GastroIntestinal Endoscopy

newsletter the insider

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Celebrating 30 years

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Our Mission at GastroIntestinal Endoscopy is to deliver experienced and accessible endoscopy services with the highest quality of healthcare standards to improve the health outcomes of patients and the communities we serve.

GIE operates an Open Access Endoscopy Service from four locations:

- **SUNNYBANK** – Brisbane Endoscopy Services
- **CHERMSIDE** – Chermside Day Hospital
- **EVERTON PARK** – North West Private Hospital
- **AUCHENFLOWER** – The Wesley Hospital

Dr Roderick Roberts

Treatment of Eosinophilic Oesophagitis

There are three approaches to treatment of Eosinophilic Oesophagitis. These are dietary measures, pharmacological measures and where necessary, dilatation of the oesophagus.

Dietary Therapy

The dietary approach, with avoidance of particular food stuffs, seems more effective in children than in adults. The most commonly noted food triggers seem to be:

Milk	Wheat
Eggs	Peanuts/tree nuts
Soy	Fish/shellfish

From this is derived the classic six food elimination diet for the treatment of Eosinophilic Oesophagitis. While this approach seems more effective when applied in children, some motivated adults benefit from this dietary approach. Dietary treatment is, of course, appealing as it is non-pharmacologic. It is worth noting that some, patients will notice specific foods that regularly trigger symptoms and sometimes, just the avoidance of these foods is sufficient to contain symptoms.

Pharmacotherapy

The pharmacological approach involves the use of proton pump inhibitors, which are normally used to treat acid reflux but are effective in Eosinophilic Oesophagitis as well. This may be because GORD and Eosinophilic Oesophagitis seem to overlap in some individuals. In adults, the use of PPIs is often the most effective initial approach. This approach has appeal as we are generally familiar with this class of drug.

The second and complimentary pharmacological approach to treatment involves the use of swallowed glucocorticoid such as flixotide or budesonide. One common approach in adults is to use budesonide as respules using between two – four 0.5mg/2ml Pulmicort Respules mixed as a slurry with Sucralose (for example, Splenda). In adults, a typical dose would be 2mg (that is 4 Pulmicort Respules), mixed with 10 x 1g packets of Splenda in water, to make a slurry with a total volume of about 8–10mls. This slurry should then be administered twice per day. As an alternative to the Splenda slurry, a similar volume of honey can be used. Patients should not eat or drink for about 30 minutes after taking the Budesonide suspension. A typical course of this slurry might be over a 4–6 week time period.

Oesophageal Dilatation

Some patients require oesophageal dilatation, particularly where there is a significant component of fibrosis in the oesophagus, and where the glucocorticoids and proton pump inhibitors are not improving swallowing sufficiently. This is done at the time of endoscopy with a sequence of dilators of appropriate size.

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10 Minute Liver Consult: Jaundice

Dr Tony Rahman and Dr Samuel Chan , The Prince Charles Hospital

Jaundice (icterus) is the accumulation of bile pigments in serum and tissues including sclerae and skin and is usually clinically detectable once serum bilirubin exceeds 50µmol/l. In adults, jaundice serves as a marker for potentially serious haematologic or hepatobiliary dysfunction.

Physiology

Comprehending normal bilirubin metabolism is crucial to understanding the pathologic conditions that cause jaundice (Figure 1).

Causes of jaundice

The causes of jaundice are classically divided into pre-hepatic, hepatocellular and cholestatic causes, but overlap is common, especially in the critically ill.

Pre-hepatic jaundice

Pre-hepatic jaundice occurs when the liver's capacity to process bilirubin is exceeded. This is either related to excess breakdown of haem pigments in red cells or congenital abnormalities in the bilirubin conjugation pathway or bile salt export pump malfunction. Common causes of intravascular haemolysis include haemoglobinopathies, red cell membrane defects, microangiopathic haemolytic anaemia, drugs and sepsis. Because unconjugated bilirubin is not water soluble, it does not appear in the urine.

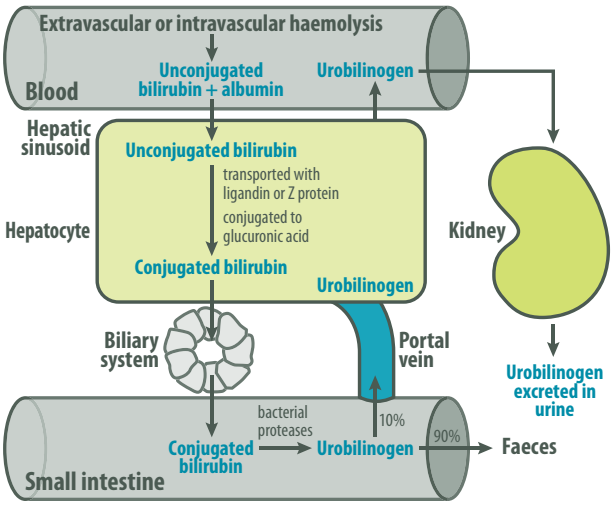

Figure 1: Summary of bilirubin metabolism

Gilbert's syndrome is a benign condition affecting 2–7% of the population and is characterized by mild unconjugated hyperbilirubinaemia in response to fasting or stress. Critical illness will almost inevitably precipitate hyperbilirubinaemia in patients with Gilbert's syndrome. Investigations including liver enzymes are normal. Other causes of congenital hyperbilirubinaemia including Dubin–Johnson, Rotor or Crigler–Najjar syndromes are rare. Mutations of the genes encoding the bile salt export pump underlying the familial intrahepatic cholestasis syndromes, which cause progressive cholestasis and liver damage, have been characterized.

Intrahepatic jaundice

Jaundice may be caused by hepatocellular dysfunction or intrahepatic cholestasis. Any cause of acute or chronic liver injury may cause jaundice. The most common causes in Australia are acute viral hepatitis and drug reactions. Acute hepatitis with renal failure may also complicate leptospirosis. Congestive hepatopathy occurs secondary to right heart failure or constrictive pericarditis. A salient cause of acute hepatocellular jaundice in ICU is following an episode of hypotension or cardiac dysrhythmia. Jaundice may also occur as a marker of sepsis in the critically ill patient.

Idiosyncratic drug reactions may be predominantly hepatocellular characterized by elevated transaminases (ALT and AST) or cholestatic (rise in alkaline phosphatase). Drug-induced liver injury is unpredictable and may be difficult to diagnose since there can be considerable latency between drug intake and the clinical presentation. Hepatocellular injury is defined as alanine transaminase (ALT) > 3 x upper limit of normal; cholestatic injury is defined as alkaline phosphate (ALP) 2 x upper limit of normal.

Extrahepatic jaundice

Extrahepatic jaundice occurs as a consequence of obstruction of the biliary tree distal to the biliary canaliculi. This may be secondary to gallstone disease, biliary strictures (benign or malignant), or extrinsic compression (pancreatitis or pancreatic carcinoma).

History and examination

Jaundice is not a diagnosis but a physical manifestation of elevated serum bilirubin. The jaundiced patient often presents with a related symptom such as abdominal pain, pruritus, vomiting or substance ingestion. The cause for jaundice must be sought. In hepatic and post-hepatic causes of jaundice, enterohepatic circulation of bile products is interrupted; hence stools will be pale whilst the urine is dark secondary to conjugated bilirubin. Right upper quadrant pain suggests either biliary obstruction or liver capsular stretching. Biliary obstruction may be complicated by cholangitis with fevers, rigors and features of sepsis. A history of biliary surgery or trauma may be apparent.

Risk factors for acute viral hepatitis should be sought. A full drug history including the use of over-the-counter, herbal or Chinese remedies, and recreational drug use is important.

Stigmata of chronic liver disease may be observed. Hepatomegaly is uncommon in chronic liver disease and suggests hepatic congestion, or infiltration. Splenomegaly suggests long-standing liver disease complicated by portal hypertension. A palpable gallbladder is suggestive of malignant biliary obstruction.

Investigations

Blood tests

Liver function tests confirm the diagnosis of jaundice and may differentiate between an obstructive and hepatocellular cause. Tests of liver synthetic function (albumin, prothrombin time) are important to stratify the severity of the liver injury. Deficiency of fat-soluble vitamins,

including vitamin K, is common in cholestasis, so clotting may be deranged in the absence of significant synthetic dysfunction. Serology for viruses (hepatitis A, B and C, EBV and CMV) should be checked.

Imaging

In the normal patient population, ultrasound is a sensitive method of diagnosing biliary obstruction, with a diagnostic accuracy of up to 79% for common bile duct stones. The technique is less accurate in the critically ill where biliary dyskinesia is common, but it is portable, non-invasive and does not require IV contrast, making it a suitable first-line investigation. CT imaging may diagnose the presence and level of biliary obstruction and is better at visualizing the pancreas than ultrasound. Magnetic resonance cholangiopancreatography (MRCP) has impressive accuracy for the diagnosis of biliary tract stones but is of limited applicability in the critically ill.

Treatment

In pre-hepatic jaundice the underlying cause should be identified and treated appropriately. For intrahepatic causes of jaundice, care is supportive with treatment of the underlying condition.

Drug reactions

All drugs with the potential to cause jaundice or hepatotoxicity should be withdrawn. In patients on multiple medications, this may be difficult, but efforts should be made to stop as many drugs as possible or substitute for less hepatotoxic agents. In most cases of drug-induced hepatitis, the AST falls by 50% within eight days of stopping the culprit drug, but liver injury may worsen or follow a protracted course. In cholestatic dug injury, it may take several months for LFTs to normalize. Rechallenge should not be performed. In cases of potential adverse effects of ceasing a drug, expert advice should be sought.

Extrahepatic jaundice

Itching is common in obstructive jaundice. Troublesome symptoms may respond to oral antihistamines or cholestyramine (4g tds).

Sepsis is common in biliary obstruction, and broad-spectrum antibiotics with Gram-negative cover should be given. Coagulopathy corrects with parenteral vitamin K.

Decompression and drainage of the biliary tree is a priority in extrahepatic biliary obstruction. For patients who are stable, ERCP performed under sedation will allow dilatation and stenting of strictures and diagnostic cytology. Patients who are too unstable to tolerate ERCP should be referred for radiological percutaneous biliary drainage, with a view to definitive drainage and internalization once their clinical condition has improved.

Further reading

Brienza N, Dalfino L, Cinnella G, *et al*. Jaundice in critical illness: promoting factors of a concealed reality. *Intensive Care Med* 2006; 32: 267–74.

Williams EJ, Taylor S, Fairclough P, *et al*. Are we meeting the standards set for endoscopy? Results of a large-scale prospective survey of endoscopic retrograde cholangiopancreatograph practice. *Gut* 2007; 56: 821–9.

Kaplan MM. Clinical aspects of serum bilirubin determination. Up To Date, 2015 (Review).

Treatment of Eosinophilic Oesophagitis

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Maintenance

Most patients should consider taking some long term maintenance therapy; particularly where dysphagia is a predominant symptom or food impaction is occurring intermittently. If single dietary triggers can be identified, then these should be avoided. Proton pump inhibitors can be taken long term. Additionally a lower dose of glucocorticoid (for example 1 mg of Budesonide as Pulmicort Respules) can be used daily.

It is important to remember we are still in a learning phase when dealing with this condition which seems to be increasing in incidence across all age groups and both sexes. We are learning about atypical presentations of Eosinophilic Oesophagitis, and we are learning more about the varied, natural history of the condition. Undoubtedly, more research will lead to a better definition, and a more tailored approach to the treatment of Eosinophilic Oesophagitis in coming years. Hopefully we can learn to avoid the acute presentation with bolus obstruction of the oesophagus and mitigate the need for oesophageal dilatation.

Joining GIE...

GastroIntestinal Endoscopy is delighted to welcome three gastroenterologists to the team. These experienced gastroenterologists will provide regular relief of open access endoscopy lists at GIE locations across Brisbane.

Please address your patient referrals to GastroIntestinal Endoscopy for rapid access to experienced, efficient and accessible endoscopy services.

DR GEORGIA HUME
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DR TONY RAHMAN
MA DIC PHD FRCP FFICM FRACP

DR RUTH HODGSON
BA (HONS) MA MBBS MRCP FRACP (OXON)

Frequently Asked Questions Dr Neville Sandford

Q Do you routinely tattoo lesions that can't be removed or are planned for surgery?

A Polyps which are suspicious of malignancy, require close follow-up or cannot be resected colonoscopically and will require surgery are usually tattooed unless they are in an anatomical position which is easily identified (e.g. caecum or rectum). This is particularly relevant for laparoscopic surgery.

Q What is the therapeutic approach to active bleeding at colonoscopy?

A There are a number of options available. If bleeding occurs during polypectomy, active pressure with forceps is sometimes all that is required. If bleeding continues, injection of adrenalin or electrocautery can be used. Haemoclips are now the main therapeutic approach to either prevent or stop active bleeding. Argon Plasma Coagulation is used for treatment of bleeding angiodysplastic lesions and also for bleeding after endoscopic mucosal resection which can also be controlled with haemostatic forceps. Electrocautery is used less for resection of small polyps as it increases the risk of secondary bleeding. Endoloops are used less frequently now. In most cases the bleeding can be stopped at colonoscopy and only very rarely do we have to resort to radiologic or surgical options.

Q Do patients with Coeliac disease or suspected Coeliac disease need to cease a gluten free diet prior to endoscopy?

A Undiagnosed patients with suspected Coeliac disease in whom a firm diagnosis is required ideally should be on a diet containing gluten for six weeks before an endoscopy. Once Coeliac disease is diagnosed they should remain on a GFD indefinitely.

Q Do patients presenting for upper endoscopy have to cease their PPI's for two weeks prior to their procedure?

A This depends on the clinical situation. Patients on PPIs should not routinely discontinue their medication prior to endoscopy but it may result in false negative Urease tests for Helicobacter detection. If a patient is being investigated for causes of dyspepsia and has not previously had an endoscopy, it would be quite reasonable to cease the PPIs so that the presence of Helicobacter can be accurately assessed, but in a patient in whom an endoscopy is being done for follow-up of a previously diagnosed condition (eg GORD), PPIs should be continued.

If you require A5 referral pads, please contact one of our four locations below. Electronic referral templates can be downloaded from our website www.gastros.com.au

GIE practice locations and contact details For all appointments, call 1300 4 GASTRO (1300 442 787)

Brisbane Endoscopy Services

Suites 16–18
McCullough Centre
259 McCullough Street
Sunnybank QLD 4109

Phone: 07 3344 1844
Fax: 07 3344 2739

Chermside Day Hospital

Level 1
Chermside Medical Complex
956 Gympie Road
Chermside QLD 4032

Phone: 07 3120 3407
Fax: 07 3120 3443

The Wesley Hospital

3rd Floor, East Wing
451 Coronation Drive
Auchenflower
QLD 4066

Phone: 07 3870 3799
Fax: 07 3870 5069

North West Private Hospital

Endoscopy Unit
137 Flockton Street
Everton Park
QLD 4053

Phone: 07 3353 3322
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