Winter 2012 | Volume 33 | Number 6
A Peer-Reviewed Publication

Diabetes Care and Education
a dietetic practice group of the
Academy of Nutrition and Dietetics

ON THE CUTTING EDGE
Diabetes Care and Education

LESS WELL-KNOWN CO-MORBIDITIES OF DIABETES

Message from the Theme Editor: Mamie Lausch, MS, RN, RD, CDE
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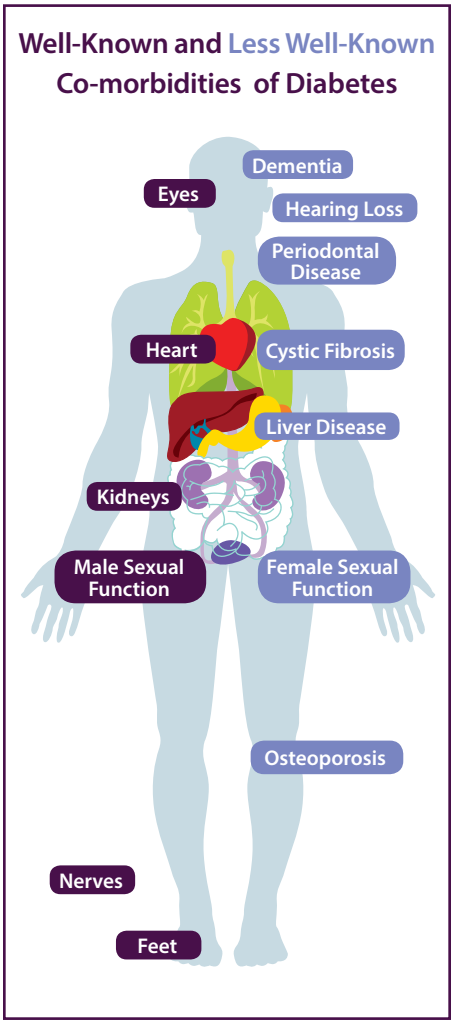
We know that uncontrolled diabetes can lead to numerous complications, as documented by the unquestionable evidence from the Diabetes Control and Complications Trial and the United Kingdom Prospective Diabetes Study. The common complications of diabetes involve the eyes, kidneys, heart, and nerves, but other body parts also can be affected. This edition of *On The Cutting Edge* is devoted to raising awareness and understanding about those "less common" complications and co-morbidities of diabetes: hearing loss, peridontal disease, dementia, hepatic complications, female sexual disorders, bone fracture and cystic fibrosis-related diabetes.



When is the last time you thought about your patient's vestulocochlear nerve? Any time you had to speak really loud and repeat yourself when talking with a patient, the cause could have been damage to this nerve, which leads to sensorineural hearing loss. Hearing loss can be treated, in the case of conductive issues, or adapted to with the use of hearing aids. However, too many patients are wearing hearing aids that

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*News*FLASH and *On the Cutting Edge* are bi-monthly publications of the Diabetes Care and Education (DCE) Dietetic Practice Group of the Academy of Nutrition and Dietetics (the Academy).

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Chicago, IL 60606-6995

Payable to Academy of Nutrition and Dietetics/DCE noting preferred mailing address.

Library of Congress National Serials Data Program
ISSN #1070-5945, issued 7/93.

MISSION
DCE members are the most valued authorities on nutrition and diabetes prevention, education, and management.

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are not turned on or that have dead batteries. Hearing loss is just one more barrier that can interfere with effective education efforts. Margaret McLellan, MS, RD, CDE, LDN, gives tips on working with patients with hearing loss.

Diabetes and dental disease is the perfect embodiment of a chicken-and-egg situation. Poorly controlled diabetes can lead to periodontal disease, and dental disease can lead to poor diabetes control. Lisa F. Mallonee, MPH, RDH, RD, LD, a dietitian and dental hygienist, explains the process of periodontal disease and describes risk factors, symptoms, and clinical application. After reading this article, you probably will make your own dental appointment!

David Randal, PsyD, LP, CDE, discusses the relationship between diabetes and cognitive function. One of his goals is to make you more alert to signs of cognitive decline in your patients (and others). Cognitive decline might explain poor diabetes management that you have observed in some patients. I recall the phone call I received from a patient's wife who was concerned when her husband woke up one day, looked at his glucose meter, and said, "What is this?" Before his cognitive decline, he had been totally independent with his glucose monitoring on a daily basis. Now the wife asked, "What do I do?"

You may have heard the term "fatty-liver," but do you know what it means? Kathleen Briggs Early, PhD, RD, CDE, and Jessica DiBari, BS, OMS3, give good descriptions of nonalcoholic fatty liver disease and nonalcoholic steatohepatitis. They share the good news that intensive lifestyle interventions (moderate calorie restriction, increased physical activity, and weekly goal setting) can dramatically reduce the incidence of liver disease; just one more good reason to follow a healthy lifestyle.

How many of you ask your female patients about problems in the bedroom? This is a very personal conversation, but if a woman cannot talk about this with a health professional, chances are that she is not talking to anyone about it. Female sexual problems can affect intimacy and self-esteem and ultimately may affect diabetes management. Janis Roszler, MSFT, RD, CDE, LD/N, helps us understand the issues and provides helpful advice for your patients.

STRATEGIC PRIORITY AREAS

GOAL 1: Sustain and grow a high level of satisfaction and retention among members.

- Use electronic technology to engage new and existing members.
- Promote and support member professional development.
- Maintain a high value of membership.

GOAL 2: Advance DCE's unique position as the authority in nutrition and diabetes prevention, education and management.

- Promote and maintain new DCE image.
- Develop domestic and global alliance and stakeholder relationships.
- Promote and support evidence-based practice and research.

We are pleased that the winner of the *Journal of the Academy of Nutrition and Dietetics'* 2012 Huddleson Award has written an article on another less well-known co-morbidity of diabetes: bone disease. Corri Hanson, PhD, RD, explains why a dual-energy x-ray absorptiometry scan does not necessarily help identify increased risk of bone fractures in those with type 2 diabetes. Understanding the physiology can help us direct our patients to the best preventive treatments.

Last but not least, Carol Brunzell, RD, LD, CDE will explain the differences between MNT for cystic fibrosis-related diabetes (CFRD) and MNT for type 1 and type 2 diabetes. CFRD is becoming more common as life expectancy has increased for people with CF.

Enjoy this issue of *OTCE* and don't forget to get your continuing education credit!

OTCE Winter 2012 Acknowledgments

THANK YOU!

To the following people for assisting with the development of this issue of *On the Cutting Edge*:

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Hearing Loss and Diabetes: Does Your Patient Hear You Clearly?

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Abstract

Analysis of data from the National Health and Nutrition Examination Survey (NHANES) 1999 - 2004 has shown that diabetes more than doubles the incidence of hearing loss. Most hearing loss in the general population is sensorineural. This is also true of diabetes-related hearing loss, which recent research has associated with the microvascular and neuropathic changes of diabetes. A hearing deficit creates an additional challenge to education for both the patient with the hearing loss and the registered dietitian (RD). Taking steps to ensure that the RD's message is received clearly, such as eliminating background noises and facing the patient directly, are outlined in the article.

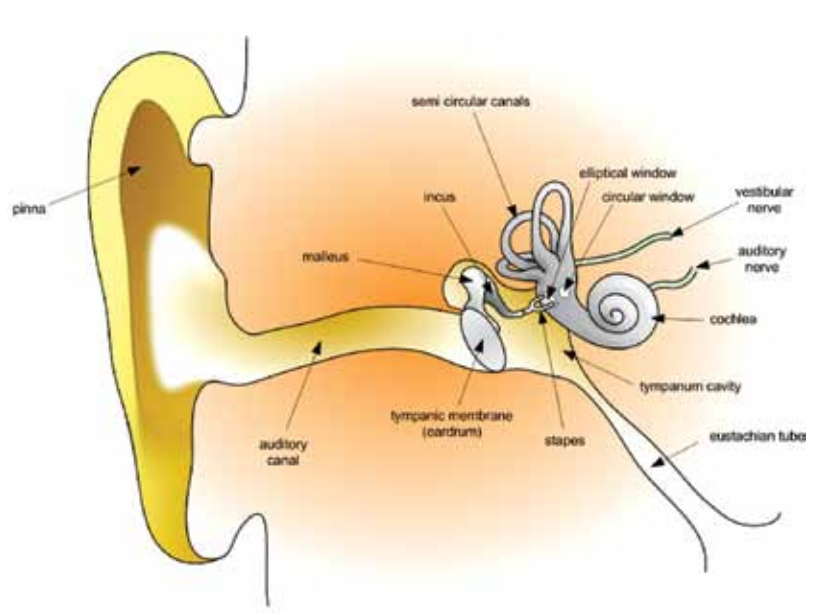
Introduction

Approximately 17% (36 million) of American adults reported difficulty hearing in 2006 (1). Recent research has shown a higher incidence of hearing loss among individuals with diabetes (2–8). Statistics from the National Institute on Deafness and Other Communication Disorders indicate that only 1 in 5 individuals who could benefit from a hearing aid actually wear one (9). Hearing impairment not only has a negative impact on an individual's ability to learn the skills necessary for successful diabetes self-management, but it also negatively affects quality of life and promotes isolation (10–12). The RD needs to be aware of possible undiagnosed or unmentioned hearing loss in clients and determine how to communicate successfully with hearing-impaired individuals (10).

Anatomy of the Ear

The ear is divided into the external ear, middle ear, and inner ear (Figure). The external ear includes the pinna (auricle) and the auditory canal, and its function is considered primarily protective. The middle ear is between the tympanic membrane ("eardrum"), which separates the external ear from the middle ear, and the ellipitical (oval) window of the osseous labyrinth. The middle ear is air-filled and contains the three smallest bones in the human body (malleus, incus, and stapes), which transduce sound waves from the tympanic membrane to the oval window of the fluid-filled inner ear. At this point, sound is amplified 30 to 40 dB. The inner ear, deep inside the temporal bone, includes the cochlea with the organs for hearing, the vestibular apparatus with organs for balance, and the vestibulocochlear (acoustic) nerve. The channels of the cochlea are lined with hair cells that are organized by the sound frequency they transmit and are rich in nerve fibers that synapse with the auditory nerve at the spiral ganglion (10,12).

Figure 1. Anatomy of the ear.



www.sciencekids.co.nz/pictures/humanbody/eardiagram.html

Literature Review: Causes of Hearing Loss

Causes of hearing loss are classified into conductive, sensorineural (comprising more than 90% of cases of hearing loss), and mixed. Conductive hearing loss can be caused by fluid buildup in the middle ear, ear wax in the ear canal, perforated ear drum, and problems with the middle ear bones. Most conductive hearing loss can be medically treated. Sensorineural hearing loss is a type of hearing loss whose root cause lies in the vestibulocochlear nerve (cranial nerve VIII), the inner ear, or central processing centers of the brain. The cause of sensorineural or nerve deafness in a specific patient, on the other hand, is often unknown. Exposure to loud noises, genetics, certain viral infections, aging, exposure to ototoxic medications, and smoking have all been found to increase hearing loss. More recently, cardiovascular disease, hypertension, and diabetes have been implicated as well. With age, most individuals develop presbycusis (age-related sensorineural hearing loss) from damage, drying, and loss of hair cells in the cochlea (10–12). This process appears to be accelerated in those with diabetes (2–4). The other class of hearing loss, mixed hearing loss, is a combination of sensorineural and conductive hearing loss.

Diabetes and Sensorineural Hearing Loss

Diabetes is a significant risk factor for hearing loss, more than doubling the incidence. An analysis by Bainbridge and associates (2) of data from the NHANES 1999–2004 found that the prevalence of mild or greater hearing loss of low or mid- frequency sounds was 9% for adults without diabetes and 21% for those with diabetes. The prevalence of mild or greater hearing loss of high frequencies was 32% for those without diabetes and 54% for those with diabetes. Overall, those with diabetes had 2.3 times the incidence of hearing loss of the general population. In addition, those with prediabetes, as measured by fasting glucose, had a 30% higher rate of hearing loss. When broken down by age, the difference in prevalence of high-frequency hearing loss between those with and without diabetes is greatest during the third and fourth decades of life. Extrapolation of the data to the general population suggests that 70% of individuals with diabetes between the ages of 50 and 69 years have high-frequency hearing impairment and 33% have low or mid-frequency hearing loss.

Estimates of diabetes-associated hearing loss range from an increased incidence of 1.3 to 5 times the incidence of those without diabetes (Table). Agrawal and associates (3,4), evaluating NHANES data from 1999 through 2002, found a twofold incidence of hearing loss across the spectrum of frequencies in individuals with diabetes. A retrospective study by Kakarlapudi and colleagues (5) of both audiometric data and diagnosis of diabetes data from more than 65,000 individuals seen at the Veterans Affairs Maryland Health Care System found that 13.1% of those with diabetes had a hearing loss compared with 10.3% without diabetes. The severity of the hearing loss increased with progression of diabetes, as measured by serum creatinine. In a retrospective review of 990 patient charts, Handzo and coworkers (6) found a statistically significant correlation between a diagnosis of diabetes and hearing impairment in females but not males. In a meta-analysis of 11 observational studies, Horikawa and associates (7) concluded that diabetes substantially contributes to the progression of hearing loss. In a small study of 50 individuals with diabetes who were age- and gender-matched with 50 individuals without diabetes, Ismail and Venkatesan (8) found that 94% of those with diabetes and 18% without diabetes had hearing deficits.

Research regarding the cause of the increased incidence of hearing loss in those with diabetes is ongoing. Diabetes is a systemic disease, known to cause microvascular and nerve damage, and it is postulated that both diabetes-related microvascular and neuropathic changes in the cochlea explain diabetes-associated hearing loss. Studies in diabetic animals found thickening of the capillary basement membranes in the ear. Autopsy studies documented damage to both the nerves and blood vessels of the inner ear in humans with diabetes (10,13). Cardiovascular disease and risk factors for cardiovascular disease, including hypertension and hyperlipidemia, are associated with an increased incidence of hearing loss. The greater incidence of hearing loss in humans with diabetes may be associated with the increased risk of cardiovascular disease (14). Another hypothesis is the presence of a genetic component that predisposes an individual to both diabetes and hearing loss. Several known genetic mutations result in both hearing loss and diabetes (15,16).

Clinical Application: Educating Patients With Hearing Loss

Whatever the cause of hearing loss, a hearing deficit adds to the challenges faced by both the individual with diabetes and the RD. A suggested

Table. Studies on Reported Incidence of Hearing Loss

Author	Population	Frequency Decibel (dB) Range Definition of Hearing Loss	Results
Agrawal et al (3)	3,527 age 20 to 69 years who participated in NHANES 1999-2002 3206 without DM 321 with DM	0.5 to 8 kHz -10 to 120 dB Loss if >25 dB before heard tone	Odds Ratio (for those with DM)* • 2 (low frequency) • 1.2 (mid-frequency) • 3.2 (high frequency)
Bainbridge et al (2)	5,140 age 20 to 69 years who participated in NHANES 1999-2004 4,741 without DM 399 with DM	0.5 to 8 kHz -10 to 120 dB Mild to moderate (>25 dB) and Moderate or greater (>40 dB) before heard tone	• Odds ratio 1.82 (low or mid-frequency) • Odds ratio 2.29 (high frequency) for those with DM
Handzo et al (6)	Retrospective study or 990 patients who had audiograms between 2000 and 2008	Did not define measure of hearing loss in abstract Defined good/poor control as "by American Diabetes Association"	Females younger than 60 years: • Odds ratio of 1.35 for good control • Odds ratio of 1.49 for poor control Females age 60 to 75 years: • Odds ratio of 1.14 with good control • Odds ratio of 1.25 with poor control No differences in males, although hearing worse in males than females
Horikawa et al (7)	Meta-analysis of 11 observational studies 28,459 total 3,388 with DM 5 of 11 studies performed in United States	Hearing impairment defined by cut-off values including 2 kHz.	• Pooled odds ratio of 1 for those without DM • Odds ratio of 1.99 with diabetes • Odds ratio of 2.62 for those <60 years with DM
Ismail et al (8)	50 with DM 50 matched for age/sex without DM Research performed in India	Classified hearing loss as mild to severe, but kHz tested and dB not defined in abstract	• 18% without DM had hearing loss • 94% with DM had hearing loss • Odds ratio of 5.2 with DM Correlated with increased incidence of hearing loss: length of time since diagnosis of DM, age >50 years, and HbA1c >7%
Kakarlapudi et al (5)	53,461 without DM 12,575 with DM Age-matched from Maryland Veterans Affairs electronic database	0.5 to 4 kHz 0 to 100 dB Measured average decibel needed to hear pure tone in each group	• 10.3% without DM had hearing loss • 13.1% with DM had hearing loss • Odds ratio of 1.27 with DM • Creatinine <1.0 hears on average at 51.7 dB • Creatinine >2.5 hears on average at 58 dB (P<0.05)

DM=diabetes mellitus

*Odds ratios mean that a person in one group with a characteristic or condition is X times as likely as a person of the same age and gender without the characteristic/condition. For example, in the Agrawal citation, a person with diabetes is 2 times as likely to have low frequency hearing loss as a person of the same age and gender without diabetes.

approach is to ask, "Can you hear me?" upon first meeting a patient. Individuals with hearing deficits do not always recognize the condition and upon discovery are often hesitant to mention it to the educator. Because those with diabetes-related and age-related hearing loss have the most difficulty hearing high frequencies, they have more difficulty hearing a female educator than a male speaker. Some clues suggesting a hearing deficit are frequent requests that information be repeated, responses that do not match the question asked, short answers, and limited participation in group classes. As the population ages, presbycusis is becoming epidemic, and it occurs at an earlier age and higher prevalence in people with diabetes. Because hearing ability affects the educational process, hearing should be assessed, just as potential visual challenges and manual dexterity deficits are evaluated, to prevent barriers to diabetes self-management education.

The educator can use many techniques to improve communication with individuals with hearing deficits (see "Tips" on page 7). Vision and hearing are both important for understanding speech. People do not hear with their ears but with their brains. Individuals may "speech read," meaning that they watch the lips and the rest of the face for clues as to what is being said. Often they are unaware that they are doing this. They should be seated where they can see the face of the individual who is speaking and wear glasses if needed. With one-on-one counseling, the RD should face the individual with nothing obstructing the view of the educator's face. Seating in a circle works best in a group setting.

Tips for Working With the Hearing Impaired (10,12,17)

1. Eliminate as much background noise as possible (e.g., discontinue background music, reduce the volume of the computer, and close doors to the room).
2. Determine that you have the patient's attention. Await a response to your greeting.
3. Seat the patient close and directly facing you. In a group situation, use a circle so the hearing impaired can see others' faces. Many "speech read" and may not even realize they do so.
4. Assure that your face is easy to see and that your mouth is not covered.
5. Speak clearly but normally. Yelling does not help and distorts the sound of words.
6. If asked to repeat what you have said, rephrase it with different words.
7. Ask the patient to restate what you said or ask a question to verify that what you said was heard correctly.
8. Give the patient written information to reinforce what is said and keep all written materials at a reading level that can be understood by your audience.
9. In a group setting, ensure that only one person speaks at a time.
10. If the patient has a hearing aid and still has difficulty hearing, ask if the hearing aid is working properly. Hearing aids all need periodic cleaning, adjustment of settings, and replacement batteries.
11. Remember that an individual with a hearing deficit may also have a vision deficit. To communicate well, both deficits must be addressed.
12. Consider that not everyone with a hearing deficit is aware of their hearing loss. Further, those aware of a hearing deficit do not always bring it to the attention of the educator.

Limiting background noise includes turning the volume off on the computer, as much as possible, and making sure only one person is speaking at a time. If someone has a hearing deficit, that person should avoid sitting next to or below the heating and cooling vent or projector fan. The educator should enunciate clearly and speak at a normal rate, neither rapidly nor slowly. Speaking very loudly or yelling distorts the sounds of words. Educators should be careful not to cover their mouths and make sure lighting is adequate.

As with any client, when asked to repeat what you said, rephrase it, ask the learner to restate what you said, or ask a question to verify understanding and provide written materials that are at an appropriate level for the audience. Educators should keep in mind that the individual with a hearing deficit may have a vision deficit or limited manual dexterity as well.

Clinicians should consider not only whether a client has a hearing loss, but also whether he or she has

hearing aids and whether the hearing aids work and are used. There are a wide variety of hearing aids. Some are placed in the ear, some are in the ear canal, and some sit behind the ear. The effectiveness and cost varies widely. The more sophisticated hearing aids have various settings that can be selected via a remote control device for group conversation, phone conversation, or watching TV alone.

Most insurance, including Medicare, pays for an audiologic evaluation or audiology examination every few years. Insurance coverage for the hearing aids tends to be limited to very few plans. Cost ($1,000 to $4,000 per ear on average) may be a factor in whether hearing is corrected and to what degree it is corrected. Because hearing aids, which are small and have very small batteries, require frequent cleaning, limited vision and manual dexterity may make their use difficult or require assistance. For more information on hearing aids and organizations for the deaf and hard of hearing, Gallaudet University has an extensive resource list that can be downloaded online (17).

Hearing loss can be very isolating. Loss of hearing affects both quality of communication and often quantity; those with a hearing loss often avoid social occasions that require conversations. In one study, participants with mild hearing loss were three times as likely and those with a moderate to severe hearing loss were almost eight times as likely to report difficulty with communicating compared to those without hearing loss (11). Self-reported difficulties with communication correlate with a reported reduced quality of life, including the frustration involved in attempting to communicate and feeling left out because people do not hear what is going on around them (10,11). By taking steps to ensure their message is heard, the diabetes educator not only communicates effectively but lessens the psychological and social burden on the individual being counseled for diabetes.

Summary

Hearing loss is about twice as prevalent in those with diabetes as those without diabetes. The cause of diabetes-associated hearing loss is being investigated and is hypothesized to be related to both the microvascular changes and neuropathy of other diabetes complications. The diabetes educator should be aware that along with vision loss and decreases in manual dexterity, hearing loss can be a barrier to diabetes care and education. Steps can be taken, such as asking "Can you hear me?" and looking for appropriate responses to questions to ascertain whether the patient has a hearing loss. The diabetes educator also should take steps to accommodate the hearing-impaired patient. Making sure the individual has an unobstructed view of the speaker in individual or group settings, minimizing background noise, and asking the patient to rephrase what has been said can aid in ensuring that the educator's message is received.

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Oral Health and Diabetes Mellitus

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Abstract

Multiple oral signs and symptoms are associated with diabetes. Literature indicates a bidirectional relationship between periodontitis and diabetes. Glycemic control can greatly affect the progression of periodontal disease and may be more pronounced in patients with diabetes than unaffected individuals. More frequent preventive dental care may be recommended for those with diabetes to monitor any oral changes that could put them at risk for periodontal disease. A working knowledge of the oral complications of diabetes is beneficial for the registered dietitian (RD). Collaboration among the person with diabetes, the RD, the medical provider, the dental team, and other members of the health care team can enhance oral health and desired quality-of-life outcomes.

Introduction

Peridontal disease, xerostomia, dental caries, *Candida* infection, burning mouth syndrome, lichen planus, fruity breath, poor wound healing, and asymptomatic parotid gland swelling are among the signs and symptoms associated with diabetes that are commonly noted by oral health professionals when providing a comprehensive screening examination (Table 1) (1). Such symptoms may be present in both those who have diagnosed and undiagnosed diabetes. A comprehensive examination, usually performed annually, may involve but is not limited to a thorough review of the medical history, dental history, periodontal assessment, and radiographs.

Table 1. Oral Manifestations Associated With Diabetes

• Xerostomia (dry mouth)
• Increased caries risk
• Taste alterations
• Burning mouth syndrome
• Glossydnia (pain in the tongue)
• Candida infection
• Gingivitis
• Poor healing
• Periodontal disease
• Periapical abscesses
• Lichen planus
• Fruity breath
• Asymptomatic parotid gland swelling

The literature suggests a two-way interaction between periodontal disease and diabetes. Periodontal infection is associated with poorer glycemic control in people with diabetes and individuals with diabetes who have poor glycemic control are at higher risk of periodontitis (2,3). Periodontal disease has been touted as the sixth complication of diabetes (4,5). Alterations in host response, subgingival microflora, and hereditary factors are among the proposed mechanisms that increase susceptibility. Periodontal disease is two times more likely to occur in a young adult with diabetes compared

to unaffected individuals, and severe periodontal disease is three times more likely to occur in those with poorly controlled diabetes than in those without the disease (3,6). The negative effect of tobacco on periodontal health is well established (7). Individuals with diabetes who smoke have an increased rate of microvascular complications (8). An adult 45 years of age or older with poorly controlled diabetes who smokes is approximately five times more likely to have severe periodontitis than those who do not smoke (6). Accordingly, advising patients with diabetes not to smoke is prudent (8).

From an oral health perspective, individuals with diabetes can be categorized into three groups: 1) the diagnosed and well controlled, 2) the diagnosed and poorly controlled, and 3) the undiagnosed (9). The latter two categories have the greatest risk of oral complications, highlighting the importance of the clinician discussing how periodontal health and diabetes can affect each other. Symptoms can include any of the oral manifestations noted in Table 1 or periodontal disease that is unresponsive to nonsurgical mechanical debridement through scaling and root planing (nonsurgical periodontal therapy or deep cleaning) (9). Such unresponsiveness suggests the potential for an underlying systemic issue that requires further evaluation. Patients should be referred to their medical providers for further evaluation if they experience excessive thirst, urination, or hunger; a recent unexplained weight loss; or vision changes.

Periodontal Disease

Periodontal disease is divided into two categories: gingivitis and periodontitis. The two categories differ by severity. Gingivitis is a milder form of periodontal disease whose key indicator is bleeding gums. It is primarily the result of accumulated biofilm, which is defined as "a complex community of bacteria that forms on any surface that is exposed to the fluids in the mouth" (10). This sticky film of bacteria emits toxins that can damage gum tissue. Supragingival biofilm is located above the gum line and subgingival biofilm is located below the gum. Biofilm accumulates when oral hygiene, such as brushing, flossing, or use of other interdental aids, is inadequate. Dental calculus is formed when the biofilm mineralizes. The only effective method of removing the mineralized deposit is via mechanical debridement performed by a dentist or dental hygienist. In addition to biofilm, hormones, medications, or genetics can contribute to gingivitis. Most adults are affected by gingivitis, and the condition can be reversed with treatment. However, untreated gingivitis progresses to periodontitis. Periodontitis is irreversible and involves destruction of connective tissue and loss of the maxillary and/or mandibular alveolar bone.

Risk Factors, Signs, and Symptoms of Periodontal Disease

Risk factors for periodontal disease include smoking, hormonal changes, diabetes, medications (e.g., oral contraceptives, antidepressants, and certain heart medications), genetics, and certain illnesses such as cancer or acquired immunodeficiency syndrome. Red, swollen, bleeding gums can be a sign of gingivitis or periodontitis. Gingival recession, sensitive teeth, and loose or separated teeth may indicate periodontal disease. Evidence of bone loss may be observed on radiographs. The presence or history of abscesses, halitosis, and missing teeth are additional indicators.

Severe periodontal disease is defined as having 5 mm of attachment loss when a periodontal probe is inserted between the gingiva (gum) and the teeth. Patients with diabetes and periodontitis often experience greater difficulty maintaining optimum blood glucose values (9). Thorough assessment and treatment of periodontal disease is indicated in patients with diabetes (8). Some have suggested that treatment of periodontal disease may help achieve glycemic control, but the evidence presented thus far is inconclusive (8,11–14). A nonsurgical approach that employs scaling and root planing may improve periodontal health, but the long-term impact on metabolic control is uncertain. Education about the importance of frequent dental visits and a well-established home care regimen are key points of discussion for all people with diabetes (controlled *and* uncontrolled).

The Progression of Periodontal Disease

When the bacteria within biofilm come in contact with the gingiva, they secrete toxins that break down tissue between the tooth and the gum. This area becomes ulcerated, triggering the body's immune system. Neutrophils are activated to eradicate the spread of the bacteria and toxins. If successful, the process stops at this point and is referred to as gingivitis. However, if the bacteria continue to damage the epithelial lining of the gum tissue, the immune system responds by sending in larger white blood cells known as macrophages. These white blood cells secrete substances that regulate

inflammation as well as substances that further break down the gum tissue and heighten the inflammatory response. As the ulceration continues deeper into the periodontal pocket, epithelial attachment and connective tissue are lost, resulting in periodontitis. With disease progression, inflammation continues to destroy the periodontal attachment that holds the teeth in place. Mobility ensues and eventual tooth loss can occur.

Impact of Periodontal Disease on Diabetes

Increased inflammation contributes to white blood cell dysfunction. Reduced vascular supply and other alterations in immune system function seen in those who have diabetes predispose them to periodontal disease. The inflammatory intermediaries, produced as periodontal disease is exacerbated, spill over into the bloodstream and add to systemic inflammation. This influx of inflammation is believed to contribute to insulin resistance by impairing the body's ability to effectively lower blood glucose concentrations (15).

The increased inflammation present in progressive periodontal disease has an impact on glycemic control in those who have diabetes. The periodontium, the tissues that surround and support the teeth, are extremely vascular. Deteriorations in the subgingival tissues create a heightened inflammatory state that affects glucose and lipid metabolism (16). The additional proteins released from the infection caused by periodontal disease increase insulin resistance and challenge glycemic control (17).

Clinical Application

Patients with diabetes require frequent medical, nutritional, and dental evaluations. Moreover, detailed patient education and consistent educational reinforcement by all health care providers is vital (Table 2) (12,13). Regular preventive dental care is recommended and should be scheduled at intervals of 3, 4, or 6 months. The frequency is determined by the patient's periodontal health. A daily regimen of brushing twice daily and flossing at least once should be encouraged.

Patients with diabetes should eat something with a balance of carbohydrate, protein, and fat at least 1 hour before dental appointments. One of the primary concerns during dental treatment is hypoglycemia related to disruption of the normal pattern of food intake. Depending on the procedure performed, oral pain may alter food intake for extended hours or days. It is essential to discuss modified food intake after procedures; a soft or liquid diet may be required during recovery. A medical consultation with a physician is indicated for all patients who have insulin-controlled diabetes as well as those who have evidence of poor glucose control. A medical consultation is needed for patients with blood glucose readings of 175 to 399 mg/dL before any invasive dental procedures. An individual with a blood glucose reading greater than 400 mg/dL should be immediately referred to a physician (18).

The dental health professional may advise the person with diabetes to contact his or her medical provider weeks in advance of scheduled procedures to discuss necessary adjustments in medications and postprocedure recommendations. During the dental appointment, the patient should be questioned about the most recent food intake and when it was consumed. Glucose can be measured in the dental office with a glucometer finger stick to determine whether there is a cause for concern before providing treatment.

Oral health professionals need to be informed of medications being used by individuals with diabetes. The action of the medication, typical dosage, adverse effects, and peak action of insulin are important considerations when planning dental treatment. Before treatment, it is important to verify that medications have been taken as usual. Appointment

Table 2. The Registered Dietitian's Role in Oral Health

- Encourage regular dental checkups
- Advise about the importance of proper oral hygiene
- Encourage patients to perform monthly oral examinations to identify any changes in their mouths
- Make referrals to a dentist as needed

When to Make a Dental Referral

- Patient unable to consume food due to mouth pain
- Patient reports sore, swollen, or bleeding gums
- Obvious facial swelling due to gum infection
- Patient reports avoidance of certain foods due to loose teeth
- Patient reports sensitive teeth
- Patient complains of recurrent mouth ulcers
- Patient complains of bad breath
- Obvious caries identified during nutrition-focused physical examination
- Patient reports he or she does not regularly see a dentist

scheduling is often dictated by the diabetic medications prescribed. Peak insulin activity should be considered for those who are taking insulin, with appointments scheduled either before or after the peak activity.

Smoking cessation programs for individuals who smoke and systematic evaluations of the gingiva to assess for early signs of disease are strongly advocated (8). Finally, the dental team should screen patients with diabetes for medical complications such as cardiovascular disease and renal impairment or nutritional risk factors. A comprehensive medical history that reviews all body systems should be used to identify both diagnosed and undiagnosed medical concerns. Referral to a physician or an RD for medical nutrition therapy may be indicated (19). Consultation with the treating physician for patients who have type 1 diabetes is recommended before dental treatment. A medical clearance must be obtained before initiating any treatment because these individuals are taking insulin and are at risk for hyperglycemia and hypoglycemia during a dental procedure.

Antibiotic therapy may be recommended following a dental procedure to decrease the risk of infection due to the slower wound healing capacity of those with poorly controlled diabetes. However, antibiotic therapy is not typically indicated following routine dental treatment. If an acute infection is present, antibiotics may be prescribed. The standard prescription is 100 mg doxycycline once daily for 14 days (1,13,14). *Candida* infection can result if an individual is placed on frequent antibiotic therapy, and individuals with uncontrolled diabetes are especially prone to *Candida* infections. The proposed theory is that increased glucose concentrations in the saliva provide a substrate for fungal growth (1,20).

Summary

Educating patients who have diabetes about their increased risk of periodontal disease is critical to successful oral health outcomes. Patients must be made aware that glycemic control is essential for both proper management of the metabolic aspects of the disease and to minimize oral disease risk. Communication between the diabetes team and the dental team is crucial to ensure consistent messages to the patient. The dental team should be informed of the patient's glycemic control, overall management of disease, duration of disease, and presence of any other diabetic complications before providing care. Conversely, the diabetes team should be made aware of any oral risk factors identified by the dental team and consulted before initiating any complex dental procedures.

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CPE CREDIT
ANSWER KEY

See the CPE credit self-assessment questionnaire on page 30.

1. A
2. D
3. B
4. D
5. A
6. C
7. B
8. E
9. D
10. A

Is There A Relationship Between Dementia and Diabetes?

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Abstract
As people live longer with diabetes, the link between diabetes and cognitive decline in later years has become increasingly clear. Some authors estimate a 1.5 to 2.5-fold increased risk of developing dementia in people with diabetes (1). This article briefly reviews the current issues associated with cognitive decline and diabetes in the elderly. Theories that attempt to explain the association are often contradictory and are worthy of review. Suggested approaches for mitigating the impact of cognitive decline are presented. Interesting and potentially promising findings regarding Alzheimer's dementia (AD) are introduced.

Introduction
Until recently, a connection between all forms of cognitive decline and diabetes was unclear. Evolving research strongly suggests a number of associations, but the nature of these associations should be viewed with caution. Vascular dementia (VD) is a known possible complication of both type 1 and 2 diabetes related to microvascular damage to the brain. This suggests that poorly controlled diabetes can cause the conditions that lead to VD (2). Unfortunately, research to date has not determined if reduced VD is associated with well-controlled diabetes. Alzheimer's dementia (AD) has only recently been identified as a likely complication of poorly controlled diabetes, but the potential for societal impact is worrisome. Projections suggest that both diabetes and AD are likely to continue to surge as the Baby Boom generation ages, and each requires tremendous resources to manage. Part of the reason that AD was not previously identified as a complication of diabetes likely is that most people with diabetes did not live long enough to develop AD. As treatment for diabetes and other conditions have improved, life is extended, and more people are diagnosed with AD and other geriatric conditions.

Dementia is a condition of the brain in which cognitive functions are impaired (3). Memory, reasoning, language, behavior, and thinking can all be affected. Dementia is usually not reversible, although some types can be at least partially reversed. In addition to AD and VD, other types of dementia include Dementia with Lewy bodies (DLB) and mild cognitive impairment (MCI). In addition, several other conditions, including Parkinson's disease and multiple sclerosis, can cause dementia.

VD is attributed to small strokes that damage discreet areas of the brain in a fairly random pattern. In addition to diabetes, tobacco use and hypertension are identified causes of VD. VD may be prevented through cessation of tobacco use and effective management of blood pressure and diabetes. Although the cause of AD is still not

fully understood, it tends to follow an established pattern of stages. DLB is the third most common form of dementia and is differentiated by the presence of Lewy bodies, which are clumps of proteins in neurons. Although each type of dementia may be associated with diabetes, only VD and AD currently have clearly established links. Because VD is relatively well understood, AD is the focus of this article.

Literature Review

Current research is both producing some answers and creating many other questions. As they investigated the premise that microvascular damage might influence the development of dementia in people with diabetes, researchers for the Edinburgh Type 2 Diabetes Study found that people with retinopathy had a higher risk of cognitive decline (4). They noted that the cause of this risk is not yet defined but hypothesized that it is associated with cerebral microvascular disease (4). Other researchers in the same study group found that people with type 2 diabetes who have a history of severe hypoglycemia are more likely to have cognitive losses late in life (5). Yet another aspect of the same study suggested that people with diabetes who have heightened inflammatory markers have a higher incidence of cognitive declines (6).

Data from the Minority Aging Research Study and the Memory and Aging Project showed no differences in cognitive decline based on whether the patient is Caucasian or of African-American origin (7). A study in the *British Journal of Psychiatry* indicated that in addition to increasing the risk of developing cognitive impairment and dementia, diabetes also increases the risk of progression of cognitive decline (8).

Another part of the Edinburgh Type 2 Diabetes Study focused on rheologic (related to flow of matter) factors and determined that increased blood viscosity is associated with cognitive decline, possibly because of effects on cerebral blood flow (9). A group of eminent researchers have asserted that probably multiple factors are involved in the development of dementia in the elderly with diabetes, including severe hypoglycemia, rheologic factors, inflammatory processes, and dysregulation of the hypothalamic-pituitary-adrenal axis (1).

Emerging Research

Based on the metabolic processes involved in glucose metabolism in the brain, some have suggested that insulin treatment may have positive effects on slowing the process of decline with AD. Plastino and associates (10) showed a benefit for insulin treatment while acknowledging that the question is controversial and unresolved. However, even a chance that treatment with insulin might slow the progression of dementia suggests that the premise will continue to be explored. Contrary to other research findings, a study with 63 patients offered limited evidence of a slower rate of cognitive decline in people with diabetes, even when insulin is not involved (11). The evidence of associations with inflammatory processes (6,12) support continued focus on managing inflammation in people with diabetes or even people who do not have diabetes (1,6).

Some investigators have suggested that the nature of cognitive decline in diabetes involves significantly different processes that are unique to diabetes and warrant a new label: type 3 diabetes (13). They also have referred to it as "brain diabetes" because it primarily affects brain functioning and produces a condition that is essentially the same as AD. This theory has contributed to the concept that insulin or insulin sensitization may help to delay or even counteract decline in cognitive function in AD, but that hypothesis has been challenged by research showing increased proclivity for and progression of cognitive decline in people with diabetes (9).

Clinical Application

Each study investigating the issue of cognitive decline in people with diabetes cites the need for much more research. Epidemiologic and long-term studies should offer additional answers. Each new study provides a deeper understanding of the issue, and the longitudinal studies should provide extremely valuable insights as they are updated. The complexity of the issue adds to the difficulty in finding a definitive answer.

Both diabetes and cognitive decline involve multiple known or theoretical causes. Both lead to potentially devastating consequences and substantial financial burdens. Both conditions were rare in the elderly and have now reached epidemic proportions, with projections of even greater numbers (14). Treatment options for both problems continue to be developed as research progresses. Possibly research on cognitive decline in people with diabetes may lead to improved treatment for all people with cognitive decline. In addition, improved treatments for diabetes may result in declines in the overall rates of cognitive losses among the elderly. Cures for either type 1 or type 2 diabetes may result in a reduction in the number of people diagnosed with dementia if some of the theories mentioned previously prove to be correct.

For now, clinicians should focus on the potential for cognitive decline in people with diabetes and be prepared to intervene early. Early identification and treatment may delay cognitive decline and allow the person a longer period of managing self-care independently. If extreme variations in glucose concentrations may worsen cognitive decline, the reduced ability to manage diabetes when changes in cognition are not recognized and treated may create a greater risk of further cognitive decline.

Health professionals have an opportunity to recognize the problem and arrange assistance. If a person with diabetes presents with inexplicable problems following the treatment plan or has evidence of memory lapses, a simple cognitive assessment is warranted and may aid in identifying a patient experiencing cognitive decline. Because screening for VD is more difficult due to the random nature of cognitive losses, developing a list of people who can conduct the screening may be helpful. The Mini-Mental State Exam is often used for screening (15), but it is typically not as effective for mild cognitive decline because it is not as sensitive as some other tools (16). Instruments such as the Neurobehavioral Cognitive Status Examination or Montreal Cognitive Assessment can provide a more complete screening to help identify evidence of early cognitive decline (17, 18). Further information on diagnosis and treatment of dementia can be obtained from the American Geriatrics Society (19).

Summary

A growing body of research has shown that diabetes is associated with cognitive decline in the form of VD and AD. A hypothetical condition termed type 3 diabetes has been proposed and postulated to represent a different form of AD (3). Suggested causes of this cognitive decline in people with diabetes include cerebral microvascular disease, inflammation, severe hypoglycemia, sustained hyperglycemia, and rheologic changes. Evidence suggests that complications such as retinopathy are predictive of cognitive decline. Some researchers provide evidence that the progression of AD is slowed in people with type 2 diabetes and that insulin treatment may help to slow that progression, although these studies have not yet been substantiated. Clinicians armed with this information should be attentive to signs of cognitive decline in their patients and intervene early. Screening can be completed quickly, inexpensively, and noninvasively. Early intervention can positively affect the quality of life for people with diabetes by reducing some of the hardship associated with cognitive decline and possibly slowing the progression of the loss.

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Hepatic Complications of Diabetes: Nonalcoholic Fatty Liver Disease and Nonalcoholic Steatohepatitis

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Abstract

Nonalcoholic fatty liver disease (NAFLD) and nonalcoholic steatohepatitis (NASH) are common hepatic complications among people who have type 2 diabetes (T2DM), and although not as common, problematic among those who have type 1 diabetes (T1DM). When present, NAFLD and NASH increase the risk for cardiovascular disease (CVD). The mechanisms leading to the development of NAFLD are poorly understood but are clearly related to insulin resistance. Studies have shown that intense lifestyle intervention (ILI) can have a positive impact on the prevention of adverse liver-related outcomes in T2DM. This article outlines the current understanding about the development of diabetes-related NAFLD and NASH as well as possible options for prevention and management.

Introduction

A healthy and well-functioning liver is capable of cell regeneration and holds approximately 13% of the body's blood supply at any one point in time. The liver is centrally involved in an assortment of metabolic functions involving macronutrient metabolism and blood glucose regulation, including handling large influxes of dietary glucose or breaking down glycogen when glucose is needed. Performing gluconeogenesis and synthesizing ketones in times of starvation are other key regulatory activities under hepatic control. The liver's primary fuel during the fed state is glucose; it switches to fat utilization via beta-oxidation during fasted states. The liver is commonly accepted as a key organ involved in glucose homeostasis, and chronic hyperglycemia contributes to excessive triglyceride (TG) deposition, leading to insulin resistance and metabolic dysregulation (1). However, the specific mechanisms involved in hepatic fat deposition in diabetes are less well established.

Nonalcoholic Fatty Liver Disease

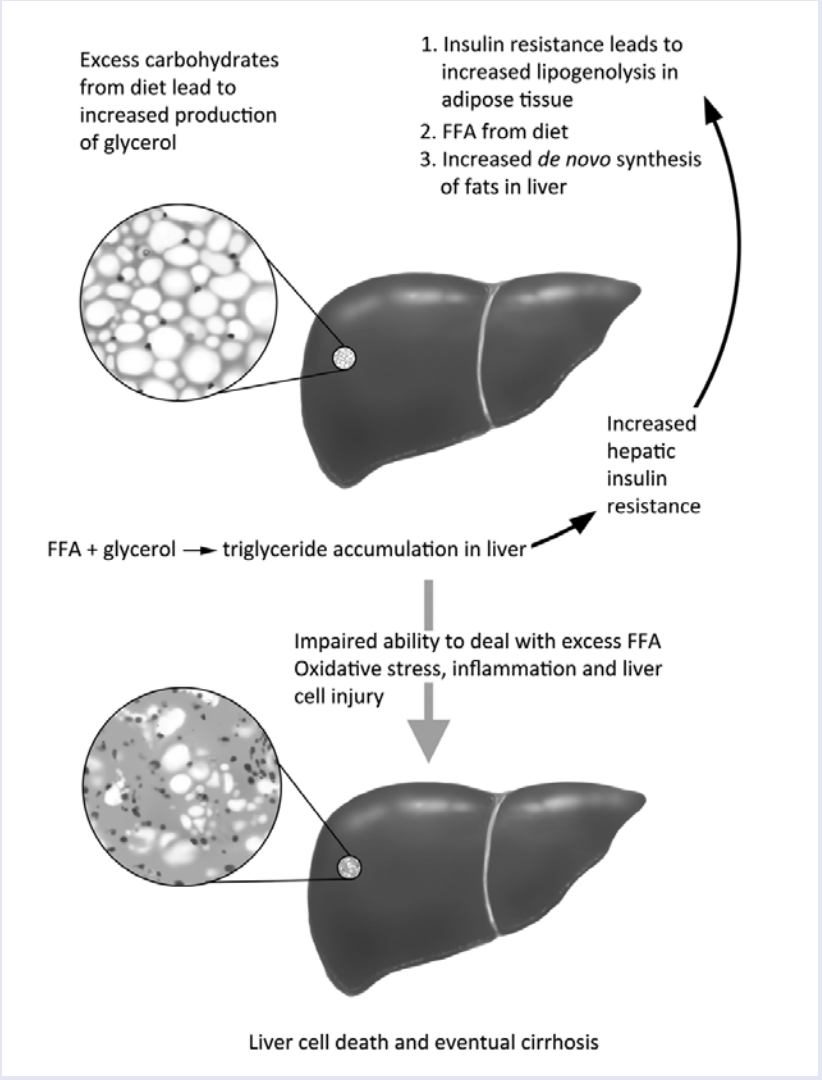
NAFLD, characterized by accumulation of fat in the liver in the absence of alcohol consumption, encompasses a broad range of disorders. On one end of the spectrum is simple fatty liver disease, but NAFLD also includes the more severe form of NASH, which can progress to liver cirrhosis and fibrosis. NAFLD has a prevalence of approximately 30% in the general population, 44% in those who have T1DM, and 50% to 70% in those who have T2DM (2–4). NAFLD is the most

common cause of liver abnormalities in the Western hemisphere, with an increasing prevalence that parallels the rise in obesity and T2DM (5). The presence of NAFLD has also been identified as a serious concern in children who are obese and is strongly linked to the presence of insulin resistance (6).

The pathogenesis of NAFLD and NASH is not yet fully elucidated, but insulin resistance and oxidative stress are believed to be key factors (Figure) (7). According to the recently proposed "three hit hypothesis" of NASH pathogenesis, the first "hit" involves steatosis (hepatic accumulation of fat in the form of TGs), the second "hit" is due to inflammation, and the third "hit" occurs when liver cells die (apoptosis) (8). Steatosis is believed to be strongly correlated with an excess concentration of free fatty acids (FFA) that the liver combines with glycerol to form triglycerides. Sources of FFA include those generated during lipolysis in adipose tissue, dietary sources, and de novo synthesis by the liver.

Under normal physiologic conditions, insulin inhibits lipolysis in adipose tissue, but when adipose tissue is resistant to the effects of insulin, lipolysis is increased, producing a greater influx of FFA to the liver (9). Additionally, insulin resistance alters metabolic function in the liver, leading to more de novo synthesis of FFA. The capability of the liver to dispose of FFA can become saturated, and abnormalities in mitochondrial oxidation of FFA can lead to the generation of reactive oxygen species (10). Recent analysis of enzymes involved in the oxidative stress response among patients with T2DM showed lower levels of key proteins and mitochondrial enzymes (11). In addition, hepatocytes do not repair and replicate as well under oxidative stress, resulting in impaired regeneration and increased cell death, likely contributing to liver fibrosis (8).

Figure 1. Progression of NAFLD.
FFA=free fatty acids. Original artwork by Delbert R. Sanchez.



Diagnosing NAFLD and NASH is challenging, and the only current method of definitive diagnosis of NASH is liver biopsy. Despite the growing prevalence of NAFLD, neither a standardized definition nor screening method currently exists. Most patients with NAFLD are asymptomatic and only come to clinical attention after discovery of elevated liver enzymes on unrelated laboratory testing or hepatic changes by ultrasonography (12). Furthermore, many patients with NAFLD do not have elevated liver enzymes, so clinicians must be aware of the possibility of NAFLD (13).

More intense monitoring of cardiovascular health indicators is required when patients with diabetes are diagnosed with NAFLD to avoid the poor prognosis associated with CVD. In a large study evaluating the prevalence of NAFLD and its association with CVD in T2DM, significantly higher prevalences (n = 1,974) of coronary, cerebrovascular, and peripheral vascular disease were found in patients with NAFLD

compared to those without NAFLD (14). Similar associations between CVD and NAFLD have been reported in studies of T1DM (2). These studies demonstrate that NAFLD is an important risk factor for CVD, independent of the classic diabetes risk factors (e.g., hyperglycemia, elevated TGs, increased waist circumference).

Strategies for Reducing the Risk of NAFLD

Insulin resistance appears to be a critical factor in the pathogenesis of NAFLD and NASH. Unfortunately, no standardized treatment exists for NAFLD or NASH. However, weight loss and dietary changes have been associated with encouraging results for preventing liver disease in diabetes.

The most promising strategy for controlling the progression toward NAFLD in T2DM is intensive lifestyle intervention (ILI), as described by the Look AHEAD Research Group. The benefits of the ILI strategy on the prevention of hepatic steatosis were investigated in the Fatty Liver Ancillary Study, a randomized controlled trial (RCT) involving 96 people within the Look AHEAD trial (15). After 1 year of treatment, people in the ILI group (moderate caloric restriction, increased physical activity, and weekly goal setting meetings) (Table) were compared to people who had received traditional diabetes support and education. Only 3% of participants in the ILI group developed NAFLD compared with 26% of people in the group receiving traditional diabetes support and education (15).

The exact role of pharmacologic treatment in T2DM and NAFLD has not been established. Treatment with metformin appears to provide some benefit, largely mediated through its effect on weight loss, as demonstrated by Krakoff and colleagues (16). As part of the Diabetes Prevention Program, 2,153 people at risk for development of diabetes were randomized to placebo or treatment with metformin and were monitored for elevations of alanine aminotransferase (ALT) as a marker for NAFLD (the dietary intervention group only had baseline ALT measurements). Over the course of 3 years, the placebo group developed significantly higher elevations of ALT, but the difference between the groups could be attributed to the difference in weight loss rather than the effects of metformin alone.

Weight loss surgery has been shown to improve steatosis and to reduce the prevalence of T2DM in morbidly obese patients. In a recent clinical trial involving 284 morbidly obese (body mass index >40) people, 106 of whom had T2DM, 89 people demonstrated complete remission of NAFLD based on liver biopsy approximately 18 months after obesity surgery (Roux-en-Y gastric bypass, adjustable banding, or biliopancreatic diversion with duodenal switch) (17). The prevalence of T2DM was also significantly reduced from 42% to 2%.

Table. Summary of Intensive Lifestyle Intervention Treatment Group in Liver Ancillary Study of the Look AHEAD Trial

Caloric restriction	1,200 to 1,500 kcal/day for individuals weighing <114 kg and 1,500 to 1,800 kcal/day for those weighing >114kg
	<30% calories from fat
	<10% calories from saturated fat
Physical activity	Goal of 175 min/wk of moderate-intensity physical activity
First 6 months	Weekly dietary counseling meetings and three group sessions per month
Months 7 through 12	Monthly individual dietary counseling and group sessions
Specific weight loss goal	At least 10% of initial weight after 12 months

Clinical Application

Partnering with patients to develop customized plans that lead to increased physical activity and weight loss is of utmost importance in the prevention and management of both NAFLD and NASH. Increased physical activity is helpful even in the absence of weight loss, as shown by a recent study involving patients with NASH. Participants had improved liver enzymes and metabolic indices that were independent of weight loss when they increased or maintained their level of physical activity to at least 150 min/wk (primarily walking) (18). In an RCT designed to study the effect of weight loss on NASH, those who achieved 7% to 10% weight loss after 48 weeks of intervention showed improvements in liver histology and liver function test results (19).

Intake of specific nutrients may aid in the treatment of steatosis or contribute to the severity of the disorder. Omega-3 polyunsaturated fatty acid (PUFA) supplementation appears to decrease hepatic fat in patients with NAFLD, although the specific mechanisms and exact dosages have yet to be established. In a meta-analysis of nine studies involving the effects of omega-3 PUFA supplementation on hepatic lipid processing (20), the duration

of intervention and doses varied dramatically between studies (doses ranged from 0.8 to 13.7 g/day and duration of intervention ranged from 8 weeks to 12 months). In one of the included studies, 5 g/day of omega-3 PUFA did not appear to be better than 2 g/day over the same duration (6 months). The authors concluded that benefits are seen with at least 0.83 g/day but emphasized that further RCTs are needed before making specific recommendations regarding supplementation.

Contrary to the benefits seen with omega-3 fats, consumption of a high-fructose diet appears not only to be a risk factor for the development of NAFLD but may also worsen the severity of the condition. In a study of 49 people with biopsy-diagnosed NAFLD, consumption of high-fructose corn syrup was 2 to 3 times higher than in controls without NAFLD matched for sex, age, and body mass index (360 kcal/day vs. 170 kcal/day) (21). Another study demonstrated that daily fructose consumption in patients with NAFLD was associated with a greater degree of liver damage (22). Therefore, encouraging patients to eat fish rich in omega-3 fats and minimize foods high in fructose (e.g., soft drinks) should help improve their liver health.

Conclusions

The liver is a major metabolic center that is significantly affected by diabetes. NAFLD and NASH are clinically important adverse outcomes that are commonly associated with diabetes and are increasing in prevalence. Many patients with NAFLD do not exhibit overt symptoms or have elevated liver enzymes. Liver biopsy is the gold standard for diagnosing NAFLD, but ultrasonography and other noninvasive alternatives have shown remarkable accuracy. Despite the fact that the exact mechanisms leading to diabetes-related NAFLD are largely unknown, insulin resistance and oxidative damage appear to play a key role in its development. Treatment options are limited for existing NAFLD, but lifestyle and dietary interventions have provided promising results in preventing the incidence of liver disease in T2DM. Less success has been found among persons with T1DM. The lack of data, difficulty in diagnosis, and limited patient symptoms underscore the importance of clinical judgment and heightened awareness of the risk factors for diabetes-related NAFLD to prevent progression to liver cirrhosis and fibrosis.

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Diabetes in the Bedroom – Women's Issues

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Abstract

Female sexual dysfunction (FSD) occurs in 10% to 30% of females who do not have diabetes, but it is a problem in approximately 50% of women with diabetes. Vaginal dryness occurs twice as often as in healthy women. Painful intercourse (dyspareunia), urinary tract infections, and depression may also be more prevalent. The incidence of orgasm and libido changes is similar to that of women without diabetes. Except for urinary tract infections, no known relationship exists between a woman's glycolated hemoglobin (HbA1c) value and her risk of FSD. Microvascular damage and neuropathies may indirectly cause FSD, but women without these complications have a similar incidence of FSD. Depression may be predictive of FSD. Treatments include vaginal lubricants, hormones, relationship counseling, and adherence to the Mediterranean diet (in type 2 diabetes).

Introduction

The issue of diabetes-related FSD has long been ignored by the medical community. Even the medical term used for the female symptom of painful intercourse, dyspareunia (Greek for "badly mated"), suggests that this problem is related more to a couple's incompatibility than to any medical issue. Yet some form of FSD, which occurs in almost 50% of women with diabetes, (1) can undermine a women's self-esteem, create stress in her intimate relationship, and weaken the emotional support she receives from her partner (2). This lack of support can make it more difficult for a woman to care for her diabetes and maintain healthy glucose control (3).

The purpose of this article is to introduce registered dietitians (RDs) to a category of diabetes-related complications that is rarely discussed. Many patients share concerns with RDs that they are hesitant to mention to other health care providers. Knowing more about diabetes-related sexual complications and their possible treatments can help RDs provide support and guidance that may make a difference in the emotional and physical health of their female patients.

Literature Review

Until recently, studies on sexuality and chronic disease were unreliable. They lacked standardized definitions of sexual dysfunction, did not employ well-validated scales, and almost totally ignored female sexual dysfunction (4). The relationship between diabetes and sexual complications received even less attention. Newer studies estimate that almost 50% of women with diabetes experience some form of sexual complication (1). The number of affected women may be higher because female sexual complications

are far more challenging to quantify as a consequence of female physical arousal not correlating well with subjective arousal (5).

Symptoms

Female sexual challenges affect 10% to 30% of the nondiabetic female population (6). Historically, women with diabetes have complained of a variety of sexual symptoms, including poor libido, slowed arousal, vaginal dryness, painful intercourse, more frequent urinary tract infections, and orgasm difficulties (7). Only three of these complaints appear with greater frequency in women who have diabetes: urinary tract infections, painful intercourse, and vaginal dryness (4). Women with diabetes have vaginal dryness twice as often as women without diabetes (4). Many investigators have linked women's diabetes-related sexual complications with small blood vessel damage and neuropathies, but current research does not support this assumption. These complications may have an indirect effect on the development of these issues, but women with diabetes who have no complications have a similar incidence of FSD (8).

Emotional and Physical Issues

Both psychological and physical issues play roles in the development of FSD. A woman's mood can significantly affect her ability to relax and enjoy the sexual experience. If she doesn't feel "in the mood" for sex, her body will not respond appropriately during intimacy (8). Depression may be predictive of the presence of FSD (9). A chronic disease such as diabetes can also strain a woman's intimate relationship, creating imbalance and a lack of fulfillment. Such an imbalance could develop if the healthy partner assumes a parental role and begins to supervise the partner's diabetes self-care management. On the other hand, a positive and supportive relationship can enhance a woman's satisfaction and quality of life (10).

Glucose Control, Sexual Dysfunction, and Gender Differences

For men who have diabetes, a clear relationship exists between sexual challenges and age, smoking, poor glycemic control, untreated hypertension, and other somatic factors (11). Except for an increased incidence of vaginal infections, the relationship between FSD and "age, BMI, duration of diabetes, glycaemic control, hormonal therapy, diabetic complications, or menopausal status" either does not exist or has not yet been demonstrated (2). In other words, a normal A1C value and lack of other known diabetes-related complications does not protect a woman from developing sexual complications.

Effect on Diabetes Control

RDs recognize that individuals with diabetes are better able to care for their disease when they have a supportive and loving partner. When diabetes enters the bedroom, however, a couple's relationship may suffer. A woman, for example, might respond to her partner's intimate advances less enthusiastically if she worries that a pregnancy will upset her diabetes control. She might also become tense during intercourse if she does not want to accidentally become pregnant before her A1C is in her target range. If a woman finds intercourse painful or feels less attractive due to weight gain or injection bruising, she may also be more reluctant to engage in intimate activity. This can cause stresses in the relationship that may negatively affect a woman's ability to adhere to her diabetes self-care practices and maintain target glycemic control (3).

Diabetes Management and FSD

Female patients should be encouraged to maintain healthy diabetes control, which can affect a woman's energy level during sexual activity. Sex is physical activity. If a woman's glucose level is inadequate at the start of sexual activity, her glucose level may drop further in response to her physical participation. Such a decrease may cause her to become drowsy or fall asleep and neglect to treat her lower glucose level or adjust her altered insulin need (9). As with other types of physical activity, it is appropriate to recommend more frequent glucose monitoring along with increased carbohydrate intake, as needed. It is also helpful to remind patients to limit their alcohol intake before sexual activity to help prevent the occurrence of alcohol-related hypoglycemia.

Clinical Application

RDs can provide women who suffer from FSD with an opportunity to share their frustrations and assure them that their experiences are very similar to many women who do not have diabetes. Asking about the problem removes it from the shadows and may relieve some of the stress that can develop for the person guarding a troubling secret (12). RDs can also encourage the use of over-the-counter vaginal lubricants to help relieve vaginal dryness and urge patients to meet with a gynecologist for evaluation and possible hormone treatment, if needed. It is also appropriate for RDs to suggest that patients seek counseling for marital stresses, if they exist.

Sex and Diabetes – For Him and For Her by Roszler and Rice (American Diabetes Association) contains additional information about diabetes-related sexual complications and offers tips, techniques, and guidance that can help the couple support one another emotionally and physically. Dr. Ruth Westheimer has also authored numerous books on sexual activity that are accessible and informative, including *Sex for Dummies* and *Dr. Ruth's Sex After 50*: *Revving Up the Romance, Passion and Excitement*.

Recent research on the Mediterranean diet, which is rich in olive oil, vegetables, grains, legumes, and fish and lower in animal products, suggests that women with type 2 diabetes who follow this regimen enjoy greater sexual satisfaction and may experience some improvement in desire, arousal, vaginal lubrication, orgasm, and intercourse-related pain (13). Two possible mechanisms may be responsible for these improvements: (1) the dietary fiber found in the Mediterranean diet may have anti-inflammatory properties and (2) individuals who follow the Mediterranean diet may have better self-care behaviors (13). Those who adhere to this meal planning regimen should adjust their portion sizes and food choices to meet their diabetes control requirements or ask their RD to help them with the adjustment (14). An updated version of this meal plan can be found at the Mediterranean Diet Foundation website www.fdmed.org.

Summary

Diabetes follows people wherever they go…even into the bedroom. Up to half (or more) of female patients with diabetes suffer from some type of diabetes-related sexual complication that may negatively affect the emotional support they receive from their partner. Without this important emotional support, the diabetes care and guidance that RDs offer may be more difficult to follow. The presence of sexual complications can also create additional stress in the lives of these patients. RDs do not have to be trained sex therapists or marriage counselors to provide patients with a compassionate ear and basic information about diabetes-related sexual complications and possible treatment options.

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Bone Health and Diabetes

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Abstract

Osteoporosis (decreased bone mass) is associated with significant morbidity and mortality following fractures. For reasons that are still not clear, people with both type 1 (T1DM) and type 2 (T2DM) diabetes have a higher incidence of bone fracture than the general population. Just as T1DM and T2DM differ in their pathophysiology and characterization, these diseases also appear to have different effects on bone. T1DM is usually associated with reduced bone mineral density (BMD), but normal or even increased BMD is seen in T2DM. Although this would seem to indicate that persons with T2DM are at decreased risk for fractures, several studies have demonstrated an increase in fracture risk. For any given BMD, bones in persons with diabetes appear to be less strong and more likely to fracture. With an increasing prevalence of diabetes in the United States, diabetes-related fractures may also increase, making fracture prevention strategies in such persons an important public health issue.

Introduction

Osteoporosis, the most common disorder of the bone, affects more than 10 million Americans and is associated with significant morbidity and mortality. This chronic condition is characterized by reduced bone strength, low bone mass, and higher risk of fracture. In adults, small amounts of bone minerals are removed daily for physiologic homeostasis. This must be balanced by an equal deposition of new minerals if bone strength is to be preserved. Osteoporosis can be prevented or slowed, but once bone damage occurs, it can be difficult to reverse. Osteoporosis-related fractures are a major public health concern, with 25% of hip fracture patients age 50 and older dying within the year following their fractures (1).

Diabetes is also an increasingly prevalent disease. Currently, 25.8 million children and adults in the United States, representing 8.3% of the population, have diabetes. This number is expected to increase twofold by 2025 (2). The total cost of diabetes is estimated at $174 billion (3), and this figure is expected to increase as the number and lifespan of people with diabetes rises.

For reasons that are still not clear, people with both T1DM and T2DM experience a higher incidence of bone fracture than the general population. This association may not be widely recognized or addressed as part of routine care for patients with diabetes. There is no formal definition of diabetic osteoporosis and no clear evidence that glycemic control can influence changes in the bones of patients with T1DM or T2DM. The purpose of this article is to review the associations between diabetes and bone fractures and to provide clinical applications of the information.

Literature Review

T1DM is characterized by insulin deficiency, while T2DM can be a state of insulin resistance, with resulting elevated circulating levels of insulin. Just as T1DM and T2DM differ in their pathophysiology and characterization, they also appear to have different effects on bone. T1DM is usually associated with reduced BMD (2,4). The duration of diabetes appears to have the greatest influence on BMD, with the lowest BMD being found in those who have had diabetes for more than 5 years (2).

Two recent meta-analyses of diabetes and risk of fracture have contributed significantly to understanding of these relationships. Both meta-analyses found a significant increase in the risk for fractures for patients with T1DM. In a 2007 meta-analysis, Vestergaard (4) found a 6.9 times higher risk of hip fracture in patients with T1DM compared to people without diabetes (relative risk=6.94, 95% confidence interval: 3.25–14.78). BMD was also decreased in those who had T1DM (4). A case-control study by the same authors in 2009 reported a trend toward increased fracture risk when diabetic kidney disease was present (5). The second meta-analysis by Janghorbani and associates (6) reported similar results, concluding that the risk of hip fracture in T1DM is 6.3 times greater than for a person with no diabetes

(relative risk=6.3, 95% confidence interval: 2.6–15.1).

Normal or even increased BMD is possible in T2DM. Obesity, which is highly prevalent in T2DM, could increase BMD through mechanical loading. Further, insulin is believed to be anabolic to bones and, therefore, the hyperinsulinemia present in some cases of T2DM may promote bone formation. Studies have shown an increase in BMD in T2DM, supporting the theory that an increased body mass index increases BMD (2). Although such findings would seem to indicate that persons with T2DM would be at decreased risk for fractures, several studies have, in fact, demonstrated an increase in fracture risk. Recent meta-analyses have reported a 1.4 to 1.7 times increased fracture risk in patients with T2DM when compared to those without diabetes, regardless of increased BMD (4,6). Vertebral risk fracture for T2DM is also increased, but BMD is not as useful in assessing this risk (7).

The Women's Health Initiative observational study compared postmenopausal women clinically diagnosed with T2DM to those without this diagnosis and found higher fracture risk at all fracture sites computed, including hip/pelvis/upper leg, spine/tailbone, and foot, after adjustment for multiple risk factors (8). This increased risk was present despite a greater BMD in those who had diabetes and was also highest among black women. This is one of the first studies to document a racial disparity in fracture risk among people with diabetes (8).

For any given BMD, bones in people with diabetes appear to be weaker and more likely to fracture than those in people without diabetes. There are several theories about this observation. Hyperglycemia itself can be detrimental to bone. In vitro models suggest that hyperglycemia may have negative effects on osteoblasts (9) (cells responsible for bone formation), although clinical evidence is limited. Meta-analysis examining the relationship between diabetes and fractures found no effect on BMD with glycemic control, as measured by glycolated hemoglobin (4). However, glycosylated hemoglobin is reflective of average blood glucose values over only the previous 2 to 3 months, and changes in BMD occur over years.

Another theory questions whether the accumulation of advanced glycation end products (AGEs) in collagen reduces bone strength. AGEs are proteins or lipids that form at a constant rate over time in the body but accumulate at an accelerated rate with diabetes because of increased exposure to glucose (10). Prime targets of AGE accumulation are the structural components of the connective tissue, which could alter collagen function, thereby altering bone function. Studies have demonstrated that AGEs have detrimental effects on osteoblasts and enhance the production of osteoclasts (cells responsible for bone breakdown) (11). Collagen fibers form beneficial covalent cross-links in bone matrix, but glycation of collagen changes the formation of this matrix, increasing the bone brittleness.

New evidence is also emerging from epidemiologic studies regarding BMD and diabetes-related comorbidities. These studies suggest that alterations in peripheral nerve function, vascular function, and kidney function are related to BMD, bone loss, and fracture in a dose-dependent manner (5,12).

Complications from diabetes (e.g., impaired eyesight, decreased balance related to neuropathies, and altered hemodynamics of the cardiovascular system) may increase the risk of falls and subsequent bone fractures. Decreased BMD could also be related to complications resulting from impaired vitamin D metabolism in kidney disease and decreased blood supply to the bones from vascular disease (5).

Hyperglycemia can impair renal calcium absorption, resulting in hypercalciuria, a condition that can resolve with the resolution of hyperglycemia. The development of renal insufficiency with associated impaired vitamin D metabolism and secondary hyperparathyroidism can also have detrimental effects on bone (13). Other theories involve the role of inflammatory cells and inflammation associated with both T1DM and T2DM. Investigators have reported enhanced expression of cytokinesis in diabetes that is capable of stimulating bone resorption, which could be partially responsible for the increased fracture risk (14).

The effectiveness of diabetes medical management may also contribute to bone health. Although insulin is believed to be anabolic to bones, treatment with insulin possibly may be associated with increased risk of fracture and falls. However, this may be reflective of more poorly controlled diabetes and autonomic neuropathy or episodes of hypoglycemia leading to falls. Some of the insulin-sensitizing agents used in the past in the treatment of T2DM, such as pioglitazone and rosiglitazone (thiazolidinediones), have been associated with decreased BMD and increased fracture risk (15–17).

Clinical Application

Diabetes is listed in the National Osteoporosis Foundation (NOF) guidelines as an endocrine disorder that may cause or contribute to osteoporosis and fractures (1). However, there are no specific guidelines for screening for fracture risk in patients with diabetes. Current NOF guidelines call for screening BMD measurements using dual-energy x-ray absorptiometry (DEXA) scanning for all women older than 65 years and all men older than 70 years, regardless of clinical risk factors. Younger postmenopausal women and men ages 50 to 69 with higher risk factor profiles, including smoking, low calcium intake, and inadequate physical activity, should also be evaluated.

Fractures can occur at higher BMDs in patients with diabetes. Although reduced BMD may be present in those with T1DM, evidence points to reduced bone strength that may not be detectable using current methods to quantify bone strength. BMD is considered the gold standard for evaluating fracture risk in patients who do not have diabetes, but fracture risk in T2DM is not reflected by this assessment. Patients with T2DM may have a high fracture rate without reduced BMD.

Based on the literature, the presence of diabetes must be part of the assessment when considering a patient's fracture risk. Patients with diabetes would benefit from counseling that includes the risk for fractures and osteoporosis. Guidelines for adequate intake of calcium (1,200 mg/day) and vitamin D (800 to 1,000 IU/day), as suggested by the NOF (Table), are advised. Achieving adequate glycemic control is critical to modifying the risk for fractures; data suggest that the development of other complications of diabetes can lead to falls and subsequent fractures. Avoidance of other risk factors for osteoporosis, such as smoking and excessive alcohol intake, also deserves emphasis.

Table. National Osteoporosis Foundation Nutrition Guidelines for Osteoporosis Prevention

Patient Group	Calcium	Vitamin D
Women (<50 years)	1,000 mg	400 to 800 IU
Women (≥50 years)	1,200 mg	800 to 1,000 IU
Men (<50 years)	1,000 mg	400 to 800 IU
Men (50 through 70 years)	1,000 mg	800 to 1,000 IU
Men (≥71 years)	1,200 mg	800 to 1,000 IU

Summary

Both T1DM and T2DM are associated with an increased risk for fractures and falls, but there is no clear definition of osteoporosis in diabetes. Although T1DM is associated with decreased BMD, those with T2DM can have normal or increased BMD. With the growing prevalence of diabetes, the contribution of diabetes to fractures may increase, making fracture prevention strategies in people with diabetes an important public health issue.

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Medical Nutrition Therapy for Cystic Fibrosis-related Diabetes

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Abstract

Achieving and maintaining good nutritional status is the cornerstone of treatment for people of all ages with cystic fibrosis (CF) and is essential for optimal lung function and survival. Critical differences in medical nutrition therapy (MNT) for CF-related diabetes (CFRD), gestational diabetes (GDM), and prediabetes are important to understand to guide people with CF to optimal health and survival.

Introduction

Approximately 1 in 3,500 children in the United States is born with CF each year. CF affects all racial and ethnic groups but is more common in Caucasians. About 30,000 people in the United States have CF. In the 1950s, life expectancy for CF was less than 5 years. By 2011, life expectancy had increased to 36.8 years, with many people living well into their sixth and seventh decades. The oldest person in the United States in 2011 with CF was 81 years (1). In 2011, more than 48% of people with CF in the United States were adults, and that number continues to increase.

CFRD is the most common comorbidity in people with CF, occurring in 20% of adolescents and 40% to 50% of adults (2). The etiology for CFRD is due to underlying insulin deficiency from fibrosis of beta-cells and reduced beta-cell mass (3). MNT for CFRD drastically differs from that recommended for type 1 and 2 diabetes in terms of energy, fat, protein, sodium, and supplemental vitamins and minerals. In addition, MNT differs from that used for GDM and prediabetes. Adequate energy intake to maintain the recommended body mass index (BMI) for those who have CF is critical for health and survival. Insulin therapy has been shown to be superior to oral glycemic agents in achieving and maintaining nutritional status and survival (4).

Literature Review

Malnutrition in CF results from a combination of factors: 1) inadequate energy and micronutrient intake complicated by malabsorption due to pancreatic exocrine deficiency that requires pancreatic enzyme replacement therapy, 2) increased resting metabolic rate due to decreased pulmonary function and increased work of breathing, 3) insulin deficiency with catabolism of lean body mass and adipose stores from fibrosis of beta-cells and reduced beta-cell mass, and 4) recurrent sinopulmonary infections and chronic inflammation (3,5,6). Higher energy intakes are needed for people with CF to maintain weight and nutritional status. Improved survival in adults at least 20 years of age has been demonstrated in women with BMIs of at least 22 and men with BMIs of at least 23 (7). For children, the goal is achievement and maintenance of the 50th percentile for weight and stature-for-age (7).

The diagnosis of CFRD does not change the usual CF MNT recommendations. Normalization of blood glucose is essential to optimize nutrient metabolism and to improve BMI and lean body mass (4). Calories should almost never be restricted. People with CF require routine vitamin and mineral supplementation due to malabsorption as well as a high-sodium diet due to losses of this element in sweat. Low-sodium diets can lead to hyponatremia, seizures, and death (7). No deaths have been reported from atherosclerotic cardiovascular disease (CVD) among those who have CF, so standard MNT for CVD is not applicable in this population (8). Microvascular complications do occur in CFRD and are related to duration and metabolic control of diabetes. Protein restriction is not recommended if nephropathy occurs because such restriction increases the risk of malnutrition (9). Insulin is superior to oral glycemic agents in reversing protein catabolism, weight loss, and pulmonary function decline because of its anabolic effects (4).

GDM in CF

Women with CF are at higher risk for GDM because of underlying insulin deficiency (9). Women who have both CF and GDM have increased energy needs and require close

monitoring of weight gain and nutritional status. The use of oral supplements may be necessary to promote adequate weight gain. Because of the risk of suboptimal weight gain, MNT recommendations do not restrict calories or carbohydrate, and most affected women require insulin. Insulin should be matched to carbohydrate intake to optimize blood glucose control (9).

Prediabetes in CF

Prediabetes in CF is due to underlying insulin deficiency and cannot be prevented. MNT for prediabetes in the general population is not applicable to people with CF. Weight loss is almost never recommended. Exercise is beneficial for overall health but will not slow the progression toward CFRD due to the progressive insulin deficiency. Spreading intake of carbohydrates throughout the day and replacing empty calorie-carbohydrates with nutrient-dense carbohydrates is recommended. Maintenance of a healthy weight and nutritional status must be monitored closely (9).

Clinical Application

Typical MNT recommendations for type 1 and 2 diabetes, GDM, and prediabetes do not apply to people with CF (Table). Educational materials and webcasts specific to CFRD are available from the Cystic Fibrosis Foundation (10,11).

Summary

People with CFRD should be collaboratively managed by their pulmonary and endocrinology teams. Registered dietitians from both teams should communicate regularly to assist people with CFRD toward optimal management for improved health and survival.

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Table. Comparison of Nutrition Recommendations for Type 1 and 2 Diabetes and CFRD (9)

Nutrient	Type 1 and 2 Diabetes	CFRD
Energy (Calories)	As needed for growth, maintenance, or reduction diets.	1.2 to 1.5 times DRI for age; individualized based on weight gain and growth.
Carbohydrate	Individualized. Monitor carbohydrates to achieve glycemic control. Choose from fruits, vegetables, whole grains and fiber-containing foods, legumes, and low-fat milk. Sugar alcohols and non-nutritive sweeteners are safe within FDA established consumption guidelines.	Individualized. Monitor carbohydrates to achieve glycemic control. Use artificial sweeteners sparingly due to lower calorie content.
Fat	Limit saturated fat to <7% of total calories, minimize intake of trans-fat, limit dietary cholesterol to <200 mg/day. Consume two or more servings per week of fish high in n-3 polyunsaturated fatty acids.	No restriction on type of fat. High fat necessary for weight maintenance. Aim for 35% to 40% total calories.
Protein	15% to 20% of total calories; reduce to 0.8 to 1.0 g/kg with nephropathy.	Approximately 1.5 to 2.0 times the DRI for age; no reduction for nephropathy.
Sodium	<2,300 mg/day for blood pressure control.	Liberal, high-salt diet, especially in warm conditions and/or when exercising.
Vitamins, Minerals	No supplementation necessary unless deficiency noted.	Routine supplementation with CF-specific multivitamins or a multivitamin and additional fat-soluble vitamins A, D, E, and K.
Alcohol	If consumed, limit to a moderate amount: 1 drink per day for women, 2 drinks per day or less for men.	Consult with physician due to the higher prevalence of liver disease in CF and possible use of hepatotoxic drugs.
Special Circumstances	**Type 1 and 2 Diabetes**	**CFRD**
Gestational Diabetes	Restricted energy/carbohydrate for weight and blood glucose control.	No energy or carbohydrate restriction. Provide adequate energy for weight gain.
IGT/Prediabetes	Weight loss of 5% to 10% recommended, low-fat diet.	No weight loss, spread carbohydrate intake throughout day, consume nutrient-dense beverages

CF=cystic fibrosis, CFRD=cystic fibrosis-related diabetes, DRI=dietary reference intake, FDA=United States Food and Drug Administration, IGT=impaired glucose tolerance

After reading this issue of *On the Cutting Edge*, "Less Well-known Co-Morbidities of Diabetes," DCE members can earn 3.0 hours of free continuing professional education units (CPEUs level II) approved by the Commission on Dietetic Registration (CDR). CPE eligibility is based on active DCE membership status from June 1, 2012 to May 31, 2013.

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CPE Credit Self-Assessment Questionnaire

1) Individuals with diabetes-related hearing loss have the most difficulty hearing:
   a. High frequencies, and therefore have more difficulty hearing a female educator than a male speaker.
   b. In a large group of people, so should always sit in the front row of seats.
   c. Low frequencies, and therefore hear a male educator's voice more clearly.
   d. The educator clearly, but rarely have vision problems.

2) Which of the following steps should the diabetes educator take to accommodate the hearing impaired patient?
   a. Ask the patient to rephrase what has been said to ensure the educator's message is received.
   b. Make sure the patient has an unobstructed view of the speaker in individual or group settings.
   c. Minimize background noise.
   d. All of the above

3) Select the correct statement below:
   a. Gingivitis is a severe form of periodontal disease.
   b. Glycemic control can greatly affect the progression of periodontal disease.
   c. Individuals with diabetes need the same frequency of preventive dental care visits as the general population.
   d. Periodontal disease is rarely a complication of diabetes.

4) Which of the following statement is correct?
   a. Alzheimer's dementia is rarely a concern for individuals with diabetes.
   b. Smoking is not a cause of vascular dementia.
   c. There is no evidence that poorly controlled diabetes can cause conditions that lead to vascular dementia.
   d. Vascular dementia is a known possible complication of both type 1 and 2 diabetes related to microvascular damage to the brain.

5) Women with diabetes experience which of the following female sexual symptoms more frequently than women without diabetes:
   a. Vaginal dryness and urinary tract infections
   b. Vaginal dryness and poor libido
   c. Painful intercourse and slowed arousal
   d. Painful intercourse and orgasm difficulty

6) The incidence of osteoporosis for persons with diabetes compared to the incidence in the general population is:
   a. Greater for persons with type 1 diabetes and less for persons with type 2 diabetes
   b. Greater for persons with type 2 diabetes and less for persons with type 1 diabetes
   c. Greater for persons with either type 1 diabetes or type 2 diabetes
   d. There is no difference in incidence for persons with diabetes compared to the general population

7) A possible role(s) of the RD in treating a female with diabetes and female sexual dysfunction (FSD) is:
   a. Recommend hormone treatment regimen
   b. Suggest the use of over the counter vaginal lubricants
   c. Provide counseling addressing the issues of marital stress that may revolve around the issue of FSD
   d. It is inappropriate for the RD to ask questions about this issue

8) Which of the following guidelines is an appropriate recommendation to assess or reduce the risk of osteoporosis for persons with diabetes?
   a. Consume twice the recommendations for adequate intake of calcium and vitamin D
   b. For patients with T2DM, obtain a dual-energy x-ray absorptiometry (DEXA) to assess bone mineral density (BMD) and fracture risk
   c. Avoid smoking and excessive alcohol intake
   d. Be sure patients are aware of the recommendations for an adequate intake of calcium and vitamin D
   e. Both C and D

9) Which statement most accurately describes MNT for cystic fibrosis-related diabetes:
   a. Is the same as MNT for type 2 diabetes
   b. Is a calorie restricted diet to help reduce insulin resistance
   c. Includes sodium restriction of <2300 mg/sodium
   d. Provides about 1.5 times more calories and protein to prevent malnutrition

10) The most promising strategy for controlling the progression of non-alcoholic fatty liver disease (NAFLD) is
   a. Intensive lifestyle intervention
   b. Insulin therapy
   c. DPP-4 Inhibitors
   d. Avoidance of alcohol

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Diabetes Care and Education
a dietetic practice group of the
Academy of Nutrition and Dietetics

PRINTED ON RECYCLED PAPER

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PUBLICATION SUPPORTED BY AN EDUCATIONAL GRANT FROM:

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