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BCMJ
A Doctors of BC Publication

Gastroesophageal reflux disease and functional dyspepsia:

Two articles review diagnosis and management in a primary care setting

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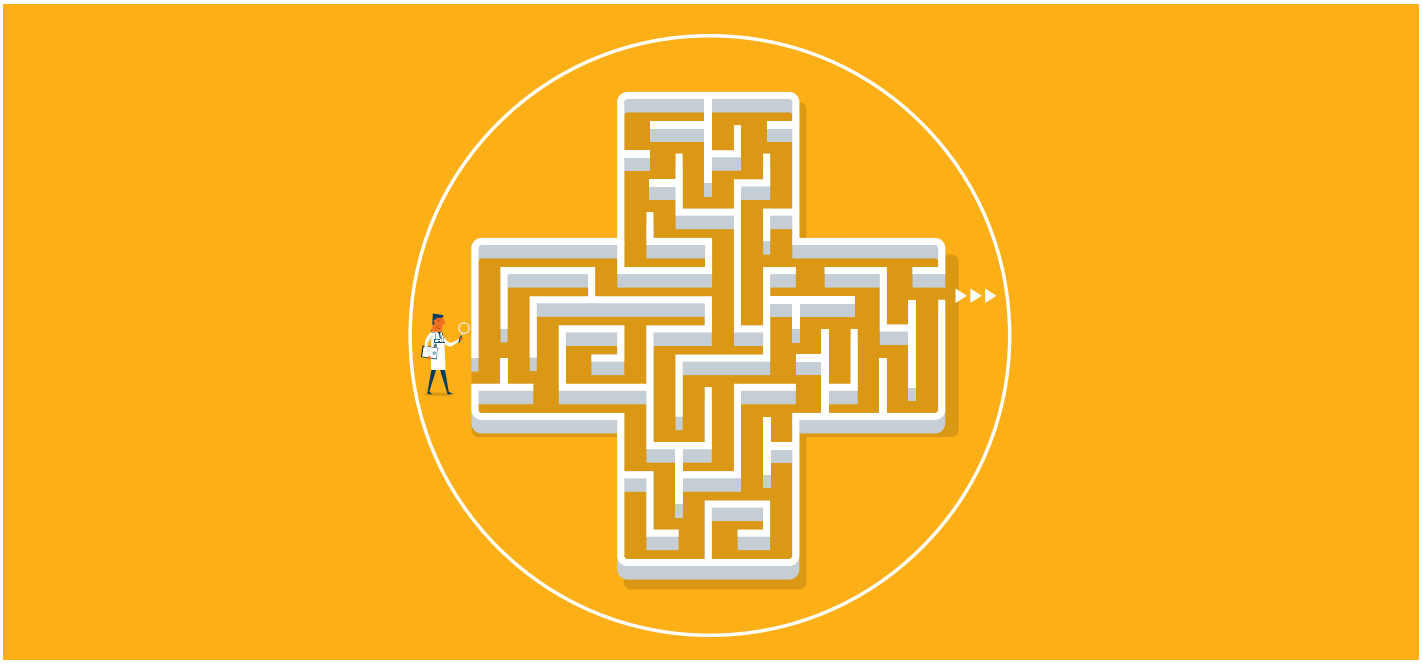

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IV hydration spas: Health hack or pricey pee?

IV hydration spas are everywhere. I first encountered one in Las Vegas, which seemed fitting for the city—and the circumstances patrons may have endured the night prior. However, what once felt like a novelty now appears embedded in the wellness landscape. A recent *JAMA* article titled "IV hydration spas are gaining popularity, but are they safe?"[1] crystallized a growing concern: IV treatments have quietly migrated from medical settings to nearly every street corner, marketed as routine self-care. For a few hundred dollars, IV therapies claim to rehydrate, boost immunity, improve nutrient absorption, enhance energy, flush toxins, and speed athletic recovery. Boutique spas offering vitamin IV infusions appear to have emerged around 2008,[2] gaining momentum in 2012 when celebrity endorsements—most notably a photo of Rihanna receiving a so-called "party-girl drip"—helped make the trend mainstream.[3] Today, IV drips are marketed as offering customized, high-dose vitamin therapy tailored to "individual needs."

Are IV hydration spas safe? Who is ensuring they are?

These businesses appear to operate in a grey zone, under a patchwork of regulatory bodies. For example, medical health professionals such as physicians, nurses, and naturopaths have provincial colleges with standards of care. Drugs and IV products must be authorized by Health Canada. Yet many medical spas function in practice like compounding pharmacies, often without the oversight, quality controls, or reporting requirements expected in traditional health care settings. This raises legitimate concerns about dosing errors, vitamin toxicity, medication interactions, and contamination.[4]

A similar regulatory ambiguity exists in the United States. While commercially manufactured IV fluids are regulated by the US Food and Drug Administration (FDA), hydration spas frequently use compounded versions of approved products, allowing them to operate outside of standard FDA oversight by classifying themselves as independent compounding entities.[1]

Common additives to "hydration therapy" include magnesium, glutathione, nicotinamide, adenine dinucleotide, and high-dose vitamins, as well as active pharmaceuticals like ketorolac, ondansetron, and, increasingly, glucagon-like peptide-1 receptor agonists.[10]

Is there a proven benefit?

Even if IV hydration spas could be shown to operate safely, a fundamental question remains: Is there a proven benefit? The promise of IV hydration spas is rooted in the perceived health benefits of vitamin supplementation, long promoted as a pathway to enhanced wellness and longevity. In Canada, about 40% of adults report using multivitamins.[5] However, high-quality evidence increasingly challenges the assumption that more vitamins translates to better health.

Research published in *JAMA* in 2024 found that vitamins do not help people live longer.[6] The study followed 390 124 generally healthy adults for up to 27 years, examining multivitamin use and mortality outcomes.[6] The investigators carefully adjusted for potential confounders, including physical activity, alcohol intake, and diet quality, to mitigate the healthy-user effect. Vitamin use was more common among college-educated women with a lower body mass index and healthier diet. Over the follow-up period, approximately 42% of participants died, most commonly from cancer, cardiovascular disease, or cerebrovascular disease.

The findings were striking. Daily multivitamin use was not associated with reduced mortality. In fact, regular use was associated with a 4% higher mortality risk. The accompanying commentary appropriately emphasized nuance.[7] The authors cautioned that these data did not capture benefits unrelated to longevity—for example, beta-carotene, vitamins C and E, and zinc for age-related macular degeneration; multivitamins for slowing cognitive decline or post–bariatric surgery supplementation; or supplementation to prevent frank deficiencies like scurvy (vitamin C) and beriberi (thiamine). Yet beyond some clearly defined indications, the authors concluded that there is little health rationale for the use of multivitamin supplements in otherwise healthy individuals. As they noted, micronutrients are most healthfully obtained from food.[7]

Against this backdrop, IV hydration spas seem difficult to justify as a meaningful health intervention. They offer costly, invasive treatments with unproven benefits, variable oversight, and potential for harm, wrapped in the language of wellness.

What do you think? Are IV hydration spas the latest tool in our collective quest for vitality and longevity? Or are they simply a more expensive way to produce urine? ■

—Caitlin Dunne, MD, FRCSC

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Tending the garden: Reflections on family medicine

I recently began to learn about gardening. There was unused space in my yard and bare soil ready to be filled, and the idea of growing a beautiful garden was appealing. What I did not expect was how difficult gardening could be, and how the lessons learned would reflect my work in family medicine.

One of the first lessons gardening teaches is that you cannot rush growth. No amount of watering will make a seed sprout faster, and no amount of attention will make a plant grow before it is ready. In family medicine, we are often asked to take on more patients in our panel, see more patients each day, and do more administrative tasks like completing forms and writing reports. But so much of what we do and what is centrally important is building relationships with patients over time. Managing chronic diseases, supporting mental health, and encouraging healthy lifestyle choices all take time and effort. We are chipping away, one piece at a time, and this cannot be rushed.

Another lesson became clear when many of my plants failed to thrive. The issue was not the plants themselves, but the soil and the environment. Until I improved drainage, added nutrients, and allowed areas to recover, the garden struggled. The same applies to medicine. The system we work in determines our ability to provide effective care. In British Columbia, ongoing challenges such as limited access to primary care, long wait times, and inequitable health care delivery all directly impact patient care. Asking physicians to thrive without first addressing these issues is like blaming a plant for struggling in depleted soil. With the introduction of the Longitudinal Family Physician Payment Model, which has been a system-wide change in family medicine, both physicians and patients have benefited greatly.

Gardening has also taught me the importance of restraint. Too much intervention can be harmful. Overwatering kills more plants than neglect does. In family medicine, we see this lesson often. Overinvestigation and overprescribing can create unintended harm and put unnecessary strain on our system. Doing more does not necessarily equate to providing better care. Early in my training, I often felt pressure to act—to order tests or to offer treatments—because patients are often looking for immediate answers. But in family medicine, we are in a unique position to practise longitudinal care and develop an understanding of patients over time. With experience, I have learned when observation and monitoring can be appropriate and effective. Knowing when to step back is also an important skill.

Finally, the quiet, hard work of tending to a garden may feel isolating at times, like in family medicine, where fatigue and burnout can also feel isolating. Family medicine is not about dramatic lifesaving moments; rather, it is about showing up, season after season, caring for our patients in small, meaningful ways, but making a lasting impact. This is the true power of family medicine.

Gardening has reinforced several truths about family medicine: growth cannot be rushed, strong support systems are essential, and restraint is often necessary. As spring approaches, I encourage colleagues to take time to slow down and admire the nature and beauty our home of British Columbia has to offer. Maybe even take up gardening. You may find, as I did, that it has as much to teach us about medicine as it does about plants. ■

—Yvonne Sin, MD

"IV hydration spas"

References continued from page 52

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Letters to the editor

We welcome original letters of less than 500 words; we may edit them for clarity and length. Email letters to journal@doctorsofbc.ca and include your city or town of residence, telephone number, and email address. Please disclose any competing interests.

Re: Failing health care delivery in Canada is the result of an outdated operating model

I read Dr Tevaarwerk's article [*BCMJ* 2025;67:359-364] with interest. I'm a family physician (GP from the UK, really) who moved to Canada 3 years ago. I worked in National Health Service (NHS) management and strategy for several years, and I have a PhD in epidemiology, so how different health systems work is of great interest to me. I don't know much about the Dutch health system, but of course I know a great deal about the UK's NHS, and I have studied other systems. I don't think the Canadian system (or the British Columbian system, specifically) is actually that similar to the UK's, and I don't think "command and control" is that good of a description of the BC system, certainly not compared with the NHS. It would be more accurate to describe the NHS as a highly centralized commissioner–provider system. Canada has a much more federated system (like Germany), which works to its advantage. Yes, it's also commissioner–provider, but that's not command and control—it seems designed to allow more flexibility in the system to account for huge geographic and population differences. There's very little command or control over the primary care system, which doesn't even require family physicians to attach all patients who apply (as is required in the UK, which is why it has universal GP coverage).

I would also have thought that a major difference between the Dutch and Canadian systems is the massive geographic and population challenges, such as the fact that Canada is more than 200 times the size of the Netherlands. Providing anything resembling universal health care across such a wide area and variable population is always going to be much more expensive.

The Netherlands does indeed generally outperform Canada in health care, but it isn't a huge difference by any means, as seen in the *Mirror, Mirror 2024* report from the Commonwealth Fund.[1] For what it's worth, Canada even slightly outperforms the Netherlands in reducing mortality and administrative barriers to care.

From what I've seen, the Canadian system (at least in BC) isn't perfect by any means, but it continues to get a lot of things right and is noticeably more sustainable and robust than the one I left in the UK. I'm sure it can improve, and better integration is certainly one way it could, but I don't think paying doctors more is particularly part of the answer.

—Paul Park, MB, BChir, MRCGP
New Westminster

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Re: Presentation of pediatric cannabis ingestion in the emergency department

We read with interest the recently published article "Presentation of pediatric cannabis ingestion in the emergency department" by Sage and colleagues[1] and commend the authors for this important work. It highlights important information on the increasing prevalence of emergency department (ED) visits due to exploratory ingestions of and pediatric exposure to edible cannabis products in Canada. It also discusses the importance of prevention as a primary strategy for reducing the availability of these products to young children, including implementing strict package warnings, labeling standards, and promotional limitations, thereby reducing pediatric cannabis exposures.[1]

Tackling the packaging of edibles as a preventive strategy to reduce cannabis exposure in young children is an important aspect of this public issue in North America. Many labels used for cannabis products include bright, colorful figures and are highly attractive to young children exploring their environments. Moreover, much of the packaging is made to resemble popular non-cannabis-containing candy and snacks, further increasing children's risk for consumption. Reducing the recognizability of packages using labeling standards and plain materials and design are critical tools that could reduce children's exposure to cannabis ingestion.

Another important area for prevention of harm is regulating the amount of cannabis contained in one packaged edible. Sage and colleagues report that there is no exact dose–response relationship for cannabis, but oral bioavailability of tetrahydrocannabinol (THC) is higher in children than in adults.[1] This difference in the clinical pharmacological properties of THC among children has been reported with the increase in severe toxicity cases, resulting in more ED visits and pediatric ICU admissions following the legalization of marijuana.[2] Many edibles are packaged with multiple "doses" in each package, and children who accidentally ingest the edible are at risk of consuming the entire product, when the intent is for

the product to be rationed into distinct "doses." As an example, a single square of chocolate or a single gummy may contain one "dose," but a package can contain an entire chocolate bar or several handfuls of gummies. Some packages of edibles contain up to 500 mg of THC, a highly toxic dose for children, since 100 mg of THC is considered a very high dose for an adult.[3]

We agree that pediatricians have an important role in counseling families about safe storage of edibles at home, but without increased public health attention and legislative drive, preventable ingestions will continue. Packaging that is not visually appealing to children and childproofing are two critical methods of reducing pediatric exposure to marijuana and visits to the ED for symptoms of toxicity.

—Hannah Zwiebel, MD, MPH
Atlanta, Georgia

—Ran D. Goldman, MD, FRCPC
Vancouver, BC

—David Greenky, MD
Atlanta, Georgia

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Re: Ethical considerations around the use of artificial intelligence in health care

Studies have shown that critical reasoning atrophies when using artificial intelligence (AI), even if the intent is to be diligent.[1] Microsoft has confirmed this finding.[2] Deep knowledge of statistics and computers is not needed to understand the negative impact of AI on cognitive abilities.[3]

It doesn't matter how well you prompt a chat bot; it will still get a staggering number of answers wrong. The example prompt offered in the article ["I am a family physician in Vancouver. What is the best antihypertensive medication for my 55-year-old Indigenous patient with comorbidities including heart failure and chronic kidney disease? Search PubMed for relevant publications and provide references for your answer. Select medications covered by non-insured health benefits."], like any similar prompt, is subject to which medication has the most aggressive marketing in the data set. This assumes the data set is not limited to peer-reviewed articles, with all conflicts of interest accounted for.

A "good prompt" should consider the risk of violating patient privacy and confidentiality, promote transparency, and meet professional standards of practice.[4]

—Chris Whittington, MB BS, MBA, FCFP, FM & FACRRM, FACTM
Abbotsford

This letter was submitted in response to the COHP article in the November issue of the BCMJ *(2025;67:326,328).* –Ed

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Being human as a physician

I should probably start by admitting that I'm writing this in early January, even though you're reading it in March. That's the reality of print deadlines. I'm also writing while traveling to Vancouver. The Comox airport is having one of those days—forgotten boarding passes, equipment not working, a delayed barista to make my coffee. Plenty of opportunities to feel frustrated, and plenty of people I feel like blaming.

Because it's January, and because this is my first trip as president, I'm reflecting on my career. Early in my medical career, mistakes were often criticized—if not first quietly hidden—and almost always accompanied by shame. If something went wrong, someone had to be at fault. This way of thinking can become a habit. Even today, I notice an instinct to look for blame when things don't go smoothly. I work hard to counter this, usually by reminding myself of my own history of mistakes, both in and out of medicine. I am human, after all.

As physicians, we are trained to aim high. From the first days of medical school, we are taught that details matter, excellence is expected, and errors can have serious consequences. That culture of high standards has saved countless lives. But alongside that, often unintentionally, we have created something far less healthy: a culture in which mistakes are tightly bound to shame.

The truth is that mistakes in both medicine and leadership are inevitable. They happen despite good intentions, deep knowledge, and careful practice. They happen to trainees and experienced clinicians alike, in hospitals, clinics, and even the Doctors of BC boardroom. Yet many of us experience mistakes not as opportunities to learn, but as personal failures—evidence that we are not good enough. Carrying mistakes this way takes a toll. It is one of the quieter drivers of burnout, anxiety, and loss of joy in our profession.

Shame thrives when we feel alone. When we believe we are the only ones who have made an error, we withdraw. We replay events over and over, questioning our competence and worth. We avoid talking about what happened, not because we don't care, but because we care deeply—and because we fear judgment even more. This isn't weakness. It's a predictable response in a culture that too often equates error with blame.

The cost of that culture can be high. Shame does not make us safer clinicians; it makes us more isolated ones. It discourages openness, stifles curiosity, and blocks improvement. When mistakes are hidden, systems can't learn. When honesty feels risky, the profession becomes less resilient—as do the people within it.

It's important to say this clearly: acknowledging mistakes does not mean abandoning accountability. Accountability and compassion are not opposites. We owe our patients and each other transparency, responsibility, and reflection. But accountability rooted in fear leads to defensiveness and silence. Accountability rooted in learning leads to safer care, stronger systems, and healthier clinicians.

Changing this culture starts with everyday conversations. The words we choose after an adverse event matter. Do we ask "Who made the mistake?" or do we ask "What happened, and what can we learn?" Do we rush to judgment or do we try to understand the context and pressures involved? As Carl Jung put it, "Know all the theories, master all the techniques, but as you touch a human soul, be just another human soul."

We also need to pay attention to how we treat each other after a mistake. The idea of the "second victim" is well recognized: clinicians involved in adverse events often experience guilt, anxiety, depression, and burnout. Leadership empathy should be routine, not remarkable. Sometimes hearing "I've been there" can make the difference between coping and struggling.

Psychological safety is not a soft concept. It is essential for both patient safety and physician wellness. Teams that feel safe to speak up identify risks earlier and adapt better. Systems that support clinicians instead of shaming them see more reporting and less harm. This isn't about lowering standards; it's about meeting them with honesty and compassion.

As I walk alongside you this year, I believe that not only is it acceptable for me to acknowledge mistakes, it is essential. Imperfection is compatible with professionalism. Our patients benefit when we are reflective and supported. And we benefit when we work in a profession that understands that excellence is not the absence of mistakes but the presence of integrity, curiosity, and caring for ourselves and each other.

My flight is being called. Now, where did I put my boarding pass? ■

—Adam Thompson, MD
Doctors of BC President

Everyone deserves better health care

Five priorities for Canadian health system improvement from medical leaders and trainees in BC.

Elsie Jiaxi Wang,* BSc, Joban Bal,* MD, Kathleen Ross, MD, Brian Yang, MD, Randeep Gill, MD, Kendall Ho, MD, John Pawlovich, MD, Gregory dePape, MD

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This article has been peer reviewed.

ABSTRACT: Primary care, the doorway to Canada's publicly funded health system, has been eroded significantly over the past decade, leaving millions of people without consistent, timely services. In 2023, only 86% of Canadians had a regular family doctor—the lowest among peer OECD countries—with the greatest gaps among young, low-income, and racialized populations. This crisis in access has led to rising chronic disease complexity, worsening emergency department burden, hospital overcrowding, and system-wide strain, with the workforce shifting further away from longitudinal family practice. Despite mounting evidence of the need for reform, federal leadership debates have largely ignored health care, revealing a lack of a coordinated national strategy.

As medical trainees and health care leaders from across British Columbia, we call for urgent, collaborative action across the federal, provincial/territorial, and regional levels to rebuild Canada's health system. We highlight five priorities in this article: expanding team-based primary care; adopting national licensure for stronger workforce planning and retention; investing in infrastructure; accelerating digital integration; and advancing equity for rural, remote, and Indigenous communities.

Erosion of access to primary care

Over the past decade, Canada has witnessed a marked decline in access to primary care, a foundational element of its publicly funded health system. In 2023, only 86% of adults reported having a regular primary care provider, the lowest rate among 10 high-income countries surveyed, down from 93% in 2016.[1] This erosion translates to millions of Canadians without consistent access to essential health services, with the disparities most pronounced among young adults, those with lower incomes, and racialized groups. The situation is particularly acute in areas of Canada such as Nunavut and the Northwest Territories, where nearly 58% and 41% of adults, respectively, reported lacking a regular provider in 2023, compared with the national average of 17%.[2] The consequences of this access gap are profound. Individuals without a primary care provider are more likely to experience unmanaged chronic conditions, poorer health outcomes, increased reliance on emergency departments, and increased hospitalizations.[3] Even those who are attached to a provider often face lengthy waits for appointments, delaying timely care and further straining the system.[4]

Systemic strain and declining capacity

Canada's hospital infrastructure reflects a similar pattern of decline. In 2021, the country had just 2.6 hospital beds per 1000 people, far below the Organisation for Economic Co-operation and Development average of 4.3 beds per 1000, and significantly less than countries such as Japan (12.6 beds per 1000) and South Korea (12.8 beds per 1000).[5] This capacity has steadily decreased from about 7.0 beds per 1000 people in 1970.[6] As a result, hospitals are frequently overcrowded, creating backlogs and worsening the long waits for both emergency and elective care.

The primary care workforce is also under significant strain. Nearly 30% of family physicians now practise predominantly outside of primary care, further exacerbating access issues and leaving many communities underserved.[7] Despite some provincial reforms and efforts to expand training or broaden scopes of practice, the lack of a unified federal strategy has resulted in fragmented delivery, persistent inequities, and a growing burden of preventable suffering.[8]

Political inattention

Despite the mounting evidence of a crisis, there was a deafening silence on the topic of health system change in the 2025 Canadian leadership debate and election campaign. The dedicated topic of health care was excluded from both the English and French national leaders' debates, a decision that drew public criticism from health professionals, labor leaders, and advocacy organizations for failing to address one of Canadians' top concerns.[9,10] The Canadian Medical Association advocated through its Fighting for Care campaign with calls to keep health care at the forefront of the national political agenda, urging all parties to commit to system transformation and sustainable funding.[11] Simultaneously, the Canadian Nurses Association and the Canadian Health Coalition also expressed alarm, noting that previous campaigns, such as those in the early 2000s and 2015, featured explicit commitments to the Canada Health Act, national wait time strategies, and investments in primary care and pharmacare.[10] In contrast, the 2025 campaign offered only brief mentions of health care, with little substantive discussion of primary care reform, workforce planning, or hospital capacity.[2,9,10] This lack of focus stands in stark contrast to earlier eras, when federal campaigns featured robust debate and policy proposals on health care reform.

Although provinces and territories hold much of the responsibility for health service delivery, with some notable exceptions including the RCMP, most First Nations health services, and BC's First Nations Health Authority, that does not absolve the federal government of responsibility.[12] Current fragmented attention at the national level through Health Canada, the Public Health Agency of Canada, the Canadian Forces Health Services Group, and initiatives such as Canada Health Infoway is inadequate, risking further entrenchment of disparities in areas such as artificial intelligence innovation and adoption and health care workforce planning, and undermining coordinated responses to the urgent needs of Canadians.

Priorities for government action

We call on the federal government to prioritize the following areas in the next term to strengthen primary care and improve health for all Canadians. Coordinated action with federal prioritization, provincial/territorial recognition, and regional responsibility is required to facilitate necessary change in this ever-worsening primary care and health care crisis that threatens every Canadian.

Expanding team-based primary care

The federal and provincial/territorial governments should push for additional team-based models of care, suggests Dr Randeep Gill. Dr Gill asserts that in BC, an inadequate tertiary care system with overrun emergency departments, long wait times for specialist care, and inadequate bed spaces in hospitals requires a shift toward interprofessional team models that leverage the skills of physicians, nurses, social workers, and allied health professionals to provide comprehensive, coordinated primary care.

Real-Time Virtual Support (RTVS) is a virtual service provided by specialist and primary care physicians across the province and supported by the Rural Coordination Centre of BC, the BC Ministry of Health, and the First Nations Health Authority. It was founded by Dr Kendall Ho, Dr John Pawlovich, Dr Ray Markham, and Mr John Mah. Dr Ho highlights that the current health care system is a bridge, but not a well-paved bridge—it is full of holes, and each patient's journey is interrupted by areas full of potential gaps. One solution to alleviate health care system pressure is to effectively triage patients virtually before they reach the emergency department via programs such as HealthLink BC 8-1-1. A 2021 study highlights that out of "7531 calls, 2548 (33.8%) callers were advised to attempt home treatment, 2885 (38.3%) to contact a primary care physician within 1 week, 1131 (15.0%) to attend an emergency department immediately, and 538 (7.1%) to attend their primary provider now."[13] By 2025, 176 000 callers were reached, representing all 231 of BC's Community Health Service Areas.[14] Other pathways within RTVS foster connections from provider to patient by "bringing the family physician, specialist, and patient together in [one] appointment via virtual care to facilitate timely referral and patient management,"[14] while also offering provider-to-provider support, including examples where urgent-care physicians "have supported overnight emergency department coverage to prevent diversions in [12] communities."[14] Not only do such models in critical and urgent care, maternity, and pediatrics improve patient outcomes by decreasing health care fragmentation, but, as Dr Pawlovich describes, they also democratize health care access for those who would otherwise not receive it. Team-based care on the ground alongside virtual models within RTVS reduces clinician burnout by offering provider-to-provider support, decreasing unnecessary emergency visits and improving early diagnosis and management.

Adopting national licensure for stronger workforce planning and retention

Canada faces a critical shortage of skilled health care workers, from physicians and nurses to technologists and support staff. The federal government must invest in provincial prioritization of increased training positions; strategic recruitment, including international talent; and retention strategies that prioritize fair compensation, safe working conditions, and meaningful expert provider involvement in effective system planning. Dr Gregory dePape emphasizes that a chronic lack of workforce planning has left long-term care, inpatient care, and emergency departments

understaffed. He cites that there is also a decrease in the recruitment of full-scope family physicians, where providers practise across outpatient and inpatient settings. More recently, a closure of the Port Alberni Diabetes Education Centre due to insufficient staffing left patients resorting to virtual care or having to drive to nearby cities. It is alarming that health care systems in BC and across Canada are facing chronic staffing shortages, despite expansion efforts, due to inadequate workforce planning, particularly in emergency and longitudinal primary care. National licensure for health care professionals would help address regional disparities in rural and remote locations and transfers between provinces and territories, ensuring that providers can work where they are most needed.

Investing in infrastructure

Targeted federal investment in infrastructure is essential, particularly in rapidly growing and historically underserved communities such as the Fraser Health region. Dr Gill emphasizes that this includes building new hospitals, diagnostic hubs, and urgent care centres, as the emergency department has become a microcosm of every systemic failure upstream. Not only are these communities growing, but they are also absorbing disproportionate health care burdens with insufficient capacity. Currently, Royal Columbian Hospital in New Westminster is operating at capacity, with up to 30% of patients awaiting long-term care; Dr Brian Yang also points out that his patients in the Fraser Health region are becoming more complex, with an ongoing need for preventive care. It is key to expand sub-acute and home-based care to transition patients who no longer require acute care out of hospitals. According to a local health review, "mortality rates have seen a large increase in Alberni-Clayoquot from 78.1 per 10 000 population in 2013–2017 to 97.6 per 10 000 population in years 2019–2023."[15] Addressing these capacity gaps is vital to meet the demands of a growing and aging population and to relieve pressure on existing facilities.

Accelerating digital integration

Digital innovation and health data interoperability must be accelerated federally to enable seamless sharing of patient information, support quality improvement, and drive system-wide efficiency. The federal government should also encourage research and development in artificial intelligence and health technology, supporting Canadian innovators through grants, start-up projects, health care technology investment funds, and prioritized procurement of homegrown solutions. Dr Kathleen Ross underscores that frameworks with measurable improvements, such as the Working Together to Improve Health Care for Canadians bilateral agreements,[16] should be used to share knowledge and facilitate a learning health care system, where successes can be rapidly scaled, accountable spending monitored, and effective solutions shared. As well, the Health Data Coalition encourages learning from community-based practices that host extensive data to address issues such as administrative burden.[17] These investments will not only improve care efficiency and decrease paperwork demands but also foster economic growth and job creation in the health care sector. Accountability and outcome-focused funding are crucial across the country. Funding at all levels of government must be tied to meaningful, targeted outcomes for both patients and providers.

Advancing equity for rural, remote, and Indigenous communities

The federal government must support the infrastructure needed for collaborative health care reform and ongoing reconciliation across provinces, territories, and unceded territories. This includes installing high-speed Internet to allow for technology adoption for timely care, alongside medical supply improvements in blood products, laboratory services, and health care facilities, and promoting connectivity to timely services that support rural, remote, and Indigenous groups. Dr Pawlovich highlights that despite enhancing telemedicine to minimize disparity in health care services, the country lacks connectedness due to a lack of broadband Internet, which worsens the challenges that disadvantaged individuals face in accessing telemedicine, primary care, acute care services, and even childbirth.

Conclusions

The next federal government must take a leadership role in rebuilding Canada's health care system, moving beyond incremental change to bold, coordinated action. By investing in primary care, workforce planning, infrastructure, digital innovation, and equity, and by holding the system accountable to outcomes, Canada can move toward a technologically advanced, sustainable, and equitable health care future for all its communities.

As a medical student, Ms Elsie Jiaxi Wang wants to begin her career in family medicine with hope, continuous policy response to Canadians' needs, and an ever-evolving health system. However, as described by Dr Joban Bal, the country is now the patient—delayed, deteriorating, and in need of lifesaving care. ■

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Real-Time Virtual Support: A network designed to support us all

The Canadian health care system is bursting at its seams. Symptoms of system challenges include frequent ER and clinic closures, physician burnout, and an ever-increasing population without access to care. We need human resources, but the number of physicians graduating from medical schools across Canada is far lower than the number needed to sustain what we have, much less enable the system to thrive. Is there a solution? Real-Time Virtual Support (RTVS) may be an answer—at least for rural health care sustainability.

RTVS is a network of specialists providing instant, accessible, consultative support to physicians, residents, nurses, nurse practitioners, and midwives for rural and remote patients in British Columbia. Operated by the Rural Coordination Centre of BC, RTVS is funded by the Ministry of Health and the Joint Standing Committee on Rural Issues, a partnership between the Government of British Columbia and Doctors of BC through the Physician Master Agreement. RTVS has five dedicated lines: RUDi and VERRa for emergency medicine, CHARLiE for pediatrics, ROCCi for critical care and internal medicine, and MaBAL for maternal health. Our aim is to support care for patients close to home, often at nursing stations and in rural emergency departments. Our physicians reside and practise in BC, with expertise and experience in rural medicine. We have intimate knowledge of the strengths and struggles each community experiences. RTVS enhances pivotal in-person care, providing a safety net for rural providers while helping enable recruitment and retention.

Many urban and tertiary-care physicians are understandably unaware of RTVS and its impact. They are buried in the never-ending to-dos of patient care, administration, and continuing education, not to mention life outside of medicine (*gasp*). On a good day, it is overwhelming.

The effects of RTVS are felt every day.

And yet, how different our days could be. Imagine being a locum in the Burns Lake emergency room with several patients waiting to be seen. To your dismay, a 4-year-old child presents with respiratory distress. You haven't cared for an acute pediatric case since graduating, much less in a remote centre. You start the child on oxygen, and you panic internally. While preparing to call the Patient Transfer Network for a transfer to a tertiary centre, the nurse working alongside you suggests that you call CHARLiE. They help place a Zoom call, and you are instantly greeted by an experienced pediatrician with "Hello; how can I help?" They guide your history taking and examination to find the child is having an asthma exacerbation. They virtually support management at the bedside the entire time. The child responds beautifully and is later discharged home. You feel relieved and have increased confidence in your remote setting with newfound support.

Previously, this call would have gone to a tertiary site physician. Instead, they can continue to focus on their long list of local patients and consultations without having to navigate additional rural cases. While it is within their scope, chances are that without the immediate dedicated care that CHARLiE provided, the child would have been transferred for ongoing care, perhaps after decompensating.

There is a positive ripple effect across the system. The transport physician with the Patient Transfer Network did not hear about the patient and could focus on their pending transfers. The child's parents did not miss work or spend time and money on travel to a centre 3 hours away. BC taxpayer dollars were not spent on an unnecessary patient transfer. The government saved much-needed dollars from a needless transfer and retained a returning locum, who will provide care to thousands in the coming years.

The effects of RTVS are felt every day. RTVS has supported over 100 000 calls from rural and remote providers in the last 5 years, with call volumes increasing annually. RTVS has prevented over 13 000 hours of rural emergency room closures. It is estimated to have reduced patient-borne costs of more than $34 million (and counting). Patients stay close to home, local physicians are retained, and tertiary-care physicians can be sustained.

The classic brick-and-mortar health care system does not work with the current resources, at least not in isolation. We need a network of care, an ecosystem to support each entity to survive and thrive. Every patient deserves timely, specialized care within their chosen community, and with the innovative and welcoming minds of RTVS and its providers, it's possible.

To learn more, visit https://rccbc.ca/initiatives/rtvs/. ■

—Kayla Parker, MD, FRCPC
CHARLiE Provider

This article is the opinion of the Joint Collaborative Committees (JCCs) and has not been peer reviewed by the BCMJ *Editorial Board.*

Gastroesophageal reflux disease: Diagnosis and management in a primary care setting

Most patients with gastroesophageal reflux disease can achieve excellent outcomes in a primary care setting if given a history-based diagnosis and appropriate pharmacological therapy, lifestyle counseling, and follow-up.

Estello Nap Hill, MD, FRCPC, Gunisha Kalra, MD, Sarvee Moosavi, MD, FRCPC, EdM, AGAF

ABSTRACT: Gastroesophageal reflux disease is one of the most common upper gastrointestinal disorders and accounts for considerable symptom burden and health care use. If left untreated, it can lead to complications such as esophagitis, strictures, Barrett's esophagus, and esophageal adenocarcinoma. However, most patients can be managed in primary care using a structured approach. The disease is generally diagnosed clinically; in patients with typical symptoms of heartburn and regurgitation without alarm features, an empiric trial of proton pump inhibitors serves as first-line therapy. Diagnostic endoscopy is reserved for those with alarm features, inadequate response to optimized therapy, or risk factors for Barrett's esophagus. Patients with persistent or atypical symptoms despite optimized therapy warrant further evaluation, such as upper endoscopy and esophageal function testing, including esophageal manometry and pH testing. Antireflux surgery may be considered in carefully selected patients, and laparoscopic fundoplication can offer relief in selected patients. Newer options such as magnetic sphincter augmentation and endoscopic techniques have limited roles.

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Gastroesophageal reflux disease (GERD) is one of the most common gastrointestinal disorders affecting Canadians; it impacts up to 1 in 5 adults and is responsible for a large proportion of primary care visits.[1] Its symptoms, often dismissed as benign heartburn or indigestion, can erode quality of life; disrupt sleep; and lead to complications such as esophagitis, strictures, Barrett's esophagus, and esophageal adenocarcinoma if left untreated. Given its high prevalence, GERD is a condition that primary care providers are uniquely positioned to manage effectively. With a thoughtful approach that emphasizes history-based diagnosis, rational use of pharmacological management, lifestyle counseling, and appropriate follow-up, most patients can achieve excellent outcomes in a primary care setting without specialist referral. This article provides an evidence-based approach for primary care clinicians to navigate GERD confidently and helps them recognize when reassurance and education suffice and when escalation or further investigation by a gastroenterologist is warranted.

Clinical cases, part 1

Case 1

Aarti, a 38-year-old woman, reports a 2-year history of heartburn one or two times per week, often after eating spicy food or late-evening meals. She takes 20 mg of omeprazole as needed with meals, when symptomatic. She finds some relief, but symptoms continue to recur. She has no dysphagia, weight loss, anemia, or alarm features **[Box]**. She prefers to avoid daily medication if possible. How would you manage Aarti's current symptoms and proton pump inhibitor use?

Case 2

David, a 46-year-old man, has had daily heartburn for years. He has a 50-pack-year history of smoking. His BMI is 31. He has been on 40 mg of esomeprazole twice daily for 6 weeks, with minimal improvement. He reports no dysphagia, odynophagia, weight loss, or gastrointestinal bleeding. What are the next steps in his management, and does he need an upper endoscopy?

Pathophysiology

The Montreal Consensus defines GERD as a multifactorial condition in which gastric contents reflux into the esophagus, causing troublesome symptoms and/or complications.[2] The esophagogastric junction, comprising the lower esophageal sphincter and crural diaphragm, normally prevents

reflux, but it can be compromised by transient lower esophageal sphincter relaxations; low basal lower esophageal sphincter pressure; or anatomical defect, such as hiatal hernia.[3] Acid and bile exposure can trigger mucosal injury via inflammatory mediators.[3] Impaired clearance from upper gastrointestinal dysmotility or xerostomia, as in Sjögren's syndrome, may prolong refluxate contact time. Symptom severity varies with mucosal sensitivity and may not correlate with acid exposure.[3]

Chronic acid and bile reflux can erode the esophageal squamous epithelium, leading to inflammation and ulceration (reflux esophagitis).[3-5] Repeated injury and subsequent fibrosis during the healing process can cause stricture formation and significant dysphagia. This cycle of injury and healing in the distal esophageal squamous lining can cause progenitor cells at the gastroesophageal junction to accumulate somatic mutations and reprogram themselves, forming metaplastic columnar epithelium, which is more resistant to acid.[6,7] This specialized intestinal metaplasia, known as Barrett's esophagus, represents a premalignant condition in approximately 1% to 2% of the general population and up to 10% to 15% of patients with chronic GERD.[6] Barrett's esophagus progresses along a metaplasia–dysplasia–carcinoma sequence, from nondysplastic metaplasia to low-grade dysplasia, high-grade dysplasia, and eventually invasive esophageal adenocarcinoma.[6,7] Although the annual risk of progression to cancer in nondysplastic Barrett's esophagus is low (approximately 0.5% per year), the high prevalence of Barrett's esophagus in the population makes it the predominant precursor lesion for esophageal adenocarcinoma, with most cases arising through this metaplastic–dysplastic pathway.[7]

Clinical presentation and diagnosis

Symptoms

Typical GERD symptoms include heartburn (substernal burning toward the throat); regurgitation of sour/bitter contents; and chest pain, which can mimic cardiac pain.[5] Regurgitation should be distinguished from rumination, in which involuntary abdominal contractions cause the return of recently ingested food. The latter usually occurs while eating or shortly after eating, where the content tastes similar to the material just ingested; the content is often chewed again and swallowed back down.[5]

The Lyon Consensus, an expert statement that provides objective diagnostic thresholds for GERD, also describes gastric belching, a physiologic venting of air that may precipitate reflux, and supra-gastric belching, a behavioral pattern of rapid air entry/expulsion before entry into the stomach.[5] Differentiating these symptoms is important in patients who are thought to have proton pump inhibitor–refractory GERD, in which case their esophageal symptoms are persistent despite good compliance with a course of single- or double-dose proton pump inhibitor, taken 30 minutes before a meal, for 4 to 8 weeks.

Patients may report cough, hoarseness, throat clearing, globus sensation, asthma-like symptoms, nausea, and/or abdominal pain.[5,6] In the absence of typical reflux symptoms, these have low sensitivity and specificity for GERD in isolation, and acid suppression is often ineffective.[6] Chronic cough usually has multiple causes, including upper airway cough syndrome, postinfectious respiratory diseases, postnasal drip, environmental exposures, ACE inhibitors, nonacid reflux, irritable larynx syndrome, and other cardiopulmonary pathologies. In addition, placebo response to proton pump inhibitors in this patient population can be high, with up to 42% of patients in placebo groups in randomized trials reporting adequate relief of laryngeal or cough symptoms.[8] When cough or laryngitis occurs without typical GERD symptoms, objective reflux testing (pH with impedance monitoring) off proton pump inhibitor therapy should precede antacid therapy.[5] Globus hystericus is more often linked to visceral hypersensitivity and hypervigilance and often responds better to neuromodulators, such as low-dose tricyclic antidepressants.[6] If atypical symptoms accompany typical GERD symptoms in the absence of alarm features, an up-front 8- to 12-week proton pump inhibitor trial is reasonable; nonresponders should be referred for objective reflux monitoring.[5]

BOX. Alarm features in gastroesophageal reflux disease (GERD).

The following symptoms are rare in GERD and warrant further investigation:

- Dysphagia
- Odynophagia
- Gastrointestinal bleeding or anemia
- Weight loss
- Recurrent vomiting
- Choking

Evaluation and diagnosis

Alarm features—dysphagia, odynophagia, gastrointestinal bleeding or anemia, weight loss, recurrent vomiting, or choking—are uncommon in GERD and warrant further investigation, including early upper endoscopy.[4] Without alarm features, a 4- to 8-week proton pump inhibitor trial is diagnostic of GERD if symptoms resolve. Partial relief may justify twice-daily dosing for another 4 to 8 weeks. No response should prompt further investigation.[4] In resource-rich settings, up-front esophageal pH monitoring off proton pump inhibitor therapy is suggested for atypical presentations or proton pump inhibitor nonresponse before long-term therapy or invasive management is initiated.[4,5] Generally, an upper endoscopy is indicated before any esophageal function testing, which allows objective evaluation for alternative diagnoses, such as esophageal dysmotility, functional heartburn, or esophageal hypersensitivity.

Up-front upper endoscopy is recommended for patients with alarm features or Barrett's esophagus risk factors: older than 50 years of age, male sex, central obesity, Caucasian race, tobacco use, GERD for 5 years or longer, or a family history of Barrett's esophagus or adenocarcinoma.[4]

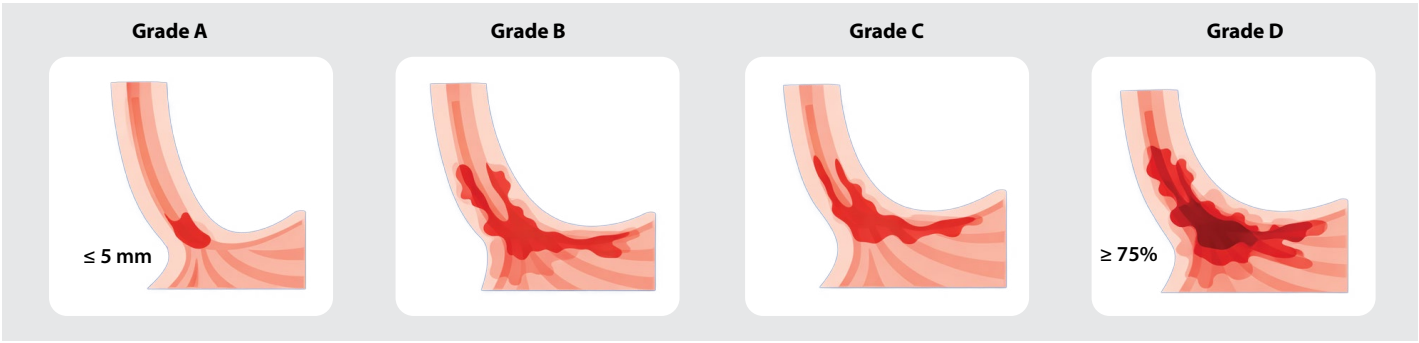

FIGURE 1. Simplified schematic representation of the Los Angeles Classification of reflux esophagitis. Grades are shown according to the extent and circumferential involvement of mucosal breaks at the distal esophagus. The classification system is based on the original definitions described by Lundell and colleagues.[9] This figure was created by the authors for educational purposes and does not reproduce or adapt any previously published illustration.

The Los Angeles Classification grades endoscopic appearance of erosive esophagitis from A (≤ 5 mm mucosal breaks not bridging folds) to D (≥ 75% circumferential injury) **[Figure 1]**.[9] Objective endoscopic evidence of GERD includes the presence of grade B, C, or D esophagitis; Barrett's esophagus; peptic stricture; or previous abnormal pH monitoring off proton pump inhibitor therapy.[5] The yield of initial upper endoscopy for GERD can increase with discontinuing proton pump inhibitor therapy for 2 to 4 weeks.[4] The absence of visible esophageal mucosal injury on endoscopy may suggest nonerosive GERD and represents most GERD cases. If clinically relevant, it can be followed up with pH testing to objectively document the presence of pathologic GERD[4,5]—for example, in cases of proton pump inhibitor nonresponders. However, given the limited access to esophageal function testing, judicious use of resources should be considered.

Barrett's esophagus—salmon-colored, velvety mucosa with histologic intestinal metaplasia—confirms GERD and requires surveillance.[4] Barrett's esophagus is graded endoscopically using the Prague C&M Classification (outlining the circumferential extent and maximal length of involved tissue) and requires careful 4-quadrant biopsies every 1 to 2 cm per the Seattle Protocol, with specific care to target suspicious-appearing lesions.[10] Endoscopic surveillance intervals depend on the presence of dysplasia. When there is no dysplasia, upper endoscopy is recommended in 3 to 5 years; if it is indefinite for dysplasia after review by two dedicated gastrointestinal pathologists, a repeat endoscopy should be done in 6 to 12 months after adequate acid suppression.[10] Preferably, low-grade dysplasia should be referred for endoscopic eradication (otherwise, surveillance should be conducted every 6 to 12 months).[10] Visible high-grade dysplasia or intramucosal carcinoma must be endoscopically resected, with close endoscopic follow-up at 3, 6, and 12 months, and annually thereafter.[10]

The updated Lyon Consensus defines "actionable GERD" as Los Angeles grade B, C, or D esophagitis; long-segment Barrett's esophagus (longer than 3 cm); peptic stricture; more than 80 reflux episodes; or distal esophageal acid exposure time greater than 6% on a 24-hour catheter-based study.[5] Further details of these investigations are outside the scope of this article. Overall, this physiologic framework helps direct therapy and avoid overtreatment, especially with prolonged proton pump inhibitor use in functional heartburn or reflux hypersensitivity.

Noninvasive testing

Ambulatory esophageal pH or pH–impedance monitoring is the gold standard for confirming conclusive pathologic GERD, defined as distal esophageal acid exposure time greater than 6% or more than 80 reflux episodes over 24 hours off proton pump inhibitor therapy.[4,5] Testing is indicated when diagnostic uncertainty exists; when there is incomplete proton pump inhibitor response; and before antireflux surgery in patients with typical GERD symptoms who have minimal or no endoscopic findings, yet symptoms persist despite optimal use of proton pump inhibitors.[4] In a resource-rich region where access to esophageal function testing is readily available, patients with atypical reflux symptoms, such as cough, sore throat, or globus, with no heartburn or acid regurgitation, should undergo up-front esophageal manometry and pH testing off proton pump inhibitors.

Catheter-based pH–impedance monitoring detects acidic and nonacidic reflux and allows the correlation of reflux episodes with reported symptoms.[4] Another modality includes wireless capsule monitoring (Bravo test), implanted in the distal esophagus during upper endoscopy, which allows recording of distal acid exposure time up to 96 hours. Barium swallow is not recommended solely for reflux diagnosis, because reflux seen on esophagram is not diagnostic of pathologic GERD.[4]

Currently, the motility lab at Vancouver General Hospital serves as the centre for excellence in BC; it accepts referrals for patients with gastrointestinal motility disorders throughout the province, while offering evidence-based, up-to-date reporting of esophageal function testing, based on rapidly evolving literature. The lab is also involved in research on esophageal disorders, including esinophilic esophagitis and esophageal motility disorders. However, resources remain limited; therefore, patients are reviewed and accepted from specialty

services based on stringent criteria, outlined on the BC centralized referral form.

Management

Lifestyle and dietary measures

Adjunctive lifestyle changes are recommended, although evidence of their benefits is modest. Even moderate weight loss in overweight patients consistently improves symptoms.[4,11] Other measures include elevating the head of the bed, particularly for nocturnal symptoms; avoiding recumbency after meals for 4 hours; and limiting late-night eating or snacking.[4,11] Smoking cessation and reduced alcohol intake are advised, because both smoking and alcohol impair lower esophageal sphincter function.[3,4] Trigger-food avoidance is reasonable when individualized; common culprits include caffeine, chocolate, peppermint, spicy foods, citrus, and fatty meals.[4] Lifestyle measures may reduce symptoms but rarely replace pharmacological therapy in severe reflux.

Pharmacotherapy

Proton pump inhibitors are first-line therapy for reflux esophagitis and are superior to H2 blockers for symptom control and mucosal healing.[4,5] Standard dosing is once daily, 30 minutes before breakfast, with improvement expected in 4 to 8 weeks; erosive esophagitis usually requires 8 weeks or longer, with more than 80% healing.[4] Severe esophagitis (Los Angeles grade C or D) or Barrett's esophagus may warrant higher-dose therapy (twice a day or double strength),[5] with indefinite use likely required to avoid recurrence and risk of further progression of Barrett's dysplasia to esophageal cancer.[10,12,13]

Beyond symptom control, proton pump inhibitors may provide chemopreventive benefit in Barrett's esophagus. Observational studies and meta-analyses suggest that long-term proton pump inhibitor therapy is associated with a reduced risk of neoplastic progression, likely through sustained acid suppression and reduction of inflammation-mediated DNA injury.[12] The AspECT trial indicated that high-dose proton pump inhibitor therapy (40 mg of esomeprazole twice daily), particularly when combined with aspirin (300 to 325 mg daily), significantly prolonged the time to the composite endpoint of high-grade dysplasia, esophageal adenocarcinoma, or death, compared with standard-dose proton pump inhibitor therapy alone.[13] However, routine use of high-dose aspirin for chemoprevention is not recommended in current gastrointestinal practice due to bleeding risk in patients without another clear indication for chronic antiplatelet therapy.[14]

Proton pump inhibitors are first-line therapy for reflux esophagitis and are superior to H2 blockers for symptom control and mucosal healing.

Patients metabolize CYP2C19 at varying rates, so their GERD response can vary depending on the proton pump inhibitor used. First-generation proton pump inhibitors (e.g., omeprazole, lansoprazole, pantoprazole) are more affected by rapid-metabolizer phenotypes; in nonresponders, switching to second-generation proton pump inhibitors (e.g., rabeprazole, esomeprazole, dexlansoprazole) that are less influenced by CYP2C19 metabolism polymorphism should be considered if their higher cost is acceptable to the patient.[15]

Clinically important adverse effects from chronic proton pump inhibitors are rare, and in high-quality randomized, placebo-controlled studies conducted over multiple years, they were generally not statistically different from placebo; a small excess of enteric infections may occur.[16] In meta-analyses, proton pump inhibitors have been linked to *Clostridioides difficile* infection, but effect sizes are modest (typical pooled odds ratio ~1.3 to 2.3 [i.e., < 3]) and subject to confounding.[16,17]

H2 blockers can relieve mild or breakthrough symptoms, but they are less effective than proton pump inhibitors in healing erosive esophagitis and are prone to tachyphylaxis.[5] A notable side effect is gynecomastia, particularly with cimetidine.[18]

Alginates form a "raft" barrier, are safe in pregnancy, and are useful as adjuncts in partial proton pump inhibitor responders or those with nonacid reflux.[19] Baclofen reduces transient lower esophageal sphincter relaxations but is limited by CNS effects.[4] Antacids provide only short-term relief by neutralizing acid and require caution and monitoring in patients with renal impairment.[20] Prokinetics (e.g., metoclopramide, domperidone) are reserved for coexisting gastroparesis or functional dyspepsia.[14] For metoclopramide, Food and Drug Administration labeling limits use to a maximum of 12 weeks due to tardive dyskinesia risk and advises caution for QT prolongation and arrhythmia risk; RCT and observational data show effects on QT dynamics.[21,22] The same potential cardiac side effect may be seen with domperidone.[22]

Vonoprazan (a potassium-competitive acid blocker) is Food and Drug Administration approved in the United States for *Helicobacter pylori* eradication (dual/triple packs) and for erosive GERD/maintenance; currently, the class is not approved in Canada.[23,24]

Once symptoms are controlled, we recommend stepping down proton pump inhibitors to the lowest effective dose. If GERD symptoms are under control, consideration can be given to using proton pump inhibitors on demand, or intermittently in nonerosive reflux disease. Severe esophagitis or chronic symptoms may require long-term therapy and reassessment on a case-by-case basis **[Table]**.

Surgery

If symptoms persist, first confirm adherence to, dosing of, and timing of proton pump inhibitor therapy, which should be 30 to 60 minutes pre-meal dosing. True refractory GERD—persistent symptoms despite twice-daily proton pump inhibitor therapy for 4 to 8 weeks—requires objective testing: upper endoscopy followed by esophageal manometry and pH–impedance monitoring off proton pump inhibitor

TABLE. Pharmacological agents used in the treatment of gastroesophageal reflux disease (GERD). Adapted from Gyawali and colleagues.[5]

Class/medication examples	Indication	Dosing	Risks/side effects	Monitoring
Proton pump inhibitors (PPIs): • Omeprazole • Esomeprazole • Lansoprazole • Pantoprazole • Rabeprazole • Dexlansoprazole	First-line therapy for typical GERD, erosive esophagitis, Barrett's esophagus	• Omeprazole 20 mg by mouth daily • Esomeprazole 40 mg by mouth daily • Lansoprazole 30 mg by mouth daily • Pantoprazole 40 mg by mouth daily • Rabeprazole 20 mg by mouth daily • Dexlansoprazole 30 mg by mouth daily; 4- to 8-week initial trial	Side effects: • Headache • Diarrhea • Nausea Long-term risks: • Hypomagnesemia • B12 deficiency • Infection Documented side effects are rare	Symptom response; consider periodic labs for long-term use
H2 receptor antagonists: • Famotidine • Cimetidine • Nizatidine	Mild–moderate GERD, nocturnal symptoms; adjunct to PPI	• Famotidine 20 mg by mouth twice a day • Cimetidine 400 mg by mouth twice a day • Nizatidine 150 mg twice a day	Headache, dizziness, constipation, gynecomastia, tachyphylaxis with chronic use	Symptom response; renal function (dose adjust for glomerular filtration rate < 50)
Antacids: • Aluminum/magnesium hydroxide • Calcium carbonate	On-demand relief of mild/episodic heartburn	As needed	Diarrhea, constipation, electrolyte imbalance (chronic use)	Not required for short-term use
Alginate compounds: Sodium alginate	Adjunct for postprandial or breakthrough symptoms	2–4 tablets by mouth four times a day	Constipation, diarrhea	Symptom response
Mucosal protectants: Sucralfate	Pregnancy; adjunct in refractory GERD	1 g by mouth two to four times a day	Constipation, nausea, drug interactions	Not routinely required
Potassium-competitive acid blockers: Vonoprazan (not FDA* approved)	Refractory GERD, erosive esophagitis (where available)	20 mg by mouth daily	Similar to PPIs	Symptom response
Transient lower esophageal sphincter relaxation reducer: Baclofen	Refractory regurgitation, belch-predominant symptoms	10–20 mg by mouth three times a day	CNS effects: drowsiness, confusion, dizziness	CNS side effects, renal function
Prokinetic agents: • Metoclopramide • Domperidone	GERD with coexistent gastroparesis	5–10 mg by mouth every 6–8 hours	Extrapyramidal symptoms, drowsiness, fatigue, QT prolongation	Neurologic side effects

* United States Food and Drug Administration.

if esophagogastroduodenoscopy shows no abnormalities.[5] This is to distinguish esophageal dysmotility and pathologic reflux from esophageal hypersensitivity or functional heartburn, where the latter often responds better to neuromodulators such as low-dose tricyclic antidepressants.[4] In patients who remain symptomatic despite optimized therapy, CYP2C19 genetic variability should be considered. Rapid and ultrarapid metabolizers can demonstrate increased hepatic clearance and subtherapeutic acid suppression; switching to proton pump inhibitors that are less affected by CYP2C19 metabolism (e.g., esomeprazole, rabeprazole, dexlansoprazole) can improve response.[17] In proven GERD with good symptom association on pH testing that is partially or totally unresponsive to optimized pharmacological and dietary therapies, antireflux surgery may be considered in carefully selected patients. Laparoscopic fundoplication offers durable relief in selected patients (e.g., young patients with severe erosive disease or large hiatal hernia),[4] although 30% to 40% resume the use of proton pump inhibitors and 4% to 10% require reoperation for complications such as gas bloat, dysphagia, or wrap failure within a few years after surgery.[4,14] Newer options—magnetic sphincter augmentation and endoscopic techniques (e.g., transoral incisionless fundoplication, Stretta)—have limited roles and should preferably be considered in expert centres.[4] Careful selection and physiologic testing are essential in ensuring a successful outcome.

Clinical cases, part 2

Case 1 recommendation

Aarti has uncomplicated GERD with no alarm features. However, her proton pump inhibitor use can be further optimized by taking the proton pump inhibitor 30 to 60 minutes before a meal for a 4- to 8-week

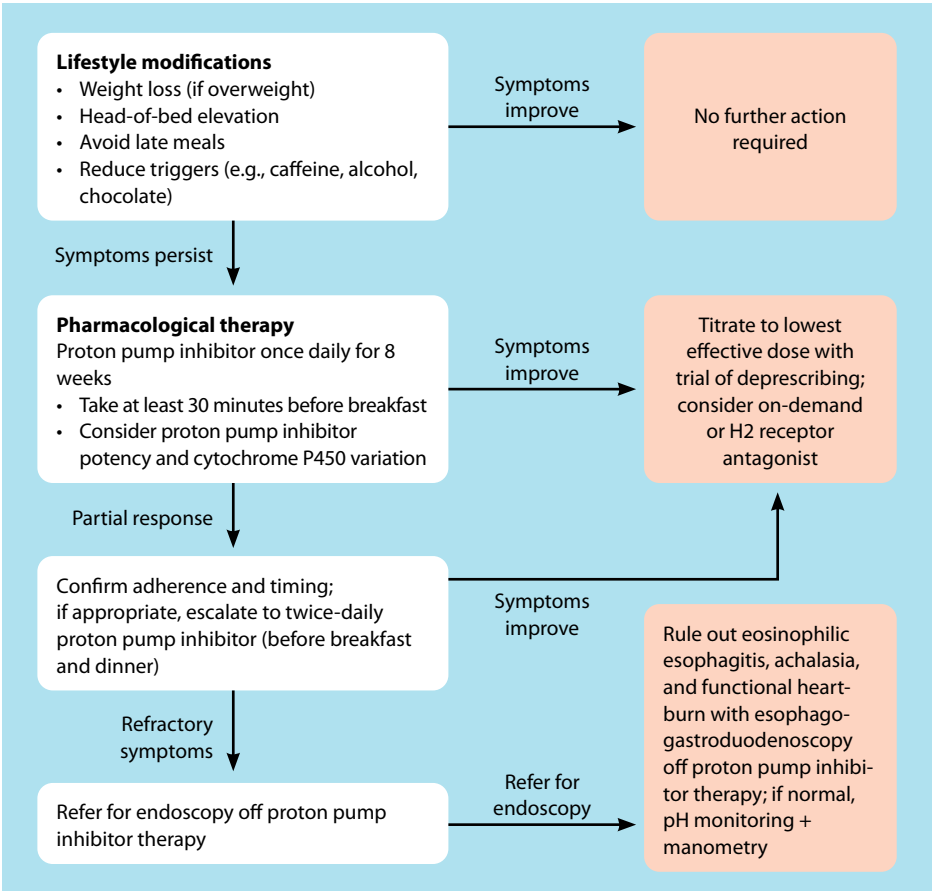

FIGURE 2. Typical gastroesophageal reflux disease (GERD) management algorithm. Adapted from Kahrilas and colleagues.[8]

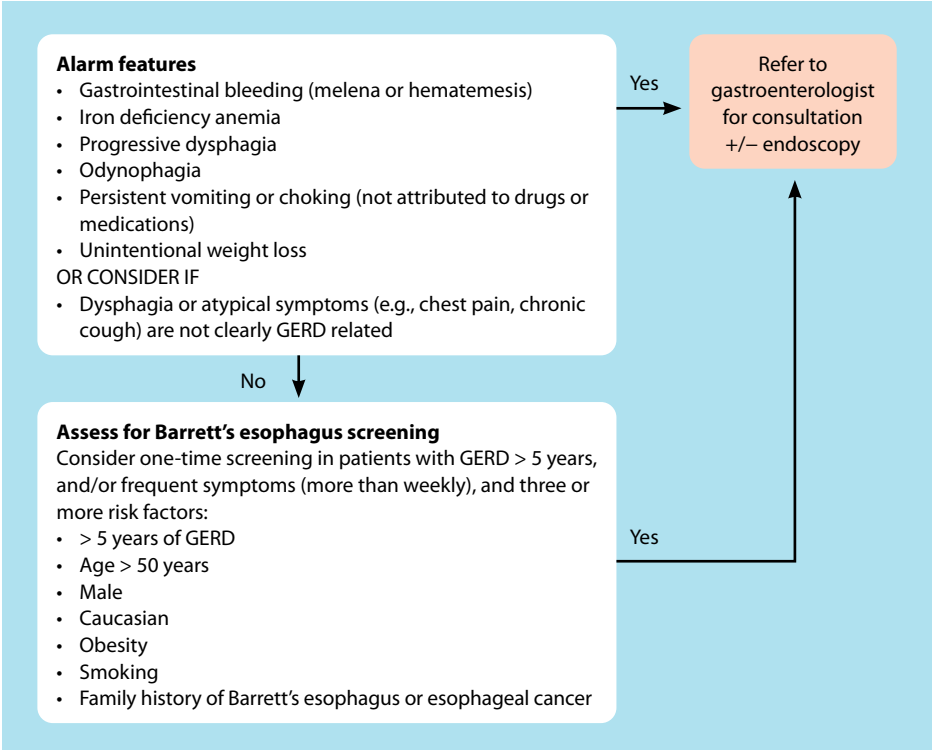

FIGURE 3. When to refer to gastroenterology. Adapted from Gyawali and colleagues.[5]

trial. If effective, she can switch to antireflux medications as needed, if symptoms recur. She should be counseled on adjunctive lifestyle measures, as discussed above **[Figure 2]**.

Case 2 recommendation

David has proton pump inhibitor–refractory GERD, defined as persistent typical symptoms of GERD, despite optimized twice-daily proton pump inhibitor therapy for 4 to 8 weeks. He should be referred for consideration of upper endoscopy with esophageal biopsies, ideally off proton pump inhibitor therapy for 2 to 4 weeks to increase the yield of biopsies **[Figure 3]**. Endoscopic evaluation will allow clinicians to rule out mimickers such as eosinophilic esophagitis or peptic ulcer disease. If there are no abnormalities, esophageal manometry may be indicated to assess esophageal motility disorders, followed by ambulatory pH or pH–impedance monitoring off proton pump inhibitor therapy to differentiate pathologic GERD from esophageal hypersensitivity or functional heartburn. Proton pump inhibitor doses should not be taken more than twice a day; adjuncts such as baclofen, alginates, or neuromodulators may be considered after testing.

Conclusions

Gastroesophageal reflux disease is a prevalent and often chronic condition that can be effectively managed in the primary care setting using a structured, evidence-based approach. Most patients achieve symptom control through accurate clinical diagnosis, lifestyle modification, and rational use of proton pump inhibitors. Primary care physicians play a pivotal role in early recognition, appropriate empiric therapy, and patient education to prevent complications such as erosive esophagitis or Barrett's esophagus. Diagnostic endoscopy should be reserved for those with alarm features, inadequate response to optimized therapy, or risk factors for Barrett's esophagus. Patients with persistent or atypical symptoms despite optimized therapy warrant further evaluation, such as upper endoscopy

and esophageal function testing, including esophageal manometry and pH testing.

By applying current guideline-based management—emphasizing stepwise therapy, periodic reassessment, and judicious investigation—primary care clinicians can manage GERD confidently and reduce unnecessary referrals, and thus optimize both patient outcomes and health care resource use. ■

Competing interests

None declared.

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Functional dyspepsia: Diagnosis and management in a primary care setting

Management of functional dyspepsia requires a multipronged approach, including nonpharmacological lifestyle changes, pharmacological therapies with a stepwise approach, and a multidisciplinary approach with dietitians and psychologists.

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CORRECTION: Postpublication, authors requested a change to their placement of the bulleted lists of symptoms in each circle in the Venn diagram in Figure 2. In June 2026, the symptoms in the two circles were reversed.

ABSTRACT: Functional dyspepsia is a common condition that involves a complex of symptoms, such as epigastric pain, postprandial fullness, and/or early satiety. While the pathophysiology is not completely understood, it is likely a complex interplay between the gut–brain axis, gut microbiome, and motor and sensory functions of the gastrointestinal tract. Endoscopy is not a mainstay of diagnostic testing and should be reserved for patients who are 60 years of age or older, or for high-risk patients based on individual cases. Clinical diagnosis can be made using the Rome IV criteria. Dietary modifications should be considered as first-line therapy prior to the use of pharmacological therapies. Other nonpharmacological treatments, such as exercise, psychological therapies, and patient counseling, should also be considered. Pharmacological treatments involve the use of proton pump inhibitors, prokinetics, and neuromodulators. Management may also require a multidisciplinary approach that includes dietitians and psychologists.

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Functional dyspepsia is a chronic, complex constellation of symptoms that include epigastric pain or burning, postprandial fullness, and/or early satiety. Given the spectrum of symptoms, it is a common presenting complaint in primary care; the estimated prevalence is 8% in Canada and 20% globally.[1,2] In addition to its burden on quality of life, functional dyspepsia is associated with significant health care costs: the annual economic impact is estimated at $18 billion in the United States alone.[3] Symptom overlap with other gastrointestinal disorders, such as gastroesophageal reflux disease, gastroparesis, and peptic ulcer disease, leading to misdiagnosis, unnecessary investigations, patient frustration, and significant impact on quality of life.[4] Given that functional dyspepsia affects almost 1 in every 12 Canadians,[1,2] primary care providers are uniquely positioned to diagnose and manage it effectively. Our aim is to provide a structured clinical pathway for diagnosing and managing functional dyspepsia in the primary care setting, which will allow family physicians to build on their expertise to both optimize patient outcomes and eliminate unnecessary wait times and testing.

Clinical cases

Case 1

Mohammad is a 51-year-old man who has had ongoing abdominal pain for several years. His medical history includes dyslipidemia on statin therapy and generalized anxiety disorder not on medical therapy. He experiences epigastric and right upper quadrant pain three to four times per week in both fasting and unfasting states, with no clear food triggers. He has no personal or family history of gastrointestinal disease and no weight loss; however, during these pain episodes, he often takes time off work. His physical examination suggests no abnormalities. He has been on proton pump inhibitor therapy with escalating doses to twice daily, and he has cut out acidic foods, caffeine, and wine, with no effect. What should the next steps be in the management of his condition?

Case 2

Samantha is a 43-year-old woman who experiences chronic postprandial fullness with bloating and discomfort on most days. This is often associated with nausea, and less frequently with vomiting. Her medical history includes hypothyroidism, which is well managed on levothyroxine, and fibromyalgia, for which she takes gabapentin. Due to abdominal pain, she has been started on hydromorphone as needed. Her physical examination shows a mildly distended abdomen with mild left-side discomfort.

She has tried proton pump inhibitor therapy with no effect. What should the next steps be in the management of her condition?

Pathophysiology

Due to the multifaceted nature of functional dyspepsia, its pathophysiology is complex and not yet fully understood. A number of potential mechanisms have been proposed; functional dyspepsia is likely a result of a complex interplay between them.

Gut–brain axis dysfunction

The gut–brain axis, communicated via the hypothalamic–pituitary axis, is modulated by stress, immune function, and the gut microbiome.[5] Central signaling through corticotropin affects gut permeability, with eosinophilic and mast cell activation contributing to barrier dysfunction.[5,6] Adverse early life experiences can also contribute to functional gut disorders,[7] which underscores the gut–brain relationship.

Altered gut microbiome

Increased duodenal bacterial load is correlated with meal-related symptoms and thus links the small-intestine microbiome with dyspeptic symptoms.[8] Dysbiosis also alters the composition of bile acids, which promotes pro-inflammatory bacterial overgrowth.[9] This relationship between the microbiome and dyspepsia is underscored by the postgastroenteritis dyspepsia phenomenon[10] and the effectiveness of *Helicobacter pylori* eradication in resolving dyspeptic symptoms.[8]

Immune dysfunction

Low-grade duodenal inflammation, evidenced by abnormal populations of inflammatory cells in duodenal samples of functional dyspepsia patients,[11] impairs duodenal mucosa integrity,[11] which leads to delayed gastric emptying[12] and decreased neuronal responsiveness,[13] and thus disrupts gastrointestinal neuroregulation.[5]

Motor and sensory dysfunction

While poor gastric emptying was thought to be directly correlated with dyspepsia, associations between functional dyspepsia and gastric emptying are inconsistent.[14] Pasricha and colleagues showed that functional dyspepsia and gastroparesis are clinically and pathologically indistinguishable in tertiary centres, which suggests they represent a spectrum of gastric neuromuscular disorders.[15] Additionally, visceral hypersensitivity, modulated by mechanical inputs, receptors, and enteral hormones,[16,17] contributes to epigastric pain and is associated with nonpainful symptoms of fullness, bloating, and belching.[16-18]

Risk factors for functional dyspepsia include female sex, smoking, use of NSAIDs, *H. pylori* infection,[1] acute gastroenteritis,[19] and psychiatric comorbidities such as anxiety and depression.[20]

Clinical presentation and diagnosis

Functional dyspepsia should be considered in patients who present with symptoms of postprandial fullness, early satiety, bloating, and/or epigastric pain or burning in the absence of alarm features **[Figure 1]**.[21] Epigastric burning in particular should be further characterized in terms of location, timing, and modifying factors, because it is often confused with gastroesophageal reflux disease. Recurrent or cyclic vomiting or symptoms related to defecation suggest alternative diagnoses and warrant appropriate workup, although other gut–brain interaction disorders could coexist. Once functional dyspepsia is suspected, diagnosis requires a detailed history and, in appropriate patients, can be made clinically using Rome IV criteria **[Figure 2]**,[21] which will minimize the use of generally low-yield endoscopy and delays in care.

Rome IV criteria for the diagnosis of functional dyspepsia include one or more of bothersome postprandial fullness, early satiation, epigastric pain, and epigastric burning; symptom duration of the past 3 months, with onset 6 months prior to diagnosis; and no evidence of structural disease as an alternative etiology.[21] Once functional dyspepsia has been diagnosed, it can be further classified into two subtypes: postprandial distress syndrome and epigastric pain syndrome.[21] The former is characterized by bothersome postprandial fullness and/or early satiation at least 3 days per week; the latter is characterized by bothersome epigastric pain or burning at least 1 day per week **[Figure 2]**.[21] In certain patients, the two subtypes may overlap.[22] Determining subtypes may be beneficial in management.

As per the American College of Gastroenterology and Canadian Association of Gastroenterology guidelines, endoscopy is not routinely required to diagnose functional dyspepsia and should be reserved for specific patient populations: symptomatic patients 60 years of age or older, with a lower threshold of 55 years of age or older in patients from Southeast Asia, given the higher prevalence of upper gastrointestinal malignancy in that population **[Figure 1]**.[23] In patients who are younger than 60 years of age, routine endoscopy is not recommended, even in the presence of alarm features **[Figure 1]**.[23] However, in patients from Southeast Asia who have a family history of upper gastrointestinal malignancy or a prominent alarm feature (e.g., rapidly progressive dysphagia, significant unintentional weight loss), the decision to use endoscopy should be individualized.[23]

Aside from endoscopy, diagnostics for functional dyspepsia are limited. All patients should undergo *H. pylori* testing, via either gastric biopsies during endoscopy for patients 60 years of age or older or noninvasive testing for patients younger than 60 years of age, with treatment and confirmation of eradication.[23] It is also prudent to rule out celiac disease by ordering anti–tissue transglutaminase serology. In patients with predominant symptoms of nausea and vomiting or significant early satiety and postprandial fullness refractory to therapy, gastroparesis should be considered.[24] To establish the diagnosis, a 4-hour solid phase gastric emptying study is recommended; however, upper endoscopy should be performed first to rule out structural causes.[24] The use of 2-hour studies is discouraged due to false negatives.[24]

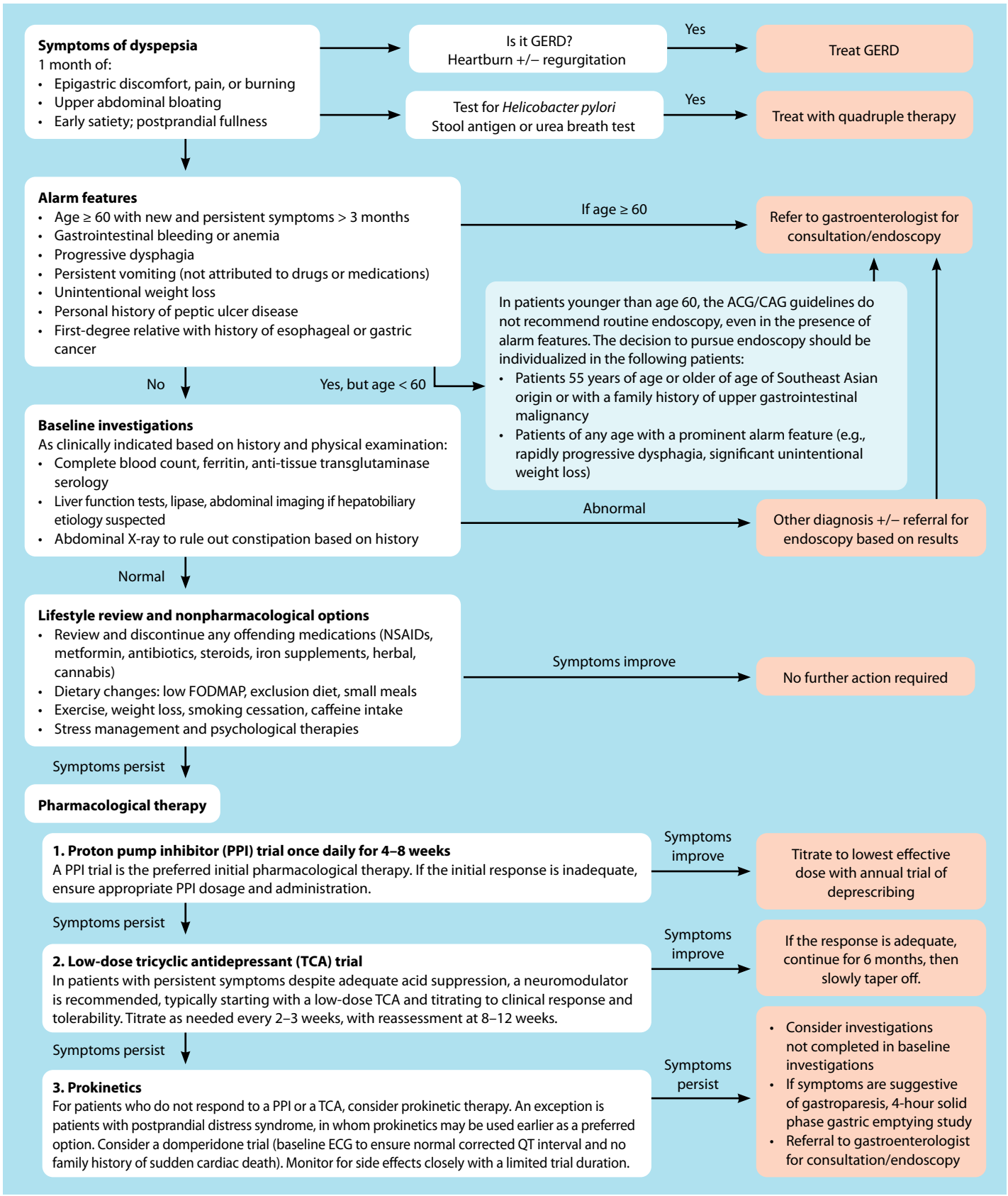

FIGURE 1. Functional dyspepsia primary care pathway. Adapted from Alberta Health Services.[40]

GERD = gastroesophageal reflux disease; ACG/CAG = American College of Gastroenterology/Canadian Association of Gastroenterology; TTG = tissue transglutaminase; LFT = liver function tests; FODMAP = fermentable oligosaccharides, disaccharides, monosaccharides, and polyols.

H. pylori testing

The American College of Gastroenterology and Canadian Association of Gastroenterology guidelines recommend noninvasive *H. pylori* testing in patients younger than 60 years of age who have dyspepsia, because it is a cost-effective strategy that minimizes unnecessary endoscopy[25] and reduces gastric cancer rates in Southeast Asian populations.[26] In patients who do not undergo endoscopic evaluation, noninvasive testing with the urea breath test or stool antigen testing is preferred;[27] the choice of modality is contingent upon local resource availability and patient preference. Serologic testing has lower specificity for acute infection and thus is not preferred. Patients should be off proton pump inhibitor therapy for 2 weeks prior to testing. If testing is positive for *H. pylori*, patients should be treated with quadruple therapy, and a confirmation of eradication test should be conducted after treatment.[25] We refer readers to the American College of Gastroenterology guideline for *H. pylori* treatment recommendations and pharmacotherapy regimens,[25] because this is outside the scope of this article.

Management

The approach to managing functional dyspepsia is multipronged and may involve nonpharmacological interventions, pharmacological treatments, and alternative pharmacological therapies. A stepwise approach is required **[Figure 1]**, often with the involvement of dietitians and psychologists.

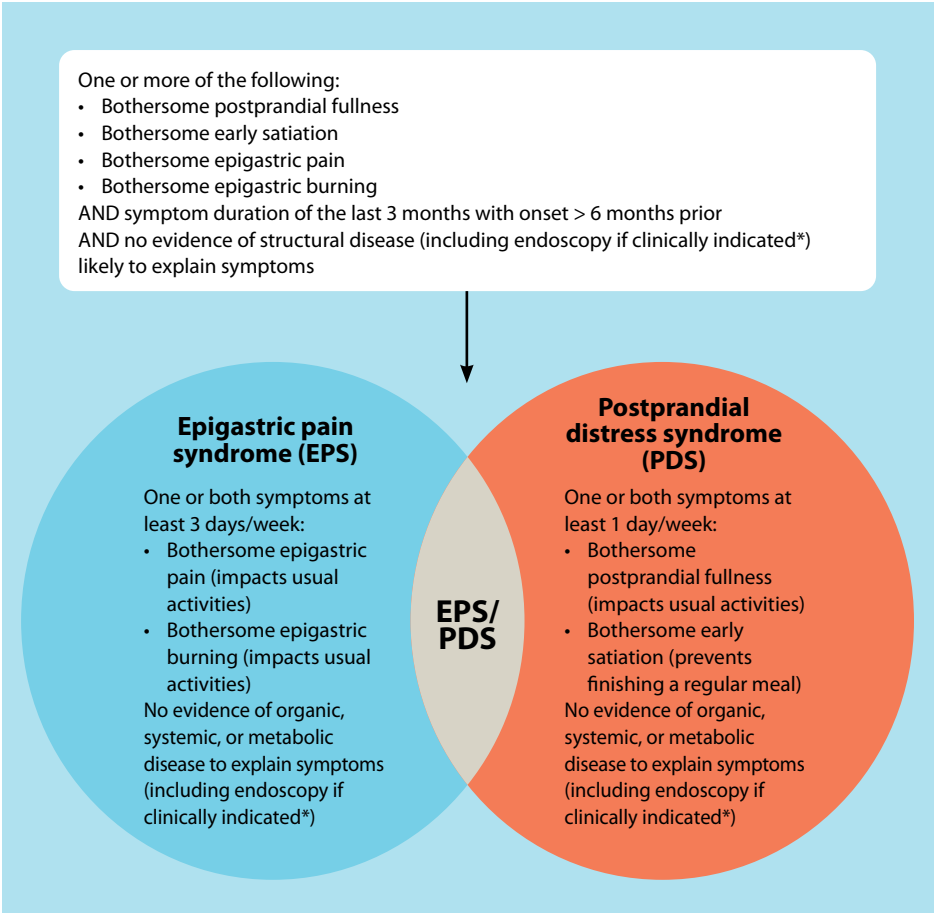

FIGURE 2. Rome IV criteria for functional dyspepsia. Adapted from the Rome IV criteria for functional dyspepsia, with supportive features for postprandial distress syndrome (PDS)/epigastric pain syndrome (EPS) excluded[21] and endoscopic investigation recommended only if clinically indicated as per the American College of Gastroenterology (ACG) and Canadian Association of Gastroenterology (CAG) guidelines.[23]

* As per the ACG/CAG guidelines, endoscopy is indicated in symptomatic patients 60 years of age or older; the decision for endoscopy should be individualized in patients 55 years of age or older of Southeast Asian descent and in patients of any age in whom an alarm feature is prominent.[23]

Nonpharmacologic therapies

Diet and exercise: Managing functional dyspepsia through dietary modifications should be considered first-line therapy prior to the use of pharmacological treatments. Patients whose diets are low in fermentable oligosaccharides, disaccharides, monosaccharides, and polyols (FODMAP) show symptomatic improvement, particularly those with postprandial distress syndrome.[28] Similarly, patients who follow a Mediterranean diet have shown an associated decrease in dyspeptic symptoms.[29] Conversely, alcohol, coffee, spicy food, and gluten are common triggers for functional dyspepsia[29] and, depending on individual patient response, should be avoided. Although the data are primarily from observational studies, small meals, low-FODMAP diets, and avoidance of trigger foods are generally recommended, and specific dietary choices (e.g., gluten-free, Mediterranean) should be individualized.[28,29]

Systematic reviews that evaluated the effectiveness of exercise—from aerobic exercise or walking to traditional Chinese exercises—found improvements in symptoms of epigastric fullness and pain, enhanced quality of life and sleep, and reduction in depressive symptoms;[30] however, many studies included concurrent pharmacological or psychological interventions.[30] Thus, while the benefit of exercise alone remains unclear, given the lack of harm, it is a reasonable first-line lifestyle intervention.

Other: Before initiating pharmacological interventions, the patient's medications and any associated side effects should be carefully reviewed. Medications commonly associated with functional dyspepsia symptoms, such as NSAIDs, steroids, metformin, glucagon-like peptide-1 agonists, antibiotics, and opioids, should be discontinued.[5] Recreational drug use, specifically cannabis, should be reviewed and discouraged. There is a lack of evidence that smoking cessation, weight management, and alcohol cessation improve functional

dyspepsia symptoms, but they are important general health measures.

Psychological therapies: While pharmacological therapies are the mainstay of treatment, a significant portion of patients may benefit from psychological therapy, given the coexisting mental health disorders.[23] While systematic reviews and randomized controlled trials have shown statistically significant benefits of psychological therapies,[31] including cognitive-behavioral therapy, meditation, gut-directed hypnosis, and relaxation therapy, the quality of the data is very low due to study heterogeneity, methodological limitations, and study bias.[23]

Pharmacological therapies

Acid suppression therapy: Given the presence of abnormal inflammatory cells, the immune response in the duodenum,[11] and hypersensitivity to gastric acid, acid suppression therapy is a recommended first-line treatment in functional dyspepsia.[25] While the use of proton pump inhibitors is preferred,[23] H2 receptor antagonists are reasonable alternatives if proton pump inhibitors are not tolerated or are ineffective; however, evidence suggesting their equal efficacy is limited by trial heterogeneity and misclassification of diagnoses.[23] Proton pump inhibitor dosing is recommended as once daily; further escalation is unlikely to confer benefit in the functional dyspepsia population.[23] Response should be reassessed in 8 weeks, and attempts to taper off or discontinue proton pump inhibitor therapy should be made every 6 to 12 months.[23]

Neuromodulators: In patients who do not respond to proton pump inhibitors or achieve only partial response, the use of neuromodulators is recommended.[23] Specific therapies include tricyclic antidepressants, which are preferred as first-line therapy, and mirtazapine.[23] Patients should be started at the lowest dose. The dose should be titrated up every 2 to 3 weeks as needed, then reassessed at 8 to 12 weeks.[32] If efficacious, tricyclic antidepressants should be continued for 6 months, then slowly tapered off; they can be re-initiated in recurrent dyspepsia at the previous lowest effective dose.[32] In patients who cannot tolerate tricyclic antidepressants or have concurrent weight loss, mirtazapine may improve both symptoms and early satiety.[23] Selective serotonin reuptake inhibitors have not shown any benefit, and there is currently no role for them in functional dyspepsia.[33]

Patients should be counseled that functional dyspepsia is a common condition that causes pain, discomfort, and/or a feeling of fullness in the upper belly, and they can feel bloated, full quickly, and nauseated.

Prokinetics: In cases where the above therapies have failed, prokinetic agents, such as metoclopramide and domperidone, are third-line options, but there is limited improvement in quality of life.[34] Consideration could be given to using prokinetic agents as first-line therapy in patients with postprandial distress syndrome subtype. Notably, prokinetic agents, particularly metoclopramide, have significant adverse effects, such as tardive dyskinesia, which affects up to 30% of patients.[23] Therefore, trial use with limited duration and close monitoring are advised. We recommend a baseline corrected QT interval prior to initiating therapy; cardiac arrhythmias and sudden cardiac death have been reported with the use of domperidone. Additional agents, such as buspirone, a 5-HT1A receptor agonist that causes fundal relaxation, have not shown significant improvement in dyspeptic symptoms and only modest improvement in bloating severity and early satiety;[35] however, they could be considered in patients with predominant postprandial distress syndrome subtype that have failed to respond to other therapies.

Other: Other pharmacological therapies, including gabapentin and rifaximin, as well as nonpharmacological therapies such as FDgard, a duodenal-release caraway oil/menthol formulation commonly used to treat irritable bowel syndrome, have shown some benefit in single trials or retrospective case series;[36-38] however, further study is needed before definitive conclusions can be drawn, and they are not currently recommended for the management of functional dyspepsia. Herbal medications, such as rikkunshito, artichoke leaf extract, and Zhizhu Kuanzhong, have been used traditionally to treat upper gastrointestinal symptoms. Symptomatic improvement has been demonstrated in controlled trials and systematic reviews, but concerns remain regarding toxicity, quality control, and dosage regulation; thus, there are no current recommendations for the use of these herbal medications in functional dyspepsia management.[39]

Patient counseling

Patients would benefit from counseling and validation from their providers, given that symptoms may be dismissed in the absence of structural disease. Patients should be counseled that functional dyspepsia is a common condition that causes pain, discomfort, and/or a feeling of fullness in the upper belly, and they can feel bloated, full quickly, and nauseated. This does not mean there is something structurally wrong with their stomach or intestines. Rather, due to the complexity of the gastrointestinal tract nervous system, visceral hypersensitivity and interplay of the gut microbiome, and gut–brain interaction, patients may experience dyspeptic symptoms. Their symptoms can also be due to infection, such as by *H. pylori*, and can be exacerbated by medications such as NSAIDs. Emotional and psychological cofactors such as anxiety and depression may also play a large role in symptomatology. Finally, diet can also play a role in symptom manifestation. Treatment includes dietary changes, regular exercise, and managing concurrent mental health disorders and may require a multidisciplinary approach with a dietitian and

psychologist. Treatment with medications includes acid reflux medications to calm down the lining of the gut, antidepressant medications that help regulate the gut–brain interaction, and prokinetics that stimulate gastric emptying. Opioids should be avoided, as they carry risk of dependence and tolerance and can further lead to narcotic bowel syndrome; they have no role in the management of functional dyspepsia. Management of functional dyspepsia may require trial of various therapeutic options, as what works for one person may not work for another, but with the right care, most people with functional dyspepsia can manage their symptoms successfully.

Clinical case follow-up

Case 1

Mohammad was misdiagnosed with gastroesophageal reflux disease and thus is nonresponsive to escalating doses of proton pump inhibitors. The lack of food triggers, association with meal times or positional changes, and localization to epigastric pain rather than typical retrosternal heartburn symptoms point away from gastroesophageal reflux disease. He meets Rome IV criteria for epigastric pain syndrome. Given his age, lack of alarm features, chronicity, and stability of symptoms, he does not require urgent upper endoscopy; a clinical diagnosis of functional dyspepsia can be made. He should be offered noninvasive *H. pylori* testing and treatment. If he is treated and his symptoms persist or the *H. pylori* test is negative, given his lack of response to proton pump inhibitor therapy, he should be started on low-dose tricyclic antidepressant therapy, such as amitriptyline, 10 to 15 mg by mouth every bedtime, or nortriptyline, 10 to 25 mg by mouth every bedtime, with reassessment at 8 to 12 weeks.

Case 2

Samantha meets Rome IV criteria for functional dyspepsia, favored postprandial distress syndrome. Given her abdominal exam and opioid use, an abdominal X-ray is performed, which shows constipation. She is treated with osmotic laxative polyethylene glycol. While this mildly improves her symptoms, her quality of life remains impaired. She is counseled on tapering off her opioids to avoid dependence and narcotic bowel and is started on tricyclic antidepressant therapy with nortriptyline, 10 mg by mouth every bedtime for 12 weeks, but shows little improvement.

Management of functional dyspepsia may require trial of various therapeutic options, as what works for one person may not work for another.

Given her intermittent nausea and vomiting, an upper endoscopy and 4-hour gastric emptying study (off opioids) are performed, which show mild delayed gastric emptying with 15% retention of a standardized meal at 4 hours. She is then started on domperidone, 10 mg by mouth three times a day, 30 minutes before meals, which significantly improves her symptoms. She is counseled on following a low-fibre, low-fat diet (gastroparesis diet) and is referred to a dietitian for further diet optimization. Her dose will be reassessed in 2 months.

Summary

In patients with functional dyspepsia, it is important to test for and treat *H. pylori*. Dietary modifications should be considered first-line therapy prior to the use of pharmacological treatments. However, the use of acid reflux medications can help by calming the lining of the gut. If the patient receives no relief, medications such as tricyclic antidepressants, which help regulate the gut–brain interaction and visceral hypersensitivity, should be considered. If symptoms are mainly postprandial bloating fullness, prokinetics that stimulate gastric emptying may be considered as first-line therapy. The use of opioids should be avoided because they are associated with the risk of tolerance and dependence and can further lead to narcotic bowel syndrome. They have no role in the management of functional dyspepsia. Various therapeutic options may be needed to manage functional dyspepsia, because what works for one patient may not work for another, but with the right care, most patients can manage their symptoms successfully. In addition, management may require a multidisciplinary approach that includes dietitians and psychologists. ■

Competing interests

None declared.

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Common pitfalls in the diagnosis and management of bacterial infections

A review of the literature to determine how clinicians may fall victim to cognitive traps when working through the diagnostic and therapeutic process, with guidance on how to avoid these pitfalls when managing adult patients with suspected infections.

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ABSTRACT: Diagnosing infectious diseases is often not straightforward. Clinicians may be misled by nonspecific or irrelevant symptoms and signs, and they may misinterpret test results. The appropriateness of the prescribed treatment hinges entirely on the accuracy of the working diagnosis. Even when the diagnosis is correct, pitfalls are frequently encountered during the management stage of the infection. This review of the literature shows how clinicians may fall victim to cognitive traps when working through the diagnostic and therapeutic process and provides guidance on how to avoid these pitfalls when managing adult patients with suspected infections.

Infections are one of the most common conditions encountered in both inpatient and outpatient settings, with infections of the respiratory tract, urinary tract, and integumentary system frequently diagnosed.[1-3] Proving the presence of an infection is not usually possible in routine clinical practice, because a biopsy of the affected tissue or organ is needed to demonstrate histological invasion and the destruction of host cells by pathogens. Therefore, medical practitioners rely on history, physical exam findings, and laboratory tests to determine whether an infection might exist. This data-gathering process is akin to the work of a detective putting together pieces of the puzzle to solve a diagnostic mystery. Sometimes the puzzle pieces don't fit perfectly with each other, irrelevant or distracting information clouds the clinical picture, or not all of the data are available for a clinician to be 100% confident about the diagnosis. The threshold at which diagnostic certainty impacts a clinical decision varies from person to person. Some might be comfortable proceeding with treatment when they are only 50% confident about the diagnosis, while others might need more certainty before doing so. Unlike the diagnosis of malignancies, where tissue biopsy (the gold standard) is required to confirm the disease before prescribing chemotherapy, the diagnosis of common bacterial infections is typically made without confirmation when antibiotic treatment is initiated.

If we cannot prove with a tissue biopsy that an infection is present, how often do we get the diagnosis wrong? And, consequently, how often are we mistreating patients with antibiotics? I will consider these questions using three common infections as examples: respiratory tract infections, urinary tract infections, and skin and soft tissue infections. These examples and the studies referenced apply to adult patients only.

Respiratory tract infections

Consider this scenario: "A previously healthy 35-year-old woman who smokes tobacco presents with 5 days of fatigue, productive cough, worsening shortness of breath, temperatures to 38.9 °C, and decreased breath sounds in the lower right field. She has a heart rate of 105 beats/minute, but vital signs are otherwise normal."[4] What is the probability that she has pneumonia? In this survey study (N = 553), nearly all respondents overestimated the probability at a median of 80%,[4] while the evidence-based answer is roughly 30% using the Heckerling diagnostic score **[Figure]**, assuming the pretest probability of pneumonia is 5% for a patient with a cough.[5,6] What is the probability if the chest X-ray is either positive or negative? The median answers were 95% and 50%, respectively, while the evidence-based estimates are roughly 55% and 15%, respectively.[4]

In all scenarios, respondents grossly overestimated the probability of pneumonia, uncovering a fundamental weakness with diagnostic reasoning and test interpretation. Due to the presence of cognitive biases that

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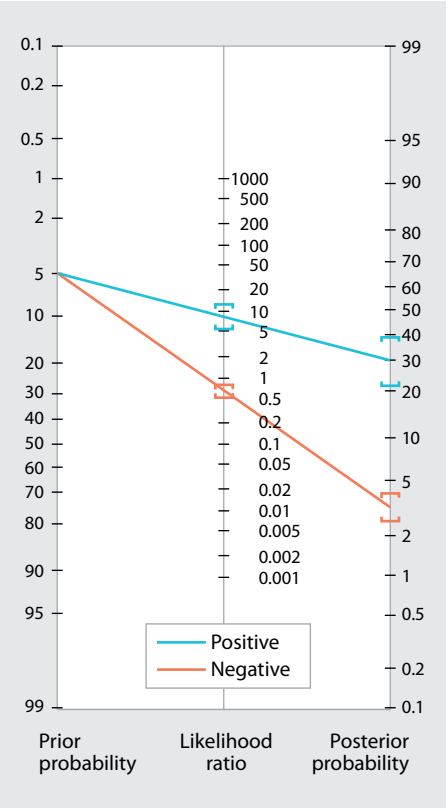

FIGURE. Likelihood nomogram showing the pretest (prior) and posttest (posterior) probability of pneumonia with 4 or 5 Heckerling diagnostic criteria met (positive) versus less than 4 criteria met (negative). Source: Schwartz A. Diagnostic test calculator [free software available under a Clarified Artistic License]. http://araw.mede.uic.edu/cgi-bin/testcalc.pl.

can impair probabilistic and statistical reasoning, including base rate neglect, anchoring bias, and confirmation bias, humans have a tendency to overestimate, which results in diagnostic errors.[4] Overdiagnosis subsequently leads to overtreatment.

The most common tool we depend on to diagnose pneumonia is chest X-ray, but how reliable is it? Unfortunately, its sensitivity and specificity are mediocre, at 69% and 78%, respectively.[7] Given the inter-observer variability in physical exam findings and the imperfect accuracy of chest X-ray, it is not surprising that pneumonia is often misdiagnosed, with 34% of outpatients being labeled with it when they don't have it.[8]

Antibiotic treatment for pneumonia is also prone to errors. In one study of outpatients with community-acquired pneumonia (N = 341), only 31% received a guideline-concordant regimen.[8] Treatments were deemed inappropriate due to the wrong choice of drug (77%) and/or the wrong duration—almost always too long (39%). Lack of awareness about the microbial etiology of pneumonia might be tied to selection of the wrong drug. According to data from the United States, the most frequently detected pathogens in community-acquired pneumonia are viruses (22%), followed by bacteria (11%).[9] Because differentiating between viral and bacterial pneumonia can be challenging, I wonder what proportion of viral infections are being treated inappropriately with antibiotics.

It is also important to recognize the limitations of microbiological testing. A nasopharyngeal swab is performed to detect viruses, while a sputum culture is sometimes obtained to identify bacteria. Caution must be exercised when interpreting a positive result. Viral testing employs a nucleic acid amplification technique, which cannot distinguish between viable and dead organisms.[10] Moreover, identifying a virus from the nasopharynx does not necessarily prove causation of pneumonia or exclude a bacterial infection. Sputum cultures also have limited utility, because isolating the culprit bacteria from a nonsterile site is difficult. Further complicating the matter, for positive sputum cultures, determining whether the organism is pathogenic or simply a colonizer is problematic.[10] For these reasons, guidelines recommend against obtaining routine sputum cultures in the management of community-acquired pneumonia, except in patients hospitalized with severe infection or if there is concern for drug-resistant pathogens.[11] However, the isolation of certain organisms, including some atypical bacteria (e.g., *Nocardia*), fungi (e.g., *Cryptococcus*), and mycobacteria (e.g., *Mycobacterium tuberculosis*), is almost always considered pathogenic and warrants further investigation and treatment.

Urinary tract infections

Let's start with a clinical case. "A 65-year-old man is seen for osteoarthritis. He has noted foul-smelling urine but no pain or difficulty with urination. A urine dipstick shows trace blood."[4] What is the probability of a urinary tract infection (UTI)? In this survey, the median estimated pretest probability by participants was 20%.[4] If a urine culture is either positive or negative, respondents predicted a median chance of UTI of 80% and 5%, respectively.[4] Foul-smelling urine does not count as a UTI symptom, and, by definition, asymptomatic bacteriuria is not a UTI, so the evidence-based probability is essentially 0% regardless of the urine culture result. Not only did clinicians overcall the probability, but the impact of a positive urine culture on their estimate was also alarmingly high.

It is intriguing that medical practitioners have such strong faith in a positive urine culture as robust evidence for a UTI. It appears that the concept of asymptomatic bacteriuria is poorly understood, as evidenced by studies that found that 71% of respondents would prescribe unnecessary antibiotic for this condition[12] and 83% of hospitalized patients with asymptomatic bacteriuria received inappropriate antibiotic.[13]

Dysuria, urinary frequency, hematuria, back or lower abdominal pain, and absence of vaginal discharge or irritation are the only evidence-based UTI symptoms.[14] An exception is the isolation of *Staphylococcus aureus* from urine culture, which might be indicative of *S. aureus* bacteremia seeding the urinary tract. Some experts recommend obtaining blood cultures even if asymptomatic to exclude an occult bacteremia in this setting.[15] Contrary to popular belief, altered mental status and foul-smelling or cloudy urine are not true UTI symptoms.[16]

Interpreting urine tests can also be tricky. The absence of pyuria strongly argues against a UTI, while the presence of nitrite and/or leukocyte esterase is not specific for infection.[17] Misdiagnosis of UTIs is high. In one study (N = 264), 66% of females presenting to the emergency department with genitourinary symptoms were diagnosed with a UTI, but only half were correctly diagnosed.[18] Some patients tested positive

for a sexually transmitted infection instead. Diagnosing UTIs in elderly patients is even more challenging, because up to 50% of females and 40% of males in long-term care have asymptomatic bacteriuria, and often pyuria as well.[17] Urine testing in this population is less helpful in diagnosing UTIs, as abnormal urine results can be expected. Thus, a urine sample should be collected only when true UTI symptoms are present, as recommended by recent guidelines.[16,19] A common indication to collect a urine sample is delirium, but other causes, such as dehydration, drug side effects, and sleep disturbances, should be ruled out before attributing the altered mental state to a UTI.[16] Regarding UTI treatment, one study found that about half of outpatient antibiotic prescriptions were consistent with guideline-recommended first-line therapy, while over 75% of prescribed durations were too long.[20]

Skin and soft tissue infections

Cellulitis and erysipelas are common bacterial infections of the skin. There is no single piece of history, physical exam finding, or lab test that can confirm the diagnosis with a high degree of accuracy.[21] Aside from a skin biopsy, which is not routinely performed, there is no way to prove that the skin or subcutaneous tissues are infected. As a result, the clinical gold standard is history and physical exam, but can any clinician provide the gold standard in diagnosing skin infections? In a randomized controlled trial (N = 175) examining the accuracy of cellulitis diagnoses where a dermatology consult was considered the gold standard, of all patients admitted to hospital with cellulitis, 30% had an alternative dermatological diagnosis such as eczema or dermatitis.[22] Patients treated by dermatologists had more rapid clinical improvement and reduced exposure to antibiotics. Similarly, in another randomized controlled trial (N = 29) of outpatients who were labeled by their primary care provider as having cellulitis, only 5 (17%) were judged to have been correctly diagnosed according to a dermatologist.[23]

Skin infections can be challenging to diagnose because there are many mimickers, also known as pseudocellulitis conditions. These include venous stasis dermatitis, contact dermatitis, eczema, deep vein thrombosis, gout, hematoma, erythema migrans, and peripheral artery disease.[21,22] When an infection is present, there are only two major pathogens to consider: beta-hemolytic streptococcus in nonpurulent infections and *Staphylococcus aureus* in purulent disease.[21,24] Coverage for Gram-negative organisms is indicated only in select circumstances, including moderate to severe diabetic foot infections, perineal infections, animal bite infections, aquatic-related injury, surgical site infections of the groin or axilla, severely immunocompromised patients, and necrotizing infections.[21,25]

It is expected that the choice of antibiotic should reflect the microbiological understanding of this disease. However, studies have shown that almost half of hospitalized patients receive unnecessary broad-spectrum antibiotic (most commonly piperacillin-tazobactam), vancomycin use is inappropriate in 75% of cases, up to half of patients who are eligible for oral antibiotic receive IV treatment longer than required, and over half of patients are treated longer than the guideline recommendation of 5 to 7 days.[22,26-28] When treating cellulitis, it is paramount to understand its natural progression to avoid treatment mishaps. Some clinicians might broaden the antibiotic or switch to IV therapy unnecessarily when they judge that the initial oral treatment is failing. The symptoms and signs of inflammation can worsen in the first 1 to 2 days while on effective therapy, and it can take up to 3 days before a clinical response is observed.[29] Misdiagnosing a treatment failure too early in the course of illness results in inappropriate modifications to treatment and might lead clinicians to falsely attribute the improvement to the new therapy.[30] Furthermore, clinicians often misinterpret persistent symptoms and signs of inflammation of the affected limb as evidence for ongoing infection and, as a result, extend the antibiotic treatment beyond the guideline-recommended duration. Residual inflammation at the end of treatment is normal in up to 60% of patients, and prolonging the course of antibiotic is not useful, as the bacteria are likely dead by that point.[29] Tissue repair and immune activation can persist well after the bacteria have been killed.

Conclusions

Pneumonia, urinary tract infections, and cellulitis in adult patients tend to be overdiagnosed and overtreated. Potential reasons include inadequate training in and modeling of diagnostic reasoning, the presence of cognitive biases, cultural normalization of antibiotic overuse, lack of awareness of recent evidence and guidelines, and clinical inertia. A positive test does not diagnose a disease; it is the clinician interpreting the relevant data in the right clinical context who does. Answers in medicine are rarely black and white, and operating in the grey zone demands a reasonable understanding of probabilistic rationalization and an ability to incorporate best evidence into clinical decision making. A rational approach is for clinicians to critically appraise their knowledge and skills to continually refine their competency, and thereby deliver the right amount and type of care for each patient. ■

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Tuberculosis diagnostics in the 21st century

Tuberculosis (TB) diagnostics relied on clinical acumen until the late 19th century, when microscopy and culture were developed. This was followed by susceptibility testing, which was developed in parallel to antibiotic therapy. Recently, molecular techniques have been changing centuries-old methods **[Figure 1]**. Yet, TB remains the most common cause of infectious mortality, with close to 1.25 million deaths worldwide in 2024.[1] New laboratory techniques are key to reaching the elimination goal of less than 1 person per million with TB globally by 2050.[2]

Molecular techniques provide faster and more accurate information about TB, including direct detection and sequencing for epidemiology and susceptibility prediction. This year marks a turning point in TB diagnostics. As of 5 January 2026, most susceptibility testing in British Columbia will rely on genomic prediction rather than phenotypic. This change speeds up the availability of results from 21 days to as few as 7 days following culture positivity.

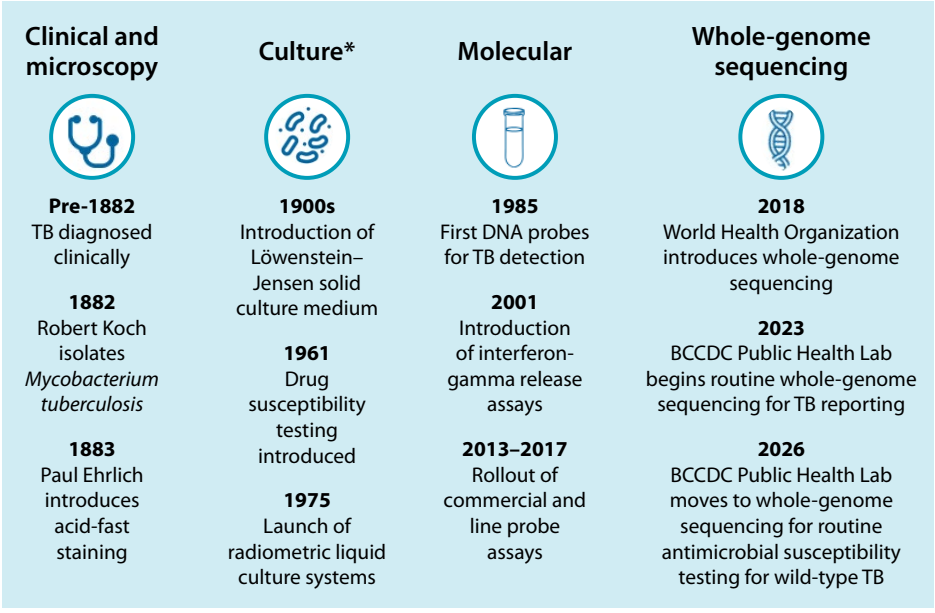

FIGURE 1. Timeline of tuberculosis diagnostics.
* Culture for TB was developed in the late 1800s but applied for clinical use in the 1900s, with widespread use starting in the 1930s, with standardized protocols.

Highlights of recent TB diagnostic developments

Direct detection by polymerase chain reaction

Polymerase chain reaction (PCR) was introduced in the 1980s and has been used widely for TB diagnostics since the early 2000s. This marked the first time it was possible to confirm TB without relying on culture. Early PCR had variable sensitivity, often lower than that of standard microscopy. In the early 2010s, improved sensitivity allowed for reliable and reproducible detection of TB from sputum. New GeneXpert testing reduces complexity and speeds turnaround time, enabling clinical teams to quickly be informed of positive specimens. The most notable advance in TB PCR diagnostics in the last 10 years is resistance detection for the antibiotics isoniazid and rifampin. These tests can help identify patients with multidrug-resistant TB to rapidly optimize treatment and reduce the risk of onward transmission.

Next-generation sequencing to identify nontuberculous mycobacteria species

While TB is the primary target of detection and treatment programs, nontuberculous mycobacteria (NTM) continue to cause disease and confuse TB diagnosis. Historic identification and differentiation of these organisms have relied on phenotypic and biochemical behavior, taking weeks to months. Molecular advances, particularly next-generation sequencing, enable the use of sequenced regions to classify NTM within 2 to 3 days of growth, often to the level of subspecies or variants. This technique can also identify TB and mixed infections, thereby avoiding the need for complex procedures to separate mixed bacterial populations. The assay used at the BCCDC Public Health Laboratory also identifies common resistance mutations. Rapid and accurate identification of NTM species allows TB to be ruled out and enables decisions about the clinical significance of the organism for the patient.

Whole-genome sequencing

Since entering clinical use in 2009, whole-genome sequencing (WGS) has transformed medicine. Use for TB diagnostics began in 2012 to support cluster

This article is the opinion of the BC Centre for Disease Control and has not been peer reviewed by the BCMJ *Editorial Board.*

Sample	1	2	3	4	5	6	7	8	9
1		0	59	66	66	66	64	65	65
2	0		59	66	66	66	64	65	65
3	59	59		67	67	67	64	66	66
4	66	66	67		0	0	72	73	73
5	66	66	67	0		0	72	73	73
6	66	66	67	0	0		72	73	73
7	64	64	65	73	72	72		1	1
8	65	65	66	73	73	73	1		2
9	65	65	66	73	73	73	1	2	
10	65	65	66	73	73	73	1	2	0

FIGURE 2. Example of whole-genome sequencing data to show individual single-nucleotide differences between isolates (the darker border indicates closely related isolates).

investigations, replacing older methods that relied on repeats and other short genetic sequences and lacked granularity. WGS allows the identification of single-nucleotide differences between strains, which can identify transmission events and uncover previously undetected transmission networks **[Figure 2]**. WGS was first used in a research capacity in BC in 2018 and was transitioned to regular clinical application in 2023—the BCCDC Public Health Laboratory was the first provincial laboratory to rely on WGS results. We now have over 2000 isolates sequenced, providing a rich data set to understand epidemiology and confirm accuracy. Techniques and workflows are continually refined to minimize time to definitive results and support traditional epidemiology by confirming cases and clusters for further investigation.

In addition, WGS offers the ability to predict susceptibility. Since 2018, the World Health Organization has collated and published resistance mutations, permitting bioinformatics teams to link sequences to phenotypic behavior. The advantages of WGS are that results can be made available in several days to weeks, and multiple antibiotics can be assessed with a single test. The BCCDC Public Health Laboratory has validated the first-line agents compared with phenotypic methods, finding excellent concordance. Negative predictive values for wild-type TB to first-line medications are 99.5% for isoniazid, 99.6% for pyrazinamide, 100% for ethambutol, and 100% for rifampin. Fully susceptible organisms are now reported with confidence within 10 days of detection, an improvement of 10 to 14 days.

All these advances rely on a large team of individuals with advanced skills—laboratory personnel, bioinformaticians, informatics specialists, front-line clinicians, epidemiologists, and many others all play key roles. We are grateful for the wonderful team working together to eliminate TB. ■

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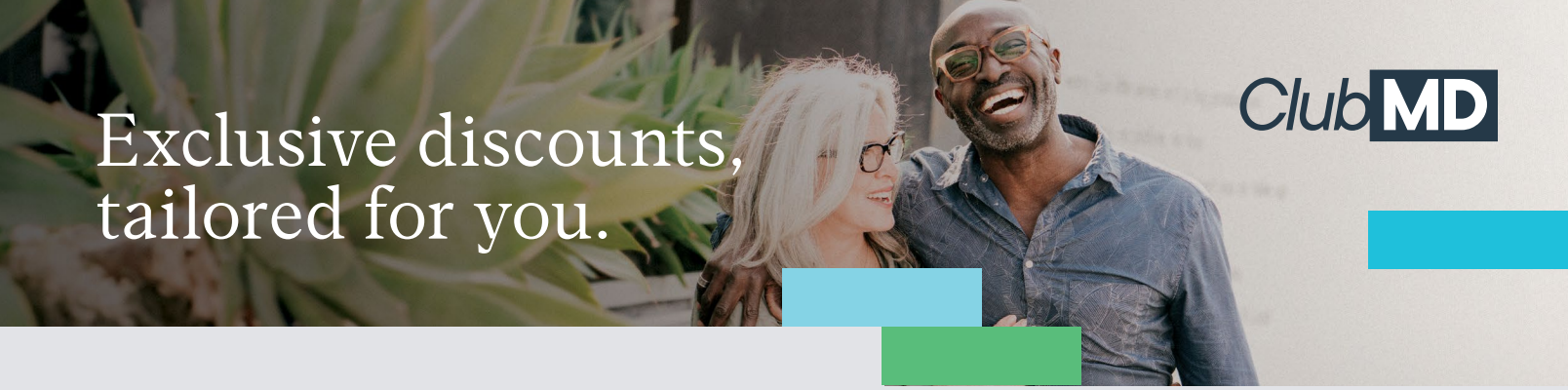

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