SPRING 2014

The Wesley HOSPITAL
Caring for you for life

Medilink

Medical Professionals' direct link
to programs and services at the Wesley

Next generation genetics for the next generation: Dr David Coman

Articles in this issue:

Welcome

Dr Luis Prado

Director of Medical Services

Welcome to the Winter 2014 edition of Medilink.

The Wesley Hospital's GP continuing medical education program is a very important part of our commitment to the GP community both in Brisbane and throughout Queensland.

So far this year we have held seven Continuing Professional Development events (CPDs), four regional CPDs (at Mackay, Hervey Bay, Gold Coast and Sunshine Coast) and two Active Learning Modules (ALMs). These popular events provide a regular forum to update our referring doctors on medical advancements at The Wesley Hospital and to network with their peers. I am very pleased by how successful the program has become. Since launching 10 years ago, our GP education program has gone from strength to strength with more than 80 GPs taking part in our most recent ALM on General Medicine.

One of the highlights of the year to date was our 'Q&A' with the ABC's Tony Jones, exploring the topic of palliative care. This is the second year that the renowned broadcaster has hosted the panel discussion and we look forward to staging a third Q&A with Tony next year. As not every GP can attend these educational events, we are pleased to offer in this publication a selection of articles written by our specialists on topics ranging from paediatrics to gastroenterology.

Along with professional development, improving our services and facilities to ensure the best patient outcomes is a top priority. The newly expanded Wesley Emergency Centre (WEC) is now fully operational with 17 patient bays and an expanded waiting room, making WEC the largest private emergency centre in the state.

Phase one of The Wesley's expanded theatre complex opens in October, with three new operating theatres including a state-of-the art hybrid theatre for endovascular surgery. The $20 million redevelopment project, due for completion in March, also sees the enlargement and refurbishment of four existing theatres, bringing the total number of theatres to 19, making the complex the largest in a private hospital setting in Queensland. The new theatre complex has been named after surgeon Professor Russell Stitz in honour of his dedication to the Wesley. We are particularly excited about the advanced imaging technology in the new hybrid theatre which will greatly enhance our cardiac and vascular services.

The Wesley has also installed a second da Vinci robot system as the hospital's specialists advance further into the areas of gynaecology, colorectal surgery and general surgery. In yet another Australian first for the hospital, Dr David Cavallucci performed the first partial hepatectomy in June, an outstanding achievement for the doctor and his team. ■

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The Wesley's expanded theatre complex opens in October, with three new operating theatres including a state-of-the-art hybrid theatre for endovascular surgery

Business Development Update

Vicki Goss

Business Development Manager

Our second annual Q&A with ABC journalist Tony Jones on palliative care was a huge success. Tony was initially hesitant about the topic as he thought it may be too sombre to discuss at a dinner – but this was certainly not the case. Dr Ralph McConaghy, Director of The Wesley Palliative Care Service, made it very light and engaging for the audience, and after the event Tony said he would like to explore the topic of palliative care on to his television show, so we are thrilled that he wants to bring this often neglected subject to a national audience.

For the remainder of the year, we will be stepping up our program of GP Continuing Medical Education events to fortnightly. Please see the schedule for the dates for the remainder of 2014. At GPs request, we have added a Cardiology CPD event in October, our second for the year, and are having a Paediatric CPD event in November. With The Wesley's new high-tech hybrid theatre opening in October, our CPD on Vascular in November is likely to be very well attended. We look forward to seeing you there.

If you are interested in having specialists visit your practice or would like more information on our CPD program please contact me via email vicki.goss@uchealth.com.au or 07 3232 7258. ■

1. Host Tony Jones addresses the conference
2. Dr Ralph McConaghy
3. Ms Vicki Goss, Mr Tony Jones and Associate Professor Luis Prado
4. Dr Richard Kidd ponders the topic of treating the terminally ill
5. The panel: Dr Ralph McConaghy, Director of Wesley Palliative Care Service; Mr Terence Seymour, Chief Strategy Officer, UnitingCare Health; Dr Luis Prado, Chief Medical Officer, UnitingCare Health and Director of Medical Services, The Wesley Hospital; Associate Professor Deborah Parker, Director, UQ/Blue Care Research and Practice Development Centre; Dr Richard Kidd, GP; ABC journalist Tony Jones; Reverend Helen Dick, Director of Mission, UnitingCare Health; Associate Professor Greg Scalia, Cardiologist, Heartcare Partners; and Dr David Schlect, Radiation Oncologist, The Wesley Hospital.

Clinical Education 2014

CPD The Wesley Hospital	ALM The Wesley Hospital	Regional
16 SEPTEMBER Infectious Disease		**10 SEPTEMBER** Prostate Cancer Rockhampton
2 OCTOBER Cardiology Part 2	**11 OCTOBER** CPR	
21 OCTOBER Intensive Care		**1 NOVEMBER** ALM – Cardiology Hervey Bay
6 NOVEMBER Paediatric		
18 NOVEMBER Vascular		

Please note: Topics and dates are subject to change

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Wesley Emergency Centre expands to match demand

The Wesley Hospital has completed a substantial renovation and expansion of its emergency centre underlining its 20-year commitment to private emergency medicine.

Ongoing high demand for the Wesley Emergency Centre has driven the expansion now making it the largest private emergency department in Brisbane.

Dr Gavan Doig, Director of Emergency Medicine at The Wesley Hospital, said the growth in demand for emergency services at the hospital had well and truly outstripped the prediction made two decades ago.

"We now have 19 emergency beds including resuscitation, consultation, paediatric and procedure rooms along with the latest state-of-the-art patient monitoring equipment," Dr Doig said.

"The hospital's substantial investment to increase capacity is about improving the patient and their relatives' experience in the centre with increased privacy and greater efficiency.

"Our emergency centre treats a wide range of patients from paediatric through to geriatric, orthopaedic, respiratory and cardiac with excellent backup from the hospital's inpatient teams. We also handle the myriad of minor injuries and common illnesses seen in all emergency departments," he said.

The Wesley Emergency Centre is accessed directly from the hospital's main entrance in Chasely Street. An adjacent X-ray facility and on-site pathology laboratories allow for rapid imaging and pathology results with minimum delays for unwell patients.

The Wesley Emergency Centre is staffed by a team of nearly 50 medical and nursing staff including 14 permanent emergency doctors three of whom have worked at the centre since it opened in 1994.

To contact Wesley Emergency Centre call 3232 7333 or visit www.wesley.com.au ■

The Wesley Hospital tops Press Ganey patient survey

A comprehensive survey measuring patient satisfaction has ranked The Wesley Hospital the highest for private hospitals of its size in Australia and New Zealand.

The Press Ganey survey results from 717 patients discharged from the Wesley between 1 August 2013 and 30 November 2013 revealed the hospital has increased its satisfaction levels significantly across almost all key areas since the previous year's survey.

"I am thrilled to announce that the Wesley was ranked the highest in the group of private hospitals of our size of more than 300 beds," Wesley General Manager Ann Maguire said.

"We attribute a large part of our success to the introduction of the Living Values program which includes initiatives such as hourly rounding and patient communication noticeboards."

The greatest improvements are recorded in the areas of waiting time for admission to ward, staff involving family in their care, pre-admit preparing the patient for their stay, promptness response to call button, and nurses keeping patients informed.

Ms Maguire said the impact of these incremental across-the-board improvements in both clinical and non-clinical services had clearly resulted in the rise in the hospital's overall rating. ■

PSMA PET-CT scanning latest technology in prostate cancer detection

Wesley Medical Imaging (WMI) has installed its own Gallium68 generator.

The generator allows the Department of PET and Nuclear Medicine to produce several novel PET tracers not previously available including Ga68 PSMA (Prostate Specific Membrane Antigen).

The first clinical Ga68 PSMA PET-CT scan was performed on 16 July at The Wesley Hospital – proudly a Queensland first.

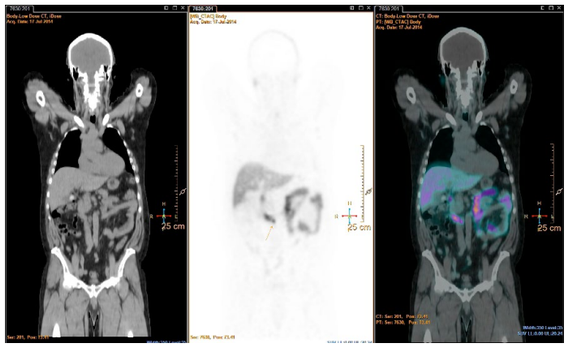
PSMA PET-CT is able to detect small volume recurrent prostate carcinoma when there is a PSA rise on follow-up after prostatectomy or other definitive treatment for prostate carcinoma. The use of PSMA PET-CT in the initial staging in prostate carcinoma is uncertain at this time but WMI radiologists will be in a unique position to undertake clinical research to establish the role of PSMA PET-CT in the management of patients with newly diagnosed prostate carcinoma.

The availability of clinical PSMA PET-CT imaging is very limited in Australia and worldwide presently. PSMA PET-CT does not attract a Medicare rebate but Wesley Medical Imaging will make this new and exciting technique as affordable as possible to patients.

The first PET scan in Queensland was performed at The Wesley Hospital on 8 February, 1999. The availability of PSMA PET-CT imaging at Wesley Medical Imaging reaffirms our position as the premier site for private PET-CT imaging in Brisbane.

The addition of PSMA PET-CT complements the internationally recognised multi-parametric 3T prostate MRI service at Wesley Medical Imaging and further enhances The Wesley Hospital's reputation as a centre of excellence in the diagnosis and management of patients with prostate carcinoma. ■

Sarah Daniel, Nuclear Medicine technologist, Wesley Medical Imaging, with the Gallium68 generator.



PSMA PET-CT showing suspected small malignant retroperitoneal node in a patient with prior radical prostatectomy with low-level PSA rise on follow-up.

Wesley Sleep Disorders Centre gains national recognition

The Wesley Hospital Sleep Disorders Centre has received accreditation with the NATA/ASA Sleep Disorders Services Accreditation Program in 2014, the first private hospital service in Australia to meet this standard.

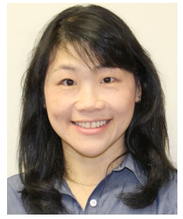
The Thoracic and Sleep Group QLD (TSGQ) provide a team of specialist physicians including Dr Andrew Scott and Dr John Feenstra, sleep scientists and nurses managing patients in the rapidly growing area of sleep health.

For more information, contact the Centre at scott.reception@tsgq.com.au ■

Welcome to our new
Visiting Medical Practitioners

Dr Wen-Yi Chew-Lai

Developmental Paediatrician
BMBS, FRACP, MPH



Dr Wen-Yi Chew-Lai commenced practice as a Developmental Paediatrician at The Wesley Hospital in July 2014. She comes to the hospital with considerable experience in developmental paediatrics with a special interest in children with developmental delays, learning difficulties, autism and attention deficit disorder. Dr Chew-Lai has dual training in both General Paediatrics and Community Child Health encompassing Developmental Paediatrics, Child Protection and Population Health. Other languages spoken include Malay and Cantonese.

Following her graduation from Flinders University in South Australia, Dr Chew-Lai worked in various hospitals and community settings in South Australia and Queensland.

In 2009 she completed a fellowship at the Mater Children's Hospital Child Development Clinic, followed by a brief consultant position at the clinic, and practised for three years as a community paediatrician with Queensland Health's Metro South Children's Developmental Services.

Dr Chew-Lai obtained her Master of Public Health in 2011 and has undertaken Autism Diagnostic Observation Schedule (ADOS) training. She is a Fellow of the Royal College of Physicians (Paediatrics and Child Health division) and member of the Chapter of Community Child Health and Neurodevelopmental and Behavioural Paediatric Society of Australia.

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Dr Melinda Heywood

Obstetrician and Gynaecologist
BSc, MBBS(Hons), FRANZCOG

Dr Melinda Heywood is an Obstetrician and Gynaecologist who joined our extensive team of medical specialists this year.

After graduating from The University of Queensland, Dr Heywood went on to become a Fellow of The Royal Australian and New Zealand College of Obstetricians and Gynaecologists (RANZCOG).

She worked for Queensland Health for 11 years, most recently at Redcliffe Hospital, before joining eXXpectations, a general obstetrics and gynaecology practice based at The Wesley Hospital.

Away from work, she enjoys spending time with her husband, three children, extended family and friends.

Dr Heywood welcomes all referrals and looks forward to working in close collaboration with you to provide holistic multidisciplinary care for all patients. She is available for discussion and advice regarding any concerns you may have.

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Dr Agnieszka Malczewski

Medical Oncologist
MBBS, FRACP

Dr Agnieszka Malczewski is a Medical Oncologist who comes to the Wesley with considerable experience in treating all solid tumour types including breast, gastrointestinal, genitourinary, lung, brain and melanoma.

After graduating from James Cook University in 2005 with a Bachelor of Medicine, Bachelor of Surgery (Hons 1) and the University Medal, she completed her oncology training in Brisbane in 2012, with rotations through the Princess Alexandra, Prince Charles, Redcliffe and the Royal Brisbane and Women's hospitals.

She completed her fellowship at the University of Oxford in 2013, where she had a strong focus on early-phase clinical trials and melanoma. At the same time she conducted laboratory research at the Ludwig Institute for Cancer Research in Victoria, investigating the molecular biology of melanoma. Her experience within this area extends to the new, targeted melanoma treatments and immunotherapies.

Dr Malczewski holds the position of senior lecturer with the University of Queensland.

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Dr Simon Quinn

Vascular and Endovascular Surgeon
Bthty (Hons), MBBS (Hons), FRACS (Vasc)

Dr Simon Quinn is a Vascular and Endovascular Surgeon with interests in the minimally invasive treatment of both arterial and venous pathology, and in complex arterial reconstruction of both aneurysmal and occlusive arterial disease.

Dr Quinn grew up in Brisbane and attended the University of Queensland. After completing a degree in Physiotherapy he undertook a postgraduate Bachelor of Medicine and Bachelor of Surgery, graduating with Honours. He completed his surgical training throughout Queensland and Victoria and was awarded Fellowship of the Royal Australasian College of Surgeons in Vascular Surgery.

Dr Quinn gained sub-specialist training in advanced endovascular techniques for the management of aortic aneurysms while working at St Vincent's and the Epworth hospitals in Melbourne. After his training Dr Quinn visited international quaternary vascular surgical units at the Groningen University Medical Centre and St Mary's Hospital, London to gain further experience in emerging endovascular techniques. He is a consultant Vascular Surgeon at the Royal Brisbane and Women's Hospital and The Prince Charles Hospital and is a senior lecturer at the University of Queensland.

He has particular interest in the treatment of: aortic aneurysm – open and endovascular; carotid artery disease – endarterectomy and stenting; lower limb arterial disease – endovascular and bypass; varicose veins – open, endovenous and sclerotherapy; renal access surgery and diabetic foot management.

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Dr Marion Thomas

Paediatrician
MBChB, MRCP, FRACP

The Wesley is pleased to welcome Dr Marion Thomas who brings a wealth of experience in childrens' and adolescent health to the hospital.

Dr Thomas is a Paediatrician with more than 22 years experience both in the UK and Australia who has recently commenced work at the Wesley Medical Centre.

Her area of speciality is children and adolescents up to 14 years of age working across the spectrum of General Paediatrics, with an emphasis on family-centred holistic care. Complex children with developmental delay and special needs and early feeding and nutritional problems are her special interest area.

Dr Thomas holds dual Australian and British qualifications in Paediatrics and has worked in the UK, at the Royal Children's Hospital in Melbourne, and the Mater and Royal Children's hospitals in Brisbane.

In 2013, she began working at the Wesley Medical Centre with Dr Bruce Lewis and Dr David Coman.

She lives in Brisbane and has four school-aged children including twins.

Dr Thomas invites you to contact her to discuss any questions related to your patients.

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GP Referral Advisory Service

The Wesley Hospital offers a 24/7 referral advisory service to assist GPs in selecting the appropriate specialist in Brisbane for your patients' needs.

For urgent referrals email gp.wesley@uchealth.com.au and we will reply within one hour (during business hours) with appropriate specialist contact details, or call 07 3232700 and page the Director of Medical Services on-call.

To assist with your enquiry please provide the following patient details:

- Age
- Clinical background
- Presenting complaint

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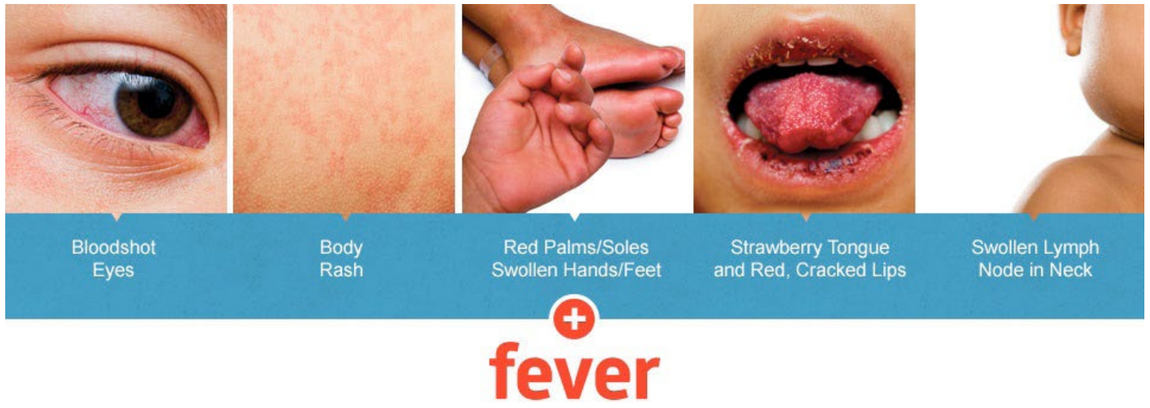
Kawasaki Disease

Dr Marion Thomas

Paediatrician

Kawasaki Disease is the commonest cause of acquired heart disease in children in Australia and the developed world with steadily increasing incidence.

Kawasaki Disease was first described in Japan by Tomisaku Kawasaki in 1967. The first Australian case was reported in 1974.



Prognosis in Kawasaki Disease relates to extent of cardiac involvement, with significant mortality and lifelong morbidity associated with inflammatory changes, fragility and aneurysm formation of the coronary arteries which occurs in up to 25 per cent of untreated cases. The pathogenesis is an acute multi-system vasculitis: its aetiology remains an enigma with a suspected interplay of host and environmental factors.

Increasing incidence of the disease and new therapeutic interventions which effectively reduce cardiac sequelae make awareness of potential cases of Kawasaki Disease imperative. Recent updated NICE Guidelines for Childhood Fever highlight this principle with consideration of Kawasaki's recommended in any child with persistent unexplained fever.

Diagnosis is made clinically based upon defined criteria:

+ Fever - classically greater than 5 days*

Plus four of the following:

+ Mucous membrane changes - dry fissured injected lips, strawberry tongue, pharyngeal injection
+ Extremity changes - erythema, oedema and desquamation of hands and feet
+ Conjunctivitis - bilateral and non-exudative
+ Skin rash - polymorphous erythematous
+ Cervical lymphadenopathy

(American Heart Association Criteria 2004)

(Japanese Circulation Society Criteria 2008 - *atypical or incomplete cases recognised with shorter duration of fever)

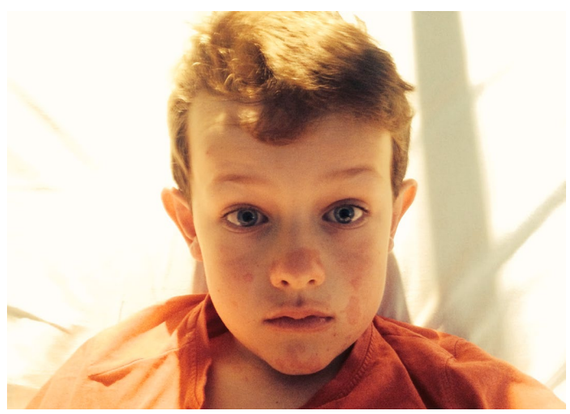
Other non-specific clinical features include extreme irritability with inconsolability, aseptic meningitis, vomiting and diarrhoea, abdominal pain, uveitis, gallbladder hydrops, urethritis, non-specific cough and development of induration at BCG scar site.

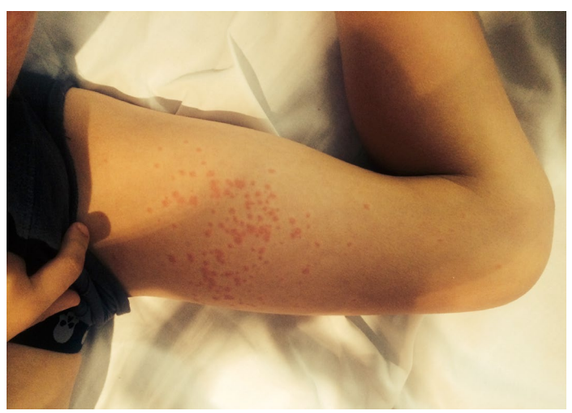
Laboratory findings are supplementary to the diagnosis with typically thrombocytosis, hypoalbuminaemia, normochromic normocytic anaemia, elevated alanine aminotransferase, neutrophilia, elevated ESR and CRP.

The median age of onset is 2.3 years with 80 per cent of cases occurring between six months and two years. Cases rarely occur in neonates and adults, presenting significant diagnostic challenges. There are multiple differential diagnoses for consideration including streptococcal and staphylococcal infection, juvenile idiopathic arthritis, a drug adverse reaction and it can be difficult to distinguish from a myriad of common febrile childhood illnesses. There is frequently coexisting infectious illness in up to a third of children, and cases may be unrecognised or diagnosed late in the disease when treatment is of limited benefit.

Kawasaki Disease was first described in Japan by Tomisaku Kawasaki in 1967. The first Australian case was reported in 1974. Surveillance demonstrates an incidence of approximately 50 to 60 cases per year in Australia (4 per 100, 000 <5years age) compared to 230 per 100, 000 in Japan. This marked variation in incidence in different ethnic groups, maintenance of rates of genetic susceptibility in migratory populations and sibling concordance all suggest a genetic aetiology. However seasonal variation patterns suggest a link to an environmental infectious agent and several other environmental hypotheses have been suggested as a potential trigger of a cascade of autoimmune mediated inflammation in a genetically susceptible child.

The cardiac complications in Kawasaki disease are due to blood vessel intimal inflammation and secondary coronary artery dilatation, with peak onset at day 10 of the fevers with progression coinciding with persistence of fever. Sudden death can occur due to thrombosis or myocardial ischaemia. If untreated 25 per cent of cases progress to aneurysm formation with associated lifelong morbidity.





Typical skin rash, above and right, associated with Kawasaki disease. Photos courtesy of Kawasaki Disease Foundation Australian kdfoundation.org.au

Treatment

The mainstay of current treatment for Kawasaki disease is intravenous immunoglobulin (IVIG), which has demonstrated efficacy, reducing coronary artery complications tenfold from 25 per cent to 2.5 per cent. It has greater impact given early - preferably prior to day 10 of the illness. There is a rapid response in 80 per cent of cases and a smaller proportion is responsive to a second dose. The mechanism of action is anti-inflammatory. Aspirin is the oldest treatment used historically; there is no proven cardioprotective action. Administration is at a high anti-inflammatory dose (80-100 mg/kg/day) initially followed in medium to long-term at a lower anti-platelet dose (3-5 mg/kg/day) after two days of cessation of fever. Use of steroids is controversial in Kawasaki disease, with uncertainty of efficacy and dosing despite long-term anecdotal use. Practically steroids currently have their main use in cases refractory to two doses of IVIG in the form of pulse methylprednisolone.

A recent advance in treatment for Kawasaki's is Infliximab, a biologic monoclonal antibody, which is a specific antagonist of tumour necrosis factor alpha. Early data for Infliximab suggests a very promising role in treatment for IVIG and steroid refractory cases. Long term sequelae of use are not clearly defined with prospective studies in progress.

Future challenges for Kawasaki's disease are the aims of increased general awareness and early clinical recognition, allowing prompt initiation of intervention and prevention of cardiac sequelae. Treatment options are expanding, collection of surveillance data and the long term follow-up of affected children into adulthood will provide more knowledge of the aetiology and natural history of this important and enigmatic condition.

For more information on the disease, go to kdfoundation.org.au

Dr Marion Thomas is a paediatrician with more than 22 years experience, both in the UK and Australia. For contact details, see previous page.

Acute rhinosinusitis

Definition, Aetiology, Management

Dr Brian Wilson-Boyd

Ear, Nose and Throat (ENT) Surgeon

Acute rhinosinusitis is a common presentation in the winter months. It is mostly a viral illness requiring supportive care for resolution. Acute bacterial rhinosinusitis is less frequent and may require more aggressive management with a low incidence of complication. Acute bacterial sinusitis is often predisposed by anatomical obstruction to the sinus ostia.

Definition

Rhinosinusitis in adults is defined as inflammation characterized by two or more symptoms and either endoscopic signs or radiological changes. One of the symptoms should be either nasal obstruction or nasal discharge with either facial pain or a reduction of loss of smell. Endoscopic signs are polyposis, muco-purulent discharge or mucosal oedema. CT evidence is reflected in mucosal changes of the sinus ostia or paranasal sinuses.

Aetiology

Acute rhinosinusitis (ARS) comprises viral ARS (common cold) and post-viral ARS. A small percentage of the patients with post-viral ARS will have bacterial ARS. Acute viral rhinosinusitis is defined as a duration of symptoms less than 10 days. Acute post-viral rhinosinusitis is defined as increase of symptoms after five days or persistent symptoms after 10 days.

Acute bacterial sinusitis is suggested by three symptoms or signs of discolored discharge (with unilateral predominance), severe local pain (with unilateral predominance) fever (>38ºC), elevated ESR/CRP, 'Double sickening' (i.e. a deterioration after an initial milder phase of illness.

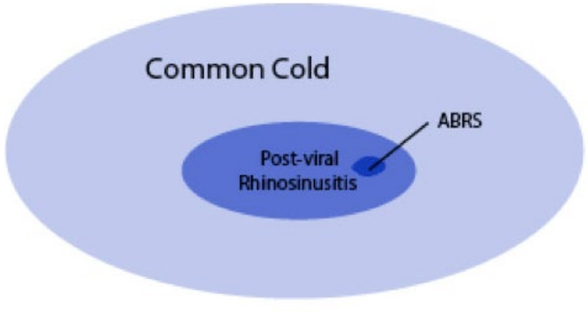
The primary cause are viruses with only 0.5-2.0% of patients developing acute bacterial rhinosinusitis secondary to a viral infection. Allergic inflammation and cigarette smoke exposure predispose patients to ARS possibly via changes to ciliary motility and function. Acute bacterial pathogens are S. pneumoniae, H. influenzae, S. pyrogenes, M. catarrhalis, and S aureus.

Anatomical obstruction to sinuses is a significant factor in the aetiology of ARS. Anatomical factors associated with ARS are Haller cells (infraorbital cells), concha bullosa (pneumatized middle turbinate), septal deviation, choanal atresia, nasal polyps and hypoplasia of sinuses. These may require surgical correction to prevent recurrent acute rhinosinusitis.

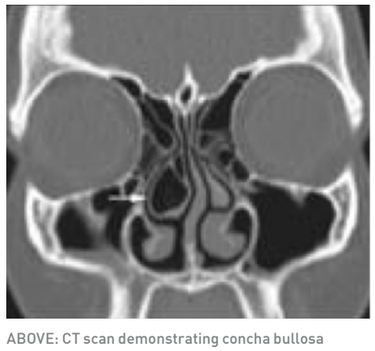
Complications are rare but often early in the course of the illness with severe symptoms. Antibiotic treatment does not prevent complications of which there are three main categories, orbital, intracranial, and bony.

Warning symptoms of complications are:

+ Periorbital oedema/erythema
+ Displaced globe
+ Double vision
+ Ophthalmoplegia
+ Reduced visual acuity
+ Severe unilateral or bilateral frontal headache



ABOVE: Acute rhinosinusitis can be divided into Common Cold and post-viral rhinosinusitis. A small subgroup of the post-viral rhinosinusitis is caused by bacteria.



ABOVE: CT scan demonstrating concha bullosa (arrow), Haller cells and Septal deviation.

+ Frontal swelling
+ Signs of meningitis
+ Neurological signs
+ Reduced consciousness

Management

ARS resolves without antibiotic treatment in most cases. Antibiotics are indicated for high fever or severe (unilateral) facial pain. It is estimated that about 80% of cases of mild ARS respond to amoxicillin and the remainder to Amoxicillin/Clavulanic acid. Intranasal corticosteroids are effective bd and oral corticosteroids can be used in severe ARS.

Nasal decongestants, nasal washouts or irrigations, NSAIDs, aspirin, paracetamol, Probiotics (10 RCT reduction in frequency and severity of symptoms) and Zinc shorten symptoms of the common cold.

Antihistamines have a marginal effect in reducing symptoms of runny nose and sneezing at two days in viral rhinosinusitis. There is no indication for the use of antihistamines in the treatment of post-viral ARS.

Steam inhalation has not shown any consistent benefits in the treatment of the common cold.

Echinacea: There are 10 RCTs performed on the efficacy on Echinacea of which five found that Echinacea significantly reduced overall symptom score compared with placebo and five RCTs found no significant difference between groups.

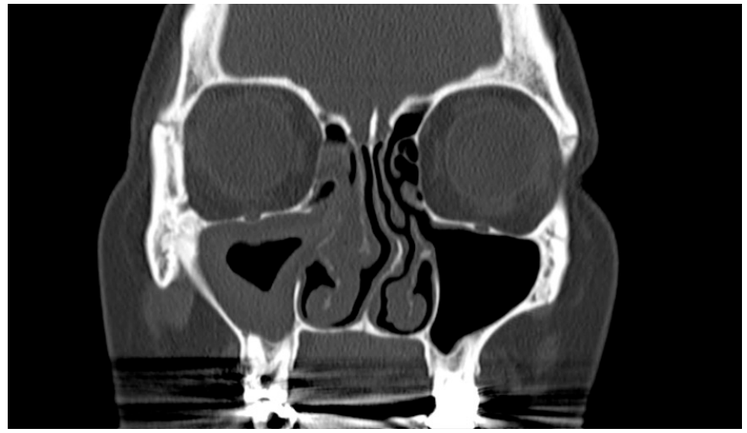
Summary

Acute rhinosinusitis is common, only 2% are bacterial and associated with more severe, unilateral symptoms and fever. Complications are rare and associated with severe early symptoms. Topical treatments are beneficial including corticosteroid, analgesia and selective antibiotic.

Reference

European Position Paper on Rhinosinusitis and Nasal Polyps 2012. Rhinology supplement 23: 1-298, 2012

Case Study



KW is a 54-year-old woman with recurring acute right maxillary sinusitis.

KW presented with a four-week history of acute sinusitis, with right maxillary and dental pain, nasal obstruction, yellow rhinorrhea and fever. Treatment included five courses of antibiotic and a decongestant spray. There is a history of recurrent sinusitis once or twice a year, always on the right hand side. KW is a non-smoker.

CT scan demonstrated septal deviation to the left, with right maxillary mucosal oedema and occluded maxillary infundibulum. There was a compensatory right middle turbinate concha bullosa (pneumatised middle turbinate) causing maxillary infundibulum obstruction.

KW's acute treatment included a 50 mg daily dose of prednisone (0.5 – 1 mg / kg) for seven days with a 10-day course of Augmentin duo forte. Topical treatment included five days of bd nasal decongestant, twice daily nasal inhaled steroid and bd saline irrigation.

KW's acute symptoms resolved with this treatment with no need for antral washout or acute sinus surgery. She presented electively for right-sided endoscopic sinus surgery to the maxillary and ethmoidal sinuses and reduction of the lateral lamella of the pneumatised right middle turbinate. As there was no history of left acute sinusitis no surgery was performed on this side despite the significant septal spur to the left. Following maxillary antrostomy a peanut-coloured mucosal coating was evident on the maxillary mucosa with fungal hyphae suggestive of allergic fungal sinusitis. Microscopy grew Aspergillus fumigatus with no bacteria seen on microscopy.

KW remained well post-operatively with no pain and good nasal airflow. She continued on nasal irrigation for six weeks post-operatively until the right maxillary and ethmoidal sinus mucosal completely healed and there was no residual debris in the nasal passage or sinuses. There was no going mucosal inflammation to suggest a diagnosis of allergic fungal sinusitis. However, the Aspergillus will have predisposed KW to recurrent acute sinusitis as did her anatomical predisposition. ■

Dr Brian Wilson-Boyd is an Ear, Nose and Throat (ENT) Surgeon specialising in Otolaryngology and Head and Neck Surgery. He consults at the Wesley Private Hospital, North West Private Hospital, Bywater Medical Centre – Oxley and Cadogan House – Nundah.
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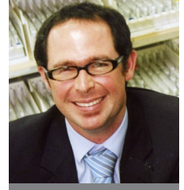
New approach to testing for inflammatory bowel disease



Dr Daman Langguth

Clinical Immunologist

Since its introduction in 2010 at Sullivan Nicolaides Pathology, faecal calprotectin has been proving itself a useful non-invasive test able to identify those patients most likely to need endoscopy for suspected inflammatory bowel disease (IBD). It may help identify those patients who need endoscopy, those who are likely to have an underlying gastrointestinal pathology, and help in monitoring inflammatory bowel disease.

For the past two years, the Immunology Laboratory at Sullivan Nicolaides Pathology has offered faecal calprotectin testing with next day results, five days a week. It requires only a stool sample from the patient.

Faecal Calprotectin helps discriminate between symptoms of inflammatory bowel disease and irritable bowel syndrome, and may help rule out inflammatory disease.

Calprotectin is a calcium-binding protein in the cytosol of neutrophils with anti-microbial activity. High levels reflect intestinal inflammation with increased numbers of neutrophils in the bowel wall. It provides more objective assessment of inflammation than faecal microscopy and bypasses the difficulty of identifying stool leukocytes. Calprotectin performs better as a marker of bowel inflammation than standard systemic markers such as CRP and ESR.

One meta-analysis suggested sensitivity of 95%, specificity 91% overall. A normal calprotectin, in the absence of 'red flag' symptoms, and in the context of positive Rome criteria carries a high likelihood of absence of a non-organic diagnosis in subsequent investigation. However, a small number of patients with organic disease may be missed and a small number with IBS (no detectable inflammation by other means) may have mildly raised levels due to significant daily variation.

The cut-off chosen may vary depending on the situation. Its discriminative power appears less in children and a higher threshold (200-300 ug/g) has been suggested as having improved diagnostic efficiency (Henderson, 2012).

Faecal calprotectin testing at Sullivan Nicolaides Pathology's Immunology Department using a Phadia ImmunoCAP 250 immunoassay analyser

Calprotectin is non-specific for G.I. inflammation of any cause, so may also be raised in other pathologies (e.g. NSAID-induced, bacterial/viral infection, microscopic colitis, diverticulitis, untreated celiac disease or colonic malignancy). While levels are commonly elevated in patients with symptomatic colon cancer (median level about 350 ug/g), its low specificity hinders its use as a marker for colon cancer screening.

In patients with inflammatory bowel disease calprotectin levels correlate well with clinical activity and with definitive tests such as colonoscopy and mucosal biopsy. Levels may normalise with mucosal healing and it has been used as a surrogate marker of remission in patients treated with anti-inflammatory agents. For IBD patients in clinical remission faecal calprotectin can predict the likelihood of relapse over the following year (sensitivity about 78% and specificity 73% in one meta-analysis).

It appears to be a better marker of disease activity for ulcerative colitis than for Crohn's disease, however it has not been found useful for discriminating between the two and its ability to predict relapse is similar (Smith, 2012).

Spot faecal samples appear as reliable as 24-hour samples, as calprotectin is evenly distributed through the faeces. However, clinical use can be hampered by difficulties in extraction from a stool sample (avoiding particulate matter such as seeds and fibre) and by problems with international standardisation. With high-volume diarrhoea, a single sample may underestimate the degree of bowel inflammation. Recent changes in testing methodology have improved the ability to gain the correct results in all stool types.

Case study

Mary is a 25-year old woman with intermittent bloating, diarrhoea and on occasions, abdominal pain. She sometimes passes stool several times per day. There has been no weight loss and she has no other medical problems. She doesn't take non-steroidal anti-inflammatory drugs (NSAIDs) and apart from the oral contraceptive pill is on no regular medication.

This is a frequent clinical problem and is most likely to reflect Irritable Bowel Syndrome though rarely in cases like this where there remains a possibility of underlying inflammatory bowel disease or GIT pathology, it may be difficult to

1) reassure the patient, and
2) stratify who can be observed to need early investigation.

Case Results

Mary returned a normal faecal calprotectin and, given she had been appraised as low risk using the Rome criteria for IBS, a colonoscopy was avoided.

Information for clinicians

http://www.snp.com.au/media/330449/guide_to_inflammatory_bowel_disease.pdf

Specimen collection guide

http://www.snp.com.au/media/255882/specimen_collection_guide.pdf

Patient information

http://www.snp.com.au/media/330452/new_test_for_crohns_disease_and_ulcerative_colitis.pdf ■

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Van Rheenen PF, et al.
Faecal calprotectin for screening of patients with suspected inflammatory bowel disease: diagnostic meta-analysis. BMJ 2010; 341: 3369 (doi: 10.1136/bmj.c3369)

Henderson P, et al.
The diagnostic accuracy of fecal calprotectin during the investigation of suspected pediatric inflammatory bowel disease. Am. J. Gastroenterol. 2012; 107: 941-9

http://www.romecriteria.org/assets/pdf/19_RomeIII_apA_885-898.pdf

Dr Daman Langguth is an immunologist and Head of the Immunology Department at Sullivan Nicolaides Pathology, Brisbane. He is also a clinical immunologist visiting the Wesley Hospital. He has particular expertise in the investigation of auto-immune disease, allergy, and immune deficiency and has special interests in idiopathic inflammatory myositis, Vasculitis, IgG4-related systemic disease and e-Health.
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Next generation genetics for the next generation

Next Generation Sequencing is the latest genetic testing technology on offer for clinicians, but there is reason to be cautious.

In approximately 80% to 90% of clinically diagnosed complex genetic diseases the causative gene variation is not found by single gene testing. This is due to the fact that the classical sequencing of gene by gene is expensive and time consuming. The last five years has seen an explosion of novel genetic testing technologies that have transitions from the bench side as research tools to the bedside as clinical tools. The most notable example has been the evolution of Next Generation Sequencing (NGS).

NGS is in essence a massive parallel gene sequencing technique, which means that millions of small fragments of DNA can be sequenced simultaneously. Previous methods could sequence one DNA fragment at a time i.e. examining one gene at a time which is both expensive in time and financial considerations.

NGS allows the sequencing of massive amounts of genetic material from the one blood sample, allowing the time and cost effective collection of a significant amount to genetic information. Genetic laboratories are in the early phases of introducing NGS into clinical practise; however the use of this technology requires careful consideration.

Currently the NGS technologies can be tailored towards:

a) "diagnostic gene panels" i.e. known genes associated with a specific clinical phenotype, or

b) Genome Wide Exome Sequencing (GWES) i.e. the entire coding DNA is sequenced.

NGS in healthcare – Diagnostic Gene Panels

Diagnostic Gene Panels are an exceptionally powerful clinical tool for a tightly defined clinical phenotype in which there is genetic heterogeneity i.e. multiple genes associated with the clinical phenotype e.g. there are over 130 genes currently known to be associated with an infantile encephalopathy phenotype. NGS testing of these known genes as part of a "diagnostic panel" offers an attractive diagnostic tool in terms of financial cost, comprehensiveness of genes screened, and turn-around time.

Distinct clinical advantages for the utilisation of a diagnostic panel in an appropriate patient include:

a) secure an accurate and confirmed clinical/molecular diagnosis
   + institute early therapeutic intervention where possible
   + provide the family/patient an understanding of the disease, natural history and prognosis

b) to offer a targeted examination of other at-risk family members i.e. cascade testing.

Some examples of clinically useful diagnostic gene panels being offered by a range of service providers include:

a) Hereditary cancer syndromes
   + Breast cancer
   + Colon cancer and polyposis syndromes (Lynch syndrome)
   + Prostate cancer
   + Endocrine tumours

b) Epilepsy syndromes and migraines

c) Sensorineural Hearing Loss (SNHL)
   + nonsyndromic SNHL
   + autosomal recessive SNHL
   + autosomal dominant SNHL
   + X-linked SNHL

d) Congenital brain malformations

e) Neuromuscular diseases (e.g. Charcot-Marie-Tooth disease)

f) Ion channel diseases (e.g. Cardiac arrhythmias, Myotonia congenita)

g) Connective tissue diseases (e.g. Ehlers-Danlos Syndrome)

h) Genetic ocular diseases (e.g. Retinitis pigmentosa)

i) Cardiac disease (e.g. Dilated and hypertrophic cardiomyopathy)

j) Inborn errors of metabolism (e.g. Mitochondrial diseases, Lysosomal storages diseases)

k) Dysmorphic syndromes with a known molecular/genetic basis

NGS in healthcare – GWES

Extending this NGS further, it is becoming possible to undertake whole exome genome analysis for select patients. In this type of analysis, it is possible for scientists to look at the entire genetic make-up of a patient. This type of testing is most often utilized in the research setting while looking for a causative gene. Careful patient selection and genetic counseling is imperative as GWES can provide unforeseen collateral information e.g. carrier status for diseases such as cystic fibrosis, or mutations in cancer predisposition genes. GWES is only a tool for Clinical Geneticists at this stage.

NGS challenges

The devil is in the detail of the bioinformatics analysis of these complex genetic tests. Cost is still a limiting factor for many families. A targeted NGS diagnostic panel may cost as little as $1000, while GWES can cost $3000 to $5000 in the current climate. The ethical utilisation of NGS technologies in "personalised genomics" and in assisted reproductive fertility streams of medicine are areas for ongoing caution and careful consideration. ■

Associate Professor David Coman

Metabolic Physician/Clinical Geneticist/General Paediatrician

Associate Professor David Coman is Medical Director of Paediatrics at The Wesley Hospital. He is involved in multiple research projects aimed at novel disease discovery, improved diagnostic testing and treatments for children with inherited genetic disorders and holds academic appointments at The University of Queensland, Griffith University and Bond University.
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Remarkable milestones

Congratulations to the Wesley's Cardiac Service on three remarkable milestones in 2014!

+ Cardiology 30 years
+ Cardiac Catheter Lab 25 years
+ Cardiac Surgery 21 years

Paediatric Coeliac Disease

A gluten vaccine is among the novel treatments being explored as an alternative to gluten-free diets for people with coeliac disease

Dr Richard Muir

Paediatric Gastroenterologist

Coeliac disease (CD) is an immune-mediated systemic disorder elicited by gluten and related prolamines in genetically susceptible individuals. Whilst an enteropathy with symptoms of malabsorption is the classic presentation, CD can have protean manifestations, reflecting its systemic nature.

CD affects approximately 1.25% of the Australian population, but only around 25% of patients with coeliac disease are currently diagnosed. Paediatric onset of CD can occur at any age, from about 12 months of age onwards. Recent population reviews have suggested that breastfeeding has a protective effect on CD risk, and early introduction of gluten (less than four months of age) increases CD risk.

The presentation of CD can be varied, from the most severe manifestation, coeliac crisis, through to relatively asymptomatic patients (up to be 80% in some populations). A significant proportion of patients (30%) will be diagnosed through familial screening, emphasizing its importance.

Apart from classical gastrointestinal symptoms of CD in the paediatric population - such as constipation, diarrhea, abdominal pain and failure to thrive - it is important to be aware of extra-intestinal manifestations. Up to 70% of CD patients will have oral involvement, either recurrent aphthous stomatitis or dental enamel defects- such as horizontal ridging or staining of permanent dentition.

Certain groups are at higher risk of CD and should be screened regularly. These include IDDM (10-15%), Down Syndrome (10%), and Williams and Turner Syndrome (up to 5%). Other high risk group which should be screened are first degree relatives of CD patients as up to 15% can be affected (10% sibling risk, 6-9% parental risk).

Appropriate screening includes anti-tTG IgA antibody (tTG-IgA), Deamidated Gliadin IgG (DGP-IgG), and total serum IgA. The sensitivity and specificity of the tTG-IgA antibody is around 95%. DGP-IgG has inferior sensitivity and specificity compared to tTG-IgA but has previously been thought to have some clinical utility in IgA deficient patients. A recent review suggested a sensitivity of around 81 per cent in IgA deficient patients and that an elevated DGP-IgG in the absence of an elevated tTG-IgA performs poorly as a predictor for coeliac disease. (1)

It should be remembered that these are sceening tests only. No serological marker has 100% Positive Predictive Value (PPV) for CD, so the caveat with their use, is that if the clinical suspicion of coeliac disease remains high, endoscopic assessment is the most appropriate investigation, even in the setting of normal coeliac serology. Anti gliadin-IgG or IgA serology (as opposed to anti-tTG IgA or DGP-IgG) should no longer be used in clinical practice. This includes in patients less than two years of age. There is no definitive evidence that they are superior to tTG or DGP in this age group.

In patients who are already on a gluten-free diet and the diagnosis of coeliac disease is entertained, an appropriate diagnostic algorithm would be to first assess DQ2/DQ8 haplotype. Given its high Negative Predictive Value (NPV), a negative haplotype excludes CD. A positive result dictates the need for a gluten challenge. An appropriate challenge is at least one gluten-containing meal per day (e.g two slices of bread, bowl of pasta, serve of cereal) for six weeks and then appropriate serological assessment. It is important to emphasise again, that if there is a high clinical suspicion of CD, endoscopic assessment is appropriate, even in the setting of normal coeliac serology.

Appropriate endoscopic assessment of the patient with suspected coeliac disease includes collecting at least six biopsies from the small bowel, including the duodenal bulb. Coeliac disease can have a patchy histological distribution so it's crucial that adequate sampling occurs. Up to 30% of patients can have other coexistent gastrointestinal diseases diagnosed at initial endoscopy.

Management

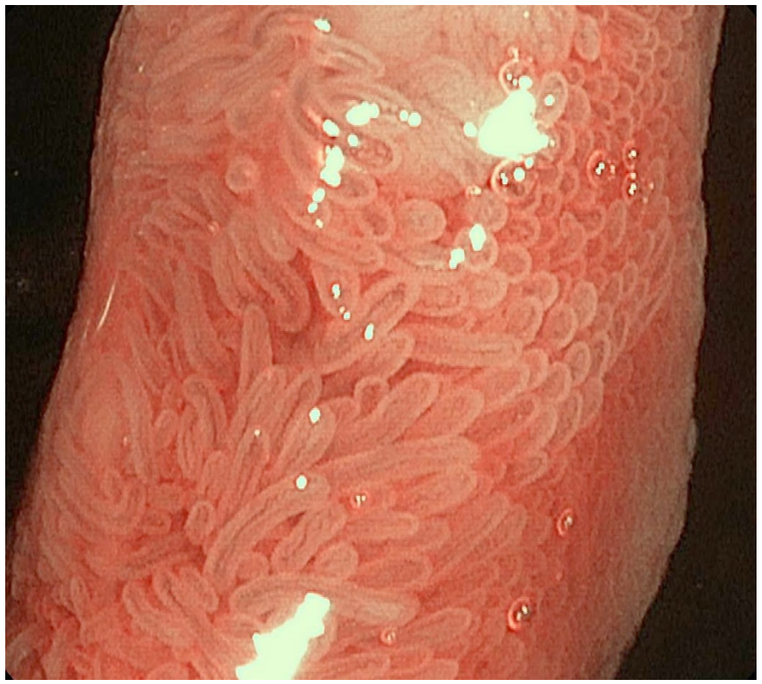
After diagnosis of CD, management should focus on appropriate education of the family, including dietetic review. Screening of first degree family members should also occur, including for DQ2/DQ8 haplotype, anti-tTG IgA, DGP-IgG, and total serum IgA. Siblings with a positive DQ2/DQ8 haplotype and negative coeliac serology, should have repeat serological screening every three years or if symptomatic. Those with negative haplotype do not need any further screening.

Parents should expect a clinical response in symptomatology within two to four weeks of commencement of a gluten-free diet. Patients may have a transient lactose intolerance due to villous atrophy which should resolve.

Bone Mineral Density (BMD) assessment through DEXA scanning is not routinely performed in paediatric patients as the BMD will normalize within one year on a GFD and be comparable to a peer without coeliac disease. The exception to this would be for a patient with a history of fractures despite incidental trauma. This response is maintained in the longer term on a GFD. Final adult height will not be affected in patients diagnosed before 10 years of age. Catch up growth after diagnosis is maximal in the first six months of commencing a GFD (Weight>Height).

Compliance with a GFD is monitored chiefly by anti-tTG IgA and secondarily DGP-IgG. Anti-tTG IgA should halve by six months and normalize within 12 months on a GFD. Failure of serological response to a GFD warrants repeat review of the patient's dietary intake with a dietitian, and repeat serology (anti-tTG IgA and DGP-IgG) in three months time. Repeat endoscopic assessment of patients with CD is not routinely performed in children unless there is failure of symptomatic response to a GFD or persistently abnormal serology after more than one year on a GFD. Complete mucosal healing can take up to two years to occur in children, so repeat endoscopy inside of a year on a GFD has little diagnostic utility.

> "Recent population reviews suggest breastfeeding has a protective effect on Coeliac Disease risk."



Intestinal cilia

Key predictors of long term compliance in paediatric patients with coeliac disease are:

+ younger age at diagnosis (<10 years of age)
+ if patients are symptomatic on gluten ingestion
+ patient and parent knowledge around a gluten-free diet and
+ regular follow up with their health practitioner and dietitian.

Membership of a dedicated patient support group has also been shown to increase long-term compliance with a GFD.

Although the current and only treatment for coeliac disease is a life-long adherence to a gluten-free diet, there are several other potential novel therapies in various stage of development around the world.

The gluten vaccine is based around the same principles of desensitization therapy for traditional allergic conditions. It is based on the view of peptide-based therapies promoting tolerance by inducing regulatory T Cells. Brisbane has been fortunate to have been involved in the first in human study with this drug and continues to be part of the international phase one trial being conducted by ImunsanT. The vaccine trial in Brisbane is still open to adult volunteers.

To make an appointment to be seen in the Coeliac Clinic located at St Andrews' War Memorial Hospital please phone 3636 1010 or fax 3367 1075. If you would like more information about the coeliac vaccine trial please phone 1300 774 276

For more information of the diagnosis, management and follow up of paediatric coeliac disease, the recent ESPHAGN guidelines are a useful reference guide. ■

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Dr Richard Muir is a Paediatric Gastroenterologist and began practicing at the Wesley Hospital in 2012. He has a special interest in Inflammatory Bowel Disease, Eosinophillic Oesophagitis and Coeliac Disease.Dr Muir was the recipient of a Royal Children's Hospital Foundation Clinical Fellowship Grant. He practices at the Paddington Medical Centre. Telephone: 33671065

Pancreatic Cystic Lesions

Pancreatic cysts are being detected with increasing frequency over recent years as a result of a massive growth in cross-sectional imaging studies such as CT and MRI.

Often, the pancreatic lesion is incidental, and therefore termed a "victim of medical imaging technology" (VOMIT). Nonetheless, certain cystic lesions, even asymptomatic, do carry malignant potential, and therefore cannot be ignored. True pancreatic cystic neoplasms account for less than 10 per cent of all pancreatic neoplasms.

Most cystic lesions are asymptomatic and found incidentally, but can cause or result from pancreatitis, or if malignant, present with jaundice and weight loss.

Generally, cystic pancreatic lesions can be divided into those with no malignant potential (pseudocysts and serous cystadenomas) or those that are pre-cancerous - mucinous cystic neoplasms (MCN) and intraductal papillary mucinous neoplasms (IPMN). Any solid tumour whether benign or malignant can also have a cystic component, or undergo cystic degeneration, but the scope of this short article is limited to true cystic lesions.

Despite advances in imaging technology, it remains difficult to make the distinction between mucinous and non-mucinous lesions on their radiologic characteristics alone. Endoscopic Ultrasound (EUS) has emerged over the last 10 years as the mainstay in assessment of lesions within the pancreas, both solid and cystic. EUS provides better detection of one or more lesions (up to 40% of solid lesions <2cm in size can be missed on CT scans), clarify communication with the main pancreatic duct and assess the presence of associated masses or mural nodules. In addition, it allows sampling for both cyst fluid biochemical analysis as well as for cytology. Whilst it remains an imperfect test, cyst fluid CEA (carcino-embryonic antigen) levels >192 ng/ml has a 79% accuracy to identify mucinous, pre-malignant lesions.

Pseudocysts

Pseudocysts are the most common non-mucinous lesions – often easy to identify clinically given an antecedent history of pancreatitis. These lesions have low CEA and markedly elevated amylase levels, and resolve spontaneously in a large proportion of cases. If symptomatic, they can be drained endoscopically into the stomach or duodenum under EUS guidance.

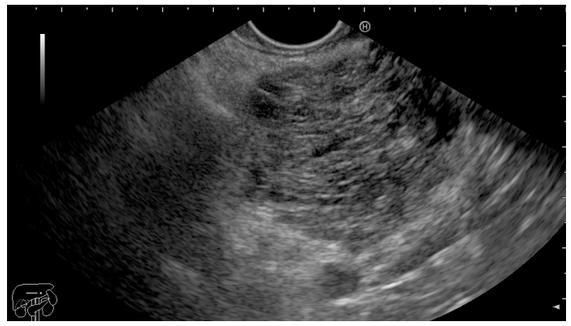


Figure 1. Classical microcystic (honeycomb) appearance of a serous cystadenoma.

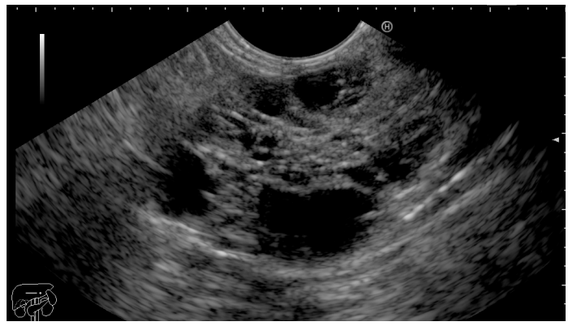


Figure 2. Variant of micro- and macro-cystic serous cystadenoma

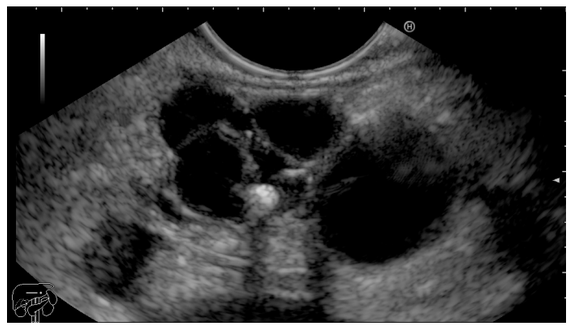


Figure 3. Oligocystic variant of serous cystadenoma. Note the central calcification.

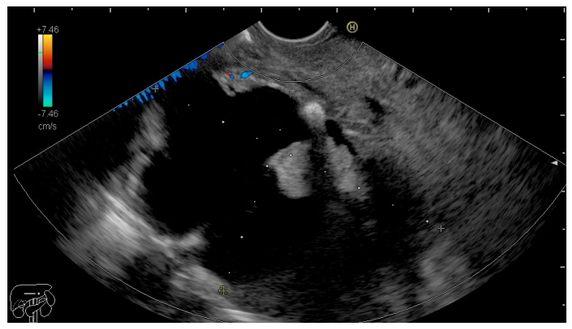


Figure 4. Mucinous cystic neoplasm. Note the eccentric calcification and internal (mural) mass.

Serous cystadenomas

Accounting for around 30% of cystic neoplasms, these generally occur in middle-aged and older females. They classically have a honeycomb or microcystic appearance at EUS with or without central calcification. These multiple septations can be confused as a solid lesion on cross-sectional imaging. They have a glycogen-rich cuboidal cell lining, and are often quite vascular. Biochemical analysis of fluid shows low CEA and amylase levels.

Mucinous cystic neoplasms

These lesions carry the highest malignant potential, and occur almost universally in women. They account for 40% of pancreatic cystic neoplasms, and have a columnar, mucous producing lining supported by ovarian stroma. Cyst fluid analysis shows elevated CEA but low amylase levels. Once diagnosed or suspected, in suitable patients surgical resection is the treatment of choice.

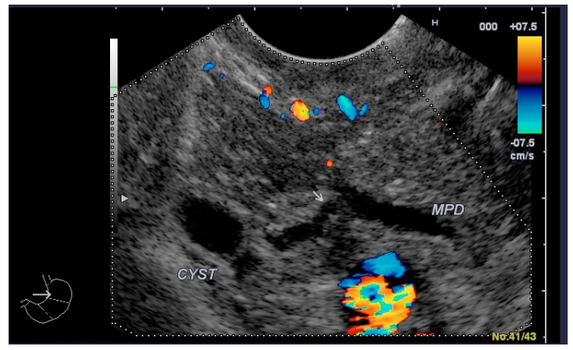


Figure 5. Branch duct IPMN. Note the clear communication with main duct (arrow).

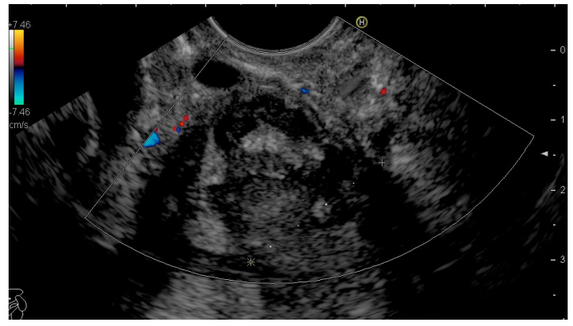


Figure 6. Malignant main-duct type IPMN. Callipers marks out diameter of main pancreatic duct

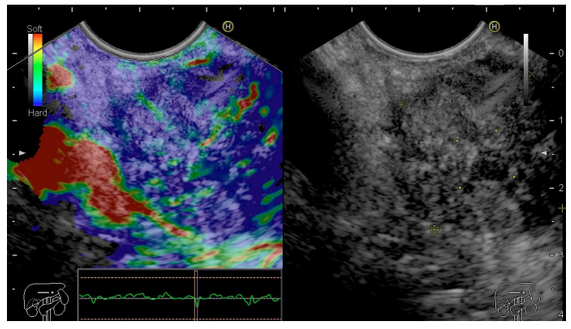


Figure 7. Mass associated with main-duct type IPMN. The blue colour represents a discrete hard lesion indicative of malignancy

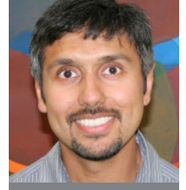
	Pseudocyst	Serous Cystadenoma	IPMN	Mucinous Cystic Neoplasm
CEA	Low	Low	High	High
AMYLASE	High	Low	High	Low
CYTOLOGY	Inflammatory cells	Cuboidal cells	4 histologic subtypes	Columnar cells & ovarian stroma

Table 1. Fine needle aspirate fluid analysis characteristics of cystic pancreatic lesions

IPMN (Intraductal papillary mucinous neoplasm)

These can be divided into main duct or branch duct types, and occasionally a mixed form. The lifetime cancer risk is over 50% in main duct types, and 15-25% for branch duct variants. International consensus guidelines have been recently revised for assessment and surveillance of these lesions. Branch duct IPMN are classified as being "high risk", displaying "worrisome features" or "low risk". This has implications on surveillance strategies and intervals, as well as identification of those who should be considered for surgery. Biochemical analysis shows high CEA and amylase (as they by definition arise from either main pancreatic duct or its side branches).

In conclusion, cystic lesions are uncommon, but found with increasing frequency and often in asymptomatic individuals. Some do have malignant potential, but all universally result in anxiety for both the doctor and the patient. Endoscopic Ultrasound (EUS) is presently the best way to clarify the nature of these lesions and stratify the risk to the individual. Not all are VOMIT! ■



Dr Saurabh Gupta

Gastroenterologist and Therapeutic Endoscopist

Dr Saurabh Gupta is a Gastroenterologist specialising in advanced diagnostic and therapeutic endoscopic procedures. Dr Gupta also has a special interest in Upper GI endoscopy and colonoscopy, endoscopic ultrasound, ERCP, endoscopic mucosal resection of advanced neoplasia and enteroscopy. He is located on the Wesley campus.
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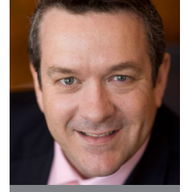
Post-operative complications in patients with obstructive sleep apnoea

Since the mid-1990's the medical literature has reflected a growing awareness of the risks of post-operative pain medications for patients with obstructive sleep apnoea (OSA). In patients with OSA, the airway completely or partially occludes during sleep despite respiratory effort. Arousal from sleep temporarily reopens the upper airway, but falling back asleep may close it and start the process of airway closure and arousal again. This process can produce significant hypoxemia, but the dangers are greatly increased when the patient is sedated by pain medications.



Dr Andrew Scott

Thoracic and Sleep Physician

Pain medication in a post-operative patient is meant to reduce pain perception, but will also result in sedation. For a normal patient the sedation may be a welcome relief from the pain and an opportunity to sleep. In a patient with OSA the sedation further reduces pharyngeal muscle tone, The risk is that there will be reduced pharyngeal tone coupled with a reduced response to low O2 and high CO2 leading to increased airway obstruction. If not controlled appropriately there can be frequent airway collapse with further worsening OSA, recurrent severe hypoxia, which increases cardiorespiratory complications.

In a study, published in Chest, a significant number of patients were observed to proceed to surgery while at risk of undiagnosed OSA. Why is this a problem? The study reported that in a random sample of 1759 patients, 16% had OSA.

Those at intermediate/high risk of cardiorespiratory complications had the following predictors:

+ Patients that were older: mean age of 55.9 years
+ Predominantly men
+ Higher BMI (38.3 vs 33 without OSA)
+ Higher ASA class
+ Higher level of co-morbidities (COPD/ lung disease, hypertension, coronary artery disease, diabetes)

The following complications were noted:

+ Postoperative hypoxemia (>10% desat) (Odds Ratio OR x7.9)
+ ICU transfer due to RF > 35 (OR x4.4)
+ Longer length of stay (OR x1.7)

Of these patients: 57% required CPAP therapy for stabilisation.

Other complications that were noted:

+ Myocardial ischemia and infarction, atrial fibrillation
+ Stroke, mental confusion, delirium
+ Wound breakdown
+ Reintubation,and ICU admission.

Keys to avoiding such outcomes:

+ Awareness on the part of the physicians and post-operative care providers that the patient suffers from OSA
+ Reducing systemic opioids as much as possible. This can include use of regional analgesia; neuroaxial analgesia; NSAIDs
+ Observe patients in recovery for longer postoperative period— 3 hours has been suggested
+ Place them on continuous pulse oximetry postoperatively, and provide continuous supplemental oxygen after surgery, even if not hypoxemic
+ Place patient in the lateral (side) position, not supine
+ Start CPAP if patients were on it at home; if they weren't, consider starting it in the hospital.

For surgery patients who are admitted with a diagnosis of OSA, the standards for post-operative treatment are relatively clear: careful attention must be paid to the pain medications being used and there must be monitoring of the oxygen level. If the patient uses a CPAP machine, the machines should be made available, and be used, if considered appropriate in the post-operative period. ■

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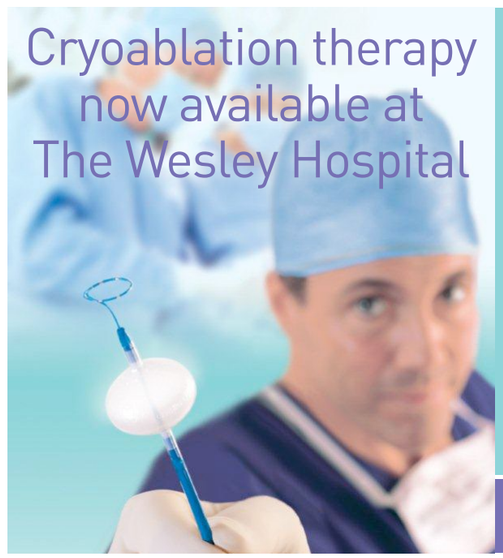
Wesley Pain and Spine Centre

The Wesley Hospital launched the Wesley Pain and Spine Centre in January 2014. The Centre replaces the Wesley's Pain Management and Back Rehabilitation programs and offers a more comprehensive range of services.

The Wesley Pain and Spine Centre's services include:

+ Specialist pain medicine physician review
+ Multidisciplinary rehabilitation (occupational therapy, physiotherapy, psychology, exercise physiology, nutrition and nursing care)
+ Interventional procedures
+ Referral for surgical review
+ Referral for psychiatric review
+ Referral for addition physician review
+ Medical imaging where appropriate
+ Individual allied health management

To make a referral, please call 3232 6190 or fax 3232 6189 or email twh.rehabcentre@uchealth.com.au



Cryoablation therapy now available at The Wesley Hospital

Cryoablation is a new method being used for the treatment of Paroxysmal Atrial Fibrillation (PAF).

Unlike radiotherapy techniques, cryoablation uses refrigerant instead of heat to isolate the pulmonary vein and freeze tissue to terminate unwanted electrical pathways.

It has similar efficacy in the treatment of PAF as radiofrequency catheter ablation with a cure rate of 70%.

Advantages of cryoablation over radiofrequency catheter ablation:

+ Performed in less time that RF ablation as pulmonary veins can be isolated with a single balloon application
+ A light sedation can be used for patients with high anaesthetic risk
+ Patients tend to make a quicker recovery

Appointments are available from Dr Vince Deen Telephone 3876 8285

Dr Deen will be speaking on Cryoablation at the Cardiology CPD on October 2. To register email Wesley.bdm@uchealth.com.au

Eosinophilic oesophagitis

Clinical, endoscopic histological features and treatment

Dr Matthew Remedios

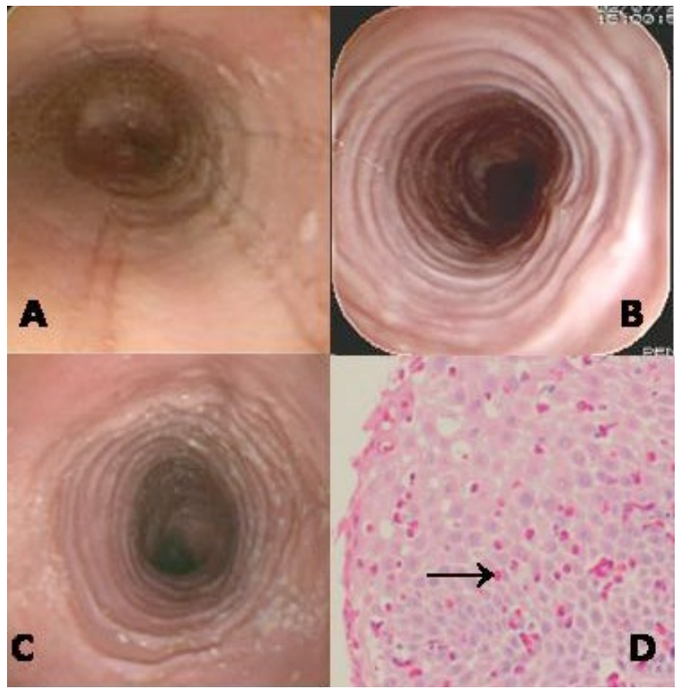
Gastroenterologist

In the last decade there has been appreciation of a new oesophageal condition, eosinophilic oesophagitis (EE): defined by histology of >15 eosinophils/high power field (HPF) isolated to the oesophageal mucosae associated with upper gastrointestinal symptoms where primary gastro-oesophageal reflux is excluded.

It is recognised in most countries with a male predominance of 3:1. Familial trends have been reported. Adults often present before age 40. There is an increasing prevalence and recognition. The rise in EE has paralleled atopy supporting the "hygiene hypothesis". Affected individuals may have asthma (often childhood), allergic rhinitis, atopic dermatitis and various food or drug allergies. Seasonal variation is described correlating with seasonal allergens. Multiple food sensitivities may be present with true food-induced anaphylaxis between 9-24%.

Diagnosis is based on symptoms, endoscopic features and histology (secondary causes of eosinophilic infiltration excluded). Symptoms in children include heartburn, abdominal pain, vomiting, isolated nausea, refusal to feed and failure to thrive. Adults often present with heartburn (despite acid suppression), chest pain, dysphagia and food impaction. Half of adults with food bolus obstruction will have EE; a third of untreated patients with EE may experience food bolus impaction requiring endoscopic removal. Symptom severity is variable - from asymptomatic to debilitating.

Patients have characteristic endoscopic findings (Fig. 1). Histology is essential for diagnosis. Proximal and distal oesophageal biopsies are required. A diagnostic mean threshold is ≥ 15 eosinophils/HPF; the peak density is often higher. The eosinophil is absent from the normal oesophagus and should be <5 eosinophils/HPF distally with absence proximally in GORD.



Patients have characteristic endoscopic findings (Fig. 1) including linear furrows (A), multiple rings (B), eosinophilic abscesses (C) and a narrow caliber fibrotic oesophagus. Histology is essential for diagnosis (D).

Therapy

Therapy is required for the symptomatic patient. EE is a chronic disease like asthma and requires a multi-pronged strategy of dietary manipulation and topical steroids +/- acid suppression. Long-term efficacy and modification of the natural history with treatment has not been yet demonstrated. Therapy can be:

1. Non-pharmacological (elimination/ elemental diet) and dilatation.
2. Pharmacological

Dietary therapy is appealing as a simple non-pharmacological strategy. Skin prick and patch testing can help identify culprit foods but are not essential for the majority of EE patients as predictive value is limited. A simple dietary modification in adult clinical practice is that patients modify their diet using smaller food boluses, chewing well and washing through with adequate fluid (particularly in men).

An elimination diet is useful given the presumptive role of ingested allergens in EE. This is most successful in the paediatric population. In a study from Philadelphia 98% of 381 children with EE responded to dietary intervention. Restrictive diets in adults are less well tolerated. Specialty team supervision in paediatric EE is essential to avoid malnutrition, food aversion and psychological support. An alternative is the "six food elimination diet" – exclude the six foods most often implicated in allergy (cows milk protein, wheat, soy, egg, seafood, peanuts). This can be expanded to corn, an increasingly ubiquitous food allergen. In a series of 146 children on a directed elimination diet 77% experienced oesophageal eosinophilic infiltrate resolution; allergen reintroduction was associated with relapse. In a trial of six food elimination in 33 adults there was a 94% symptomatic and 78% histological response. Six food elimination has been compared with elemental dietary management in one study of 60 children: elimination diet was cheaper, better tolerated and equally efficacious.

"Eosinophilic oesophagitis (EE) is a cause of dysphagia, chest pain and food bolus obstruction"

Elemental diets are poorly tolerated in adults but useful in the difficult paediatric patient. The basis is to minimise food antigens via synthetic amino-acid-based formulations. There is a cost, complexity of administration and a feeding tube may be required. A study in 51 EE children documented reduced vomiting, abdominal pain and dysphagia in the majority with histological improvement using this approach. There was a similar result in a larger study where oesophageal histology normalised in 158 of 160 paediatric patients.

Endoscopic dilatation was routine but has fallen out of favour in EE due to resultant long tears, post-dilatation pain and occasional perforation. It was previously employed when ignorant of EE, presuming strictures were peptic. We reserve cautious dilatation for the minority: tight symptomatic strictures resistant to topical therapy.

Adult patients may present following an inadequate response to GORD therapy. There is a clear subset of patients with overlap of EE and reflux disease. These patients need a combination of proton pump inhibitors and topical steroid therapy.

Corticosteroids

Eosinophilic gastrointestinal disease is responsive to corticosteroids. Symptom recurrence after ceasing steroids is common (50% at 12 months); there have been no long-term studies beyond 18 months. Systemic and topical preparations have been used with success in children and adults. The long-term side effect profile of systemic corticosteroids is not desirable. The topical delivery of steroids to the "site of disease" is analogous to their use in asthma to minimise side effect with swallowed Fluticasone and viscous Budesonide used. There are recent trials on new swallowed dispersible formulations of steroids; these may become the mainstay with tolerability and ease of administration.

Swallowed fluticasone is via metered dose inhaler with ease of administration and high first pass hepatic metabolism, minimising side effect. Appropriate administration is essential: no spacer, swallowed (not inhaled) with oral intake avoided for 30 minutes post-swallow and mouth rinse at 30 minutes to reduce candidiasis. Swallowed fluticasone is established in children and adults (first published trial in 1998) with symptom and histological response. An adult series of 21 EE patients treated with swallowed fluticasone relieved dysphagia in all for at least four months. In our series of 26 adult patients, 19 received swallowed fluticasone with improved symptoms and histology in all.

Swallowed Budesonide is useful in those with difficulty using aerosolised medication delivery. In a paediatric publication, swallowed Budesonide 1-2mg in viscous sucralose solution in 20 EE patients was used for up to four months with 80% response (symptoms and histology) and no adverse effect. The same group published a controlled trial of 22 EE children with 87% response to Budesonide and no response in the placebo arm. Budesonide 2mg daily in an adult experience also produced histological remission in 61% of 18 patients.

Montelukast (Singulair) is a leukotriene D4 receptor antagonist paralysing eosinophil function. A small trial of Montelukast in 2003 provided symptomatic benefit. It should be reserved for the difficult case.

Future therapies

The future of EE therapy will be directed treatment using monoclonal antibodies. Il-5 is important in the eosinophil pathway for eosinophilic recruitment. Mepolizumab (anti-IL5 monoclonal humanised IgG antibody) is undergoing trials in hyper-eosinophilic syndromes including EE with benefit and no significant side effect.

Eosinophilic oesophagitis (EE) is a cause of dysphagia, chest pain and food bolus obstruction. There are characteristic endoscopic findings and defining histology. EE should be considered in adults with resistant reflux symptoms despite escalating P.P.I., food bolus impaction and atypical chest pain. GORD and EE may co-exist; reflux should be treated. Swallowed fluticasone is effective. We use swallowed Flixotide 250mcg for four-six weeks at two puffs BD and then decrease to lowest dose for symptom control. Swallowed Budesonide is an alternative. EE may require long-term preventative medication to maintain remission. Dietary therapy is important and should not be neglected. If associated difficult stricture, cautious dilatation has a role. Consideration can be given to immunology and allergist input in difficult cases. ■

Dr Matthew Remedios is a Gastroenterologist specialising in Interventional Endoscopy with a private practice based at the Wesley Hospital. He has a special interest in Barrett's endotherapy (endoscopic mucosal resection and HALO radiofrequency ablation), ERCP, small bowel balloon enteroscopy and large gastro-intestinal polyp resection.
Telephone: 07 3870 1722 or email remedios@wesley.com.au

The Wesley HOSPITAL

Breast Clinic

Wesley Breast Clinic
Urgent Referral Service

Doctor Hotline
07 3232 7085

Every year more than 23,000 patients attend The Wesley Breast Clinic for asymptomatic screening and diagnostics.

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FAST referrals	Referral form located under GP resources at ***www.wesley.com.au*** and fax **07 3217 8840**.
Practice enquiries	Call the Practice Manager on **07 3232 7067** or email ***Catherine.VanDongen@uchealth.com.au***
Patient bookings	Call **07 3232 7202** for a prompt appointment.