Gastroenterology Update

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Gastroenterology has progressed enormously in the past 25 years. Hirschowitz pioneered the use of the fibreoptic endoscope and used it on a patient at the Michigan University Hospital. Since then, the endoscope has become firmly established as a diagnostic instrument for both upper and lower digestive tract disorders.

This increase in its use has been established due to the ability to obtain a tissue diagnosis by multiple target biopsy, cytology and the ability to measure and keep records of a suspected lesion. The growing number of endoscopes has made endoscopy exploration possible into virtually every part of the bowel.

The diagnostic role of the endoscope has, however, widened to include a therapeutic function whereby the endoscope can be employed as a delivery system for various devices such as snares, (Polypectomy), grasping devices, (to remove foreign bodies), flexible fibres, (laser photocoagulation), papillotomes, (for papillotomy) and by the use of the above, treatment may follow endoscopic diagnosis.

Upper GI endoscopy: (Oesophagus)

One of the problems encountered in assessing oesophageal pathology is the poor correlation between oesophageal symptoms and endoscopic evidence (visual and histological) of disease.

Severe symptoms may be present with normal or only mildly abnormal mucosa and vice versa. Histology will allow a diagnosis of oesophagitis in early disease where the mucosa appears endoscopically normal.

Fibreoptic endoscopies are also used today in preference to the more dangerous rigid oesophagoscope, thereby eliminating the need for anaesthesia and allowing examination of the cardia from below. The endoscope is also being used to dilate strictures, both benign and malignant and to place an oesophageal tube in position.

Sclerotherapy for oesophageal varices is now being widely practised, making use of the fibreoptic endoscope with an overtube. A lot of pioneering work has been done by Prof Terblanche of Cape Town.

Endoscopic sclerotherapy may be used in acute variceal haemorrhage and to completely obliterate the varices - in so doing, to reduce the number of bleeding episodes. Terblanche et al (1979a) controlled bleeding in over 90% of hospital admissions.

Stomach

Recent advances in endoscopic examination of the stomach includes its wider effective use in X-ray-negative dyspepsia, and the increased detection of early gastric cancer.

Emergency endoscopy in acute upper GI haemorrhage is controversial. However, the majority of people feel that endoscopic assessment should be performed within 24 hours to ascertain the bleeding site with certainty.

In particular, erosive gastritis, Mallory Wise tears and bleeding in patients with portal hypertension are best evaluated by endoscopic means. Current majority view is that a more accurate diagnosis should be achieved, as with time, improved management will improve mortality.

Endoscopic control of alimentary bleeding is now a reality and thermal techniques are being implemented. The technique showing the most promise is laser photocoagulation, employing either argon ion or Neodymium YAG Laser. Uncontrolled clinical studies employed ND YAG laser have reported 93% success in 106 cases of alimentary bleeding including 39 cases of varices with two perforations.

ERCP

This is a widely accepted diagnostic tool in patients with jaundice or pancreatic disease.

In hepatic disorders, it is being used to assess the cause of obstructive jaundice and to ascertain the presence or absence of common duct stones.

It is also helpful in diagnosing periampullary carcinoma, cholangiocarcinoma and post-operative strictures. Following a diagnosis of retained common duct stones, endoscopic papillotomy may be performed, using the flexible papillotome to excise the papillary sphincter.

This is then followed by spontaneous passage of stones in 80% of cases. Occassionally a Dormia basket can be used to remove the stones.

In malignant strictures of the CBD a draining catheter can be left in situ to decompress the biliary system.

Pancreatic diseases

ERCP continues to be of assistance in diagnosing chronic pancreatitis, pancreas divisum (congenital duct anomalies) and along with ancillary aids like CT scan and ultrasound in diagnosing ca. pancreas.

The initial enthusiasm for its use in diagnosing ca. pancreas has however, been dampened by experience.

Colonoscopy and Flexible Sigmoidoscopy

The colonoscopy has had a profound effect on the diagnosis and management of colonic diseases.

Rectal bleeding:

50% of X-ray negative bleeds can be diagnosed by colonoscopy. Overall, 267 of 578 patients with barium-negative rectal bleeding were diagnosed by colonoscopy, i.e. 46% out of these 77 (13%) were carcinomas.

Colonoscopy in diverticular disease is valuable in concomitant cancers which are often difficult to confirm or exclude.

In one series of 22 patients, suspected of having concomitant sigmoid cancer, only ten were shown by colonoscopy to have malignant disease, whereas two infiltrating ca's were discovered in patients with diverticular disease with no radiological suspicion of malignancy.

In inflammatory bowel disease, colonoscopy is valuable in assessing disease severity plus extent. Also using the criteria of Morson for dysplasia, patients with pancolitis of more than 7 - 10 years duration, are being followed with

annual or biannual colonoscopies to assess their risk of developing malignancy, and thus allowing prophylactic surgery to take place before overt cancer develops.

Colonic Polyps

Colonoscopy has established a major role in the therapeutic removal of malignant or potentially malignant adenomatous polyps.

In one study of 239 barium-negative patients with persistent rectal bleeding, 39/95 patients with endoscopically proven lesions, had polyps.

Although total colonoscopy is desirable from the point of view of screening for further polyps, the new flexible 60cm. fibreoptic sigmoidoscope has much to commend it. These instruments can be used in the consulting room after administering a simple fleet enema, and with a lot of practice, can be passed to 60cm in a few minutes.

They can be used to perform polypectomies, and to diagnose lesions often up to splenic flexure, and as the majority of colonic cancers occur on the left side, the diagnostic yield is high.

Peptic ulceration

Two recent developments have greatly increased our knowledge about the course of peptic ulcer disease. Firstly, fibreoptic endoscopy has improved documentation of ulcer craters, and secondly, many controlled clinical trials have been performed using endoscopy.

The cause of gastric and duodenal ulcer has still not been established with certainty, and although they may have different causes, both require the presence of acid (and possibly pepsin) for development and perpetuation.

Therapy of ulcer is directed at either reducing acidity or enhancing mucosal resistance.

Suggested management of Peptic Ulcers

The decision of whether to use Cimetidine, antacids, Sucralfate or bismuth preparations, as primary Rx for peptic ulcer, is predictable primarily on considerations such as cost, side-effects and patient compliance.

The cost of a course of Cimetidine is about the same as a course of a large dose of potent antacid regimen.

Based on available data, I recommend that duodenal ulcer may be treated with either Cimetidine, Sucralfate or antacids in adequate dose (±230ml./24 hours).

Recent evidence indicates that a bedtime dose of anacid is far less effective than Cimetidine in reducing nocturnal acidity.

Therefore, if a patient experiences night-time ulcer pain, consideration should be given to substituting a nocturnal Cimetidine dose for the antacid dose.

Treatment should continue for four to six weeks. If at that time the patient is symptom-free, therapy may be discontinued. If the patient is still symptomatic, then a combination regimen of Cimetidine/antacid or Sucralfate/Cimetidine is in order for several weeks.

If serious symptoms persist, consider surgery in those patients with a long history of ulcer disease. Before this however, the physician must be sure that the diagnosis is correct and medication is being taken regularly, and all aggravating factors are avoided.

Endoscopy is reserved for patients not responding to Rx or where surgery is contemplated, to ensure symptoms are ulcer-related. Endoscopy, to confirm ulcer-healing is apparently unwarranted.

Gastric ulcer may be treated with either antacid, Sucralfate or Cimetidine. The dose of a potent antacid has been shown to compare with Cimetidine in lowering gastric acidity if given one to three hours after meals and at bedtime. Whether all G.U's should be endoscopies, is only a problem if expert endoscopy and radiology are both available, otherwise endoscopy is preferred.

Once treatment is instituted, progress can be monitored endoscopically (or radiologically) if endoscopy is not available).

Rx should continue to healing which should be within 12 weeks. If the ulcer enlarges at six weeks or fails to heal at 12 weeks, re-endoscopy and biopsy to exclude malignancy, is indicated.

If the ulcer is still considered benign; then one of the two approaches may be considered.

Firstly combination Rx on the premise that previous Rx was inadequate. Secondly, surgery may be contemplated in chronic ulcers.

Infectious Diarrhoea

A patient probably has an infectious form of diarrhoea if he complains of an illness of acute onset that consists of four or more unformed bowel movements in twenty-four hours, together with at least one associated symptom, such as fever, dehydration, moderate to severe nausea, vomiting or abdominal discomfort.

Partial immunity-inoculum size, or the ingestion of large quantities of classically non-pathogenic bacteria, all play a role in dictating the clinical outcome.

An important enquiry in the patient with acute diarrhoea depends on whether or not antibiotics have been taken.

The onset of antibiotic associated Colitis (AAC) may occur many days following cessation of antibiotics.

The syndrome may be marked by fever and profuse diarrhoea with or without bloody stools.

A polymorphonuclear leucocytic exudative response in the stool, is common but may be absent especially early in the course.

It is now accepted that therapy with most antibiotics can be associated with Colitis including the Penicillins, Cephalosporins, Lincomycin/Clindamycins, Tetracyclines, Trimethoprim - Sulphamethoxozate and Choramphenicol. Relative to the amount of prescribed antibiotics, Lincomycins carry the greatest risk, but this is still well below 1 - 2% and nowhere near the frequency described earlier.

Current indications are that most AAC is mediated by an Exotoxin elaborated by **Cl. Difficule,** i.e. PMC. The presence of Exotoxin in stool can be assessed by tissue culture. A crude stool filtrate will cause a cytopathologic effect in the LA cells.

This effect can be neutralized by Gasgangren Antitoxin containing antibody to **CL. Sordelli** which cross-reacts with antibody to **CL. Difficule** toxin.

The diagnosis of PMC is difficult. The principal method of identifying the disorder is by sigmoidoscopic appearances, histology or by demonstrating toxin in faeces.

The typical sigmoidoscopic appearances are of multiple white plaques adhering to the mucosa. The intervening mucosa is normal, and there is no blood or purulent material in the lumen.

The biopsy result is not diagnostic and can be difficult to distinguish from ischaemic colitis.

The principal clinical presentation is diarrhoea, usually watery with excessive mucosa, unassociated with bleeding. Fever is present in 67% of cases.

The treatment of confirmed cases of PMC is Vancomycin orally 250mg. six hourly by 7 - 14 days. A few resistant cases have been treated with Bacitracin.

Vancomycin for ten days costs in the region of R800.

Non "A" non "B" Hepatitis

The common types of virus hepatitis can now be identified. Hepatitis "A" is diagnosed by Serum IgM antibody which is detected in the acute attack and for the next few weeks.

Hepatitis "B" is diagnosed by the Hepatitis "B" surface antigen (H B s A g.)

Cytomegalovirus and Epstein Barr infections are recognised by standard techniques.

There remains a large number of patients with virus hepatitis where the cause is unknown.

These have been designated as due to Non "A" Non "B" viruses. The agents have not been identified although various candidates have been postulated. Non "A" Non "B" hepatitis represents disease due to more than one agent. Non "A" Non "B" hepatitis is particularly transmitted by blood and blood products.

Spread of Non "A" Non "B" Hepatitis

Blood and its products; Transfusion; Factors VIII and IX; Renal Units and Transplants; Needle pricks; Waterborne (India) - may account for 89% of post-transfusion Hepatitis occuring in areas where routine screening of donors has virtually eliminated Hepatitis "B".

The spread of Non "A" Non "B" hepatitis resembles hepatitis "B" more than hepatitis "A". However, in addition to spread related to parenteral innoculations and to blood products Non "A" Non "B" Hepatitis may be responsible for 25% of sporadic cases of virus Hepatitis.

The nature of the infecting agent

Probably more than one agent is involved. There seems to be at least two types designated long and short incubation types.

INCUBATION PERIOD

	(Antigen)	(Incubation)	(Chronicity)
A long incubation	present	weeks	%
		7 - 10	10
B Short	No	1 - 3	50 -90%
C Waterborne (Kasemir India)	No	3 - 4	0

The clinical picture of Non "A" Non "B" Hepatitis

Only 25% of cases become jaundiced. Initial symptoms include lethargy, nausea, rarely vomiting, abdominal discomfort, and finally jaundice. Many patients are virtually asymptomatic, the diagnosis being made on blood tests.

Colon cancer

No other aspect has been the subject of such a major promising and truly international research effort in the past three years.

The position can probably be summarised by saying that the epidemiological evidence points to an environmental cause for colon cancer, but the specific causal agent has not been identified. Diet appears to play a major role in promoting tumour-formation.

Study of the incidence of bowel cancer shows a 10x variation on occurrence. It is common in the USA (particularly Connecticut) New Zealand, Scotland and Denmark, while rare in Nigeria and Brazil.

Migrants from low incidence areas (Japan) to high incidence areas (USA) acquire the local bowel cancer rate after one to two generations suggesting an environmental cause.

Rectal and recto-sigmoid cancer account for about 35% of total large bowel cancer and they become more common with advancing age.

Certain ethnic groups like the Jews and Parsi (of Bombay) have a high risk while the 7th Day Adventists of the USA have lower risk.

Ischaemic disease of the colon Principal causes

- Surgical interruption of blood supply.
- Radiological injury.
- Spontaneous thrombosis of colonic vessels.
- Small vessel disease.
- Low flow states.
- Obstruction.
- Venous disease.
- Infarction of unknown origin.

Two principal pictures emerge

Gangrene of the colon: The patient is usually a middle-aged or elderly patient with a background of degenerative cardiovascular disease, such as diabetes etc., with episodes of LV failure or myocardial infarction.

The onset of the illness is sudden and dramatic, with severe generalised abdominal pain which is at first colicky, but then becomes continuous. Vomiting and diarrhoea is frequent, early on.

The clinical picture then progressively gives way to one of abdominal distension, thirst, restlessness and circulatory collapse. Abdominal examination reveals signs of peritonitis with diffuse tenderness and absent bowel sounds. Rectal examination may reveal dark blood.

Ischaemic colitis: This is a non-gangrenous form of the disease which varies from a transient episode of inflammation to more severe disease which gives rise to a fibrous stricture.

One of three things may happen when the recommended conservative regimen is followed, (rest, fluids and antibiotics).

- Progression to gangrene.
- Resolution.
- Stricture formation (50%).

Crohn's Disease

The group of patients described by Crohn et al (1932) had lesions of the terminal ileum, ie. regional ileitis. However, even in the 1930's it became apparent that other parts of the bowel could be affected, especially when the "skip" lesions were recognised. Nevertheless, the disease appeared to affect the small intestine predominantly with colonic sparing.

However, nowadays, the majority of newly diagonised cases will have ileocolitis in continuity. The next largest group are those with SB involvement: However, 25,6% of cases seen in Radcliff have colonic involvement alone.

The apparent decline of cases confined to SB can partially be due to the discovery that many cases of acute ileitis formerly regarded as due to Crohn's disease, are in reality due to an acute infection, particularly with **Yersinia Enterocolotica.**

The apparent increase in colonic Crohn's disease can be explained by greater awareness of the condition, coupled with improved diagnosis especially in relation to distinguishing it from UC.

The progressive nature of Crohn's disease and its ability to recur after resectional surgery, has made the surgeon more conservative, especially since it is now felt that there is a widespread abnormality of the GIT in Crohn's disease. □