Your Guide to Hospital Care 2017

Pancreatic exocrine insufficiency

Pancreatic exocrine insufficiency (PEI) can be caused by a number of conditions, including cystic fibrosis, chronic pancreatitis, acute pancreatitis, pancreatic cancer, and upper gastrointestinal surgery, including pancreatectomy and gastrectomy.[1,2]

When PEI is suspected, a pancreatic function test should be performed.[2] The most widely used non-invasive test is faecal elastase-1 (FE-1) level. It is reliable in patients with moderate-to-severe PEI. A single stool sample is used for analysis with concentrations of less than 200 mcg/g of stool indicating mild PEI, and less than 100mcg/g indicating severe PEI.

Use of pancreatic enzyme replacement therapy

Pancreatic enzyme replacement therapy (PERT) is the pharmacological mainstay of treatment of PEI. Newer preparations contain pancreatic extract encapsulated in microtablets or mini-microspheres with pH-sensitive enteric coating. The enzymes mix intragastrically with the chyme yet are protected from acid degradation by the enteric coating. The higher pH found in the duodenum dissolves the coating to release the enzymes at the appropriate site.[2]

The Australasian Pancreatic Club updated their guidelines on the management of PEI in late 2015. Motivating this update was the knowledge that some patients are not being prescribed enzymes when their use has clear clinical benefits (eg, reduction in pain and steatorrhea) and a desire to evaluate new evidence since the previous guidelines were published.[2]

Key recommendations for the use of PERT[2]

- Commence on 25,000 to 40,000 units lipase with each meal (adults)
- Titrate dosage up to 75,000 to 80,000 units lipase with each meal according to presence of malabsorption (adults)
- Trial acid suppressing agents in patients failing to respond to high-dose PERT

Australasian Pancreatic Club:
www.tinyurl.com/zz7xpvq

1. Lindkvist B. World J Gastroenterol 2013;19:7258-66. **2.** Australasian Pancreatic Club. (Accessed February 29, 2016, at www.tinyurl.com/zz7xpvq) **3.** Lohr JM, et al. United European Gastroenterol J 2013;1:79-83

Preventing pests in hospitals and nursing facilities

Hospitals have a vested interest in maintaining a clean environment, free of infectious agents and pests. In their unrelenting search for food, pests contaminate medical supplies and equipment such as dressing materials, intravenous drips, syringes, catheters; hygiene sensitive surfaces and drains or waste bins, through feeding, droppings and the shedding of skin or tissue. With any pest infestation easily leading to disease outbreaks, particularly vector-borne and food contamination diseases, ongoing pest management is an essential requirement for hospitals.

The problem

Hospitals and nursing facilities are especially vulnerable to pests due to the multitude of access points through constant deliveries, storage and preparation of food, high patient or resident turnover, frequent visitors and structural defects.

Any evidence of pests inevitably leads to a profound loss of trust on the part of patients or residents, relatives, and employees who will typically link an infestation to poor hygiene standards.

Integrated pest management programs

An integrated pest management (IPM) program is key to overcoming the challenges and potential risks associated with pest infestation. IPM programs essentially anticipate and prevent pest activity through the integration of various strategies with the ultimate goal of achieving a long-term solution. The key components of an IPM program are:[1]

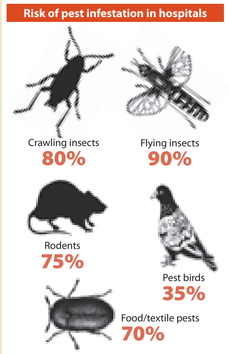
- Education
- Structural repair
- Proper waste management
- Biological and mechanical control techniques
- Judicial use of pesticides
- Ongoing maintenance

Good housekeeping and sanitation practices are the first steps in preventing pest infestation.

As part of an IPM program, these and other measures can help prevent the consequences of pest infestation in the hospital setting, including:

- Contamination of medical equipment
- Infections resulting in serious medical complications
- Litigation in the event of non-compliance with applicable hygiene or health and safety legislation
- High costs of treating a full-blown pest infestation in comparison with the relatively modest cost of a preventative pest control program

Risk of pest infestation in hospitals



1. Kunst RL. In: Robinson WH, Bajomi D, editor. Sixth International Conference on Urban Pests; 2008; Budapest, Hungary: OOK-Press Kft; 2008. p. 287-90

Reliably detecting AF

Automated blood pressure monitors that detect pulse irregularity are being more widely used to screen for AF.[1] In fact the UK National Institute for Health and Care Excellence (NICE) recommends the use of one such device in the primary care setting – the WatchBP Home A device – to increase the rate of detection of AF in patients having their blood pressure (BP) measured.[2]

How the device works

The WatchBP Home A device automatically detects pulse irregularity that may be caused by AF. It has an embedded algorithm that calculates the irregularity index (standard deviation divided by mean) based on interval times between heartbeats. If the index is above a defined threshold, AF is likely and an icon indicating AF is displayed on the device screen.[2]

Clinical evidence

Pooled data from 6 studies on the diagnostic accuracy of the algorithm embedded in the device estimated a sensitivity of 0.98% (95% CI 0.95, 1.00) and specificity of 0.92 (0.88, 0.96). Taking 3 sequential readings with at least 2 detecting AF gave the highest diagnostic accuracy. A single study of paroxysmal AF screening demonstrated sensitivity of 99% and specificity of 93%. Another study suggested that AF detected in >15% of all readings has a high probability of AF diagnosis, requiring confirmation by 24-hour electrocardiography.[3]

Candidates for screening

Regular use of this device is likely to detect prolonged asymptomatic episodes of AF.[1] Patients admitted with trans-ischaemic attacks and congestive heart failure should be considered candidates for screening, as should patients who have experienced a stroke. At discharge, at-risk patients should be advised to continue monitoring for AF in the outpatient setting either through regular GP visits or by purchasing an appropriate device to use in the home setting.

Atrial Fibrillation Association Australia:
www.tinyurl.com/oa6xp5c

Irregular heartbeat detection vs. AFIB technology

- There is confusion in the market about the difference between the 'irregular heartbeat detection' type technology, and the AFIB detection technology.
- Irregular heartbeat detection is a technical function designed, not to diagnose arrhythmias, but rather to serve as a warning message indicating that the BP reading may not be accurate because of the presence of arrhythmia.[3]
- AFIB technology is a clinical function designed to specifically detect the presence of AF.[3]

1. Wiesel Jet al. Am J Hypertens 2009;22:848-52. **2.** National Institute for Health and Clinical Excellence (NICE). WatchBP Home A for opportunistically detecting atrial fibrillation during diagnosis and monitoring of hypertension. January 2013. **3.** Verberk WJ, et al. Int J Cardiol 2016;203:465-73.

NOACs – attitudes and adherence

For many years oral Vitamin K antagonists (VKAs) such as warfarin were the mainstay of therapy to risk the reduction of stroke in patients with non-valvular atrial fibrillation (NVAF). The introduction of non-VKA oral anticoagulants (NOACs), which offer non-inferiority to warfarin for stroke prevention and systemic embolism and generally favourable risk-benefit profiles, has seen these agents become firstline therapy for most patients.[1] Two recent studies shed light on physician and patient preferences regarding OACs[1] and adherence rates to individual agents.[2]

Preferences

In one study, physicians and patients were asked to evaluate factors relating to the use of NOACs, including effectiveness in stroke prevention, requirements for regular blood testing, dosing regimen, and known interactions with foods and/or other drugs. Patients rated efficacy, safety, and recommendation by their physician as the most important factors.[1]

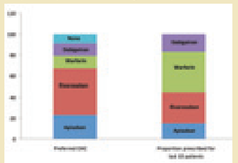
Among physicians, the efficacy of the agent, the evidence base for its use, and safety profile were ranked as the most important attributes. Rivaroxaban was the preferred OAC for stroke prevention (44% vs. 23%, 12%, and 12% for apixaban, dabigatran, and warfarin, respectively; P<0.001). Interestingly however, warfarin was the most frequently prescribed agent, at least among the last 10 patients (Figure 1).[1]

Adherence

In the previous study, patients taking once-daily medications demonstrated better compliance with their OAC therapy. Around 30% of medications prescribed twice-daily were taken once-daily, with significantly more missed doses. Patients taking rivaroxaban were significantly less likely to consider stopping treatment compared with those taking other OACs (8% vs. warfarin 18% [P=0.022]; dabigatran 18% [P=0.080]; and apixaban 27% [P=0.022]).[1]

In another study of health claims data, 73% of patients taking rivaroxaban were adherent compared with 67% of dabigatran and 70% of apixaban users (P<0.001).[2]

Figure 1. Physician-preferred OAC and relative proportion prescribed among last 10 patients.[1]



Australian Atrial Fibrillation Association
www.tinyurl.com/oa6xp5c

1. Andrade JG, et al. Can J Cardiol 2016;32:747-53. **2.** McHorney CA, et al. Curr Med Res Opin 2015;31:2167-73.

Zeroing in on xerostomia

Xerostomia is generally defined as the subjective complaint of dry mouth,[1] affecting around 20% of the population.[2] The prevalence rises to around 30% in people ages 65 years and older,[3] with studies suggesting around 60% of hospitalised elderly patients with physical disabilities[4] and around 70% of terminally ill patients experience dry mouth.[5]

Causes

Xerostomia may be caused by systemic diseases, salivary gland dysfunction, and head and neck radiotherapy. However it is most frequently associated with the use of certain medications, and can affect 70% of adults taking some type of medication.[2] A study of elderly patients demonstrated a significant independent association between xerostomia and antidepressants (P=0.03) and benzodiazepines (P=0.01).[3]

Classes of medications associated with xerostomia:[2]

- Anticholinergics, antimuscarinics
- Diuretics
- Antihypertensives
- Antidepressants and antipsychotics
- Antihistamines
- Sedatives and anxiolytics
- Muscle relaxants
- Opioid analgesics
- Non-steroidal anti-inflammatory drugs

Sequelae

Xerostomia can be associated with pain, dental caries, reduced taste and smell, and increased risk of oral infections.[6] Compared with patients with mild xerostomia, those with severe dry mouth are 2.3 to 4.9 times more likely to experience a negative impact on health.[7]

In addition, xerostomia can have a negative impact on quality-of-life, particularly in cancer patients receiving treatment, where it can be a significantly troubling and ongoing problem.[8]

Addressing the symptoms

If underlying conditions have been identified and treated, yet xerostomia persists, saliva substitutes may be helpful in relieving the symptoms. These mimic the key properties of saliva, including its viscosity, lubrication, wetting properties, and antimicrobial effects. Oral gel formulations that have a thicker texture may provide a longer-lasting moisture sensation and thus are recommended for use at night.[1]

1. Sasportas LS, et al. Oral Surg Oral Med Oral Pathol Oral Radiol 2013;116:e37-51. **2.** Miranda-Rius J, et al. Int J Med Sci 2015;12:811-24. **3.** van Eijk J, et al. J Am Geriatr Soc 2013;61:1836-7. **4.** Avcu N, et al. Arch Gerontol Geriatr 2005;41:69-79. **5.** Chaushu G, et al. Cancer 2000;88:984-7. **6.** Feng J, Coppes RP. ScientificWorldJournal 2008;8:959-62. **7.** Matear DW, et al. J R Soc Promot Health 2006;126:79-85. **8.** van den Beuken-van Everdingen MH, et al. J Pain Symptom Manage 2009;38:216-33.

NSAIDs and postoperative pain management

The mainstay of managing moderate-to-severe postoperative pain are opioids, generally delivered by patient-controlled analgesia (PCA). However, adequate pain relief with these agents is not guaranteed[1] and side effects limit their use in many patients.[2] Another disadvantage of opioids is that they do not interrupt the inflammatory component of pain.[3] On the other hand non-opioids, such as non-steroidal anti-inflammatory drugs (NSAIDs), affect the chemical changes at the site of injury that typically result in inflammation and increased pain sensitivity.[4] NSAIDs inhibit the conversion of arachidonic acid to prostaglandins and thus prevent sensitisation of pain receptors in response to injury[3-5] and also have antipyretic properties.

The efficacy of IV ibuprofen for post-operative pain has been investigated in three key Phase III randomised, placebo-controlled, double-blind studies in orthopaedic, abdominal, and gynaecological surgery. (All patients had access to morphine PCA)[3,6-8]

Orthopaedic patients receiving IV ibuprofen 800mg experienced significant reductions:[6,7]

P<0.001 for all 3 primary efficacy outcomes for IV ibuprofen 800 mg vs. placebo

Gynaecological surgery patients also reported a 19% reduction in the use of morphine among patients receiving ibuprofen 800 mg IV every 6 hours (*P*<0.001) and significant reductions in pain at rest and movement. Also, time to ambulation was significantly shorter in the IV ibuprofen group (23.4 hours vs. 25.3 hours; *P*=0.009).[8]

Compared with placebo, IV Ibuprofen was generally well tolerated, with no significant differences in bleeding, renal and gastrointestinal side effects (*P*-values not reported).[3,6-9]

Role of NSAIDs in multimodal therapy

A multimodal approach to pain relief has the advantages of:

- Combining different agents that act at different receptor sites, producing synergistic or additive effects[2,5]
- Lowering the dose of opioids required, thereby reducing the potential for opioid-related side effects[5,6]
- Intravenous administration of an NSAID as part of a multi-modal postoperative pain regimen has the potential to halt the inflammatory cascade associated with surgery, thus reducing or preventing post-operative pain[5,6]

1. Sadhasivam S, Chidambaran V. Pharmacogenomics 2012;13:1719-40. 2. Corke P. Aust Prescr 2013;36. **3.** Southworth S, et al. Clin Ther 2009;31:1922-35. **4.** E-med Experts. (Accessed June 15, 2016, at **www.tinyurl.com/lv4tyo**) **5.** McIntyre PE. Schug SA. CRC Press; 2015. **6.** Singla N, et al. Pain Med 2010;11:1284-93. **7.** Caldolor approved product information, May 2015. Parkville, VIC: bioCSL Pty Ltd. **8.** Kroll PB, et al. Pain Pract 2011;11:23-32. **9.** Southworth SR, et al. J Pain Res 2015;8:753-65.

Codeine dependence

Codeine is used as an analgesic in both prescription and over-the-counter (OTC) preparations in Australia. It is often combined with paracetamol or ibuprofen, non-steroidal anti-inflammatory drug (NSAIDs) codeine-containing cough suppressants.[1]

The main reasons why codeine-containing preparations can be problematic when misused:[2]

- Codeine can be addictive.
- If codeine-containing medicines are taken in doses higher than recommended, more of the other active ingredients in the medicine are also being consumed, which can cause serious side effects.
- Codeine may be associated with side effects, such as nausea, constipation, dizziness, and, in some cases, respiratory depression.

Dependence and misuse

Although codeine has a role to play in the management of pain relief, misuse of codeine-containing preparations may lead to the development of a physical and psychological dependence and has been associated in some cases with increased mortality.[3] Experts have often referred to codeine dependence as the "hidden addiction" because codeine is readily available with some restrictions.

Many people with codeine dependence may have a high prevalence of comorbid conditions, including pain, mental health, and substance abuse problems; so it is clearly necessary to increase the capacity to identify those at high-risk and to respond more effectively to their needs.

Recognising codeine dependence

Hospital clinicians have a key role to play in identifying codeine dependence. Patients showing any of the following behaviours may benefit from being evaluated and closely monitored for signs of opioid painkiller dependence:

- Use of multiple opioid medications.
- Reports of opioid withdrawal symptoms such as:[4]
  - Cravings for codeine
  - Dilated pupils
  - Abdominal cramps, diarrhoea, nausea, vomiting
  - Lack of appetite
  - Runny nose and sneezing
  - Yawning and difficulty sleeping
  - Trembling, aching muscles and joints
  - Goosebumps, fever, chills, sweating
  - Restlessness, irritability, nervousness, depression

Clinicians should ensure that patients prescribed codeine-containing preparations at discharge are aware of the potential for abuse and offer patients clear guidance on how long the medication should be taken. In addition, physicians responsible for patients in the outpatient setting should be made aware through discharge letters that their patients have been prescribed a codeine preparation.

Treating dependence

There are a number of approaches to treating codeine-dependence. These include abstinence-focused programs, behavioural interventions, and self-directed intervention such as participation in Narcotics Anonymous. In situations where these measures are unsuccessful or are not appropriate, Medication Assisted Therapy or MAT (formerly known as opioid substitution therapy) is considered.[5] MAT is indicated for opioid withdrawal and long-term maintenance. To manage withdrawal symptoms, reducing doses of an opioid substitute (eg, buprenorphine, methadone) are administered for approximately a week, followed by referral to counselling services.[5] Relapse following detoxification alone is common, thus most patients embark on abstinence-oriented treatment and substitution maintenance treatment after initial detoxification.[6]

If you suspect one of your patients may be dependent on codeine or other opioids, try and discuss your concerns and refer the customer to an addiction specialist for further assessment and appropriate treatment.

1. Crews KR, et al. Clin Pharmacol Ther 2014;95:376-82. **2.** NPS Medicinewise. (Accessed September 6, 2016 at **www.tinyurl.com/zljwx5c**) **3.** Roxburgh A, et al. Med J Aust 2015;203:299 **4.** DrugInfo. Codeine Facts. (Accessed September 6, 2016 at **www.tinyurl.com/z5rhx35**) **5.** McDonough M. Aust Prescr 2013;36:83-7. **6.** World Health Organization, United Nations Office on Drugs and Crime, UNAIDS. (Accessed September 6, 2016 at **www.tinyurl.com/hbnkyol**)

Turn to Help www.turntohelp.com.au

PBS INFORMATION: Authority Required (STREAMLINED).
Refer to PBS Schedule for full authority information.

PLEASE REVIEW THE FULL PRODUCT INFORMATION BEFORE PRESCRIBING. THE APPROVED PRODUCT INFORMATION IS AVAILABLE UPON REQUEST FROM BAYER AUSTRALIA LIMITED AND CAN BE ACCESSED AT WWW.BAYERRESOURCES.COM.AU/RESOURCES/UPLOADS/PI/FILE9466.PDF.

Minimum Product Information. XARELTO® (rivaroxaban) INDICATIONS: Prevention of venous thromboembolism (VTE) in adult patients who have undergone major orthopaedic surgery of the lower limbs (elective total hip replacement, treatment for up to 5 weeks; elective total knee replacement, treatment for up to 2 weeks); 10 mg tablet once daily. Prevention of stroke and systemic embolism in patients with non-valvular atrial fibrillation and at least one additional risk factor for stroke; 20 mg tablet once daily (15 mg for patients with CrCl 30-49 mL/min). Treatment of deep vein thrombosis (DVT) and pulmonary embolism (PE) and for the prevention of recurrent DVT and pulmonary embolism (PE); 15 mg tablet twice daily for 3 weeks, followed by 20 mg tablet once daily. Xarelto 15 mg and 20 mg tablets should be taken with food. Tablets may be crushed and administered orally (mixed with water or applesauce) or given through gastric tubes. See full PI for details. CONTRAINDICATIONS: Hypersensitivity to rivaroxaban or to any of the excipients, clinically significant active bleeding, lesions at increased risk of clinically significant bleeding and patients with spontaneous impairment of haemostasis, significant hepatic disease which is associated with coagulopathy, dialysis or severe renal impairment with a creatinine clearance < 15 mL/min for Xarelto 10 mg or < 30 mL/min for Xarelto 15 mg and 20 mg, concomitant treatment with strong inhibitors of both CYP 3A4 and P-glycoprotein, Pregnancy, Lactation. PRECAUTIONS: Increased bleeding risk such as general haemorrhagic risk (see PI for list), bronchiectasis or history of pulmonary bleeding, renal impairment, hepatic impairment, surgery and interventions, spinal/epidural anaesthesia or puncture, patients with prosthetic valves (no clinical data), haemodynamically unstable PE patients or patients who require thrombolysis or pulmonary embolectomy, lactose intolerance. INTERACTIONS WITH OTHER MEDICINES: Care to be taken if concomitantly used with medicines affecting haemostasis; concomitant administration with NSAIDs, platelet aggregation inhibitors, other anticoagulants. ADVERSE EFFECTS: Please refer to PI for a complete list. Very common and common adverse reactions (≥ 1%) include post procedural haemorrhage, increased transaminases, gingival bleeding, constipation, diarrhoea, nausea, pyrexia, oedema peripheral, contusion, pain in extremity, headache, dizziness, haematuria, menorrhagia, epistaxis, haematoma, anaemia, rectal haemorrhage, fatigue and ecchymosis, haemoptysis, pruritus, conjunctival haemorrhage, abdominal pain, dyspepsia, gastrointestinal haemorrhage, syncope, hypotension, increased gamma-glutamyltransferase, tachycardia, vomiting, asthenia, wound haemorrhage, subcutaneous haematoma and rash. Less frequent but serious adverse reactions include: urticaria, hypersensitivity, hyperglycaemia, cerebral, cerebellar and intracranial haemorrhage, haemorrhagic transformation stroke, jaundice, eye haemorrhage, loss of consciousness, angioedema, allergic oedema, cholestasis, hepatitis and thrombocytopaenia. DOSAGE AND ADMINISTRATION: see INDICATIONS above. BASED ON PI DATED: 09 Nov 2015.

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