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A Doctors of BC Publication

THEME ISSUE

Pancreatic cancer, part 1



IN THIS ISSUE

Guest editorial: Pancreatic cancer, part 1

Pancreas screening in high-risk individuals

Pancreatic cystic lesions in adults: Diagnosis and management

Pancreatic neuroendocrine tumors in British Columbia: A review

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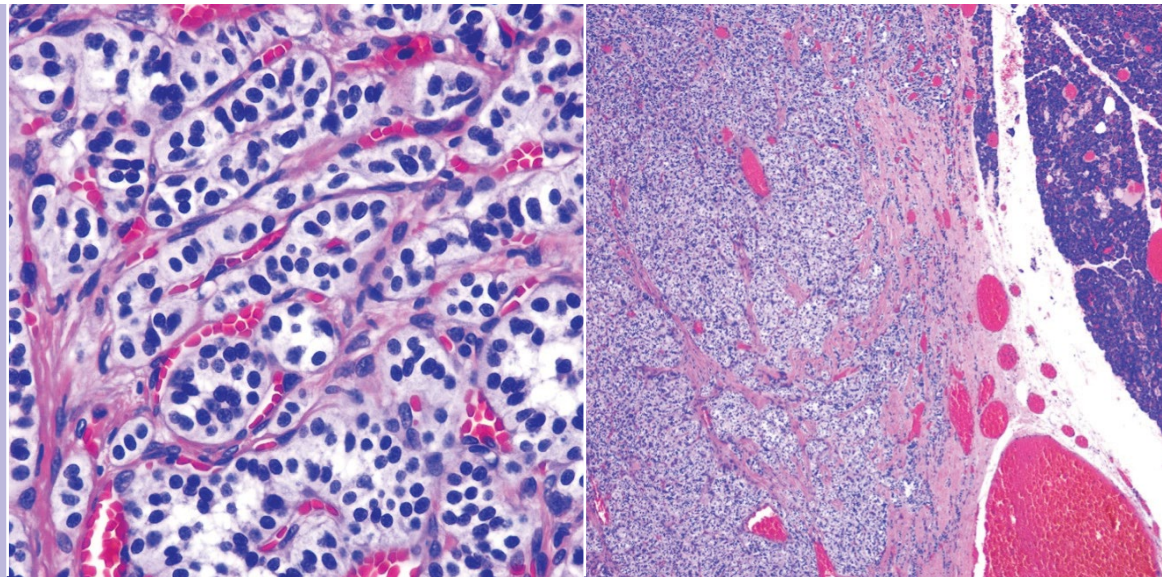


Figure 1 from the article "Pancreatic neuroendocrine tumors in British Columbia: A review." Pancreatic neuroendocrine tumor after endoscopic ultrasound fine-needle biopsy. Images on the left (40x) and right (4x) show immunohistochemical stains of the biopsies. Findings indicate a well-differentiated neuroendocrine tumor. The theme issue on pancreatic cancer begins on page 312.

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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ON THE COVER
Sometimes called the "lion of the abdomen," the pancreas is powerful and dangerous when provoked. True to the fierce association, pancreatic cancer is the deadliest cancer in BC. In this theme issue, three articles from a multidisciplinary group of physicians, nurse practitioners, educators, and researchers cover various aspects of the disease. The guest editorial begins the section on page 312.

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The histories of three provincial institutions reflect the evolving social, ethical, legal, and therapeutic approaches that have shaped mental health care in BC for generations. Pictured is Woodlands in New Westminster, c. 1878. "A missing tale of psychiatry in British Columbia" begins on page 308.

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Shining the spotlight on BC physicians

There is extraordinary work happening in medicine around British Columbia. Some of it is happening in large academic centres, some in small rural communities. Some involves cutting-edge research or innovative clinical care. Some is simply a physician who has found a better way to solve a problem, support their patients, or bring a community together. But how often do we hear about it?

As physicians, we spend much of our professional lives focused on our own patients, practices, and institutions. We may work a few kilometres or several hundred kilometres from colleagues doing remarkable things, without ever knowing their stories.

Last year, we introduced a new article type called Physician Spotlight. It is part of a refreshed group of article types that also includes BC Stories, Clinical Images, and Beyond Medicine. The changes were informed by our reader survey and website data, but they were also driven by something more fundamental: We want the *BCMJ* to help physicians learn from one another.

Perhaps you are a rural physician who has inspired connections in your community, or a researcher whose question began with something you wanted to solve in your practice. Perhaps you have a story about your career decision, mentorship of younger students, or an unexpected path in life that changed how you practise medicine. These stories matter.

There is a saying that "We need to see it to be it." Understanding what drives our colleagues' work can expand our sense of what is possible. I believe that we can all benefit from hearing how any one of us has built a fulfilling career in medicine. The *BCMJ* is uniquely positioned to create this communication—to share your stories of success, failure, surprise, controversy, hardship, and teamwork.

Understanding what drives our colleagues' work can expand our sense of what is possible.

Take a moment to consider whether you know a BC physician whose work deserves a wider audience. Tell us about them, or write about yourself. Physician Spotlight is designed to highlight the person behind the process. Here are a few example questions you can use to lead an interview with a colleague, as a jumping-off point for an essay, or to answer about yourself:

- What is your greatest achievement or the proudest moment of your career?
- What is an initiative you want more people to know about?
- What brings you the most happiness?
- What is your motto?
- What do you wish you knew when you were a medical student?
- What medical advance do you most anticipate?
- What is something you've learned from a patient?
- How have you been inspired by one of your heroes?

Submission guidelines are provided at https://bcmj.org/submit-content. And please, send pictures! A photo of the physician doing the work they care about, with colleagues, or in the community.

In this issue, a spotlight on Dr Birinder Narang highlights his love of service, faith, and family.

If you've got a great story but it doesn't relate to the practice of medicine, there's the BC Stories section, where physicians share their adventures from their off-hours. We look forward to hearing from you. ■

—Caitlin Dunne, MD, FRCSC

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The patient behind the chart

I know many of my patients' medical charts remarkably well. I know when their last mammogram was done, how their A1c has changed over the years, which medications they can't tolerate, and which investigations still need follow-up. But sometimes I find myself wondering whether I know enough about the *person* whose chart I know so well.

Family medicine is built on the foundation that knowing the patient matters. Yet, as the need for thorough documentation increases, what is often sacrificed is the amount of time you are able to spend getting to know your patients. I wonder if we are becoming better at knowing the chart but not necessarily better at knowing the person.

There are many reasons our documentation has become more detailed. We need accurate records for continuity of care, specialist referrals, insurance company requests, WorkSafe and ICBC documentation, billing, regulatory requirements, and medicolegal protection. Thorough documentation is essential, and I am certainly not suggesting that we should be less thorough.

But documentation takes more than time. It also takes attention. During a typical visit, I am listening to and examining a patient while forming a differential diagnosis, considering treatment options, checking medications, reviewing previous investigations, ordering tests, and making sure my notes accurately reflect what happened. By the end of the visit, I want the patient to leave safely and with a clear understanding of the plan. Despite the hundreds of things going on in my head during the visit, I often wonder about what I did not ask.

A medical chart can tell us a great deal, but it can never tell us everything. It may show that a patient has diabetes, depression, hypertension, or chronic pain. It may show that they missed appointments, stopped taking a medication, or declined an investigation. What it may not tell us is why.

Maybe the patient is working two jobs and cannot easily make appointments. Maybe they are caring for an elderly parent and putting that person's needs ahead of their own. Maybe they cannot afford the medication. Maybe they had a bad experience with the health care system and have become reluctant to seek care. Something that looks like "non-adherence" in the chart can make much more sense once we understand what is happening in the rest of a patient's life.

Family medicine has always been at its best when we understand illness in the context of someone's life.

That kind of understanding has always been one of the strengths of family medicine. Over time, we learn things that are difficult to capture in a note: what matters to someone, what they are worried about, who they rely on, and what is happening in their family or at work. These details may not appear on a problem list, but they often shape how we understand a patient's health and how we care for them.

I am certain that I provide evidence-based and comprehensive care to my patients. I know patients appreciate the fact that their doctor is competent, organized, and respectful of their time. But I also hope my patients feel listened to and feel that I know them as a whole person. With the time constraints of family medicine, I sometimes feel that I am not allowing enough time to do so thoroughly.

We have access to more information now than ever before. We have electronic medical records, specialist reports, hospital records, and PharmaNet readily available. We also have increasingly powerful artificial intelligence capable of summarizing years of medical history in seconds. These tools can help us practise better medicine. But having more information about someone is not the same as understanding them.

Perhaps that is where artificial intelligence may eventually help the most. Its greatest contribution to family medicine may not be helping us create longer or more detailed notes. It may simply be giving us some of our attention back. The question is, what will we do with that time?

If technology allows us to finish documentation more quickly, maybe we can listen a little longer. Maybe we can become a little more curious about the person sitting in front of us. Of course, there is also the possibility that every gain in efficiency will be filled with more tasks, more paperwork, and more patients. I hope that is not where we end up.

Family medicine has always been at its best when we understand illness in the context of someone's life. That understanding takes time, attention, and trust. The patient sitting across from us is more than a list of problems, a collection of test results, or a well-documented encounter. Their health is intertwined with every aspect of their lives.

I hope I continue to know my patients' charts well. But years from now, I hope my patients feel that I also knew the person behind the chart well, and that knowing them helped me care for them better. ■

—Yvonne Sin, MD

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: Rising demand for psychiatric diagnosis in urban British Columbia: Implications for the BC health care system

Thank you, Dr Chete, for an interesting and timely article [*BCMJ* 2026;68:234-239].

For the past few years, I have been diagnosing, treating, and longitudinally managing patients with increasingly complex mental health and neurodevelopmental presentations—often combinations of attention-deficit/hyperactivity disorder, autism spectrum disorder, posttraumatic stress disorder, generalized anxiety disorder, depression, bipolar disorder, and other comorbidities. I generally continue to follow these patients until they are stable, sometimes for several months, and then refer them back to their primary care physician for ongoing prescription refills.

Over the years, after seeing many of these patients, I started noticing some very interesting clinical patterns, particularly in patients with overlapping neurodevelopmental and psychiatric conditions.

I continue doing this work largely because I see the need and because of the enormous gratitude I receive from patients and families.

Am I happy with the current system? No.

Financially, I could probably be better off if I had remained in longitudinal family practice under the Longitudinal Family Physician Payment Model and focused on less complex cases. The amount of time and clinical work involved in managing these complex patients is simply not well recognized or well compensated in the current system.

Do I feel that this work is seen or appreciated at a system level? Unfortunately, absolutely not.

I have written repeatedly to Doctors of BC, the Ministry of Health, and the former Ministry of Mental Health and Addictions about these issues, with very few results.

Your article resonated with me because I believe there is an important role for family physicians with focused expertise and experience in mental health to help fill this gap—particularly physicians who are willing to assess and diagnose these complex patients, initiate and adjust treatment, follow them until they're stable, and then return them to their primary care physician.

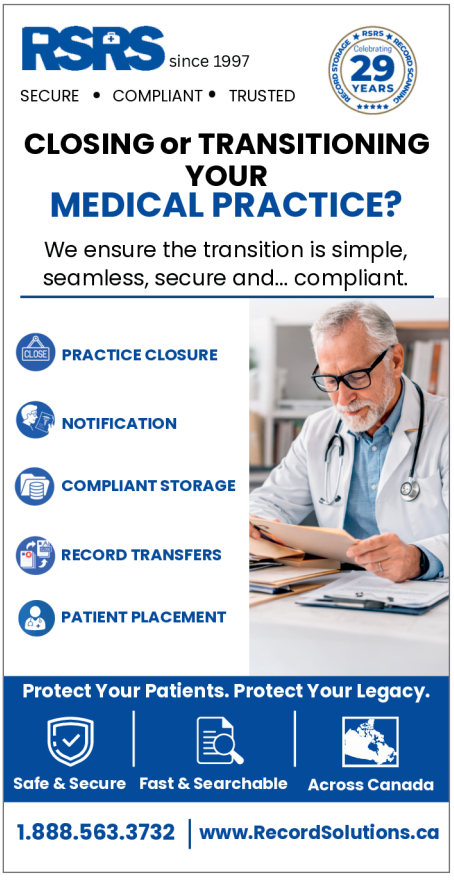
—Mitra Motamedi, MD
Vancouver

Re: Patient portals and radiology reports: Toward patient-centred communication

The recent article about the need for clear language in radiology reports that are to be understandable by the average patient [*BCMJ* 2026:68:211-213] put me in mind of a report I received in the 1970s: "The patient presented for a barium enema. The barium was administered. However, the procedure could not be completed as the individual absquatulated."

With a mental image of an examining room covered in a frightful mess, I reached for my dictionary, discovering that this early-19th-century American term indicated that the patient had simply pushed off and left the facility, full of contrast material.

—Anthony Walter, MB BCh
Coldstream



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Medical leadership: The value of lived experience

Medical leadership cannot be learned entirely in a boardroom, in a leadership or management course, or from a strategy document. To lead medicine well, you need to understand medicine. And there is no substitute for having lived it.

The strongest medical leaders are actively working in the health care system or have a meaningful recent history of doing so. They understand what it feels like to sit with a worried patient, make a difficult clinical decision, navigate an overbooked day, complete paperwork after hours, and carry the responsibility that comes with caring for another human being. They are beside their colleagues, supporting them, and experiencing the day-to-day challenges, whether in longitudinal family practice, in the hospital, or in a community specialist clinic. That lived experience matters.

Health care is complex. The pressures facing physicians today are enormous: workforce shortages, increasing demand, financial constraints, administrative burdens, changing models of care, and growing expectations. These challenges cannot be understood solely through data or policy papers. They need to be understood through the experiences of those delivering care. This is how leaders develop empathy, a key component of effective leadership.

This does not mean that every medical leader needs to practise full-time. Leadership inevitably requires time away from clinical work. But the further removed we become from the clinical environment, the greater the risk of losing sight of the people and experiences that should inform our decisions.

For me, medical leadership is fundamentally about stewardship. We have inherited a profession built over generations by physicians who cared deeply about their patients, their communities, and one another. Our responsibility is not to merely manage the profession today but to leave it stronger for those who come after us.

The strongest medical leaders are actively working in the health care system or have a meaningful recent history of doing so.

That means advocating for the conditions that enable physicians to provide excellent care, while recognizing our responsibility to patients and the broader health care system. It means caring about physicians' well-being—not because physicians are more important than the people we care for, but because a healthy, supported, and respected medical profession is better able to provide that care.

It also means remembering that medicine is a community. We are colleagues, teachers, learners, mentors, advocates, and, ultimately, patients ourselves. The culture we create matters. How we treat one another matters. Whether people feel heard, valued, and able to speak openly matters. Good leaders foster that culture by listening, not by leading with their opinions.

Leadership is about connection, credibility, and trust.

The best medical leaders I have encountered are not necessarily those with the biggest titles or the loudest voices. They understand the work, stay connected to those doing it, listen before they speak, and are prepared to stand up for the profession when it matters. They lead because they care.

Perhaps that is the most important quality of medical leadership: caring enough to stay connected to the work, the people who do it, and the future of the profession.

Many of those leaders are you—out in your communities.

As members of Doctors of BC, we all have a role in shaping that future, including choosing the physician leaders who will represent our profession and help guide our association. Voting in the 2026 Doctors of BC election is open from 3 to 18 November, and I encourage you to learn about the candidates and make your voice heard. You can learn more on the Doctors of BC elections web page (www.doctorsofbc.ca/elections).

Doctors of BC and the profession of medicine need leaders who live the work, understand its realities, listen to their colleagues, and care deeply about our profession's future. Ultimately, medical leadership is about being part of the profession and taking responsibility to help it thrive. ■

—Adam Thompson, MD
Doctors of BC President

A missing tale of psychiatry in British Columbia

The histories of three provincial institutions—Woodlands in New Westminster, Riverview Hospital in Coquitlam, and the psychiatric services at Vancouver General Hospital—reflect the evolving social, ethical, legal, and therapeutic approaches that have shaped mental health care in BC for generations.

Austin Lam, MD

During my psychiatry residency at the University of British Columbia, I found myself drawn to the histories of the institutions that have shaped mental health care in the province—histories sometimes unfamiliar to the clinicians and trainees who work in the spaces those institutions left behind. As the philosopher Martin Heidegger suggested, good historical knowledge can guard against uncreative repetition:[1] the risk of redesigning what has already been tried or forgetting what has already been learned, at considerable cost. Three institutions offer a window into how psychiatric care in British Columbia evolved across more than a century: Woodlands in New Westminster, Riverview Hospital in Coquitlam, and the psychiatric services at Vancouver General Hospital.



Woodlands, New Westminster. (Provincial Asylum for the Insane c. 1878.) Photograph by S.J. Thompson. Public domain. https://commons.wikimedia.org/wiki/File:NewWestminsterAsylum1878.jpg.

Woodlands, New Westminster (1878–1996)

The oldest of these institutions began in 1878 in New Westminster as the Provincial Lunatic Asylum (also referred to as the Provincial Asylum for the Insane), established to relieve overcrowding at an earlier facility in Victoria.[2] From its earliest years, the institution was marked by inadequate staffing, overcrowding, and conditions that drew criticism, including the use of handcuffs, leather mitts, and straitjackets.[2] In 1897, the name was changed to the BC Public Hospital for the Insane.[2] Conditions at the hospital prompted reform: Dr G.H. Manchester officially introduced the era of "moral treatment of insanity" in the 1902 annual report of the BC Public Hospital for the Insane, with principles that included medicine, good food, regular habits, employment, and recreation.[2,3]

In 1950, the facility was renamed Woodlands School, reflecting its focus on education and rehabilitation for people with intellectual disabilities, with other patients previously transferred to Riverview Hospital (then Essondale Hospital).[4] It reached its highest population in the late 1950s, housing over 1400 residents, including children and youth with developmental, behavioral, and psychiatric needs, as well as wards of the state.[4,5] Deinstitutionalization gradually reduced Woodlands' population, and it closed in 1996, making British Columbia the first province in Canada to close large institutions for people with developmental disabilities.[4]

Closure did not end the institution's public reckoning. In 2000, former residents came forward with accounts of

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This article has been peer reviewed.

physical, sexual, and emotional abuse. Former Ombudsperson Dulcie McCallum's 2001 report *The Need to Know* found evidence of systemic abuse and recommended a formal apology and further investigation.[5] A class action lawsuit was filed, and a settlement was reached in 2009; in 2018, the provincial government provided *ex gratia* compensation to eligible survivors of Woodlands.[6] The main building was demolished in 2011, witnessed by survivors who had spent years seeking acknowledgment. A memorial garden now marks part of the site, recognizing the more than 3300 former residents buried in an associated cemetery, many of whose grave markers were removed and discarded in 1977.[4]

Riverview Hospital, Coquitlam (1913–2012)

In 1904, the provincial government designated a 405-hectare site in Coquitlam for the proposed Hospital for the Mind at Mount Coquitlam. In 1913, before the hospital opened, the name was changed to Essondale Hospital, after the provincial secretary, Dr Henry Esson Young, to avoid confusion with the nearby town of Coquitlam. West Lawn, the first building, opened on 1 April 1913, and 340 male patients were later transferred from the BC Public Hospital for the Insane in New Westminster. Dr Young was influenced by ideas about the therapeutic value of nature, fresh air, and occupational activity, principles associated with the tradition of "moral treatment," which emphasized humane psychosocial care over confinement.[7] As Andrew Merrill documented in *Riverview Heritage Inventory*, each decade brought new buildings, programs, and thinking about psychiatric care. The Essondale campus was officially renamed Riverview Hospital in 1965, the name by which it became widely known until its closure in 2012.[7] At its peak in 1956, the hospital housed more than 4300 patients and employed approximately 2200 staff members.[7]

The latter half of the 20th century brought fundamental change. The Canadian Mental Health Association's 1963 report *More for the Mind* advocated for community-based care, and deinstitutionalization proceeded across Canada in the decades that followed.[8] In British Columbia, the number of psychiatric hospital beds decreased from 6371 to 3372 between 1965 and 1981, a decline of just over 47%.[9] Concerns about patient conditions were documented in the BC ombudsperson's 1994 report *Listening: A Review of Riverview Hospital*.[10] A prospective study of 189 former Riverview patients found that the majority achieved stable community placements, although a subset had needs that could not adequately be met outside a tertiary hospital setting.[11]

In the early 2000s, concerns about electroconvulsive therapy (ECT) prompted an audit; the resulting Ministry of Health report concluded that ECT was being administered safely and effectively.[12,13] The episode reflected tensions between therapeutic innovation, institutional oversight, and public accountability. Among those who shaped Riverview's later history was Dr Ralph Arrowsmith, known for his advocacy for marginalized populations and his commitment to psychiatric education. His reflections were documented in the 2012 film *Consciousness*, part of the Riverview Hospital Story Project, one of the few sustained efforts to preserve firsthand accounts of the institution before its closure.[14]

Riverview closed in 2012, a decision former BC attorney general Wally Oppal later described as the wrong one, "with 20/20 hindsight."[15] The limited expansion of community-based mental health services relative to institutional closure contributed to ongoing challenges surrounding homelessness, addiction, and access to care.[16] The Riverview lands, known as səmiq̓ʷəʔelə ("Place of the Great Blue Heron"), are now home to the Red Fish Healing Centre for Mental Health and Addiction and the Provincial Assessment Centre, on the traditional territories of the kʷikʷəƛ̓əm (Kwikwetlem First Nation).[7] Growing up near what became the Riverview Heights neighborhood, I often passed these buildings before understanding their historical significance, a reminder that institutional histories can persist in a landscape long after the institutions are gone.

The shift toward hospital-based psychiatric services reflected a longer reorientation in psychiatry. As Dr Robert Nathan



Riverview Hospital, West Lawn building, Coquitlam. Item EH.003—Essondale's west lawn—straight-on view ca. 1914. CA CCOQ C5-S01-SS02-EH.003, City of Coquitlam Archives. Republished with permission from City of Coquitlam Archives; public domain. https://searcharchives.coquitlam.ca/index.php/essondales-west-lawn-straight-on-view-ca-1914.

has argued, a model of the "psychopathic hospital," situating psychiatric care in general hospitals rather than stand-alone asylums, had emerged in the late 19th century, reflecting psychiatry's effort to align with general medicine and treat patients voluntarily.[17] This model positioned psychiatry as a branch of medicine rather than a system of custodial management, with implications for how patients were admitted, treated, and discharged. The development of psychiatric services at Vancouver General Hospital was, therefore, part of a much older professional transformation.

Psychiatric services at Vancouver General Hospital (1915–present)

Psychiatric services at Vancouver General Hospital (VGH) developed alongside broader shifts in Canadian psychiatry toward treatment in general hospitals rather than stand-alone asylums. In 1915, VGH opened its first psychiatric ward, followed in 1920 by the establishment of a psychiatric clinic staffed by a psychiatrist, psychologist, and psychiatric social worker.[18]

By the early 2000s, CBC News investigations highlighted deteriorating conditions in VGH's psychiatric facilities, including overcrowding, aging infrastructure, inadequate security, poor ventilation, and limited patient space.[19,20] Hospital figures reportedly showed that dispensing of antipsychotic medications had more than doubled over a 2-year period, a pattern the BC Nurses' Union characterized as a form of "chemical restraint" in an overcrowded environment that staff felt could not otherwise be managed.[19] The director of mental health services acknowledged publicly that the building was "falling apart" and that renovation was no longer viable.[19] These conditions made visible a gap between the principles guiding psychiatric care and the physical environments in which care was delivered. The Joseph and Rosalie Segal and Family Health Centre, which subsequently opened on the VGH campus, reflected changing expectations about what a therapeutic environment should provide: private rooms, secure outdoor space, and a physical setting designed around the dignity and safety of patients.

The evolving legal framework

Alongside these institutional histories, legal frameworks governing psychiatric care evolved across successive generations in response to legal challenges, changing standards of consent, and expanding conceptions of patient rights. While reviewing provincial ECT guidelines published in 2002,[21] I encountered reference to the Health Care and Care Facility Review Board, established under the Health Care (Consent) and Care Facility (Admission) Act and later dissolved following legislative amendments in the early 2000s.[22] Its disappearance from the legislative landscape is a small illustration of how oversight mechanisms can be created, modified, and dissolved in a single generation, often without leaving clear traces in clinical practice or institutional memory. More recent decisions, including *A.H. v. Fraser Health Authority*, have continued to shape frameworks surrounding detention, consent, and patient autonomy.[23] These developments are easy to overlook in day-to-day clinical practice, yet they define the conditions under which patients are admitted, treated, and discharged.

Conclusions

These histories trace the arc of mental health care in BC, from the era of large custodial institutions through deinstitutionalization and into the present. Each institution reflects the social values, therapeutic assumptions, and political priorities of its time and carries forward unresolved questions about who receives care, under what conditions, and who bears the cost when standards of safety and dignity are not met.

Institutional memory fades quickly once physical spaces disappear. Woodlands has been demolished, Riverview's grounds are being gradually repurposed, and the older VGH psychiatric wards are now green space. New facilities and policies do not emerge in isolation; they are shaped, whether acknowledged or not, by the generations of clinicians, patients, administrators, and communities that preceded them. The tale of psychiatry in British Columbia is not missing because nothing happened. It is missing because the histories embedded in these institutions have not yet been fully told. ■



Vancouver General Hospital. Item : CVA 586-4385—Vancouver General Hospital [on] West 12th Avenue. Williams Bros. Photographers Collection, City of Vancouver Archives. Public domain. https://searcharchives.vancouver.ca/vancouver-general-hospital-on-west-12th-avenue.

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Competing interests

None declared.

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Pancreatic cancer, part 1

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This editorial has been peer reviewed.

Pancreatic cancer is the deadliest cancer in British Columbia, with a 5-year survival rate of only 16%.[1] Although it is the 11th-most diagnosed cancer in the province, it is the 3rd-leading cause of cancer death.[2]

In this issue of the *BCMJ*, I am pleased to present three articles on pancreatic cancer from a multidisciplinary group of physicians, nurse practitioners, educators, and researchers from across BC.

Increasing availability of genetic testing for patients and families of patients diagnosed with cancer has led to the identification of pathogenic germline variants associated with a susceptibility to pancreatic cancer. Screening of the pancreas is recommended for certain high-risk individuals and is available at specialized centres. The first article aims to increase awareness of hereditary pancreatic cancer syndromes, the indications for genetic testing, and current recommendations for pancreas screening.

The lethal nature of pancreatic cancer often creates anxiety among both patients and physicians when an abnormality is detected in the pancreas. Cross-sectional abdominal imaging has become increasingly common and more accurate, which has allowed for the detection of even very small pancreatic lesions. Most of these are cystic lesions. Although only a minority of pancreatic cystic lesions will progress to a high-risk precursor or pancreatic cancer, that possibility weighs heavily on both patients and clinicians. Uncertainty regarding the natural history of these lesions, conflicting guidelines, and inconsistent messaging across radiology reports and among gastroenterologists and surgeons have made the management of cystic lesions challenging. The second article reviews the spectrum of pancreatic cystic lesions, summarizes current evidence and guidelines, and offers a pragmatic approach to evaluation and management.

Pancreatic neuroendocrine tumors are increasing in incidence but remain a less common form of pancreatic malignancy, with a natural history and management strategy distinct from pancreatic adenocarcinoma. The third article provides a comprehensive review of pancreatic neuroendocrine tumors, including their classification, specific tests used to establish a diagnosis, and oncologic management.

Our goal in this issue is to highlight recent advances in the diagnosis of patients who have an increased risk of pancreatic cancer and review pancreatic neuroendocrine tumors. The December issue will discuss the management of patients with pancreatic adenocarcinoma. We hope this collection of articles provides clinicians with practical guidance for patient care while increasing awareness of available diagnostic services and patient resources across BC. ■

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Pancreas screening in high-risk individuals

Pancreas screening of high-risk individuals has the potential to save lives, but the ideal screening strategy is not currently fully realized.

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ABSTRACT: Pancreas screening is recommended for individuals who are at higher risk of developing pancreatic cancer due to family history or a documented germline pathogenic variant predisposing them. Screening entails annual imaging of the pancreas and screening for diabetes mellitus. The goals of screening are to identify high-risk precursor lesions and early-stage pancreatic cancer that is amenable to surgical resection, thereby reducing deaths due to pancreatic cancer. Pancreas screening is limited by the ability of current imaging technology to accurately distinguish low- and high-risk precursor lesions. Research is underway to develop a serum test for pancreas screening.

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Most pancreatic cancer is pancreatic ductal adenocarcinoma (PDAC). Although PDAC has the 10th-highest incidence rate in BC, it is the 3rd-leading cause of cancer-related mortality, after lung and colorectal cancers,[1] and the 5-year survival rate is 10%.[2]

Screening for PDAC has been evaluated by the United States Preventive Services Task Force, with an evidence review of the potential benefits and harms of pancreas screening. The task force recommended against screening average-risk individuals.[3] However, individuals at increased risk of PDAC due to family history and/or a documented germline pathogenic variant or a likely pathogenic variant in an established PDAC susceptibility gene are recommended to consider pancreas screening.[4-9] Cancer susceptibility genes that confer moderate to high PDAC risk through rare pathogenic variants include *ATM*, *BRCA1*, *BRCA2*, *CDKN2A*, *MLH1*, *MSH2*, *MSH6*, *PMS2*, *EPCAM*, *PALB2*, *STK11*, and *TP53*, as well as several genes associated with hereditary pancreatitis.[10] Although efforts are underway to develop polygenic risk scores based on common genetic factors associated with PDAC susceptibility, they are not currently in clinical use.[11]

Key points

Assessment and management of individuals at risk for pancreatic cancer:

- Assess family history.
- Modify risk factors.
- Discuss the benefits, harms, and limitations of screening.
- Screen for diabetes.
- Refer to the BC Cancer Hereditary Cancer Program.
- Refer to an endoscopic ultrasound centre.

Other important risk factors for PDAC include smoking, alcohol use, obesity, chronic pancreatitis, and new-onset diabetes mellitus.[12] Individuals over 50 years of age who have had a diagnosis of diabetes in the last 3 years have a 7.9-fold increased risk of pancreatic cancer.[13] Similarly, obesity, defined as a BMI of 30 or higher, is associated with a 1.19-fold increased risk per unit increase in BMI, and alcohol use is considered to have a dose-dependent risk association exclusively in men, with heavy alcohol consumption (≥ 35 units/week) and a history of binge drinking (≥ 35 units/week, > 1 month) conferring a 2.6-fold and 3.5-fold increased risk of pancreatic cancer, respectively.[14,15]

Pancreatic neuroendocrine tumors are a less common type of pancreatic cancer and are discussed in detail by Sanders and colleagues in another article in this issue of the

BCMJ. Several hereditary cancer syndromes are known to increase the risk of pancreatic neuroendocrine tumors. Although most are sporadic, 10% to 20% occur in the setting of an inherited syndrome, including multiple endocrine neoplasia type 1 and von Hippel-Lindau disease. Multiple endocrine neoplasia type 1 is an inherited predisposition to neoplasms of the parathyroid gland, pituitary gland, thymus gland, and lung, and to pancreatic neuroendocrine tumors. Patients with multiple endocrine neoplasia type 1 are offered pancreatic cancer screening starting between 8 and 15 years of age, with MRI or endoscopic ultrasound every 1 to 3 years.[16]

Who to screen

Eligibility criteria for pancreatic cancer screening vary slightly between guidelines.[4-8] Because the risk of pancreatic cancer varies across genes and family history of pancreatic cancer is both an independent and an additive risk factor, current screening guidelines reflect this complexity by varying recommendations according to the underlying gene and, for some genes, the presence or absence of a family history of pancreatic cancer **[Table]**. Screening generally commences at 50 years of age. Individuals with familial atypical multiple mole and melanoma syndrome or hereditary pancreatitis are recommended to begin pancreas screening at 40 years of age, while those with Peutz-Jeghers syndrome are recommended to begin screening at 35 years of age. For individuals with family members diagnosed with PDAC at younger ages, screening begins when the individual is 10 years younger than the age of diagnosis of the youngest affected family member.

Familial pancreatic cancer

Familial pancreatic cancer is defined as cancer that occurs in a pair of first-degree relatives (parent and child or two siblings) without a pathogenic variant in a pancreatic cancer susceptibility gene. Individuals who are a first-degree relative to one of the affected family members are eligible for screening. Familial pancreatic cancer yields an 8% to 12% lifetime risk of PDAC, while three or more first-degree relatives equates to a 40% lifetime risk.[17] Furthermore, an earlier age of familial pancreatic cancer diagnosis confers an increased risk of early-onset pancreatic cancer in high-risk individuals, which suggests a risk escalation based on age of onset.[18,19]

Hereditary pancreatic cancer

In addition to familial risk, documented pathogenic variants in pancreatic cancer susceptibility genes are an important risk factor for PDAC. The lifetime risk of PDAC varies depending on the affected gene **[Table]**. The risk is further increased by a family history of PDAC and varies depending on the number of relatives affected, the closeness of the relationship, and the age of diagnosis. Lifestyle and environmental factors further influence the risk.

The following syndromes are autosomal dominant hereditary cancer susceptibility syndromes in which inheriting one copy of the gene with a pathogenic variant is sufficient to significantly increase cancer risk, even if a normal second copy of the gene is also inherited. Once a patient is found to have a pathogenic variant, their parents, siblings, and children are at a 50% risk, and genetic counseling and testing are recommended.

Peutz-Jeghers syndrome

Peutz-Jeghers syndrome is caused by pathogenic variants in the *STK11* gene. The syndrome is characterized by mucosal pigmentation in the mouth and perioral skin and pathologically distinct hamartomas throughout the gastrointestinal tract. Individuals with Peutz-Jeghers syndrome are at high risk of PDAC as well as cancers involving the gastrointestinal tract, breast, ovary, uterus, cervix, and testes.[6]

Familial atypical multiple mole and melanoma syndrome

Familial atypical multiple mole and melanoma syndrome is associated with pathogenic variants in the *CDKN2A* gene and presents with multiple moles and melanomas.

TABLE. Hereditary syndromes and risk of pancreatic ductal adenocarcinoma (PDAC).[4,17]

Syndrome	Criteria/gene	Lifetime risk of PDAC (%)	Age to screen (years)*
Familial pancreatic cancer	A pair of first-degree relatives with PDAC	8–12	50
Peutz-Jeghers syndrome	*STK11*	11–36	35
***PRSS1*-associated pancreatitis**	*PRSS1*	10	40
Familial atypical multiple mole melanoma syndrome	*CDKN2A*	16–20	40
Hereditary breast and ovarian syndrome	*BRCA1*† *BRCA2* *PALB2*	3–5 5–8 5–8	50
Ataxia-telangiectasia mutated	*ATM*†	5–10	50
Li-Fraumeni syndrome	*TP53* + family history	Unknown	50
Lynch syndrome	*MLH1*, *MSH2*, or *MSH5* + family history	0.5–7.0	50

* Or 10 years younger than the age of PDAC diagnosis in the youngest affected relative, whichever comes first. For hereditary pancreatitis, screening may begin 20 years after the onset of clinical pancreatitis or at 40 years of age, whichever comes first.

† Guidelines disagree about whether screening should be offered only to those with a family history of PDAC.

Hereditary breast and ovarian cancer syndrome

Pathogenic variants in the *BRCA1*, *BRCA2*, and *PALB2* genes are associated with a high risk of breast, ovarian, pancreatic, and prostate cancer. Estimates range from 1 in 200 to 1 in 400 individuals that carry a pathogenic variant in the *BRCA1* or *BRCA2* gene, but there is a higher frequency in some populations.[20] In the Ashkenazi Jewish population, approximately 1 in 40 individuals are carriers,[21] which has prompted local awareness campaigns to inform individuals about genetic testing options, such as the BRCAinBC initiative (https://brcainbc.ca/).

Ataxia-telangiectasia syndrome

Individuals with a single pathogenic variant in *ATM* are at increased risk of PDAC, breast cancer, and prostate cancer and represent approximately 1/242 of the population.[22] In addition to the autosomal dominant risk of cancer, individuals with a pathogenic variant in both copies of the *ATM* gene are afflicted with ataxia-telangiectasia syndrome, characterized by a progressive neurodegenerative disorder, oculocutaneous telangiectasias, and immunodeficiency.

Li-Fraumeni syndrome

Li-Fraumeni syndrome is caused by a rare pathogenic variant in the *TP53* gene. It is associated with a very high lifetime risk of cancer, often with early onset of disease, particularly breast cancer, sarcomas, brain tumors, and adrenocortical carcinoma, as well as several other malignancies, including pancreatic and prostate cancers.

Lynch syndrome

Lynch syndrome is another relatively common cancer predisposition. It is associated with pathogenic variants in the mismatch repair genes (*MLH1*, *MSH2*, *MSH6*, and *PMS2*) and the 3′ terminal deletions of *EPCAM* causing epigenetic silencing of *MSH2*. Individuals with Lynch syndrome represent at least 1/279 of the population and are at increased risk of uterine and colorectal cancers and, less commonly, ovarian, gastric, small intestinal, pancreatic, hepatobiliary, urinary tract, brain, and sebaceous carcinomas.[23]

PRSS1-associated pancreatitis

Pancreatitis associated with genetic risk factors, also known as hereditary pancreatitis or familial pancreatitis, may manifest as acute recurrent pancreatitis or chronic pancreatitis with the phenotype mediated by other known risk factors for pancreatitis, such as smoking and alcohol use. *PRSS1*-associated pancreatitis is inherited in an autosomal dominant fashion and carries a high associated risk of PDAC. *CFTR*-associated pancreatitis usually presents as chronic pancreatitis and appears to have a small increased risk of PDAC. *SPINK1*-associated pancreatitis presents in an autosomal recessive fashion and does not appear to significantly increase the risk of PDAC. Genetic testing should be offered to patients with pancreatitis in childhood or young adulthood, particularly if there is a family history of pancreatitis. Individuals with pancreatitis and a *PRSS1* pathogenic variant are recommended to begin screening for pancreatic cancer at 40 years of age or 20 years after the initial diagnosis of pancreatitis, whichever is earlier. Screening is not recommended for individuals with pancreatitis and negative genetic testing. Interpretation of pancreas imaging in the context of chronic pancreatitis may be challenging due to pancreatic calcifications, fibrosis, and atrophy.[24]

Guideline recommendations

There are several expert consensus guidelines for pancreas screening. They align for the highest-risk conditions: Peutz-Jeghers syndrome, familial atypical multiple mole and melanoma syndrome, familial pancreatic cancer, and *PRSS1*-associated pancreatitis. However, there is controversy about whether individuals with other inherited syndromes should undergo pancreas screening in the absence of a family history of PDAC in a first- or second-degree relative. For instance, the National Comprehensive Cancer Network guidelines recommend screening for individuals with a pathogenic variant in *ATM* or *BRCA2*, regardless of family history, but require individuals with a *BRCA1* pathogenic variant to have a family history of PDAC that is additionally presumed or confirmed to be on the same side.[4] In contrast, the American Society for Gastrointestinal Endoscopy guidelines do not require individuals with *BRCA1*, *BRCA2*, or *PALB2* pathogenic variants to have a family history of PDAC but do require this of individuals with an *ATM* pathogenic variant.[25] The Cancer of the Pancreas Screening Study guidelines recommend screening individuals with pathogenic variants in *BRCA1*, *BRCA2*, *PALB2*, or *ATM* only when a family history of PDAC is present.[5] All guidelines recommend that screening be performed only in the setting of a family history of PDAC for individuals with Lynch syndrome or *TP53* pathogenic variants.

Guidelines emphasize that pancreas screening should be conducted only after an informed discussion with the patient that addresses the limitations, potential harms, and incidence of indeterminate abnormalities. Ideally, screening is performed at a high-volume expert centre and in the setting of a longitudinal study.

Genetic testing in BC

Genetic testing in BC is available through three pathways: mainstream testing for individuals diagnosed with PDAC or a pancreatic neuroendocrine tumor, referral to the BC Cancer Hereditary Cancer Program for individuals with a family history of pancreatic cancer, and referral to the Provincial Medical Genetics Program at BC Women's Hospital and Health Centre for individuals with a history that is of concern for hereditary pancreatitis. Mainstream testing refers to genetic testing ordered and interpreted by an individual's doctor or nurse practitioner rather than the Hereditary Cancer Program.

1. Mainstream genetic testing for individuals diagnosed with pancreatic cancer

All patients with histologically confirmed PDAC or pancreatic neuroendocrine

tumors should undergo mainstream genetic testing to guide their treatment and assess their relatives' risk of pancreatic cancer.[26]

- Testing may be ordered by any physician or nurse practitioner via "mainstreaming."
  - Complete the Hereditary Cancer Multigene Panel requisition **[Box]**.
  - Blood may be drawn at any BC lab.
  - Results are available in 4 to 6 weeks.
- Referral to the Hereditary Cancer Program is NOT required.
- In Canada, genetic information is protected by the Genetic Non-Discrimination Act.
  - Individuals cannot be required to disclose the results of a genetic test.
  - Individuals are protected from discrimination on the grounds of genetic characteristics.

BOX. Referral forms and eligibility criteria.

- **BC Cancer Hereditary Cancer Program referral form:** www.bccancer.bc.ca/coping-and-support-site/Documents/Hereditary%20Cancer%20Program/HCP%20Referral%20Form.pdf
- **BC Cancer Hereditary Cancer Program eligibility criteria:** www.bccancer.bc.ca/coping-and-support-site/Documents/Hereditary%20Cancer%20Program/HCP%20Eligibility%20Criteria.pdf
- **BC Cancer Cancer Genetics and Genomics Laboratory Hereditary Cancer Multi-Gene Panel referral form (mainstream genetic testing):** https://cancergeneticslab.ca/wp-content/uploads//CACG_CGL_3017-CGL-Hereditary-Cancer-Multi-Gene-Panel-Requisition.pdf
- **BC Women's Hospital and Health Centre Medical Genetics Clinic General Referral Form:** www.bcwomens.ca/Medical-Genetics-Site/Documents/General-Triage-Referral-Form-Fillable.pdf

- Reminders to order mainstream testing are currently included in pancreatic resection pathology reports and will soon be included in pancreas biopsy pathology reports.
- If a pathogenic variant is identified, a referral is automatically submitted to the Hereditary Cancer Program for posttest genetic counseling, information, and support for the patient and their relatives.

Mainstream testing is the standard of care because it achieves higher testing rates, produces faster results, and reaches a more diverse group of patients than referrals to the Hereditary Cancer Program.[27]

Additional information on mainstream hereditary cancer testing, including patient videos and FAQs, is available from BC Cancer's Cancer Genetics and Genomics Laboratory (https://cancergeneticslab.ca/hereditary/mainstreamed-testing/).

2. Referral to the Hereditary Cancer Program for individuals with a family history of pancreatic cancer

The eligibility criteria for a Hereditary Cancer Program referral have recently been updated to prioritize individuals who are most likely to have hereditary cancer risk, including:

- People from families where a pathogenic variant has been identified.
- People with a first-degree relative (parent, child, or sibling) who has specific cancers, such as PDAC or pancreatic neuroendocrine tumors, if multigene hereditary cancer testing was not performed in their affected relative.
- People without pancreatic cancer who are predicted to have a higher risk of hereditary cancer based on validated risk prediction tools.

For more information and to access the referral form, see the resources provided in the **Box**.

Note: All the pancreatic cancer susceptibility genes listed in the **Table** (except for those related to hereditary pancreatitis) confer multisite cancer risks that exceed the risks for pancreatic cancer and may appear more prominent in the patient's personal and/or family history. Patients and family members with suspicion of these conditions may meet additional actionable mainstream genetic testing or Hereditary Cancer Program referral criteria.

Additionally, all clinicians should reinforce the importance of cascade genetic testing or familial variant testing in families in which a hereditary cancer susceptibility has been identified to enable cancer risk stratification and early detection strategies in other family members.

3. Referral to the Provincial Medical Genetics Program for individuals with a personal or family history of hereditary pancreatitis

BC Women's offers genetic testing for individuals with a history of pancreatitis that is consistent with *PRSS1* and associated genetic risk factors. Physicians and nurse practitioners may refer patients using the referral form **[Box]**.

How to screen

Guidelines recommend annual imaging of the pancreas with MRI with magnetic retrograde cholangiopancreatography (MRCP) and/or endoscopic ultrasound (EUS); CT is reserved for individuals who are unable to undergo MRI or EUS.[4-8] Screening programs often have participants alternate between MRI and EUS each year, where feasible. For instance, an individual may undergo EUS in their first year of screening, MRI the next year, and EUS in the third year.

MRI/MRCP and EUS appear to be similar in the detection of high-risk pancreatic lesions and high-risk resectable lesions.[7] Several centres in BC offer alternating EUS and MRI/MRCP to high-risk individuals undergoing pancreas screening. Other factors that determine screening strategy include local availability and expertise and patient preferences. Ideally, either screening test is performed in a standardized manner, including image capture and documentation, in a high-volume centre.[28,29]

In addition to annual pancreas imaging,

annual screening for diabetes mellitus should be conducted starting at 40 years of age in patients with a hereditary or familial predisposition that meets the threshold for pancreas screening.

Goals of screening

The primary goal of pancreas screening in high-risk individuals is to identify high-risk precursor lesions and early-stage PDAC that is amenable to surgical resection, thereby reducing PDAC incidence and mortality. Target precursor lesions are pancreatic intraepithelial neoplasia (PanIN) with high-grade dysplasia, intraductal papillary mucinous neoplasm with high-grade dysplasia, and mucinous cystic neoplasms.[30] Intraductal papillary mucinous neoplasm and mucinous cystic neoplasms are cystic lesions; they are discussed in detail by Kakkar and colleagues in another article in this issue of the *BCMJ*.

PanIN are noncystic, microscopic, flat or papillary mucinous epithelial neoplasia that are less than 5 mm and arise from the pancreatic ducts.[31] PanIN are classified as either low or high grade based on the degree of dysplasia. PanIN are commonly diagnosed in pancreatic surgical resections, but high-grade PanIN are usually identified only adjacent to PDAC.[31] More than 80% of PDAC cases are thought to arise from PanIN, with a precancerous lesion present for many years prior to cancer development.[32] Due to their small size, PanIN are not visible using current MRI or EUS technology. However, PanIN are often associated with parenchymal changes, including lobulation and atrophy surrounding the area of neoplasia. While these findings are subtle, it may be possible to detect them with EUS.[30]

Benefits of screening

Cohort studies suggest that pancreas screening in high-risk individuals leads to early detection, thereby improving survival outcomes. A multicentre study demonstrated that high-risk individuals with screen-detected cancers had a higher proportion of stage 1 cancer than matched controls from the general population and a 5-year overall survival rate of 50% compared with 10% in the control group.[33] These findings are supported by other studies.[34] However, the development of late-stage PDAC despite screening continues to account for approximately 60% of PDAC cases in high-risk individuals.[35,36]

Cohort studies suggest that pancreas screening in high-risk individuals leads to early detection, thereby improving survival outcomes.

Beyond survival benefits, a recent systematic literature review on the psychological impacts of pancreas screening in high-risk individuals reported that participants felt that screening reduced their risk of developing cancer, which reduced their cancer-related worries.[37]

Harms of screening

Adverse events associated with EUS include hemorrhage, infection, pancreatitis, and perforation, but they occur in less than 1% of patients. An older retrospective study reported adverse events in 2.5% of 355 patients who underwent EUS with fine-needle aspiration of solid pancreatic masses.[38] MRI/MRCP risk is limited to claustrophobia and allergic reactions to IV contrast; relative MRI/MRCP contraindications include implanted devices and metallic foreign bodies.[25,39]

Harms also arise when screening leads to unnecessary or low-yield surgeries due to the limitations of current technology to differentiate low- from high-grade precursor lesions. Low-yield surgeries include resections that identify low-grade mucinous cystic lesions or low-grade PanIN. In the American Society for Gastrointestinal Endoscopy's systematic review, low-yield surgeries were identified in 3% of the total screened population and in nearly 50% of high-risk individuals who underwent surgery.[25] Low-yield surgeries are associated with morbidity in approximately 20% of patients, including fistulae, postoperative diabetes, cholangitis, hernias, delayed gastric emptying, and malabsorption. This highlights the need for screening techniques that are better at distinguishing low- and high-grade precursor lesions.[25]

Cost-effectiveness of screening

A cost-effectiveness analysis of different screening strategies in high-risk individuals compared with no screening demonstrated that screening is cost-effective but depends on the amount a payer is willing to pay for one life-year gained.[40,41] MRI tends to be more cost-effective in lower-risk individuals unless the cost of MRI rises. In contrast, EUS is more cost-effective in higher-risk individuals due to the ability to perform tissue sampling with EUS.[40]

Future approaches

In addition to blood glucose monitoring, other biomarkers for detecting high-risk precancerous lesions and early-stage cancer in individuals at risk are being explored. Carbohydrate antigen 19-9 (CA 19-9) has been evaluated in pancreatic cancer diagnosis, prognosis, and postoperative recurrence, with some success. However, sensitivity and specificity are lowest for small tumors, and up to 10% of the population does not express CA 19-9.

Emerging techniques for identifying novel serum biomarkers, such as circulating tumor DNA, aberrant microRNAs, and tumor-specific proteins and metabolites, are being studied, individually and in combination with each other and CA 19-9, for several malignancies, including PDAC.[42] While some studies have shown promise, additional research on screening high-risk individuals prior to clinical implementation is needed.

At present, there is no blood-based test available to confidently screen for pancreatic cancer in either high-risk individuals or the general public.

The BC Cancer Familial Pancreatic Cancer Program/Hereditary Cancer Program is a site for the Pancreatic Cancer Early Detection (PRECEDE) Consortium (https://precedestudy.org/). The consortium is an international, multi-institutional, collaborative group of experts who are focused on increasing the survival rate of pancreatic cancer patients by improving early detection, screening, risk modeling, and prevention for those with a heritable risk of pancreatic cancer, through a novel model of collaboration and data sharing. PRECEDE's mission is to transform the early detection and prevention of pancreatic cancer, with the aim of increasing the 5-year survival rate of pancreatic cancer patients from 10% to 50% within the next 10 years.

Summary

Pancreas screening of high-risk individuals has the potential to save lives, but the ideal screening strategy is not currently fully realized. Improving our ability to identify those who are at high risk and subsequently detect early PDAC and high-grade precursor lesions will be achieved through collaborative research initiatives such as PRECEDE, innovative technology, training of skilled radiologists and endosonographers, and development of accurate biomarkers. ■

Competing interests

Ms Cremin holds a Michael Smith Health Research BC Health Professional Award for research studies related to mainstream testing and increasing access to hereditary cancer testing among patients with pancreatic cancer. The remaining authors have no competing interests to declare.

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Pancreatic cystic lesions in adults: Diagnosis and management

Early recognition, risk stratification, and coordinated surveillance are critical to the timely detection of malignant pancreatic cystic lesions.

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ABSTRACT: Pancreatic cystic lesions are being increasingly detected with cross-sectional imaging. Although most are benign, a subset carry malignant potential. Accurate classification into nonneoplastic and neoplastic subtypes is essential, because lesions such as intraductal papillary mucinous neoplasms and mucinous cystic neoplasms warrant closer monitoring or surgical evaluation. Diagnosis is guided by cross-sectional imaging. Magnetic resonance cholangiopancreatography is the preferred modality; endoscopic ultrasonography is reserved for indeterminate or higher-risk lesions. Major society guidelines use high-risk and worrisome features to direct referral, intervention, or surveillance, although thresholds vary. Most asymptomatic cysts without concerning features can be safely followed with interval imaging, whereas lesions with mural nodules, ductal dilation, or suspicious cytology require specialist assessment. Early recognition, risk stratification, and coordinated surveillance are critical to enable timely detection of malignancy. This review focuses on the adult population, in whom pancreatic cystic lesions are most prevalent.

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Pancreatic cystic lesions are being increasingly encountered in clinical practice due to the increased use of cross-sectional imaging.[1] Incidental pancreatic cysts are identified in up to 20% of patients undergoing abdominal MRI, and prevalence increases to up to 40% with age (particularly in those over age 70) and underlying comorbidities, such as diabetes or chronic pancreatitis.[2,3]

While most pancreatic cystic lesions are benign and asymptomatic, a clinically important subset represents premalignant or malignant neoplasms. The incidental discovery of a pancreatic cyst often raises concerns for both clinicians and patients, particularly given the high mortality rate associated with pancreatic adenocarcinoma. However, most lesions follow an indolent course, and distinguishing between low- and high-risk cysts is critical to avoid unnecessary interventions while ensuring timely cancer detection. Early identification of cyst type guides diagnostic testing, informs malignancy risk, and determines the need for specialist referral. Clinical decision making, however, is complicated by the variability among major society guidelines, which differ in their definitions of high-risk features, indications for further workup, and recommended surveillance intervals.[2-6]

In this context, primary care physicians play a pivotal role in the initial identification and interpretation of radiologic findings, as well as in counseling patients, initiating appropriate surveillance, and facilitating specialist referral when warranted. This review provides a practical, evidence-based overview of pancreatic cystic lesions for primary care. It focuses on classification, natural history, and current guideline recommendations for workup, surveillance, and referral.

This review focuses specifically on the diagnosis and management of pancreatic cystic lesions in adults, the population in which these lesions are overwhelmingly prevalent. Pancreatic cystic lesions are rare in children and typically differ in etiology (e.g., congenital, hereditary, or syndromic causes). When a cyst is identified incidentally in a child, magnetic resonance cholangiopancreatography (MRCP) is the imaging test of choice. It can be ordered as a dedicated pediatric pancreatic-protocol MRI/MRCP, with referral to pediatric gastroenterology or surgery given the differing management pathways in this age group.

Classification

Pancreatic cystic lesions represent a heterogeneous group of entities with varying causes, epithelial linings, mucin production, and, most importantly, malignant potential. Broadly, they can be categorized into nonneoplastic and neoplastic lesions **[Figure 1]**. This classification is clinically important, because the risk of malignancy varies markedly among subtypes, and appropriate categorization guides diagnostic workup, referral, and surveillance strategies.

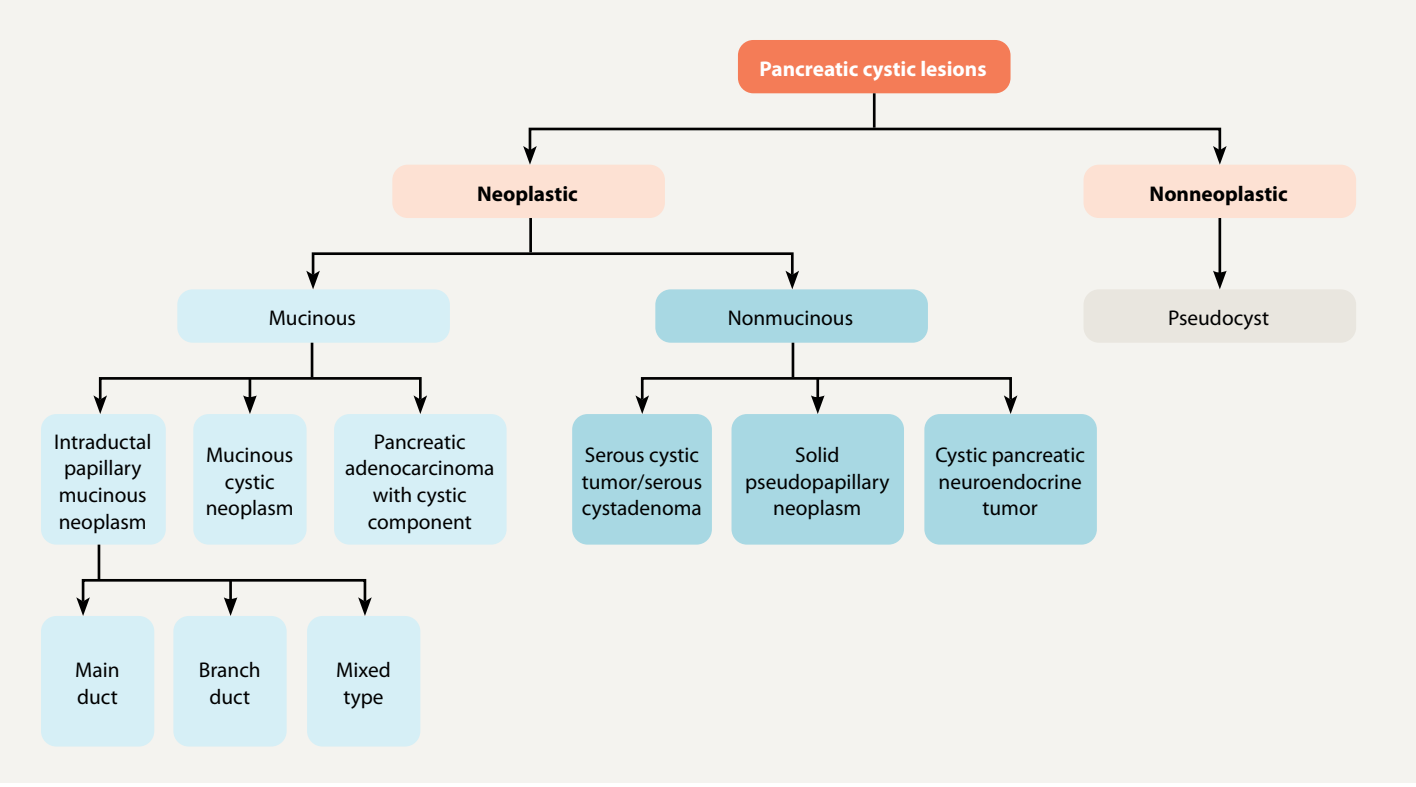


FIGURE 1. Classification of pancreatic cystic lesions.

Nonneoplastic

Most nonneoplastic lesions are pseudocysts that represent collections of pancreatic secretions that have extravasated from a duct disrupted by inflammation or obstruction.[7] They typically occur in patients with a history of acute or chronic pancreatitis or abdominal trauma.

Other benign and uncommon cyst types include simple (true epithelial), lymphoepithelial, and retention cysts. Simple cysts are unilocular, are lined by a single layer of epithelium, lack communication with the pancreatic ductal system, and carry no malignant potential.[8] Lymphoepithelial cysts typically occur in middle-aged men; they can arise anywhere in the pancreas and are often multilocular, with a mean size of approximately 5 cm. They are benign but may mimic pseudocysts on imaging.[9,10] Retention cysts represent ductal side-branch dilation secondary to obstruction (e.g., calculi, mucin). They may have mucinous lining and can resemble intraductal papillary mucinous neoplasms, although they lack neoplastic potential.[11]

Neoplastic

Neoplastic pancreatic cystic lesions encompass a diverse group of cyst-forming tumors with varying malignant potential. The main subtypes are mucinous cystic neoplasms, intraductal papillary mucinous neoplasms, solid pseudopapillary neoplasms, serous cystic neoplasms/serous cystadenomas, cystic pancreatic neuroendocrine tumors, and cystic pancreatic adenocarcinoma.

Mucinous cystic neoplasms

Mucinous cystic neoplasms are mucin-producing epithelial cysts that occur in the body and tail of the pancreas, and they do not communicate with the ductal system. They occur almost exclusively in middle-aged women and are characterized histologically by ovarian-type stroma. Recent meta-analyses have reported an overall malignancy rate of 17% in resected specimens.[12]

Intraductal papillary mucinous neoplasms

Intraductal papillary mucinous neoplasms (IPMNs) are mucin-producing neoplasms that arise from the ductal epithelium. They are subclassified into three main types: main duct, branch duct, and mixed type. Malignant potential differs significantly by type. Main-duct IPMNs have a 30% to 50% risk of malignancy, whereas branch-duct IPMNs have a 3% to 12% risk.[13] IPMNs have ductal communication by definition and are a major cause of both referral and surveillance uncertainty.

Solid pseudopapillary neoplasms

Solid pseudopapillary neoplasms are large, well-encapsulated masses with mixed solid and cystic components, and they primarily affect young women.[14] Their malignant potential has not been fully characterized, but case series have reported malignancy in up to 15% of resected lesions. Local invasion or metastasis is possible, so surgical resection is indicated.[14]

Serous cystic neoplasms/ serous cystadenomas

Serous cystadenomas are nonmucinous, glycogen-rich epithelial tumors with a

classic honeycomb, microcystic appearance on imaging. They are essentially benign and have negligible risk of malignancy.[15] Cyst fluid analysis demonstrating von Hippel-Lindau gene alterations and the presence of vascular endothelial growth factor-A support a diagnosis of serous cystadenoma, although this is not routinely required.

Cystic pancreatic neuroendocrine neoplasms

Cystic pancreatic neuroendocrine neoplasms may exhibit secondary cystic degeneration that often appears as a well-circumscribed, hypervascular cystic mass, typically in the body or tail of the pancreas.[2] Malignant potential varies by grade and proliferative index, and management is individualized.

Pancreatic ductal adenocarcinoma with a cystic component

A subset of pancreatic ductal adenocarcinomas may undergo cystic degeneration or arise within a pre-existing cystic neoplasm, such as an IPMN or mucinous cystic neoplasm. They typically present as irregular, hypoechoic masses with an abrupt main-duct cutoff and upstream dilation and represent overtly malignant lesions that require oncologic resection when feasible.[2,16]

Key points: Classification

1. Pancreatic cystic lesions are broadly divided into nonneoplastic (e.g., pseudocysts) and neoplastic lesions. Neoplastic cysts are further subdivided into mucinous (mucinous cystic neoplasm, intraductal papillary mucinous neoplasm [IPMN]) and nonmucinous (solid pseudopapillary neoplasm, serous cystic neoplasm/serous cystadenoma, cystic pancreatic neuroendocrine tumor, cystic pancreatic adenocarcinoma) subtypes.
2. Malignant potential varies by subtype, from negligible in serous cystadenomas to 30% to 50% in main-duct IPMN; therefore, accurate classification is essential to guide management.
3. Mucin-producing lesions (mucinous cystic neoplasm, IPMN) account for most clinically significant pancreatic cystic lesions and require the greatest attention given their premalignant potential.

Diagnosis

When a pancreatic cyst is discovered on cross-sectional imaging, the initial step is to confirm that it is a true cystic lesion and not an inflammatory fluid collection such as a pseudocyst. Reviewing the clinical history and any prior imaging is essential to assess chronicity and exclude pancreatitis-related causes. The subsequent diagnostic pathway is guided by lesion size, ductal communication, and presence of worrisome features **[Table]**.

If the lesion is a true cyst, the next step is dedicated MRCP, which is the preferred modality for characterization. MRCP delineates ductal communication, defines the presence of mural nodules or septations, and identifies high-risk or worrisome features with superior sensitivity compared with CT.[2-4] When MRCP is contraindicated or nondiagnostic, a pancreatic-protocol contrast-enhanced CT is an acceptable alternative; it provides information about calcifications, wall thickening, and ductal dilation.[4,5] Representative cross-sectional and endoscopic imaging findings of common pancreatic cystic lesions are shown in **Figure 2**.

For lesions with indeterminate imaging features or worrisome characteristics, endoscopic ultrasonography offers superior spatial resolution and allows for fine-needle aspiration for cyst-fluid analysis. Cytology may reveal mucinous epithelium, cuboidal glycogen-rich epithelium suggestive of serous cystadenomas, high-grade dysplasia, or even malignant cells. Cyst-fluid biomarkers can further help refine diagnosis. Carcinoembryonic antigen greater than 192 ng/mL suggests a mucinous cyst, whereas less than 5 ng/mL supports a serous cyst. Glucose less than 2.8 mmol/L favors a mucinous lesion, while amylase is typically elevated if the cyst communicates with the ductal system.[2,16-18]

Despite these advances, surgical pathology remains the diagnostic gold standard when resection is undertaken because it allows for definitive histologic subtyping and grading of dysplasia.[19]

Key points: Diagnosis

1. The initial evaluation should confirm a true pancreatic cyst and exclude inflammatory fluid collections, such as pseudocysts, based on clinical history and prior imaging.
2. Magnetic resonance cholangiopancreatography is the preferred imaging modality for cyst characterization, including assessment of ductal communication and high-risk features.
3. Endoscopic ultrasonography with fine-needle aspiration should be reserved for indeterminate or worrisome lesions, using cytology and cyst-fluid biomarkers (carcinoembryonic antigen, glucose, amylase) to refine diagnosis.

Risk stratification and surveillance

Risk stratification is central to determining whether a pancreatic cystic lesion requires surgical evaluation, endoscopic assessment, or continued surveillance. Major international guidelines have similar core criteria but differ in diagnostic thresholds and management aggressiveness. In general, lesions are evaluated for the presence of high-risk features, which warrant referral for surgical consideration, or worrisome features, which prompt further evaluation with endoscopic ultrasonography or short-interval imaging.

High-risk features indicate an increased risk of malignancy and generally prompt referral for further evaluation. These features include obstructive jaundice related to the cyst, an enhancing mural nodule 5 mm or larger, main pancreatic duct dilation 10 mm or larger, and cytology showing high-grade dysplasia or carcinoma.[2-5]

Worrisome features warrant further evaluation with endoscopic ultrasonography, with or without fine-needle aspiration,

TABLE. Risk stratification and surveillance recommendations for pancreatic cystic lesions, by lesion type.

Lesion type	How to recognize it	Malignant potential	Surveillance	When to refer
Nonneoplastic cysts				
Pseudocyst	History of pancreatitis or abdominal trauma	None	Not needed	Only if diagnosis is unclear or the cyst is symptomatic
Other benign cysts (simple, lymphoepithelial, retention)	Incidental; no ductal communication on imaging	None	Not needed once confirmed	If diagnosis is uncertain
Mucin-producing cysts				
Mucinous cystic neoplasm	Body/tail of pancreas; almost always in women	Approximately 17%	Not applicable (surgical resection)	Refer to gastroenterology/ surgery at diagnosis
Branch-duct intraductal papillary mucinous neoplasm (IPMN)	Connects to a small pancreatic duct branch	3%–12%	If no high-risk or worrisome features:* < 2 cm: MRCP every 18 months 2–3 cm: MRCP every 12 months > 3 cm: MRCP every 6 months	Any high-risk or worrisome feature* develops
Main-duct or mixed-type IPMN	Involves the main pancreatic duct	30%–50%	Not applicable (usually goes straight to resection)	Refer to gastroenterology/ surgery at diagnosis
Other cystic neoplasms				
Serous cystic lesion/ serous cystadenoma	"Honeycomb" pattern on imaging	Negligible	Not needed once confirmed	If diagnosis is uncertain
Solid pseudopapillary neoplasm	Young women; mixed solid/ cystic mass	Up to 15%	Not applicable (refer for resection)	Refer to gastroenterology/ surgery at diagnosis
Cystic pancreatic neuroendocrine tumor	Well-defined, vascular-appearing mass	Variable	Individualized; specialist directed	Refer to gastroenterology/ oncology at diagnosis
Cystic pancreatic adenocarcinoma	Irregular mass; may block the duct	Malignant	Not applicable	Urgent referral for oncologic workup

* High-risk features (refer urgently to gastroenterology/surgery): obstructive jaundice, an enhancing mural nodule ≥ 5 mm, main pancreatic duct ≥ 10 mm, or high-grade/ suspicious cytology. Worrisome features (refer for endoscopic ultrasound ± fine-needle aspiration): cyst ≥ 3 cm, mural nodule < 5 mm, main duct 5–9 mm, thickened/ enhancing cyst wall, elevated carbohydrate antigen 19-9, or interval growth. Magnetic resonance cholangiopancreatography (MRCP) intervals shown follow the Kyoto IPMN Guidelines;[3] American College of Gastroenterology and Canadian Association of Radiologists recommendations use similar but not identical size cutoffs.[2,6]

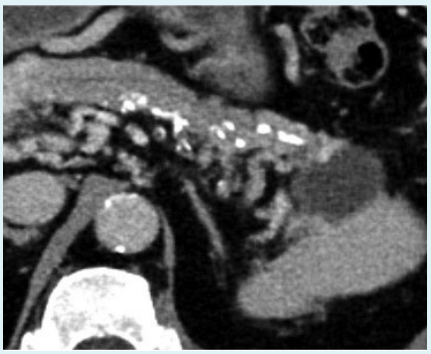
because they may signal an increased but not immediate risk. These features include cyst size 3 cm or larger, mural nodules less than 5 mm, thickened or enhancing cyst walls, main duct dilation of 5 to 9 mm, abrupt caliber change with distal atrophy, elevated carbohydrate antigen 19-9, and interval cyst growth.[2-5]

Cysts without high-risk or worrisome features can be followed with noninvasive surveillance, because most remain stable over time. MRCP is the preferred modality for longitudinal follow-up, because it accurately characterizes ductal communication and avoids radiation exposure.[2,4,6] Surveillance intervals vary across guidelines and depend on cyst size, morphology, and patient factors; these recommendations are summarized in the **Table**. Patients with an IPMN have a 3- to 5-fold increased risk of developing pancreatic ductal adenocarcinoma at an anatomically separate site from the cyst, likely due to a field defect. This highlights the importance of evaluating the entire pancreas, not only the cyst under surveillance.[20,21]

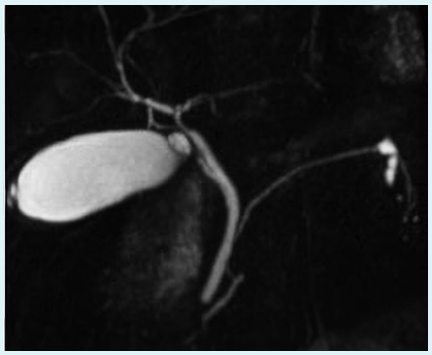
Surveillance may be discontinued in patients who are elderly, medically frail, or no longer candidates for pancreatic surgery, because continued imaging would not alter management.[2-5] Conversely, younger or surgically fit patients, particularly those with mucinous lesions or cysts 2 cm or larger, benefit from long-term follow-up, because early detection of morphologic change or emerging high-risk features enables timely referral and intervention.[2,4,6] The American Gastroenterological Association is currently planning an update to its 2015 guideline, which may further refine risk stratification and surveillance recommendations in the coming years.

Key points: Risk stratification and surveillance

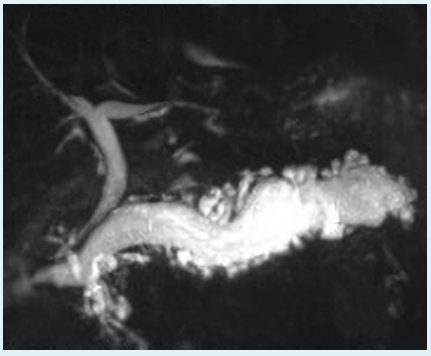
1. Risk stratification is based on the presence of high-risk and worrisome features, which determines the need for surgical referral, endoscopic ultrasonography, or continued surveillance.
2. Most asymptomatic pancreatic cystic lesions without concerning features can be safely monitored with magnetic resonance cholangiopancreatography at guideline-recommended intervals, tailored to cyst characteristics and patient factors.
3. Surveillance should continue only in patients who remain candidates for pancreatic surgery and when it aims to detect interval changes or the development of high-risk features.



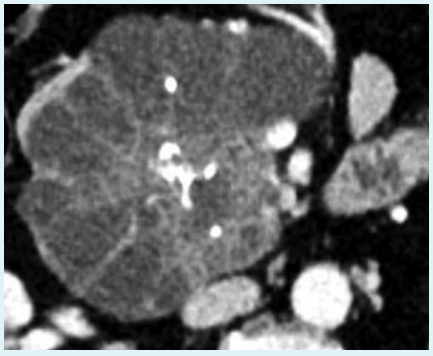
CT abdomen shows chronic calcific pancreatitis with a 2.5 cm pancreatic tail pseudocyst.



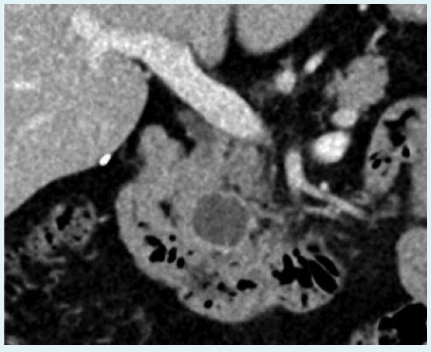
MRCP shows cystic lesions in the pancreatic tail; the largest measures 1.5 cm and communicates with a nondilated main pancreatic duct, in keeping with an IPMN with no worrisome features.



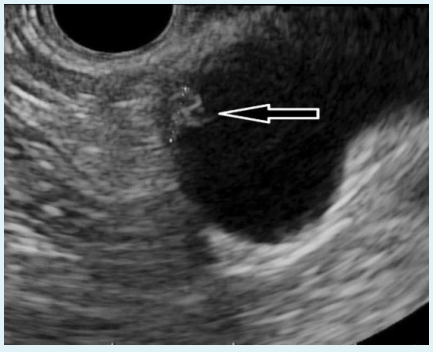
MRCP shows diffuse dilation of the main pancreatic duct up to 2.7 cm, consistent with a main duct IPMN.



CT shows a large 10 cm multiloculated pancreatic head cyst, with a central scar and calcifications consistent with a serous cystadenoma.



CT demonstrates a 1.5 cm cyst with rim enhancing in keeping with a cystic pancreatic neuroendocrine tumor.



EUS image of a pancreatic cyst suspicious for IPMN, with an intraluminal echogenic focus (arrow), representing a mural nodule or mucin ball.

FIGURE 2. Representative imaging features of common pancreatic cystic lesions on CT, magnetic resonance cholangiopancreatography (MRCP), and endoscopic ultrasound (EUS).
IPMN = intraductal papillary mucinous neoplasm

Management

Management of pancreatic cystic lesions is guided by the type of cyst, malignant potential, and presence of high-risk features, with the overarching goal of identifying lesions at risk for invasive carcinoma while avoiding unnecessary pancreatic surgery. In general, pancreatic cystic lesions with a known high risk of malignancy, such as main-duct or mixed-type IPMNs, mucinous cystic neoplasms, and solid pseudopapillary neoplasms, should be referred for surgical resection if the patient is an acceptable surgical candidate.[3,5,14,22] Surgery is also indicated when a cyst demonstrates high-risk imaging or clinical features, including main pancreatic duct dilation 10 mm or larger, an enhancing mural nodule 5 mm or larger, obstructive jaundice related to the cyst, or cytology showing high-grade dysplasia or malignancy.[2-4,23] In addition, symptomatic cysts causing pancreatitis, gastric outlet obstruction, or persistent pain warrant resection regardless of malignant potential.[2,5] A practical stepwise approach to management is outlined in **Figure 3**.

For asymptomatic lesions without high-risk features, active surveillance is preferred, because the risk of progression to advanced neoplasia remains low. MRCP is the recommended modality for longitudinal follow-up.[2-4] The frequency and duration of surveillance vary across major guidelines and depend primarily on cyst size, morphology, and interval stability; these recommendations are summarized in the **Table**. In general, MRCP is conducted 1 year after diagnosis and then every 2 years thereafter for those without worrisome features. Surveillance may be discontinued if the patient becomes unfit or unwilling to undergo pancreatic surgery.[2-5,23]

Patients who have undergone partial pancreatic resection for IPMN require ongoing follow-up of the pancreatic remnant, because metachronous cysts can develop even years later. Annual MRCP or endoscopic ultrasonography is generally recommended, particularly when the resected IPMN harbored high-grade

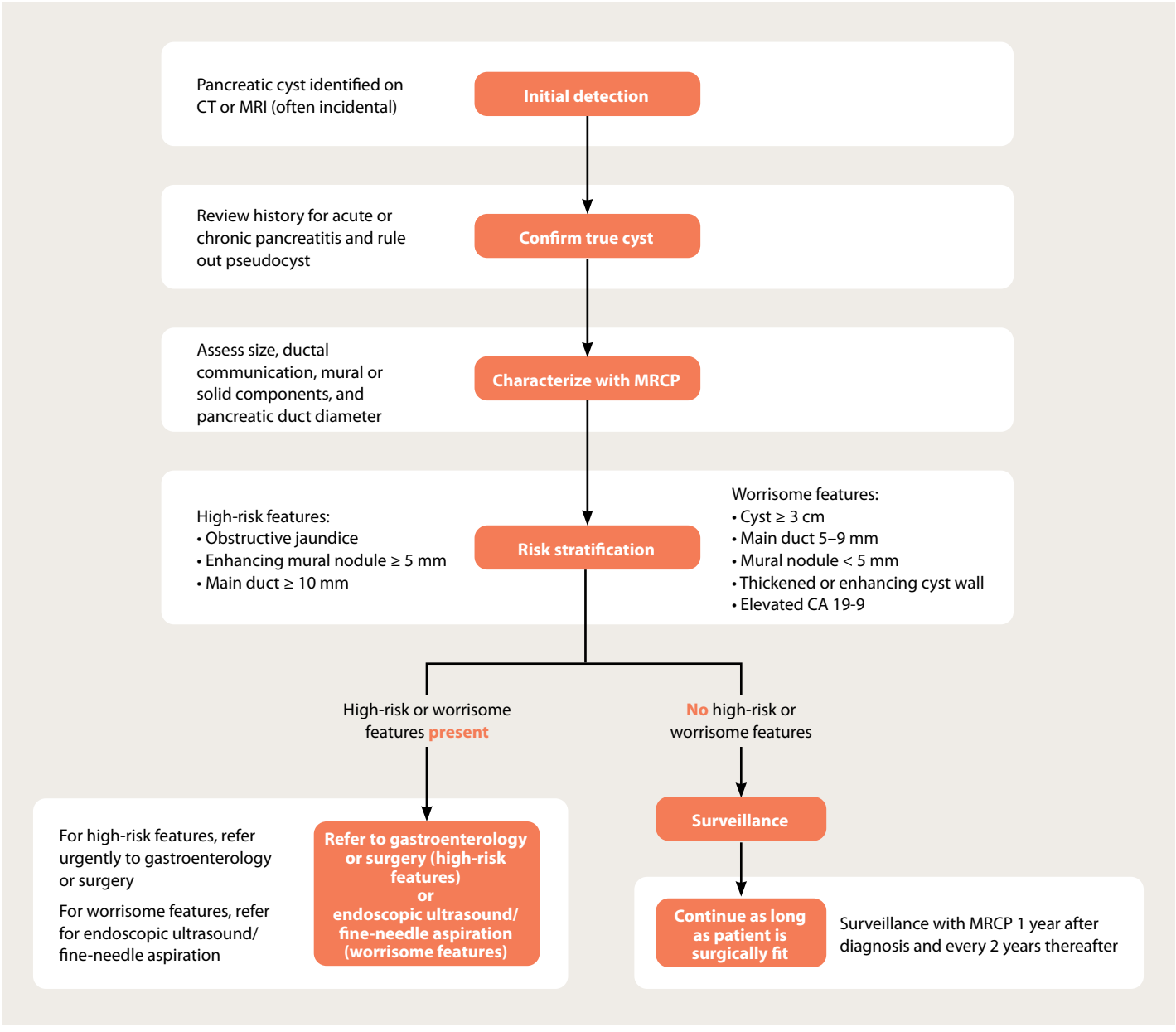


FIGURE 3. Practical algorithm for evaluation and management of pancreatic cystic lesions.
MRCP = magnetic resonance cholangiopancreatography; CA 19-9 = carbohydrate antigen 19-9.

dysplasia, because these individuals remain at elevated risk of subsequent pancreatic cancer.[24,25]

Overall, the prognosis for noninvasive cystic lesions is excellent when managed according to evidence-based pathways. Importantly, studies have shown that pancreatic cancers detected during structured surveillance are more often found at early, surgically curable stages, which reinforces the value of guideline-directed monitoring in appropriately selected patients.[25]

Key points: Management

1. Lesions with high malignant potential (main-duct/mixed-type intraductal papillary mucinous neoplasm (IPMN), mucinous cystic neoplasm, solid pseudopapillary neoplasm) or high-risk imaging/clinical features should be referred for surgical resection in appropriate surgical candidates.
2. Asymptomatic lesions without high-risk features are managed with active surveillance, typically magnetic resonance cholangiopancreatography, at 1 year after diagnosis and every 2 years thereafter **[Figure 3]**.
3. After partial pancreatectomy for IPMN, lifelong annual surveillance of the pancreatic remnant is required, given the risk of metachronous disease, especially if high-grade dysplasia was present.

Summary

Pancreatic cystic lesions are increasingly detected with modern imaging, and most are benign or indolent. However, a subset, particularly mucinous and main-duct lesions, carry a measurable risk of malignant transformation. MRCP is the preferred imaging modality for characterization and surveillance, while endoscopic ultrasonography with cyst-fluid analysis provides additional diagnostic precision when indicated. The presence of high-risk features such as mural nodules, main duct dilation, or obstructive jaundice should prompt referral to a gastroenterologist or pancreatic centre for surgical evaluation. For most asymptomatic cysts that lack these features, structured surveillance following established guideline intervals is safe and effective. Early recognition, risk stratification, and coordinated follow-up between primary care and specialist teams are key to ensuring timely detection of malignant change while minimizing unnecessary intervention. ■

Competing interests

None declared.

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Pancreatic neuroendocrine tumors in British Columbia: A review

Given the nuances in evaluating patients with pancreatic neuroendocrine tumors, referral for specialty consultation is advised.

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ABSTRACT: Pancreatic neuroendocrine tumors are neoplasms arising from the endocrine cells of the pancreas. Although these lesions are neoplastic, there are significant differences in prognosis and management depending on tumor characteristics; thus, they present a diagnostic challenge to the clinician and can create confusion for patients. Most lesions can be worked up with appropriate imaging. Endoscopic ultrasound is a sensitive modality for tissue diagnosis, risk stratification, and may play an evolving role in management. Given the nuances in evaluating patients with pancreatic neuroendocrine tumors, referral for specialty consultation is advised.

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BC Cancer estimates the average-risk patient's lifetime risk of pancreas ductal adenocarcinoma at 1.3%. In contrast, pancreatic neuroendocrine tumors (NETs) are rare. The pancreatic NET incidence was less than 1 case per 100 000 population per year,[1,2] and pancreatic NETs account for only 1% to 2% of all pancreatic neoplasms.[3,4] However, clinicians should be aware of these lesions because of increasing incidence, attributed to improved access to cross-sectional imaging.[5,6] One in five incidental pancreatic lesions on CT are pancreatic NETs.[7]

The nomenclature of pancreatic NETs has changed over time. Prior to the 1990s, these lesions were called "islet cell tumors" or "carcinoids." The World Health Organization recommended the standardized terminology "endocrine tumor"[8] and then "neuroendocrine tumor." Medical school curricula tend to highlight the hereditary syndromes of multiple endocrine neoplasia type 1, von Hippel-Lindau disease, tuberous sclerosis complex, and neurofibromatosis type 1; however, 90% of pancreatic NETs are sporadic and are not associated with an inherited syndrome.[9]

Pathology and classification

Pancreatic NETs show expression of both epithelial markers (keratins) and neuroendocrine markers, such as chromogranin A and synaptophysin.[10,11] Hormone-producing pancreatic NETs with symptoms are called functional tumors.[12] Less than 20% of pancreatic NETs are functional.[13] Functional pancreatic NETs include insulinomas, gastrinomas, vasoactive intestinal peptide–secreting tumors (VIPomas), glucagonomas, pancreatic NETs producing carcinoid syndrome, and somatostatinomas.[10,14]

Pancreatic NETs can be divided into well-differentiated NETs (grades 1 to 3),[13] neuroendocrine carcinomas (small cell or large cell), and mixed neuroendocrine–nonneuroendocrine neoplasms **[Table]**. Mixed lesions have both neuroendocrine and nonneuroendocrine components.[11] The most common nonneuroendocrine component is adenocarcinoma.[11] Histologically, well-differentiated NETs are monomorphic neoplastic proliferations with salt-and-pepper chromatin most often arranged in trabeculae and nests **[Figure 1]**. These lesions also have a Ki-67 proliferation

TABLE. Pancreatic neuroendocrine tumor classification, features, and prognosis.

	Well-differentiated neuroendocrine tumors	Neuroendocrine carcinoma
Pathologic features	Well-differentiated morphology Lower proliferative index	High-grade morphology High proliferative index
Prognosis	Slowly progressive Prognosis based on size, resectability, and World Health Organization grade (1 to 3)	Rapidly progressive Poor prognosis

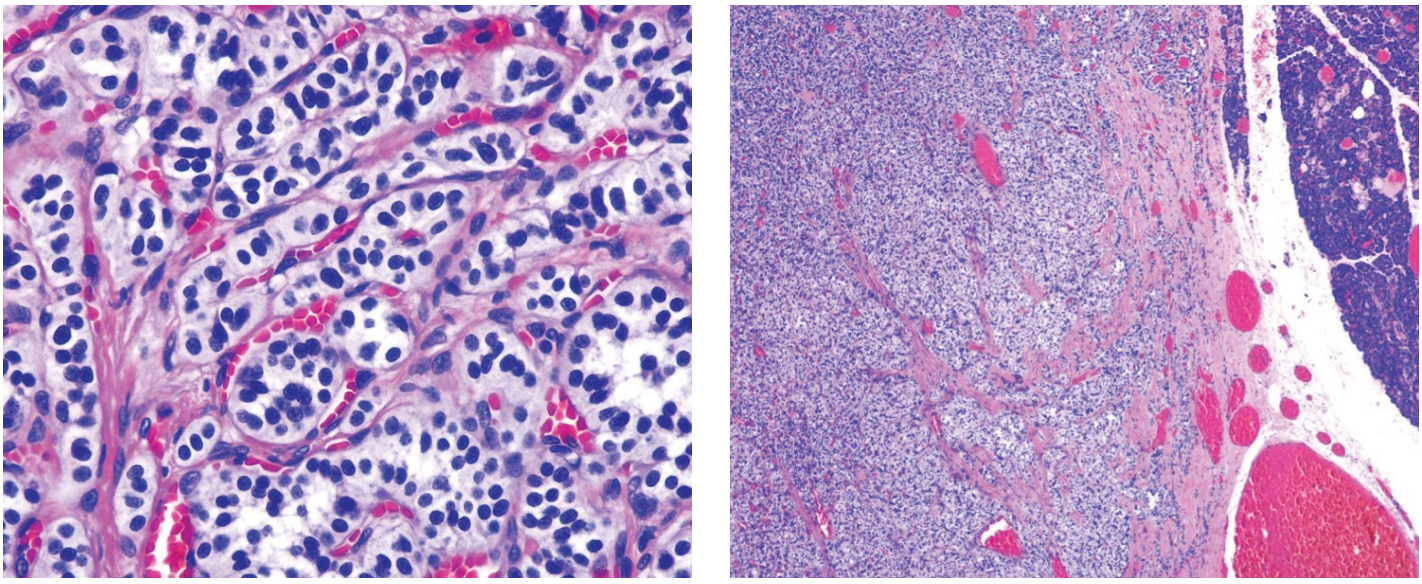


FIGURE 1. Pancreatic neuroendocrine tumor after endoscopic ultrasound fine-needle biopsy. Images on the left (40x) and right (4x) show immunohistochemical stains of the biopsies. The cells are strongly cytokeratinAE1/AE3-positive, with a granular cytoplasmic pattern of staining and paranuclear accentuation in many cells. Findings indicate a well-differentiated neuroendocrine tumor.

rate of less than 50%.[11,15,16] Common molecular alterations in well-differentiated NETs are in the *MEN1*, *DAXX*, *ATRX*, *mTOR*, *CDKN2A*, and *SET2* genes.[17] All pancreatic NETs are considered malignant; however, small, well-differentiated T1 tumors have an excellent prognosis.[11]

Neuroendocrine carcinomas are distinct malignancies from the well-differentiated NETs **[Table]**. They can be divided into small-cell and large-cell carcinomas. Small-cell carcinomas are characterized by closely packed cells with a high nuclear-to-cytoplasmic ratio, hyperchromasia, nuclear molding, high mitotic rate, and abundant apoptosis. Large-cell neuroendocrine carcinomas show large, closely packed cells with vesicular chromatin and a prominent nucleolus. The threshold between high-grade (grade 3) well-differentiated NETs and large-cell neuroendocrine carcinomas can be difficult. Rarely, some well-differentiated NETs transform into neuroendocrine carcinomas.[16-18] Neuroendocrine carcinomas frequently show mutations in both the *p53* and *Rb1* genes. They may also demonstrate gene mutations seen in pancreatic adenocarcinoma, such as *KRAS*, *CDKN2A*, and *SMAD4* mutations.[17]

Diagnostic evaluation

The clinical presentation of pancreatic NETs can be diverse. Insulinomas can cause intermittent hypoglycemia. Abdominal pain, heartburn, peptic ulcers, or diarrhea can direct clinicians to look for a gastrinoma in Zollinger-Ellison syndrome. VIPomas can cause a rare syndrome of hypokalemia, hypochlorhydria, and watery diarrhea. Nonfunctioning pancreatic NETs can cause complications such as weight loss, abdominal pain, and jaundice from mass effect. Nonfunctioning pancreatic NETs are often found when imaging is being done for another indication.

A thorough elevation of pancreatic NETs starts with imaging, includes a functional assessment, and is confirmed by cytology or histology. Routine CT with portal venous phase may miss a pancreatic NET, because the lesion can appear isodense or only mildly hyperdense to the background parenchyma.[19] On arterial phase CT, these lesions demonstrate arterial phase hyperenhancement.[14] Therefore, multiphase cross-sectional imaging is essential to define these lesions **[Figure 2]**. On MRI, the lesions are hypointense on intrinsic T1 phases and high signal on T2-weighted sequences and demonstrate avid arterial phase enhancement on postcontrast T1 sequences.[20,21] Other arterial enhancing lesions can mimic pancreatic NETs on CT, including solid pseudopapillary neoplasms, pancreatic metastases (e.g., renal cell carcinoma), acinar cell carcinomas, pancreatic hamartomas, pseudoaneurysms, intrapancreatic splenules, and exophytic gastrointestinal stromal tumors.[22,23] The most appropriate way to initiate investigation of a pancreatic lesion is a pancreatic-protocol CT scan.

Depending on the diagnostic evaluation, further noninvasive imaging may be indicated, including an octreotide scan or PET. Three PET radiopharmaceuticals are available for suspected pancreatic NETs: gallium-68 DOTA-Tyr(3)-octreotate (Ga-68 DOTATATE), gallium-68 DOTA-D-Phe1-Tyr(3)-octreotide (Ga-68 DOTATOC), and copper-64 DOTA-Tyr(3)-octreotate (Cu-64 DOTATATE).[24]

Functional testing with biomarkers is usually performed by a local specialist in endocrinology, gastroenterology, hepatopancreaticobiliary surgery, or oncology. The specialist interrogates symptoms, elicits a family history, and examines the patient for signs of multiple endocrine neoplasia type 1, von Hippel-Lindau disease,

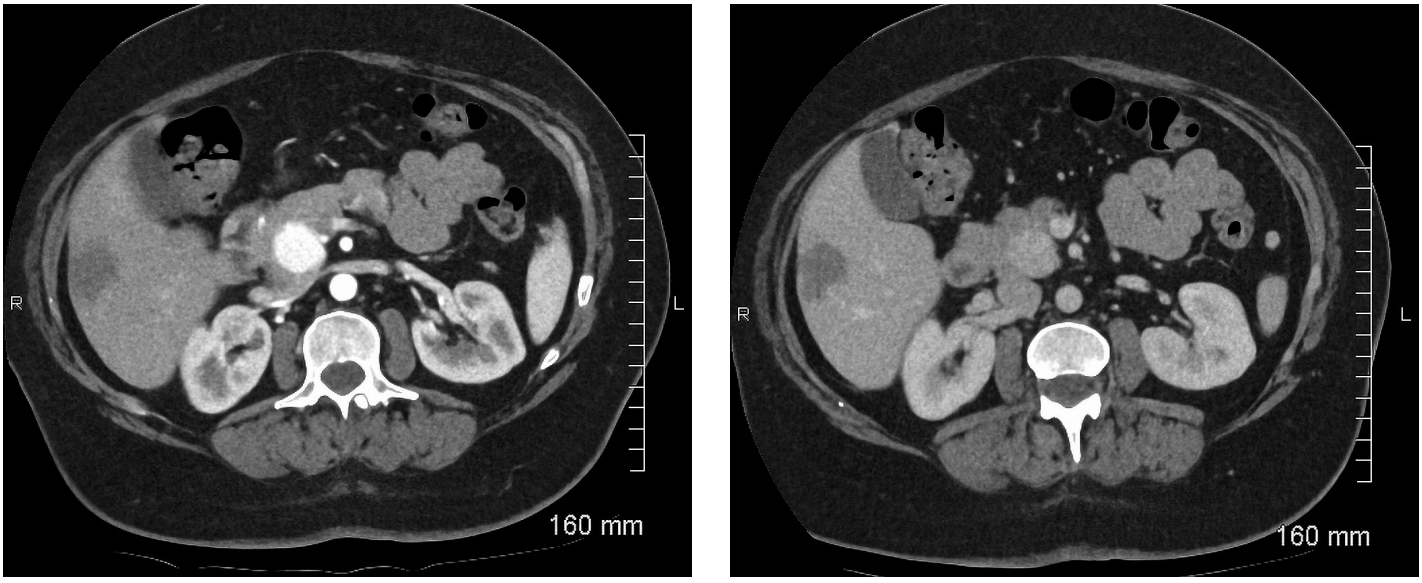


FIGURE 2. Multiphase CT imaging of the pancreas with arterial phase (left) and portal venous phase (right). There is a 2.6 × 2.7 × 2.4 cm circumscribed hypervascular mass in the medial head of the pancreas. Enhancement of these masses is most avid on the arterial phase, where it is similar in attenuation to arterial blood pool.

neurofibromatosis type 1, or tuberous sclerosis complex.[13] North American guidelines recommend against routine use of nonspecific tumor markers such as chromogranin A.[13]

Endoscopic ultrasound

In British Columbia, endoscopic ultrasound and fine-needle biopsy are available at Victoria General Hospital, Vancouver General Hospital, St. Paul's Hospital, Royal Columbian Hospital, Surrey Memorial Hospital, Royal Inland Hospital, and Kelowna General Hospital. Endoscopic ultrasound has good accuracy for pancreatic NETs[26,27] and facilitates tissue acquisition for most patients.[28] Endoscopic ultrasound is more sensitive than CT[29] and is superior to MRI and somatostatin receptor scintigraphy for pancreatic NET localization; it has reported sensitivities of 82% to 93% and is recommended in guidelines for pancreatic NET diagnosis.[30] Even small pancreatic NETs should be confirmed on biopsy if possible.[31] Endoscopic ultrasound prior to surgery is beneficial. A meta-analysis of 612 patients showed a preoperative sensitivity for pancreatic NET detection of 97%.[32]

For small nonfunctioning pancreatic NETs with low proliferative activity, surveillance with endoscopic ultrasound or cross-sectional imaging is often appropriate. Novel technologies in endoscopic ultrasound that use IV contrast, tissue harmonics, and elastography may be useful in differentiating pancreatic NETs from benign pancreatic lesions in the future. Endoscopic ultrasound can also play a role in the treatment of pancreatic NETs using endoscopic ultrasound–guided tattooing or fiducial insertion to target radiotherapy and surgical treatment. For nonsurgical candidates, endoscopic ultrasound–guided ablation therapy with ethanol and radiofrequency ablation have been studied for local tumor control.[33]

Prognosis

Pancreatic NETs encompass a spectrum of disease, ranging from low-grade indolent tumors to rapidly progressive disease. Tumor histology, grade, Ki-67 score, and clinical stage are important features for estimating prognosis and response to treatment. For localized, resectable disease, 5-year overall survival rates are 97%, 87%, and 73% for stages I, II, and III, respectively, by European Society of Neuroendocrine Tumors staging.[34] Ki-67 is a protein found in dividing cells and is useful for quantifying the risk of tumor growth and spread. Tumor grade, Ki-67 score, and differentiation are strong predictors of prognosis.[35] Based on surveillance, epidemiology, and end results data for patients with stage IV or metastatic disease, 5-year overall survival rates are 51%, 45%, and 12% for tumor grades 1, 2, and 3, respectively.[6] In contrast, pancreatic neuroendocrine carcinomas have a poor prognosis, with a median overall survival of 8 to 17 months.[36] Therefore, when counseling a patient regarding a diagnosis of pancreatic NET, it is important to incorporate both clinical and pathologic features.

Management

Surgery is advised for functional pancreatic NETs.[25] Active surveillance with annual imaging has been advised for patients with nonfunctional pancreatic NETs that are less than 1 cm in size.[25,31] In a prospective study of patients with documented pancreatic NETs that were less than or equal to 2 cm, only 11% of patients showed an increase in pancreatic NET size of at least 0.5 cm/year, which was a study indication for surgical referral, over a median follow-up of 17 months.[37] The management of patients with nonfunctional pancreatic NETs 1 cm to 2 cm, without pancreatic duct obstruction,

can be tailored based on surgical risks and tumor biology. Referrals for surgery should be made when the nonfunctional pancreatic NET is 2 cm or larger or is associated with main pancreatic duct dilation.[25] Curative intent surgery for nonfunctional pancreatic NET has a 5-year survival rate of more than 70%.[38] Given the potential for late recurrences, the North American Neuroendocrine Tumor Society recommends surveillance imaging for at least 10 years after resection. Individual recommendations are based on tumor size, Ki-67 score, and the number of lymph nodes involved.[13]

Systemic therapy

Systemic therapy is reserved for patients with unresectable or metastatic disease. Currently, there is no evidence to support the use of systemic therapy in the adjuvant setting. The choice of systemic therapy depends on symptoms, somatostatin receptor status, tumor grade, and histology.

For patients with well-differentiated tumors, a Ki-67 score of less than 10%, and somatostatin receptor–positive disease, somatostatin analogues alone can be considered for first-line therapy with long-term responses reported.[39] However, for patients with well-differentiated pancreatic NETs and a high proliferation rate (Ki-67 > 10%), chemotherapy with capecitabine and temozolomide can be considered.[40,41] New data support the use of peptide receptor radionuclide therapy as first-line treatment in patients with a Ki-67 score of 20% or higher in combination with a somatostatin analogue or as second-line therapy for patients whose disease has progressed on somatostatin analogues.[42,43] Subsequent lines of therapy include tyrosine kinase inhibitors such as sunitinib, cabozantinib, or a mechanistic target of rapamycin inhibitors such as everolimus.[44-46]

In contrast, pancreatic neuroendocrine carcinomas are associated with aggressive behavior and poor prognosis. Neuroendocrine carcinomas are treated with cisplatin and etoposide or fluoropyrimidine-based doublet chemotherapy.

Summary

Pancreatic malignancy is a challenging topic to discuss with patients. This is particularly true if there is a delay in diagnosis. Most patients do not interact with an active group of advocates and survivors. Advocacy programs for other cancers have improved outcomes.[47] Finding the balance between informing a patient and not creating panic is a constant focus for physicians in this field.

Accurate prognostication and awareness of the stark contrast between the management of pancreatic NETs and that of pancreatic adenocarcinoma are critical. A pancreatic-protocol CT with arterial phase imaging is the first step in evaluating a patient with a suspected pancreatic NET. Subsequent referral for endoscopic ultrasound will enable tissue diagnosis and risk stratification. Endoscopic ultrasound providers will refer the patient to a pancreas surgeon or to BC Cancer based on staging data and functional testing. For oncologists and surgeons, deciding between active surveillance with yearly imaging and surgical resection often requires a multidisciplinary review. ■

Key points

1. A pancreatic-protocol CT is the starting imaging modality for a suspected pancreatic neuroendocrine tumor (NET).
2. Functional testing should be initiated by local specialists in endocrinology, gastroenterology, hepatopancreaticobiliary surgery, and oncology.
3. A referral for a local endoscopic ultrasound can facilitate detection, tissue diagnosis, and risk stratification.
4. The decision between surveillance and surgery is dictated by clinical factors, pancreatic NET size, functional status, stage, and patient goals.

Competing interests

Dr De Korompay has been a speaker and consultant for Penumbra, Inc. and Cook Medical. The remaining authors have no competing interests to declare.

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Making the most of my screen time

Reflections from a resident doctor's experience in Real-Time Virtual Support.

Jen-Ai Lai, MD, FRCPC



The longhouses and monumental totem poles of Kitselas Canyon reflect the enduring connection between the Kitselas people, their culture, and this landscape. **Kitselas** ***is derived from the Sm'algyax word*** **Gitselasu,** ***meaning "People of the Canyon."***

By the time you reach the 5th year of your emergency medicine residency, you start to feel like you're getting the hang of it. You've run traumas, managed shock, and intubated a crashing patient at 3 a.m. You've learned to think in parallel and act decisively.

Then you open the door to something entirely new.

For me, that was participating in Real-Time Virtual Support (RTVS), a provincial network that provides instant virtual peer-to-peer consultative support to physicians and other health care providers in rural communities. By connecting frontline clinicians with experienced colleagues in real time, RTVS has become an essential component of equitable emergency care in British Columbia, strengthening local care through partnership, shared decision making, and support for the expertise already present on the ground.

Despite training in large urban academic centres, I've always gravitated toward rural and remote practice. I'm currently completing an area of concentrated expertise in rural and remote emergency medicine, which has taken me across our beautiful province and into Canada's far north. But until recently, virtual emergency medicine was uncharted territory for me.

Through RTVS, I worked within two pathways administered by the Rural Coordination Centre of BC. RUDi (Rural Urgent Doctor-in-aid) provides 24/7 virtual emergency support to rural physicians, remote nursing stations, and other frontline clinicians by reviewing cases, assisting in critical moments, and collaborating in real time when help is needed most. VERRa (Virtual Emergency Room Rural assistance) is one of the newer RTVS pathways and provides overnight virtual emergency department coverage to small rural hospitals facing physician staffing challenges.

Working alongside Drs Ian Ricketson and Brydon Blacklaws, two seasoned RTVS physicians, we supported communities across British Columbia, including Kitkatla, Atlin, Tsay Keh Dene, Fort Nelson, Tatla Lake, Txalgiuw (Hartley Bay), Bella Coola, and Ahousaht. Some of these communities are accessible only by boat. Others are a 6-hour drive from the nearest hospital with advanced imaging and subspecialty services—that is, *if* the roads aren't too icy or flooded. Geography is not a backdrop; it's an integral part of the clinical picture.

I gained a deep appreciation for the unique skill set required in virtual emergency medicine. Some of it is deceptively simple. How you show up on screen matters, as does the cadence of your speech. Pausing for long enough to allow others to hear you, but not so long that they think the connection has frozen, keeps communication clear. Narrating your decision making aloud ensures that everyone is on the same page.

But beyond the mechanics of a virtual

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platform is the nuance. Coming from a tertiary care emergency department, we often see virtual medicine gone wrong—patients who present to the ER because something was missed or inappropriate. It's easy to develop a jaundiced view of virtual medicine. But what if the real comparison isn't virtual care versus in-person care but virtual care versus no access to care at all?

When it comes to rural communities, I learned that virtual care is not about replacing hands-on clinical medicine; it's about navigating risk and amplifying the skill and courage already present in these communities. Like an air traffic controller, virtual medicine guides what must land immediately and what can safely circle.

Even if it isn't face-to-face, you still speak with every patient. Even if you never physically touch the patient, you still examine them, noting their color, mentation, work of breathing, and vitals. You learn what can and cannot be done safely over a virtual network. And sometimes the safest plan is reassessment in the morning when in-person care is available.

Some presentations are particularly challenging in this arena. Abdominal pain and orthopaedic ailments—conditions heavily reliant on physical examination—can stretch the limits of virtual care. Surprisingly, others, such as acute chest pain, can feel more straightforward: Obtain a focused history, vitals, ECG, and labs if available. An ST-segment elevation myocardial infarction is diagnosed: Administer acute coronary syndrome medications, initiate thrombolysis, and coordinate urgent transfer. The principles of emergency medicine remain the same, but the delivery is different. Acknowledging the limitations of virtual care is key to practising well.



Dr Ricketson receiving an RTVS advice call from a remote nursing station located near the Ulkatcho First Nation (Anahim Lake). Photo credit: Rebecca Connop Price, RTVS communications officer.



Dr Lai in Gingolx, a Nisga'a village where the Nass River meets Portland Inlet. Until 2002, the community was accessible only by boat or plane. The completion of a road connection just over 2 decades ago brought Gingolx into the provincial highway network. Despite this connection, geographic isolation continues to shape access to health care across the province.

Geography and logistics add layers of complexity that I find intellectually stimulating. Even the most bread-and-butter cases become interesting when your patient is hours away from the nearest hospital, weather permitting. A child presenting with a fever and a cough isn't just another viral illness when follow-up is uncertain. Decisions about antibiotics, observation, and transfer shift when distance and delay are part of the equation. RTVS physicians must operate with a high tolerance for uncertainty while accepting that the margin for error is essentially zero.

Relationships are the foundation that makes this work. The success of RTVS is grounded in trust between patients, nurses, and physicians. I had a patient in Haida Gwaii with new-onset atrial fibrillation and congestive heart failure who didn't want to travel into town. To many of us, transfer to a larger centre with more specialized care may seem like the obvious choice. But for her, the matriarch of her community, being torn from her responsibilities and support system for yet another long hospitalization was untenable.

As Dr Ricketson often says, "Assumptions don't work in rural care." Some patients don't want to leave their communities. Others absolutely do. Sometimes it is who you would least expect. Regardless, the door must always remain open. Our role is not to impose a rigid standard of care but to work in partnership with each

patient—to understand what matters most to them, explain risk clearly, and co-create a plan that is medically sound while respecting each patient's unique context.

Delivering this kind of care requires deep collaboration. For RTVS physicians, the nurses on the ground become your eyes and hands. I remember guiding one nurse through repairing a deep scalp laceration, a procedure she had done only twice before. Her determination to spare our patient a long winter transfer was truly humbling. Moments like this stand out to me because they capture the collaborative spirit of virtual care at its best: standing behind the people delivering care on the ground.

Sometimes the impact extends beyond a single case. One night while covering Fort Nelson through VERRa, I learned that Dr Ioana Lupu was one of only two doctors in the entire community covering the 24/7 ER. For many physicians, working in rural and remote communities is more than a full-time job. They cover clinics by day and the ER by night. If a VERRa physician–nurse team can give that doctor a full night of uninterrupted rest, that matters. Over time, that may be what keeps physicians in their communities, prevents burnout, and keeps rural emergency departments open.

As I prepare to graduate, I am reflecting on my journey through residency. Working with RTVS has reshaped how I think about distance in medicine and what becomes possible when people are willing to show up for each other across it. I believe that earlier exposure to this kind of work during residency would make a meaningful difference.



The vivid blue waters of Kitsumkalum Lake, photographed during a worthwhile detour from Dr Lai's commute between Ksyen Regional Hospital in Terrace and Prince Rupert Regional Hospital.

It builds comfort with uncertainty, communication at a distance, and the realities of rural practice. It also develops a different kind of clinical confidence—less about having every resource immediately available, and more about safely navigating the situation when you do not. These are skills that extend well beyond rural medicine. Whether you ultimately practise in rural or urban settings, experiences like these foster relationships between clinicians, deepen our appreciation for the realities of rural care, and help bridge a divide that extends beyond geography alone.

In many ways, my experience with RTVS reaffirmed why I was drawn to emergency medicine in the first place: It is one of the rare fields in medicine where we have the privilege of caring for people from all walks of life, including those who might not otherwise be able to access health care. Emergencies don't discriminate, nor should access to care.

To me, RTVS embodies that principle. It isn't just a technology platform; it is a commitment to being present and showing up, even when you are hundreds of kilometres away. It strengthens the remarkable work already happening in rural communities by fostering collaboration, sharing expertise, and supporting the clinicians who know their communities best. It expands access in ways that are pragmatic, relational, and sustainable, ensuring that rural communities are supported—not just in moments of crisis, but as an ongoing partnership. ■



School-based health hubs bring care to BC classrooms

Your third patient this morning is Kaitlin. She is worried about Casey, her child in grade 8, who takes a 20 km roundtrip bus ride to and from school every day in rural BC. Casey's older sibling has had issues with petty theft and alcohol use. Their father is currently unemployed due to a slowdown in the logging industry. Kaitlin has a part-time job at a community grocery store.

Casey struggles socially and turns to social media extensively. Casey often complains about feeling anxious and lonely. Kaitlin knows that Casey is experimenting with alcohol and cannabis, and she is asking for your help.

The problem

As many as one in eight children and youth in BC meet the criteria for a mental health disorder, including anxiety, attention-deficit/hyperactivity disorder (ADHD), substance use, and depression. In a review by Schwartz and colleagues, the authors found that only 44% of children with a mental health disorder received any mental health services.[1] This is especially alarming when coupled with substance use: 67% of children in BC who died from illicit drug toxicity in 2025 also struggled with mental health issues.[2]

What can we do?

School-based health hubs (SBHHs) are one way to improve access to care by providing care directly at a young person's school. An SBHH is a collaborative team—usually composed of doctors, nurses, counselors, and other mental and physical health care professionals—that provides child and youth health care in an elementary or secondary school. Throughout BC, there are currently 16 SBHHs. In all instances, they were initiated by physicians, nurse practitioners, and other health care and education professionals as a local, community-based response to the medical needs of children and youth, particularly those suffering from mental health and substance use issues.

SBHHs do not replace established relationships with longitudinal care in the community; rather, they enhance those services and aid children and youth who are disconnected from care. As every community is unique, each SBHH is tailored to its needs and resources, while still sharing a common goal of increasing access to health care for students.[3,4]

SBHHs help with the following:

- Bridging gaps: They provide services in a familiar, accessible, and safe school environment. The accessibility of SBHHs also fosters trust between students, families, and health care professionals, encouraging earlier intervention and preventive care.[5]
- Improving health outcomes: By connecting students and families to community resources and providing culturally competent care, SBHHs contribute to long-term health equity by addressing underlying social determinants of health, such as poverty and discrimination.[6,7]
- Promoting health equity: SBHHs address critical barriers to health care access, ensuring that students, particularly those from isolated or marginalized communities, receive the medical, mental, and social support they need to thrive.[5-7]
- Enhancing educational engagement: SBHHs improve student attendance, academic performance, and overall educational engagement through accessible primary care appointments, mental health counseling, chronic disease management, and health education.[5,7]
- Increasing graduation rates: Regular school attendance and feeling safe and supported improve student learning and success. When students finish school and graduate, they are more successful in acquiring meaningful employment and life stability.[8]
- Reducing emergency department visits: SBHHs meet the needs of pediatric patients, reducing emergency department visits for minor conditions and maintaining capacity for critical situations.[9]
- Improving collaboration: SBHHs strengthen relationships between school staff and physicians and other health care professionals by bringing services closer together and developing a network of support. One family physician expressed that this network is the only thing stopping them from burning out.

Next steps

Building on their collective learning from SBHHs across 16 communities through the Child and Youth Mental Health and Substance Use Community of Practice, the Shared Care Committee is now supporting a funded spread initiative. With two communities in the Interior Health region eager to start, implementation began on 9 September 2026 at the first site and is scheduled for January 2027 at the second site.

Additionally, physicians and health care professionals will continue to engage with health authorities, Divisions of Family Practice, the Ministry of Health, the Ministry of Children and Family Development, and other partners to support the growth and

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adoption of SBHHs and improve pediatric mental health and substance use care throughout BC.

If you are interested in learning how to initiate your own SBHH, reach out to Sonia Virk, advisor, communities of practice, for more information at svirk@doctorsofbc.ca. ■

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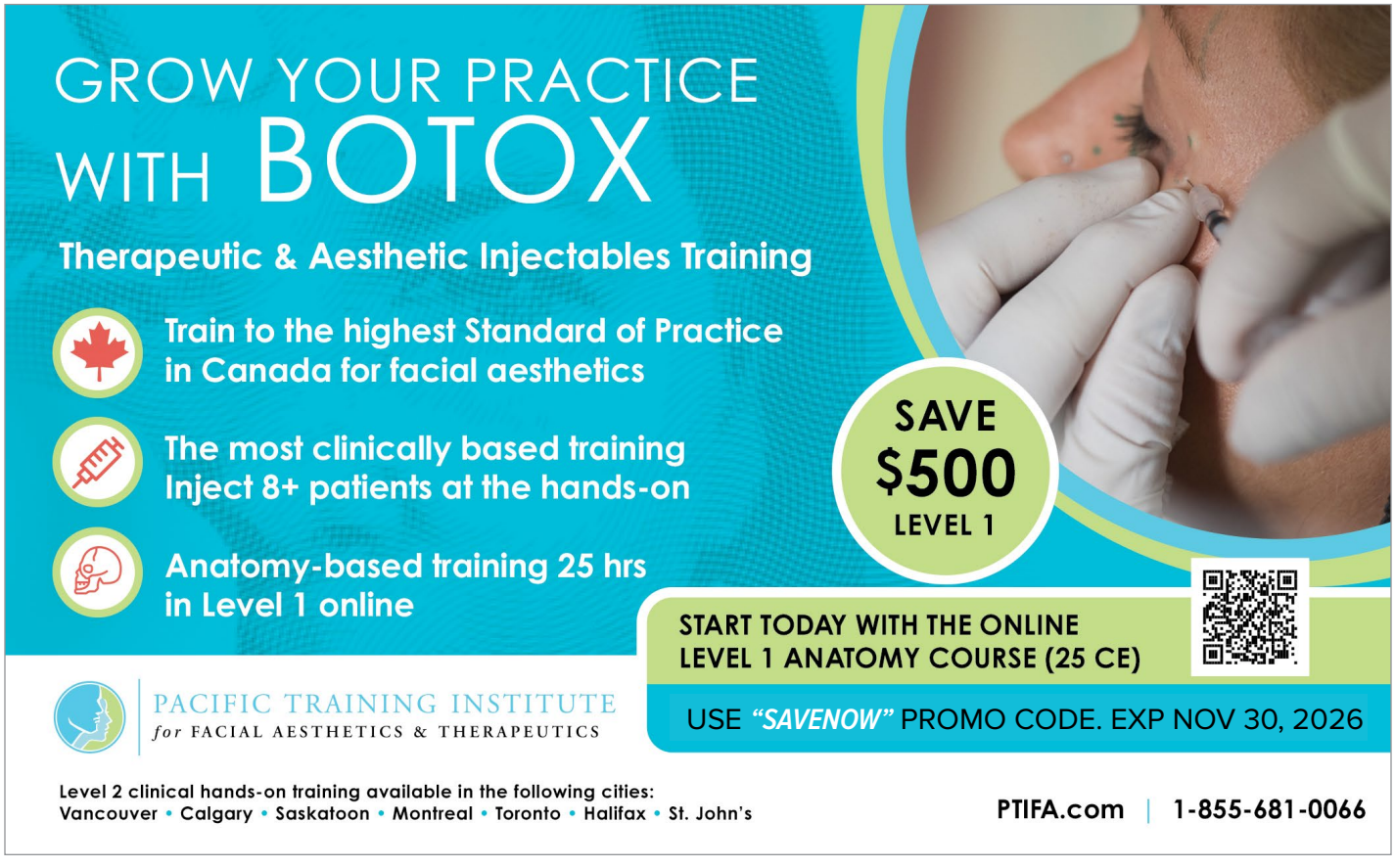
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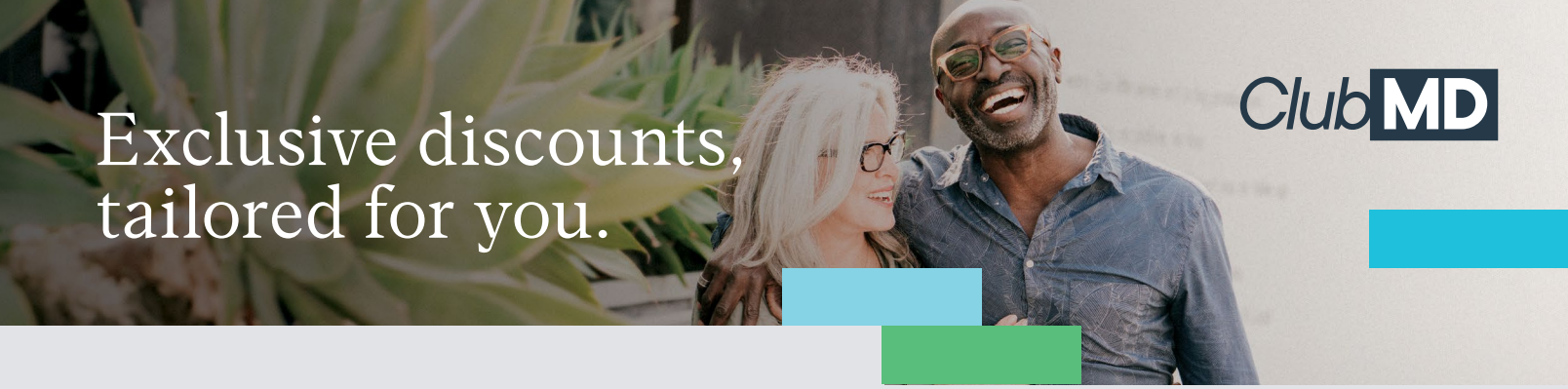
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Smoking and radon in British Columbia: Overlapping risks of lung cancer

Radon is a carcinogenic gas released during the decay of uranium that exists naturally in soil and bedrock. It has no smell, taste, or color, and it accumulates indoors, where it and its radioactive progeny are inhaled and directly impact lung epithelium. Radon is a leading environmental cause of lung cancer, with risk increasing with exposure time and magnitude.[1] Fortunately, testing indoor levels is easy and inexpensive. In British Columbia, the BC Centre for Disease Control (BCCDC) collects indoor radon measurements and displays them on an interactive public map (https://reporting.bccdc.ca/bcradonmap/) to promote testing and awareness.[2]

Radon levels in BC

Uranium levels in soil and bedrock vary dramatically across BC, with the highest levels found in the Interior and Northern Health regions. This pattern is clearly visible on the BCCDC's interactive radon map, where an estimated one-third of homes in the Interior Health region had radon concentrations exceeding Health Canada's guideline of 200 Bq/m^3. However, radon levels vary on the background of these general patterns. Local geographic features can increase radon levels, and some homes have construction elements that promote radon accumulation (e.g., poor ventilation, below-grade basements, foundation cracks, gaps around pipes, porous walls). For example, 10% of measurements collected from some municipalities in the Vancouver Coastal Health region exceeded 200 Bq/m^3, despite the area on average having very low indoor radon levels. This is why Health Canada and the BCCDC recommend that every home be tested for radon.

Radon, lung cancer, and smoking

Exposure to radon increases the risk of lung cancer, but this increase in risk is not equal in everyone. Radon and tobacco smoke interact synergistically. Smokers who are exposed to high radon levels face a much higher risk of lung cancer than would be expected if radon and smoking acted independently to increase risk. In fact, the lifetime risk of lung cancer among nonsmokers exposed to high radon levels is about 2 per 100, compared with about 15 per 100 among smokers exposed to high radon. Consequently, about 85% of radon-attributable lung cancers are in smokers.[3] Whether former smokers or those exposed to secondhand smoke have the same increased risk from radon as current smokers is not yet known.

Routine radon testing is warranted

Importantly, areas of BC with higher smoking rates overlap with areas with higher radon measurements. In 2023–24, about 12% of the population in high-radon health regions (Northern Health and Interior Health) reported smoking daily, compared with 7% to 8% of the population in other health regions.[4] These risks compound; therefore, radon poses a higher risk to the population than it might otherwise.

However, awareness of this risk is low. In a 2023 national survey, only 60% of BC households had heard of radon, and, of those, only 7% reported testing their homes.[5] We do not have good insight into whether smokers are aware of their increased risks.

Physicians and other health care providers can help raise awareness about radon, particularly among smokers, and discuss radon testing while encouraging smoking cessation. Patients with a smoking history should be made aware of their additional risk and the steps they can take to reduce radon exposure. This includes providing education on the health risks of radon exposure, encouraging regular screening for radon at home, and sharing available

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resources, such as those found in the Box, with patients so they can test their homes and take mitigating steps where possible. ■

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BOX. Additional information about radon.

- **Radon, BC Centre for Disease Control:** www.bccdc.ca/health-info/prevention-public-health/radon.
- **Radon and smoking, Take Action on Radon:** https://takeactiononradon.ca/smoking-and-radon/.
- **Test kit information, Take Action on Radon:** https://takeactiononradon.ca/provinces/british-columbia/.
- **Find a professional, Canadian National Radon Proficiency Program:** https://c-nrpp.ca/find-a-professional/.

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BC moving to simplified IV N-acetylcysteine protocol for acetaminophen overdose

The BC Drug and Poison Information Centre (DPIC) will be transitioning to a standardized pan-Canadian protocol for IV N-acetylcysteine (NAC) for the treatment of acetaminophen overdose.

The current three-bag regimen will be replaced by a single concentration given in two steps. DPIC is prepared to launch the provincewide transition to the revised protocol by 1 November 2026, although individual health authorities may launch at earlier or later dates. The goal is for all BC health authorities to complete the transition by the end of 2026.

The change is in response to safety concerns raised by the Institute for Safe Medication Practices Canada, which identified inconsistent protocols and complex dosing stages as contributors to NAC infusion errors.[1] The traditional three-bag regimen required three separate preparations and concentrations, and medication errors have been reported in up to 33% of cases.

The new regimen uses a standardized 3% (30 mg/mL) solution, and the simplified protocol involves administering a loading dose of 150 mg/kg over 1 hour, then reducing the infusion rate to 15 mg/kg/hour as a continuous maintenance infusion for at least 20 hours. The total dose of NAC delivered over the first 21 hours is 450 mg/kg, compared with 300 mg/kg with the three-bag protocol, providing greater protection in cases of massive overdose or established hepatotoxicity. In addition, the revised protocol no longer requires ad hoc dose adjustment in massive overdoses or for patients on hemodialysis.

The new protocol also includes clearer stopping criteria, with the infusion continued until all of the following are met:

- Acetaminophen is below 66 µmol/L.
- Alanine aminotransferase and/or aspartate aminotransferase is normal or declining by more than 25% from peak.
- INR is below 2.0 and falling (without the use of fresh frozen plasma).
- The patient is clinically well, without evidence of encephalopathy.

Order sets, infusion references, and monographs in each health authority will be aligned with the revised protocol. Clinicians who manage acetaminophen overdose are encouraged to review the changes and the order sets at their local institutions.

DPIC is available 24 hours a day at 1 800 567-8911 for advice on suspected or confirmed acetaminophen overdose and for questions about the transition.

For more information from DPIC, see the N-acetylcysteine (NAC) 2 Dose for Acetaminophen Toxicity Resource Page: www.dpic.org/content/n-acetylcysteine-nac-2-dose-acetaminophen-toxicity-resource-page.

—Daniel Ovakim, MD, FRCPC
Medical Toxicologist, BC Drug and Poison Information Centre

Reference

1. Institute for Safe Medication Practices Canada. When the antidote causes harm: Preventing errors with intravenous acetylcysteine. ISMP Canada Safety Bulletin 2023;23(7). Accessed 19 September 2026. https://ismpcanada.ca/bulletin/when-the-antidote-causes-harm-preventing-errors-with-intravenous-acetylcysteine/.

For the love of service, faith, and family: Dr Birinder Narang

Dr Narang stays focused on his patients while also advocating for meaningful, sustainable change to advance primary care for the benefit of a diverse spectrum of communities.

Sujatha Nilavar, MD, CCFP, ABFM

For Dr Birinder Narang, the communities he serves are his greatest reward. While he channels his energy into caring for patients as a Burnaby-based family doctor, he also advocates for improved conditions for the profession and ensuring that all people can access a family doctor—work he will continue as incoming president of BC Family Doctors. Dr Narang is also helping shape the next generation of doctors as a clinical assistant professor in the University of British Columbia Department of Family Practice and Simon Fraser University's Stephens Family School of Medicine. Beyond these roles, Dr Narang looks to his faith and his deep connection to the Sikh community as the foundation of his purpose. The Sikh principle of "oneness," or an energy that connects all people, guides his daily interactions with patients, treating them equally with dignity and compassion.

Dr Nilavar is a physician representative on Doctors of BC's Community Physician Health and Safety Oversight Working Group, a community physician lead for the Physician Quality Improvement initiative in the Fraser Health Authority, a family physician at Azure Medical Clinic, and past co-chair of the Surrey-North Delta Division of Family Practice board of directors.

This article has been peer reviewed.

Born in the United Kingdom, Dr Narang moved to Halifax at age 6 before eventually settling in British Columbia. Growing up with a psychiatrist father and an occupational therapist mother gave him early exposure to the health care field. While their careers influenced his path, he points to his Sikh upbringing as his main motivation. Sikhi encourages a life of service to the community, and Dr Narang saw family medicine as a route to practising a community-based discipline. He completed medical school in Australia and residency training in Winnipeg, experiences that shaped his understanding of how deeply health care delivery is influenced by local systems. "The real world of medicine," he reflects, "is far more complex than what we learn in training. [It's] something I also impart on my students."

Commitment to service

Dr Narang serves patients across age groups, cultures, and backgrounds, but he is drawn to helping marginalized communities that have barriers to seeking traditional care. In line with his pursuit of a community approach to medicine, he works at the REACH Community Health Centre in East Vancouver. The centre provides community-based health care and emphasizes a collaborative, compassionate, and culturally sensitive approach to patient care.

For Dr Narang, building trust and patience are key to providing compassionate care to all patients. "You learn quickly that trust goes a long way," he says. "It shapes how decisions are made, especially when patients are navigating complex or difficult circumstances." Through his patients, he has developed a deeper understanding of the realities of how people from different backgrounds manage their health.

Collaboration through crisis

Collaboration is another area that fills Dr Narang with pride. During the COVID-19 pandemic, he contributed to the South Asian COVID Task Force, along with several other well-respected Canadian physicians. The purpose was to provide accurate information about the virus and vaccine safety in several South Asian languages to ensure these communities were able to access lifesaving information. This was rewarding work. As Dr Narang remarked, "It was an opportunity to work together to provide myth-busting information in a culturally appropriate way."

In addition, Dr Narang and the task force helped launch a national campaign called "This Is Our Shot" to stamp out vaccine hesitancy across Canada, with the support of several celebrities, including BC's own Ryan Reynolds. The campaign aimed to communicate with diverse audiences, and its campaign slogan T-shirt was translated into 36 languages. It was a successful awareness-building campaign, selling 65 000 T-shirts and amplifying confidence-building information on vaccines. The campaign was a perfect fit for the task force, because it ensured that South Asian communities were included in this important conversation.

Future of family medicine

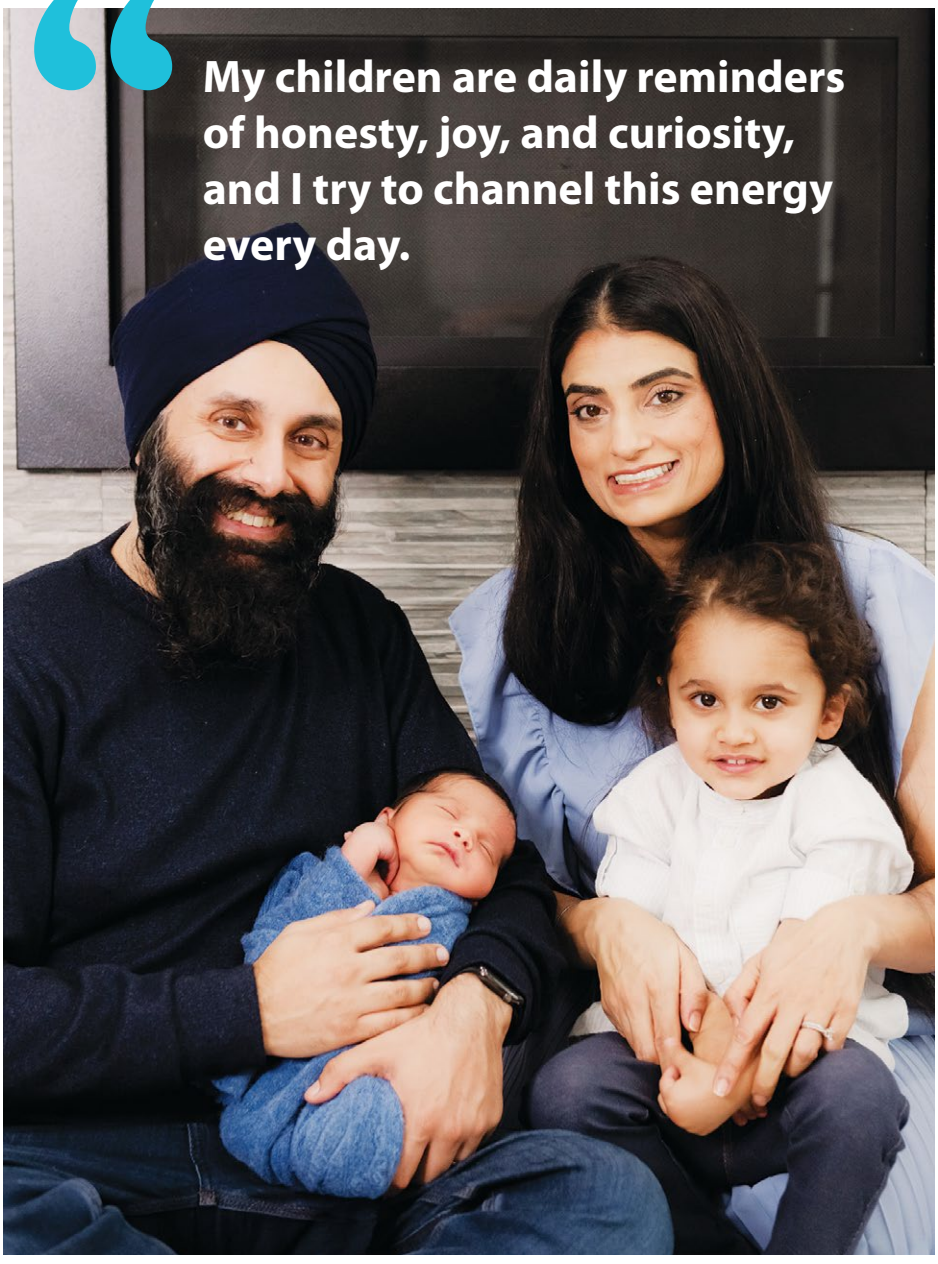
In Dr Narang's opinion, the future of family medicine is bright, most notably because of digital health. Digital health uses technology and digital tools to connect different parts of the health care system. These tools help people manage their health and well-being while enabling clinics and hospitals to share information, coordinate services, and provide better-quality care. As co-chair of Doctors of BC's Joint Executive Table Digital Referrals and Orders program and chair of Doctors of BC's Digital Health Caucus, Dr Narang is helping lead efforts to modernize how health information is digitally captured and exchanged and expand the reach to patients and fellow physicians across British Columbia. As part of this work, he supports improving the governance of electronic medical records in BC to help achieve interoperability. The Digital Referrals and Orders program has a goal of enhancing patient safety and reducing administrative burden for physicians.

The promise of digital health, artificial intelligence–enabled documentation tools, and other technologies designed to reduce administrative burden are all areas anticipated to make a difference in health care delivery. As Dr Narang states, "It's not the technology itself that is exciting but the potential to improve care for both patients and family doctors."

Grounded in faith and family

A key theme in Dr Narang's story is his commitment to faith. His greatest inspiration comes from the examples set by those around him in the Sikh community. Growing up, he was shaped by individuals who showed that it was possible to pursue Western opportunities while remaining rooted in faith, values, and service.

Many of these mentors came through the Sikh Research Institute (SikhRI), a global not-for-profit dedicated to Sikh education and research. Dr Narang's mother played a foundational role in establishing SikhRI in Canada, helping secure its not-for-profit status and serving as chair. Today, Dr Narang continues that connection through his service on SikhRI's Advisory Council, supporting the organization's broader work to make Sikh wisdom, history, and thought more accessible to communities around the world.



Family photo, left to right: Dr Birinder Narang holding Giaan Narang, Dr Jaspreet Mudhar, and Sevak Narang.

In addition to Dr Narang's faith, family is where his heart is. He always looks forward to evenings and weekends when he can be at home with his wife and two children, a toddler and a newborn. "My children are daily reminders of honesty, joy, and curiosity, and I try to channel this energy every day."

Looking ahead

While Dr Narang is always involved in several projects advancing primary care, he is not interested in becoming removed from the realities of care. He plans to stay close to his patients while advocating for meaningful, sustainable change. He intends to continue his community approach to medicine while advocating for improvements that benefit a diverse spectrum of communities. ■

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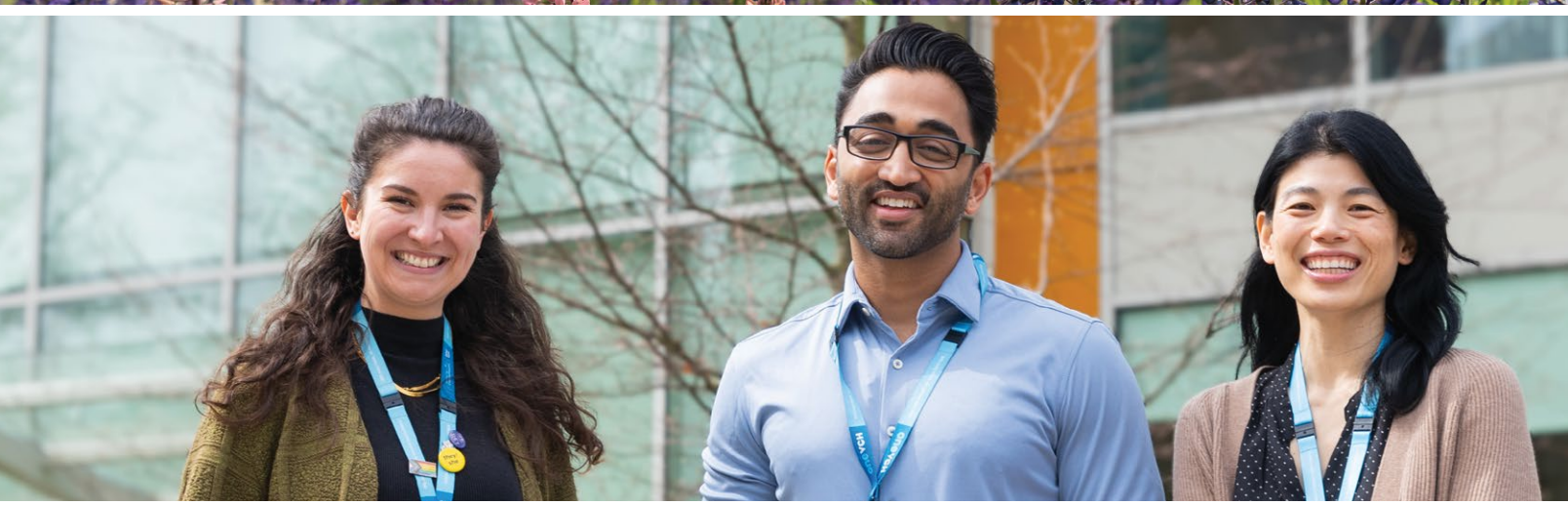
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