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Collagenous colitis

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COLIAGENOUS COLITIS

Pages with reference to book, From 227 To 229

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The collagenous colitis was first described by Lindstrom in 1976, in a 48 year old lady, who presented with cramps, abdominal pain and watery diarrhoea for 2-1/2 year. Her rectal biopsy showed a thickened layer of sub-epithelial collagen. Now this entity is being increasingly recognized as a cause of persistent watery diarrhoea Routine colonic biopsy despite endoscopically normal mucosa and increasing awareness of this condilion would help in the detection of this disease entity.

CASE REPORT

65 year old lady presented with intermittent watery diarrhoea for the last 4 years. The bowels were open 5-6 times a day and were associated with urgency and episodes of incontinence. Antacids would aggravate the symptoms. There was no complaint of passing any mucous or blood in stools. She was operated for thyrotoxicosis in 1968 and now she was on thyroxine replacement. Her physical examination was unremarkable. Serum biochemistry and haematology were normal, so was the thyroid stimulating hormone level. Stool examinations were negative for any mucous, blood, pus or parasites. Bacterial cultures were also negative. Sigmoidoscopy showed normal looking mucosa. Biopsies were taken. There were focal degenerative changes in the surface epithelium with inflammatory cell infiltration. The sub-epithelial region showed a distinct collagen band. (Figure).

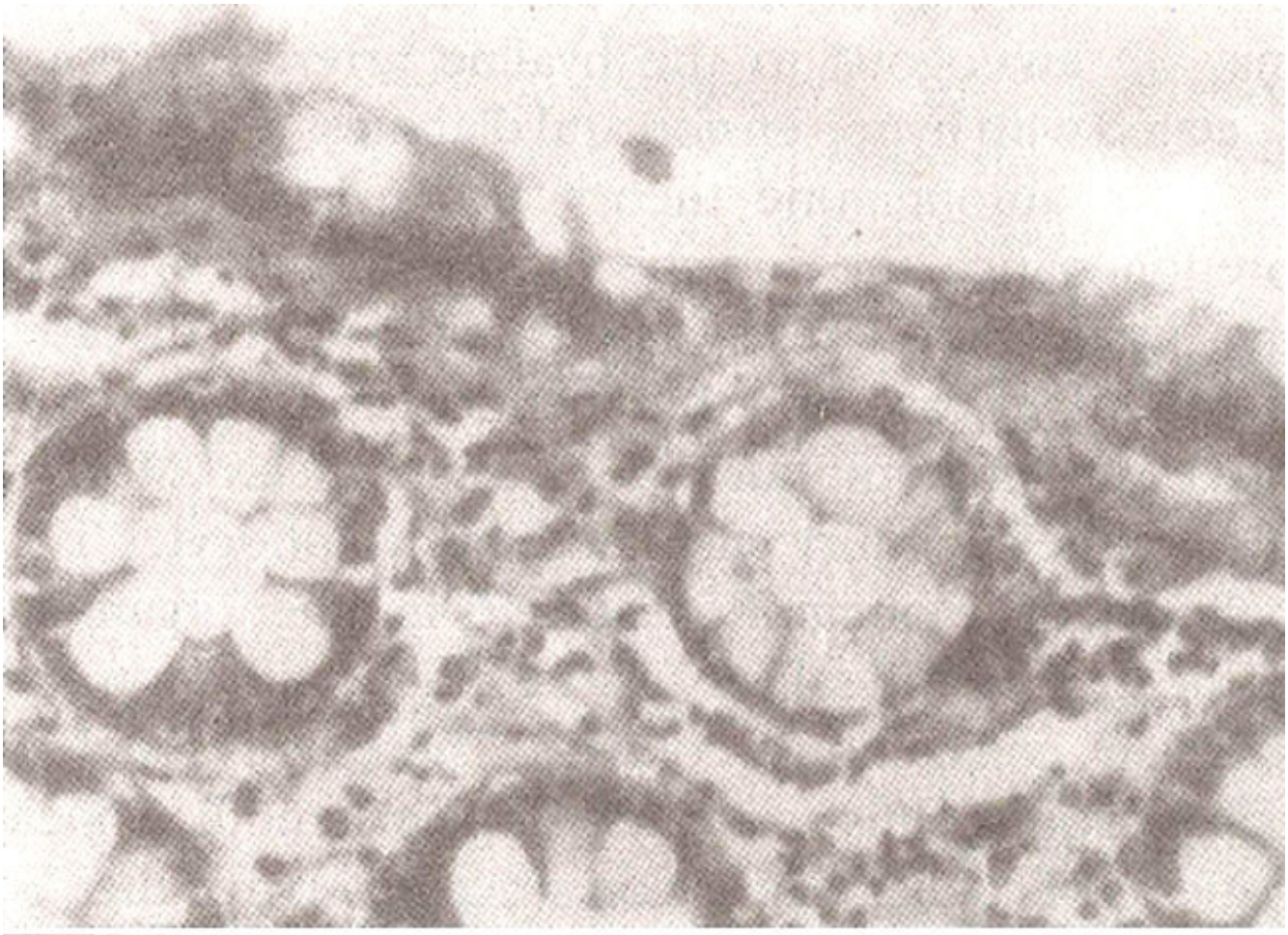

Figure showing dark sub-epithelial collagen band.

The lamina propria was infiltrated with moderately increased chronic inflammatory cells. Tubular glands did not show any mucin depletion or distortion. She was put on lspaghula husk (Psyllium) and loperamide. Patient improved symptomatically and bowel movements decreased to two stools per day. She is being followed in gastrointestinal clinic.

DISCUSSION

Collagenous colitis usually presents as chronic watery diarrhoea and predominantly involves middle aged and elderly women[2,3], though no age appears immune and disease has also been reported in children[4,5].

The hallmark of the disease is broad collagen band immediately subjacent to the surface epithelium. This band characteristically spares the crypts and is confined to the intercryptal area. There is moderate lymphocyte and plasma cell infiltration and patchy surface epithelial injury without ulceration or erosion[6]. Normal thickness of sub-epithelial collagen table has been reported 0.4 4.6 um by Van Den Oord et al[7] and 4.6 - 6.0 um by Bogomeletz and colleagues[8]. In collagenous colitis it exceeds 15 um and is usually 30-70 um in thickness[6]. Gledhill and Cole have shown that thickness wider than 15 um correlates strongly with watery diarrhoea[9].

The cause of collagenous colitis is debated. An inflammatory cause has been suggested and apparent transition from inflammatory to collagenous colitis have been reported[3]. Microscopic colitis and

collagenous colitis might be variants of the same disease[10]. The collagenous colitis may represent end stage of microscopic colitis[11]. The superficial inflammatory reaction may be seen involving the surface epithelium in active disease both before and after development of collagen band[12]. Increased number of mast cell reported, might have a role in providing sub-epithelial fibrosis by releasing mediators[13,14]. Normal crypts are surrounded by. a thin collagenous layer that is synthetized by the pericryptal fibroblast sheath. A functional disturbance of this apparatus could be the cause of excessive collagen deposit[13]. Kingham et al have postulated three mechanisms leading to collagen deposition[3]: (a) reduction in the turnover of fibroblasts with longer life span and more collagen production; (b) the band may represent a "healed" area of excessive scaring following previous inflammation and ulceration; (c) the process may be analogous to the hyaline arteriosclerosis associated with hypertension and diabetes.
An autoimmune theory also exists. The female predominance, the non-specific abnormality of connective tissue blood tests, associated thyroid disease as found in our case, and therapeutic response to steroids make auto-immunity an attractive hypothesis2. Moreover the epithelial injury mediated by lymphocytes is the hallmark of autoimmune disorders in other organs such as the thyroid and salivary glands. Such infiltration is also seen in collagenous colitis[14]. There are reports of collagenous colitis as a cause of persistent diarrhoea in patients of coeliac disease on strict gluten free diet. So it is unlikely that gluten toxicity causes collagen deposit in colon as it does in jejunum[16,17]. Whether any other dietary factor is involved, is not known[18].
What is the pathophysiology of diarrhoea? It seems that in the collagenous colitis normal colonic secretory process of chlorides and fluid by the undamaged crypts continues while the absorption of sodium and fluids by the damaged surface epithelium is decreased[19]. Increased levels of prostaglandin E2 have been found in stools[19] and jejunal aspirates[20]. Prostaglandins produced. by the damaged colon might be a pathogenitic mechanism of diarrhoea[12,19]. These may also circulate systemically, secondarily influencing fluid balance in the small bowel[2]. As the disease progresses, the collagen thickness increases and it may act as a diffusion barrier which contributes further to diarrhoea[12].
The course of disease is variable. It may persist as chronic diarrhoea, or may have relapsing and remitting course with variable response to treatment[21] Spontaneous resolution may occur and the collagen band may disappear[22], or persist[20]. Treatment with sulphasalazine has caused disappearance of band in some cases[22]while others showed favourable response only after steroids were introduced[5,23] but all remedial measures may fail[3]. Our patient responded to symptomatic treatment with psyllium and loperamide. So it may be worthwhile to try these measures before introducing anti-inflammatory drugs.

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