The NEW GASTROENTEROLOGIST

INSIGHTS FOR FELLOWS & YOUNG GIs A Quarterly Supplement to GI & Hepatology News | Winter 2016

Managing Celiac Disease

A Brief Overview **12**

Letter
FROM THE EDITOR

Bryson W. Katona is an instructor of medicine in the division of gastroenterology at the University of Pennsylvania.

Dear Colleagues,

With the increasing prevalence of celiac disease in our society, proper diagnosis and management of this condition has become an important part of every gastroenterologist's practice. In this issue of *The New Gastroenterologist*, Laura Pace, David Kunkel, and Sheila Crowe from the University of California, San Diego, have prepared an excellent overview covering the current state of diagnosis and management of celiac disease, which will certainly serve as a useful tool for improving our clinical practice.

As we look back, it is very apparent that 2015 was a tremendously productive year throughout the field of gastroenterology. In this issue, Hassan Siddiki, Marcelo Vela, Amy Foxx-Orenstein, and Jonathan Leighton from the Mayo Clinic, Scottsdale, Ariz., review the key findings from the most important, practice-changing papers over the last year in the fields of esophagology, inflammatory bowel disease, and motility.

This issue also contains an inspiring piece about the importance and effectiveness of advocacy in our field, written by Erin Forster Perlini from the University of Miami and Peter Margolis from Brown University, as well as a useful primer on delivering effective medical lectures authored by Peter Buch from the University of Connecticut. Finally, retirement planning should always be a key part of a GI's financial portfolio, and this issue features an informative article on the "nuts and bolts" of individual retirement accounts (IRAs).

As always, our goal is to tailor the content of *The New Gastroenterologist* to the interests and needs of the young GI community. Therefore, if you have any suggestions, or would be interested in contributing content to a future issue, please e-mail me at bryson.katona@uphs.upenn.edu or our editorial assistant, Ryan Farrell, at rfarrell@gastro.org.

Sincerely,
Bryson W. Katona, M.D., Ph.D.
Editor in Chief

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The NEW GASTROENTEROLOGIST

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ON THE COVER

Inlay: Dr. David C. Kunkel, Dr. Sheila E. Crowe, and Dr. Laura A. Pace.
Photo courtesy of Dr. Peter B. Ernst
Background: Immunofluorescence pattern of endomysial antibodies.
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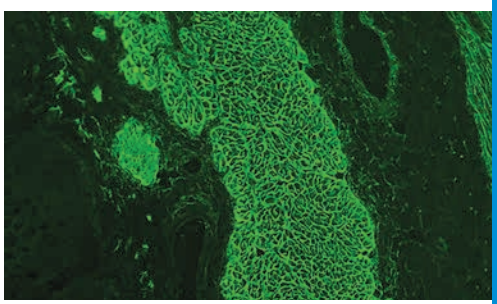


DDSEP® 7
Digestive Diseases Self-Education Program®

QUESTIONS

// Answers on page 30

Q1: A 46-year-old man is referred for abnormal liver function tests. His past medical history includes obesity and depression, a body mass index of 31 kg/m^2, AST 67, IU/L; ALT, 79 IU/L; transferrin saturation, 70%; ferritin, 1393 microg/L; WBC, 6.2 × 1000/mm$^{3;}$ hemoglobin, 14.2 g/dL; platelets 125,000/mm^3, HBsAb negative, HBsAg positive, HBcAb positive, HBeAg negative, and HBV DNA unmeasurable. Autoimmune work up was negative. HFE gene test revealed C282Y/C282Y.

The most appropriate next step would be:

A. MRI iron quantification of the liver
B. Phlebotomy
C. Liver biopsy
D. Tenofovir
E. Weight loss

Q2: A 70-year-old patient is admitted to the hospital with periumbilical abdominal pain, nausea with vomiting, and bloody diarrhea. She has a history of coronary artery disease and coronary artery bypass grafting 3 years earlier. Her cardiac ejection fraction is 40% and she has a history of paroxysmal atrial fibrillation. She was recently treated with ciprofloxacin for a urinary tract infection. On exam, her abdomen is soft with tenderness and guarding with palpation in the periumbilical area. She was found to have a WBC 15 × 1000/mm^3, hematocrit 45%, and platelets 303,000/mm^3. She was also found to have metabolic acidosis. She underwent an arteriogram and was found to have ischemia of the superior mesenteric artery. She then was noted to have worsening of her abdominal pain and peritoneal signs on exam and was taken to the operating room for exploratory laparotomy with subsequent extensive resection of her small bowel.

The following are factors that determine the likelihood of successful transition to an enteral diet, except:

A. Length of remaining small intestine
B. Presence of the ileocecal region
C. Intestinal adaptation
D. Presence of colon
E. Presence of jejunum rather than ileum

For more information about DDSEP© visit gastro.org/ddsep

News from the AGA

AGA Helps Prepare for Life After Fellowship

AGA is holding five free 1-day regional workshops for GI fellows in 2016. During these practice skills workshops, which will be held throughout the U.S., senior and junior GI leaders will guide you through the various practice options and address topics rarely discussed during fellowship.

These topics include employment models, partnerships, hospital politics, billing and coding, compliance, contracts, and more.

This is a great opportunity to develop an effective action plan for achieving your career goals and to ensure you are on track for success as you transition into practice. Find an AGA workshop near you and mark your calendars today.

- **San Diego –** Sat., Feb. 20, 8 a.m.–1 p.m., Univ. of California, San Diego; Course Director: Sheila E. Crowe, M.D., AGAF.
- **New York** – Sat., Feb. 20, 8 a.m.–1:30 p.m., Mount Sinai Hospital, Hatch Auditorium; Course Director: Brijen Shah, M.D.
- **Houston** – Sat., Feb. 20, 8 a.m.–1 p.m., Baylor College of Medicine, McNair; Course Directors: Avinash Ketwaroo, M.D., and Mohamed Othman, M.D.
- **Boston** – Fri., March 18, 9:30 a.m.–2:30 p.m., Beth Israel Deaconess Medical Center; Course Director: Adam Cheifetz, M.D., AGAF.
- **Philadelphia** – Wed., April 6, 8 a.m.–12:30 p.m., Univ. of Pennsylvania; Course Directors: Jan-Michael Klapproth, M.D., and Laurel Fisher, M.D., AGAF.

To find out more information and to register, visit http://www.gastro.org/news_items/2015/12/03/prepare-for-life-after-fellowship-with-aga. Contact Carol Brown at cbrown@gastro.org with any questions. ■

Attend the 2016 AGA Tech Summit

Register for the seventh annual AGA Tech Summit (http://www.gastro.org/in-person/2016/3/31/2016-aga-tech-summit), which will take place March 31 through April 1, 2016, at the InterContinental hotel in Boston. The summit addresses critical elements that impact the likelihood of success in developing and obtaining adoption and coverage of new medical technologies in today's GI marketplace.

Attendees will learn how AGA is creating a more supportive environment for new technologies, through resources such as the AGA Center for GI Innovation and Technology (http://www.gastro.org/cgit) and initiatives that support innovation in digestive and metabolic diseases.

Sessions will explore obtaining funding for new medical devices and technology, the impact of personalized medicine on the future of care delivery, diagnostics in chronic diseases, novel transplant technologies, and more.

Companies and entrepreneurs with an innovative technology or FDA-regulated product looking to get it financed, licensed, or distributed are also encouraged to submit an application for an opportunity to present during the "Shark Tank" portion of the conference. A panel of business development leaders, investors, entrepreneurs, and other strategic partners will provide valuable feedback. Apply now at http://netforum.gastro.org/eweb/DynamicPage.aspx?WebCode=TC.

Also, make sure to attend How to Innovate in Digestive Health, a supplement to the AGA Tech Summit, on April 2, 2016, to hear from leaders in the physician and medtech communities on the process of innovation, protecting intellectual property, and strategies for achieving success. Learn more at http://www.gastro.org/in-person/2016/04/02/how-to-innovate-in-digestive-health. ■

Save the Date: 2016 AGA Postgraduate Course

Mark your calendars for the 2016 AGA Postgraduate Course, which will take place Saturday, May 21 and Sunday, May 22, 2016, in San Diego. It will be held in conjunction with Digestive Disease Week® 2016 (www.ddw.org).

Attend this course to hear world-renowned experts present cognitive and technical skills for today's gastroenterologist. Six general sessions, 13 clinical challenge sessions, and 12 lunch breakout sessions will keep you informed about the critical updates for 2016. You will leave this course with the newest advances that will help you make confident decisions for your patients.

Members-only registration opens Jan. 6, 2016 (Jan. 13 for nonmembers). AGA members can register for the course at reduced registration rates. Register early to secure your spot and save $75 on the registration fee.

View the agenda and learn more at http://www.gastro.org/in-person/2015/10/27/2016-aga-postgraduate-course. ■

Help Your Patients Manage Chronic Constipation

AGA has published the latest in a series of guideline-based resources for members to provide their patients. Based on the published AGA guideline on constipation, (http://www.gastrojournal.org/article/S0016-5085(12)01545-4/pdf) the new patient guide explains how a gastroenterologist can help when diet and lifestyle modifications fail.

The guide also explains defecatory disorders, slow-transit and normal-transit constipation, as well as information about tests and medication options in language that is accessible to patients.

AGA's evidence-based patient guides can help improve the efficiency of office visits and the value you provide patients.

If your patients are suffering from chronic constipation, make sure to let them know about this helpful AGA patient guide (www.gastro.org/info_for_patients/2015/10/29/a-patient-guide-managing-chronic-constipation). ■

Learn How to Avoid a Reimbursement Loss

The Physician Quality Reporting System (PQRS) will apply a negative 2% payment adjustment in 2017 if practices do not report data on quality measures for covered professional services furnished to Medicare beneficiaries in 2015. By participating in AGA's Digestive Health Recognition Program™ (www.agarecognition.org) or DHRP, members can report on quality measures and work to avoid this negative payment adjustment.

To learn more about DHRP, register for a free, 30-minute webinar taking place during these two dates (attendee.gotowebinar.com/rt/2452555612218433025)

• Feb. 25, 2016, 12 p.m. ET

The deadline to enroll for the 2015 PQRS year through DHRP is Feb. 26, 2016. Members pay $300 to enroll.

DHRP is made possible by support from AbbVie; Gilead Science, Inc.; Janssen Biotech, Inc. and Janssen Therapeutics, Division of Janssen Products, LLP; and Shire Pharmaceuticals. ■

What's Your Diagnosis?

An unusual cause of hematochezia in an elderly man

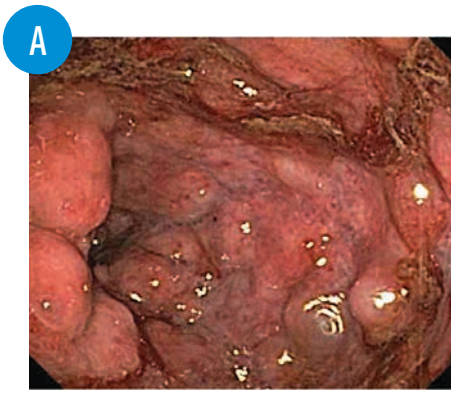
Published previously in Gastroenterology (2014;147:943-1188)

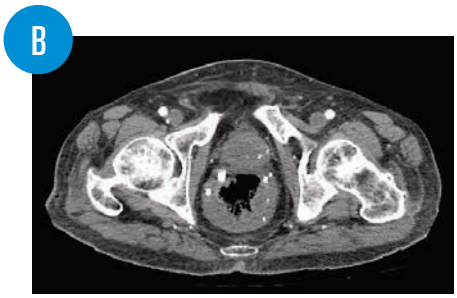
By Greg S. Cohen, M.D.

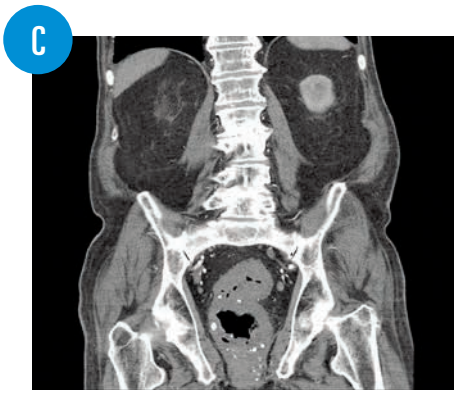
An 82-year-old man with congestive heart failure was transferred from an outside hospital for further management of recurrent hematochezia. He had a history of recurrent rectal bleeding since he was a young child, with one episode of hematochezia every week on average. He was diagnosed with ulcerative colitis as a child, but was never treated for it. He continued to have recurrent hematochezia throughout adulthood, but it had become significantly worse in the 6 months before admission. He reported hematochezia "every 6 minutes" the week before admission. He had no history of proctalgia or tenesmus. He was taking aspirin 81 mg daily until shortly before admission. At the outside hospital his admission hemoglobin was 8.9 g/dL. Platelets, prothrombin time, and partial thromboplastin time were normal. A colonoscopy was performed at the outside hospital with findings of "purplish inflammation of the rectum from the anal verge to a distance of 10 cm" (Figure A) with active rectal bleeding treated with a bipolar probe. Biopsies from this area were normal. Computed tomography (CT) of the abdomen and pelvis showed concentric rectosigmoid wall thickening with clusters of calcification in the colon wall (Figures B, C). ■

What is the diagnosis?

Dr. Cohen is in the Department of Medicine, Division of Gastroenterology, Northwestern University, Chicago.







Courtesy AGA Institute

See ***The Answer*** on page 31

AGA Outlook

For more information about upcoming events and awards deadlines, please visit www.gastro.org

Upcoming Events

Feb. 20; Mar. 18; Apr. 6, 2016
Practice Skills Workshops
These workshops are targeted to GI fellows, and will provide valuable insight into and information about how to start a successful career in a variety of practice settings. These workshops will be held at five separate locations.
San Diego, CA (2/20), Houston, TX (2/20), New York, NY (2/20), Boston, MA (3/18), Philadelphia, PA (4/6)

Feb. 25-26, 2016
Psychosocial Care Integration in Inflammatory Bowel Disease & Chronic Illness Management
This conference features national experts presenting on psychosocial care integration and health care reform for GIs and related professionals.
Universal City, CA

Mar. 5-6, 2016
2016 Gut Microbiota for Health World Summit
Join a prestigious faculty of researchers and clinicians for the latest insight on advances in human gut microbiota research.
Miami, FL

Mar. 11, 2016
2016 Partners in Quality Meeting
Experts in the field will show you how to best implement quality improvement techniques that can help your practice flourish within an accountable care market.
Washington, D.C.

Mar. 11-12, 2016
AGA-AASLD Academic Skills Workshop
Take advantage of valuable tools to shape a successful career in the highly competitive environment of medical academia. This enriching learning opportunity will provide future physician-scientists career/life guidance via mentor-mentee pairings.
Phoenix, AZ

Mar. 26, 2016
20th Annual Virginia Liver Symposium & Update in Gastroenterology
State-of-the-art talks in gastroenterology, hepatology, and nutrition.
Richmond, VA

Mar. 31-Apr. 1, 2016
2016 AGA Tech Summit
Join leaders in the physician, investor, regulatory, and medtech communities as they examine the issues surrounding the development and delivery of new GI medical technologies.
Boston, MA

Apr. 2, 2016
How to Innovate in Digestive Health
Experienced physician innovators and leaders in the medtech community provide an introduction on the process of innovation – revealing exactly what's needed to take an idea from concept to reality.
Boston, MA

May 21-22, 2016
2016 AGA Spring Postgraduate Course
The 2016 AGA Spring Postgraduate Course, Cognitive and Technical Gastroenterology for Patients' Needs, is a clinically focused, multi-topic course that offers immediately applicable information. Held in conjunction with DDW®.
San Diego, CA

May 21-24, 2016
Digestive Disease Week® (DDW)
The premier meeting for the GI professional. Every year it attracts approximately 15,000 physicians, researchers, and academics from around the world who desire to stay up-to-date in the field.
San Diego, CA

Nov. 1, 2016
ABIM® Gastroenterology Certification Exam
Registration dates are from March 1 to May 16, 2016 (with late registration from May 17 to June 1, 2016).

Nov. 2, 2016
ABIM® Transplant Hepatology Certification Exam
Registration dates are from March 1 to May 16, 2016 (with late registration from May 17 to June 1, 2016).

Awards Application Deadlines

AGA/AGA-GRG Fellow Travel and Abstract of the Year Awards
Deadline: Feb. 26, 2016

AGA-Moti L. & Kamla Rustgi International Travel Awards
Deadline: Feb. 26, 2016

AGA Student Abstract Prizes
Deadline: March 4, 2016

Research Scholar Award (RSA)
Deadline: Sept. 23, 2016

Retirement Strategies: The Differences Between Traditional and Roth IRAs

By Ross Cameratta

Mr. Cameratta is a financial advisor at North Star Resource Group, Philadelphia.
He can be reached at Ross.Cameratta@northstarfinancial.com

Planning for retirement is oftentimes a main concern for physicians as they begin building the financial plan for their future. A common question when saving for retirement is that if you are taking advantage of your employee-sponsored plans, what other savings vehicles are available? In this article we will touch on using traditional and Roth IRAs to continue to build your retirement savings.

What are traditional and Roth IRAs?

Traditional and Roth IRAs are individual retirement accounts that allow you to save outside of your employer-offered retirement plans. Both of these investment vehicles have very unique benefits and, for certain people, can be a beneficial part of a diversified financial plan. The current maximum contribution limit for both the traditional and Roth IRA is $5,500 per year. For people who are age 50 or older, the government allows you to make "catch-up contributions" increasing the maximum to $6,500 per year.

Am I eligible to fund a traditional IRA or Roth IRA?

To be eligible to fund a traditional IRA you need to have taxable income for that year and fund this account before you reach age 70½.

The Roth IRA has additional eligibility restrictions compared to the traditional IRA. The factors that go into deciding whether or not you are eligible to fully fund a Roth IRA are your income and tax filing status. For a single individual, you have to make $117,000 or less to be eligible. As a married couple filing jointly, you have to make a combined income of $184,000 or less to be eligible. If you are married and filing separately, you have to make less than $10,000 a year to be eligible to fund a Roth IRA. If you fall into the married filing separately category, there is a conversion method you can use called the "Backdoor Roth IRA" that we will discuss later.

What are the tax benefits for a traditional IRA vs. Roth IRA?

The first tax benefit for the traditional IRA is the ability to make pre-tax contributions much like a 401(k) or 403(b). By making these contributions on a pre-tax basis, you will be able to fully or partially deduct your contribution for that year off of your gross income and lower your taxable income. You should consult your tax advisor to determine whether you are able to fully or partially deduct your contribution depending on the rules set by the IRS. After you make your annual contribution into your account, the funds that are being invested grow on a tax-deferred basis, which allows the funds to grow without being taxed until you withdraw them in retirement. Once you are retired and ready to begin making withdrawals from this account, it will be subject to federal income tax.

When utilizing a Roth IRA, the con-

tributions made during the year are done with after-tax dollars and grow tax deferred like the traditional IRA. The main advantage of this account is that in retirement you are able to withdraw these funds completely tax free, which can offer you tax diversification in retirement when deciding where to withdraw income.

When can I withdraw money from my traditional IRA and Roth IRA without penalty?

You are able to begin withdrawing from your traditional IRA without incurring a 10% penalty once you reach the age of 59 ½; however, you will still be taxed on the withdrawal at your ordinary income rate. For people under the age of 59 ½, there are a few exceptions of where you are able to withdraw funds without incurring a penalty. These exceptions are made for a first-time home purchase, qualified educational expenses, death or disability, unreimbursed medical expenses, and health insurance if you are unemployed. If you elect not to withdraw from your traditional IRA and reach the age 70½, the owner will have to begin taking mandatory minimum required distributions from the account or incur penalties.

If you are contributing to a Roth IRA, you are able to begin withdrawing funds tax and penalty free at age 59½. Much like the traditional IRA, you have a few exceptions that allow you to withdraw funds without penalties (e.g., death, disability, or a qualified first-time home purchase). Unlike traditional IRAs, Roth IRAs are not subject to minimum required distributions during the lifetime of the original owner of the account.

Once my income goes above the income restrictions, can I still fund my Roth IRA?

As mentioned briefly in the beginning of this article, if you do not qualify to contribute to a Roth IRA directly (based on income restrictions

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or filing married but separately), you can still utilize the "Backdoor" method to fund the account. The first step in this strategy is to open up a nondeductible traditional IRA which you will fund with after-tax dollars. Once you make the contribution to the nondeductible traditional IRA you will have to fill out a conversion form, which will then convert the money to your Roth IRA.

This strategy can also be used if you have old retirement accounts from past employers, as you are able to roll over an old 401(k) or 403(b) to your nondeductible traditional IRA and then convert these funds into your Roth IRA. One thing to consider before moving forward with this strategy is that, based on the funds in your 401(k) or 403(b) not being taxed yet, you will owe taxes on the amount you decide to convert into your Roth IRA. This can be beneficial if you have old retirement accounts during training as you will be able to convert this money and get a jump start on building tax-free savings for retirement and will pay the taxes at your lower income bracket. If you are currently thinking of taking advantage of this strategy you should consult your financial advisor to make sure this is an efficient strategy for your plan.

Developing a plan and deciding what retirement vehicles are most efficient for you are important steps in creating a bright future and providing for the lifestyle you are working to obtain. There are many resources that you can take advantage of to learn more about these vehicles, and being proactive in developing a strong strategy can provide the retirement you always imagined. ■

References

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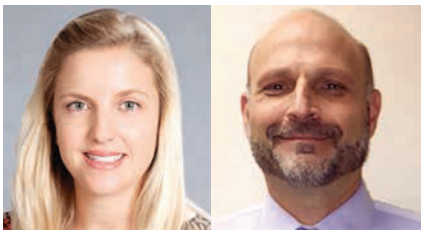
http://www.rothira.com/

https://www.irs.gov/Retirement-Plans/

Advocacy: Why It Matters

By Erin Forster Perlini, M.D., MPH, and Peter Margolis, M.D., AGAF



Dr. Perlini is a gastroenterology fellow PGY-5, at the University of Miami/ Jackson Memorial Hospital. Dr. Margolis is Chair, AGA Government Affairs Committee, and a clinical assistant professor of medicine, Brown University School of Medicine, Providence, R.I.

Many physicians are perplexed by the question, "What is advocacy?"

Our preferred answer is: advocacy is our community's unified voice for the greater good – both for patients and the profession of gastroenterology and hepatology. Typically, advocacy involves the process of educating both the public and policy makers about gastrointestinal illness and the importance of ensuring general access to GI specialty care. One such example is highlighting the success that access to screening and increasing awareness has had in reducing America's colon cancer rate and burden of disease. Rarely are we given such an opportunity, as no other screening modality can make similar claims.

Participation in advocacy is akin to being a good citizen – it serves as an investment now that pays dividends for quite some time. The passage of the Affordable Care Act represents the greatest political evolution in American health care since the establishment of Medicare. As a result of the current heated political climate, involvement in advocacy has become more critical than ever before. The future of medicine depends on advocacy. Practitioners must realize medical care should be determined by those who understand the patient/physician obligation and not by bureaucrats or insurance company executives who are obligated only to the financial bottom line. The time to act is NOW!

Far beyond procedural reimbursement, effective advocacy allows for health care issues to remain focused on patient-provider relationships – which is where they belong. Even medical schools and residency programs recognize advocacy as an educational need and are working to update curricula to reflect this and prepare physicians for their future roles. We have participated in advocacy days, both at the state and federal level, on behalf of a variety of organizations, including the AGA. These face-to-face meetings with lawmakers and their staff really drive home the importance of GI care, as it allows us to connect on a personal level about affected family members as well as our patients. Hearing directly from their constituents regarding important issues – such as access to colorectal cancer screening, appropriate funding for NIH, and graduate medical education – makes it difficult for policy makers to ignore. Speaking directly to your lawmaker has a profound impact on their understanding of the issues.

Participation in advocacy is akin to being a good citizen – it serves as an investment now that pays dividends for quite some time.

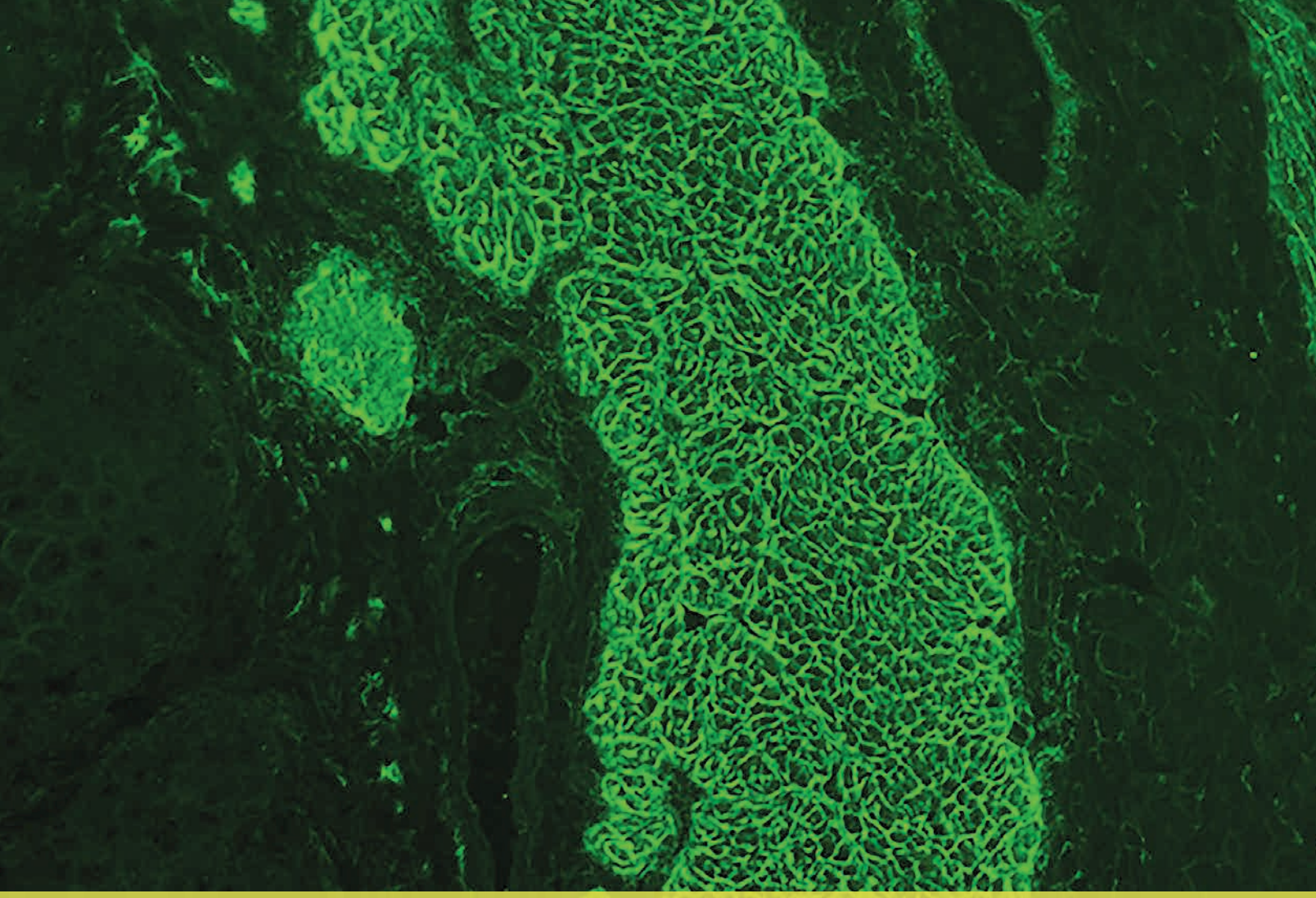
The AGA is the go-to GI society for politicians seeking educational materials on health care issues. As stakeholders, we need to capitalize

on our unique position to effectively bring about change both in gastroenterology and medicine more broadly. Past successes of AGA-driven advocacy include improving access to subspecialty care, more accurate billing, Medicare transparency, and ensuring that there is an adequate supply of well-trained gastroenterologists to treat a diverse population. The AGA has also produced clear, concise issue briefs on the value of colonoscopy in order to best inform policy makers – stating "its ability to save lives." By citing accessible and easy-to-understand statistics, colorectal cancer screening can become an issue upon which everyone can take action to improve access. The AGA has also worked toward securing and increasing NIH funding to ensure the future of unbiased drug development, clinical trials, and emphasizing the importance of fair reimbursement for endoscopic procedures requiring advanced training. These monies also support advanced fellowships such as transplant hepatology and advanced endoscopy; without them, fellows would need to seek outside funds if they wished to pursue further training after the 3-year GI fellowship, perhaps limiting opportunities for those unable to do so.

Getting involved in advocacy efforts is easier than you might think. Myriad opportunities exist from volunteering for local events – such as walks for colon cancer awareness and involvement in state gastroenterological societies – to more traditional programs such as the AGA Government Affairs Committee. Also, the AGA hosts its annual "Advocacy Day" in Washington, D.C., during which physicians meet with members of Congress and their staffers to discuss key issues related to gastroenterology during prime-time legislative sessions. Not only does this experience teach practitioners about what happens on Capitol Hill in general, but also how seemingly unrelated issues such as insurance coverage for diagnostic versus screening colonoscopy can impact adherence to colonoscopy guidelines.

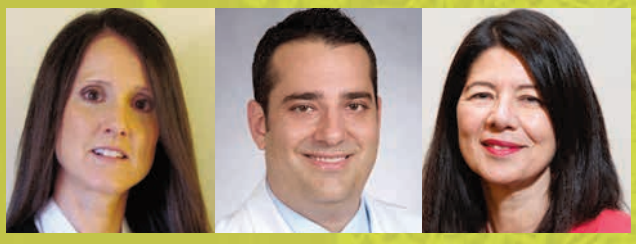
The simplest way to become involved is to financially support the AGA Political Action Committee (PAC). This is our society's way to support lawmakers who support our patients' interests and our profession, and understand the importance of good GI care. The AGA PAC has always offered bipartisan support for lawmakers who understand our specialty's priorities. The best thing about your financial support is that 100% of the funds go to support legislators who are champions of GI. It is the only gastroenterology PAC where such a financial relationship exists, because the AGA itself supports the overhead costs of the PAC.

The decision facing you now is not whether it is necessary to get involved, but how to do it! Exercise your right to a voice – it's a measure of your strength and dedication to patient care. You can begin by visiting our Advocacy and PAC pages at https://gastroadvocacy.org/actionalerts.aspx and http://www.gastro.org/take-action/aga-pac. ■



Managing Celiac Disease: A Brief Overview

By Laura A. Pace, M.D., Ph.D., David C. Kunkel, M.D., and Sheila E. Crowe, M.D., AGAF



Dr. Pace is a fourth-year GI fellow, University of California, San Diego; Dr. Kunkel is an assistant professor, University of California, San Diego; and Dr. Crowe is a professor of medicine, University of California, San Diego.

Historical perspective

Celiac disease (CD) is an immune-mediated small intestinal enteropathy that occurs in a small fraction of genetically susceptible individuals upon exposure to dietary gluten. Symptoms consistent with CD have been described in the literature as early as 250 B.C., and archeological evidence suggests that phenotypic forms of the disease were present in the first century A.D.[1]

Approximately 20%-30% of the United States population carries the HLA-DQ2/HLA-DQ8 genes that confer a genetic risk for the development of CD.[2] The overall prevalence of CD is approximately 1% in the general population, therefore only approximately 2%-3% of genetically susceptible individuals will develop CD over their lifetime.[3]

There has been a fivefold increase in the prevalence of CD over the past 50 years, rising from approximately 0.2% in the 1950s to at least 1% today.[4] CD was previously considered a disease of childhood, yet now it is understood that it can develop and be diagnosed at nearly any age. This suggests that there are other factors beyond genetic risk that result in the manifestation of CD. The past 50 years have also seen a dramatic increase in the prevalence of other autoimmune diseases, food allergies, and the emergence of an entirely new allergic disease, eosinophilic esophagitis (EoE).[5-7]

Table 1

Test Name	Sensitivity	Specificity	Clinical Use
TTG IgA	~76%-95%	~95%-98%	First line test
TTG IgG (IgA sufficiency)	~30%-70%	98%	Only useful in IgA deficiency*
TTG IgG (IgA deficiency)	~95%	~95%	Only useful in IgA deficiency*
DGP IgA	~74%-95%	~95%	May detect TTG seronegative individuals
DGP IgG	~60%-95%	~98%	Immunofluorescence test is very specific
Endomysial antibody IgA	89%-92%	~99%	Immunofluorescence test is very specific
Anti-Gliadin IgA, IgG	52%-100%	47%-100%	No longer routinely available, replaced by DGP antibodies

**Individuals with partial IgA deficiency can typically mount an adequate IgA serologic response. Check total IgA levels if concerned for complete IgA deficiency.*

Clinical presentation

CD is a heterogeneous disorder ranging from asymptomatic phenotypes to severe cases of malnutrition.[3] It is also considered a systemic illness, with some individuals reporting a predominance of gastrointestinal symptoms and others reporting extraintestinal symptoms such as fatigue, joint pain, and cognitive impairment. The diagnosis of CD is complicated by this heterogeneity in phenotype and presentation, along with the rapidly evolving prevalence rates and the diverse populations affected.

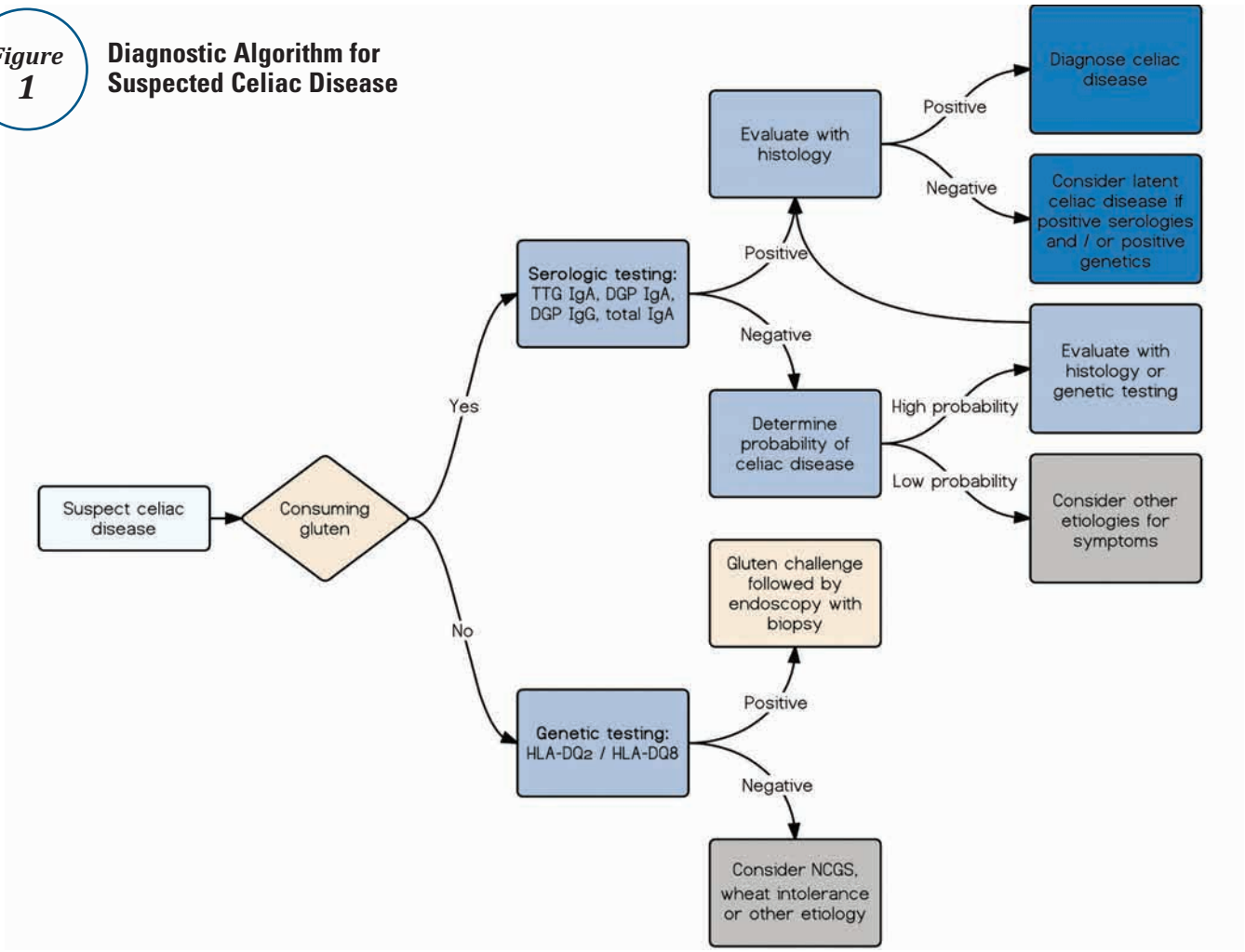
CD should be assessed in individuals with symptoms suggestive of a food-related disorder, such as abdominal pain, bloating, diarrhea, or dyspepsia that occurs in association with food consumption. Even in the absence of gastrointestinal symptoms, CD should be considered in individuals with unexplained iron-deficiency anemia, elevated transaminases, and micronutrient deficiencies as well as early-onset osteopenia/osteoporosis.

Typically, the diagnosis of CD is confirmed using a combination of serological tests and duodenal mucosal histologic findings in the appropriate clinical setting.[3] The initial evaluation should begin with a thorough medical history focusing on the onset of symptoms; detailed description of symptoms; any associated symptoms such as cognitive disturbances; comorbid conditions such as autoimmune disorders, micronutrient deficiencies, anemia, skin rashes, oral lesions, and a detailed family history for CD, diabetes mellitus type I (DM-I), and other autoimmune/rheumatologic disorders.

Initial screening in the symptomatic individual should begin with CD serologic tests while consuming a gluten-containing diet; specifically tissue transglutaminase (TTG) IgA, deamidated gliadin peptide (DGP)

There has been a fivefold increase in the prevalence of CD over the past 50 years, rising from approximately 0.2% in the 1950s to at least 1% today.

Figure 1 **Diagnostic Algorithm for Suspected Celiac Disease**



Non-celiac gluten sensitivity and wheat intolerance refer to disorders in which symptomatic manifestations are precipitated by ingestion of wheat/gluten in individuals without CD or wheat allergy. Latent CD is typically defined as abnormal CD serologies, symptomatology with gluten ingestion, and normal mucosal histology in a genetically susceptible individual.

IgA/IgG, along with measurement of total IgA.[8-10] If the TTG or DGP are elevated, endoscopy with mucosal biopsy should be pursued (see above). Approximately 2%-5% of patients with CD are IgA deficient and may not have the expected increase of TTG IgA or DGP IgA (refer to Table 1).[8]

Screening with both TTG IgA and DGP antibodies may be helpful to increase the overall sensitivity despite the increased cost.[2,3,11] Additionally, approximately 10%-15% of individuals with true CD will be seronegative.[2] Thus, patients with normal serologic tests and a higher probability of CD based on symptoms, associated disorders, family history, and other indications should undergo evaluation by endoscopy with biopsy.

In some instances, such as poor candidates for endoscopy, genetic testing could be the next step in the evaluation of elevated CD serologic markers. In these special circumstances, endoscopy with mucosal biopsy would only be considered if the HLA-DQ2/HLA-DQ8 genes were present. The absence of HLA-DQ2/HLA-DQ8 effectively rules out the possibility of CD.[3] However, the presence of these genes only informs on the individual's genetic risk for potentially developing celiac disease

In some instances, such as poor candidates for endoscopy, genetic testing could be the next step in the evaluation of elevated CD serologic markers.

and not whether they have active disease.

Falsely elevated TTG antibodies can be encountered in patients with other autoimmune disorders such as DM-I, Hashimoto's thyroiditis, psoriatic arthritis, and rheumatoid arthritis, and in the presence of heart failure or liver disease.[12] Therefore, histology is always needed to confirm the diagnosis of CD (refer to Figure 1).

Endoscopy with mucosal biopsy

Endoscopy with at least five to six mucosal biopsies from the duodenum using standard biopsy forceps should be obtained and sent for histologic evaluation; four from the second portion of the duodenum and two from the first portion of the duodenum, all collected into the same jar.[3,13] While endoscopic abnormalities are not present in all individuals with CD, the most common endoscopic findings include duodenal villous atrophy, scalloping and/or notching of the duodenal mucosal folds, and fissuring of the duodenal mucosa.[8] Therefore, biopsies must always be obtained during endoscopy, even if the mucosa appears endoscopically normal.[14]

When CD is suspected, the stomach should always be biopsied to evaluate for concurrent lymphocytic gastritis; at least two biopsies should be obtained from the gastric antrum and two from the gastric body, all collected into the same jar. The esophagus should also be biopsied if there are any symptoms of heartburn, dysphagia, odynophagia, or a history of food impaction given the association of CD with EoE. At least four biopsies should be obtained from the proximal esophagus and four biopsies from the distal esophagus; biopsies from the proximal and distal esophagus should be collected into separate jars.[15]

Histologic findings of celiac disease

Typical findings of CD described on duodenal pathology include an increased presence of intraepithelial lymphocytes and villous atrophy.[16] However, it is important to note that CD is not the only disorder that gives rise to these histologic features and therefore the differential diagnosis must include *Helicobacter pylori* infection, small intestinal bacterial overgrowth (SIBO), or drug-induced injury from olmesartan or valsartan.[17,18] For a more exhaustive list, refer to Table 2.[16]

Table 2

Histologic Mimics of Celiac Disease
Helicobacter pylori infection
Small intestinal bacterial overgrowth
Autoimmune enteropathy
Combined variable immunodeficiency
Drug induced (e.g., olmesartan and valsartan)
Eosinophilic enteritis
Crohn's disease
Tropical sprue
Infectious disorders (e.g., tuberculosis, giardiasis)
Whipple disease
Lymphoma
Malnutrition

Screening high-risk asymptomatic individuals

There are increased rates of CD in certain populations who lack the classic presentation of CD. Thus, screening should be considered in high-risk asymptomatic individuals (refer to Table 3).[3] In these groups, screening should be approached in a different manner than those with symptoms, and the initial screening test should be with HLA-DQ2/HLA-DQ8 genetic testing.[3] In individuals with a negative genetic test, no further screening will ever be required. However, individuals with a positive genetic test should be periodically screened with serological tests.

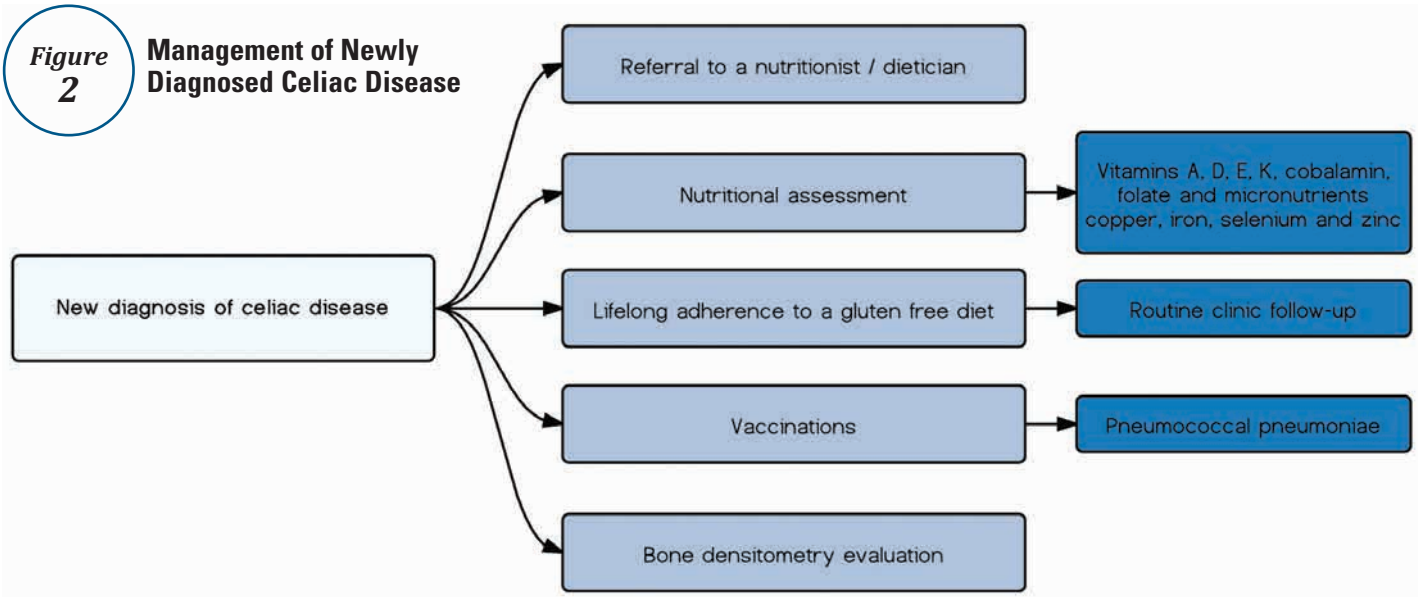
Table 3

Asymptomatic Individuals at Risk of Celiac Disease
First-degree relative with biopsy-proven celiac disease
Diabetes mellitus type I
Syndromes (Down, Turner, William)
Premature osteopenia / osteoporosis
Unexplained iron deficiency anemia
Unexplained elevated transaminases
Unexplained micronutrient deficiencies

A new diagnosis of celiac disease

A patient with newly diagnosed CD should be counseled on the importance of life-long adherence to a gluten-free diet (GFD). It is also important to carefully explain that CD will not be cured by the GFD and that the only treatment currently available is life-long total gluten avoidance. The goal of treatment is to minimize the adverse effects of gluten toxicity and to allow for mucosal healing. Most individuals will achieve complete mucosal healing within 2 years of commencement of a GFD.[3]

All patients with newly diagnosed CD should be referred to a registered dietitian (RD) experienced in the management of CD.[3] Any patient with concerns for inadvertent gluten consumption should also be referred to



an expert RD to carefully review their diet, medications, and lipsticks or other items that can be ingested.

After a period of adherence to a GFD, celiac serological testing should be repeated to verify that they have normalized or are trending towards improvement. These tests should be repeated every three to six months until they have completely normalized (refer to Figure 2).[3] Provided that the serologic studies, other abnormal laboratory studies, and associated symptoms improve with adherence to a gluten-free diet, there is no specific recommendation to repeat endoscopy with biopsy to confirm mucosal healing.

Health care maintenance of the celiac patient

At diagnosis, a complete nutritional assessment should be undertaken as individuals with CD have high rates of micronutrient deficiencies. This assessment should include vitamins A, D, E, K, cobalamin, and folate, along with the micronutrients copper, iron, selenium, and zinc.[3] Any deficiencies should be repleted and repeat testing used to confirm achievement of adequate levels. Since vitamin D is not found in a normal diet, this nutrient should be checked on an annual basis. At diagnosis, and periodically, bone densitometry testing should be undertaken as individuals with CD are at an increased risk for diminished bone mineral density.

The British Society of Gastroenterology suggests that newly diagnosed CD patients also receive pneumococcal and influenza vaccination.[19] Individuals with CD have a slight increased lifetime risk of developing small intestinal lymphoma of T-cell origin and small intestinal adenocarcinoma. There are no guidelines for screening practices for small intestinal malignancy in individuals with well-controlled CD since the risk is low. However, individuals with refractory celiac disease (RCD) have a much higher risk for transformation to a small intestinal T-cell lymphoma and should be managed at a center with expertise in CD. The complex management of these patients goes beyond the scope of this brief review.

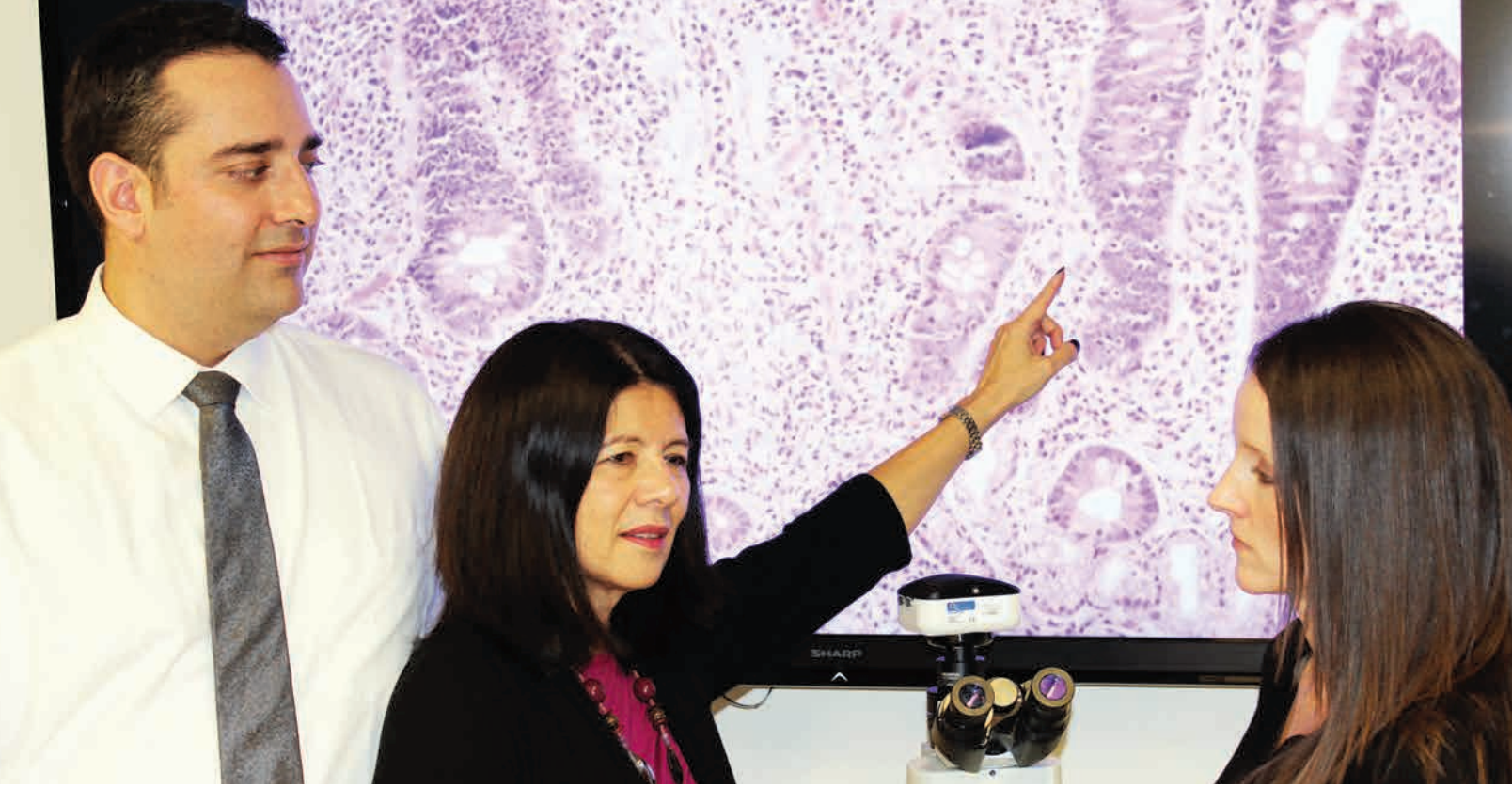
Individuals with CD have a slight increased lifetime risk of developing small intestinal lymphoma of T-cell origin and small intestinal adenocarcinoma.

Screening for celiac disease in first-degree relatives

Screening of all first-degree relatives of individuals with CD should be considered regardless of symptoms because of the high rates of disease within families.[3] Refer to the section on "Screening High-Risk Asymptomatic Individuals" for more details.

Approach to the celiac disease patient with recurrent symptoms

For most individuals with CD, adherence to a GFD results in rapid



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Courtesy Dr. Peter B. Ernst

improvement in their gastrointestinal complaints, along with improvement of their extra-intestinal symptoms such as fatigue, cognitive impairment, and joint pain. An elevation of serologic markers associated with a period of recurrent symptoms strongly suggests gluten exposure. Recurrent symptoms after initial resolution of symptoms mandate a careful evaluation of an individual's diet and medications in an attempt to identify inadvertent gluten consumption.

If gluten exposure is ruled out and there is the presence of new-onset diarrhea as the predominant symptom, the co-occurrence of microscopic colitis should be considered. Inflammatory bowel disease (IBD) should also be considered in the appropriate clinical setting, as both CD and IBD can rarely occur in the same individual.

There are many gluten-free processed foods available on the market and while this has eased the dietary limitations of individuals with CD, it also has contributed to other problems of excess carbohydrate consumption and inadvertent gluten exposure. Gluten-free processed foods sometimes contain an abundance of carbohydrates and/or small amounts of gluten that can lead to mucosal injury. Consumption of excess carbohydrates can result in gastrointestinal symptoms of abdominal pain, bloating, and diarrhea that can be confused with recurrent symptoms associated with CD. Individuals with symptoms related to excess carbohydrate consumption from gluten-free processed foods should instead focus on consuming more fresh vegetables, fruits, nuts, and lean proteins.

SIBO should also be considered in individuals reporting abdominal pain, bloating, or diarrhea after food consumption. SIBO may occur at higher rates in individuals with CD due to altered dietary patterns such as excess carbohydrate consumption, changes in intestinal motility, or changes in the small intestinal microbiota (refer to Figure 3).

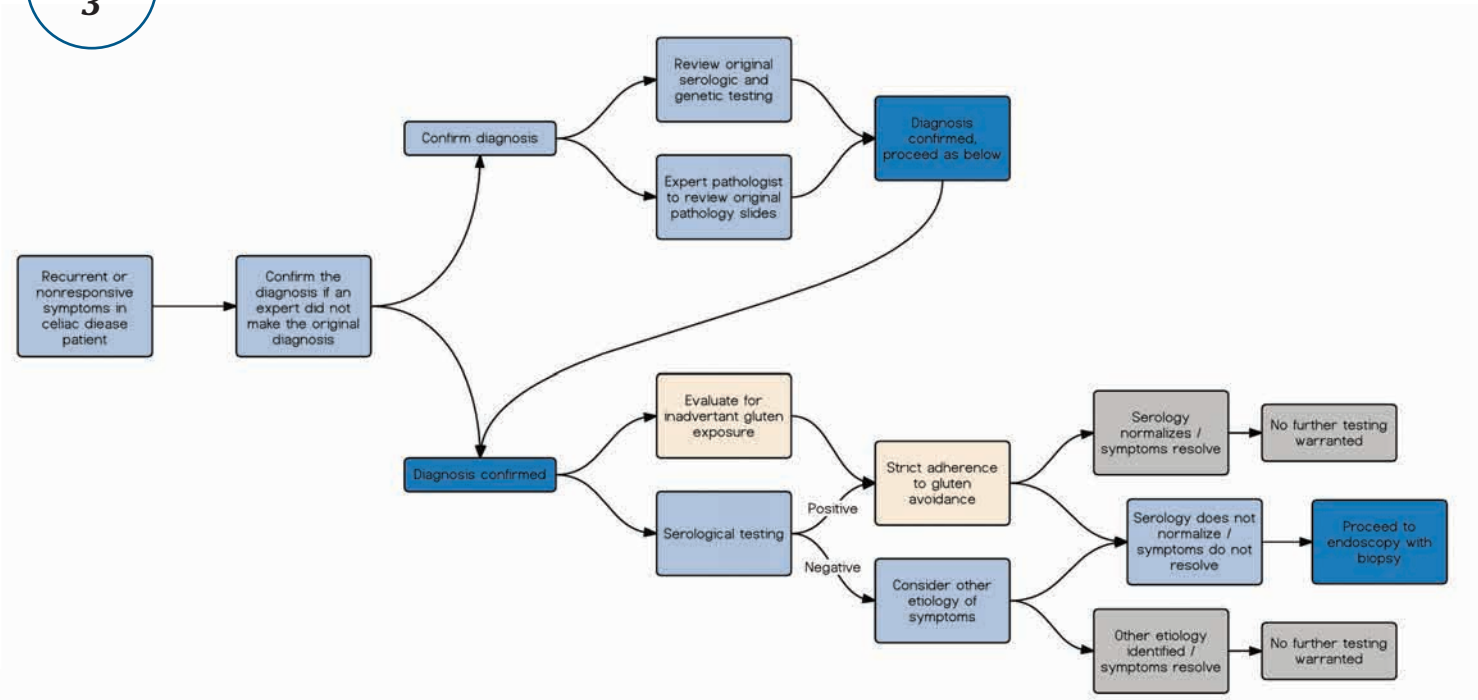
What is refractory celiac disease?

RCD is a rare complication of CD and can occur anytime during the course of disease. RCD is defined as the persistence or recurrence of malabsorption and duodenal mucosal

TAKE-AWAY POINTS

- Celiac disease can present at any age
- Celiac disease can occur in any race
- Celiac disease can present in the absence of gastrointestinal symptoms
- Celiac disease can occur in patients of any weight
- Celiac disease can occur in the presence of constipation
- Screen all first-degree family members for celiac disease if willing and will derive benefit if treated
- Refractory celiac disease should be managed at an expert center

Figure 3 **Evaluation of Non-Responsive or Recurrent Symptoms in Celiac Disease**



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Individuals with symptoms related to excess carbohydrate consumption from gluten-free processed foods should instead focus on consuming more fresh vegetables, fruits, nuts, and lean proteins.

injury after prolonged strict gluten avoidance in the absence of other causes.[20] RCD should be considered in the persistently symptomatic patient, even when serologies remain normal as inadvertent gluten exposure is typically not driving this process.[20] There are two forms of RCD: type I (RCD-I) which has a normal intra-epithelial lymphocyte phenotype and better overall prognosis and type II (RCD-II) which has an abnormal intra-epithelial lymphocyte phenotype and a poor prognosis. Five-year survival rates are estimated at 50% for RCD-II. Cases of RCD should be managed at expert centers and further discussion of this topic is beyond the scope of this brief review.

New advances on the horizon

There are several novel treatments in development for the management of CD beyond strict gluten avoidance.[21-23] Most strategies will never

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replace strict gluten avoidance for the long-term management of CD. However, endopeptidases provide a method to possibly manage small amounts of gluten exposure that may occur in such situations in which the individual has less control over the gluten content in their food, such as when consuming food prepared outside the home or when traveling.

Conclusions

CD is an immune-mediated small intestinal enteropathy that occurs in a small fraction of genetically susceptible individuals upon exposure to dietary gluten. It is a heterogeneous disorder both in presentation and phenotype and is associated with several other autoimmune and allergic disorders – all of which complicate the diagnosis. Individuals with CD are at an increased risk for vitamin and micronutrient deficiencies.

The mainstay of treatment is lifelong dietary gluten avoidance and repletion of vitamin and micronutrient deficiencies. RCD is a rare complication of CD and should be managed at expert centers. ■

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2015: A Year in Review in Esophagus, Inflammatory Bowel Disease, and Motility

By Hassan Siddiki M.D., M.S.; Marcelo F. Vela, M.D., MSCR;
Amy Foxx-Orenstein, D.O., FACG, FACP; and Jonathan A. Leighton, M.D.

Dr. Siddiki is a clinical fellow in the department of gastroenterology and hepatology, Mayo Clinic; Dr. Vela is the director of esophageal disorders in the division of gastroenterology and hepatology, Mayo Clinic; Dr. Foxx-Orenstein is professor of medicine, co-director of motility, director of Mayo Clinic Weight And Wellness Solutions, department of gastroenterology and hepatology, Mayo Clinic; and Dr. Leighton is professor of medicine, division of gastroenterology and hepatology, Mayo Clinic, Scottsdale, Ariz.

In this overview, we summarize the key original research published in 2015 on esophagus, inflammatory bowel disease, and motility. This research has influenced clinical practice and indicates the future of gastroenterology.

Esophagus

In 2015, endoscopic anti-reflux therapy was shown to be a feasible alternative treatment for a subset of patients with proton-pump inhibitor (PPI) refractory gastroesophageal reflux disease (GERD) symptoms. The RESPECT trial compared transoral fundoplication with a sham procedure plus PPI (n = 87 vs. 42); transoral fundoplication showed improvement in regurgitation on 6-month follow-up.[1] In addition, transoral fundoplication was also associated with a reduction in esophageal acid exposure as measured by pH-metry. The authors attributed the success of the trial to optimization of technique by deploying more fasteners compared to the prior negative trials. Outcomes from the National Registry data created for this new device will help establish if the trial findings are reproducible. Another exciting development in the area of GERD diagnostics is the use of impedance measured with a through-the-scope catheter that comes in direct contact with mucosa; this technique was shown in a study to reliably diagnose GERD – in the absence of erosive esophagitis – from non-GERD (3,252 Ω vs. 4,806 Ω, *P* less than or equal to .001), suggesting the greater role that endoscopy may have in the future management of this disorder.[2]

A study published in PLoS One created headlines when it reported a "signal" linking PPIs with elevated risk of myocardial infarction.[3] A computer algorithm for data mining was employed but there was no control for biases originating from covariates

Transoral fundoplication was also associated with a reduction in esophageal acid exposure as measured by pH-metry.

that are known to modulate cardiovascular risk, like obesity. Given these methodological weaknesses, PPIs should not be discontinued for fear of cardiovascular complications in patients with a legitimate indication for acid suppression. However, infection risk from PPI use remains a valid risk. In a study of 188 cirrhotic patients hospitalized for an index infection, it was reported that PPI use was an independent predictor (odds ratio, 2.94) of development of a different infection within 6 months of follow-up.[4] A small recent trial (n = 6) may provide biological plausibility of infection risk with PPI therapy by showing that – even in healthy subjects – PPI use increases Enterococcaceae and Streptococcaceae taxa that are associated with infections.[5] In summary, these studies show that we should critically evaluate use of PPIs and discontinue them if there is no indication for their continued use.

In 2015, we also arrived at a better understanding of the behavior of dysplasia in patients with Barrett's esophagus. A large study of more than 4,900 patients showed that increasing Barrett's length and a higher baseline pathologic grade were associated with an increased risk of esophageal adenocarcinoma (EAC).[6] In another study of 125 patients with low-grade dysplasia (LGD), radiofrequency ablation (RFA) reduced progression to high-grade dysplasia (HGD)/EAC from 26% to 1.5%, with a NNT = 4.[7] Multivariate analysis from this elegant study showed that progression of LGD was linked to nodularity and multifocality – providing insight as to which LGD cases should be targeted for RFA. In our practice, we are performing RFA on all patients with HGD and patients with LGD (after confirmation by two GI pathologists) that is persistent on endoscopic follow-up, especially when there are additional risk factors

Table 1

Author, Journal	Therapy, Target Disease	Mechanism of Action, Study Design, Size	Results
Vermeire et al., Lancet[9]	Etrolizumab for moderate to severe ulcerative colitis (UC)	Humanized monoclonal B7 integrin antibody antagonizing egress of lymphocytes from mucosal vasculature. Double-blinded placebo controlled phase II RCT, n = 124.	Clinical remission in 21% (8/39) at week 10 ($P < .05$) for 100 mg. More effective in anti-TNF-naive subjects, so further trials may enhance remission rates.
Lang et al., CGH[10]	Add-on curcumin for mesalamine resistant mild UC	Inhibits TNF and nuclear factor-kappaB secretion, activates STAT-3 and p38 mitogen-activated protein kinase and TH1 cytokines. Double-blinded placebo controlled phase I trial, n = 50.	53.8% achieved clinical remission at week 4 ($P = .01$). Clinical response 65.3% vs. 12.5% and endoscopic remission 38% vs. 0% receiving placebo ($P = .04$).
Sandborn et al., Gastro[11]	Budesonide foam enema for ulcerative proctitis or proctosigmoiditis	Anti-inflammatory effect from high potency second generation corticosteroids. Two randomized double-blinded placebo controlled phase III studies, n = 546.	Remission (endoscopic or clinical) achieved at week 6 in 38.3% vs. 25.8% in proctitis and 44% vs. 22.4% in proctosigmoiditis.
Monteleone et al., NEJM[12]	Mongersen for moderate to severe CD	Oral oligonucleotide that hybridizes to human SMAD 7 messenger RNA to increase immunosuppressive TGFB1. Multicenter randomized double-blinded placebo controlled phase II study, n = 166.	Clinical remission (CDAI score) at day 28 was 65% in the 160-mg group vs. 12% in the 10-mg group ($P < .001$).
Molendijk et al., Gastro[13]	Allogenic mesenchymal stromal cells for perianal fistulizing CD	Local administration of multipotent cells able to down-regulate mature dendritic cells and promote tissue healing. Randomized double-blinded phase II placebo controlled dose escalating clinical trial, n = 21.	Healing observed in (66.7%; 85.7%; and 28.6% in groups 1; 2; and 3 vs. 33.3% in placebo $P = .06$).
Fedorak et al., CGH[14]	VSL#3 for preventing recurrence of post-op CD	High number of viable bacteria (900 billion/sachet) and a mixture of 8 different bacteria generating a probiotic effect. Randomized placebo-controlled double-blinded multicenter study, n = 120.	Reduced inflammatory cytokine levels at day 90 ($P < .05$). Endoscopic recurrence was not different than placebo group. Trend for protective effect if used in early post-op toward endoscopic recurrence ($P = .09$).
Yoshimura et al., Gastro[15]	AJM300 for induction therapy for moderately active UC	Oral alpha4 integrin antagonist in patients with IBD. Double-blind placebo-controlled phase 11a study, n = 102.	Clinical response rates were 62.7% at week 8 in the AJM300 group as compared to 25% with placebo (OR = 5.35; $P = .0002$).

A large population-based study supported that biologics alone do not increase overall risk of malignancy and that malignancy risk for combination therapy appears to be related to thiopurine exposure.

like a smoking history. In the newly published clinical guidelines for Barrett's, ablative therapy or 1-year surveillance have been suggested to be equally acceptable options for patients with LGD.[8]

Inflammatory bowel disease

Exciting new inflammatory bowel disease (IBD) therapies came on the horizon in 2015, with some that are administered orally, which could prove to be game changing (Table 1). For existing therapies, focus remained on employing drug-level monitoring to prevent loss of response, which occurs in half of patients started on biologic agents. Post hoc analyses of the SONIC and TAXIT trials show that monotherapy with anti–tumor necrosis factor (TNF) drugs can be highly successful if mucosal inflammation can be neutralized quickly; as evidenced by adequate drug levels both at the end of induction and during the maintenance phase. Infliximab target trough level of greater than 3.0 microg/mL at week 30 predicts both long-term steroid-free remission and mucosal healing (odds ratio, 3.34).[16] In the maintenance phase, levels between 3 and 7 microg/mL are considered optimal, with dose intensification if levels are low.[17]

Several studies highlighted known malignancy concerns with thiopurines (TPs) and reassuring data on the safety of biologics. A large population-based study supported that biologics alone do not increase overall risk of malignancy and that malignancy risk for combination therapy appears to be related to thiopurine exposure.[18,19] A meta-analysis of 18 studies reports that there is a sixfold increased risk of lymphoma with exposure to TPs especially in men under 30 or patients more than or equal to 50, with a number needed to harm of 1/377.20 The good news is that the risk is high only after more than 1 year of exposure and goes back to baseline if discontinued. So how can we stop TPs for patients on combination therapy? Safe de-escalation was shown to be a possibility for at least a subset of patients who objectively demonstrate well-controlled disease and have adequate anti-TNF trough levels, greater than 5 microg/mL for infliximab.[21,22]

In an effort to alter the natural history of IBD, treatment targets continued to evolve in 2015, moving away from symptom-based scores like Crohn's Disease Activity Index to more objective therapeutic targets of intestinal inflammation.[23,24] Fecal calprotectin (FC), a calcium-binding protein, may offer a noninvasive and cost-effective target. Two prospective studies of 135 and 86 postoperative Crohn's disease patients show that serial FC can indicate early recurrence and hence, for low-risk patients, it is ideal for surveillance of recurrence.[25,26] FC is also useful to make management decisions in symptomatic IBD patients (sensitivity, 0.88) if used in the context of pretest probability of endoscopic inflammation.[27] For instance, in a patient with high pretest probability of endoscopic disease, an elevated FC can give the gastroenterologist further confidence for escalating therapy. Other clinical scenarios in which FC could be employed include predicting pouchitis for patients who have undergone restorative proctocolectomy even before they become symptomatic and differentiating IBD from irritable bowel syndrome (IBS).[28] A meta-analysis of 12 studies showed that an FC cutoff of 40 effectively rules out IBD.[29]

Table 2

Factors Associated with Symptom Improvement	Factors Associated with Lack of Symptom Improvement
Male sex	Obesity
More than 50 years of age	Use of pain modulators
Life-long nonsmokers	Abdominal pain (moderate to severe)
Initial infectious prodrome	Severe GERD
Antidepressant use	Moderate to severe depression
Four-hour gastric retention more than 20%	

Motility and functional disorders

The concept of IBS being a brain-gut-axis disorder further solidified in 2015, confirming that psychological distress and IBS can synergistically make symptoms more severe. In an elegant study on the effect of somatization on postprandial symptoms, anxiety and depression were both associated with bloating, fullness, and higher levels of abdominal pain. Furthermore, the severity of depression increased the rate of symptom onset in the postprandial phase, indicating that meal ingestion and depression have a potentiating effect on quicker worsening of symptoms.[30]

A double-blinded randomized controlled trial scientifically supported the common practice of using tricyclic antidepressants (TCA) for functional dyspepsia (FD) and showed that TCAs provide twofold relief in symptoms as compared to therapy with selective serotonin reuptake inhibitors (SSRIs) and that SSRIs are no better than placebo.[31] More importantly, it was recognized that FD is a heterogeneous disorder that can present as either an ulcer/pain-like phenotype or as postprandial distress. TCAs work only for patients who have pain symptoms, which is consistent with the established role of TCA-analgesia for several other chronic pain syndromes. TCAs are ineffective for the postprandial distress variant of FD. Along the same lines, FD with evidence of delayed gastric emptying (GE) does not respond to TCAs.

Investigators continue to gather more evidence that the GE test is highly variable so care should be taken before labeling a patient gastroparetic on GE testing alone. A prospective 27-year- long study on diabetic gastroparesis showed that long-term hyperglycemia is a risk for development or worsening of delayed GE.[32] Yet while 48% of study subjects had GE abnormalities, the majority were asymptomatic; and when symptoms did occur they were mostly mild. The same authors reported that two-thirds of diabetic patients have an asymptomatic abnormal GE study but improving hemoglobin A_{1C} did not improve GE at 6 months.[33] These results challenge the concept of treating GE abnormalities in isolation or that better glycemic control improves gastric function. For symptomatic gastroparetic patients, though the clinical outcomes are disappointing (less than 30% improvement), we now know the factors that portend worse outcomes (Table 2).[34] On the horizon is relamorelin, a ghrelin receptor agonist, which, in a randomized controlled trial published in 2015, showed that this new class of prokinetic agent enhanced GE as compared with placebo.[35] It remains to be seen if patients will have significant symptom relief along with improving GE in further larger studies.

With a critical analysis of the studies summarized above, we can improve our understanding of some of the most common gastrointestinal diseases as well as deliver the best care to our patients. For those newly introduced diagnostic or treatment possibilities, larger prospective studies published in coming year will help take them off the bench and into clinical practice. ■

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Tips For Delivering an Effective Medical Lecture

By Peter Buch, M.D., AGAF, FACP

Dr. Buch is an associate clinical professor in the department of gastroenterology and hepatology at the University of Connecticut School of Medicine, Farmington, Conn.

Teaching is my passion. Throughout my 34 years as a gastroenterology educator, I've had the privilege of teaching medical students, house staff, GI fellows, physician assistants (PAs), advanced-practice registered nurses (APRNs), and practicing clinicians. As a young GI, you will likely be involved with educating others by offering consults, teaching trainees, and being invited to give lectures. While it is important to not let the consults go by without a teachable moment, more formal lectures are truly unique and rewarding experiences. Use them diligently, especially early in your career when you have more free time. Each speaking opportunity offers the chance to improve patient care, educate your staff and provide for better teamwork, and ultimately highlight your talents and willingness to take time out of your busy schedule to help others. Be sure to get out there and shine – you will generally have more time to lecture now than at any other point in your career. Below are some lessons I've learned in giving effective and meaningful lectures.

Keep the message simple and practical

During each lecture, I always strive to focus on a few important "take-home" points. I never present over-complicated slides; four points per slide is fine. Furthermore, as a clinician, I have encountered patients whose diagnosis remains elusive. I use these cases as practical examples by working through differential diagnoses with the audience. In addition to giving them insight into my thought process, it opens up the presentation to discussion and occasional debate. This level of interactivity promotes audience engagement and allows me to modify my slide deck for future use.

Know your audience

Each audience has its own personality. This will be reflected in their level of participation and sense of enthusiasm. Seemingly trivial factors, such as the room's ambient temperature and the timing of the lecture (especially if scheduled after lunch) can have a tremendous influence on success.

As educators, we need to learn as much as possible about our audience in advance. Oftentimes, before the lecture, I introduce myself to several attendees and ask them what they're looking to learn. This individual contact can often lead to very meaningful discussions during my presentation. Also, if appropriate, I begin my lecture with some humor before shifting to a higher energy level. As Robin Williams proved in "Good Morning, Vietnam," humor can be effective in grabbing and maintaining attention. In the end, tone can make or break the

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learning environment; learn to use it to your advantage.

Additionally, you must be able to adjust your presentation on the fly. When you see that the audience is drifting, begin to course-correct immediately. I would note that body language is an important indicator in this regard. The above-mentioned incorporation of humor, small stretch breaks, or questions – particularly multiple choice queries via acclamation or an audience response app – are all wonderful ways of making sure your teaching points are being understood.

Know your topic and include your own experiences

In the age of smartphones and Google, being aware of the breaking literature is crucial. I can assure you that someone in the audience will use their device to fact-check! However, don't have unrealistic expectations of yourself; you shouldn't be afraid to say "I don't know." Moreover, be sure to address both your successes and failures. Often, I detail my clinical mistakes to the audience. This acknowledgement of fallibility makes me more approachable and promotes wider discussion.

In the age of smartphones and Google, being aware of the breaking literature is crucial. I can assure you that someone in the audience will use their device to fact-check!

Be receptive to feedback

Incorporating audience feedback into my talks has made me a better lecturer. This is why I love live discussions. Be aware of audience reception and take note of any potential feedback. When there are questions, follow-up to make sure the topic has been truly understood. Learn to enjoy positive feedback and acknowledge the validity of criticism (none of which may be related to you as a speaker). Should there be negative feedback, think about the topic in relation to the audience. It might beneficial to rework your approach or consider alternative modalities – like rounds, small group discussions, or simulation – for future lectures. When questions that I cannot answer arise, I always take audience members' contact information, do the necessary follow-up research, and get back to them.

Conclusion

Use every possible lecturing opportunity to not only improve GI care in your institution/practice and the community, but to also promote your own special talent. Utilize your down time. Reach out to surgeons, floor nurses, PAs, APRNs, and whoever you can think of to collaborate and develop lectures that will benefit everyone. Enjoy what you are doing! This is the fuel you need to succeed. ■

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Snapshots from the AGA Journals

GI and Liver Diseases Remain Public Health Burden

December Gastroenterology (doi: 10.1053/j.gastro.2015.08.045)

Key clinical point: Gastrointestinal and liver diseases remain a major cause of health care utilization and associated costs in the United States.

Major finding: Hospital admissions and associated costs for *Clostridium difficile* infection, inflammatory bowel disease, and liver disease all rose substantially between 1993 and 2012.

Data source: Analysis of surveillance data from the Centers for Disease Control and Prevention, Agency for Healthcare Research and Quality, and National Cancer Institute.

Disclosures: The National Institutes of Health helped fund the work. The investigators reported having no conflicts of interest.

Commentary

Dr. Philip S. Schoenfeld is a professor of medicine and director, training program in GI epidemiology, division of gastroenterology at the University of Michigan, Ann Arbor. He has no conflicts of interest.

In the excellent study by Peery and colleagues, statistics on health care utilization in the ambulatory and hospital settings, incidence and mortality from GI cancers, and mortality associated with other GI illnesses from 2007 to 2012 were collected using data from multiple complementary databases. This is the ideal methodology for this type of study because it quantifies utilization data from several complementary national databases. Of course, these data may be limited by systematic errors in ICD coding and costs are estimated using Medicare's cost-to-charge ratio. Nevertheless, these data provide the best "snap shot" of trends in the burden of gastrointestinal and liver illness as of 2012.

What are the key points? First, the increase in the burden of GI and liver illness probably reflects the aging of the "baby boomer" population. Furthermore, since the Affordable Care Act is expanding access to health care, the burden on gastroenterologists is also likely to expand. Second, although we're doing a good job with CRC screening, there is also room for improvement. While the incidence of CRC continues to decrease, only 58% of adults aged 50-75 years had CRC screening in 2010. Third, HCV-associated hospitalizations have doubled from 2003 to 2012. Since HCV-associated cirrhosis is likely to increase until 2030, insurers and public health officials will have to carefully weigh the initial high cost of using new and highly effective regimens of direct-acting antiviral agents versus the downstream costs of managing these individuals after developing decompensated cirrhosis. ■

Nerve Cell Transplants Studied in Hirschsprung Disease

January Cellular and Molecular Gastroenterology and Hepatology (doi: 10.1016/j.jcmgh.2015.09.007)

Key clinical point: A preclinical study took several key steps toward autologous transplantation of nerve progenitor cells to treat Hirschsprung disease.

Major finding: Neural progenitor cells from the proximal colons of patients with Hirschsprung disease divided and formed neurons and glia in their own distal aganglionic colon tissue.

Data source: Flow cytometry, reverse transcriptase-PCR, immunohistochemistry, and cell culture of proximal (neuronal) and distal (aneuronal) colon tissue from 31 affected patients, and co-culture with avian embryonic gut and mouse enteric nervous system cells.

Disclosures: The National Health and Medical Research Council, Murdoch Children's Research Institute, Graham Burke and Yvonne Spencer, the Federation of Chinese Associations, Fonds du Service de chirurgie pédiatrique et de Perfectionnement du CHUV, Fondation SICPA, and Société Académique Vaudoise funded the study. The researchers disclosed no conflicts of interest.

Commentary

Marion France, Ph.D., is a postdoctoral research fellow at Brigham and Women's Hospital and Harvard Medical School, Boston. She has no conflicts of interest.

Hirschsprung disease results from a failure of complete neural crest cell migration into the distal colon. Current therapy relies on surgical resection of the aganglionic distal colon. However, for many children, particularly those with large aganglionic segments, surgery often fails to completely normalize function.

One therapeutic approach might be to transplant new enteric neural cells into the aganglionic colon. This has been partially accomplished in rodent and avian models. However, it is not known if human smooth muscle can be colonized or if human postnatal enteric nervous system cells are capable of migration, expansion, and differentiation.

Rollo et al. show that human postnatal enteric nervous system cells isolated from the proximal, i.e., ganglionic, margin of Hirschsprung disease resection specimens can migrate and spread to colonize aganglionic smooth muscle from the distal margin of the same specimen. Remarkably, the transplanted cells differentiated into neurons and glia and formed normal-appearing neural structures.

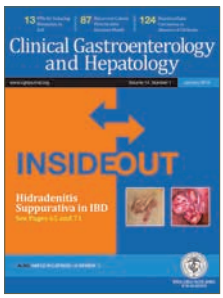
Many questions remain. If neural cells isolated from the proximal margin can migrate ex vivo, why didn't they migrate during in utero development? Do the transplanted cells restore normal motility, which requires a complex series of events that must be precisely orchestrated?

Nevertheless, the demonstration that human postnatal neural cells can be isolated from surgical specimens and used to colonize aganglionic smooth muscle suggests that it may be possible to conserve and restore motility and, potentially, to successfully treat Hirschprung disease patients without the need for surgery. ■

PPIs Caused Remission in Half of Esophageal Eosinophilia Cases

January Clinical Gastroenterology and Hepatology (doi:10.1016/j.cgh.2015.07.041)

Key clinical point: Proton pump inhibitors should be considered in the first-line treatment of esophageal eosinophilia.
Major finding: Half of patients achieved clinical and histologic remission after a trial of PPIs.
Data source: Meta-analysis of 33 studies of 619 patients with symptomatic esophageal eosinophilia indicative of eosinophilic esophagitis.
Disclosures: The authors reported no funding sources and had no disclosures.



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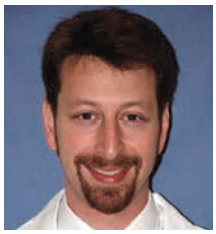

Dr. Evan S. Dellon, MPH, is an associate professor of medicine and epidemiology at the Center for Esophageal Diseases and Swallowing, division of gastroenterology and hepatology, University of North Carolina School of Medicine at Chapel Hill. He has received research funding from Meritage, Miraca, Receptos, and Regeneron and consulted for Aptalis, Banner, Novartis, Receptos, Regeneron, and Roche.

Proton pump inhibitor–responsive esophageal eosinophilia (PPI-REE) is a condition in which patients have symptoms of esophageal dysfunction (often dysphagia or heartburn), biopsies with at least 15 eosinophils per high-power field (eos/hpf), and symptomatic and histologic resolution after a PPI trial. Currently, PPI-REE and eosinophilic esophagitis (EoE) overlap substantially, but in the most recent guidelines, they are still considered to be distinct entities. The study by Dr. Lucendo and colleagues, a comprehensive and rigorously conducted review and meta-analysis of 33 studies accounting for 619 patients, found that just over 50% of patients with esophageal eosinophilia had histologic remission (less than 15 eos/hpf) and just over 60% had symptomatic improvement after PPI use. Moreover, similar responses were seen whether or not there was pathologic acid exposure on pH testing.

There are several important messages from this study. First, PPI-REE is commonly seen in patients with esophageal eosinophilia, and is not always simply due to reflux. Second, PPIs have a potent antieosinophil effect in these patients. Novel acid-independent mechanisms for this anti-inflammatory action recently have been described in other studies. Third, a PPI trial remains important before confirming the diagnosis of EoE. PPIs should be considered the first-line treatment when esophageal eosinophilia is identified. However, note that all esophageal eosinophilia is not due to EoE. If a patient responds to the PPI trial, there is no clear need to move toward topical steroid or dietary elimination therapy specifically for EoE, and starting multiple antieosinophil treatments concomitantly precludes determining which is most effective. In the future, understanding which patients with esophageal eosinophilia will most benefit from a PPI trial will be important, as we are currently unable to predict this from clinical, endoscopic, and histologic factors. Future studies and guidelines will also need to address whether EoE and PPI-REE are distinct diseases or manifestations of the same underlying process. ■

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ANSWERS

// From page 3

Q1: ANSWER: C

CRITIQUE
This patient has hereditary hemochromatosis. Risk factors for advanced fibrosis include ferritin > 1,000 microg or elevated liver enzymes. A serum ferritin level > 1,000 microg/L with an elevated aminotransferase level and a platelet count < 200 × 10^3 micro/L predicted the presence of cirrhosis in 80% of C282Y homozygotes. With a ferritin of 1,393 microg/L, elevated transaminases, and relatively low platelets, a liver biopsy would be the most appropriate next step. Phlebotomy may also be performed but does not provide clinically important and prognostic information such as the presence of cirrhosis and its implications for variceal bleeding and HCC screening. MRI for iron quantification is not required for the diagnosis of HH in light of positive gene testing and evidence of serologic iron overload and does not provide information regarding the degree of fibrosis in this case. Although obesity may be associated with elevated iron stores, this degree of ferritin elevation would be unusual. In the long term, weight loss should be encouraged, however, obesity is not the immediate cause of potentially significant liver disease. This patient is a healthy inactive carrier of hepatitis B and treatment is not indicated.

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Q2: ANSWER: E

CRITIQUE
Choices A through D of the presented answers are correct. Resection of 50% of the small intestine is usually well tolerated, whereas resection of more than 75% of the small bowel usually requires parenteral nutrition. The ileocecal region provides a brake that slows intestinal transit, allowing for absorption of nutrients. The terminal ileum is required for absorption of vitamin B_{12} and bile salts. The remaining intestine, namely the terminal ileum, can adapt by increasing the height and diameter of the villi to produce a greater surface area for absorption. Finally, the colon absorbs nutrients that are not absorbed in the small intestine as well as fluids and electrolytes. Taken together, presence of a terminal ileum, ileocecal valve, and colon in addition to length of remaining small bowel are all positive prognostic indicators of enteral nutrition tolerance. Adaptation and fluid balance is more successful with residual ileum, in contrast to patients who only have residual jejunum.

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The Answer

From ***What's Your Diagnosis?*** on page 6

This patient was diagnosed with diffuse cavernous hemangioma of the rectosigmoid colon. A flexible sigmoidoscopy was done and showed nodularity and blue-purple discoloration from the anal verge to a distance of 20 cm, suggestive of large submucosal vascular malformations. No active bleeding was seen. Pelvic magnetic resonance imaging (MRI)/magnetic resonance venogram was done and showed irregular circumferential wall thickening in the rectum and sigmoid colon with phleboliths consistent with diffuse cavernous hemangioma. Given the patient's age, a sphincter-sparing operation was not pursued and an abdominoperineal resection was performed to resect the involved rectosigmoid colon. The patient expired 2 days after discharge from the hospital from apparent septic shock.

Diffuse cavernous hemangioma of the colon is a rare, nonmalignant, vascular malformation frequently misdiagnosed as hemorrhoids or ulcerative colitis. The most common site affected is the rectosigmoid colon.[1] It is a congenital abnormality caused by embryonic sequestration of mesodermal tissue. There have been roughly 350 cases reported in the literature, with the vast majority in patients under the age of 30.[1] There is only one other case report of a patient presenting in the ninth decade of life.[2] The most common presenting symptom is recurrent painless rectal bleeding. Characteristic findings on endoscopy are a confluence of serpiginous purple areas in the rectum with some nodularity resulting from the dilated submucosal vascular structures. Biopsy is best avoided owing to the risk of bleeding, and in this case biopsies and treatment of the bleeding lesions with a bipolar probe may have contributed to the patient's ongoing bleeding. On CT scan, diffuse concentric rectal thickening with clusters of calcified phleboliths are pathognomonic findings. In the absence of phleboliths on CT, MRI can be diagnostic.[3] Moreover, MRI is much more accurate in defining the extent of involvement of the anal sphincter and adjacent structures, which can be crucial in planning surgery.[1] Definitive treatment requires surgical resection with a sphincter-sparing operation when possible based on patient factors including anal sphincter functional capacity and lack of involvement of the anal canal by hemangioma. Angioembolization has been used as well, but with limited and usually only temporary improvement in bleeding.[3] ■

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