

BCMj

A Doctors of BC Publication

CASE REPORT Cryptogenic multifocal ulcerous stenosing enteritis presenting as refractory iron deficiency anemia

ALSO IN THIS ISSUE

CASE REPORT: Serotonin syndrome presenting as posterior reversible encephalopathy syndrome in the setting of combined selective serotonin reuptake inhibitors and attention-deficit/hyperactivity disorder therapy



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Clinicians managing patients with attention-deficit/hyperactivity disorder should be vigilant about the underrecognized risk factor of polypharmacy in the development of serotonin syndrome. "Serotonin syndrome presenting as posterior reversible encephalopathy syndrome in the setting of combined selective serotonin reuptake inhibitors and attention-deficit/hyperactivity disorder therapy" begins on page 280.

269 Editorials

- Nicotine pouches:
Harm reduction or a new
generation of nicotine users?
Caitlin Dunne, MD
- *Alhghob*: A new online course
advancing Indigenous cultural
safety in primary care
Terri Aldred, MD

271 Letters

- Re: Primary care is the focus of
Simon Fraser University's new
School of Medicine
Jane McKay, MD

- Re: Primary care is the focus of
Simon Fraser University's new
School of Medicine
Paul Hertz, MD
Stephen Tsai, MD
- Author replies
David Price, MD
- Bad booking protocol
Ben R. Wilkinson, MBBS
- Re: Bad booking protocol
Inderveer Mahal, MD

273 President's Comment

The social contract: Defining how
we show up for society
Adam Thompson, MD

274 Premise

Heart failure tool kit for
BC prescribers: Initiation and
titration of medication
Michael J. Diamant, MD
Nathaniel M. Hawkins, MBChB
Valerie Barrette, RN
Rozina Chaube, RN
Fritha Munday, MPH
Kelly Mackay, RN

276 BCMD2B

Practical tips for interpreting
blood culture results
Polina Novoseltseva, MSc
Eugene Y.H. Yeung, MD

Contents continued on page 268

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For Doctors of BC, acknowledging the traditional territories is an expression of cultural humility that involves recognizing our
commitment to support the provision of culturally safe care to Indigenous Peoples in BC.

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ON THE COVER

Cryptogenic multifocal ulcerous stenosing enteritis is rare and challenging to diagnose but should be considered in patients with unexplained chronic anemia and small bowel obstructive symptoms. Article begins on page 285.

Mission: The *BCMj* is a general medical journal that shares knowledge while building connections among BC physicians.

Vision: The *BCMj* is an independent and inclusive forum to communicate ideas, inspiring excellent health care in British Columbia.

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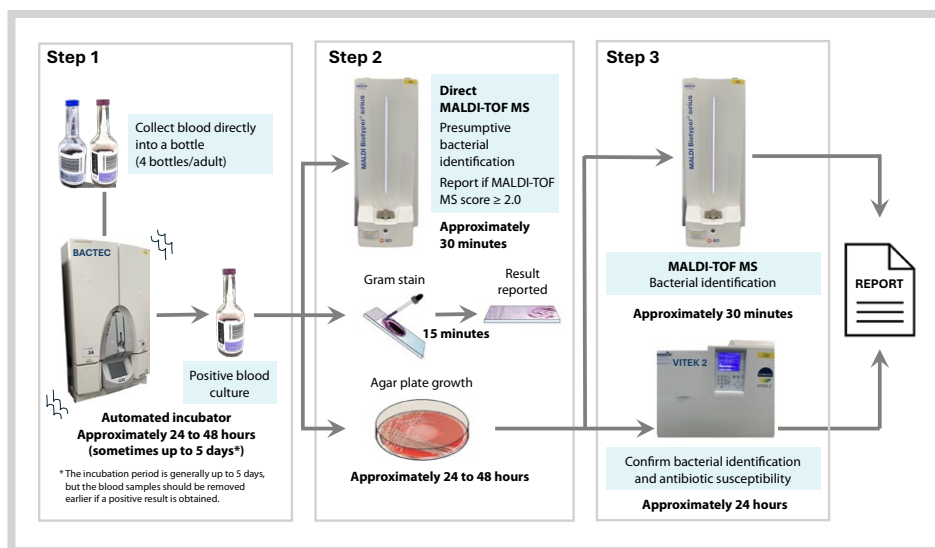
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of bc**



Adult blood culture typical laboratory protocol and workflow. "Practical tips for interpreting blood culture results" begins on page 247. (MALDI-TOF MS = matrix-assisted laser desorption/ionization time-of-flight mass spectrometry)

Contents continued from page 267

CLINICAL

CASE REPORT

- 280 Serotonin syndrome presenting as posterior reversible encephalopathy syndrome in the setting of combined selective serotonin reuptake inhibitors and attention-deficit/hyperactivity disorder therapy**
Nicole Hawe, MD
Lori Wik, MD

CASE REPORT

- 285 Cryptogenic multifocal ulcerous stenosing enteritis presenting as refractory iron deficiency anemia**
Jeraldine Chan, MD
Hui-Min Yang, MD
Diane Villanyi, MD
Lik Hang Lee, MD

289 WorkSafeBC

A rapid overview of occupational diseases from welding: Tips for family physicians
Kieran Steer, MD,
Jeanette Campbell, MSc(A)

290 BCCDC

How physicians can help improve declining childhood immunization coverage rates in BC
Elaine Chan, MPH
Michelle Takeuchi, RN
Jia Hu, MD

292 NEWS

- Doctors of BC 2026 scholarship winners
- Spotlight: Indigenous Physicians Association of Canada
Ellen Tannam

294 Obituaries

- Dr Anthony "Hugh" Robinson
- Dr John Law

297 Classifieds

Nicotine pouches: Harm reduction or a new generation of nicotine users?

My preteen son came home from summer day camp and told me his counselor had given the group two rules: “Be on time” and “Don’t ask about my mints.” Naturally, he was very curious to know what these mints were and why they were so important.

The “mints” were nicotine pouches: small, discreet sachets tucked between the upper lip and gum, where saliva releases nicotine for absorption by the oral mucosa. Unlike traditional smokeless tobacco products, such as chewing tobacco, nicotine pouches contain no tobacco leaf; their nicotine is either extracted from tobacco or produced synthetically. They may also contain flavorings, bulking agents, humectants, and pH-altering agents to facilitate absorption. Because they produce no smoke, vapor, or odor and require no spitting, they can be used almost anywhere, often without anyone noticing.¹

In Canada, because pouches do not contain tobacco and are considered nicotine replacement therapy, they fall under the Food and Drugs Act rather than the Tobacco and Vaping Products Act, a regulatory distinction that Health Canada has recognized it may need to change.² Mint-flavored Zonnic (a product of Imperial Tobacco) can be legally sold in Canada by pharmacies to people 18 years of age and older for nicotine replacement therapy, and it must have a natural product number. Yet, unauthorized nicotine pouches are readily available online and can be found in convenience stores and gas stations. These grey-market products may contain higher concentrations of nicotine, as well as flavors and additional ingredients not on the label.³

As tobacco use among young people continues to decline, nicotine pouches are

moving in the opposite direction.⁴ In the United States, the 2025 National Youth Tobacco Survey found that overall youth tobacco use had fallen to 7.2%.⁴ E-cigarettes remained the most commonly used product (5.2%), followed by nicotine pouches, which had more than doubled in prevalence over the past 4 years (0.8% in 2021 vs 1.7% in 2025).

JAMA published similarly concerning numbers from the Monitoring the Future study of US students in grades 10 and 12.⁵ Between 2023 and 2024, past-30-day use of nicotine pouches doubled from 1.3% to 2.6%, while lifetime use rose from 3% to 5.4%. Despite declining e-cigarette use, dual use of e-cigarettes and pouches increased.⁵ Canadian data tell a similar story. In a survey of nearly 14 000 Quebec secondary-school students participating in the COMPASS study, 2.6% reported using nicotine pouches in the past month, and nearly one-quarter were unsure about the health risks.⁶

The appeal to youth is not difficult to understand. Nicotine pouches are discreet, easy to conceal, and available in appealing flavors, and they can be used without obvious signs of smoking or vaping. But while they may represent a less harmful source of nicotine for adults who would otherwise smoke, they are not harmless—and nicotine is highly addictive.⁷

The World Health Organization published a report in May 2026 examining how nicotine pouches are marketed and how countries might regulate them.⁸ A *JAMA* article captured the WHO’s concern in a commentary: “Fruity flavours, festivals, and F1—WHO report reveals how nicotine pouches hook consumers.”⁷ Around the same time, in June 2026, the US Food

and Drug Administration authorized reduced-risk marketing claims for some nicotine pouches. Can both perspectives be true? Can nicotine pouches be a less harmful alternative for adults who smoke while simultaneously being perilously attractive to young people who might never have used nicotine otherwise?

The evidence that nicotine pouches help people quit smoking is surprisingly limited. A 2025 Cochrane review included only four small studies and concluded that there was “very uncertain” evidence that nicotine pouches led to slightly higher smoking abstinence compared with no intervention.⁹ Evidence about long-term health effects is also limited. The American Heart Association cautioned that smokeless oral nicotine products may negatively affect some biomarkers of cardiovascular disease risk and could increase the risk of oral cancers.¹⁰

This leaves physicians navigating a nuanced but important distinction. For an adult who smokes and cannot quit, switching from combustible tobacco to nicotine pouches may reduce exposure to harmful chemicals. But for a young person who does not use nicotine, we certainly would not recommend starting. The individual-level harm reduction message and the population-level prevention message are not contradictory, but they can become confused.

Perhaps this is a lesson we should have learned from vaping: “reduced risk” does not mean “safe.” As physicians, we may be well positioned to counsel patients on this distinction. But as a parent, making nicotine tastier, quieter, and easier to conceal scares me. I will *definitely* be asking about the “mints.” ■

—Caitlin Dunne, MD, FRCSC

References on page 275

Alhgoḥ: A new online course advancing Indigenous cultural safety in primary care

I have participated in and supported Indigenous health initiatives for the past 20 years, focusing on increasing representation of Indigenous people in health professions and increasing awareness of Indigenous health among non-Indigenous professionals. I was driven to advance Indigenous health—and later cultural safety and Indigenous-specific anti-racism initiatives—because I grew up both in the inner city and on reserve experiencing the impacts of segregation and isolation. These impacts on health included lack of access, unsafe care, and poorer outcomes. I was driven to address the disparities faced by my community by becoming a physician; however, I quickly realized that to have a wider-reaching impact, I would need to support broader system change.

In the past few years, British Columbia has formally recognized Indigenous-specific racism and implemented new regulatory standards for Indigenous cultural safety, cultural humility, and anti-racism. This has included many health organizations, including Doctors of BC, health authorities, and the College of Physicians and Surgeons of BC (CPSBC), signing declarations of commitment to cultural safety; publishing the *In Plain Sight* report; developing the Health Standards Organization's *British Columbia Cultural Safety and Humility Standard*; and publishing the CPSBC's *Cultural Safety, Cultural Humility and Anti-Racism* practice standard.¹ This foundational regulatory framework has supported the shift from acknowledging harms experienced by Indigenous people in accessing care to implementing changes to address them.

This shift became evident when I started receiving feedback from physicians

wanting to understand what this looks like at the bedside and in their practices. Their college standards called for them to be self-reflective, anti-racist, relational, and trauma informed, but many struggled with where and how to start. In my work in primary and community care, there seemed to be an even wider gap, with limited support available, few facilitators, and increasing pressures on clinicians and staff. Acknowledging this gap, in 2021, primary care teams from the First Nations Health Authority and the Ministry of Health developed a working group with representatives from across the system, including Doctors of BC, the Ministry of Health, the regulatory colleges, and leaders in cultural safety. The group identified the need for more clinical tools and skills to translate the standard into practice as the number one priority. The tools would need to be practical, accessible, and relevant to everyday scenarios that clinicians may encounter.

With this clarity of purpose, the group developed a proposal that led to the Cultural Safety, Humility, and Indigenous-Specific Anti-Racism Provincial Primary Care Project. In discussions with other cultural safety and anti-racism clinician educators, it became apparent that we were often presenting the same topics to similar groups. We determined that an asynchronous introductory course for clinicians would help expand the reach of this education. The target audience would be primary and community care clinicians, particularly those prescribing treatments. We thought it would also be helpful for those new to practice or British Columbia. The course would need to be open-access, free, and accredited to minimize barriers for busy

clinicians. It would be rooted in translating practice standards into actionable bedside teaching using case studies and reflective exercises. The course would also provide resources and next steps to encourage clinicians to continue their cultural humility and anti-racist journeys.

After almost 2 years of development, the online course, *Alhgoḥ Together as One, Together as Community: Pathway to Indigenous Cultural Safety*, was released through the UBC CPD eLearning platform on 21 June 2026 in recognition of National Indigenous Peoples Day.² *Alhgoḥ* is a Dakelh word from the Lheidli T'enneh traditional territory, which encompasses Prince George, where the idea and development of the course began. *Alhgoḥ* represents the personal and collective responsibility we have as medical professionals to provide culturally safe, respectful, and anti-racist care. To learn more, please visit the course registration page.³

Providing leadership and content for this course has deepened my own understanding of the journey toward a safer, more inclusive health care system. I was able to work with a dedicated and inspiring team committed to promoting social justice in a way that heals and connects. The next step for the project team is to develop microlearnings that build on key concepts highlighted in the course and support the development of a provincial community of practice. There is no destination with this work; it is an ongoing journey, and I'm incredibly grateful and proud to be part of it. ■

—Terri Aldred, MD

References on page 275

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: Primary care is the focus of Simon Fraser University's new School of Medicine

Congratulations to Simon Fraser University on the opening of the Stephens Family School of Medicine. I am writing to seek clarity on a statement made by the dean of medicine, Dr David Price: "We're starting with family medicine residencies, but our commitment is to increase to a number of other primary care residency specialties, like pediatrics, psychiatry, and general internal medicine" [*BCMJ* 2026;68:196-198].

As a general internist, it is a surprise to see general internal medicine considered a primary care residency specialty.

I witnessed the launch of general internal medicine as a subspecialty in my career. The subspecialty serves both the acute and chronic internal medicine care needs of many adults in British Columbia and throughout Canada. It is a growing field that will continue to provide excellence in care over time.

Many individuals struggle to access specialty care, and our specialty is no different. I don't like having a 2-year wait list, but it serves as a reflection of the incredible need for specialized care. Transferring the role of primary care to already overburdened specialties needs review and consultation. We all realize the desperate need for primary care and hope the Stephens Family School of Medicine will be a move forward in educating our future primary care experts. I can comment only on my own area—providing primary care along with specialty service would not be realistic and would drastically increase my already-too-long wait list.

—Jane McKay, MD, FRCPC

Clinical Associate Professor, UBC Division of General Internal Medicine

Re: Primary care is the focus of Simon Fraser University's new School of Medicine

As presidents of the Section of General Internal Medicine and the BC Pediatrics Society, respectively, we would like to congratulate Dr David Price and his colleagues on the exciting progress toward opening Simon Fraser University's new Stephens Family School of Medicine. We also warmly welcome the inaugural class of medical students and look forward to working with them as they begin their careers.

However, we believe it is important to clarify one point. While pediatricians and general internal medicine physicians care for patients with complex, often undifferentiated medical conditions, these are not primary care specialties, as described by Dr Price [*BCMJ* 2026;68:196-198] and in the Stephens Family School of Medicine's mission and vision statements. General internists and pediatricians are consultant specialists who provide referral-based care after 4 to 5 years of postgraduate specialty training.

This distinction is more than semantic. Accurate understanding of general internal medicine and pediatrics in BC (and across Canada) has implications not only for patients and learners but also for physician workforce planning, remuneration, credentialing, licensure, and recognition of internationally trained physicians. As health care systems increasingly recruit physicians from different training backgrounds, precise terminology is essential to ensure that physicians are recognized for the roles and expertise for which they have been trained.

We are grateful that SFU has recognized the value of general internal medicine and pediatrics and the need to strengthen

these chronically under-resourced specialties. As the new medical school takes shape, we look forward to helping educate the next generation of physicians and helping ensure that learners understand the unique and essential role that pediatrics and general internal medicine play in British Columbia's health care system.

—Paul Hertz, MD, FRCPC

President, Section of General Internal Medicine

—Stephen Tsai, MD, FRCPC

President, BC Pediatric Society

Author replies

Thank you to Drs McKay, Hertz, and Tsai for drawing attention to an important distinction in terminology in my recent interview about Simon Fraser University's Stephens Family School of Medicine.

They are, of course, correct that specialties such as general internal medicine, pediatrics, and psychiatry are not primary care specialties. My comments should have more accurately referred to primary care and other community-based specialties, and I apologize for my error.

This distinction is well understood within our school and is reflected in how we are developing our medical education and postgraduate training. We are careful to emphasize to learners the distinct and complementary roles of family medicine, general internal medicine, pediatrics, psychiatry, and other specialties in the health care system.

Our intent is to prepare new physicians to meet the needs of communities across British Columbia while recognizing and respecting the unique expertise and contributions of each discipline. I appreciate the

letter writers for providing the opportunity to clarify this important point.

—David Price, BSc, MD, CCFP, FCFP
Dean, Stephens Family School of Medicine

Bad booking protocol

As a physician who is also a patient, I feel a responsibility to draw attention to what I perceive as bad protocols or habits by physicians. On this occasion, I am speaking out against a common booking protocol used by many specialists.

When a specialist receives an email referral from another physician, they respond to the email, but they do not contact the patient at that time. They contact the patient only weeks or months later, when an appointment becomes available. Meanwhile, the patient is left with no knowledge as to whether the referral went through and no idea of how long they will have to wait for their appointment. I find this protocol to be rude and insensitive of the patient's needs.

The suggestion that the patient should contact the referring physician to find out how long they are expected to wait is unprofessional and inappropriate. So is the suggestion that the patient should phone the specialist to find out.

The appropriate protocol is for the office of the referred physician to contact the patient at the same time they contact the referring physician to tell the patient that the referral has been received and that an appointment will be made for them in however many weeks or months.

I recently discovered that some hospital departments are also following this booking protocol; from the perspective of a patient, this is a definite decline in service. Not knowing if your requested CT will be in a week or a year is very unsatisfactory.

—Ben R. Wilkinson, MBBS, FRCSC, MBA
Cedar

Re: Bad booking protocol

Dr Wilkinson's letter raises a critical concern: Patients should not be left wondering whether a referral has been received or how long they may wait for care. However, individual physicians are not solely responsible for clear, effective communication, and this is not simply a question of professionalism. It is a question of how our increasingly complex health care systems can facilitate communication in ways that improve the patient experience as well as overall system efficiency.

The College of Physicians and Surgeons of BC's Referral-Consultation Process guideline emphasizes timely, clear communication and shared responsibilities between referring and consulting physicians. These principles are important. However, achieving them requires designing a system to support the principles, rather than simply adding more administrative tasks to a physician's workload.

Doctors of BC's Council on Health Economics and Policy has repeatedly highlighted the cumulative burden of administrative work. Tasks that appear small and reasonable in isolation, such as an additional phone call, email, or form, become

substantial when multiplied across hundreds or thousands of patients. The result can paradoxically be less physician time available for direct patient care.

Referral communication is, therefore, best approached as a system-design problem. Patients should receive confirmation that their referral has been received, information about anticipated wait times when available, and clear instructions about whom to contact if their condition changes. But these functions should increasingly be supported through coordinated referral systems, technological infrastructure, and cross-platform communication that supports notifications, wait-list management, and appropriately resourced administrative teams. Our province's health care system is far too complex for this to be an individual clinician's burden.

Patients deserve transparency, but physicians also deserve systems that enable transparency without having to take on the responsibility of operationalizing it.

—Inderveer Mahal, MD
Editorial Board Member, BC Medical Journal

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The social contract: Defining how we show up for society

As I write this in mid-August, I have just had the opportunity to spend time with the 48 new medical students who are beginning their journey at Simon Fraser University's Stephens Family School of Medicine.

Nearly 2000 people applied for those 48 places. What particularly interested me was which attributes helped distinguish the successful applicants. There is, of course, a minimum bar for academic achievement. But what struck me was how much emphasis was placed on connection to community—not simply volunteering, but also being part of a sports team, playing in an orchestra, coaching, or contributing in ways that demonstrate real commitment to the community around them.

That's a shift from my experience entering medical school, when community involvement mattered mostly as another form of achievement—captaining a team, leading a society, holding a position of distinction. I was delighted to see how much this has evolved.

The Stephens Family School of Medicine is clearly seeking to develop physicians who understand that their responsibility extends beyond the individual patient in front of them—physicians who see themselves as part of, and contributing to the health of, the communities in which they practise. That feels increasingly important.

Following this visit with the students, I read an excellent article co-authored by one of my predecessors, Dr Kathleen Ross, alongside Drs Courtney Howard, Katharine Smart, and Constance LeBlanc.¹ They make a compelling case that Canada's greatest

asset is its people and that investing in the wellness of current and future generations is fundamental to building a successful Canada. It is well worth reading.

The synergy between their argument and the approach being taken at SFU is striking. Both recognize that the health of individuals and the health of communities are inseparable. It also brings us back to something we perhaps don't talk about enough: the social contract between medicine and society.

Doctors are among the most trusted professionals in Canada. In return, society expects something of us: competence, high ethical standards, putting patients first, and a responsibility for the health and well-being of the broader society in which we practise.

Doctors of BC's strategic plan includes a focus on the future of the profession and the culture of medicine. I believe this gives us an opportunity—and perhaps a responsibility—to define what that social contract looks like for the physicians of today and tomorrow.

SFU has a clear vision for the physicians it aims to graduate, but many of us graduated years, even decades, ago. We are increasingly disconnected from the environment in which we trained, even though we still have many years of our careers ahead of us. That makes me wonder: If we don't define how we want our profession to show up for society, who will?

I worry that we are already seeing the consequences of allowing others to define this for us. Changes stemming from the Health Professions and Occupations Act,

which diminish our self-regulation, and issues such as the assignment of most responsible physician status to physicians on call all raise questions about who ultimately defines the responsibilities, autonomy, and culture of our profession.

These are not simple issues, and I don't pretend there are easy answers. Over the coming months, the Board of Directors, with support from the Representative Assembly, will begin these conversations and explore what the future of our profession and its culture should look like. The conversations will not always be comfortable—there will be disagreement, including on professional autonomy and the different ways physicians choose to practise. But they are conversations we need to have, because ultimately, we should retain ownership of how our profession shows up for society.

And perhaps, if we get the culture right—by combining clinical excellence with genuine connection to the community and a renewed understanding of our social contract—physicians can play an even greater role in helping Canada leverage one of its greatest assets: its people.

That feels like a future worth working toward. ■

—Adam Thompson, MD
Doctors of BC President

Reference

1. Ross K, Smart K, Howard C, LeBlanc C. Canada's greatest asset isn't beneath our feet. It's our people. Insights. Longwoods Publishing Corporation, August 2026. Accessed 13 August 2026. www.longwoods.com/content/27919/insights/canada-s-greatest-asset-isn-t-beneath-our-feet-it-s-our-people.

Heart failure tool kit for BC prescribers: Initiation and titration of medication

Cardiac Services BC has developed a tool kit of user-friendly resources to help physicians and nurse practitioners prescribe guideline-directed medications for heart failure with reduced ejection fraction.

Michael J. Diamant, MSc, MD, FRCPC, Nathaniel M. Hawkins, MBChB, MD, MPH, Valerie Barrette, RN, MScN, Rozina Chaube, RN, BScN, Fritha Munday, MPH, Kelly Mackay, RN, BScN, MA

More than 150 000 people in British Columbia live with heart failure.¹ Considering that approximately half of all individuals with heart failure have heart failure with reduced ejection fraction (HFrEF), this translates to over 75 000 people in BC affected by HFrEF.²

Dr Diamant is medical chair in heart failure and chronic cardiovascular disease for Cardiac Services BC, heart function clinic physician lead and an advanced heart failure cardiologist at Royal Columbian Hospital, and a clinical assistant professor in the Faculty of Medicine at the University of British Columbia. Dr Hawkins is medical lead, quality and research, at Cardiac Services BC and a clinical associate professor at UBC. Ms Barrette is a provincial clinical nurse specialist at Cardiac Services BC. Ms Chaube is provincial clinical initiatives lead at Cardiac Services BC. Ms Munday is a project manager, research and knowledge exchange, at Cardiac Services BC. Ms Mackay is senior director, provincial quality, research, and integrated care, at Cardiac Services BC.

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This article has been peer reviewed.

The Canadian Cardiovascular Society's 2021 guidelines recommend treating HFrEF with four classes of medications, collectively referred to as guideline-directed medical therapy (GDMT):

- One of an angiotensin receptor-neprilysin inhibitor, an angiotensin-converting enzyme inhibitor, or an angiotensin receptor blocker.
- A beta-blocker.
- A mineralocorticoid receptor antagonist.
- A sodium-glucose cotransporter-2 inhibitor.³

To significantly reduce cardiovascular mortality and heart failure-related hospitalizations and improve symptoms and quality of life, patients with HFrEF should receive the four standard therapies, in the absence of contraindications.³

Despite the well-established benefits of GDMT, it remains underused in BC. Current estimates indicate that only one in four eligible patients with HFrEF is receiving all four recommended medication classes.⁴

HFrEF management gaps

Cardiac Services BC's provincial heart failure community of practice consists of physicians, nurse practitioners, pharmacists, and nurses. They identified the need for a practical tool for clinicians to guide rapid and safe titration of the four GDMT medication classes, as well as a concise, accessible educational resource for patients with HFrEF.

In response to these needs, Cardiac Services BC developed a tool kit of user-friendly resources designed to support BC health care providers with safe, timely, and effective prescribing of medications for HFrEF.

HFrEF GDMT tool kit

The GDMT tool kit (available at www.cardiacbc.ca/clinical-resources/heart-failure#toolkit) includes the following resources:

- *GDMT Initiation and Titration Pathway:* The pathway provides prescribers and their clinical teams with practical, evidence-based strategies to initiate and titrate all four guideline-recommended medications for HFrEF within 3 months. It also provides recommendations for managing hyperkalemia and renal dysfunction and for administering additional individualized therapies beyond quadruple therapy. The pathway is available in a Supplementary Information file at bcmj.org.
- *Guidance for the Use of the HFrEF GDMT Pathway:* This resource supports clinicians in applying the *GDMT Initiation and Titration Pathway* in clinical practice. It highlights key components of the pathway and includes practical tips such as approaches for medication affordability.

- *Medications for Heart Failure with Reduced Ejection Fraction (EF)*: Developed in partnership with the HeartLife Foundation, a patient-led advocacy and support organization, this educational handout supports clear, consistent, and respectful communication with patients and caregivers. It is available in English, French, Arabic, Farsi, Punjabi, Simplified Chinese, Traditional Chinese, Spanish, and Tagalog.

The GDMT tool kit is currently being implemented across BC's Heart Function Clinics and disseminated in both specialty and primary care settings. Patients with HFrEF benefit from optimized GDMT, and all practitioners in BC, including family physicians and nurse practitioners, are now eligible to prescribe these medications and obtain Fair PharmaCare coverage through Special Authority application. We strongly encourage BC prescribers to review and use the GDMT tool kit in their practice.

Support for BC prescribers

Cardiac Services BC is available to provide educational presentations and support to BC health care providers and their teams. Contact Ms Valerie Barrette, provincial clinical nurse specialist, at valerie.barrette@phsa.ca to request an education session for your group. ■

Competing interests

Dr Hawkins is on the advisory boards of GSK and Novo Nordisk, has received speakers bureau and honoraria from AstraZeneca, has consulted for AstraZeneca and Roche, has received grants and research support from AstraZeneca, and has been involved with a clinical trial involving AstraZeneca. No other authors declared competing interests.

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EDITORIALS

Nicotine pouches

Continued from page 269

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Alhghoh

Continued from page 270

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Practical tips for interpreting blood culture results

Exploring the time for blood culture results to be reported, interpreting whether a microorganism is a pathogen or a contaminant, and transitioning patients from intravenous to oral therapy.

Polina Novoseltseva, MSc, Eugene Y.H. Yeung, MD, MSc, FRCPC, FCCM, D(ABMM), DTM&H, CTH, CIC, LTC-CIP

Situations that would prompt a clinician to order blood cultures include suspected endocarditis, pyelonephritis, sepsis, and other serious infections.^{1,2} Blood culture results provide important clinical information, including identification of the causative organism and its antimicrobial susceptibility. These parameters are central to managing bloodstream infections.

To simplify the interpretation of blood culture results, we address common questions from clinicians, such as why antimicrobial susceptibility results are not available with the initial Gram stain, how to distinguish true infection from contamination, and why oral antibiotic options may not be reported. Using the laboratory workflow

shown in the **Figure**, we aim to help clinicians confidently incorporate blood culture results into clinical decision making.

Timing and interpretation of presumptive blood culture results

In a typical adult blood culture protocol, four blood culture bottles are collected from two venipuncture sites and placed in an automated incubator for up to 5 days [**Figure, step 1**].^{1,2} Although this remains standard practice, emerging evidence suggests that single-site venipuncture sampling may be sufficient in select settings.³ Most clinically significant bloodstream infections are detected in the first 24 to 48 hours, so cultures that remain negative beyond this point are substantially less likely to yield an infectious organism.^{1,4}

Clinically, this timeline helps frame risk. For instance, in patients whose clinical status is improving, negative cultures after 48 hours suggest a lower likelihood of a true bloodstream infection. However, when clinical suspicion remains high—such as in the presence of ongoing fever, hemodynamic instability, or sepsis—empiric antibiotic treatment may be initiated while awaiting blood culture results.

Within 1 hour of a blood culture returning a positive result, the laboratory releases the Gram stain to the ordering clinician. In many cases, the presumptive organism is also identified, using direct matrix-assisted laser desorption/ionization time-of-flight (MALDI-TOF) mass spectrometry

[**Figure, step 2**]. When confidence thresholds are met, a presumptive identification (e.g., “presumptive *Staphylococcus aureus*”) is released.^{1,5} When thresholds are not met, only the Gram stain result is reported, and identification is deferred until final testing [**Figure, step 3**].

In practical terms, clinicians can expect to receive Gram stain results, with or without presumptive organism identification, within 1 to 2 days of sample submission. At this stage, antimicrobial susceptibility data are not yet available. However, empiric antibiotics may still be selected by combining the Gram stain and presumptive organism identification with antimicrobial susceptibility tables from local laboratories and hospitals. When only Gram stain results are available, published analyses of local epidemiology and susceptibility patterns may inform initial treatment decisions [**Table 1**]. **Table 1** is based on outpatient blood culture data, which represent a subset of bloodstream infections, because blood cultures are more commonly obtained in hospitalized patients with severe illness. For example, analyses of regional bloodstream infections in British Columbia demonstrate that, among Gram-negative organisms, *Escherichia coli* predominated on Vancouver Island, whereas in the Lower Mainland, *Salmonella* species were more common than *E. coli*.⁶

Definitive organism identification and antimicrobial susceptibility testing take

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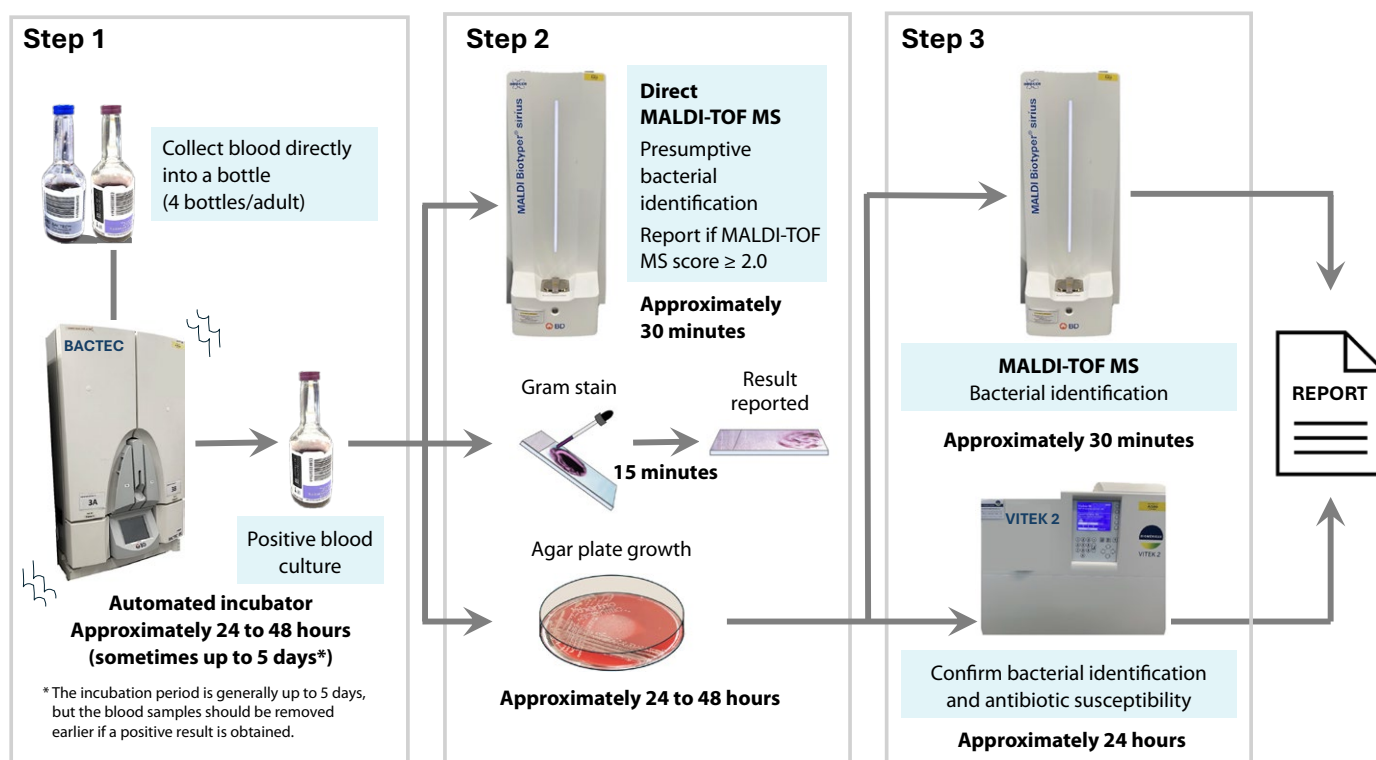


FIGURE. Adult blood culture typical laboratory protocol and workflow. Gram stain and presumptive identification results (from direct MALDI-TOF MS) are released at step 2, with final organism identification and antimicrobial susceptibility testing released at step 3.

MALDI-TOF MS = matrix-assisted laser desorption/ionization time-of-flight mass spectrometry.

longer, as they require subculture to obtain isolated bacterial colonies. This process typically adds another 24 to 48 hours to the testing timeline [Figure, step 3].² As a result, antimicrobial susceptibilities are typically available 3 to 4 days after submitting a patient's sample.

Anticipating this timeline, clinicians can plan follow-up monitoring of the changes in a patient's clinical status. Once available, the antimicrobial susceptibility can be used to confirm, narrow, or adjust initially selected empiric therapy. Ultimately, the ordering clinician is responsible for reviewing results, notifying patients, and ensuring appropriate follow-up.⁷

Distinguishing true infection from contamination

Interpreting preliminary blood culture results requires careful consideration of both the identified organism and the clinical context. Certain organisms are more

likely to be contaminants, while others are more often associated with true bloodstream infections.⁸ Organisms commonly considered to be contaminants—particularly when isolated from only one of four bottles—include *Cutibacterium acnes*, *Bacillus* species, *Corynebacterium* species, and coagulase-negative staphylococci such as *Staphylococcus epidermidis*.² In most clinical situations, these do not require antibiotic therapy. However, they may represent true infection in patients with intravascular

catheters, prosthetic joints or valves, or other implanted devices.

In contrast, isolation of organisms such as *Staphylococcus aureus*, beta-hemolytic streptococci such as *Streptococcus pyogenes* and *Streptococcus agalactiae*, *Streptococcus pneumoniae*, Enterobacteriaceae species such as *E. coli* and *Klebsiella pneumoniae*, *Pseudomonas aeruginosa*, organisms from the *Bacteroides fragilis* group, and *Candida* species should generally be considered clinically significant, even when detected

TABLE 1. Frequently encountered Gram-positive and Gram-negative organisms based on the LifeLabs outpatient blood culture data.⁶

Gram-positive	Gram-negative
Viridans group streptococci	<i>Salmonella</i> species
<i>Staphylococcus epidermidis</i>	<i>Escherichia coli</i>
<i>Staphylococcus aureus</i>	<i>Klebsiella pneumoniae</i>
<i>Enterococcus faecalis</i>	<i>Serratia marcescens</i>
<i>Staphylococcus lugdunensis</i>	<i>Enterobacter</i> species

TABLE 2. Common probable pathogens and contaminants identified in outpatient blood cultures based on LifeLabs data.⁶

Common probable pathogens	Common probable contaminants*
<i>Staphylococcus aureus</i>	<i>Staphylococcus</i> species other than <i>Staphylococcus aureus</i>
<i>Salmonella</i> species	Viridans group streptococci
<i>Escherichia coli</i>	<i>Bacillus</i> species
<i>Klebsiella pneumoniae</i>	<i>Micrococcus</i> species
<i>Serratia marcescens</i>	<i>Corynebacterium</i> species
<i>Enterobacter</i> species	<i>Cutibacterium acnes</i>

* If isolated from only one of four blood culture bottles.

in a single blood culture bottle [Table 2]. These organisms are rarely contaminants and should prompt urgent initiation of intravenous antimicrobial therapy.^{1,2}

The patient’s clinical presentation can be used as a consistency check. For example, complicated pyelonephritis that begins with urinary tract symptoms supports a urinary source, where Enterobacteriaceae species, most commonly *E. coli*, would be expected. In contrast, detection of organisms not typically associated with the presenting syndrome, such as *Streptococcus pneumoniae* or *Streptococcus pyogenes* in a presumed urinary source, should prompt consideration of an alternative focus of infection and escalation of care when appropriate.

Transitioning from intravenous to oral therapy

Bloodstream infections generally require initial intravenous antimicrobial therapy. Once the infection is clinically controlled, patients may transition to oral antibiotics to complete therapy, provided specific criteria are met. These criteria include demonstrated susceptibility to an oral agent, hemodynamic stability, adequate source control, and reliable gastrointestinal absorption.¹

The step-down from intravenous to oral therapy is an important component of antimicrobial stewardship that reduces the burden of prolonged intravenous access and catheter-related complications.^{9,10} However, because initial management of bloodstream infections is typically intravenous, oral options are often not reported

in antimicrobial susceptibility results unless specifically requested from the microbiology laboratory. Treatment decisions ultimately rest with the ordering clinician and often involve collaboration with infectious diseases and microbiology teams.⁷

Conclusions

Managing bloodstream infections relies on understanding blood culture processing timelines and results. Early interpretation of the Gram stain and presumptive identification of the organism help differentiate contamination from true infection and guide initial therapy. Final test results help identify the pathogens and potentially effective antibiotics. When combined with the patient’s clinical condition and local epidemiology, these results help the care team quickly choose the most appropriate antimicrobial therapy. ■

Competing interests

Ms Novoseltseva has no competing interests to declare. Dr Yeung has been paid to work as a physician, pharmacist, microbiologist, and clinical assistant professor. Opinions expressed are solely his own and do not represent the views of his employers.

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Serotonin syndrome presenting as posterior reversible encephalopathy syndrome in the setting of combined selective serotonin reuptake inhibitors and attention-deficit/hyperactivity disorder therapy

Clinicians managing patients with attention-deficit/hyperactivity disorder should be vigilant about the underrecognized risk factor of polypharmacy in the development of serotonin syndrome.

Nicole Hawe, MD, Lori Wik, MD

ABSTRACT: Serotonin syndrome is a potentially life-threatening toxidrome resulting from excess serotonergic activity. Posterior reversible encephalopathy syndrome is a rare and serious complication of serotonin syndrome; there are only two reported cases in the literature. We present the case of a 38-year-old woman with a history of anxiety, managed on escitalopram, and a recent diagnosis of attention-deficit/hyperactivity disorder. She had been started on lisdexamfetamine dimesylate and dextroamphetamine prior to presentation. She subsequently developed serotonin syndrome following the addition of cyclobenzaprine for musculoskeletal pain. She presented with evidence of neurologic dysfunction, and neuroimaging demonstrated watershed

ischemia and multifocal white matter changes consistent with posterior reversible encephalopathy syndrome. Extensive workup excluded alternative causes. Following discontinuation of serotonergic agents and supportive care, complete clinical and radiological resolution was achieved. This case highlights the risk of polypharmacy in patients with multiple psychiatric comorbidities and underscores the importance of vigilant medication reconciliation.

Serotonin syndrome is a potentially life-threatening drug-induced toxidrome associated with increased serotonergic activity in both the peripheral and central nervous systems.¹ It is characterized by a dose-dependent spectrum of clinical findings, including cognitive impairment, autonomic hyperactivity, and neuromuscular abnormalities.^{1,2} The spectrum of cognitive dysfunction associated with serotonin syndrome ranges from mild irritability to profound coma, while hyper-reflexivity, clonus, and tremor represent a portion of the diverse neuromuscular abnormalities.^{1,2} Autonomic manifestations include nausea, vomiting, diarrhea, dizziness, tachycardia, hypertension, mydriasis, hyperactive bowel sounds, diaphoresis, fever, and sexual dysfunction.^{1,2} While hypertension is a common feature of serotonin

syndrome, it typically falls within the mild to moderate range and does not necessitate specific therapy.² However, in some cases, severe dysautonomia can lead to hypertensive crisis, posterior reversible encephalopathy syndrome, myocardial infarction, and even death.^{2,3}

Serotonin syndrome is a diagnosis of exclusion; there is no single diagnostic test that confirms the syndrome. However, clinical diagnosis can be determined based on the Hunter Serotonin Toxicity Criteria, which require the use of a serotonergic agent and one of the following: spontaneous clonus, inducible clonus plus agitation or diaphoresis, ocular clonus plus agitation or diaphoresis, tremor plus hyperreflexia, or hypertonia plus temperature above 38 °C plus ocular clonus or inducible clonus.⁴ A summary of the features, diagnosis, and management of serotonin syndrome is provided in **Box 1**.

Over the past 2 decades, diagnoses of attention-deficit/hyperactivity disorder (ADHD) have risen steadily.⁵ Although the cause is debated, clinicians are increasingly likely to encounter patients who are receiving treatment for ADHD. Notably, 65% to 80% of children and up to 85% of adults with ADHD have at least one psychiatric comorbidity, which significantly increases the risk of polypharmacy and drug

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interactions.^{6,7} Stimulant medications, such as lisdexamfetamine dimesylate and dextroamphetamine, are commonly prescribed for the management of ADHD. These agents are thought to primarily enhance dopamine and norepinephrine signaling in the central nervous system.^{8,9} However, they may also have indirect effects on serotonin levels. When used alone, stimulant medications are generally considered safe and effective.⁸ However, caution is warranted when combining them with other medications that modulate serotonin function, because this may increase the risk of serotonin syndrome.¹

Posterior reversible encephalopathy syndrome was first identified as a distinct neurological condition in 1996.¹⁰ Over time, it has gained recognition as a compelling clinical and radiological syndrome. Among patients with posterior reversible encephalopathy syndrome, common symptoms encompass altered mental status, headache, seizures, visual disturbances, focal neurological deficits, and status epilepticus.¹¹ Posterior reversible encephalopathy syndrome is now a more recognized condition in some patients hospitalized for acute illness.^{11,12} This syndrome, first described as a possible consequence of hypertension, eclampsia, or immunosuppressive therapy, has more recently been recognized in a variety of disorders, including several of iatrogenic or neurotoxic etiology.^{10,11} Despite its recognized clinical features, the precise pathogenesis of posterior reversible encephalopathy syndrome remains elusive, although it is believed to be associated with the loss of cerebral autoregulation and endothelial dysfunction.¹³ To our knowledge, there have been only two other reported cases of serotonin syndrome leading to posterior reversible encephalopathy syndrome.^{14,15}

Posterior reversible encephalopathy syndrome is diagnosed based on clinical and radiological assessment. Neuroradiological imaging usually shows edema involving the cerebral posterior regions.^{11,12,16,17} Three common imaging patterns seen with posterior reversible encephalopathy syndrome are a dominant parieto-occipital pattern,

BOX 1. Key clinical features and management of serotonin syndrome.^{1,2,4}

Definition

Serotonin syndrome is a potentially life-threatening drug reaction resulting from excess serotonergic activity in the central and peripheral nervous systems. It is characterized by a clinical triad of:

- Mental status changes (e.g., agitation, confusion, anxiety)
- Autonomic instability (e.g., tachycardia, hyperthermia, diaphoresis, labile blood pressure)
- Neuromuscular abnormalities (e.g., clonus, hyperreflexia, myoclonus, tremor)

Diagnosis: Hunter Serotonin Toxicity Criteria

In the setting of a serotonergic agent, one of the following must be present:

- Spontaneous clonus
- Inducible clonus + agitation or diaphoresis
- Ocular clonus + agitation or diaphoresis
- Tremor + hyperreflexia
- Hypertonia + temperature > 38 °C + ocular or inducible clonus

Severity classification

- Mild: Tremor, tachycardia, diaphoresis, mydriasis, and intermittent tremor or myoclonus
- Moderate: Mild symptoms + hyperreflexia, clonus, agitation, and hyperthermia (≤ 41 °C)
- Severe: Hyperthermia (> 41 °C), rhabdomyolysis, renal failure, seizures, and respiratory failure; can be fatal

Management

- Discontinue all serotonergic agents immediately
- Supportive care: IV fluids, oxygen, and cardiac monitoring
- Benzodiazepines: First-line treatment for agitation and neuromuscular hyperactivity
- Active cooling for hyperthermia; avoid antipyretics (ineffective for this mechanism)
- Severe cases: ICU admission, intubation, or neuromuscular paralysis if refractory

a superior frontal sulcus pattern, or a holo-hemispheric watershed pattern.¹⁶ The diagnosis is usually made based on compatible clinical and radiological patterns.³ However, when the diagnosis is uncertain, the clinical and radiological improvement that occurs with appropriate treatment supports the diagnosis.¹¹ Treatment of posterior reversible encephalopathy syndrome includes correcting elevated blood pressure, discontinuing causative agents, initiating anti-epileptic therapy if seizures are present, and addressing the underlying causes.¹¹⁻¹³ The overall prognosis for posterior reversible

encephalopathy syndrome is favorable with early diagnosis and treatment, because the symptoms and imaging findings can reverse after promptly discontinuing causative agents and administering appropriate treatment.³

In this case report, we describe a patient who initially presented with nonspecific neurological symptoms. She had a preliminary diagnosis of serotonin syndrome, and her imaging findings were consistent with posterior reversible encephalopathy syndrome.

Case data

A 38-year-old woman with a history of anxiety, ADHD, and polysubstance use disorder presented with nonspecific neurological symptoms to the emergency department in a hospital in rural British Columbia. She presented after feeling unwell for approximately 1 week. Initially, she noted nausea and abdominal pain. Four days prior to presentation, she was started on indomethacin 25 to 50 mg three times a day as needed and cyclobenzaprine 10 mg daily for shoulder pain. On the morning of her presentation, she noted that her hands seemed to be slowed, as though they were not cooperating. Later that day, she developed a frontal headache with visual disturbances, light-headedness, and unsteadiness.

In the 3 months prior, this patient was diagnosed with ADHD and started on stimulant medications, which included lisdexamfetamine dimesylate 60 mg daily and dextroamphetamine 5 to 10 mg daily as needed. She also took escitalopram daily. Although she had a history of substance use,

she had not used any substances in 1 year.

Initial physical examination revealed a heart rate of 94 bpm, blood pressure of 107/73, temperature of 37 °C, and oxygen saturation of 99% on room air. Her general examination did not show any evidence of nuchal rigidity, lymphadenopathy, or skin rashes. There were no cardiac murmurs or abnormalities. Her neurological examination showed an alert and oriented female who appeared agitated. Her cranial nerves were intact, with no nystagmus. She had no sensory deficits or neglect and no motor drift. She was noted to have spontaneous choreiform-type movement in both of her upper limbs. She demonstrated bilateral dysmetria and bilateral dysidiadochokinesis. Hyperreflexia was noted in all limbs, and four beats of clonus were noted in both feet. No tremor was observed.

Initial lab tests were significant for an elevated white blood cell count at 15×10^9 cells/L, a mild C-reactive protein elevation of 45 mg/L, and a troponin elevation of 3553 ng/L. Urinalysis was within normal

limits. Urine toxicology screen was positive for tricyclic antidepressants and amphetamines. The patient's initial CT head with angiography did not show any large vascular abnormality or occlusion. Crowding of the lateral ventricles was reported and suggested possible increased intracranial pressure. Given the history of using multiple serotonergic agents, recent medication change, and clinical features that fulfilled the Hunter Serotonin Toxicity Criteria, the patient was suspected of having serotonin syndrome.⁴

The initial CT angiogram head findings, along with the patient's physical examination abnormalities, prompted a noncontrast head MRI to be performed. The diffusion-weighted sequences showed small multicentric areas of recent ischemia in a watershed distribution between the middle cerebral artery and anterior cerebral artery territory and involving the posterior occipital lobes, suggesting peripheral posterior cerebral artery territory involvement [Figure].

The differential diagnosis for these MRI findings in the context of the patient's presentation was broad, including stroke, vasculitis, and infection. Further investigations, including full cerebrospinal fluid analysis; rheumatologic workup including antinuclear antibody, rheumatoid factor, and antineutrophil cytoplasmic antibodies; and an extended infectious workup including HIV, Lyme disease, and treponemal antibodies, all returned negative. A transthoracic echocardiogram was unremarkable, and no significant valvular dysfunction was noted. A bubble study was also negative. Bilateral leg ultrasound with Doppler did not reveal any deep vein thromboses; an antiphospholipid antibody syndrome workup was also negative.

A contrast MRI was performed the following day, which again showed symmetric bilateral scattered multifocal hyperintense lesions throughout the subcortical white matter. In discussion with neurology and in the setting of a negative workup for other causes, the diagnosis of posterior reversible encephalopathy syndrome was proposed. In this clinical case, it was suspected that

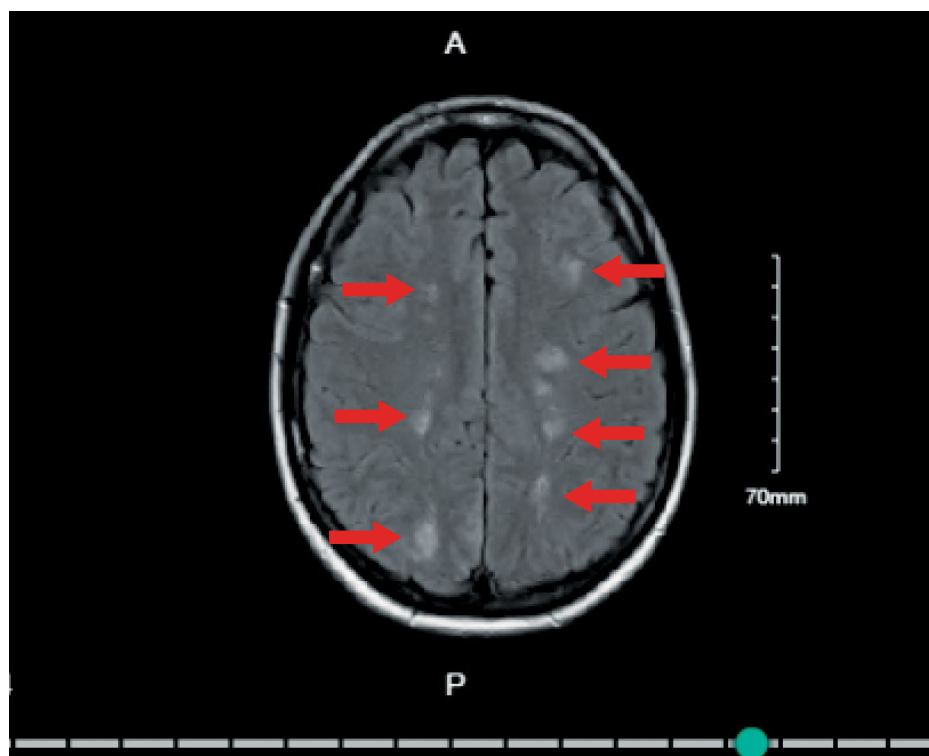


FIGURE. MRI of the brain with contrast showing restricted diffusion bilaterally in a watershed pattern (red arrows).

posterior reversible encephalopathy syndrome was a result of the patient's initial diagnosis of serotonin syndrome.

Upon admission, all serotonergic agents were discontinued, and the extensive investigations outlined above were ordered. The patient was initially treated with antibiotics, acyclovir, and aspirin. Once her cerebrospinal fluid was analyzed and did not suggest infection, her antimicrobials were discontinued. Over the following 2 weeks, she was treated supportively with intensive physical therapy. The patient's neurological symptoms slowly improved during her stay. At the time of discharge, she self-reported some cognitive difficulties and confusion, as well as ongoing visual disturbance, but her gait had improved. She was seen in the community 8 weeks after discharge and had complete resolution of all her symptoms. Her repeat MRI at this time showed complete resolution of all cortical foci. After discussion in the clinic, she elected to remain on aspirin 81 mg daily indefinitely.

Discussion

Serotonin syndrome is a potentially life-threatening toxidrome arising from excess serotonergic activity in the central and peripheral nervous systems.² It most commonly results from combinations of serotonergic agents that increase synaptic serotonin through complementary mechanisms, including increased synthesis, impaired reuptake, and direct receptor agonism that results in overstimulation of the 5-HT_{1A} and 5-HT_{2A} receptors.² The clinical presentation exists on a dose-dependent spectrum encompassing cognitive dysfunction, autonomic instability, and neuromuscular hyperactivity, with diagnosis typically established through the Hunter Serotonin Toxicity Criteria [Box 1].^{1,2,4} Autonomic dysregulation in serotonin syndrome reflects exaggerated sympathetic outflow, most commonly manifesting as mild to moderate hypertension that seldom necessitates targeted intervention.^{2,3} However, in severe cases, catecholamine surge can precipitate hypertensive crisis with end-organ consequences, including myocardial infarction,

acute kidney injury, and, in rare instances, posterior reversible encephalopathy syndrome or death.^{2,3}

Posterior reversible encephalopathy syndrome is diagnosed based on clinical and radiological assessment. Clinically, it is characterized by transient neurological symptoms, including headache, altered mental status, visual disturbances, focal deficits, and seizures.^{3,11} MRI remains the neuroimaging modality of choice, with T2-weighted sequences demonstrating greater diagnostic sensitivity than CT, classically revealing vasogenic edema predominantly involving the posterior cerebral regions.¹¹ Once considered a sequela of hypertension alone, posterior reversible encephalopathy syndrome is now recognized across a broader spectrum of precipitating conditions, including drug-induced syndromes [Box 2].^{3,11} The exact pathophysiology remains unclear, but it is believed that under normal physiological conditions, cerebral autoregulation maintains stable perfusion across a range of systemic pressures. It is hypothesized that in posterior reversible encephalopathy syndrome, this mechanism is overwhelmed or disrupted, leading to forced hyperperfusion, breakdown of the blood–brain barrier, and resultant vasogenic edema.^{3,11} Endothelial dysfunction, whether driven by hypertension, circulating toxins, or inflammatory mediators, is believed to act synergistically with autoregulatory failure.³ A distinctive characteristic of posterior reversible encephalopathy syndrome is the reversibility of clinical and radiological abnormalities after appropriate treatment and removal of precipitating factors.^{3,11}

Because ADHD diagnoses have risen steadily over the past 2 decades, clinicians are increasingly likely to encounter patients who require pharmacologic management of both ADHD and comorbid psychiatric conditions.⁵ Stimulant medications such as lisdexamfetamine dimesylate and dextroamphetamine are among the most commonly prescribed agents for ADHD; they work primarily by enhancing dopamine and norepinephrine signaling in the central nervous system and indirectly stimulate

BOX 2. Drugs and medical conditions associated with posterior reversible encephalopathy syndrome (PRES).³

Drugs associated with PRES

Oncologic

- Alkylating agents (e.g., cisplatin, ifosfamide)
- Antimetabolites (e.g., methotrexate, fluorouracil)
- Kinase inhibitors (e.g., sunitinib, ponatinib)
- Anti-vascular endothelial growth factor medications (e.g., bevacizumab, aflibercept)
- Proteasome inhibitors (e.g., bortezomib, carfilzomib)
- Hormone-modulating agents (e.g., enzalutamide)
- Chemotherapy agents (e.g., paclitaxel, vinblastine)

Miscellaneous

- Ondansetron
- Lorazepam
- Levetiracetam
- Methadone
- Lithium
- Propofol
- Phenytoin

Anti-infectious

- Hydroxychloroquine
- Voriconazole
- Acyclovir
- Linezolid
- Vancomycin

Immunosuppressants

- Calcineurin inhibitors (e.g., cyclosporin, tacrolimus)
- Corticosteroids (e.g., prednisone, dexamethasone)
- Monoclonal antibodies (e.g., ustekinumab)
- Human immunoglobulins
- Sphingosine 1-phosphate receptor modulators

Other immune modulators

- Azathioprine
- Mycophenolate
- Thalidomide

Medical conditions associated with PRES

- Uncontrolled hypertension
- Systemic lupus erythematosus
- Cryoglobulinemia
- Hemolytic uremic syndrome
- Hematopoietic stem cell transplantation
- Renal disease
- Eclampsia
- Sepsis
- Thrombotic thrombocytopenic purpura
- Solid organ transplantation
- Collagen vascular disease
- Other autoimmune diseases

the serotonin pathway.^{8,9} When combined with other serotonergic medications, such as selective serotonin reuptake inhibitors, serotonin and norepinephrine reuptake inhibitors, or monoamine oxidase inhibitors, stimulants may act synergistically on the serotonin pathway, resulting in an additive or potentiating effect that substantially elevates the risk of serotonin toxicity.^{8,9} This is particularly important to consider when such a high percentage of patients being treated for ADHD have additional comorbid psychiatric conditions in which most treatment options are serotonergic agents.⁶

We believe that in our case, the use of escitalopram, combined with the recent diagnosis and treatment of ADHD with both short-acting and long-acting stimulants, lowered this patient's threshold for serotonin syndrome. When prescribed cyclobenzaprine for her musculoskeletal injury, this was enough to tip the balance, leading to the development of serotonin syndrome, which then resulted in posterior reversible encephalopathy syndrome, which presented with neurologic dysfunction and watershed infarcts on MRI.¹⁸ In this case, the diagnosis of serotonin syndrome was based on the Hunter Serotonin Toxicity Criteria and the history of serotonergic medication use.⁴ The patient remained normotensive throughout her admission; therefore, we believe that posterior reversible encephalopathy syndrome was not a result of hypertension from serotonin syndrome but rather was secondary to autoregulatory dysfunction from serotonin syndrome. In addition, an extensive list of alternative diagnoses was considered and ruled out, which further supported the diagnosis of serotonin syndrome resulting in posterior reversible encephalopathy syndrome. Moreover, a prompt resolution of both symptoms and imaging findings with cessation of serotonergic medications further supports a diagnosis of posterior reversible encephalopathy syndrome from serotonin syndrome and excludes other secondary possibilities, because a resolution of other pathologies would not be so immediate.

To our knowledge, this is one of the first

cases of this combination of escitalopram, lisdexamfetamine dimesylate/dextroamphetamine, and cyclobenzaprine ultimately resulting in posterior reversible encephalopathy syndrome. This case highlights important principles for clinical practice. Clinicians who are managing patients with ADHD and prescribing stimulant medications should be vigilant about potential drug interactions, especially when combined with other agents that act on serotonergic pathways, such as escitalopram and cyclobenzaprine.

Summary

This case highlights important principles and the risk of polypharmacy for clinicians managing patients with ADHD and associated comorbidities. In particular, ADHD therapies are an underrecognized risk factor for the development of serotonin syndrome. This is especially relevant currently, because an increasing number of patients globally are being treated for ADHD, and many of them have additional psychiatric comorbidities. This case emphasizes the importance of a thorough medication history and awareness of potential adverse effects to prevent life-threatening complications when prescribing these medications given the increasing prevalence of polypharmacy in psychiatric and neurological disorders. ■

Competing interests

None declared.

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Cryptogenic multifocal ulcerous stenosing enteritis presenting as refractory iron deficiency anemia

Cryptogenic multifocal ulcerous stenosing enteritis is rare and challenging to diagnose but should be considered in patients with unexplained chronic anemia and small bowel obstructive symptoms.

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ABSTRACT: Cryptogenic multifocal ulcerous stenosing enteritis is a rare idiopathic disorder characterized by recurrent superficial ulcerations and multiple strictures of the small intestine, typically occurring without systemic inflammation. We report the case of a 50-year-old man with a 20-year history of postprandial abdominal pain, iron deficiency anemia, and low albumin. Initial investigations were unrevealing, and the patient was treated with iron supplementation. With progressive symptoms, endoscopic evaluation suggested eosinophilic esophagitis and enteritis; however, he did not respond to conventional therapies, including

systemic corticosteroids and dupilumab. Subsequent bidirectional endoscopy and magnetic resonance enterography were nondiagnostic. Capsule endoscopy later revealed diffuse multifocal ulcerative disease throughout the small intestine, with capsule retention at a suspected stricture. Double-balloon antegrade and retrograde enteroscopy was unsuccessful in identifying disease or retrieving the capsule. Ultimately, laparoscopic small bowel resection removed the affected segment and retrieved the retained capsule. Histopathologic examination demonstrated multifocal superficial ulcerations with associated strictures in the absence of granulomas or transmural inflammation, consistent with cryptogenic multifocal ulcerous stenosing enteritis. The patient experienced resolution of symptoms. This case illustrates the diagnostic challenges associated with cryptogenic multifocal ulcerous stenosing enteritis and underscores the importance of considering this rare entity in patients with unexplained chronic anemia and small bowel obstructive symptoms.

Cryptogenic multifocal ulcerous stenosing enteritis (CMUSE) is a rare inflammatory pathology of unknown origin, with multiple ulcerative strictures confined to the small bowel. With less than 100 published cases worldwide as of 2020,¹ CMUSE has probably been underdiagnosed or misdiagnosed, mainly as Crohn disease or NSAID-induced

enteropathy.² This case report demonstrates the complexity of diagnosing CMUSE and the importance of an accurate diagnosis to guide treatment.

Case data

A 50-year-old man had experienced postprandial abdominal pain, iron deficiency anemia, and low albumin since he was in his mid-20s. His ethnicity was half Chinese and half Central European. His medical history was significant for exercise-induced asthma and numerous food allergies. Family history was negative for gastrointestinal disorders. He had initially come to medical attention in 2002 with shortness of breath on exertion, which was attributed to an exacerbation of his known exercise-induced asthma. Despite treatment for the latter, his dyspnea did not improve.

Subsequent blood work to investigate the cause of the patient's dyspnea revealed microcytic anemia, with a hemoglobin level of 85 g/L and a ferritin level of 20 ng/mL. He had no use of NSAIDs, including both over-the-counter and prescription products. He did not use alcohol. At the time, he did not report abdominal symptoms. Bidirectional endoscopy, a barium swallow, and a barium enema did not reveal a cause of the marked anemia. He was advised to start oral iron supplementation, which helped normalize his hemoglobin.

Two years after the diagnosis of microcytic anemia, the patient developed

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intermittent postprandial left upper quadrant pain, bloating, nausea, and vomiting associated with partial small bowel obstructions. His weight was stable, with a low to normal BMI of 20; however, he was unable to gain weight despite a concentrated effort, consistent with malabsorption.

The diagnoses of eosinophilic esophagitis and eosinophilic enteritis were rendered based on biopsy findings from a repeat esophagogastroduodenoscopy in 2010. However, neither topical steroid nor proton pump inhibitor treatment improved the patient's symptoms. He had taken oral iron supplements for decades to treat his chronic iron deficiency, presumed secondary to eosinophilic esophagitis and eosinophilic enteritis; however, in 2024, he required monthly IV iron infusions because his anemia had stopped responding to oral iron supplementation and his hemoglobin was hovering around 105 g/L. The monthly IV iron infusions did not increase his hemoglobin beyond 110 g/L, and his ferritin remained low, at 10 to 20 ng/mL.

A hematology consult excluded a primary hematological cause of the anemia.

Concurrent with the decrease in hemoglobin, the patient's episodes of postprandial left upper quadrant abdominal pain became more frequent and more severe, such that he opted for a trial of treatment in 2024. Because prior biopsy had suggested eosinophilic esophagitis and eosinophilic enteritis, he was trialed on oral budesonide for several months, then dupilumab for 6 months. Neither of these treatments improved his abdominal pain, and he remained anemic and hypoalbuminemic.

In late 2024, the patient underwent updated gastrointestinal investigations, including bidirectional endoscopy and magnetic resonance enterography (a previous MRI in 2021 showed no abnormalities). The only pertinent positive finding was chronic inactive gastritis and esophagitis without eosinophils on the upper endoscopy, but this did not account for his degree of anemia and hypoalbuminemia. Colonoscopy showed no abnormalities.

Given the ongoing suspicion of gastrointestinal-related blood loss, the patient had a capsule endoscopy in February 2025, which revealed diffuse multifocal

ulcerative disease of the small bowel (jejunum) with spontaneous oozing from multiple ulcer sites. The capsule was retained at the site of involvement in a presumed stricture.

Double-balloon antegrade and retrograde enteroscopy in June 2025 did not show macroscopic or microscopic abnormalities. The capsule was not located, because it was beyond the reach of the double-balloon studies.

In September 2025, the patient's diagnosis was still uncertain. A laparoscopic small bowel resection was performed to remove the diseased section (165 cm of strictures and shallow ulcers [Figure 1]) and retrieve the capsule. Pathology revealed multifocal superficial ulcerations and strictures, negative for granulomas and transmural inflammation. There were reactive lymph nodes with abundant hemosiderin-laden macrophages [Figure 2]. This identified the diagnosis of CMUSE.

One month postoperatively, the patient's hemoglobin and albumin had normalized, yet his ferritin remained low, at 20 ng/mL. His postprandial abdominal pain had not

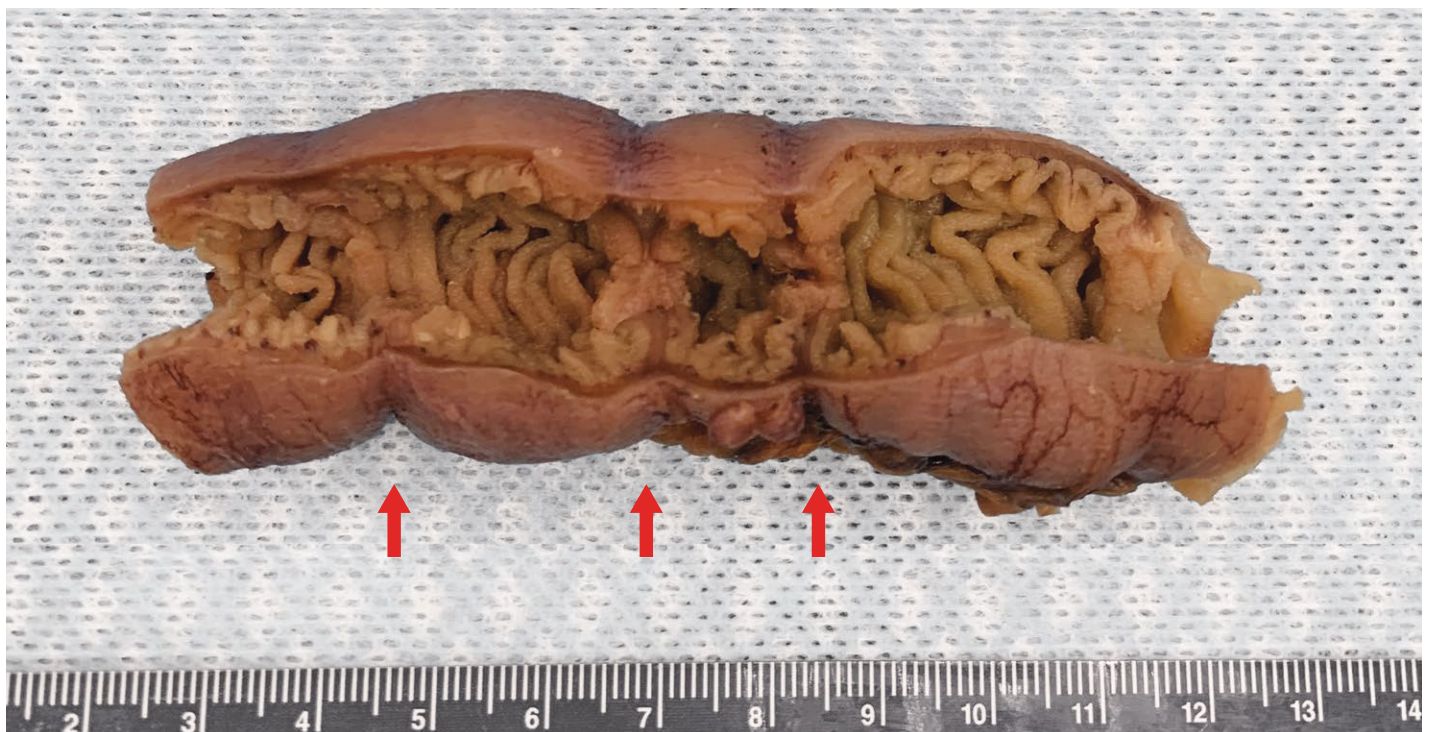


FIGURE 1. Macroscopic image of a 10 cm representative segment of the resected small intestine, demonstrating multiple thin annular strictures (red arrows).

returned since his bowel resection. Capsule endoscopy was chosen as the preferred method for surveillance going forward, because it was the only modality of investigation that had been able to identify the disease process in this patient. He was scheduled for repeat capsule endoscopy 6 months postoperatively.

Discussion

Diagnosis of CMUSE is based on history, clinical features, CT or magnetic resonance enterography, small intestinal endoscopy (capsule endoscopy or double-balloon enteroscopy), and histology of the small bowel.²⁻⁴

Chronic or relapsing subileus episodes resulting from multiple small intestinal strictures are a leading clinical symptom, along with chronic iron deficiency anemia from small bowel occult blood loss, which may precede the diagnosis for several years.² There may be fatigue, edema, or growth retardation.⁵

Blood analysis tends to show iron deficiency anemia and low albumin.⁶ Inflammatory markers and other biological signs

of systemic inflammation are usually not abnormal.^{5,6}

CT or magnetic resonance enterography may show short annular ulcerative strictures at the jejunum and/or ileum, while the inflammatory ulcerative strictures can also be recognized as multiple intestinal foci on fluorodeoxyglucose PET-CT.^{3,6}

Capsule endoscopy reveals intestinal ulcerations; however, there is an increased risk of capsule retention due to the presence of strictures.⁶ In British Columbia, capsule endoscopy can be ordered by a gastroenterologist if gastrointestinal bleeding in the small intestine is suspected, but only after other investigations have ruled out other causes (as per the Medical Services Commission Payment Schedule). Antegrade or retrograde enteroscopy can confirm the presence of one or more short ulcerative strictures with normal mucosa between;⁶ however, sometimes the involved area cannot be reached, as in the case of our patient.

Histological findings include superficial ulceration, fibrosis and nonspecific inflammation of the mucosa and submucosa,

and vascular changes without transmural inflammation.⁴

The **Table** summarizes the differential diagnoses of CMUSE.^{2,6-9} Crohn disease is an important differential diagnosis, with skip lesions and cobblestoning on macroscopic appearance, while histology reveals transmural inflammation, and the typical lesions are longitudinal and fissured ulcers, which are prone to fistula formation and intestinal perforation.^{4,5} NSAID-induced enteropathy is very similar to CMUSE on endoscopy and histology. In NSAID-induced enteropathy, there tends to be gradual resolution of disease with NSAID cessation.⁵ Genetic testing for *PLA2G4A* mutations, genes associated with prostaglandin metabolism, can also distinguish these conditions.⁶ Other differential diagnoses include intestinal tuberculosis, vasculitis, chronic ulcerative jejunitis, collagen sprue, autoimmune or eosinophilic enteritis, and chronic nonspecific ulcers of the small intestine.^{2,5,6}

Chronic nonspecific stenosing ulceration is a similar entity that affects predominantly Japanese patients and is associated

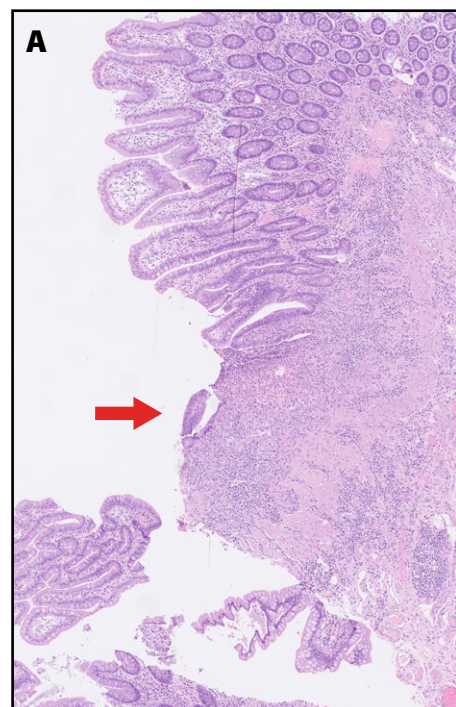
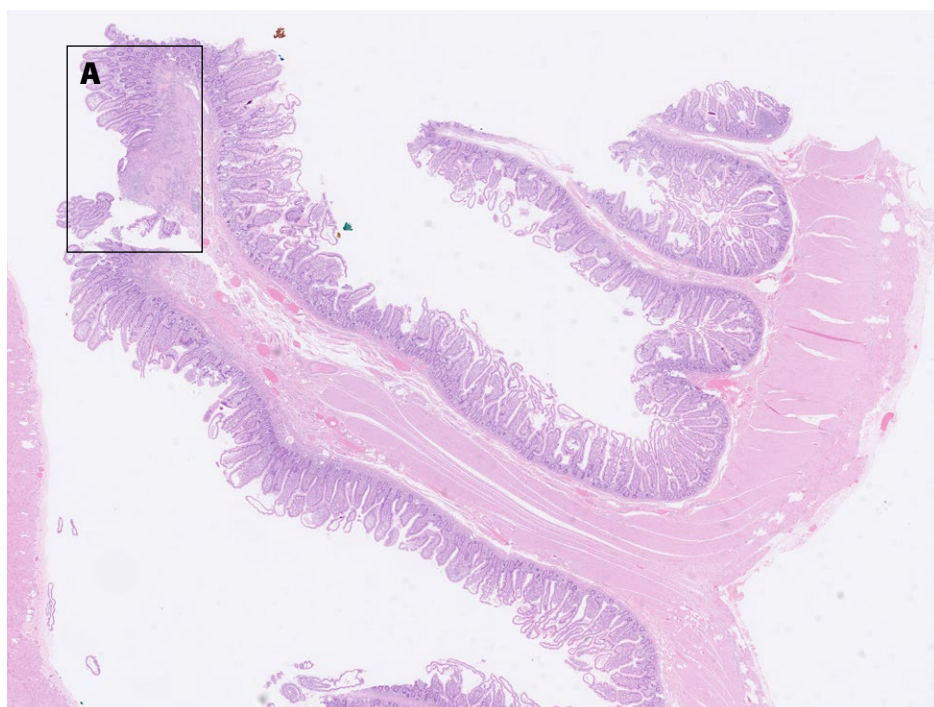


FIGURE 2. Histology of the thin, web-like mucosal septum with surface ulceration (red arrow); hematoxylin and eosin stain (1x). **(A)** Higher-power view of superficial mucosal ulcer (4x).

TABLE. Pathological findings of cryptogenic multifocal ulcerous stenosing enteritis (CMUSE)/chronic nonspecific stenosis ulceration (CNSU) and its main differential diagnoses.

	CMUSE/CNSU	NSAID-induced injury ("diaphragm disease") in the small bowel	Crohn disease
Distribution in the gastrointestinal (GI) tract	Disease limited to the small intestine	May affect all segments of the GI tract ⁷	Often ileocolonic distribution; may affect all segments of the GI tract
Macroscopic appearance	Multifocal thin circumferential strictures with superficial ulcers ⁶	The most severe stage of NSAID-induced enteropathy ("diaphragm disease") is similar in appearance to CMUSE/CNSU ⁶	Skip lesions and cobblestone appearance
Clinical presentation features	Chronic abdominal pain; partial obstruction; iron-deficiency anemia ⁶	History of NSAID use; gradual recovery after discontinuation ⁸	Presence of typical extraintestinal manifestations such as cutaneous erythema nodosum, arthritis, and eye disease ⁸
Microscopic appearance	Superficial ulceration, fibrosis, and nonspecific inflammation of the mucosa and submucosa; no transmural inflammation or granulomatous inflammation ⁶	Superficial ulceration, fibrosis, and nonspecific inflammation of the mucosa and submucosa; +/- transmural inflammation ⁷	Knife-like fissuring ulcers; noncaseating granulomas; transmural inflammation, often with lymphoid aggregates
Treatment	Glucocorticosteroids; less commonly, immunosuppressive and immune modulatory medications, endoscopic dilatation, and surgical resection; nutritional support ^{2,6}	NSAID cessation; some response to balloon dilatation ⁶	Steroids, monoclonal antibody therapies, immunomodulators, and surgery ⁹

with loss-of-function mutations in the *SLCO2A1* gene.⁶

Medical treatment consists of systemic and local glucocorticosteroids at individual-specific doses;^{2,6} however, most patients develop corticosteroid dependence,² and some will relapse or develop steroid resistance.^{5,6} There have been case reports of long-term remission using immunosuppressive and immune modulatory medications typically used in Crohn disease and ulcerative colitis, such as mesalazine, mercaptopurine, azathioprine, thalidomide, and infliximab.⁶

Endoscopic balloon dilatation can alleviate symptoms and prevent small bowel resection.^{2,5} Surgical resection is a last resort, reserved for those with small bowel obstruction or patients who do not respond to medical treatment.^{2,6} However, patients can easily relapse, and surgery poses a risk of complications such as short bowel syndrome.^{2,4}

Furthermore, supportive care by means of enteral or even parenteral nutrition and iron and vitamin supplements may improve symptoms and promote mucosal healing.^{5,6}

Summary

In this case, the patient had received systemic steroids as treatment for eosinophilic esophagitis and eosinophilic enteritis, which did not promote remission of CMUSE. Ultimately, a small bowel resection was performed to alleviate his symptoms and clarify his diagnosis. Continued follow-up is necessary to monitor for relapse. This case highlights the complexity of diagnosing CMUSE and the importance of an accurate diagnosis to guide treatment. ■

Competing interests

None declared.

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A rapid overview of occupational diseases from welding:

Tips for family physicians

As a physician, you may have patients who work as welders. Welding is fundamental to many industrial processes and occurs across a variety of industries. Here we review key chronic health outcomes related to welding and provide information on reporting related workplace exposures and diseases.

Welding and allied processes have shared health hazards, such as ergonomic stressors, noise, welding fumes, irritating gases, fires, ultraviolet and infrared radiation, eye hazards, and electrical shocks. These hazards place welders at risk of certain chronic occupational diseases. These diseases often have gradual onset over several years, which can make diagnosis and assessment of risk factors difficult.

Occupational diseases associated with welding

Respiratory diseases

Long-term exposure to welding fumes is associated with chronic obstructive pulmonary disease (COPD), chronic bronchitis, and occupational asthma, although the degree of risk depends on several factors (e.g., type of metal fumes, workplace controls).¹ Routine spirometry for welders is not required in Canada; however, it is important that the patient is aware of the signs of asthma and bronchitis and sees a physician early for persistent respiratory symptoms. For concerns about asthma or COPD, consider referring your patient for pulmonary function testing. You can

search either Pathways or the College of Physicians and Surgeons of BC's Diagnostic Accreditation Program to find accredited testing facilities where pulmonary function testing is performed.

Illnesses from chemical exposures

Exposures to chemicals like metals and solvents, including agents like chromates, nickel, epoxies, and degreasers, can vary and are highly job dependent. The patient should work with their employer to understand and effectively prevent these exposures. Medical monitoring among asymptomatic individuals is limited under BC's Occupational Health and Safety Regulation and is typically arranged by the employer.

If the patient develops signs of toxicity from a chemical exposure, laboratory testing may be indicated. The indications and interpretation of these tests can be specific, so ordering family physicians may consider consulting other specialist physicians (e.g., internal medicine or medical subspecialties), calling the BC Drug and Poison Information Centre, or contacting a WorkSafeBC physician via the RACEapp+ to discuss.

Occupational cancers

The International Agency for Research on Cancer has assessed that welding fumes are a confirmed cause of lung cancer, with positive associations with kidney cancer.² Ultraviolet radiation from welding has also been determined to cause ocular melanoma.² Patients diagnosed with these cancers should have occupational histories that inquire into welding.

Contact dermatitis

Welders have regular contact with dermal sensitizers that can lead to allergic contact

When to report to WorkSafeBC

If a patient who works or has worked as a welder develops a disease that is suspected to be work-related, initiate a claim by submitting a Form 8 to WorkSafeBC. If you have questions before or after submitting a Form 8, contact a WorkSafeBC physician on the RACEapp+.

dermatitis—notably metal dusts and fumes (e.g., nickel, chromium), epoxies, and resins.³ Workplace irritants include solvents, petrochemicals, and ultraviolet radiation. Patch testing may be indicated for welders with dermatitis to ensure that allergens are identified and managed.

Noise-induced hearing loss

Welders are often exposed to hazardous noise levels, which can cause noise-induced hearing loss.⁴ Employers have legislated responsibilities for noise, including exposure limits, hearing conservation programs, workplace controls, and audiometric surveillance. You may receive referrals to initiate medical investigations, specialist referrals, and clinical management of abnormal audiograms from workplace surveillance programs.

If you suspect occupational risks or exposures

If there are any significant concerns about preventable health risks in a workplace, you or your patient can contact WorkSafeBC's Prevention Information Line (1 888 621-7233).

Continued on page 295

How physicians can help improve declining childhood immunization coverage rates in BC

Immunization coverage assessments are conducted to measure the percentage of people who have received certain vaccines. The assessments are used to:

- Understand population-level protection against vaccine-preventable diseases.
- Explore potential reasons for low vaccine coverage.
- Identify communities at higher risk for outbreaks.
- Inform population-level interventions, including resource allocation.

Routine childhood immunization coverage is assessed annually at four key milestones: age 2, age 7, grade 6, and grade 9. Coverage estimates for individual antigens are calculated for each milestone, and the proportion of children considered up to date for all antigens is presented for ages 2 and 7. Annual coverage estimates, historical coverage trends, and reasons for nonimmunization are published on the BC Centre for Disease Control website.¹

For most routine childhood vaccines, provincial coverage rates have generally declined from 2016 to 2025.² Two-dose measles vaccine coverage at age 7 decreased from 90% in 2016 to 72% in 2025, while the proportion of children considered up to date for all routine immunizations at age 2 decreased from 73% in 2016 to 69% in 2025 [Figure].² Recent years have seen recovery from lows observed during the COVID-19 pandemic attributable to pandemic-related service disruptions, although coverage rates have not returned to prepandemic levels.²

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

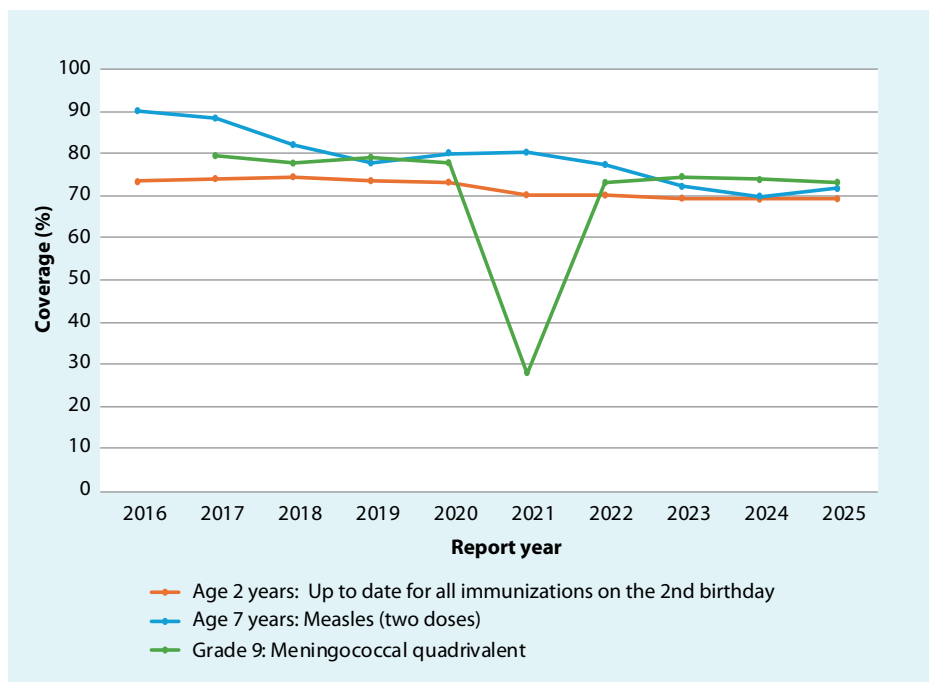


FIGURE. Provincial immunization coverage estimates from 2016 to 2025 for 2-year-olds up to date for all immunizations, 7-year-olds with two doses of measles vaccine, and grade 9 students with at least one dose of meningococcal quadrivalent conjugate vaccine in adolescence. See the BCCDC immunization coverage reports for details on definitions and methodology.¹

Uptake of the meningococcal quadrivalent conjugate vaccine in grade 9—previously holding steady at 78% in 2017 to 79% in 2020—dropped to 28% in 2021 due to interruption of school-based immunization clinics.² Rates subsequently recovered to 73% in 2022 and 74% in 2025 [Figure].² Declines in childhood immunization coverage have been similarly observed in other provinces in Canada.³

Reasons for lower immunization coverage

Reasons for lower immunization coverage estimates are multifactorial and include documentation gaps, barriers to vaccine access, and other structural and systemic

factors. Coverage estimates are based on vaccine doses recorded in the Provincial Immunization Registry. However, not all vaccinations are recorded. Doses administered by family physicians and in acute care settings are generally not recorded or are delayed being recorded unless they are reported separately to Public Health, documented using the Immunization Entry Form (via eForms), or submitted via the provincial immunization record portal. Immunization records for people new to BC (including those from another province or territory) are also not automatically captured in the registry. Of the 2024 and 2025 cohorts, 9% to 11% at ages 2 and 7 and 2% to 4% in grades 6 and 9 had no records in

the registry and are therefore reported as unimmunized.

Coverage rates may also be influenced by access to vaccination services. School-based vaccination programs have long supported access by removing the need to book appointments and travel to vaccination sites⁴ and have played a key role in vaccine uptake. This is demonstrated by the reduction in grade 9 immunization coverage as a result of service disruptions during the pandemic as well as by the increase of two-dose measles vaccination coverage from age 7 (80% in 2021) to grade 6 (89% in 2025) among roughly the same cohorts.²

While coverage rates are not stratified by sociodemographic factors, inequities in structural and social determinants of health are long recognized as contributing to unequal infectious disease burden as well as differential access to and uptake of vaccination. For example, rural, remote, and Indigenous communities face inequitable access to vaccination. People living with disabilities face various barriers to access, while newcomers face challenges finding accurate information that meets cultural and linguistic needs. Historic and ongoing medical racism and discrimination also negatively affect trust and vaccine acceptance.⁴

Moreover, while vaccine mis- and disinformation are not new phenomena, the relationship between trust, mis- and disinformation, and vaccination may be particularly complex for populations already experiencing mistrust in health care or the government, particularly in the context of increased polarization of vaccination across digital and social media platforms.⁴

What physicians can do to improve vaccination coverage

Many initiatives to support vaccination focus on individual-level factors affecting vaccine uptake rather than access to vaccines. Without considering access, differential vaccine uptake may be incorrectly attributed to low vaccine acceptance rather than structural issues, placing undue responsibility on individuals without recognizing the broader systems and determinants

influencing vaccination.⁴ Understanding structural barriers to vaccine access enables physicians to tailor their approach to populations facing intersecting inequities, including the following.

Offering and promoting low-barrier vaccination: Resources are needed to book vaccine appointments, travel to appointments, and cover other vaccination-related costs (e.g., time off work). Physicians can work with patients to identify and address these barriers by co-delivering vaccines with other health services, reducing missed opportunities for vaccination, and helping to identify accessible vaccination services in their community.⁴

Creating supportive health care environments: Vaccination is enabled by supportive health care environments, including access to a trusted physician and culturally safe, person-centred care.⁵ Reducing vaccine-related pain and fear can also positively impact vaccination experiences.^{4,6}

Fostering supportive vaccine information environments: Not all people have equitable access to supportive information environments. Access to timely and accurate information on the safety and effectiveness of vaccines and the frequency and severity of vaccine-preventable diseases from a trusted source is key for shaping knowledge, attitudes, and beliefs about vaccines.⁷ Tailored messages that are appropriate, accessible, easy to understand, and culturally relevant can increase vaccine acceptance and uptake.³

Finally, recognizing that low immunization coverage may represent gaps in documentation rather than true vaccination coverage, family physicians are strongly recommended to document vaccines in the Provincial Immunization Registry using one of the provincial tools (eForms,⁸ the immunization record portal,⁹ or electronic medical record system integrations). People whose records are not reflected in Health Gateway should also be encouraged to submit

or update their vaccination records in the provincial immunization record portal.⁹ ■

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—**Michelle Takeuchi, RN, MHA**

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News We welcome news items of less than 500 words; we may edit them for clarity and length. News items should be emailed to journal@doctorsofbc.ca and must include your mailing address, telephone number, and email address. All writers should disclose any competing interests.

Doctors of BC 2026 scholarship winners

Doctors of BC has awarded its annual \$1000 scholarships for 2026 to Ms Kate Myllyniemi and Ms Claire Padvaiskas. The recipients were chosen from a pool of inspiring applicants.



Ms Kate Myllyniemi

Ms Myllyniemi graduated from Mount Douglas Secondary School's Challenge Program and is attending the University of British Columbia in the Faculty of Science this fall. In high school, Ms Myllyniemi participated in numerous sports, including captaining her varsity basketball team and playing on the ultimate Frisbee team, which won bronze at the provincial championships. She enjoys working with younger students and athletes, whether she is mentoring other students in her school's gifted program, coaching basketball, or volunteering as a junior leader with Girl Guides of Canada. Ms Myllyniemi is grateful to have been recognized with several awards during her graduating year, including receiving the Grade 12 Service Cup and being named the senior girls' basketball most valuable player. At UBC, she hopes to explore her interest in science while working toward a career in health care. In the future, she would love the opportunity to care for children at BC Children's Hospital and volunteer internationally with Love Without Boundaries. Ms Myllyniemi looks forward to continuing to learn, meet new people, and find ways to make a difference in her community.

Ms Padvaiskas is entering her 4th year at the University of British Columbia, where she is completing her bachelor of science in neuroscience. Ms Padvaiskas is particularly interested in improving follow-up care after critical illness. Through her work in the pediatric intensive care unit at BC Children's Hospital, she has seen how recovery continues long after a child leaves the hospital. This insight led her to become interested in how health care teams can better support children and families through the transition from acute care to the community.

Ms Padvaiskas has a background in competitive dance and enjoys staying active through hiking and Pilates. She also loves learning new languages, reading, and creative writing in her spare time. Her interest in Spanish led her to volunteer with MOSAIC, where she facilitates English conversation groups for newcomers and assists with Spanish interpretation. She enjoys meeting people from different backgrounds and helping create a welcoming environment where students can gain confidence, improve their communication skills, and use language as a tool to connect with others.

Ms Padvaiskas hopes to attend medical school and eventually pursue a career in pediatric critical care, combining patient care and research to improve the transition from hospital to home for children and their families.



Ms Claire Padvaiskas

About the award

For more than 20 years, Doctors of BC has presented the scholarship award to children of members in good standing who are completing (or have completed) high school and will be (or are currently) attending a recognized postsecondary institution. For more information, visit www.doctorsofbc.ca/about-us/awards-scholarships/awards-residents-students-community.

Spotlight: Indigenous Physicians Association of Canada

Founded in 1991, the Indigenous Physicians Association of Canada (IPAC) is a member-based not-for-profit organization that aims to nurture healthy and vibrant Indigenous nations, communities, and individuals. It is supported by Indigenous physicians, Indigenous medical learners, and allies who share this goal. IPAC's priorities are interconnected and focus on advancing the health of Indigenous lands and communities and uplifting current and future Indigenous physicians. At its core, IPAC is also a national community where Indigenous physicians and learners can connect, support one another, and strengthen Indigenous leadership in medicine.

The work of IPAC is diverse and multifaceted, explains Melanie Osmack, IPAC's executive director. IPAC advocates for culturally safe health care; supports efforts to address Indigenous-specific racism in medicine; contributes to conversations on forced and coerced sterilization; and partners on initiatives that improve health across the lifespan, including men's and boys' health and menopausal care for Indigenous women.

Osmack says a key goal is to ensure that Indigenous physicians and learners help shape the future of health care in Canada. This work includes contributing to national conversations; working on national consultations; collaborating with governments,

medical schools, health organizations, and Indigenous partners; and advocating for policies that reflect Indigenous communities' priorities. As a member of the Canadian Medical Forum, IPAC connects with other national organizations, including the Canadian Medical Association, to discuss issues of priority for physicians and the Canadian health care system.

For IPAC, strengthening Indigenous representation in medicine is vital, as Indigenous physician leadership leads to stronger health care systems and better outcomes for Indigenous people. Ultimately, IPAC's goal is to ensure that Indigenous perspectives are part of shaping decisions from the beginning. This work is interconnected with IPAC's broader commitment to the health of Indigenous people, communities, and lands. Recognizing that human and environmental health are inseparable, IPAC also supports the work of partner organizations, including the Canadian Association of Physicians for the Environment.

Osmack notes IPAC's unique role in nurturing and mentoring Indigenous medical students and residents. Medicine can be isolating, particularly for Indigenous learners, who may be the only Indigenous person in their program, residency, or training environment. For many Indigenous students, residents, and physicians, IPAC is a place to connect with colleagues who can support one another, celebrate diverse cultural identities, and build a solid community. It provides mentorship-matching services for

all members, as well as Canadian Resident Matching Service information workshops and funding to attend conferences and events. It works with medical schools in Canada to ensure that Indigenous medical learners can attend its Annual Mentorship Gathering. Through its Mentorship Circle, these relationships often extend beyond career advice to become a source of shared understanding and belonging.

For physicians who want to learn more about Indigeneity in health care or become better allies, IPAC encourages curiosity, humility, and a willingness to listen. Through initiatives such as Strengthening Allyship in Action: Walking Alongside in Medicine, physicians are encouraged to move beyond good intentions by listening, reflecting, and taking meaningful action in their own practices and communities.

Through its work, IPAC is helping ensure Indigenous perspectives are included in the development of a culturally safe, respectful health care system that supports physicians, patients, and the lands on which they live. Recognizing that Indigenous people are the experts in their own lives, cultures, and communities makes better outcomes possible.

Visit the IPAC website to learn more about its work, available resources, and how to become a member (<https://ipacamic.ca>).

—Ellen Tannam
Communications Specialist,
Communications and Public Affairs,
Doctors of BC



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Obituaries

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Dr Anthony “Hugh” Robinson
1947–2026

Dr Anthony “Hugh” Robinson was born in Saltash, Cornwall, England, to Tony and Joan Robinson. He attended Sherborne School in Dorset and, as a young man, developed a lifelong love of sailing, working as a sailing instructor in Saltash.

Following in the footsteps of his father and grandfather, Hugh pursued a career in medicine, qualifying from Middlesex Hospital Medical School in London in 1972. That same year, he immigrated to Canada and began working as a family physician in Vancouver.

Hugh met his wife, Caroline, in 1975, and they married in 1976. Their daughter, Claire, was born in 1978, followed by their son, Jamie, in 1980.

Hugh worked as a full-service family physician until retiring from his practice in 2013. He cared deeply for his patients and practised medicine in the truest sense of “cradle to grave”—from delivering babies to caring for the elderly, including his work as medical coordinator at St. Jude’s Anglican Home in Vancouver.

Hugh lived life to the fullest. He was an avid skier and sailor and later became a passionate road cyclist, completing numerous races and Gran Fondos, including the Maratona dles Dolomites in Italy. He also completed three Vic-Maui Yacht Races.

Retirement did little to slow him down. Hugh enjoyed his Probus groups, investment club, and book club, and he especially valued time spent with his many dear friends. He continued to travel and seek out new adventures, including recent family trips to Patagonia, hiking in New Zealand, and travels throughout Europe.

Above all, Hugh was a wonderful husband, father, and grandfather. He was immensely proud of his three equally adventurous grandsons, William, Benjamin, and Oliver, and he loved sharing his spirit of adventure with his family. His many friendships also meant the world to him, and he was fortunate to have wonderful friends throughout his life.

Hugh is survived by his wife, Caroline; his children, Claire and Jamie; his grandchildren, William, Benjamin, and Oliver; his brother, Jo, in England; and his nephews, Charles, Miles, and Alexander.

Hugh passed away on 30 July 2026, surrounded by his family, just 11 days after being diagnosed with lung cancer. His loss was sudden and profound, and he will be deeply missed by his family and the many friends whose lives he touched.

In lieu of flowers, donations may be made in Hugh’s memory to Doctors Without Borders.

—**Claire Robinson, MD, CCFP**
Vancouver



Dr John Law
1958–2026

Dr John Law, my friend, passed away peacefully in Nanaimo on 11 March 2026 at the age of 68, surrounded by his loving family.

I write this remembrance of John from a place of grief but also with immense gratitude for the blessing of his presence in my life and the lives of many others. He embraced life fully, with curiosity and enthusiasm, and he met every person with kindness and compassion. Our lives have been enriched and transformed by John’s words and actions. He inspires us still as a model of what a physician can be and what to make of a life. John was my best friend from medical school and the physician I most admire.

John was born in Vancouver on 1 February 1958. He grew up in the Strathcona neighborhood of Chinatown in a house with no indoor plumbing except for the kitchen sink, which sat atop a dirt-bottomed cabinet. He could hear the coos of the pigeons living in the vacant suite upstairs. John’s first career was plumbing. John once said, “The

past serves me well, and I am so grateful for all that I have survived. I like to think of investing and profiting from my suffering when I encounter challenges. This is how we can learn and improve.”

At the University of British Columbia, he met his wife and best friend, Bonnie. After graduating from medical school, John served in the Canadian military as a medical officer and began his family practice in the Courtenay Medical Clinic.

Like all great physicians, John continued to refine his skills and expand his knowledge beyond medical school, motivated by his deep desire to help his patients. I know of no other physician with the breadth and depth of John’s clinical and counseling skills.

John studied and practised mindfulness, acupuncture, clinical hypnosis, trigger-point therapy, neuroproltherapy, and a variety of myofascial skills, including the Franklin Method and osteopathic techniques. He shared his learning generously with colleagues and trained international medical graduates.

John did more for his patients than any other physician I know. In addition to his full clinic practice, he taught weekly 90-minute group sessions in psychological skill building, teaching his patients practical skills such as mindfulness, couples’ therapy, motivational interviewing, trauma therapy, dialectical-behavioral therapy, addictions therapy, and cognitive-behavioral therapy (CBT). He would accept some of the most challenging patients, because he knew no other physician would care for them.

John told me, “I have realized I am so drawn to medical hypnosis, motivational interviewing, positive psychology, mindfulness, and CBT because of the empowering nature of the communication and the intent. Put together with our technological advances, wonderful outcomes can result.”

John enjoyed a rich life outside of medicine. With his lifelong curious and humble spirit, he challenged himself by studying public speaking with Toastmasters; swimming; playing guitar, ukelele, violin, piano, saxophone, and banjo; singing;

and dancing, including doing the West Coast Swing with Bonnie.

John cherished his beautiful family. He was a devoted, loving, and wise husband and father, and he inspired both of his children, Christine and Meesh, to become physicians.

John inspired his family, friends, colleagues, and patients to rise to life’s challenges. He saw and brought out the best in us. He was always cheering us on.

John did not get lost in the title, role, and work of a physician. This was his advice to me on life after an accomplished career in medicine: “Going into retirement to explore and learn skills that might enhance my quality of life meant at times being the one with the least skill in a group. It is liberating to judge myself not by my skills or accomplishments but [by] how I treat others and myself.”

After John’s retirement, he and Bonnie moved to Nanaimo, where their daughter, Christine, had established her family practice. He continued to enjoy his passions in music, exercise, gardening, and, above all else, his family.

He is survived by his loving wife, Bonnie; his children, Christine Layton (Elliot) and Meesh QX; his grandchildren, Quinn and Afton Layton; and many family members and friends. Our lives have been enriched and transformed by John. He inspires us still.

—**Davidicus Wong, MD**
Vancouver

Continued from page 289

Patients who believe they have had exposure to a harmful substance at work but haven’t experienced ill effects can file the exposure through WorkSafeBC’s Exposure Registry Program ([worksafebc.com/form-41m1](https://www.worksafebc.com/form-41m1)). ■

—**Kieran Steer, MD, FRCPC, MSc**
Medical Specialist, Occupational Diseases,
Medical Services, WorkSafeBC

—**Jeanette Campbell, MSc(A), CIH, ROH**
Senior Occupational Hygienist,
OHS Science and Technical Service,
WorkSafeBC

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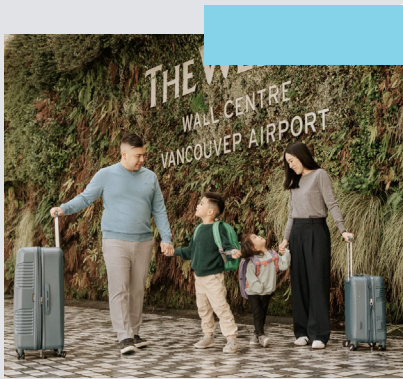
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