do excisional biopsy for greater tissue yield.

Risk factors for endometriosis include decreased BMI, menorrhagia and dysmenorrhea. Exercise may be protective against endometriosis.<sup>3</sup> The exact cause of endometriosis is not well understood and several differing theories exist. Common concepts include retrograde menstruation involving viable endometrial cells with subsequent adhesion, implantation, and proliferation within the peritoneum as well as hematogenous and lymphatic metastasis.<sup>6,8</sup> Chronic abdominal pain and infertility are potential outcomes of endometriosis.<sup>2,5</sup> This case illustrates that endometrioma is an important diagnosis to consider with all abdominal wall masses in women of reproductive age. As noted above, one of the most common extraperitoneal sites is the thorax. For this reason the incidental pulmonary nodularities observed on CT will be followed up with repeat imaging in 6 months.

Epidermal Inclusion Cyst, also referred to as epidermoid cysts, epidermal cysts, or, improperly, "sebaceous cysts," are the most common of cutaneous cysts and can occur anywhere on the body. They typically present as skin-colored dermal nodules, often with a clinically visible central punctum ranging from a few millimeters to several centimeters in diameter. Infected, fluctuant cysts tend to be larger, more erythematous, and more painful than sterile inflamed cysts, although an intense inflammatory response to cyst rupture may also present as a fluctuant nodule.

The cyst wall consists of normal stratified squamous epithelium derived from the follicular infundibulum. The cyst may be primary or may arise from the implantation of the follicular epithelium in the dermis as a result of trauma or comedone. Lesions may remain

stable or progressively enlarge. Spontaneous inflammation and rupture can occur, with significant involvement of surrounding tissue. There is no way to predict which lesions will remain quiescent and which will become larger or inflamed. The diagnosis of epidermoid cyst is usually clinical and can be confirmed by histologic examination. The cyst itself is freely movable upon palpation. The cavity is filled with laminated layers of keratinous material. In ruptured cysts, a foreign-body inflammatory granulomatous reaction due to the release of the cyst content into the dermis may result in the formation of a keratin granuloma. 9,10,11,12

Lymphadenitis refers to inflammation or infection of a lymph node, can be found anywhere on the body. Lymphadenitis occurs when the glands become enlarged by inflammation in response to bacteria, viruses, or fungi. The swollen glands are found near the site of an infection, tumor, or inflammation. Lymphadenitis can range from feeling rubbery to fluctuant depending on the underlying components. <sup>13,14,15</sup> Most commonly, the etiology is bacterial and can include staph and group A strep. MRSA should be considered in those with recent hospitalization or occupational exposure. Patients with a poor response to initial antibiotic therapy may require needle aspiration or incision and drainage of the abscess, with subsequent culture for bacterial diagnosis. Viral lymphadenitis can be due to adenovirus, rhinovirus, enterovirus, or EBV. <sup>16,17</sup>

Seroma, or a collection of serous fluid most commonly seen after surgery or after a trauma. Because it contains no or very little blood and no purulent material, it is different from a hematoma or an abscess. They are formed as plasma and other serous fluid collects at the site of tissue removal or trauma. The fluid collects at the site of the scar tissue causing pain, discomfort or an esthetically displeasing lesion. They are usually diagnosed clinically, but can be seen as fluid collections on CT or ultrasound. 19

Lipomas are a collection of adipocytes, or fat cells. These are benign tumors and are the most common soft tissue tumor. Simple lipomas are encapsulated masses made up almost entirely of fat cells.<sup>20</sup> Occasionally, they can contain other structures including blood vessels, muscle fibers and fat necrosis. While most are subcutaneous, occasionally, about 1% of the time, they will be considered deep meaning they will be found deep to the fascia and muscles. These should be treated as more suspicious as they could be liposarcomas. Also, rarely, lipomas can infiltrate surrounding tissues. When this occurs, they are referred to as intramuscular lipomas.<sup>21</sup> On physical exam, they are seen as soft, painless, subcutaneous nodules found most commonly on the trunk and upper extremities. Identified clinically, but appear as a well-circumscribed lesion with homogenous characteristic of fat surrounded by a thin capsule with MRIs being the preferred imaging to evaluate them, especially to evaluate atypical features which could indicate liposarcoma. When there are no abnormal findings, MRI is 100% specific in diagnosing lipomas.<sup>22</sup> In general, no treatment is required for these as they are benign. However, if abnormalities are seen on imaging, surgical resection is necessary. Recurrence is possible and seen in 4-5% of cases.21

Though not specifically covered in our case, other important considerations as part of a thorough differential diagnosis include: neuroma, suture granuloma, hernia, desmoid tumor, and sarcoma.<sup>24,25,26,27</sup>

#### **AUTHOR DISCLOSURE**

No relevant financial affiliations.

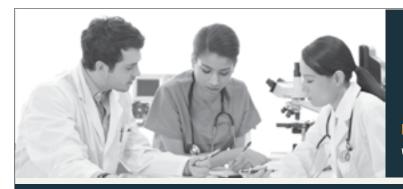
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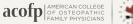
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