SCLERODERMA – GASTROINTESTINAL DISEASE

FOR PATIENTS – HAMILTON 2015

Canadian Scleroderma Research Group
Groupe de recherche canadien sur la sclérodermie
WE WILL DISCUSS...

How each part of the GI tract is affected
What symptoms you may experience
How we might measure the severity of GI disease
Nutritional aspects
Treatment
Involvement of the gut occurs with equal frequency among patients with the diffuse (dcSSc) and limited subtypes of SSc (lcSSc)

GI involvement is the leading cause of morbidity and the third most common cause of mortality in patients with SSc
MOUTH INVOLVEMENT

SSc may affect the oral and perioral tissues. Can lead to rigidity of the facial skin and tongue, and result in impaired chewing.

These functions can be further compromised when sicca syndrome (dry eyes/dry mouth) and SSc coexist, since saliva production is severely reduced with sicca syndrome.
26% of patients showed swallowing dysfunction, (e.g., oral leakage, retention, penetration, mild or moderate aspiration, and upper esophageal sphincter incoordination).

These dysfunctions were more severe in patients with prominent esophageal dysmotility which is lack of coordination of the muscles of the esophagus.

Oropharyngeal and esophageal function in scleroderma. AU Montesi A; Pesaresi A; Cavalli ML; Ripa G; Candela M; Gabrielli A SO. Dysphagia 1991;6(4):219-23.
This may lead to oral leakage, retention of food in mouth, and aspiration (food going into lungs), and result in a sense of food sticking in the throat, food coming up through nose and coughing after swallowing.

Patients develop narrowing of the oral aperture
SJOGREN’S SYNDROME

Dry eyes
Dry mouth
Specific auto-antibodies
  • SSa, SSb
Antibodies in about 15% of our patients
Overlap with other auto-antibodies
SS can occur alone or accompany several rheumatic diseases
Infiltrate of white blood cells in salivary glands
Important clue to concept of poly-autoimmunity?
ESOPHAGUS

The earliest internal manifestation to be described was esophageal disease, which remains the most common source of GI symptoms in scleroderma.

Symptoms may be produced by gastroesophageal reflux, stricture formation, and abnormal motility.

Up to 90% of patients
Food gets into your stomach even if you are lying down – not gravity!
Gastroesophageal Reflux
SYMPTOMS

Food sticks on way down
Heartburn
pain
STRICTURE !!

Narrowing of the esophagus
DIAGNOSIS

Symptoms
Manometry
Radionuclide transit studies
Barium studies with ciné radiography
gastroscopy
MANAGEMENT

Some drugs like motilium may help the muscle action of the esophagus and move food and acid down to the stomach.

The main treatment is to prevent acid from coming into esophagus.

Raise head of bed.

Use drugs that decrease stomach acid.
If a stricture forms then it has to be dilated mechanically.
STOMACH INVOLVEMENT

Stomach emptying is delayed

Early satiety – feel full quickly while eating

Rarely, severe involvement of the stomach results in gastroparesis (paralysis of stomach) with intractable vomiting, which may cause weight loss and nutritional deficiencies.
Peristalsis is lost in scleroderma
GASTRIC ANTRAL VENOUS ECTASIA (GAVE)

Massive upper gastrointestinal bleeding
an increasingly recognized complication of SSc

Watermelon stomach
the most sensitive method of detecting stomach involvement is via radionuclide gastric emptying.

In addition, a barium meal may reveal a dilated stomach that does not contract.

gastroscopy may show a lack of peristalsis or GAVE.
MANAGEMENT

Small but more frequent meals

Can try medications that increase the muscle action in stomach eg erythromycin

Nutritional supplements
SMALL INTESTINAL INVOLVEMENT

Abnormal small bowel function has been reported in 20 to 60 percent of patients with SSc underlying neuromuscular disorder similar to that in the esophagus

Decreased muscle action

Leads to growth of bacteria
Dilatation of intestinal loops is the most prominent radiographic feature when absence of peristalsis affects the duodenum and proximal jejunum.
MALABSORPTION

Approximately 10 to 30 percent of patients with SSc have evidence of malabsorption.

Intestinal stasis with overgrowth of bacteria is considered to be the major cause of this complication.

One study, for example, performed jejunal aspirates in 20 unselected patients with SSc; bacterial overgrowth was present in one-third...
CLINICAL MANIFESTATIONS

The major manifestations of small intestinal involvement are due to reduced muscle contraction with resulting stasis and intestinal dilatation.

This results in abdominal distension and pain arising from dilated bowel loops.
Bacterial overgrowth subsequently emerges due to intestinal stasis and pooling.

As fat malabsorption ensues, the patient's symptoms may change from distension, pain, bloating, and constipation to diarrhea, steatorrhea, and weight loss.
A rare complication is intestinal pseudo-obstruction, resulting in recurrent obstructive symptoms

Affected patients typically complain of an alteration in bowel habit with diarrhea, constipation, or both (intermittently).
DETECTION OF BACTERIAL OVERGROWTH

The gold standard for the detection of bacterial overgrowth is small bowel aspiration.

the glucose hydrogen breath test is a noninvasive examination
It appears to be difficult to improve the muscle action of the small bowel.

A drug called somatostatin (octreotide) may help – it improves muscle action and decreases bacterial overgrowth.

Bacterial overgrowth is treated with antibiotics.

Rarely intravenous feeding.
COLON AND ANORECTAL INVOLVEMENT

Colonic disease occurs in 10 to 50 percent of patients with SSC, with the anorectum being the most frequently affected area.

A recent study, for example, found that the colon is almost as frequently involved as the esophagus; in addition, patients with abnormal esophageal manometry (a test of muscle action) almost always had abnormal anorectal motility.
Pneumatosis Intestinalis

gas in the bowel wall
Constipation (less than two spontaneous stools per week) and fecal incontinence are common problems in SSc. Disordered anorectal function is also an early finding in SSc, and is a major factor in the development of fecal incontinence.
MANAGEMENT

Prevent constipation

Fiber supplementation can improve symptoms in patients with constipation.

bulk-forming laxatives such as psyllium or methylcellulose are the most physiologic and effective approach to therapy

Stimulant laxatives such as bisacodyl (eg, some forms of Dulcolax), senna (eg, Senokot), and sodium picosulfate (eg, Dulcolax drops)
New drugs eg Linaclotide, Prucalopride
Although diarrhea is principally due to small bowel bacterial overgrowth, its presence may contribute significantly to the development of fecal incontinence.

As a result, incontinence associated with diarrhea should be treated with antibiotics in combination with nonspecific measures including a low-residue diet, antidiarrheal agents.
ELECTRIC SACRAL NERVE STIMULATION FOR INCONTINENCE
THANK YOU FOR YOUR ATTENTION...